

ORAL ARGUMENT NOT YET SCHEDULED**IN THE UNITED STATES COURT OF APPEALS
FOR THE DISTRICT OF COLUMBIA CIRCUIT**

No. 17-1172

AMERICAN LUNG ASSOCIATION, *et al.*,
Petitioner,

v.

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY, *et al.*,
Respondents.

Petition for Review of Final Administrative Actions of the
United States Environmental Protection Agency

**MOTION FOR SUMMARY VACATUR OR, IN THE ALTERNATIVE, FOR
STAY PENDING JUDICIAL REVIEW**

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GLOSSARY OF ACRONYMS AND ABBREVIATIONS

The following is a glossary of acronyms and abbreviations used in this motion:

NAAQS National ambient air quality standards

ppb Parts per billion

INTRODUCTION AND SUMMARY OF ARGUMENT

Petitioners challenge the Environmental Protection Agency's illegal and arbitrary delay of action mandated by the Clean Air Act to protect people from ground-level ozone, a dangerous and widespread air pollutant. EPA itself has found that ozone causes deaths, hospitalizations, asthma attacks, emergency room visits, and other serious harms, and that the existing federal limit on its concentration in the outdoor air is inadequate to protect public health. Yet, in a preemptory action involving no public participation and only cursory explication, EPA recently extended its deadline for promulgating initial area air quality designations for the 2015 national ambient air quality standards ("standards" or "NAAQS") for ozone. 82 FR 29,246 (June 28, 2017), Ex.1; *e.g.*, Letter from Scott Pruitt, Adm'r, EPA, to Doug Ducey, Gov. of Ariz., at 1, Ex.2 ("Delay Letter").¹ Under the Act, such designations are the essential step that triggers statutory obligations to implement measures to protect public health and welfare.

The Designations Delay defers urgently needed cleanup of harmful ozone pollution that threatens people across the nation. EPA has estimated that compliance with the standards will—each year—save hundreds of lives, prevent

¹ As explained below, the June 28 Notice followed letters sent June 6, with both announcing EPA was extending the deadline for promulgating initial area designations by one year. We refer to these documents announcing the delay collectively as the "Designations Delay."

230,000 asthma attacks in children, avoid hundreds of hospitalizations and emergency room visits, and prevent 160,000 missed school days for children. EPA, EPA-452/R-15-007, *Regulatory Impact Analysis of the Final Revisions to the National Ambient Air Quality Standards for Ground-Level Ozone* at ES-16, tbl.ES-6 (2015), Ex.12 (“RIA”); *see also id.* ES-2 to -3. Substantial death and suffering will thus inevitably result from the Designations Delay.

The Designations Delay is illegal and irrational. The statutory provision that EPA seized upon as ostensible ground for delaying implementation of the 2015 standards applies only where EPA “has insufficient information” to promulgate designations for one or more areas. 42 U.S.C. § 7407(d)(1)(B)(i). But EPA’s Designations Delay is devoid of any showing that the copious information already before the agency is somehow “insufficient.” Indeed, EPA did not even attempt such a showing. Instead, EPA tried to convert § 7407(d)(1)(B)(i) into a blanket authorization for delay on a broad mishmash of policy grounds irrelevant to the adequacy of information for designations, such as a desire to revisit the 2015 ozone standards themselves and alleged challenges in complying with the standards. Such concerns are untethered from § 7407(d)(1)(B)(i), the sole statutory authority EPA cited for its action.

Because EPA’s Designations Delay is clearly unlawful and arbitrary, and because this unlawful deferral of the October 1, 2017, deadline for completing

designations is dangerous to public health, summary vacatur is appropriate. In the alternative, EPA's Designations Delay should be stayed pending judicial review. In light of the severe health threats at stake, Petitioners further request that the case be expedited.²

BACKGROUND

I. OZONE SERIOUSLY HARMS HUMAN HEALTH.

Ozone, the main component of urban smog, is a corrosive air pollutant that inflames the lungs and constricts breathing. *See Am. Trucking Ass'ns v. EPA*, 283 F.3d 355, 359 (D.C. Cir. 2002) (“ATA”); EPA-HQ-OAR-2008-0699-0405 (“ISA”) 2-20 to -23 tbl.2-1, Ex.14. It causes asthma attacks, emergency room visits, hospitalizations, deaths, and other serious health harms. *E.g.*, 80 FR 65,292, 65,308/3-09/1 (Oct. 26, 2015), Ex.11; EPA-HQ-OAR-2008-0699-0404 (“PA”) 3-18, 3-26 to -29, 3-32, Ex.13; ISA 2-16 to -18, 2-20 to -24 tbl.2-1. Ozone can harm healthy adults, but others are more vulnerable. *See* 80 FR 65,310/1-3. Because their respiratory tracts are not fully developed, children are especially vulnerable to ozone pollution, particularly when they have elevated respiratory rates, as when playing outdoors. *E.g.*, PA 3-81 to -82. People with lung disease and the elderly also have heightened vulnerability. *See* 80 FR 65,310/3. People with asthma suffer

² Petitioners requested EPA administratively stay the Designations Delay on July 5. Exs.16-19. EPA has not acted on that request.

more severe impacts from ozone exposure than healthy individuals do and are more vulnerable at lower levels of exposure. *Id.* 65,311/1 n.37, 65,322/3.

II. THE CLEAN AIR ACT PRESCRIBES A CAREFULLY-DESIGNED PROGRAM FOR CONTROLLING OZONE POLLUTION.

Bringing the entire country expeditiously into compliance with health- and welfare-protective air quality standards forms the driving “heart” of the Clean Air Act. *Alabama Power Co. v. Costle*, 636 F.2d 323, 346 (D.C. Cir. 1980). EPA must set “primary” and “secondary” standards for pollutants like ozone to protect public health and welfare, respectively. 42 U.S.C. §§ 7408(a), 7409(a)-(b). It must review and, as appropriate, revise these standards at least every five years. *Id.*

§ 7409(d)(1). In setting and revising them, EPA is barred from considering the costs and technological feasibility of implementing the standards. *Whitman v. Am. Trucking Ass’ns*, 531 U.S. 457, 471 & n.4 (2001); *Am. Petroleum Inst. v. Costle*, 665 F.2d 1176, 1185 (D.C. Cir. 1981).

After EPA sets a standard, the implementation process begins, which, as relevant to this case, starts with initial area air quality designations. *See ATA*, 283 F.3d at 358-59. States and Tribes first submit recommended designations, and then, “as expeditiously as practicable,” but at the latest within two years of promulgating a standard, EPA “shall promulgate the designations of all areas (or portions thereof) submitted” by states and Tribes as either violating the standard

(“nonattainment” areas) or meeting the standard (“attainment” areas). 42 U.S.C. § 7407(d)(1)(A)-(B); *see also id.* §§ 7601(d)(1), 7602(d).³ The Act specifies the relevant considerations for making designations by specifically defining each type of area. For example, nonattainment areas are those that “do[] not meet (or that contribute[] to ambient air quality in a nearby area that does not meet)” a standard for a pollutant. *Id.* § 7407(d)(1)(A)(i). The Act provides only one condition under which EPA may extend its deadline for promulgating designations—when it “has insufficient information to promulgate the designations.” *Id.* § 7407(d)(1)(B)(i) (“Such period may be extended for up to one year in the event the Administrator has insufficient information to promulgate the designations.”).

For nonattainment areas, Congress created a detailed program to ensure that air quality will attain ozone standards by specified deadlines (“attainment deadlines”). *Id.* §§ 7410(a), (c), 7502; *see also id.* §§ 7511-7511f (provisions specific to ozone nonattainment areas). Each state must adopt a “state implementation plan” that, for nonattainment areas, includes all the requirements Congress crafted for such areas. *Id.* § 7410(a)(2)(I).

³ There is a third designation—an “unclassifiable” area, which is “any area that cannot be classified on the basis of available information as meeting or not meeting” the standard—which is treated for regulatory purposes as an attainment area. *See* 42 U.S.C §§ 7407(d)(1)(A)(iii), 7471; *see also Miss. Comm’n on Env’tl. Quality v. EPA*, 790 F.3d 138, 145 (D.C. Cir. 2015) (describing “unclassifiable” designation).

Crucially, the Act-required attainment deadlines are keyed to the date of designation. *See NRDC v. EPA*, 777 F.3d 456, 465-69 (D.C. Cir. 2014). The requirements—and deadlines—for states to adopt the specific programs Congress mandated to control harmful emissions in nonattainment areas similarly depend on the areas being designated nonattainment. *See, e.g.*, 42 U.S.C. §§ 7502(b), (c), 7503 (general planning requirements for nonattainment areas kick in when area is designated nonattainment), 7511a(a)(2)(C) (requiring “new source review” permitting programs that require new and modified major factories and power plants in nonattainment areas to install state-of-the-art emission controls and compensate for emission increases with greater offsetting reductions), 7511a(b)(2) (requiring emission control on certain types of existing sources in certain nonattainment areas), 7511a(c)(2)(A) (for certain nonattainment areas, requiring plans demonstrating attainment of standard by applicable attainment deadline).

Simultaneously with their designation, ozone nonattainment areas must be classified based on the severity of their ozone pollution levels. *Id.* § 7511(a)(1) tbl.1. The higher the classification, the longer the area has to come into attainment, but the more stringent the controls it must adopt. *South Coast Air Quality Mgmt. Dist. v. EPA*, 472 F.3d 882, 887 (D.C. Cir. 2006), *amended in other parts* 489 F.3d 1245 (D.C. Cir. 2007). If an area fails to attain timely, EPA must reclassify it to a higher classification, triggering stronger pollution control requirements. *Id.* 887-88

EPA's promulgation of nonattainment designations is thus essential to triggering the Act's nonattainment provisions and bringing about the attendant health and environmental benefits. Simply put, delay of designations delays the stronger pollution controls Congress mandated to protect people in communities with unhealthy air.

III. EPA FINDS THE 2008 STANDARDS INADEQUATE AND PROMULGATES STRONGER STANDARDS.

EPA revised the ozone standards most recently on October 1, 2015, strengthening them by tightening the maximum 8-hour level of ozone allowed in the ambient air to 70 parts per billion (ppb), down from the 75 ppb allowed under the 2008 standards. 80 FR 65,292/1, 65,452/2; 73 FR 16,436, 16,436/1 (2008). After a lengthy and detailed review process, EPA determined that the 2008 standards were inadequate to protect public health and welfare. 80 FR 65,342/2-47/1, 65,389/1-90/2. Important parts of the extensive record showed that healthy young adults experienced adverse health effects with ozone exposures at levels allowed by the 2008 standards and linked ozone levels allowed by those standards to hospital visits, deaths, and other serious health harms. *Id.* 65,343/1-44/3, 65,346/2-3. In a 15-city study, EPA estimated that tens of thousands of children would still face dangerous ozone exposures even after the 2008 standards were met. *Id.* 65,344/3-47/1. EPA's independent scientific advisors likewise

unanimously found the 2008 standards were not strong enough to protect public health and welfare. *Id.* 65,346/2, 65,381/3.

Multiple parties filed petitions in this Court challenging the 2015 standards, some arguing they were overly stringent, and others that they were insufficiently protective. *See Murray Energy v. EPA*, No. 15-1385 *et al.* (D.C. Cir.). Shortly before the scheduled oral argument in those consolidated cases, EPA sought to postpone it based on the agency's stated desire for time to determine whether to reconsider the 2015 standards. EPA Mot. to Continue Oral Argument 5-6, *Murray Energy*, No. 15-1385 (D.C. Cir. Apr. 7, 2017). The Court held the case in abeyance, but the standards have not been stayed and remain in effect. Order, *Murray Energy*, No. 15-1385 (D.C. Cir. Apr. 11, 2017).

EPA's revision of the standards on October 1, 2015, meant its mandatory deadline for issuing designations is October 1, 2017. *See* 42 U.S.C. § 7407(d)(1)(B)(i). The Act prescribes a step-by-step process for promulgating designations. States must first submit recommended designations to EPA within one year of standards' promulgation. *Id.* § 7407(d)(1)(A). EPA may modify a recommended designation, but must first provide the state 120 days' notice and give the state an opportunity to rebut the proposed modification. *Id.* § 7407(d)(1)(B)(ii).

EPA, states, and Tribes have already undertaken the steps that traditionally have enabled EPA to meet prior designation deadlines. All the states and several Tribes submitted designation recommendations to EPA.⁴ The states and Tribes formulated these recommendations with guidance from EPA regarding what kind of information the agency needed to make the final designations. For example, drawing on its experience with past ozone standards, EPA promulgated a memorandum further describing the process for developing designations. Memorandum on Area Designations for the 2015 Ozone National Ambient Air Quality Standards, from Janet McCabe, Acting Ass't Adm'r, to Reg'l Adm'rs (Feb. 25, 2016), Ex.10 (“Memorandum”).⁵ Among other things, EPA explained that it bases designations on air quality monitoring data—measurements of the amount of ozone actually present in the air at stations that sample the ambient air in locations consistent with EPA regulations. Memorandum 3; *see Miss. Comm'n*, 790 F.3d at 147 (upholding designations that used this approach for 2008 ozone standards). “After identifying each monitor that indicates a violation of the 2015

⁴ <https://www.epa.gov/ozone-designations/2015-ozone-standards-state-recommendations>; <https://www.epa.gov/ozone-designations/2015-ozone-standards-tribal-recommendations>.

⁵ EPA also proposed a rule that would govern implementation of the 2015 standards. 81 FR 81,276 (Nov. 17, 2016). The comment period on that rule closed February 13, 2017. 81 FR 91,894, 91,894/1-2 (Dec. 19, 2016).

ozone NAAQS in an area, the EPA will determine which nearby areas contribute to the violation(s)” based on five factors used in prior designations. Memorandum 5-7 (factors are “air quality data, emissions and emissions-related data, meteorology, geography/topography, and jurisdictional boundaries,” with other factors potentially relevant for specific areas); *see Miss. Comm’n*, 790 F.3d at 149, 158-59 (same factors applied for initial area designations for 2008 standards).

EPA explained that states would base designation recommendations on certified, quality-assured air quality monitoring data for 2013-15 (the years needed to calculate the official air quality statistic—“design value”—to assess compliance with the standards for 2015), with preliminary data for 2016 perhaps factoring in. Memorandum 2-4. EPA expected to base designations on the 2016 design value, which use 2014-16 data. *Id.* The 2015 design values, as measured at every air quality monitor in the country, were available in July 2016,⁶ and EPA regulations required full, accurate, and quality-assured data for 2016 by May 1, 2017. 40 C.F.R. § 58.15; *see also* Memorandum 3 (explaining regulations).

⁶ https://www.epa.gov/sites/production/files/2016-07/ozone_designvalues_20132015_final_07_29_16.xlsx.

IV. EPA ABRUPTLY DELAYS IMPLEMENTING THE NEW STANDARDS.

Without notice or public input, EPA extended its deadlines for promulgating designations by a year. First, on June 6, 2017, it sent identical four-paragraph letters to state governors so informing them. Exs.2-5; *see also* EPA, *Administrator Extends Deadline for Area Designations for 2015 Ozone Standards* (as updated June 7, 2017), Ex.6 (stating that EPA extended designations deadline “[i]n a letter to Governors”); EPA, *EPA to Extend Deadline for 2015 Ozone NAAQS Area Designations* (June 6, 2017), Ex.7 (press release announcing extension). The Delay Letter asserts potential concern about various purported “issues that could undermine...compliance efforts,” and that the delay gives “the Agency time to complete its review” of the 2015 standards, but nowhere explains why the information it already has is inadequate to promulgate designations. Delay Letter 1.

EPA subsequently announced its delay action in the Federal Register. 82 FR 29,246 (“Notice”). The Notice’s single-paragraph explication for the Designations Delay rehashes similar claims as the Delay Letters, but with some minor variations. It first baldly claims that because of various “issues regarding the 2015 ozone NAAQS and its implementation,” EPA “cannot assess whether [the Administrator] has the necessary information to finalize designations.” 82 FR 29,247/2. It also contends that because of the ongoing review of the standards, EPA lacks sufficient information to promulgate designations. *Id.* 29,247/3. EPA

again provides no explanation of what specific information it lacks to allow it to promulgate designations.

ARGUMENT

I. THE DESIGNATIONS DELAY IS UNLAWFUL AND ARBITRARY AND SHOULD BE SUMMARILY VACATED.

The Designations Delay is flagrantly illegal and arbitrary.⁷ EPA relied solely on a narrow Clean Air Act provision that allows EPA to delay nonattainment designations by up to one year only when it “has insufficient information to promulgate the designations.” 42 U.S.C. § 7407(d)(1)(B)(i). But EPA nowhere identified any insufficiency of information of the sort that, under the statute, is the sole permissible basis for a delay. The factors EPA cited are extraneous to the statutory criterion, instead addressing EPA’s desire to reconsider the standards and to examine compliance issues. EPA also failed to explain why, assuming it had explained what relevant information was lacking for any area, it was delaying designations for the entire country for an entire year, despite the Act’s mandate for expeditious designation promulgation.

EPA’s attempt to convert a narrow statutory provision into a broad authorization for delay is contrary to the plain, limited language of the Act and

⁷ This Court must set aside EPA actions that are “arbitrary, capricious, an abuse of discretion, or otherwise not in accordance with law.” 5 U.S.C. § 706(2)(A).

would thwart Congress's framework for deadline-driven attainment of health standards. Because EPA's Designations Delay is clearly unlawful, arbitrary, and dangerous to public health, and particularly given the imminent October 2017 statutory deadline for EPA to complete the designations, summary vacatur is appropriate. *See Clean Air Council v. EPA*, No. 17-1145, slip op. 10-11 (D.C. Cir. July 3, 2017).

A. EPA Has Not Satisfied the Statutory Requirements for Delaying Designations.

EPA does not even attempt to tether its purported rationale for the Designations Delay to the statutory prerequisite for such action—insufficient information to allow it to make designations. EPA has up to two years to promulgate designations, with the sole basis for extending that deadline (for up to a year) being where EPA “has insufficient information to promulgate the designations.” 42 U.S.C. § 7407(d)(1)(B)(i). Under the Act, the only bases for making designations are whether an area fails to meet the standards (a simple numerical comparison based on data EPA has) and whether it contributes to another area's failure to meet the standards: if an area meets either condition, it is a nonattainment area; otherwise, it is an attainment area. *Id.* § 7407(d)(1)(A)(i)-(ii) (defining “nonattainment” and “attainment” area). The information needed to promulgate designations is thus information about air quality at monitoring sites

and information about what areas affect air quality in nearby areas with monitors that violate the standards. *See id.* § 7407(d)(1)(A)(i)-(iii).⁸ Yet EPA illegally seeks to justify the Designations Delay based on considerations unrelated to these factors. *See Massachusetts v. EPA*, 549 U.S. 497, 535 (2007) (“EPA must ground its reasons for action or inaction in the statute.”).

EPA centrally relies on the irrelevant fact that it is reviewing the standards themselves, closing the Notice by saying:

We also note that new agency officials are currently reviewing the 2015 ozone NAAQS rule. The Administrator has determined that in light of the uncertainty of the outcome of that review, there is insufficient information to promulgate designations by October 1, 2017.

82 FR 29,247/3. The Agency says that, “[a]s part of the review process,” it is examining purported “issues that could undermine associated compliance efforts,” which are background ozone levels,⁹ ozone originating abroad, and “exceptional events demonstrations.”¹⁰ Delay Letter 1 (emphasis added); *accord* 82 FR 29,247/2

⁸ *See also* Memorandum 3-7 (describing EPA’s intended practice); *Miss. Comm’n*, 790 F.3d at 147, 149, 158-59 (describing EPA’s historical practice).

⁹ EPA uses the term “background” to mean ozone pollution caused by natural phenomena anywhere or by human-caused emissions outside the United States. 80 FR 65,328/1 (“U.S. background” means ozone “that would exist even in the absence of any manmade emissions within the United States”).

¹⁰ “Exceptional events” are certain events that were shown to clearly cause exceedances of standards but were “not reasonably controllable or preventable”

(citing most of the same issues as ones EPA is “evaluating...regarding the 2015 ozone NAAQS and its implementation”). EPA frames the Designations Delay as purportedly justified “[i]n light of the analyses currently underway at the agency.” 82 FR 29,247/3. But, though EPA is free to engage in such analyses, review of the standards and supposed compliance-related considerations is wholly divorced from the sufficiency of the information about actual air quality conditions that is relevant to making designations under these still-effective standards. *See, e.g., Motor Vehicle Mfrs. Ass’n v. State Farm Mut. Auto. Ins.*, 463 U.S. 29, 43 (1983).

Indeed, Congress required EPA to review standards at least every five years, 42 U.S.C. § 7409(d)(1), so Congress cannot possibly have meant to include such reviews as a justification for one-year delays. Otherwise, EPA could halt the “engine that drives nearly all of Title I of the [Clean Air Act]” by repeatedly revising and then reviewing standards, *Whitman*, 531 U.S. at 468. This interpretation cannot be reconciled with the Act. *See id.* 485 (rejecting as unreasonable statutory interpretation that would allow EPA to “abort[.]” ozone nonattainment provisions of Clean Air Act “the day after [provisions were] enacted”); *South Coast*, 489 F.3d at 1248 (rejecting as “absurd” statutory

and “caused by human activity that is unlikely to recur at a particular location or a natural event.” 42 U.S.C. § 7619(b). Data resulting from such events may be excluded from air quality monitoring data used to determine if an area violated the standards. *Id.*

interpretation that would allow EPA to take trivial actions and “avoid ever implementing” Act-mandated pollution controls).

Moreover, EPA cannot base a delay on mere speculation that it might have insufficient information. 82 FR 29,247/2-3 (claiming EPA cannot determine if it has “necessary,” “sufficient information to finalize designations” because of review). Congress required EPA to promulgate designations “as expeditiously as practicable” and authorized delay only where EPA rationally finds the information is actually “has” is inadequate to make designations. 42 U.S.C. § 7407(d)(1)(B)(i). EPA’s assertion here that it might have inadequate information is not such a finding. *See In re Harman Int’l Indus. Sec. Litigation*, 791 F.3d 90, 103 (D.C. Cir. 2015) (pointing out “important difference between warning that something ‘might’ occur and that something ‘actually had’ occurred” (emphasis in original)); *see also Moncrieffe v. Holder*, 133 S. Ct. 1678, 1688 & n.8 (2013) (rejecting analysis that looks at what “could have” or “would have” happened, not what did happen). Because EPA has not found the information it has is insufficient, it cannot lawfully or rationally grant itself an extension under § 7407(d)(1)(B)(i). *See Chamber of Commerce v. SEC*, 412 F.3d 133, 143-44 (D.C. Cir. 2011) (where Congress requires agency to make certain assessment, agency must do so).

EPA also says it seeks more time to “consider completely all designation recommendations provided by state governors...and to rely fully on the most

recent air quality data.” Delay Letter 1; *see also* 82 FR 29,247/2-3 (claiming EPA needs time to consider such materials because of (statutorily irrelevant) review of “issues regarding the 2015 ozone NAAQS and its implementation”). But those excuses do not relate, nor do they even claim to relate, to the sufficiency of the information EPA possesses. EPA raises no concern about the sufficiency of the designation recommendations or any concern about the sufficiency of the air quality data before it. Because EPA’s decision “rests on reasoning divorced from the statutory text,” it is unlawful and arbitrary. *Massachusetts*, 549 U.S. at 532-33; *see NRDC v. EPA*, 777 F.3d at 468-69 (where EPA “explanation lacks any grounding in the statute,” it is unlawful).¹¹

B. EPA’s Purported Justifications for the Delay Are Arbitrary and Unlawful.

“Where, as here, Congress has delegated to an administrative agency the critical task of assessing the public health and the power to make decisions of national import in which individuals’ lives and welfare hang in the balance, that agency has the heaviest of obligations to explain and expose every step of its reasoning.” *American Lung Ass’n v. EPA*, 134 F.3d 388, 392 (D.C. Cir. 1998).

¹¹ Even if the statute were not so unambiguous, EPA’s attempt to import factors irrelevant to the adequacy of information would be due no deference, as it did not adopt that reading through any formalized or well-considered process. *See, e.g., United States v. Mead Corp.*, 533 U.S. 218, 228 (2001).

Here, EPA has not explained how the information it has is insufficient to promulgate designations. Nor has it explained how the purported “issues” implicated by its review of the 2015 standards relate to the adequacy of the information it has for making designations. EPA identifies no way in which “background ozone levels” or “international transport” are relevant to designations, nor can it. 82 FR 29,247/2; *see supra* p.14, n.9 (EPA defines “background” as ozone resulting from anything other than human-caused emissions within the United States). Indeed, the Act specifically deals with international transport at the post-designation stage, during actual implementation. *See* 42 U.S.C. § 7509a(a)-(b). Addressing it at the designation stage would thus conflict with Congress’s careful implementation design. EPA’s speculation that it might find new approaches for states to comply with the standards is utterly irrelevant to the issue of whether communities are currently meeting or violating the standards. 82 FR 29,247/2-3 (discussing new “Ozone Cooperative Compliance Task Force” (emphasis added)). The Designations Delay is thus arbitrary.

Even if EPA’s stated bases for wanting more time were statutorily relevant, its reliance on them here was arbitrary. EPA claimed it cannot even tell if the information it has is insufficient, *id.* 29,247/2, but it has provided no explanation, much less a rational one, about why it cannot assess the information it has when it has all the information the statute and its own guidance say EPA needs. *See supra*

pp.9-10; Letter from Dannel Malloy, Gov. of Conn., to Scott Pruitt, Adm'r, EPA, at 3 (June 20, 2017) (“There is nothing missing from past information used by EPA to designate areas after previous revisions to the ozone NAAQS.”), Ex.9.

Also, EPA has already had months to consider the designation recommendations the states provided—all but one were submitted by mid-October 2016. *See* <https://www.epa.gov/ozone-designations/2015-ozone-standards-state-recommendations>. The last one was submitted in March 2017, but it is three sentences long, with the only substantive sentence consisting of the recommendation that EPA designate the same areas nonattainment under the 2015 standards as it did under the 2008, with the same boundaries. Letter from Larry Hogan, Gov. of Maryland, to Cecil Rodrigues, Acting Regional Adm'r (Mar. 23, 2017), https://www.epa.gov/sites/production/files/2017-05/documents/md_recommendations.pdf. It is hardly credible for EPA to claim it requires an entire year to “consider completely” information it has had for months.

As well as being irrelevant, EPA’s claim that, because of the review of issues regarding the standards, it needs more time to consider “exceptional events impacting designations” as part of considering the state recommendations, 82 FR 29,247/3, is unsupported. EPA identifies no rational connection between the review and the level of consideration needed for such exceptional events. Nor does EPA cite any specific instances where attainment designations hinge on timely

submitted, yet unresolved claims of exceptional events. Even if there are such claims, EPA fails to show it lacks adequate information to resolve them before the October 1 designations deadline. Even after the announcement in April 2017 of the review, EPA swiftly fully processed and granted an exceptional event petition dated April 14, 2017. EPA-HQ-OAR-2017-0223-0004 (granting petition via letter dated May 30, 2017), Ex.8. Thus, the record evidence contradicts EPA's claim that it needs more time, rendering the claim arbitrary. *See Nat'l Lime Ass'n v. EPA*, 233 F.3d 625, 634-35 (D.C. Cir. 2000).

Nor does EPA's claim about needing time to "rely fully on the most recent air quality data," Delay Letter 1, have any record basis. Those data were due to be certified by May 1, 2017, a month before EPA decided to extend the deadline and five months before the October 1 designation deadline. 40 C.F.R. § 58.15. EPA has provided no explanation why those periods are inadequate. To the contrary, EPA has previously made ozone designations only two months after receiving certified data. *See* 77 FR 30,088, 30,091/3, 30,095/2 (2012) (EPA finalized some designations on Apr. 30, 2012, using data certified by Feb. 29, 2012); *see also Miss. Comm'n*, 790 F.3d at 157-58. Thus, as well as failing to claim that it actually lacks the most recent air quality data, EPA has not made any rational claim that it lacked time to consider that information. *See Sorenson Comms. v. FCC*, 755 F.3d

702, 709 (D.C. Cir. 2014) (rule is arbitrary and capricious where it “is not only unsupported by the evidence, but contradicted by it”).

Further, EPA’s generic desire to consider new information cannot be reconciled with the Act’s requirement that EPA promulgate designations “as expeditiously as practicable,” with delay allowed only where the information EPA “has” is “insufficient,” 42 U.S.C. § 7407(d)(1)(B)(i). EPA here contravenes Congress’s command by delaying designations without explaining why the existing information is insufficient. EPA itself has recognized that it cannot keep waiting for new information instead of making designations, explaining during the designations process for the 2008 standards that “[n]ew technical data become available on a regular basis,” so “delay ‘to consider such new information would result in a never-ending process in which designations are never finalized.’” *Miss. Comm’n*, 790 F.3d at 158 (quoting letter from EPA Administrator) (alteration in original). In an analogous circumstance, where a statute required EPA to use the “best available evidence,” this Court has already held that “EPA cannot reject the ‘best available’ evidence simply because of the possibility of contradiction in the future by evidence unavailable at the time of action—a possibility that will always be present.” *Chlorine Chemistry Council v. EPA*, 206 F.3d 1286, 1290-91 (D.C. Cir. 2000) (emphasis in original). So too here: EPA’s preference to wait for more evidence is inconsistent with the Act and arbitrary.

C. Even If Some Delay Could Be Justified for Some Areas, EPA Has Not Justified the Across-the-Board One-Year Delay It Granted Itself.

Even if some information for some areas were insufficient—a claim EPA has not made or supported—EPA has not explained why a one-year extension for all areas is justified. The Act instructs EPA to make designations “as expeditiously as practicable,” and the length of any extension beyond the two-year outer deadline is limited to being “up to one year.” 42 U.S.C. § 7407(d)(1)(B)(i) (emphasis added). Thus, a one-year extension is not a default: it is an outer bound, with EPA obligated to move as quickly as practicable to promulgate designations. Here, EPA has given no explanation why a full year’s extension is rationally justified anywhere, let alone for every single area of the nation. That is arbitrary. *See American Lung*, 134 F.3d at 392. Further, because EPA failed to address the statutory requirement to designate as expeditiously as practicable, the Designations Delay is unlawful and arbitrary. *See Public Citizen v. Fed. Motor Carrier Safety Admin.*, 374 F.3d 1209, 1216 (D.C. Cir. 2004).

EPA claims that its review of certain issues relating to the standards and their implementation, like background ozone levels, international transport of ozone pollution, and exceptional events, affects its ability to make designations. 82 FR 29,247/2-3. Even if that were true—and it patently is not, *see supra* pp.14-15—EPA has not explained why those issues are relevant over every square inch of the

nation. To the contrary, EPA identified no specific area where any such issue precluded rational designations.

Far from claiming that background ozone levels are high enough to matter for regulatory purposes everywhere in the country, EPA has made clear that “the locations that are most strongly influenced by background [ozone] are relatively limited in scope, i.e., rural areas in the intermountain western U.S.” EPA-HQ-OAR-2008-0699-4309 at 347 (EPA Response to Comments on establishment of 2015 standards). The same holds for international transport. 80 FR 65,328/2. Nor does the Designations Delay identify any specific area where designation hinges on an unresolved claim that exceedances of the standards resulted from exceptional events, much less that such claims exist for every area of the nation. EPA has thus failed to explain why it chose to delay designations for the entire country. *See, e.g., Sorenson Comms.*, 755 F.3d at 709.

If EPA lacked sufficient information for certain areas, EPA arbitrarily failed to consider its time-tested alternative to a national delay. In prior designations, EPA has issued designations for certain areas while deferring designations for others for which it lacked sufficient information. For example, in making designations under the 2012 particulate matter standards, EPA extended its deadline for 10 specific areas where it had insufficient monitoring data “to determine whether the areas are meeting or are not meeting the [standards],” and

an additional year would give it those data, but EPA promulgated designations for the rest of the country, for which it had data or for which an additional year's worth of data would still be insufficient. 80 FR 2206, 2207/3 (Jan. 15, 2015); *see also* 75 FR 71,033, 71,035/3-36/1 (2010) (similar for designations under 2008 lead standard). Assuming there were some (unexplained) factual basis for EPA's concerns about the completeness of its information, it was arbitrary for EPA not even to consider the option of making some designations but deferring others. *E.g.*, *State Farm*, 463 U.S. at 46-48 (agency failure to consider reasonable alternative renders its action arbitrary); *Del. Dep't of Nat. Res. v. EPA*, 785 F.3d 1, 17-18 (D.C. Cir. 2015) (where EPA failed to consider "reasonable alternatives" to uniform national rule, "its action was not rational and must, therefore, be set aside").

II. IN THE ALTERNATIVE, THE COURT SHOULD STAY THE DESIGNATIONS DELAY.

If the Court does not summarily vacate the Designations Delay, it should stay EPA's action pending merits review. A stay's issuance depends on balancing four factors: (1) petitioners' likelihood of success on the merits; (2) whether petitioners will suffer irreparable harm without a stay; (3) whether a stay will substantially harm other parties; and (4) the public interest. D.C. Cir. R.18(a)(1); *see, e.g., League of Women Voters v. Newby*, 838 F.3d 1, 6 (D.C. Cir. 2016). As

discussed above, the Designations Delay is clearly illegal and arbitrary, meaning the first factor strongly favors Petitioners. As discussed below, so do the other three.

A. The Designations Delay Will Irreparably Harm Petitioners.

Unless this Court summarily vacates the Designations Delay, judicial review in this case will mostly likely extend well beyond October 1, 2017, the date by which, under the Act, EPA must promulgate designations. Without a stay pending judicial review, Petitioners' members face a substantial likelihood of irreparable harm resulting from the Designations Delay.

By delaying designations, EPA delays pollution controls required by the Act to curb ozone levels EPA agrees are unsafe in communities where Petitioners' members live, work, and enjoy recreation. Such communities include areas currently designated as attainment under the 2008 standards but whose ozone levels violate the 2015 standards; because of the Designations Delay, they will not be timely designated nonattainment and thus will lack the anti-pollution protections that a nonattainment designation would accord them. *See supra* pp.6-7; Berman Decl. ¶¶ 17-34, Ex.23; Craft Decl. ¶ 14, Ex.27. These important protections include measures states adopt into their implementation plans to limit emissions of ozone-forming chemicals sufficiently for the area to come into attainment, as well as attainment deadlines, nonattainment new source review for

new or modified major sources of pollution, like factories and power plants, and pollution controls for large existing plants.

These delays will irreparably harm Petitioners' members by prolonging their exposure to ozone levels EPA has found cause deaths, asthma attacks in children, emergency room visits, hospitalizations, and other serious health harms. RIA at ES-16 tbl.ES-6; *see also id.* ES-2 to -3. The attached Declarations, Exs.20-46, demonstrate the human impacts of these harms. Krystal Henagan is a member of Petitioners Environmental Defense Fund and Sierra Club living in San Antonio, a city with ozone levels that violate the 2015 standards, but is designated attainment under the 2008 standards. Henagan Decl. ¶¶ 1-2, 4, Ex.34; Berman Decl. ¶ 19. She struggles with her 8-year-old son's asthma, which is controlled by four medications, and which has been "life threatening" in the past. Henagan Decl. ¶¶ 5-9, 12. Her son regularly must go to the doctor—12 visits in 2017 alone—and "he has missed countless school days due to poor air quality exacerbating his asthma," days during which Ms. Henagan must stay home and care for him. *Id.* ¶¶ 7-12. She fears he will suffer asthma attacks or that she "would need to rush him to the hospital due to his inability to breathe." *Id.* ¶ 8.

Rhonda Anderson, a Sierra Club member living and working in Detroit (also a city with ozone levels that violate the 2015 standards, but designated attainment under the 2008 standards, Berman Decl. ¶ 19), a grandmother, and a senior with

asthma, describes how, even as an adult, she has had to go to the hospital because of her asthma, and her daughters and granddaughters have similarly had to go (both as adults and as children). Anderson Decl. ¶¶ 4-6, Ex.21. She describes the trauma of going to the emergency room because of a child's asthma attack:

Often, I have spent no less than 4 hours waiting, all the while wondering when my child will be able to see the doctor. Once we are called, we get sent to a second crowded room with breathing machines, and every station is filled with a child having an asthma attack. It is very stressful and there is nothing that can take my mind off the fact that I have a sick child that I cannot help.

Id. ¶ 6. Family members and others she knew have died from asthma attacks. *Id.*

¶ 8.

Other declarants further detail how the health harms ozone causes affect them. Nsedu Obot Witherspoon, a member of the American Public Health Association, has a seven-year-old son with asthma. Witherspoon Decl. ¶ 5, Ex.46. He endured "his first bad asthma attack at just three years old," so bad that when they got to the doctor,

they said his oxygen levels were so low that we had to leave our car and go immediately to the ER in an ambulance. This was one of the scariest moments of my life. He was hospitalized for two nights. He was again hospitalized at the age of four.

Id. Even now, despite "a rigorous asthma management plan with frequent check-ups at the pediatrician's office," "his asthma acts up...frequently...when air quality is bad." *Id.* ¶ 6. "[H]e often has to slow down or sit out on high air pollution days,"

instead of playing outside or walking or hiking with his siblings and parents. *Id.*

¶¶ 3-4, 7.

Petitioners' members living, working, and recreating in areas attaining the 2008 standards, but with ozone levels that violate the 2015 standards routinely find their ability to breathe impaired (*see, e.g.*, Anderson Decl. ¶¶ 4-5; Brock Decl. ¶ 5 (Atlanta area), Ex.24; Einzig Decl. ¶¶ 3-5 (Baltimore), Ex.30; *see also* Berman Decl. ¶¶ 19, 22-23) and their ability to work and their children's ability to attend school impeded (*see, e.g.*, Einzig Decl. ¶ 8; Henagan Decl. ¶¶ 6-7). Because of their health concerns, they must refrain from outdoor activities they would otherwise enjoy. *See, e.g.*, Seal Decl. ¶ 10 (San Antonio), Ex.41; Einzig Decl. ¶ 7. Petitioners have tens of thousands of other members residing in such areas. *See* Stith Decl. ¶ 10, Ex.42; Berman Decl. ¶¶ 19, 22 & attach.1.

Because the attainment deadlines run from the date of designation, *see supra* p.6, the ozone pollution levels in these areas will be allowed to remain at dangerously elevated levels for an additional year because of EPA's year-long Designations Delay unless this Court stays EPA's unlawful action. *See* Craft Decl. ¶ 14. Petitioners' members living in such areas, who experience severe harms to their health and wellbeing because of ozone pollution, will thus be harmed both by the delay in mandatory pollution reductions and by the additional year that they will have to endure dangerous ozone levels.

Even in areas violating both the 2008 and 2015 standards, the Designations Delay means Petitioners' members there will receive neither the benefits of pollution reductions designed to drive compliance with the new, more protective 2015 standards nor the benefit of actual compliance with those standards as soon as they would absent the Designations Delay. For example, Jane Reardon, a nurse and member of Petitioner American Lung Association's board, lives and works in Hartford County, Connecticut, caring for patients "who are hospitalized as a result of respiratory ailments," including "many...patients...older than 65, like [her]." Reardon Decl. ¶¶ 1, 5-6, Ex.39; *see also id.* ¶ 7 (describing activities she engages in outdoors near her home, thus exposing her to dangerous ozone pollution). Hartford County violates both the 2008 and 2015 standards. *Id.* ¶¶ 1, 5; Berman Decl. ¶¶ 17, 30. As a result of the Designations Delay, implementation of the 2015 standards in Hartford County will be delayed a year, and the area's attainment deadline will, too, thus endangering her health and her patients' health. Reardon Decl. ¶ 8; *see also* Lyon Decl. ¶¶ 4-5 (describing how patients in Philadelphia with lung disease must miss medical appointments because poor air quality causes symptoms to flare up). Accordingly, the harms described above affect even more of Petitioners' members.

These human health harms resulting directly from EPA's Designations Delay are irreparable, for ultimate success on the merits cannot undo them: no

court order can enable EPA to raise the dead, undo asthma attacks, reverse a hospitalization, or restore a missed day in the classroom or at work. *See Wisc. Gas Co. v. FERC*, 758 F.2d 669, 674 (D.C. Cir. 1985). Petitioners' members, their families, and, for medical professionals, their patients have demonstrated heightened vulnerability to ozone pollution and already experience serious harms from it. The harms to them absent a stay are "likely," as well as "certain and great," "actual and not theoretical," "beyond remediation," and so "imminent that there is a clear and present need for equitable relief to prevent irreparable harm." *League of Women Voters*, 838 F.3d at 6-8; *see also Coleman v. Paccar, Inc.*, 424 U.S. 1301, 1307-08 (1976) (Rehnquist, J., in chambers) (finding "irreparable harm" where lower court stay of motor vehicle safety standards would delay "for a year or more" "[e]ffective implementation...of the congressionally mandated" program to "reduce traffic accidents and deaths and injuries").

Likewise, the Designations Delay is substantially likely to cause irreparable environmental harms, including to places Petitioners' members use and enjoy. *See Kodish Decl.* ¶¶ 2-6, 9-14, Ex.35; *Toher Decl.* ¶¶ 3-8, Ex.43. Ozone damages vegetation and forested ecosystems, causing or contributing to widespread stunting of plant growth, tree deaths, visible leaf injury, reduced carbon storage, and damage to entire ecosystems. PA 5-2 to -3; ISA 9-1; 80 FR 65,370/1-2, 65,377/3. EPA acknowledges that, "[i]n terms of forest productivity and ecosystem diversity,

ozone may be the pollutant with the greatest potential for region-scale forest impacts.” RIA 7-3. Such widespread vegetation and ecosystem losses are irreparable, as they cannot “be adequately remedied by money damages” and are of “permanent or at least of long duration.” *Amoco Production Co. v. Village of Gambell*, 480 U.S. 531, 545 (1987).

In short, without a stay pending judicial review, EPA will delay initial area designations beyond what the statute allows, making for a longer period of time that Petitioners’ members will be exposed to excessive amounts of air pollution that causes them serious harms. There can be “no do over and no redress” later, *League of Women Voters*, 838 F.3d at 9, for the irreparable health and environmental harms that Petitioners’ members (and the broader public) are virtually certain to experience absent a stay.¹²

B. A Stay Will Not Harm Other Parties.

As the agency responsible for the proper execution of the Clean Air Act, EPA cannot be substantially harmed by a stay that would prevent it from giving effect to an illegal and arbitrary action. *See Nat’l Ass’n of Farmworkers Orgs. v. Marshall*, 628 F.2d 604, 615 (D.C. Cir. 1980) (“consequences [that] are no

¹² For similar reasons, Petitioners have standing to challenge the Designations Delay. *See* Declarations.

different from [agency's] burdens under the statutory scheme” “do not constitute substantial harm for the purpose of delaying injunctive relief”).

Moreover, a stay would not prevent EPA from reviewing the 2015 standards or their implementation. The Act plainly contemplates that EPA will implement ozone standards even as it reviews them. Indeed, the Act puts the ozone standards on an implementation schedule that can last up to 20 years, while requiring EPA to review standards at least every 5 years. *Whitman*, 531 U.S. at 485 (“Congress knew” EPA could review ozone standards at any time, but still established implementation framework “reaching...far into the future”) (discussing 42 U.S.C. §§ 7409(d)(1), 7511(a), and 7511a). Thus, EPA would not experience harm from this Court’s staying the Designations Delay.

C. The Public Interest Strongly Favors Staying the Designations Delay.

EPA projects that compliance with the 2015 standards will realize significant health benefits. Outside of California, EPA estimates that compliance with the standards will result in upwards of 600 lives saved, over 250 heart attacks avoided, about 1,000 hospital admissions or emergency room visits prevented, 230,000 asthma attacks in children prevented, and 160,000 school loss days for children averted each year. RIA at ES-16 tbl.ES-6. The economic value of these benefits substantially outweighs the costs of achieving them. *Id.* ES-15 tbl.ES-5. In

making its estimate of public health benefits, EPA assumed designations would be completed in late 2017. *See id.* ES-2 to -3. These health benefits would come on top of the public health gains from achieving the 2008 standards and from several other pollution reduction requirements EPA put in place around the time it finalized the 2015 standards. *Id.* 6-1 (“The benefits...are estimated as being incremental to attaining the existing standard of 75 ppb.... These estimated benefits are incremental to the benefits estimated for several recent rules....”).

These protections are already years overdue. EPA’s deadline for reviewing and revising the 2008 standards fell in March 2013. *See* 42 U.S.C. § 7409(d)(1) (five-year review cycle); 73 FR 16,436 (standards promulgated in March 2008). EPA acted over 18 months late, and only after being sued (a suit in which it requested still more time to finalize its review and revision of the 2008 standards). Order 1-2, *Sierra Club v. EPA*, No. 13-cv-2809 (N.D. Cal. Apr. 30, 2014) (rejecting timeframe EPA sought for finishing rulemaking). Delaying the designations will cause serious harm to the breathing public and to the environment. *See supra* pp.25-31; Craft Dec. ¶ 14 (delaying designations will “lead to a longer period of inaction before measures to abate health-harming ozone are undertaken in these heavily impacted areas,” resulting in “delayed attainment and more exposure to ground-level ozone,” resulting in “more asthma attacks, hospitalizations, emergency room visits, and premature deaths in those areas”).

On the other side of the ledger, industry and states are not cognizably harmed by having to comply with the ozone implementation program Congress enacted. *See, e.g., League of Women Voters*, 838 F.3d at 12 (“There is generally no public interest in the perpetuation of unlawful agency action.”). Moreover, history shows this country can have both economic growth and air pollution reductions. *See, e.g.,* https://gispub.epa.gov/air/trendsreport/2016/#econ_growth_cleaner_air_ (over 1970-2015, emissions of the six pollutants most directly limited by national ambient air quality standards decreased 71%, and gross domestic product increased nearly 250%), Ex.15.

The public interest thus strongly favors staying the Designations Delay. The Designations Delay means that Congress’s carefully-refined ozone nonattainment provisions will not engage as quickly as EPA assumed in its regulatory analysis. The pollution reductions they are specifically designed to assure will not occur as quickly, either. Thus, without a stay, compliance with the standards will be delayed, and Congress’s promise to all residents of this country that they will have safe air to breathe will go unfulfilled even longer than it already has. Significant numbers of lives will be needlessly worsened or lost as a result. Such an outcome flouts the public interest and the purpose of the Clean Air Act. *Union Elec. v. EPA*, 427 U.S. 246, 256 (1976) (Clean Air Act is “a drastic remedy to what was perceived as a serious and otherwise uncheckable problem of air pollution”).

CONCLUSION

For the foregoing reasons, Petitioners respectfully request summary vacatur of the Designations Delay or, in the alternative, a stay of the Designations Delay and expedited review, *see* D.C. Cir., *Handbook of Practice and Internal Procedures* 33 (Jan. 26, 2017).

DATED: July 12, 2017

Respectfully submitted,

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CERTIFICATE OF COMPLIANCE WITH TYPE-VOLUME LIMIT

Counsel hereby certifies, in accordance with Federal Rules of Appellate Procedure 32(g)(1) and 27(d)(2) and D.C. Circuit Rule 18(b), that the foregoing **Motion for Summary Vacatur or, in the Alternative, for Stay Pending Judicial Review** contains 7,760 words, as counted by counsel's word processing system, and thus complies with the 7,800 word limit.

Further, this document complies with the typeface and type-style requirements of Federal Rule of Appellate Procedure 32(a)(5) & (a)(6) because this document has been prepared in a proportionally spaced typeface using **Microsoft Word 2010** using **size 14 Times New Roman** font.

DATED: July 12, 2017

/s/Seth L. Johnson
Seth L. Johnson

CERTIFICATE OF SERVICE

I hereby certify that on this 12th day of July, 2017, I have served the foregoing **Motion for Summary Vacatur or, in the Alternative, for Stay Pending Judicial Review** on all registered counsel through the court's electronic filing system (ECF) and by email.

/s/Seth L. Johnson

Seth L. Johnson

**IN THE UNITED STATES COURT OF APPEALS
FOR THE DISTRICT OF COLUMBIA CIRCUIT**

AMERICAN LUNG ASSOCIATION,)	
<i>et al.</i> ,)	
)	
<i>Petitioners,</i>)	
)	
v.)	Case No. 17-1172
)	
U.S. ENVIRONMENTAL)	
PROTECTION AGENCY, <i>et al.</i> ,)	
)	
<i>Respondents.</i>)	

**ADDENDUM TO PETITIONERS' MOTION FOR SUMMARY VACATUR
OR, IN THE ALTERNATIVE, FOR STAY PENDING JUDICIAL REVIEW**

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**IN THE UNITED STATES COURT OF APPEALS
FOR THE DISTRICT OF COLUMBIA CIRCUIT**

AMERICAN LUNG ASSOCIATION,)	
<i>et al.</i> ,)	
)	
<i>Petitioners</i> ,)	
)	
v.)	Case No. 17-1172
)	
U.S. ENVIRONMENTAL)	
PROTECTION AGENCY, <i>et al.</i> ,)	
)	
<i>Respondents</i> .)	

CERTIFICATE AS TO PARTIES, RULINGS, AND RELATED CASES

Pursuant to D.C. Circuit Rule 27(a)(4), American Lung Association, American Public Health Association, American Thoracic Society, Clean Air Council, Environmental Defense Fund, Environmental Law and Policy Center, National Parks Conservation Association, Natural Resources Defense Council, Ohio Environmental Council, Physicians For Social Responsibility, Sierra Club, and West Harlem Environmental Action (collectively, “Movants”) submit this certificate as to parties, rulings, and related cases.

(A) Parties and *Amici*

(i) Parties, Intervenors, and *Amici* Who Appeared in the District Court

This case is a petition for review of final agency action, not an appeal from the ruling of a district court.

(ii) Parties to This CasePetitioners:

The petitioners in the above-captioned case are American Lung Association, American Public Health Association, American Thoracic Society, Clean Air Council, Environmental Defense Fund, Environmental Law and Policy Center, National Parks Conservation Association, Natural Resources Defense Council, Ohio Environmental Council, Physicians For Social Responsibility, Sierra Club, and West Harlem Environmental Action

Respondents:

The respondents in the above-captioned case are the United States Environmental Protection Agency (“EPA”) and Scott Pruitt, in his official capacity as Administrator of the EPA.

Intervenors:

None at present.

(iii) Amici in This Case

None at present.

(iv) Circuit Rule 26.1 Disclosures

See Petitioners’ disclosure form filed accompanying the Petition for Review.

(B) Rulings Under Review

Petitioners seek review of the final action taken by EPA extending the deadline for promulgating initial area designations for the 2015 ozone national ambient air quality standards, which EPA announced in a Federal Register notice published at 82 FR 29,246 (June 28, 2017) and titled “Extension of Deadline for Promulgating Designations for the 2015 Ozone National Ambient Air Quality Standards” and in letters to state governors dated June 6, 2017, examples of which were attached to the Petition for Review as Attachments B and C.

(C) Related Cases

Movants are not aware of any related cases.

DATED: July 12, 2017

Respectfully submitted,

/s/Ann Brewster Weeks (w/permission)

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Association, American Thoracic
Society, National Parks Conservation
Association, Natural Resources*

Defense Council, Physicians for Social Responsibility, Sierra Club, and West Harlem Environmental Action

/s/Scott Strand (w/permission)

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/s/Sean H. Donahue (w/permission)

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Counsel for Environmental Defense Fund

Exhibit 1

withdrawn or denied. The proceeds of any such fees will be used to pay the remaining portion of the Agency's cost of providing credit assistance and the costs of retaining expert firms, including financial, engineering, and legal services, in the field of municipal and project finance, to assist in the underwriting of the Federal credit instrument. All of, or a portion of, this fee may be waived.

(d) *Servicing fee.* EPA will require borrowers to pay a servicing fee for each credit instrument approved for funding. Separate fees may apply for each type of credit instrument (e.g., a loan guarantee, a secured loan with a single disbursement, or a secured loan with multiple disbursements), depending on the costs of servicing the credit instrument as determined by the Administrator. Such fees will be set at a level sufficient to enable the EPA to recover all or a portion of the costs to the Federal Government of servicing WIFIA credit instruments.

(e) *Optional supplemental fee.* If, in any given year, there is insufficient budget authority to fund the credit instrument for a qualified project that has been selected to receive assistance under WIFIA, EPA and the approved applicant may agree upon a supplemental fee to be paid by or on behalf of the approved applicant at the time of execution of the term sheet to reduce the subsidy cost of that project. No such fee may be included among eligible project costs.

(f) *Reduced fees.* To the extent that Congress appropriates funds in any given year beyond those sufficient to cover internal administrative costs, EPA may utilize such appropriated funds to reduce fees that would otherwise be charged under paragraph (c) of this section.

(g) *Extraordinary expenses.* EPA may require payment in full by the borrower of additional fees, in an amount determined by EPA, and of related fees and expenses of its independent consultants and outside counsel, to the extent that such fees and expenses are incurred directly by EPA and to the extent such third parties are not paid directly by the borrower, in the event that a borrower experiences difficulty relating to technical, financial, or legal matters or other events (e.g., engineering failure or financial workouts) which require EPA to incur time or expenses beyond standard monitoring.

[FR Doc. 2017-13438 Filed 6-27-17; 8:45 am]

BILLING CODE 6560-50-P

ENVIRONMENTAL PROTECTION AGENCY

40 CFR PART 81

[EPA-HQ-OAR-2017-0223; FRL-9964-37-OAR]

Extension of Deadline for Promulgating Designations for the 2015 Ozone National Ambient Air Quality Standards

AGENCY: Environmental Protection Agency (EPA).

ACTION: Extension of deadline for promulgating designations.

SUMMARY: The Environmental Protection Agency (EPA) is announcing that it is using its authority under the Clean Air Act (CAA) to extend by 1 year the deadline for promulgating initial area designations for the ozone national ambient air quality standards (NAAQS) that were promulgated in October 2015. The new deadline is October 1, 2018.

DATES: The deadline for the EPA to promulgate initial designations for the 2015 ozone NAAQS is October 1, 2018.

FOR FURTHER INFORMATION CONTACT: For questions regarding this action, contact Denise Scott, Air Quality Planning Division, Office of Air Quality Planning and Standards, Mail Code C539-04, Environmental Protection Agency, Research Triangle Park, North Carolina 27711; telephone number: (919) 541-4208; email address: scott.denise@epa.gov.

SUPPLEMENTARY INFORMATION:

I. General Information

A. Does this action apply to me?

Entities potentially affected by this action include state, local and tribal governments that would participate in the initial area designation process for the 2015 ozone standards.

B. Where can I get a copy of this document and other related information?

The EPA has established a docket for designations for the 2015 ozone NAAQS under Docket ID No. EPA-HQ-OAR-2017-0223. All documents in the docket are listed in the <http://www.regulations.gov> index. Although listed in the index, some information is not publicly available, e.g., confidential business information or other information whose disclosure is restricted by statute. Certain other material, such as copyrighted material, will be publicly available only in hard copy. Publicly available docket materials are available either electronically in [http://](http://www.regulations.gov)

www.regulations.gov or in hard copy at the EPA Docket Center EPA/DC, EPA West, Room 3334, 1301 Constitution Avenue NW., Washington, DC. The Public Reading Room is open from 8:30 a.m. to 4:30 p.m., Monday through Friday, excluding legal holidays. The telephone number for the Public Reading Room is (202) 566-1744, and the telephone number for the EPA Docket Center is (202) 566-1742.

An electronic copy of this notice is also available at <http://www.epa.gov/ozone-designations> along with other information related to designations for the 2015 ozone NAAQS.

II. Designations Requirements

On October 1, 2015, the EPA signed a notice of final rulemaking that revised the 8-hour primary and secondary ozone NAAQS (80 FR 65292; October 26, 2015). The primary standard was lowered from 0.075 parts per million (ppm) to a level of 0.070 ppm. The EPA also revised the secondary standard by making it identical in all respects to the revised primary standard. (The previous ozone NAAQS were set in 2008 and remain effective.)

After the EPA establishes or revises a NAAQS pursuant to CAA section 109, the CAA directs the EPA and the states to begin taking steps to ensure that those NAAQS are met. The first step is to identify areas of the country that do not meet the new or revised NAAQS. This step is known as the initial area designations. Section 107(d)(1)(A) of the CAA provides that, "By such date as the Administrator may reasonably require, but not later than 1 year after promulgation of a new or revised national ambient air quality standard for any pollutant under section [109], the Governor of each State shall * * * submit to the Administrator a list of all areas (or portions thereof) in the State" that designates those areas as nonattainment, attainment, or unclassifiable. The CAA defines an area as nonattainment if it is violating the NAAQS or if it is contributing to a violation in a nearby area. 42 U.S.C. 7407(d)(1)(A)(i).

The CAA further provides, "Upon promulgation or revision of a national ambient air quality standard, the Administrator shall promulgate the designations of all areas (or portions thereof) * * * as expeditiously as practicable, but in no case later than 2 years from the date of promulgation of the new or revised national ambient air quality standard. Such period may be extended for up to one year in the event the Administrator has insufficient information to promulgate the

designations.” 42 U.S.C. 7407(d)(1)(B)(i).

After the states submit their recommendations, but no later than 120 days prior to promulgating designations, the EPA is required to notify a state of any intended modifications to the state’s recommended designation. The state then has an opportunity to demonstrate why any proposed modification is inappropriate. Whether or not a state provides a recommendation, the EPA must promulgate the designation that the agency deems appropriate within 2 years of promulgation of the NAAQS (or within 3 years if the EPA extends the deadline).

For the 2015 ozone NAAQS, the deadline for states to submit designation recommendations to the EPA for their areas was October 1, 2016. The EPA has been evaluating these recommendations and conducting additional analyses to determine whether it is necessary to modify any of the state recommendations.

III. Extension of Deadline for Promulgating Designations for the 2015 NAAQS

In this action, the EPA is announcing that it is using its authority under

section 107(d)(1)(B)(i) of the CAA to extend by 1 year the deadline for promulgating initial area designations for the 2015 ozone NAAQS. The new deadline is October 1, 2018. For the reasons explained in this notice, the EPA Administrator has determined that there is insufficient information to complete the designations by October 1, 2017.

Following the recent change in administrations, the agency is currently evaluating a host of complex issues regarding the 2015 ozone NAAQS and its implementation, such as understanding the role of background ozone levels and appropriately accounting for international transport. The Administrator has determined that he cannot assess whether he has the necessary information to finalize designations until additional analyses from this evaluation are available. In addition, pursuant to language in the recently-enacted Fiscal Year 2017 omnibus bill, the Administrator is establishing an Ozone Cooperative Compliance Task Force to develop additional flexibilities for states to comply with the ozone standard. It is possible the outcome of that effort could identify flexibilities that could impact

the designations process. In light of the analyses currently underway at the agency, the Administrator has determined he needs additional time to consider completely all designation recommendations provided by state governors pursuant to CAA section 107(d)(1)(A), including full consideration of exceptional events impacting designations, and determine whether they provide sufficient information to finalize designations. We also note that new agency officials are currently reviewing the 2015 ozone NAAQS rule. The Administrator has determined that in light of the uncertainty of the outcome of that review, there is insufficient information to promulgate designations by October 1, 2017.

List of Subjects in 40 CFR Part 81

Environmental protection, Air pollution control, National parks, Wilderness areas.

Dated: June 21, 2017.

E. Scott Pruitt,
Administrator.

[FR Doc. 2017–13437 Filed 6–27–17; 8:45 am]

BILLING CODE 6560–50–P

Exhibit 2



E. SCOTT PRUITT
ADMINISTRATOR

June 6, 2017

The Honorable Doug Ducey
Governor of Arizona
State Capitol
1700 W. Washington Street
Phoenix, AZ 85007

Dear Governor Ducey:

I am writing to update you on the status of the U.S. Environmental Protection Agency's efforts related to the National Ambient Air Quality Standards (NAAQS) for ozone promulgated in October 2015. Pursuant to section 107(d)(1)(B) of the Clean Air Act (CAA), I am extending the deadline for promulgating initial area designations for the 2015 ozone NAAQS by one year. I have determined that there is insufficient information, and taking additional time is appropriate in order to consider completely all designation recommendations provided by state governors pursuant to CAA section 107(d)(1)(A) and to rely fully on the most recent air quality data. This additional time will also provide the Agency time to complete its review of the 2015 ozone NAAQS, prior to taking this initial implementation step.

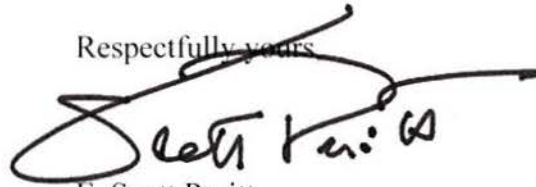
Although the new ozone standard was set on October 1, 2015, there remains a host of complex issues that could undermine associated compliance efforts by states, localities and regulated entities. As part of the review process, the Agency is evaluating these issues primarily focusing on: fully understanding the role of background ozone levels; appropriately accounting for international transport; and, timely consideration of exceptional events demonstrations. Additionally, pursuant to language in the recently-enacted FY 2017 omnibus bill, I have established an Ozone Cooperative Compliance Task Force to develop additional flexibilities for states to comply with the ozone standard.

States have made tremendous progress and significant investment cleaning up the air. Since 1980, total emissions of the six principal air pollutants have dropped by 63 percent and ozone levels have declined by 33 percent. Despite the continued improvement of air quality, costs associated with compliance of the ozone NAAQS have significantly increased. I am committed to working with you and your local officials to effectively implement the ozone standard in a manner that is supportive of your air quality improvement efforts, without interfering with local decisions or impeding economic growth.



I appreciate the information you and your staff have shared with EPA already as part of this process. I am confident this progress will continue as we work together towards our shared goal of clean air, a robust economy and stronger, healthier communities. If you have questions or concerns, please contact me or your staff may contact Troy Lyons, Associate Administrator for the Office of Congressional and Intergovernmental Relations, at lyons.troy@epa.gov or (202) 564-4987.

Respectfully yours,

A handwritten signature in black ink, appearing to read "Scott Pruitt", written over the typed name below.

E. Scott Pruitt

Exhibit 3



E. SCOTT PRUITT
ADMINISTRATOR

June 6, 2017

The Honorable Dan Malloy
Governor of Connecticut
210 Capitol Avenue
Hartford, Connecticut 06106

Dear Governor Malloy:

I am writing to update you on the status of the U.S. Environmental Protection Agency's efforts related to the National Ambient Air Quality Standards (NAAQS) for ozone promulgated in October 2015. Pursuant to section 107(d)(1)(B) of the Clean Air Act (CAA), I am extending the deadline for promulgating initial area designations for the 2015 ozone NAAQS by one year. I have determined that there is insufficient information, and taking additional time is appropriate in order to consider completely all designation recommendations provided by state governors pursuant to CAA section 107(d)(1)(A) and to rely fully on the most recent air quality data. This additional time will also provide the Agency time to complete its review of the 2015 ozone NAAQS, prior to taking this initial implementation step.

Although the new ozone standard was set on October 1, 2015, there remains a host of complex issues that could undermine associated compliance efforts by states, localities and regulated entities. As part of the review process, the Agency is evaluating these issues primarily focusing on: fully understanding the role of background ozone levels; appropriately accounting for international transport; and, timely consideration of exceptional events demonstrations. Additionally, pursuant to language in the recently-enacted FY 2017 omnibus bill, I have established an Ozone Cooperative Compliance Task Force to develop additional flexibilities for states to comply with the ozone standard.

States have made tremendous progress and significant investment cleaning up the air. Since 1980, total emissions of the six principal air pollutants have dropped by 63 percent and ozone levels have declined by 33 percent. Despite the continued improvement of air quality, costs associated with compliance of the ozone NAAQS have significantly increased. I am committed to working with you and your local officials to effectively implement the ozone standard in a manner that is supportive of your air quality improvement efforts, without interfering with local decisions or impeding economic growth.

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Respectfully yours



E. Scott Pruitt

Exhibit 4



E. SCOTT PRUITT
ADMINISTRATOR

June 6, 2017

The Honorable John Carney
Governor of Delaware
Legislative Hall
Dover, Delaware 19901

Dear Governor Carney:

I am writing to update you on the status of the U.S. Environmental Protection Agency's efforts related to the National Ambient Air Quality Standards (NAAQS) for ozone promulgated in October 2015. Pursuant to section 107(d)(1)(B) of the Clean Air Act (CAA), I am extending the deadline for promulgating initial area designations for the 2015 ozone NAAQS by one year. I have determined that there is insufficient information, and taking additional time is appropriate in order to consider completely all designation recommendations provided by state governors pursuant to CAA section 107(d)(1)(A) and to rely fully on the most recent air quality data. This additional time will also provide the Agency time to complete its review of the 2015 ozone NAAQS, prior to taking this initial implementation step.

Although the new ozone standard was set on October 1, 2015, there remains a host of complex issues that could undermine associated compliance efforts by states, localities and regulated entities. As part of the review process, the Agency is evaluating these issues primarily focusing on: fully understanding the role of background ozone levels; appropriately accounting for international transport; and, timely consideration of exceptional events demonstrations. Additionally, pursuant to language in the recently-enacted FY 2017 omnibus bill, I have established an Ozone Cooperative Compliance Task Force to develop additional flexibilities for states to comply with the ozone standard.

States have made tremendous progress and significant investment cleaning up the air. Since 1980, total emissions of the six principal air pollutants have dropped by 63 percent and ozone levels have declined by 33 percent. Despite the continued improvement of air quality, costs associated with compliance of the ozone NAAQS have significantly increased. I am committed to working with you and your local officials to effectively implement the ozone standard in a manner that is supportive of your air quality improvement efforts, without interfering with local decisions or impeding economic growth.

I appreciate the information you and your staff have shared with EPA already as part of this process. I am confident this progress will continue as we work together towards our shared goal of clean air, a robust economy and stronger, healthier communities. If you have questions or concerns, please contact me or your staff may contact Troy Lyons, Associate Administrator for the Office of Congressional and Intergovernmental Relations, at lyons.troy@epa.gov or (202) 564-4987.

Respectfully yours

A handwritten signature in black ink, appearing to read "E. Scott Pruitt", with a large, sweeping flourish extending to the right.

E. Scott Pruitt

Exhibit 5



E. SCOTT PRUITT
ADMINISTRATOR

June 6, 2017

The Honorable Andrew Cuomo
Governor of New York
State Capitol
Albany, New York 12224

Dear Governor Cuomo:

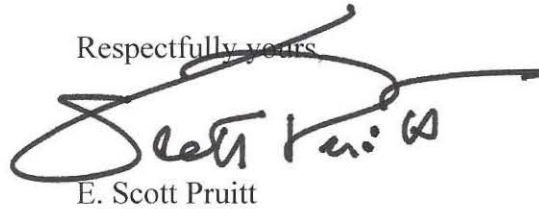
I am writing to update you on the status of the U.S. Environmental Protection Agency's efforts related to the National Ambient Air Quality Standards (NAAQS) for ozone promulgated in October 2015. Pursuant to section 107(d)(1)(B) of the Clean Air Act (CAA), I am extending the deadline for promulgating initial area designations for the 2015 ozone NAAQS by one year. I have determined that there is insufficient information, and taking additional time is appropriate in order to consider completely all designation recommendations provided by state governors pursuant to CAA section 107(d)(1)(A) and to rely fully on the most recent air quality data. This additional time will also provide the Agency time to complete its review of the 2015 ozone NAAQS, prior to taking this initial implementation step.

Although the new ozone standard was set on October 1, 2015, there remains a host of complex issues that could undermine associated compliance efforts by states, localities and regulated entities. As part of the review process, the Agency is evaluating these issues primarily focusing on: fully understanding the role of background ozone levels; appropriately accounting for international transport; and, timely consideration of exceptional events demonstrations. Additionally, pursuant to language in the recently-enacted FY 2017 omnibus bill, I have established an Ozone Cooperative Compliance Task Force to develop additional flexibilities for states to comply with the ozone standard.

States have made tremendous progress and significant investment cleaning up the air. Since 1980, total emissions of the six principal air pollutants have dropped by 63 percent and ozone levels have declined by 33 percent. Despite the continued improvement of air quality, costs associated with compliance of the ozone NAAQS have significantly increased. I am committed to working with you and your local officials to effectively implement the ozone standard in a manner that is supportive of your air quality improvement efforts, without interfering with local decisions or impeding economic growth.

I appreciate the information you and your staff have shared with EPA already as part of this process. I am confident this progress will continue as we work together towards our shared goal of clean air, a robust economy and stronger, healthier communities. If you have questions or concerns, please contact me or your staff may contact Troy Lyons, Associate Administrator for the Office of Congressional and Intergovernmental Relations, at lyons.troy@epa.gov or (202) 564-4987.

Respectfully yours,

A handwritten signature in black ink, appearing to read "E. Scott Pruitt", is written over the typed name. The signature is stylized and somewhat cursive.

E. Scott Pruitt

Exhibit 6



Administrator Extends Deadline for Area Designations for 2015 Ozone Standards

In a letter to Governors, Administrator Pruitt extended by one year the deadline for promulgating initial areas designations for the 2015 ozone National Ambient Air Quality Standards. A sample letter is attached here.

You may need Adobe Reader to view files on this page. See EPA's [About PDF page](#) to learn more.

- [Letter to Governor Doug Ducey, AZ \(PDF\)](#) (2 pp, 349 K, 06/06/2017)

LAST UPDATED ON JUNE 7, 2017

Exhibit 7



News Releases from Headquarters

EPA to Extend Deadline for 2015 Ozone NAAQS Area Designations

06/06/2017

Contact Information:

U.S. EPA Office of Media Relations (press@epa.gov)

WASHINGTON – U.S. Environmental Protection Agency (EPA) Administrator Scott Pruitt sent a letter to governors today to inform them of EPA’s efforts related to the National Ambient Air Quality Standards (NAAQS) for ozone promulgated in October 2015. EPA is extending the deadline for promulgating initial area designations, by one year, for the 2015 ozone NAAQS.

“States have made tremendous progress and significant investment cleaning up the air. We will continue to work with states to ensure they are on a path to compliance,” **said Administrator Scott Pruitt.**

The National Ambient Air Quality Standard (NAAQS) for ground-level ozone is an outdoor air regulation under the Clean Air Act. As part of the process to determine what areas of the country are able to meet the current air quality standards, states are currently submitting their proposals for area designations under the 70 parts per billion (ppb) standard, which was lowered from 75 ppb in 2015. Areas designated as being in “nonattainment” of the standard face consequences, including: increased regulatory burdens, restrictions on infrastructure investment, and increased costs to businesses.

EPA is giving states more time to develop air quality plans and EPA is looking at providing greater flexibility to states as they develop their plans. And, pursuant to the language in the recently-enacted FY2017 Omnibus funding bill, Administrator Pruitt is establishing an Ozone Cooperative Compliance Task Force to develop additional flexibilities for states to comply with the ozone standard.

Additionally, the Agency is taking time to better understand some lingering, complicated issues so that air attainment decisions can be based on the latest and greatest information. This additional time will also provide the agency time to review the 2015 ozone NAAQS, prior to taking this initial implementation step.

Although the new ozone standard was set on October 1, 2015, there remains a host of complex issues that could undermine associated compliance efforts by states and localities. The Agency is evaluating these issues, primarily focused on:

- Fully understanding the role of background ozone levels;

- Appropriately accounting for international transport,

- And, timely consideration of exceptional events demonstrations.

“We share the goal of clean air, a robust economy and stronger, healthier communities. We are committed to working with states and local officials to effectively implement the ozone standard in a manner that is supportive of air quality improvement efforts without interfering with local decisions or impeding economic growth,” **said Administrator Pruitt.**

Since 1980, total emissions of the six principal air pollutants have dropped by 63 percent and ozone levels have declined by 33 percent. Despite the continued improvement of air quality, costs associated with compliance of the ozone NAAQS have significantly increased.

Read the letter sent to governors: <https://www.epa.gov/ozone-designations/administrator-extends-deadline-area-designations-2015-ozone-standards>

Visit ozone designations page: <https://www.epa.gov/ozone-designations>

R107

LAST UPDATED ON JUNE 7, 2017

Exhibit 8



UNITED STATES ENVIRONMENTAL PROTECTION AGENCY

REGION IX

75 Hawthorne Street
San Francisco, CA 94105-3901

MAY 30 2017

OFFICE OF THE
REGIONAL ADMINISTRATOR

Ms. Charlene Albee, Director
Washoe County Health District
Air Quality Management Division
1001 East 9th Street, Building B
Reno, NV 89512

Dear Ms. Albee:

I am pleased to concur with your determination that six exceedances of the 2015 8-hour ozone (O₃) National Ambient Air Quality Standard (NAAQS) at the Reno3 monitor meet the criteria for an exceptional event in the Exceptional Events Rule (EER).

The Washoe County Health District's submittals, dated November 10, 2016 and April 14, 2017, included documentation that the exceedances on August 18, 19, and 21, 2015, and July 2-4, 2016 were caused by exceptional events due to wildfire emissions. After thoroughly reviewing the information you provided, we agree that your submittals meet the demonstration criteria and the schedule and procedural requirements in the EER.

The basis for our concurrence is set forth in the enclosed technical support documents. My staff will enter concurrence flags for these data into EPA's Air Quality System (AQS) database. With EPA's concurrence on the six days, the 2016 design value for the Reno3 monitor (AQS ID: 32-031-0016) does not violate the 2015 8-hour O₃ NAAQS.

EPA's concurrence is a preliminary step in the regulatory process for actions that may rely on these data and does not constitute final Agency action. If EPA completes a notice-and-comment rulemaking for an action that is influenced by the exclusion of the O₃ data specified in this concurrence, EPA's concurrence letter and accompanying technical support documents would be included in the record as part of the technical basis for the proposed action. If we receive comments, we must consider and respond to those comments before taking final regulatory action. When EPA issues that regulatory action, it is a final Agency action subject to judicial review.

If you have any questions or wish to discuss this matter further, please contact Elizabeth Adams, Acting Director of the Air Division, at (415) 972-3183.

Sincerely,


Alexis Strauss
Acting Regional Administrator

Enclosures

cc (via email): Danilo Dragoni, Nevada Division of Environmental Protection
Daniel Inouye, Washoe County Health District

Exhibit 9



Dannel P. Malloy

GOVERNOR
STATE OF CONNECTICUT

June 20, 2017

The Honorable E. Scott Pruitt
Administrator
U.S. Environmental Protection Agency
William Jefferson Clinton Building
1200 Pennsylvania Ave, NW
Mail Code: 1101A
Washington, DC 20460

RE: Objection to Delay of Designations for the 2015 Ozone Standard

Dear Administrator Pruitt:

I am dismayed by your June 6th letter announcing a delay in implementation of the designation phase of the 2015 ozone National Ambient Air Quality Standards (NAAQS). Your decision is deeply troubling because it will postpone important progress to improve air quality in my home state of Connecticut, as well as states across the nation.

Pushing back the timeframe for putting in place this new, more stringent standard prolongs the risk of illness – and even death – for thousands of residents in my state who are most sensitive to unhealthy levels of ozone in our air. In addition to placing our health at risk, delay in addressing high ozone levels created by the interstate transport of air pollution from upwind states to Connecticut undermines our economy. Finally, your purported rationale for this deferral has little— if any—basis in law or fact.

Delay puts the health of Americans at risk

Noticeably absent from your letter is any mention of protection of public health – but your own science and our experience here in Connecticut demonstrate that there are real human costs to postponing the implementation of better standards. Your decision to extend the Environmental Protection Agency's (EPA's) deadline for promulgating initial area designations under the 2015 ozone NAAQS by a full year will push back long-awaited remedial actions required to clean the air Connecticut residents breathe every hour of every day. Your action is the latest evidence of EPA's failure to address ozone levels and to require all states to limit the discharge of air pollution within their borders.

Here in Connecticut we take this issue seriously. As you may recall, during this past winter's convening of the National Governors Association, I presented you with the fact that my state is already designated nonattainment for ozone and that we measure the highest levels of ozone in the Northeast. This is, however, through no fault of our own. More than 90% of this pollution blows into our state from other places. In fact, on some days, every power plant and factory in our state could shut down and Connecticut would still exceed the ozone NAAQS.

This point is worthy of repetition—if every power plant and factory within the boundaries of our state ceased operations, our air would still exceed the ozone NAAQS because of air pollution created in *other* states.

This is not fair to the people of Connecticut and it puts the health of our population at severe risk. We urge you to move faster – not slower – to implement the 2015 ozone standard so that concentrations of pollution in our air will be less of a threat to our residents.

High ozone levels have been directly linked to illnesses such as asthma, and in Connecticut, children, women, Hispanics, blacks, and residents of Connecticut's five largest cities are disproportionately affected by this condition. Connecticut is above the national average in asthma sufferers, and in 2014, Connecticut incurred over \$135 million in acute care charges due to asthma.

Delay hurts the state economy

Your letter also talks about not wanting to “imped[e] economic growth.” But delay means that Connecticut – a state where extensive investments have been made to limit pollution – will remain at a competitive economic disadvantage against states that ignore critical air quality issues in an effort to maintain a low-cost environment for their businesses.

In a nonattainment area, industries face additional administrative and air pollution control costs when applying for a new permit or expanding their business. Industries in our state are required to install the most stringent emissions controls in the country when building a plant, and must purchase pollution credits to offset new emissions. This can add millions of dollars to the cost of doing business.

The cost of removing additional pollution in Connecticut, where we already have stringent requirements in place, is estimated at \$10,000 to \$40,000 per ton. Compare this to the estimated cost of as little as \$500 to \$1,200 per ton it takes to remove the same amount of pollution in upwind states, where even some basic control technologies have not been installed at various facilities.

Delay contradicts the facts

Your argument for the delay is weak and unjustified. Contrary to claims in your June 6 letter, the EPA has all of the information and authority it needs to act.

Specifically, ambient ozone concentrations measured by over 1,100 monitoring stations located throughout the country were certified and submitted to EPA by May 1, 2016. States submitted

their designation recommendations as required by the October 2016 deadline. There is nothing missing from past information used by EPA to designate areas after previous revisions to the ozone NAAQS. Your agency is currently in possession of all the necessary information to promulgate the required initial designations immediately.

The other factors cited in your letter – background ozone levels, international transport, and exceptional event demonstrations – are immaterial to the initial area designations, but instead are considerations for the next phase—the implementation phase. In fact, Congress addressed these matters through separate provisions distinctly different from area designation requirements,¹ which provide EPA adequate flexibility to address the implementation considerations without delaying the significant public health benefits that your own Regulatory Impact Analysis shows greatly outweigh the costs of implementation.²

For the sake of public health and economic fairness, I urge you to reconsider the unnecessary one-year extension and to promulgate initial attainment and nonattainment designations under the 2015 ozone NAAQS as required by CAA section 107(d)(1)(B), and finalize the implementation rule proposed on November 16, 2016.

Connecticut looks forward to working with EPA to achieve our shared air quality goals while also ensuring that the necessary emissions reductions are secured in an equitable manner from contributing states.

Sincerely,



Dannel P. Malloy
Governor, State of Connecticut

¹ Specifically, Clean Air Act section 179B related to international transport, and section 182(h) related to rural transport areas, apply to state implementation plans or revisions that are due after an area's designation. Section 179B has in fact already been successfully applied by Texas to address international transport after the El Paso area was designated nonattainment. See "Approval and Promulgation of Implementation Plans for Texas; Approval of Section 179B Demonstration of Attainment, Volatile Organic Compounds and Nitrogen Oxides Motor Vehicle Emissions Budgets for Conformity for the El Paso Ozone Nonattainment Area." 69 Federal Register 32450-32454 (June 10, 2004).

² See EPA-452/R-15-007; September 2015. The Regulatory Impact Analysis concludes that the revised standard will avoid 320-660 premature deaths annually and provide important benefits for those with asthma and chronic lung disease, avoiding 230,000 asthma attacks, 160,000 lost school days, and 28,000 missed work days each year. Overall, EPA estimates annual benefits of \$2.9-\$5.9 billion, outweighing estimated annual costs of \$1.4 billion. The one-year designation delay will unnecessarily cause substantial health and economic damages while needlessly deferring benefits. These are EPA's national estimates (excluding California) for 2025.

Exhibit 10



UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

FEB 25 2015

OFFICE OF
AIR AND RADIATION

MEMORANDUM

SUBJECT: Area Designations for the 2015 Ozone National Ambient Air Quality Standards

FROM: Janet G. McCabe *JGM*
Acting Assistant Administrator

TO: Regional Administrators
Regions 1-10

The purpose of this guidance is to provide information on the schedule and process for initially designating areas for the purpose of implementing the 2015 primary and secondary ozone national ambient air quality standards (NAAQS). In addition, this memorandum identifies important factors that the Environmental Protection Agency intends to evaluate in making final nonattainment area boundary decisions for these standards. The EPA recommends that states and tribes also consider these factors in making their recommendations for area designations and nonattainment area boundaries. As for designations for prior ozone NAAQS, the EPA will also consider any other relevant information in making designation determinations. Please share this memorandum with state and tribal air agencies in your region.

On October 1, 2015, the EPA promulgated revised primary and secondary ozone NAAQS (80 FR 65292, October 26, 2015). In that action, the EPA strengthened both standards to a level of 0.070 parts per million, while retaining their indicators, averaging times, and forms. The EPA revised the ozone standards based on an integrated assessment of an extensive body of new scientific evidence, which substantially strengthens our knowledge regarding ozone-related health and welfare effects, the results of exposure and risk analyses, the advice of the Clean Air Scientific Advisory Committee, and consideration of public comments.

The revised primary standard provides increased protection for children, older adults, and people with asthma or other lung diseases, and other at-risk populations against an array of adverse health effects including reduced lung function, increased respiratory symptoms and pulmonary inflammation and asthma exacerbations; effects that contribute to emergency department visits or hospital admissions; and mortality. The revised secondary standard provides protection of natural forests from adverse growth-related effects and is expected to provide increased protection from other effects of potential public welfare significance, including crop yield loss and visible foliar injury.

Clean Air Act Designation Requirements

Section 107(d) of the Clean Air Act (CAA) governs the process for initial area designations after the EPA establishes a new or revised NAAQS. Under section 107(d) of the CAA, states are required to submit area designation recommendations to the EPA. This submission must happen by a date specified by the EPA, which cannot be sooner than 120 days, or later than 1 year, after promulgation of the new or revised NAAQS. If, after careful consideration of these recommendations, the EPA intends to promulgate a designation different from a state's recommendation, then the EPA must notify the state at least 120 days prior to promulgating the final designation and must provide the state an opportunity to comment on the intended modification. The EPA may choose to modify a state's recommended designation as it relates to the status of an area or as it relates to the boundaries of an area. The CAA requires the EPA to complete the initial designation process within 2 years of promulgation of a new or revised NAAQS, unless the Administrator has insufficient information to make initial designation decisions in the 2-year time frame. In such circumstances, the EPA may take up to 1 additional year to make initial area designation decisions (i.e., no later than 3 years after promulgation of the standard). While section 107(d) of the CAA specifically addresses the designations process between the EPA and states, the EPA intends to follow the same process to the extent practicable for tribes that choose to make initial designation recommendations pursuant to section 301(d) of the CAA regarding tribal authority and the Tribal Authority Rule (TAR) (63 FR 7254, February 12, 1998). To provide clarity and consistency in doing so, in December 2011, the EPA issued a guidance memorandum concerning the involvement of tribes in the designations process.¹ In accordance with the TAR and the December 2011 tribal designations guidance, and in consultation with the tribes, the EPA intends to designate tribal areas on the same schedule as designations for states. If a state or tribe does not submit designation recommendations, then the EPA will promulgate the initial designations that the agency deems appropriate.

Schedule for Initial Ozone Area Designations

State governors should submit, and tribes can choose to submit, their initial designation recommendations for the 2015 ozone NAAQS to the EPA no later than 1 year following promulgation of the revised NAAQS, i.e., by October 1, 2016. Because the form of the 2015 ozone NAAQS relies on a 3-year average, we recommend that states and tribes base their recommendations on air quality data from the 3 most recent years of quality assured monitoring data available at that time, i.e., 2013 to 2015. However, states and tribes may also have preliminary information about 2016 monitoring data that could help inform their recommendations. Based upon these monitoring data and any other available information, states and tribes should identify areas as attainment, nonattainment, or unclassifiable.² If

¹ Guidance to Regions for Working with Tribes during the National Ambient Air Quality Standards (NAAQS) Designations Process. Memorandum from Stephen D. Page, Director, EPA OAQPS to Regional Administrators, Regions I-X. December 20, 2011. Available at <http://www.epa.gov/ttn/oarpg/t1/memoranda/20120117naaqsguidance.pdf>.

² For the initial area designations for the 1997 ozone NAAQS and the 2008 ozone NAAQS, the EPA used a designation category of "unclassifiable/attainment" for areas that were monitoring attainment and for areas that did not have monitors but for which the EPA had reason to believe were likely attainment and were not contributing to nearby violations. The EPA reserved the category "unclassifiable" for areas where the EPA could not determine based on available information whether the area was meeting or not meeting the NAAQS and the EPA had not determined that the area contributed to a nearby violation. While states can submit recommendations identifying areas as "attainment," the EPA expects to continue to use the "unclassifiable/attainment" category for designations for the 2015 ozone NAAQS.

the EPA believes it is necessary to make any modifications to a state's or tribe's initial recommendations, including area boundaries, then the EPA will notify the state or tribe by letter of the intended modification no later than 120 days prior to finalizing the designation. These notifications are commonly known as the "120-day letters." Consistent with the statutory requirement that the EPA designate areas no later than 2 years following promulgation of a revised NAAQS, the EPA expects to complete the initial area designations by October 1, 2017. Thus, the EPA intends to issue the 120-letters no later than June 2, 2017. If a state or tribe has additional information that it wants the EPA to consider with respect to a designation recommendation that the EPA plans to modify, then the EPA requests that such information be submitted no later than 60 days from the date of the 120-day letter. This schedule will ensure that the EPA can fully consider any such additional information prior to issuing final designations. Also, although section 107(d) of the CAA explicitly exempts the designation process from the public notice and comment rulemaking process, the EPA intends to consider public input in the designation process. Accordingly, we plan to provide a 30-day public comment period immediately following issuance of the 120-day letters responding to the designation recommendations from states and tribes.³ Attachment 1 summarizes this anticipated schedule.

Identifying Nonattainment Areas

Section 107(d)(1) of the CAA directs the EPA to designate an area "nonattainment" if it is violating the NAAQS or if it is contributing to a violation of the NAAQS in a nearby area. Thus, the first step in the designation process is to identify air quality monitoring sites with data that show a violation of the 2015 ozone NAAQS. Violations are identified using data from Federal Reference Method (FRM) and Federal Equivalent Method (FEM) monitors that are sited and operated in accordance with 40 CFR part 58. Procedures for using the air quality data to determine whether a violation has occurred are given in 40 CFR part 50 Appendix U, as revised in conjunction with the final rule for the 2015 ozone NAAQS (80 FR 65292, October 26, 2015). For designations for the 2015 ozone NAAQS, the EPA intends to evaluate areas using the most recent complete three consecutive calendar years of quality-assured, certified air quality data in the EPA Air Quality System (AQS).⁴ In accordance with 40 CFR 58.15, states are required to certify their air monitoring data for the previous year by May 1 of each year. Although generally the EPA will use such data only if they have been certified by the reporting organization, data not certified by the reporting organization can nevertheless be used if the deadline for certification has passed and the EPA judges the data to be complete and accurate. We expect that in providing designation recommendations to the EPA by October 1, 2016, states and tribes will review and rely on air quality data from 2013 to 2015. States and tribes may also review and consider preliminary 2016 data, although those data cannot be relied on until they are either certified in accordance with 40 CFR 58.15 or the date for certification has passed. Air quality monitoring data from 2016 are required to be certified and quality assured by May 1, 2017. Because the certification date will have passed and the data will be available, the EPA expects to base final designation decisions by October 1, 2017, on data

³ Section 107(d)(2) explicitly provides that designations are exempt from the notice and comment provisions of the Administrative Procedure Act (APA). Likewise, designations under section 107(d) of the CAA are not among the list of actions that are subject to the notice and comment procedures of CAA section 307(d). Thus, neither the CAA nor the APA require notice and comment rulemaking for promulgation of the designations for these or any other NAAQS. However, the EPA intends to solicit direct public comment on its preliminary responses to the initial area designation recommendations of the states and tribes because we believe this process will be useful to gather additional information and to assure that the agency is more directly aware of issues raised by initial area designations.

⁴ This information is available on the EPA's website at <http://www2.epa.gov/aqs>.

from 2014 to 2016.⁵ For this reason, the EPA encourages states and tribes to review and consider preliminary 2016 air quality data in their designation recommendations. States and tribes may also update their designation recommendations based on 2016 data once the data have met the certification requirements.

The EPA notes that in past designations, some states have chosen to certify air quality data prior to the certification deadline (i.e., “early certify”) so that the EPA could rely on the newer data for designations. For multistate nonattainment areas, there have been situations where some, but not all, of the states with portions in the area have chosen to early certify their data. In such cases, the “most recent air quality data” for the area is a mix of two different 3-year periods – an earlier time period for those states that did not early certify data and a later time period for those states that chose to early certify. The most common situation is where one state that is part of the multistate area early certifies data that show attainment of the NAAQS. The other is where one state early certifies data that show a violation. The EPA’s position is that the agency cannot review mixed years of data to conclude that an area is attaining the standard; the decision must be based on the same 3-year period for all portions of the area. In contrast, if the early certified data for one state’s portion of a multistate area indicate a violation of the NAAQS, the EPA’s position is that the agency must consider the violating monitor and assess what nearby areas contribute to the violation.⁶

The process for evaluating the appropriate designation for areas that are not violating the NAAQS, but may be contributing to the violations of the NAAQS in a violating area, is discussed below in connection with the process for determining appropriate nonattainment area boundaries.

Exceptional Events and Designations

When certain criteria are met, the CAA and the EPA’s implementing regulations specified in the Final Rule on the Treatment of Data Influenced by Exceptional Events (72 FR 13560, March 22, 2007)⁷ allow for the exclusion of air quality monitoring data from design value calculations when there are exceedances caused by exceptional events. A design value describes the air quality status of a given location relative to the level of the NAAQS. A design value calculated using a data set from which exceptional event-influenced data have been excluded has the potential to affect initial area designations and nonattainment area classifications for the 2015 ozone NAAQS.

In the 2015 ozone NAAQS final rule, the EPA established schedules for air agencies to flag data influenced by exceptional events and submit related documentation for data that will be used in the initial designations process for the 2015 ozone NAAQS (*see* Attachment 2). Although some of these deadlines are accelerated compared to the general schedule timelines in the 2007 Exceptional Events

⁵ In the final rule for the 2015 ozone NAAQS, the EPA also finalized changes to the ambient air monitoring requirements applicable to the ozone NAAQS. In 32 states and the District of Columbia, the final rule extends the ozone season. The new ozone season requirements do not take effect until January 1, 2017.

⁶ The Court of Appeals for the D.C. Circuit upheld this approach as reasonable. *Miss. Comm’n on Env’tl. Quality v. EPA*, 790 F.3d 138, 160 (D.C. Cir. 2015).

⁷ On November 10, 2015, the EPA proposed revisions to the 2007 Exceptional Events Rule and announced the availability for public comment of a draft guidance document, which applies the proposed rule revisions to wildfire events that could influence monitored ozone concentrations. *See* 80 FR 72840, November 20, 2015. The EPA intends to finalize these rule revisions and the wildfire guidance by the October 1, 2016, date by which states, and any tribes that wish to do so, are required to submit their initial designation recommendations for the 2015 ozone NAAQS.

Rule, they were promulgated to align closely with the timing of the initial designations recommendations from states and tribes in October 2016 and/or the EPA's expected issuance of 120-day letters pertaining to designations by June 2017. These schedules reflect the EPA's interest in ensuring that we can fully consider exceptional events claims that could influence the final designations decisions.

The EPA regional offices are encouraged to work with states and tribes with exceptional events claims to prioritize and expedite the demonstration development and review process for those claims that have the potential to influence regulatory decisions, such as the initial designations process. Similarly, the EPA encourages states and tribes to contact and collaborate with the appropriate EPA regional office after identifying any exceptional events that influence ambient air quality concentrations in a way that could potentially affect designations for the 2015 ozone NAAQS. The EPA has developed interim exceptional events implementation guidance documents that air agencies can use when reviewing potential exceptional events and developing appropriate exceptional event demonstrations. Additional information and examples of exceptional event submissions and best practice components can be found at the EPA's exceptional events website located at <http://www2.epa.gov/air-quality-analysis/treatment-data-influenced-exceptional-events>.

Nonattainment Area Analyses and Boundary Determination

The EPA believes that the boundaries for each nonattainment area should be evaluated and determined on a case-by-case basis considering the specific facts and circumstances unique to the area. Section 107(d) explicitly requires that the EPA designate as nonattainment not only the area that is violating the pertinent standard, but also those nearby areas that contribute to the violation in the violating area. After identifying each monitor that indicates a violation of the 2015 ozone NAAQS in an area, the EPA will determine which nearby areas contribute to the violation(s).

Ground-level ozone is not emitted directly into the air, but is formed by chemical reactions primarily between oxides of nitrogen (NO_x) and volatile organic compounds (VOC) that are attributable to a variety of emission sources commonly found throughout urbanized areas. Because ozone and its precursor emissions are pervasive and readily transported, the EPA believes it is important to examine ozone-contributing emissions across a relatively broad geographic area associated with a monitored violation. Thus, for analyzing whether nearby areas contribute to a violating area, the EPA intends to consider information relevant to designations associated with the counties in the Combined Statistical Area (CSA) or, where appropriate, the Core Based Statistical Area (CBSA) in which the violating monitor(s) are located. The CSAs and CBSAs are delineated by the Office of Management and Budget (OMB) as part of their Metropolitan and Micropolitan Statistical Area program.⁸ The CBSA is a collective term that refers to both Metropolitan Statistical Areas (MSAs) and Micropolitan Statistical Areas (Micropolitan Areas), which are distinguished by size. An MSA has at least one urban area with a population of at least 50,000. A Micropolitan Area has at least one urban area with a population of at

⁸ OMB adopted revised standards for defining Metropolitan and Micropolitan Statistical Areas on December 27, 2000 (65 FR 82229). These standards established the terms CSA and CBSA. In 2010, OMB further revised the standards for delineating Metropolitan and Micropolitan Statistical Areas (75 FR 37246, June 28, 2010). The statistical areas are delineated based on U.S. Census Bureau information. The EPA intends to use the 2010 standards and the associated lists of CSAs and CBSAs issued in February 2013. These lists and their geographic components are provided at <http://www.census.gov/population/metro/>.

least 10,000, but less than 50,000. Each CBSA consists of a county or counties associated with at least one urban core, plus adjacent counties having a high degree of social and economic integration with the core as measured through commuting ties with the counties containing the core.⁹ A CSA includes two or more adjacent CBSAs.

The EPA previously reviewed relevant information associated with OMB statistical area boundaries when analyzing nonattainment areas for the 1997 and 2008 ozone standards. We believe this is a reasonable approach to ensure that the nearby areas most likely to contribute to a violating area are evaluated.¹⁰ The EPA emphasizes it does not intend the statistical area boundary to be a presumed nonattainment area boundary. The area-specific analyses may support nonattainment boundaries that are smaller or larger than the CSA or CBSA.¹¹ Where a violating monitor is not located in a CSA or CBSA, the EPA intends to review relevant information associated with the county containing the monitor and, if appropriate, other adjacent nearby counties. The EPA will determine the nonattainment area boundaries through a weight-of-evidence analysis for the area based on synthesizing the assessments of the five factors identified below. In relatively urbanized areas, the nonattainment area boundary may include an entire metropolitan area. In rural locations, the nonattainment area boundary may include one or more small population centers, each with sources that contribute to a violating monitor. In some cases, the boundary for a nonattainment area may include portions of two or more states, thus resulting in a multistate area. This approach to designations has been upheld by numerous courts under a variety of challenges.

Consistent with past designations for ozone NAAQS, for area-specific analyses through which the EPA intends to determine area boundaries, the EPA will evaluate information relevant to five factors: air quality data, emissions and emissions-related data, meteorology, geography/topography, and jurisdictional boundaries. The EPA also recommends that states and tribes base their boundary recommendations on an evaluation of information relevant to these five factors. Attachment 3 describes these factors in general and provides guidance regarding analyses relevant to each of these factors.¹² Additionally, the EPA, states and tribes may identify and evaluate other relevant information or circumstances specific to a particular area to support nonattainment area boundary recommendations.

⁹ The geographic components of CBSAs are counties and equivalent entities (boroughs and census areas in Alaska, parishes in Louisiana, independent cities in Maryland, Missouri, Nevada, and Virginia, and municipios in Puerto Rico).

¹⁰ The EPA notes that for the purpose of the designations for the 1-hour ozone standards at the time the CAA was amended in 1990, CAA section 107(d)(4)(A)(iv) and (v) specified the use of the OMB statistical areas as the boundaries that applied by operation of law for the then-existing nonattainment areas classified as Serious, Severe, and Extreme, unless a governor made a demonstration to the satisfaction of the EPA Administrator that a portion did not contribute.

¹¹ The Court of Appeals for the D.C. Circuit upheld the EPA's interpretation of the term "nearby" as being reasonable and consistent with the statute. *Miss. Comm'n on Env'tl. Quality v. EPA*, 790 F.3d 138, 160 (D.C. Cir. 2015).

¹² In the designation guidance for the 2012 PM_{2.5} NAAQS, the EPA used these same five factors. In prior designation guidance for the ozone and PM_{2.5} standards, the EPA identified nine factors to consider in making designation recommendations: emissions data, air quality data, population density and degree of urbanization, traffic and commuting patterns, growth rates and patterns, meteorology, geography/topography, jurisdictional boundaries, and level of control of emission source. In the area analyses to support the designations for the 2008 ozone standards, the EPA grouped the emissions-related factors together in the emissions and emissions-related data factor, resulting in five overall factors. The Court has upheld the EPA's use of a multi-factor test for designations multiple times. See *Mississippi Commission on Env. Quality v. EPA*, 709 F.3d 138 (D.C. Cir. 2015); *ATK Launch Sys., Inc. v. EPA*, 669 F.3d 330 (D.C. Cir. 2012); *Catawba Cnty., v. EPA*, 571 F.3d 20 (D.C. Cir. 2009).

While the EPA generally believes it is appropriate to include the entire violating or contributing county in an ozone nonattainment area, we recognize that, in some cases, an assessment of relevant information may support inclusion of only part of a county. For example, as has been the case in past designations, there may be low elevation areas (e.g., valleys) with poor air quality in violation of the NAAQS due to restricted atmospheric dispersion where higher elevations (e.g., mountainous areas) in the same county can be shown not to have sources of emissions that contribute to the violation. Alternatively, partial county boundaries may be appropriate in situations where the sources located in a contributing county are located only in a portion of a large county that is otherwise not contributing to the nearby violations. Particularly in the western United States where counties are large, including only partial counties in a designated nonattainment area may be appropriate. For defining partial county boundaries, the EPA recommends the use of well-defined legal jurisdictional boundaries such as townships, census blocks, immovable landmarks (e.g., major roadways), or other permanent and readily identifiable boundaries.

In addition, as provided for in the December 20, 2011, guidance titled, “Policy for Establishing Separate Air Quality Designations for Areas of Indian Country,” tribes may recommend that the EPA designate areas of Indian country separately from the adjacent state areas.¹³ This guidance provides for a nationally consistent approach for evaluating such designation recommendations from tribes. The policy was designed to recognize tribal sovereignty in air quality management matters affecting Indian country.

Nonattainment Area Classifications

As provided in CAA section 181(a)(1), at the time of initial designations, the EPA will classify all nonattainment areas according to the severity of the ozone air quality problem. The classification categories are Marginal, Moderate, Serious, Severe-15, Severe-17 and Extreme. The EPA previously interpreted the air quality thresholds associated with each classification through rulemaking for both the 1997 and 2008 ozone NAAQS. We intend to take a similar approach for the 2015 ozone NAAQS and will finalize the rulemaking no later than the promulgation of the final designations.

Under CAA section 181(a)(4), the EPA has the discretion to reclassify a nonattainment area to a higher or lower classification (also known as a bump up or a bump down) within 90 days of the effective date of the initial designation and classification if the area would have been classified in another category had the area’s design value been 5 percent greater or 5 percent less than the level on which the initial classification was based. The EPA does not intend to exercise its authority independently to initiate a reclassification of an area to a higher or lower classification. Rather, the EPA intends to rely on a state or tribe to submit a request for such a reclassification. As part of the action to designate and classify areas in 1991 for the 1-hour NAAQS, the EPA developed criteria for evaluating a state’s request to reclassify a particular area to a lower classification. *See* 56 FR 56698, November 6, 1991. The EPA intends to continue to use the same approach for purposes of evaluating a request to reclassify an area to a lower classification for the 2015 ozone NAAQS. In the *Federal Register* action to designate areas for the 2015 ozone NAAQS, the EPA will provide the schedule for submitting a reclassification request under section 181(a)(4) that would allow sufficient time for the EPA to make a determination within the 90-day period allowed under the CAA.

¹³ Memorandum from Stephen D. Page, Director, EPA OAQPS to Regional Administrators, Regions I-X. December 20, 2011. Available at <http://www.epa.gov/ozone-designations>.

Section 181(b)(3) of the CAA allows a state to voluntarily request that the EPA reclassify a nonattainment area in that state to a higher classification. The EPA must grant the request. Multistate nonattainment areas present a special case because the area is not wholly in one state and classifications apply areawide. For multistate nonattainment areas, the EPA strongly encourages all of the states with a portion included in the nonattainment area to consult and agree prior to submission of a reclassification request. Section 181(b)(3) does not place a time limit on the opportunity for a state to request a voluntary reclassification of a nonattainment area to a higher classification. These voluntary reclassifications can be done at any time.

Rural Transport Areas

The EPA recognizes that violations of the ozone standards in some rural areas may be almost entirely attributable to emissions from upwind areas and/or sources of background ozone. Section 182(h) provides the EPA with the discretion to treat an ozone nonattainment area as a “rural transport area” (RTA), provided the area meets certain criteria. Regardless of the area’s classification under section 181(a), an RTA is deemed to have fulfilled all ozone-related planning and control requirements if it meets the CAA’s planning requirements for areas classified as Marginal.¹⁴ To qualify as an RTA, the EPA must determine that the nonattainment area boundary does not include and is not adjacent to any part of an MSA¹⁵, and that the area does not contain VOC and NO_x emissions sources that make a significant contribution to monitored ozone concentrations in the area or in other areas. A nonattainment area that includes, or is adjacent to, any part of a Micropolitan Statistical Area or that is too sparsely populated to be included in a statistical area, may be able to qualify as an RTA.

States and tribes that believe a potential nonattainment area qualifies for treatment as an RTA are encouraged to request, as part of their recommendations, that the EPA use the section 182(h) authority and to work with the EPA to develop and review information that would satisfy the CAA’s RTA criteria. In general, the EPA expects a rural nonattainment area that has few or insignificant sources of ozone precursors to encompass a relatively small geographic area due to the lack of emission sources. Therefore, partial county boundaries may be appropriate. The EPA expects this to be especially relevant in the western United States, where many of the counties are large. A partial county nonattainment area located in a county that is adjacent to an MSA may still be able to qualify as an RTA provided that the nonattainment area boundary is not adjacent to the MSA boundary. The EPA intends to respond to any RTA request submitted during the designation process at the time the EPA promulgates the initial area designations. However, the EPA notes that a state or tribe may also request RTA treatment for a nonattainment area after the initial designations are completed. Attachment 3 provides information on conducting an analysis to support an RTA request.

¹⁴ The requirements applicable to ozone transport regions supersede the Marginal requirements for RTAs.

¹⁵ The rural transport area criteria in section 182(h) restrict rural transport areas to those nonattainment areas that do not include and are not adjacent to any part of a “MSA” or “CMSA” as defined at the time of the 1990 CAA amendments. The OMB issued revised statistical area standards in 2000 that replaced the pre-existing MSA and CMSA definitions and established the terms “CBSAs” and “CSA.” In 2010, OMB further revised the standards. The CBSA is a collective term that includes MSAs and Micropolitan Statistical Areas. The EPA interprets the references to both MSA and CMSA in CAA section 182(h) to refer to OMB’s current definition of MSA. *See* 80 FR 12264, March 6, 2015. The EPA believes this interpretation of CAA section 182(h) is consistent with the original scope of CAA section 182(h) as promulgated in 1990.

Unclassifiable Areas

In certain cases, there may be insufficient information to support a designation of nonattainment or attainment for an area. For example, there may be monitors that indicate an exceedance of the NAAQS, but the monitoring data may be incomplete or the monitors may not be sited and operated in accordance with the regulatory requirements of 40 CFR part 58. In recommending boundaries for an unclassifiable area, states should consider which nearby areas contribute to ambient air quality within the impacted area. The EPA notes that if sufficient information later becomes available indicating a monitor in the unclassifiable area is violating the NAAQS and the EPA redesignates the area to nonattainment, the EPA likely would conduct a weight-of-evidence analysis as described in Attachment 3 of this guidance to determine the appropriate area boundaries.

Attainment Areas

Once the EPA has determined the boundaries for nonattainment areas (areas that are violating the NAAQS or contributing to a nearby violation) and any unclassifiable areas, the EPA intends to designate the remainder of the state as unclassifiable/attainment.¹⁶ The EPA requests that states and tribes recommend how they would like the boundaries drawn for their unclassifiable/attainment areas. For designations for the 1-hour and two previous 8-hour ozone NAAQS, states have elected to draw boundaries for the unclassifiable/attainment areas in a variety of ways, including as “rest of state” or “entire state,” by Air Quality Control Regions, by county, by previous nonattainment area boundaries, or by a combination of methods. The EPA recommends that the boundaries of unclassifiable/attainment areas generally not be smaller than a county.

Summary

This memorandum provides the EPA’s preliminary views on the process for determining initial area designations and boundaries for the 2015 ozone NAAQS. Any guidance contained herein is not binding on states, tribes, the public or the EPA. The EPA will make the designations determinations and nonattainment area boundary decisions in the final action that designates all areas for the 2015 ozone standards. When the EPA promulgates the initial area designations, those decisions will be binding on states, tribes, the public and the EPA as a matter of law.

Three attachments provide additional information relevant to the initial ozone area designations process. Attachment 1 is an anticipated timeline of important milestones in the initial area designations process for the 2015 ozone NAAQS. Attachment 2 identifies the promulgated exceptional event schedule for initial data flagging and submission of exceptional event demonstrations. Attachment 3 provides information on the five factors that the EPA intends to consider in evaluating and making decisions on nonattainment area boundaries and provides guidance regarding analyses relevant to support each of the factors. Attachment 3 also provides information on conducting an analysis to support an RTA request.

¹⁶ As indicated in footnote 2, in the initial designations for previous ozone NAAQS, the EPA used a designation category of “unclassifiable/attainment” for areas that were monitoring attainment and for areas that did not have monitors but for which the EPA had reason to believe were likely attainment and were not contributing to nearby violations. The EPA expects to continue this approach for designations for the 2015 ozone NAAQS.

Staff in the EPA's Office of Air Quality Planning and Standards are available for assistance and consultation throughout the initial area designation process. Questions on this guidance may be directed to Carla Oldham at (919) 541-3347 or Denise Scott at (919) 541-4280.

Attachments (3)

1. Anticipated Timeline For 2015 Ozone NAAQS Designation Process
2. Revised Schedule For Exceptional Event Flagging And Documentation Submission For Data To Be Used In Initial Area Designations For The 2015 Ozone NAAQS
3. Factors the EPA Plans to Consider in Determining Nonattainment Area Boundaries in Designations for the 2015 Ozone NAAQS, and Guidance on Analyses to Support these Factors

ATTACHMENT 1

ANTICIPATED TIMELINE FOR 2015 OZONE NAAQS DESIGNATION PROCESS	
Milestone	Date
The EPA promulgates 2015 Ozone NAAQS rule	October 1, 2015
States and tribes submit recommendations for ozone designations to the EPA	No later than October 1, 2016
The EPA notifies states and tribes concerning any intended modifications to their recommendations (120-day letters)	No later than June 2, 2017 (120 days prior to final ozone area designations)
The EPA publishes public notice of state and tribal recommendations and the EPA's intended modifications, if any, and initiates 30-day public comment period	On or about June 9, 2017
End of 30-day public comment period	On or about July 10, 2017
States and tribes submit additional information, if any, to respond to the EPA's modification of a recommended designation	No later than August 7, 2017
The EPA promulgates final ozone area designations	No later than October 1, 2017

ATTACHMENT 2

Revised Schedule for Exceptional Event Flagging and Documentation Submission for Data to be Used in Initial Area Designations for the 2015 Ozone NAAQS

NAAQS Pollutant/ Standard/(Level)/ Promulgation Date	Air Quality Data Collected for Calendar Year	Event Flagging & Initial Description Deadline	Detailed Documentation Submission Deadline
Ozone/ Primary and Secondary 8-hour Standards (0.070 parts per million) Promulgated October 1, 2015	2013, 2014, 2015	July 1, 2016	October 1, 2016
	2016	May 31, 2017	May 31, 2017

ATTACHMENT 3

Factors the EPA Plans to Consider in Determining Nonattainment Area Boundaries in Designations for the 2015 Ozone NAAQS, and Guidance on Analyses to Support these Factors

For initial area designations for the 2015 ozone national ambient air quality standards (NAAQS), the Environmental Protection Agency will rely on monitoring data to identify areas to be designated nonattainment due to monitored violations of the standard. Consistent with the directives of the Clean Air Act (CAA) and with previous area designation processes, the EPA will then determine the appropriate nearby¹ areas to include within the nonattainment area boundary for the violating area, based on emissions that contribute to these violations. For each monitor or group of monitors indicating a violation of the NAAQS, the EPA intends to assess information related to five factors for the purpose of establishing the appropriate geographic boundaries for designated ozone nonattainment areas. The EPA will evaluate relevant information from the entire area (i.e., Combined Statistical Area / Core Based Statistical Area) containing the violating monitor(s) and any adjacent counties or nearby areas that have the potential to contribute. For those portions of the area where an evaluation of the available information clearly establishes that emissions sources do not contribute to exceedances at the violating monitor(s), the EPA believes it would be appropriate to exclude that portion of the area from the nonattainment area. This weight-of-evidence approach to determining area boundaries could result in nonattainment areas consisting of an entire metropolitan area, single counties, or, in cases supported by relevant evidence, partial counties, including partial counties within larger urban areas or in relatively isolated locations. While technical assessments can help to define the magnitude or relative magnitude of contribution from nearby areas, the EPA is not setting a threshold contribution level or “bright line” test for determining whether a contributing area should be included within the boundaries of a given nonattainment area. Section 107(d) of the CAA does not require the EPA to set a threshold contribution. As was done in prior NAAQS designations, the EPA believes that the contribution determination should be made through a case-by-case evaluation of the relevant facts and circumstances in each nonattainment area.

As a framework for area-specific analyses to support nonattainment area boundary recommendations and final boundary determinations, the EPA believes it is appropriate to evaluate the following five factors:

1. air quality data,
2. emissions and emissions-related data,
3. meteorological data,
4. geography/topography, and
5. jurisdictional boundaries.

The EPA notes that these five factors are comparable to the factors that states and tribes and the EPA have used successfully for analytical purposes in prior designations. The recommendation of these factors is not intended to indicate that other relevant information should not be considered in the initial area designations process, as appropriate. Where a state or tribe includes additional information or analysis as part of its recommendation, the EPA will evaluate that information as part of its review in determining the appropriate nonattainment area designation.

¹ The Court of Appeals for the D.C. Circuit upheld the EPA’s interpretation of the term “nearby” as being reasonable and consistent with the statute. *Miss. Comm’n on Envtl. Quality v. EPA*, 790 F.3d 138, 160 (D.C. Cir. 2015).

This attachment is intended to provide guidance regarding available data that states and tribes may wish to assess when evaluating these five factors. This guidance also provides insight into the EPA's subsequent review and evaluation of the state and tribal nonattainment area boundary recommendations. The guidance offers suggestions about techniques and approaches; it does not contain requirements to be strictly followed and should not be read as prescriptive with respect to the specific techniques recommended.

The EPA recognizes that some of the recommended assessments can be resource intensive. To help mitigate this potential concern, the EPA intends to provide an Ozone Designations Mapping Tool to assist air agencies in developing their area designation and nonattainment boundary recommendations and to provide the relevant data to facilitate the analyses. The EPA will make the Ozone Designations Mapping Tool available on the ozone designations website.² The table below outlines the datasets that the EPA expects to make available to the public on the ozone designations website and the expected date of availability. Design values for the 3-year period 2012 – 2014 are currently available³ and will also be posted on the ozone designations website. The EPA will update this website during the initial area designations process as other relevant datasets are identified.

Datasets the EPA will Provide via the EPA Ozone Designations Website

Dataset	Expected Availability Date
2013 – 2015 Ozone Design Values	Summer 2016
2014 – 2016 Ozone Design Values	Summer 2017
Nitrogen Oxide (NO _x)/VOC Point sources and county level emissions and Vehicle Miles Traveled (VMT) from 2011 National Emissions Inventory (NEI) ⁴ version 2	March 2016
County and Census Tract Population	March 2016
HYSPLIT Trajectory data *	March 2016
Geography/Topography *	March 2016
Jurisdictional Boundaries *	March 2016

* Separate datasets will not be provided. The information will be part of the web-based Ozone Designations Mapping Tool.

This guidance also offers recommendations concerning how states and tribes may wish to describe the basis for their initial designations recommendations. The EPA recommends that states and tribes articulate those recommendations in a narrative format. Thus, this guidance provides some direction regarding the content and structure of a narrative that describes the problem in a potential nonattainment area with monitors violating the NAAQS. A comprehensive narrative would articulate a conceptual model of the area that explains the nature and causes of the ozone air quality problem in the specific area, identifies the scope and scale of the air quality problem in that area, and describes all nearby emission sources that contribute to the problem.⁵ For multistate or multi-jurisdictional areas, the EPA

² <http://www.epa.gov/ozone-designations/>

³ <http://www3.epa.gov/airtrends/values.html>

⁴ The 2014 NEI may not be available for initial designation recommendations. If it becomes available, then it will be considered in lieu of the 2011 NEI.

⁵ Chapter 2.1 of the EPA's Draft Modeling Guidance for Demonstrating Attainment of Air Quality Goals for Ozone, PM_{2.5}, and Regional Haze has a detailed description of how to develop a sound conceptual description of an air quality problem. The document is located at: http://www3.epa.gov/ttn/scram/guidance/guide/Draft_O3-PM-RH_Modeling_Guidance-2014.pdf.

encourages states and tribes to work collaboratively to develop a single narrative. However, states or tribes with areas contributing to potential multistate or multi-jurisdictional nonattainment areas could also develop a conceptual model that describes only the contribution from the areas within their jurisdiction to the larger nonattainment area, rather than attempting to describe the scope and scale of the air quality problem throughout the entire area. Where a single area-wide narrative on the causes of the ozone air quality problem is not developed, the EPA will collectively use the information in all relevant submittals, along with other relevant data, to make its decision on the extent and designation of the multi-state area.

The underlying analytical framework of the recommended narrative can be summarized as follows:

- Determine violating monitors with design values greater than the NAAQS and gather data that enables an assessment of potential nearby contributing areas and the emissions sources (NO_x and VOC) in those areas.
- Assess and characterize the spatial and temporal differences in ozone concentrations within the area using data from Federal Reference Method (FRM)/Federal Equivalent Method (FEM) ozone monitors, as well as data from other FRM/FEM ozone monitors in nearby areas, if available.
- Areas may find it useful to assess and characterize the area-specific sensitivity of ozone formation to NO_x and VOC emissions. The amount of ozone formed in any given area depends on the amount of NO_x, VOC, and sunlight available to interact in a set of complex chemical reactions to form ozone. Depending on the local situation, peak ozone concentrations may be NO_x-sensitive, VOC-sensitive, or a mix of the two depending upon other conditions. Understanding the relative role of local NO_x and VOC emissions sources to ozone formation in the area violating the NAAQS helps identify which nearby emissions sources may be contributing to the monitored violations. Ambient data analyses and/or photochemical modeling simulations can be used to assess and characterize local ozone sensitivities.
- The information identified in the previous bullets can be evaluated in conjunction with emissions data and emissions-related data (e.g., vehicle miles traveled and population) to determine which source categories and source regions are contributing to the monitored violations.
- Once the emissions and air quality assessments have been evaluated, it is valuable to then assess the meteorology during the ozone season in the violating area. Weather patterns will have a large impact on the determination of contributing source regions. This analysis may further help to identify the relative magnitude of contributions from emission sources in nearby areas.
- Additionally, it may be useful to assess any geographic/topographic information, which could have consequences for transport, meteorology, and ozone formation in the area.
- Finally, all of the above assessments would be aggregated or synthesized into a consistent narrative that describes the relationship between sources in the analysis area and the measured exceedances. It will also be useful to assess jurisdictional considerations that could be relevant in identifying a nonattainment area boundary. This synthesis should represent a collective “weight-of-evidence” regarding the most appropriate boundaries for the nonattainment area.

While the general 5-factor framework is expected to be comprehensive and provide the foundation for each assessment of area boundaries, the extent of the analyses may vary on an area-by-area basis based on the nature, cause, and extent of the ozone air quality problem. This guidance suggests analyses of certain data sets that can be useful to assess which nearby areas contribute to nonattainment in a given area. In cases where more highly-resolved or newer data sets are available that are not explicitly mentioned in this guidance, states and tribes should consider their use. If these data are used, the EPA recommends that the states or tribes fully describe the data and their derivation in their supporting documentation for the designation recommendation.

The following sections provide more detail on the five factors and the weight-of-evidence approach that the EPA plans to consider when evaluating state and tribal recommendations and determining nonattainment area boundaries for the 2015 ozone NAAQS.

1. Air Quality Data

Ozone in the troposphere is a secondary pollutant formed by photochemical reactions of precursor gases and is not directly emitted from specific sources. Ozone is formed by atmospheric reactions involving two main classes of precursor pollutants: VOCs and NO_x. The formation of ozone is a complex, nonlinear function of many factors, including the intensity of sunlight, atmospheric mixing, the concentration of ozone precursors in the air, and the rates of chemical reactions of these precursors. Ozone is largely regional in nature with some higher values occurring in locations with ozone-conducive emissions, meteorological conditions, or transport patterns.

The first step in identifying an area to be designated nonattainment and to determine an appropriate nonattainment area boundary is to identify all monitored violations of the NAAQS using the most recently available design values. The EPA determines NAAQS compliance by considering the design value for each air quality monitoring site. The design value for the 2015 ozone NAAQS is the 3-year average of the annual 4th highest daily maximum 8-hour average concentrations.⁶ Only ozone measurement data collected in accordance with the quality assurance (QA) requirements⁷ using approved FRM/FEM monitors can be used for NAAQS compliance determinations. The EPA uses FRM/FEM measurement data residing in the EPA's Air Quality System (AQS) to calculate the ozone design values. Individual measurements that the EPA determines to be "exceptional" in accordance with the Exceptional Events Rule⁸ (such as days with poor air quality caused by wildland fire) are not included in these calculations. State and tribal monitoring agencies are required to annually certify data submitted to AQS by May 1st of the subsequent year.⁹ A tribal monitoring agency must certify its data if the tribe is monitoring for regulatory purposes. A tribe may also be specifically required to certify its data under terms of a grant from EPA. Tribes should consult with the appropriate Regional office on questions regarding regulatory monitoring and the certification process. The EPA typically extracts ambient data from AQS and calculates official design values for regulatory purposes shortly after the

⁶ The specific methodology for calculating the ozone design values, including computational formulas and data completeness requirements, is described in 40 CFR part 50, Appendix U.

⁷ The QA requirements for ozone monitoring data are specified in 40 CFR part 58, Appendix A.

⁸ Final Rule on the Treatment of Data Influenced by Exceptional Events (72 FR 13560, March 22, 2007). Note, on November 10, 2015, the EPA proposed revisions to the 2007 Exceptional Events Rule and issued a draft guidance document for wildfire ozone events. The EPA intends to finalize the rule revisions and guidance before the October 1, 2016, deadline for state and tribal designations recommendations.

⁹ Data certification requirements can be found in 40 CFR, part 58.15. The EPA has developed guidance related to the data certification process that can be found at: <http://www3.epa.gov/ttn/amt/c/qacert.html>.

certification due date. The design values calculated using this data undergo review by the EPA regional offices, and the final design values are then posted on a public website.¹⁰ Initial state and tribal designation recommendations due October 1, 2016, should focus on design values based on air quality data from 2013 to 2015; however, the EPA intends to make final designation decisions using design values based on the 2014 to 2016 certified air quality data.

In addition to identifying monitors where the most recent design values violate the NAAQS, examining historical ozone air quality measurement data (including previous design values) can improve our understanding of the nature of the ozone ambient air quality problem in an area and thereby, inform decisions regarding the nonattainment area boundary. Since ozone concentrations are substantially impacted by meteorological conditions, including local wind patterns and synoptic weather patterns, the frequency and spatial distribution of exceedances of the standards can vary from year-to-year. This can be revealed by examining how frequently exceedances of the standard have occurred at the monitor with the highest design value for the area and at other monitor locations in the area under consideration, and how the spatial pattern in ozone concentrations across the area varies over time. This information can help to identify spatial and temporal patterns in the air quality of a given area and, when combined with other information from the 5-factor review, can help identify nearby areas with emissions sources contributing to an area with a monitored violation.

2. Emissions and Emissions-Related Data

The sources and levels of emissions of ozone-precursor pollutants are important factors in the initial area designations process. As noted above, ambient ozone is formed through complex atmospheric processes. Air quality in a nonattainment area is also typically the result of a combination of regional and local emissions. In the designations process, for each area with a violating monitor, the EPA evaluates the current emissions data from nearby counties to assess each county's potential contribution to ozone concentrations at the violating monitor(s) in the area under evaluation. It should be noted that while ozone can be transported many hundreds of miles and sources of emissions that are very distant from the potential nonattainment area may also contribute to monitored ozone levels, these far upwind emissions are not considered in the designation determination to be "nearby" sources.¹¹ Therefore, the evaluation of the area is also a means to differentiate between the impact of emissions from more distant sources and from sources in nearby areas that should be included as part of the designated nonattainment area. For initial area designations, we intend to examine current emissions of identified sources of NO_x and VOC, as guided by the local conceptual description of NO_x- and VOC-limited areas. The EPA expects that some local NO_x and VOC emissions contributions from mobile and stationary sources and transport from nearby areas can contribute to higher ozone levels at the violating monitors. Analyses should include reviewing data from the latest NEI and other relevant sources, as available. The analysis should also include examining the magnitude of county-level emissions and the geographic locations of NO_x and VOC sources.

Analyzing the magnitude and spatial extent of emissions provides information about potential spatial gradients in ozone precursor emissions. Combining these analyses (e.g., magnitude of emissions and point of release) with meteorological information can inform the evaluation of the degree of contribution from nearby areas. In addition, if the most recent emission inventories do not reflect conditions for the

¹⁰ Design values for ozone can be found at: <http://www3.epa.gov/airtrends/values.html>.

¹¹ The Court of Appeals for the D.C. Circuit upheld the EPA's interpretation of the term "nearby" as being reasonable and consistent with the statute. *Miss. Comm'n on Envtl. Quality v. EPA*, 790 F.3d 138, 160 (D.C. Cir. 2015).

same time period as the air quality data being used to determine the nonattainment designation, then information provided on changes in emissions will be considered. These changes may include emissions reductions due to permanent and enforceable emissions controls and may include emissions increases from new sources or at existing sources.

The EPA believes that it will be appropriate to use 2011 NEI version 2 data because that will be the most recent national emissions inventory information available at the beginning of the designations process.¹² The NEI includes data, generally on an annual basis at the county level. Emissions from large stationary sources at a specific location are also available. More detailed inventories (higher resolution than county estimates) may also be available for some areas, although not in the NEI.¹³ To supplement the NEI county-level data, the EPA will provide information that could be used to understand spatial allocation within a county including the location and magnitude of large point sources. Additionally, states and tribes may wish to review gridded emissions data, which are generally available at 12 km grid resolution. These data, which can be provided by the EPA, have been created to cover emissions levels in the contiguous 48 states for 2011. These gridded emissions data can be provided by the EPA on an annual basis or for shorter time periods such as the ozone season.

Additionally, states or their regional organizations may submit their own emissions information or versions of gridded emissions for more recent years.

Population and degree of urbanization

The EPA has consolidated population and degree of urbanization within the emissions and emissions-related data factor as these elements supplement and help to inform the analysis of emissions data. The EPA intends to provide data such as population by county and census tract. An analysis of population and degree of urbanization may provide indicators of the location of emissions-related activities within the county.

The EPA expects that states and tribes may have independently developed datasets to better inform these elements. The EPA believes that population information such as the location and recent trends in population growth and the patterns of residential and commercial development can serve as potential indicators of the probable location and magnitude of emissions sources that may contribute to ozone concentrations in a given nonattainment area.

Traffic and commuting patterns

The EPA recommends examining the location of major transportation arteries and information on traffic volume and commuting patterns in and around the area containing a violating monitor. This may include examining the number of commuters in each nearby county who drive to a county within the area that has a violating monitor, the percent of total commuters in each county who commute to other counties

¹² The 2014 NEI may not be available for initial designation recommendations. If it becomes available, then it will be considered in lieu of the 2011 NEI.

¹³ The EPA develops gridded emissions by applying temporal (e.g., seasonal variations in emissions as reported to the NEI) and spatial (e.g., incorporates latitude and longitude location information as reported to the NEI) adjustments to the county-based NEI estimates to produce the more finely resolved gridded emissions. These emissions are generally available at a 12 km resolution, but may be available at finer resolutions for certain localities that have been the focus of special modeling studies.

with violating monitors within the metropolitan area, and the total VMT for each county. Areas with higher VMT and commuting activity can be an indicator of the location of mobile source emissions that may contribute to ozone concentrations at the violating monitor.

The NEI is one source of the county-wide VMT data and facilitates relative comparisons of traffic and commuting patterns between counties in a larger area.¹⁴ However, more detailed assessments provided by states or tribes could help to highlight the magnitude and location of emissions activity. The EPA will provide gridded VMT data; however, these estimates may not correspond directly with VMT data developed by state or local agencies.

3. Meteorology

Evaluation of meteorological data helps to assess the fate and transport of emissions contributing to ozone concentrations and to identify areas potentially contributing to the monitored violations. Results of meteorological data analysis may support determination of nonattainment area boundaries.

One basic type of meteorological analysis involves assessing potential source-receptor relationships in the area on days with high ozone concentrations using wind speed and wind direction data. A more sophisticated and accurate assessment involves modeling air parcel trajectories to help understand complex transport situations. The HYSPLIT (HYbrid Single-Particle Lagrangian Integrated Trajectory) modeling system may be useful for some areas to produce trajectories that illustrate the 3-dimensional paths traveled by air parcels to a violating monitor. The EPA will provide back trajectories for violating monitors, for each day of high ozone concentration (i.e., daily maximum 8 hour values that exceed the NAAQS) at those monitors. States or tribes can choose to do additional HYSPLIT modeling and guidance is provided below. If a trajectory model other than HYSPLIT is used, states or tribes should provide detailed information about the technique, how it is used, and why it is preferred over HYSPLIT. *Preparing and running a HYSPLIT modeling analysis*

Atmospheric trajectory models use meteorological data and mathematical equations to simulate 3-dimensional transport in the atmosphere. Generally, the position of particles or parcels of air with time are calculated based on meteorological data such as wind speed and direction, temperature, humidity, and pressure. Model results depend on the spatial and temporal resolution of the atmospheric data used, and also on the complexity of the model itself. The HYSPLIT model¹⁵ is frequently used to produce trajectories for assessments associated with determining nonattainment area boundaries. HYSPLIT contains models for trajectory, dispersion, and deposition; however, analyses recommended here only use the trajectory component. The trajectory model, which uses existing meteorological forecast fields from regional or global models to compute advection (i.e., the rate of change of an atmospheric property caused by the horizontal movement of air) and stability, is designed to support a wide range of simulations related to the atmospheric transport of pollutants.

¹⁴ NEI county-level VMT estimates are developed in a top-down approach from Federal Highway Administration estimates of statewide VMT by road class that are allocated to counties based on surrogates. Accordingly, the NEI estimates do not always compare well to detailed area-specific studies that are developed in a more robust way (e.g., travel demand model data).

¹⁵ <http://ready.arl.noaa.gov/HYSPLIT.php>

HYSPLIT trajectories may be produced for various combinations of time and locations. When HYSPLIT trajectories are produced for specific monitor locations for days of high ozone concentrations (e.g., daily maximum 8-hour values that exceed the NAAQS), the results illustrate the potential source region for the air parcel that affected the monitor on the day of the high concentration.

While HYSPLIT is a useful tool for identifying meteorological patterns associated with exceedance events, HYSPLIT trajectories alone do not conclusively indicate contribution to measured high concentrations of ozone. Therefore, they cannot be used in isolation to determine inclusion or exclusion of an area within a nonattainment boundary. While a HYSPLIT trajectory analysis alone cannot yield a conclusion that a particular region contributes to ozone concentrations, a set of HYSPLIT trajectories that show no wind flow from a particular region on any day with high ozone concentration measurements might provide support for discounting that region as contributing to ozone concentrations. HYSPLIT trajectories are very useful in combination with information on the location and magnitude of ozone precursor emissions sources.

A HYSPLIT backward trajectory, the most common trajectory used in assessments associated with determining nonattainment area boundaries, is usually depicted on a standard map as a single line extending in two dimensional (x,y) space from a starting point, regressing backward in time as the line extends from the starting point. An individual trajectory can have only one starting height; HYSPLIT can plot trajectories of different starting heights at the same latitude/longitude starting point on the same map, automatically using different colors for the different starting heights. HYSPLIT will also include a vertical plot of the trajectories in time, with colors corresponding to the same trajectory in the (x,y) plot. This display can be easily misinterpreted as having finer accuracy than the underlying model and data.

It is important to observe the overall size of the plot, its width and length in kilometers, and consider the size of an individual grid cell in the input meteorological data set. These input grid cells are usually 40 km in width and length, so the total area of a trajectory plot may be limited. It is also important to understand the trajectory line itself. The line thickness is predetermined as a user option, so its thickness does not imply coverage other than to represent the centerline of an air parcel's motion calculated to arrive at the starting location at the starting time. Uncertainties are clearly present in these results, and these uncertainties change with trajectory time and distance traveled. One should avoid concluding a region is not along a trajectory's path if the center line of that trajectory missed the region by a relatively small distance.

Detailed information for downloading, installing, and operating HYSPLIT can be found at these websites:

<http://ready.arl.noaa.gov/HYSPLIT.php>

http://www.arl.noaa.gov/documents/reports/hysplit_user_guide.pdf

<http://www.arl.noaa.gov/documents/reports/arl-224.pdf>

HYSPLIT's many setup options allow great flexibility and versatility. However, careful selection and recording of these options is necessary to provide reviewers the ability to reproduce the model results. The following paragraphs describe the options that should be recorded, at a minimum, to enable another party to reproduce a HYSPLIT model run.

Model Version. If the HYSPLIT trajectory is produced via the NOAA Air Resources Laboratory (ARL) website (http://ready.arl.noaa.gov/HYSPLIT_traj.php), note the "Modified:" date in the lower-left corner of the webpage, as well as the date the trajectory was produced. If the trajectory is produced using a stand-alone version of HYSPLIT, note *the release date*, which will be displayed after exiting the main graphical user interface (GUI) screen.

Basic Trajectory Information. Note the *starting time* (YY MM DD HR), the *duration of the trajectory* in hours, and whether the trajectory is *backward or forward*. Note the *latitude and longitude*, as well as the *starting height*, for each *starting location*. Starting height is given by default in meters above ground level (AGL) unless another option is selected. Starting heights are typically no less than 100 meters AGL to avoid direct interference of terrain, and are typically no greater than 1500 meters AGL to confine the air parcel within the mixed layer. Some trajectories can escape the mixed layer, and this result would be considered in the interpretation.

Starting height and starting location will identify the 3-dimensional location of the trajectory's latest endpoint in time if a backward trajectory is selected (i.e., the start of a trajectory going backward in time). Backward trajectories used in analyses associated with designations typically have a trajectory duration of 24 hours. Considering the geographic proximity of areas under consideration in ozone designations, air parcel locations within this proximity are almost always within the last 24 hours of travel to the trajectory endpoint. Air parcel locations more than 24 hours prior to trajectory end time are rarely found within this proximity.

Input Meteorological Data Set. Note the *input meteorological data set* used in the HYSPLIT model run. The *original file name* provides sufficient information to identify the data set.

Meteorological data fields to run the model are already available for access through the HYSPLIT menu system, or by direct FTP from ARL. The ARL web server contains several meteorological model data sets already converted into a HYSPLIT compatible format in the public directories. Direct access via FTP to these data files is built into HYSPLIT's graphical user interface. The data files are automatically updated on the server with each new forecast cycle. Only an email address is required for the password to access the server. The ARL analysis data archive consists of output from the Global Data Analysis System (GDAS) and the NAM Data Analysis System (NDAS - previously called EDAS) covering much of North America. Both data archives are available from 1997 in semi-monthly files (SM). The EDAS was saved at 80 km resolution every 3-hours through 2003, and then at 40 km resolution starting in 2004. Detailed information on all meteorological data available for use in HYSPLIT can be found in the HYSPLIT4 Users Guide.¹⁶

It is possible to run the stand-alone HYSPLIT program on user-supplied meteorological data. This could be advantageous when the horizontal resolution or model physics used by ARL is inferior to other existing datasets. If a state or tribe chooses to use meteorological data not already on the ARL web server, the state or tribe should document the reason for this choice and should provide detailed information about the substituted meteorological dataset.

¹⁶ http://www.arl.noaa.gov/documents/reports/hysplit_user_guide.pdf

Vertical Motion Options. HYSPLIT can employ one of five different *methods for computing vertical motion*. A sixth method is to accept the vertical motion values contained within the input meteorological data set, effectively using the vertical motion method used by the meteorological model that created the data set. In a typical HYSPLIT application, EPA selects the option to accept the vertical motion values contained within the input meteorological data set. The user should note which method was selected as well as the value chosen for *the top of the model*, in meters AGL.

Trajectory Display Options. The HYSPLIT trajectory model generates a text output file of end-point positions. The end-point position file is processed by another HYSPLIT module to produce a Postscript display file or output files in other display formats. Some parameters, such as map projection and size, can be automatically computed based on the location and length of the trajectory, or they can be manually set by the user. While these display options do not directly affect the trajectory information itself, noting these options will eliminate possible misinterpretation of identical trajectories because of differing display options. An important display option is the choice of *vertical coordinate*, usually set to meters AGL for these assessments.

4. Geography/topography

Consideration of geography or topography can provide additional information relevant to defining nonattainment area boundaries. Analyses should examine the physical features of the land that might define the airshed. Mountains or other physical features may influence the fate and transport of emissions as well as the formation and distribution of ozone concentrations. For example, valley-type topographical features can cause local stagnation episodes where vertical temperature inversions effectively “trap” air pollution. Under these conditions, emissions can accumulate leading to periods of elevated ozone concentrations. These inversions may be limited in extent and, therefore, the areas with inversions may need to be separated from areas at altitudes above the top of the inversion layer in locations where exceedances are associated with this type of event. Conversely, higher altitude mountaintop sites might experience a greater influence from long range transport and associated transport episodes in comparison to nearby areas at a lower altitude. Similarly, the absence of any such geographic or topographic features may also be a relevant consideration in selecting boundaries for a given violating area.

5. Jurisdictional boundaries

Once the geographic extent of the violating area and the nearby area contributing to violations is determined, existing jurisdictional boundaries may be considered for the purposes of providing a clearly defined legal boundary and carrying out the air quality planning and enforcement functions for nonattainment areas. Examples of jurisdictional boundaries include, but are not limited to: counties, air districts, areas of Indian country, metropolitan planning organizations, and existing nonattainment areas. If an existing jurisdictional boundary is used to help define the nonattainment area, it must encompass all of the area that has been identified as meeting the nonattainment definition. Where existing jurisdictional boundaries are not adequate to describe the nonattainment area, other clearly defined and permanent landmarks or geographic coordinates should be used.

Weight-of-Evidence Analysis Based on the Five Factors

In making designations recommendations for violating areas or contributing areas, and the nonattainment area boundaries for such areas, the EPA recommends that states and tribes consider the five recommended factors together and use a weight-of-evidence approach for this analysis. As explained above, the starting point for evaluating the factors is the air quality analysis. Of particular importance are the location(s) of the violating monitor(s) based on 2013-2015 data¹⁷ and the characteristics of those violations. Once the characteristics of the violations are established, one can begin to assess which nearby emissions sources and source regions may have contributed to those violations. This contribution evaluation should generally consider the location and magnitude of emissions, and the potential for these emissions to contribute to the ambient conditions at the violating monitors as informed by the meteorological and geographical/topographical analysis factors. The guiding principle for this evaluation should be to include, within the boundaries of the nonattainment area, nearby areas with emissions of ozone precursors (NO_x and VOC) that contribute to the violating monitor on days that exceed the NAAQS. The final factor, jurisdictional boundaries, should be considered to refine the nonattainment area boundary to ensure meaningful air quality planning and regulation during the NAAQS implementation phase. As in prior designations for ozone NAAQS, the EPA believes that it is appropriate to use already-established air planning boundaries where possible, to assure continued effective planning and implementation.

The EPA believes that the 5-factor analysis described here is generally comprehensive and intends to use the weight-of-evidence approach based on these five factors in establishing the nonattainment boundaries for the 2015 ozone NAAQS. As noted earlier, the EPA intends to provide an Ozone Designations Mapping Tool to assist air agencies in developing their area designation and nonattainment boundary recommendations and to provide the relevant data to facilitate the analyses. The EPA will make the Ozone Designations Mapping Tool available on the ozone designations website.

The EPA also recognizes the potential value of additional data or methodologies not already specified in this guidance that states or tribes may elect to submit to qualitatively describe or quantify the relative contributions from contributing areas to violating monitors. In some cases, these supplemental methodologies (e.g., source apportionment modeling) may be used to synthesize the various factors, such as air quality, emissions, and meteorological data into quantitative estimates of the contributions from specific areas.

Source Apportionment Modeling

Source apportionment modeling refers to an augmented instrumentation of traditional regional photochemical Eulerian models which allows the model to track the impacts of NO_x and VOC emissions from user-defined source regions on predicted ozone concentrations in a particular grid cell. Emissions are tracked with source apportionment through ozone formation, transport, and deposition processes in the host photochemical model.^{18,19} Source apportionment modeling combines into a single analysis

¹⁷ The EPA intends to consider 2014-2016 data as soon as these data are available.

¹⁸ Dunker, A. M., Yarwood, G., Ortman, J. P., and Wilson, G. M. Comparison of source apportionment and source sensitivity of ozone in a three-dimensional air quality model, *Environ. Sci. Technol.*, 36, 2953–2964, 2002.

¹⁹ Kwok, R.H.F., Baker, K.R., Napelenok S.L., Tonnesen, G.S. Photochemical grid model implementation and application of VOC, NO_x, and O₃ source apportionment, *Geoscientific Model Development*, 8(1), 99-114, 2015.

several of the factors that the EPA believes are important for determining nonattainment area boundaries: air quality data, emissions, meteorology, and geography/topography. Consequently, this modeling may help identify possible areas for inclusion in the nonattainment area because of their contribution to violations in nearby areas with violating monitors.

The EPA does not require states or tribes to conduct source apportionment modeling as part of the initial area designations process for the 2015 ozone NAAQS. However, some states used source apportionment modeling in their boundary determinations for the 2008 ozone NAAQS. The EPA is not producing source apportionment modeling assessments for any areas as part of the initial area designations process for the 2015 NAAQS. Like other aspects of the factor analyses, source apportionment modeling produces information that can help to determine potential boundaries for the area that should be designated nonattainment. Where provided by states or tribes, source apportionment results will be considered as just one part of an overall assessment of the potential nonattainment area boundaries. The EPA recognizes that while there are uncertainties associated with interpreting source apportionment outputs, it can be a useful technique for comparing the relative contribution of individual county emissions of ozone precursor emissions in a more sophisticated manner.

If a state chooses to conduct source apportionment modeling, the EPA recommends that model episodes are of sufficient duration to capture the entire range of meteorological and emissions conditions that can lead to ozone violations in a particular area. Further, we recommend that states and tribes follow the relevant EPA guidance for photochemical modeling attainment demonstrations²⁰ when establishing their source apportionment modeling platform. In establishing the parameters of a source apportionment modeling exercise, the violating monitor(s) would typically comprise the receptor(s) in the analysis. When summarizing the outputs from the source apportionment modeling, it is suggested that the relative contributions from nearby source regions be compared against one another. It is expected that the focus of the source apportionment modeling would be identifying each source region's contribution to ozone levels near or exceeding the level of the ozone NAAQS. While the EPA does not believe it is appropriate to establish an *a priori* threshold contribution level, a relative comparison of the modeled contribution of each source region should reveal where there are potential contributing sources that should be included within the nonattainment area.

Rural Transport Areas

Section 182(h) of the CAA identifies a category of ozone nonattainment areas referred to as rural transport areas (RTAs). An RTA is treated as a Marginal area for purposes of ozone-related planning and control requirements, regardless of the area's classification. In order for an area to qualify as an RTA, the nonattainment area must meet two criteria. First, the nonattainment area cannot be adjacent to, or include any part of a metropolitan statistical area, as defined by the Office of Management and Budget. Second, the NO_x and VOC emissions from sources within the area cannot make a significant contribution to ozone concentrations in the area itself, or in other areas. The first criterion was discussed earlier in this guidance memo. This portion of the document provides guidance to states and tribes regarding the information that should be submitted to the EPA as part of a demonstration for the second criterion. The EPA believes that a multi-factor, weight-of-evidence approach is needed to demonstrate

²⁰ Draft Modeling Guidance for Demonstrating Attainment of Air Quality Goals for Ozone, PM_{2.5}, and Regional Haze. December 2014. Located at: http://www3.epa.gov/ttn/scram/guidance/guide/Draft_O3-PM-RH_Modeling_Guidance-2014.pdf.

that emissions within a potential RTA do not contribute significantly to the local ozone nonattainment problem or to ozone nonattainment downwind. The factors are similar in nature to the ones described above to guide development of nonattainment designation boundaries: air quality data, emissions estimates, meteorological transport patterns, and geography/topography.

In most instances, the first step in demonstrating that the NO_x and VOC emissions in a potential RTA do not significantly contribute to ozone in the area itself is the development of a conceptual description of the nature of ozone exceedances in the area.²¹ This conceptual description should summarize the spatial and temporal patterns of ozone exceedances in the area and begin to identify hypotheses as to which processes and sources are likely most responsible for those high ozone values. To the extent that the conceptual description suggests that transport from upwind areas is largely responsible for the local ozone problem, the RTA demonstration should then further analyze existing ambient monitoring data, meteorological transport patterns, and local and regional emissions estimates to construct a weight-of-evidence argument that concludes the upwind contributions dominate any local contributions.

When compiling a weight-of-evidence based RTA demonstration, it may be valuable to consider an analysis of regional surface ozone monitoring data to see if there is a clear signal of an ozone plume being generated over an upwind area and being transported downwind as the day proceeds, reaching the potential RTA area after the time in which local photochemical production of ozone would have ceased. It also may be useful to look at any available ozone precursor data in or near the local area as a way to assess the chemical nature of a particular air mass. One indication of a photochemically-aged ozone plume that was likely formed from upwind emissions and transported away from its source origin, would be situations in which high ambient ozone and total reactive nitrogen (NO_y) values were observed in locations with relatively low ambient concentrations of NO_x. In other cases, there may be data available about the 3-dimensional chemical state of the atmosphere (e.g., from aircraft, satellites, or other relevant instrumentation) that can help characterize the role of transported ozone from upwind areas.

In terms of the meteorological factor, using HYSPLIT to estimate the back trajectories of air parcels on high ozone days can provide valuable information about the transport path and potential origin of the ozone pollution. We expect that for most areas that would qualify for treatment as an RTA, most, if not all, back trajectories on high ozone days would suggest long-path trajectories with source origins well away from the local area and with little potential for recirculation of the local emissions.

Finally, for the emissions factor, the relative magnitude of local emissions in any potential RTA is also a key consideration in determining if local sources contribute significantly to the ozone problem in the area. If the NO_x and VOC inventories for a particular area are appreciably less than those for other areas for which there is evidence demonstrating contribution to the ozone nonattainment problem (i.e., from the ambient and meteorological analyses), this provides support for concluding that the transport component is overwhelming any local ozone production. A simple approach to assessing the potential importance of local emissions is to compile county-level emissions inventory estimates for each county potentially along the trajectories that are expected to contribute to ozone in the potential RTA. If the emissions from upwind contributing counties are substantially larger than what is being emitted locally, then this suggests that the impact of the local emissions may not be significant. The EPA recommends that any comparative assessments of emissions be based on the most current available inventories.

²¹ Chapter 2.1 of EPA's Modeling Guidance for Demonstrating Attainment of Air Quality Goals for Ozone, PM_{2.5}, and Regional Haze has a detailed description of how to develop a sound conceptual description of an air quality problem. http://www3.epa.gov/ttn/scram/guidance/guide/Draft_O3-PM-RH_Modeling_Guidance-2014.pdf.

It is also possible to assess the contribution of local NO_x and VOC emissions to the ozone in the area using photochemical air quality modeling. “Zero-out” modeling can provide an estimate of the total local impact by calculating the difference between the model estimates from a base case run and the estimates from a simulation in which the man-made emissions of NO_x and VOC are removed from the potential RTA. If the response of the model is small (i.e., even with zero local emissions, there is still a local ozone problem due to transport), it would support a determination that local emissions sources make a small contribution to ozone concentrations in the area. Additionally, source apportionment modeling can be used to estimate the contributions of user-defined source regions (or source categories) to total modeled ozone in an area. These types of modeling analyses can be resource-intensive and the EPA does not expect areas to rely on these models unless they have already been completed for other purposes. In some cases, there may be existing regional or national modeling simulations that can be leveraged to support an RTA demonstration. States and tribes are encouraged to consult with their EPA regional office on potentially available information.

The analyses described above focus on showing that local emissions do not significantly impact high ozone in the local area. Similar analyses would be appropriate to demonstrate that local emissions do not significantly impact ozone concentrations in other areas. It is unrealistic to expect that a state or tribe could analyze impacts on every possible downwind area. Instead, we recommend that the state or tribe consider the effects of local emissions on the nearest potential nonattainment areas, in a qualitative sense using some of the data analyses described above.

In general, the EPA believes the geographical restrictions of section 182(h)(1) will limit the number of areas eligible for treatment as an RTA. States or tribes requesting that the EPA treat an ozone nonattainment area as an RTA are encouraged to conduct the technical analyses discussed above as part of a multi-factor, weight-of-evidence demonstration. Documentation that describes each analysis performed and the aggregate determination that emissions in the candidate area do not make a significant contribution to ozone concentrations in that area or in other downwind (current or potential) nonattainment areas should be submitted to the appropriate EPA regional office. Any state or tribe seeking an RTA determination for an area is encouraged to work closely with the appropriate EPA regional office to coordinate the analytical plan for such a demonstration.

Exhibit 11



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Part II

Environmental Protection Agency

40 CFR Part 50, 51, 52, et al.

National Ambient Air Quality Standards for Ozone; Final Rule

ENVIRONMENTAL PROTECTION AGENCY

40 CFR Parts 50, 51, 52, 53, and 58

[EPA-HQ-OAR-2008-0699; FRL-9933-18-OAR]

RIN 2060-AP38

National Ambient Air Quality Standards for Ozone

AGENCY: Environmental Protection Agency (EPA).

ACTION: Final rule.

SUMMARY: Based on its review of the air quality criteria for ozone (O₃) and related photochemical oxidants and national ambient air quality standards (NAAQS) for O₃, the Environmental Protection Agency (EPA) is revising the primary and secondary NAAQS for O₃ to provide requisite protection of public health and welfare, respectively. The EPA is revising the levels of both standards to 0.070 parts per million (ppm), and retaining their indicators (O₃), forms (fourth-highest daily maximum, averaged across three consecutive years) and averaging times (eight hours). The EPA is making corresponding revisions in data handling conventions for O₃ and changes to the Air Quality Index (AQI); revising regulations for the prevention of significant deterioration (PSD) program to add a transition provision for certain applications; and establishing exceptional events schedules and providing information related to implementing the revised standards. The EPA is also revising the O₃ monitoring seasons, the Federal Reference Method (FRM) for monitoring O₃ in the ambient air, Federal Equivalent Method (FEM) analyzer performance requirements, and the Photochemical Assessment Monitoring Stations (PAMS) network. Along with exceptional events schedules related to implementing the revised O₃ standards, the EPA is applying this same schedule approach to other future new or revised NAAQS and removing obsolete regulatory language for expired exceptional events deadlines. The EPA is making minor changes to the procedures and time periods for evaluating potential FRMs and equivalent methods, including making the requirements for nitrogen dioxide (NO₂) consistent with the requirements for O₃, and removing an obsolete requirement for the annual submission of Product Manufacturing Checklists by manufacturers of FRMs and FEMs for monitors of fine and coarse particulate matter. For a more detailed summary, see the Executive Summary below.

DATES: The final rule is effective on December 28, 2015.

ADDRESSES: EPA has established a docket for this action (Docket ID No. EPA-HQ-OAR-2008-0699) and a separate docket, established for the Integrated Science Assessment (ISA) (Docket No. EPA-HQ-ORD-2011-0050), which has been incorporated by reference into the rulemaking docket. All documents in the docket are listed on the www.regulations.gov Web site. Although listed in the docket index, some information is not publicly available, e.g., confidential business information or other information whose disclosure is restricted by statute. Certain other material, such as copyrighted material, is not placed on the Internet and may be viewed, with prior arrangement, at the EPA Docket Center. Publicly available docket materials are available either electronically in www.regulations.gov or in hard copy at the Air and Radiation Docket and Information Center, EPA/DC, WJC West Building, Room 3334, 1301 Constitution Ave., NW., Washington, DC. The Public Reading Room is open from 8:30 a.m. to 4:30 p.m., Monday through Friday, excluding legal holidays. The telephone number for the Public Reading Room is (202) 566-1744 and the telephone number for the Air and Radiation Docket and Information Center is (202) 566-1742. For additional information about EPA's public docket, visit the EPA Docket Center homepage at: <http://www.epa.gov/epahome/dockets.htm>.

FOR FURTHER INFORMATION CONTACT: Ms. Susan Lyon Stone, Health and Environmental Impacts Division, Office of Air Quality Planning and Standards, U.S. Environmental Protection Agency, Mail code C504-06, Research Triangle Park, NC 27711; telephone: (919) 541-1146; fax: (919) 541-0237; email: stone.susan@epa.gov.

SUPPLEMENTARY INFORMATION:

General Information

Availability of Related Information

A number of the documents that are relevant to this action are available through the EPA's Office of Air Quality Planning and Standards (OAQPS) Technology Transfer Network (TTN) Web site (http://www.epa.gov/ttn/naaqs/standards/ozone/s_o3_index.html). These documents include the *Integrated Science Assessment for Ozone* (U.S. EPA, 2013), available at http://www.epa.gov/ttn/naaqs/standards/ozone/s_o3_2008_isa.html; the *Health Risk and Exposure Assessment* and the *Welfare Risk and Exposure Assessment for Ozone*, Final

Reports (HREA and WREA, respectively; U.S. EPA, 2014a, 2014b), available at http://www.epa.gov/ttn/naaqs/standards/ozone/s_o3_2008_rea.html; and the *Policy Assessment for the Review of the Ozone National Ambient Air Quality Standards* (PA; U.S. EPA, 2014c), available at http://www.epa.gov/ttn/naaqs/standards/ozone/s_o3_2008_pa.html. These and other related documents are also available for inspection and copying in the EPA docket identified above.

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References

Executive Summary

This section summarizes information about the purpose of this regulatory action, the major provisions of this action, and provisions related to implementation.

Purpose of This Regulatory Action

Sections 108 and 109 of the Clean Air Act (CAA) govern the establishment, review, and revision, as appropriate, of the NAAQS to protect public health and welfare. The CAA requires the EPA to periodically review the air quality criteria—the science upon which the standards are based—and the standards themselves. This rulemaking is being conducted pursuant to these statutory requirements. The schedule for completing this review is established by a federal court order, which requires that the EPA make a final determination by October 1, 2015.

The EPA completed its most recent review of the NAAQS for O₃ in 2008. As a result of that review, EPA took four principal actions: (1) Revised the level of the 8-hour primary standard to 0.075 ppm; (2) expressed the standard to three decimal places; (3) revised the 8-hour secondary standard by making it identical to the revised primary standard; and (4) made conforming changes to the AQI.

In subsequent litigation, the U.S. Court of Appeals for the District of Columbia Circuit (DC Circuit) upheld the EPA's 2008 primary standard but remanded the 2008 secondary standard (*Mississippi v. EPA*, 744 F. 3d 1334 [D.C. Cir. 2013]). With respect to the primary standard, the court held that the EPA reasonably determined that the existing primary standard, set in 1997, did not protect public health with an adequate margin of safety and required

revision. In upholding the EPA's revised primary standard, the court dismissed arguments that the EPA should have adopted a more stringent standard. The court remanded the secondary standard to the EPA after finding that the EPA's justification for setting the secondary standard identical to the revised 8-hour primary standard violated the CAA because the EPA had not adequately explained how that standard provided the required public welfare protection. In remanding the 2008 secondary standard, the court did not vacate it. The EPA has addressed the court's remand with this final action.

This final action reflects the Administrator's conclusions based on a review of the O₃ NAAQS that began in September 2008, and also concludes the EPA's reconsideration of the 2008 decision that it initiated in 2009 and subsequently consolidated with the current review. In conducting this review, the EPA has carefully evaluated the currently available scientific literature on the health and welfare effects of O₃, focusing particularly on the new literature available since the conclusion of the previous review in 2008. Between 2008 and 2014, the EPA prepared draft and final versions of the Integrated Science Assessment, the Health and Welfare Risk and Exposure Assessments, and the Policy Assessment. Multiple drafts of these documents were subject to public review and comment, and, as required by the CAA, were peer-reviewed by the Clean Air Scientific Advisory Committee (CASAC), an independent scientific advisory committee established pursuant to the CAA and charged with providing advice to the Administrator.

The EPA proposed revisions to the primary and secondary O₃ NAAQS on December 17, 2014 (79 FR 75234), and provided a 3-month period for submission of comments from the public. In addition to written comments submitted to EPA, comments were also provided at public hearings held in Washington, DC, and Arlington, Texas, on January 29, 2015, and in Sacramento, California, on February 2, 2015. After consideration of public comments and the advice from the CASAC, the EPA has developed this final rulemaking, which is the final step in the review process.

In this rulemaking, the EPA is revising the suite of standards for O₃ to provide requisite protection of public health and welfare. In addition, the EPA is updating the AQI, and making changes in the data handling conventions and ambient air monitoring, reporting, and network

design requirements to correspond with the changes to the O₃ NAAQS.

Summary of Major Provisions

With regard to the primary standard, the EPA is revising the level of the standard to 0.070 ppm to provide increased public health protection against health effects associated with long- and short-term exposures. The EPA is retaining the indicator (O₃), averaging time (8-hour) and form (annual fourth-highest daily maximum, averaged over 3 years) of the existing standard. This action provides increased protection for children, older adults, and people with asthma or other lung diseases, and other at-risk populations against an array of adverse health effects that include reduced lung function, increased respiratory symptoms and pulmonary inflammation; effects that contribute to emergency department visits or hospital admissions; and mortality.

The decisions on the adequacy of the current standard and the appropriate level for the revised standard are based on an integrative assessment of an extensive body of new scientific evidence, which substantially strengthens what was known about O₃-related health effects in the last review. The revised standard also reflects consideration of a quantitative risk assessment that estimates public health risks likely to remain upon just meeting the current and various alternative standards. Based on this information, the Administrator concludes that the current primary O₃ standard is not requisite to protect public health with an adequate margin of safety, as required by the CAA, and that revision of the level to 0.070 ppm is warranted to provide the appropriate degree of increased public health protection for at-risk populations against an array of adverse health effects. In concluding that a revised primary standard set at a level of 0.070 ppm is requisite to protect public health with an adequate margin of safety, the Administrator relies on several key pieces of information, including: (a) A level of 0.070 ppm is well below the O₃ exposure concentration shown to cause the widest range of respiratory effects (*i.e.*, 0.080 ppm) and is below the lowest O₃ exposure concentration shown to cause the adverse combination of decreased lung function and increased respiratory symptoms (*i.e.*, 0.072 ppm); (b) a level of 0.070 ppm will eliminate, or nearly eliminate, repeated occurrence of these O₃ exposure concentrations (this is important because the potential for adverse effects increases with frequency of occurrence); (c) a level of 0.070 ppm

will protect the large majority of the population, including children and people with asthma, from lower exposure concentrations, which can cause lung function decrements and airway inflammation in some people (*i.e.*, 0.060 ppm); and (d) a level of 0.070 ppm will result in important reductions in the risk of O₃-induced lung function decrements as well as the risk of O₃-associated hospital admissions, emergency department visits, and mortality. In addition, the revised level of the primary standard is within the range that CASAC advised the Agency to consider.

The EPA is also revising the level of the secondary standard to 0.070 ppm to provide increased protection against vegetation-related effects on public welfare. The EPA is retaining the indicator (O₃), averaging time (8-hour) and form (annual fourth-highest daily maximum, averaged over 3 years) of the existing secondary standard. This action, reducing the level of the standard, provides increased protection for natural forests in Class I and other similarly protected areas against an array of vegetation-related effects of O₃. The Administrator is making this decision based on judgments regarding the currently available welfare effects evidence, the appropriate degree of public welfare protection for the revised standard, and currently available air quality information on seasonal cumulative exposures that may be allowed by such a standard.

In making this decision on the secondary standard, the Administrator focuses on O₃ effects on tree seedling growth as a proxy for the full array of vegetation-related effects of O₃, ranging from effects on sensitive species to broader ecosystem-level effects. Using this proxy in judging effects to public welfare, the Administrator has concluded that the requisite protection will be provided by a standard that generally limits cumulative seasonal exposures to 17 ppm-hours (ppm-hrs) or lower, in terms of a 3-year W126 index. Based on air quality analyses which indicate such control of cumulative seasonal exposures will be achieved with a standard set at a level of 0.070 ppm (and the same indicator, averaging time, and form as the current standard), the Administrator concludes that a standard revised in this way will provide the requisite protection. In addition to providing protection of natural forests from growth-related effects, the revised standard is also expected to provide increased protection from other effects of potential public welfare significance, including crop yield loss and visible foliar injury.

Thus, based on all of the information available in this review, the Administrator concludes that the current secondary O₃ standard is not requisite to protect public welfare as required by the CAA, and that this revision will provide appropriate protection against known or anticipated adverse effects to the public welfare.

Provisions Related to Implementation

As directed by the CAA, reducing pollution to meet NAAQS always has been a shared task, one involving the federal government, states, tribes and local air agencies. This partnership has proved effective since the EPA first issued O₃ standards more than three decades ago, and is evidenced by significantly lower O₃ levels throughout the country. To provide a foundation that helps air agencies build successful strategies for attaining new O₃ standards, the EPA will continue to move forward with federal regulatory programs, such as the final Tier 3 motor vehicle emissions standards. To facilitate the development of CAA-compliant implementation plans and strategies to attain new standards, the EPA intends to issue timely and appropriate implementation guidance and, where appropriate and consistent with the law, new rulemakings to streamline regulatory burdens and provide flexibility in implementation. Given the regional nature of O₃ air pollution, the EPA will continue to work with states to address interstate transport of O₃ and O₃ precursors. The EPA also intends to work closely with states to identify locations affected by high background concentrations on high O₃ days due to stratospheric intrusions of O₃, wildfire O₃ plumes, or long-range transport of O₃ from sources outside the U.S. and ensure that the appropriate CAA regulatory mechanisms are employed. To this end, the EPA will be proposing revisions to the 2007 Exceptional Events Rule and related draft guidance addressing the effects of wildfires.

In addition to revising the primary and secondary standards, this action is changing the AQI to reflect the revisions to the primary standard and also making corresponding revisions in data handling conventions for O₃, extending the O₃ monitoring season in 33 states, revising the requirements for the PAMS network, and revising regulations for the PSD permitting program to add a provision grandfathering certain pending permits from certain requirements with respect to the revised standards. The preamble also provides schedules and information related to implementing the revised standards.

The rule also contains revisions to the schedules associated with exceptional events demonstration submittals for the revised O₃ standards and other future revised NAAQS, and makes minor changes related to monitoring for other pollutants.

I. Background

A. Legislative Requirements

Two sections of the CAA govern the establishment and revision of the NAAQS. Section 108 (42 U.S.C. 7408) directs the Administrator to identify and list certain air pollutants and then to issue air quality criteria for those pollutants. The Administrator is to list those air pollutants that in her “judgment, cause or contribute to air pollution which may reasonably be anticipated to endanger public health or welfare;” “the presence of which in the ambient air results from numerous or diverse mobile or stationary sources;” and “for which . . . [the Administrator] plans to issue air quality criteria” Air quality criteria are intended to “accurately reflect the latest scientific knowledge useful in indicating the kind and extent of all identifiable effects on public health or welfare which may be expected from the presence of [a] pollutant in the ambient air” 42 U.S.C. 7408(b). Section 109 (42 U.S.C. 7409) directs the Administrator to propose and promulgate “primary” and “secondary” NAAQS for pollutants for which air quality criteria are issued. Section 109(b)(1) defines a primary standard as one “the attainment and maintenance of which in the judgment of the Administrator, based on such criteria and allowing an adequate margin of safety, are requisite to protect the public health.”¹ A secondary standard, as defined in section 109(b)(2), must “specify a level of air quality the attainment and maintenance of which, in the judgment of the Administrator, based on such criteria, is requisite to protect the public welfare from any known or anticipated adverse effects associated with the presence of [the] pollutant in the ambient air.”²

¹ The legislative history of section 109 indicates that a primary standard is to be set at “the maximum permissible ambient air level . . . which will protect the health of any [sensitive] group of the population,” and that, for this purpose, “reference should be made to a representative sample of persons comprising the sensitive group rather than to a single person in such a group.” S. Rep. No. 91–1196, 91st Cong., 2d Sess. 10 (1970).

² Welfare effects as defined in section 302(h) (42 U.S.C. 7602(h)) include, but are not limited to, “effects on soils, water, crops, vegetation, man-made materials, animals, wildlife, weather, visibility and climate, damage to and deterioration of property, and hazards to transportation, as well

The requirement that primary standards provide an adequate margin of safety was intended to address uncertainties associated with inconclusive scientific and technical information available at the time of standard setting. It was also intended to provide a reasonable degree of protection against hazards that research has not yet identified. See *Mississippi v. EPA*, 744 F. 3d 1334, 1353 (D.C. Cir. 2013); *Lead Industries Association v. EPA*, 647 F.2d 1130, 1154 (D.C. Cir. 1980); *American Petroleum Institute v. Costle*, 665 F.2d 1176, 1186 (D.C. Cir. 1981); *American Farm Bureau Federation v. EPA*, 559 F. 3d 512, 533 (D.C. Cir. 2009); *Association of Battery Recyclers v. EPA*, 604 F. 3d 613, 617–18 (D.C. Cir. 2010). Both kinds of uncertainties are components of the risk associated with pollution at levels below those at which human health effects can be said to occur with reasonable scientific certainty. Thus, in selecting primary standards that provide an adequate margin of safety, the Administrator is seeking not only to prevent pollution levels that have been demonstrated to be harmful but also to prevent lower pollutant levels that may pose an unacceptable risk of harm, even if the risk is not precisely identified as to nature or degree. The CAA does not require the Administrator to establish a primary NAAQS at a zero-risk level or at background concentrations, see *Lead Industries v. EPA*, 647 F.2d at 1156 n.51; *Mississippi v. EPA*, 744 F. 3d at 1351, but rather at a level that reduces risk sufficiently so as to protect public health with an adequate margin of safety.

In addressing the requirement for an adequate margin of safety, the EPA considers such factors as the nature and severity of the health effects, the size of sensitive population(s)³ at risk, and the kind and degree of the uncertainties that must be addressed. The selection of any particular approach for providing an adequate margin of safety is a policy choice left specifically to the Administrator’s judgment. See *Lead Industries Association v. EPA*, 647 F.2d at 1161–62; *Mississippi*, 744 F. 3d at 1353.

In setting primary and secondary standards that are “requisite” to protect public health and welfare, respectively, as provided in section 109(b), the EPA’s task is to establish standards that are

as effects on economic values and on personal comfort and well-being.”

³ As used here with regard to human populations, and similarly throughout this document, the term “population” refers to people having a quality or characteristic in common, including a specific pre-existing illness or a specific age or lifestyle.

neither more nor less stringent than necessary for these purposes. In so doing, the EPA may not consider the costs of implementing the standards. See generally, *Whitman v. American Trucking Associations*, 531 U.S. 457, 465–472, 475–76 (2001). Likewise, “[a]ttainability and technological feasibility are not relevant considerations in the promulgation of national ambient air quality standards.” *American Petroleum Institute v. Costle*, 665 F. 2d at 1185.

Section 109(d)(1) requires that “not later than December 31, 1980, and at 5-year intervals thereafter, the Administrator shall complete a thorough review of the criteria published under section 108 and the national ambient air quality standards . . . and shall make such revisions in such criteria and standards and promulgate such new standards as may be appropriate” Section 109(d)(2) requires that an independent scientific review committee “shall complete a review of the criteria . . . and the national primary and secondary ambient air quality standards . . . and shall recommend to the Administrator any new . . . standards and revisions of existing criteria and standards as may be appropriate” Since the early 1980’s, the CASAC⁴ has performed this independent review function.

B. Related Control Programs

States are primarily responsible for ensuring attainment and maintenance of NAAQS once the EPA has established them. The EPA performs an oversight function, and as necessary takes actions to ensure CAA objectives are achieved. Under section 110 of the CAA, and related provisions, states submit, for the EPA’s approval, state implementation plans (SIPs) that provide for the attainment and maintenance of such standards through control programs directed to sources of the relevant pollutants. The states, in conjunction with the EPA, also administer the PSD program (CAA sections 160 to 169) which is a pre-construction permit program designed to prevent significant deterioration in air quality. In addition, federal programs provide for nationwide reductions in emissions of O₃ precursors and other air pollutants through new source performance standards for stationary sources under section 111 of the CAA and the federal motor vehicle and motor vehicle fuel control program under title II of the CAA (sections 202

⁴ Lists of CASAC members and of members of the CASAC Ozone Review Panel are accessible from: <http://yoosemite.epa.gov/sab/sabpeople.nsf/WebCommittees/CASAC>.

to 250), which involves controls for emissions from mobile sources and controls for the fuels used by these sources. For some stationary sources, the national emissions standards for hazardous air pollutants under section 112 of the CAA may provide ancillary reductions in O₃ precursors.

After the EPA establishes a new or revised NAAQS, the CAA directs the EPA and the states to take steps to ensure that the new or revised NAAQS are met. One of the first steps, known as the initial area designations, involves identifying areas of the country that are not meeting the new or revised NAAQS along with the nearby areas that contain emissions sources that contribute to the areas not meeting the NAAQS. For areas designated “nonattainment,” the responsible states are required to develop SIPs to attain the standards. In developing their attainment plans, states first take into account projected emission reductions from federal and state rules that have been already adopted at the time of plan submittal. A number of significant emission reduction programs that will lead to reductions of O₃ precursors are in place today or are expected to be in place by the time revised SIPs will be due. Examples of such rules include the Nitrogen Oxides (NO_x) SIP Call and Cross-State Air Pollution Rule (CSAPR),⁵ regulations controlling on-road and non-road engines and fuels, hazardous air pollutant rules for utility and industrial boilers, and various other programs already adopted by states to reduce emissions from key emissions sources. States will then evaluate the level of additional emission reductions needed for each nonattainment area to attain the O₃ standards “as expeditiously as practicable,” and adopt new state regulations as appropriate. Section VIII of this preamble includes additional discussion of designation and implementation issues associated with the revised O₃ NAAQS.

C. Review of Air Quality Criteria and Standards for O₃

The EPA first established primary and secondary NAAQS for photochemical oxidants in 1971 (36 FR 8186, April 30, 1971). The EPA set both primary and

secondary standards at 0.08 ppm,⁶ as a 1-hour average of total photochemical oxidants, not to be exceeded more than one hour per year. The EPA based the standards on scientific information contained in the 1970 *Air Quality Criteria for Photochemical Oxidants* (AQCD; U.S. DHEW, 1970). The EPA initiated the first periodic review of the NAAQS for photochemical oxidants in 1977. Based on the 1978 AQCD (U.S. EPA, 1978), the EPA published proposed revisions to the original NAAQS in 1978 (43 FR 26962, June 22, 1978) and final revisions in 1979 (44 FR 8202, February 8, 1979). At that time, the EPA revised the level of the primary and secondary standards from 0.08 to 0.12 ppm and changed the indicator from photochemical oxidants to O₃, and the form of the standards from a deterministic (*i.e.*, not to be exceeded more than one hour per year) to a statistical form. This statistical form defined attainment of the standards as occurring when the expected number of days per calendar year with maximum hourly average concentration greater than 0.12 ppm equaled one or less.

Following the EPA’s decision in the 1979 review, the city of Houston challenged the Administrator’s decision arguing that the standard was arbitrary and capricious because natural O₃ concentrations and other physical phenomena in the Houston area made the standard unattainable in that area. The U.S. Court of Appeals for the District of Columbia Circuit (D.C. Circuit) rejected this argument, holding (as noted above) that attainability and technological feasibility are not relevant considerations in the promulgation of the NAAQS. The court also noted that the EPA need not tailor the NAAQS to fit each region or locale, pointing out that Congress was aware of the difficulty in meeting standards in some locations and had addressed this difficulty through various compliance related provisions in the CAA. See *API v. Costle*, 665 F.2d 1176, 1184–6 (D.C. Cir. 1981).

In 1982, the EPA announced plans to revise the 1978 AQCD (47 FR 11561; March 17, 1982), and, in 1983, the EPA initiated the second periodic review of the O₃ NAAQS (48 FR 38009; August 22, 1983). The EPA subsequently published the 1986 AQCD (U.S. EPA, 1986) and the 1989 Staff Paper (U.S.

EPA, 1989). Following publication of the 1986 AQCD, a number of scientific abstracts and articles were published that appeared to be of sufficient importance concerning potential health and welfare effects of O₃ to warrant preparation of a Supplement (U.S. EPA, 1992). In August of 1992, under the terms of a court order, the EPA proposed to retain the existing primary and secondary standards based on the health and welfare effects information contained in the 1986 AQCD and its 1992 Supplement (57 FR 35542, August 10, 1992). In March 1993, the EPA announced its decision to conclude this review by affirming its proposed decision to retain the standards, without revision (58 FR 13008, March 9, 1993).

In the 1992 notice of its proposed decision in that review, the EPA announced its intention to proceed as rapidly as possible with the next review of the air quality criteria and standards for O₃ in light of emerging evidence of health effects related to 6- to 8-hour O₃ exposures (57 FR 35542, August 10, 1992). The EPA subsequently published the AQCD and Staff Paper for the review (U.S. EPA, 1996a,b). In December 1996, the EPA proposed revisions to both the primary and secondary standards (61 FR 65716, December 13, 1996). With regard to the primary standard, the EPA proposed to replace the then-existing 1-hour primary standard with an 8-hour standard set at a level of 0.08 ppm (equivalent to 0.084 ppm based on the proposed data handling convention) as a 3-year average of the annual third-highest daily maximum 8-hour concentration. The EPA proposed to revise the secondary standard either by setting it identical to the proposed new primary standard or by setting it as a new seasonal standard using a cumulative form. The EPA completed this review in 1997 by setting the primary standard at a level of 0.08 ppm, based on the annual fourth-highest daily maximum 8-hour average concentration, averaged over three years, and setting the secondary standard identical to the revised primary standard (62 FR 38856, July 18, 1997). In reaching her decision on the primary standard, the Administrator identified several reasons supporting her decision to reject a potential alternate standard set at 0.07 ppm, including first the fact that no CASAC panel member supported a standard level lower than 0.08 ppm and her consideration of the scientific uncertainties with regard to the health effects evidence for exposure concentrations below 0.08 ppm. In addition to those reasons, the Administrator noted that a standard set

⁵ The Cross-State Air Pollution Rule was upheld by the Supreme Court in *Environmental Protection Agency v. EME Homer City Generation, L.P.*, 134 S. Ct. 1584 (2014), and remanded to the D.C. Circuit for further proceedings. The D.C. Circuit issued its decision on remand from the Supreme Court on July 28, 2015, remanding CSAPR to EPA, without vacating the rule, for EPA to reconsider certain emission budgets for certain States (*EME Homer City Generation, L.P. v. Environmental Protection Agency*, No. 11–1302, 2015 WL 4528137 [D.C. Cir. July 28, 2015]).

⁶ Although the level of the 2008 O₃ standards are specified in the units of ppm (*i.e.*, 0.075 ppm), O₃ concentrations are described using the units of parts per billion (ppb) in several sections of this notice (*i.e.*, sections II, III, IV and VI) for consistency with the common convention for information discussed in those sections. In ppb, 0.075 ppm is equivalent to 75.

at a level of 0.07 ppm would be closer to peak background concentrations that infrequently occur in some areas due to nonanthropogenic sources of O₃ precursors (62 FR 38856, 38868; July 18, 1997).

On May 14, 1999, in response to challenges by industry and others to the EPA's 1997 decision, the D.C. Circuit remanded the O₃ NAAQS to the EPA, finding that section 109 of the CAA, as interpreted by the EPA, effected an unconstitutional delegation of legislative authority. *American Trucking Assoc. vs. EPA*, 175 F.3d 1027, 1034–1040 (D.C. Cir. 1999) (“*ATA I*”). In addition, the court directed that, in responding to the remand, the EPA should consider the potential beneficial health effects of O₃ pollution in shielding the public from the effects of solar ultraviolet (UV) radiation, as well as adverse health effects. *Id.* at 1051–53. In 1999, the EPA petitioned for rehearing *en banc* on several issues related to that decision. The court granted the request for rehearing in part and denied it in part, but declined to review its ruling with regard to the potential beneficial effects of O₃ pollution. 195 F. 3d 4, 10 (D.C. Cir., 1999) (“*ATA II*”). On January 27, 2000, the EPA petitioned the U.S. Supreme Court for *certiorari* on the constitutional issue (and two other issues), but did not request review of the ruling regarding the potential beneficial health effects of O₃. On February 27, 2001, the U.S. Supreme Court unanimously reversed the judgment of the D.C. Circuit on the constitutional issue. *Whitman v. American Trucking Assoc.*, 531 U. S. 457, 472–74 (2001) (holding that section 109 of the CAA does not delegate legislative power to the EPA in contravention of the Constitution). The Court remanded the case to the D.C. Circuit to consider challenges to the O₃ NAAQS that had not been addressed by that court's earlier decisions. On March 26, 2002, the D.C. Circuit issued its final decision on remand, finding the 1997 O₃ NAAQS to be “neither arbitrary nor capricious,” and so denying the remaining petitions for review. *American Trucking Associations, Inc. v. EPA*, 283 F.3d 355, 379 (D.C. Cir., 2002) (“*ATA III*”).

Specifically, in *ATA III*, the D.C. Circuit upheld the EPA's decision on the 1997 O₃ standard as the product of reasoned decision making. With regard to the primary standard, the court made clear that the most important support for EPA's decision to revise the standard was the health evidence of insufficient protection afforded by the then-existing standard (“the record is replete with references to studies demonstrating the

inadequacies of the old one-hour standard”), as well as extensive information supporting the change to an 8-hour averaging time (283 F. 3d at 378). The court further upheld the EPA's decision not to select a more stringent level for the primary standard noting “the absence of any human clinical studies at ozone concentrations below 0.08 [ppm]” which supported the EPA's conclusion that “the most serious health effects of ozone are ‘less certain’ at low concentrations, providing an eminently rational reason to set the primary standard at a somewhat higher level, at least until additional studies become available” (283 F. 3d at 378, internal citations omitted). The court also pointed to the significant weight that the EPA properly placed on the advice it received from CASAC (283 F. 3d at 379). In addition, the court noted that “although relative proximity to peak background O₃ concentrations did not, in itself, necessitate a level of 0.08 [ppm], the EPA could consider that factor when choosing among the three alternative levels” (283 F. 3d at 379).

Independently of the litigation, the EPA responded to the court's remand to consider the potential beneficial health effects of O₃ pollution in shielding the public from effects of UV radiation. The EPA provisionally determined that the information linking changes in patterns of ground-level O₃ concentrations to changes in relevant patterns of exposures to UV radiation of concern to public health was too uncertain, at that time, to warrant any relaxation in 1997 O₃ NAAQS. The EPA also expressed the view that any plausible changes in UV-B radiation exposures from changes in patterns of ground-level O₃ concentrations would likely be very small from a public health perspective. In view of these findings, the EPA proposed to leave the 1997 primary standard unchanged (66 FR 57268, Nov. 14, 2001). After considering public comment on the proposed decision, the EPA published its final response to this remand in 2003, re-affirming the 8-hour primary standard set in 1997 (68 FR 614, January 6, 2003).

The EPA initiated the fourth periodic review of the air quality criteria and standards for O₃ with a call for information in September 2000 (65 FR 57810, September, 26, 2000). The schedule for completion of that review was ultimately governed by a consent decree resolving a lawsuit filed in March 2003 by plaintiffs representing national environmental and public health organizations, who maintained that the EPA was in breach of a nondiscretionary duty to complete review of the O₃ NAAQS within a

statutorily mandated deadline. In 2007, the EPA proposed to revise the level of the primary standard within a range of 0.075 to 0.070 ppm (72 FR 37818, July 11, 2007). The EPA proposed to revise the secondary standard either by setting it identical to the proposed new primary standard or by setting it as a new seasonal standard using a cumulative form. Documents supporting these proposed decisions included the 2006 AQCD (U.S. EPA, 2006a) and 2007 Staff Paper (U.S. EPA, 2007) and related technical support documents. The EPA completed the review in March 2008 by revising the level of the primary standard from 0.08 ppm to 0.075 ppm, and revising the secondary standard to be identical to the revised primary standard (73 FR 16436, March 27, 2008).

In May 2008, state, public health, environmental, and industry petitioners filed suit challenging the EPA's final decision on the 2008 O₃ standards. On September 16, 2009, the EPA announced its intention to reconsider the 2008 O₃ standards, and initiated a rulemaking to do so. At the EPA's request, the court held the consolidated cases in abeyance pending the EPA's reconsideration of the 2008 decision.

On January 2010, the EPA issued a notice of proposed rulemaking to reconsider the 2008 final decision (75 FR 2938, January 19, 2010). In that notice, the EPA proposed that further revisions of the primary and secondary standards were necessary to provide a requisite level of protection to public health and welfare. The EPA proposed to revise the level of the primary standard from 0.075 ppm to a level within the range of 0.060 to 0.070 ppm, and to revise the secondary standard to one with a cumulative, seasonal form. At the EPA's request, the CASAC reviewed the proposed rule at a public teleconference on January 25, 2010 and provided additional advice in early 2011 (Samet, 2010, 2011). After considering comments from CASAC and the public, the EPA prepared a draft final rule, which was submitted for interagency review pursuant to Executive Order 12866. On September 2, 2011, consistent with the direction of the President, the Administrator of the Office of Information and Regulatory Affairs, Office of Management and Budget (OMB), returned the draft final rule to the EPA for further consideration. In view of this return and the fact that the Agency's next periodic review of the O₃ NAAQS required under CAA section 109 had already begun (as announced on September 29, 2008), the EPA decided to consolidate the

reconsideration with its statutorily required periodic review.⁷

In light of the EPA's decision to consolidate the reconsideration with the current review, the D.C. Circuit proceeded with the litigation on the 2008 final decision. On July 23, 2013, the court upheld the EPA's 2008 primary O₃ standard, but remanded the 2008 secondary standard to the EPA (*Mississippi v. EPA*, 744 F. 3d 1334). With respect to the primary standard, the court first held that the EPA reasonably determined that the existing standard was not requisite to protect public health with an adequate margin of safety, and consequently required revision. Specifically, the court noted that there were "numerous epidemiologic studies linking health effects to exposure to ozone levels below 0.08 ppm and clinical human exposure studies finding a causal relationship between health effects and exposure to ozone levels at and below 0.08 ppm" (*Mississippi v. EPA*, 744 F. 3d at 1345). The court also specifically endorsed the weight of evidence approach utilized by the EPA in its deliberations (*Mississippi v. EPA*, 744 F. 3d at 1344).

The court went on to reject arguments that the EPA should have adopted a more stringent primary standard. Dismissing arguments that a clinical study (as properly interpreted by the EPA) showing effects at 0.06 ppm necessitated a standard level lower than that selected, the court noted that this was a single, limited study (*Mississippi v. EPA*, 744 F. 3d at 1350). With respect to the epidemiologic evidence, the court accepted the EPA's argument that there could be legitimate uncertainty that a causal relationship between O₃ and 8-hour exposures less than 0.075 ppm exists, so that associations at lower levels reported in epidemiologic studies did not necessitate a more stringent standard (*Mississippi v. EPA*, 744 F. 3d at 1351–52).⁸

The court also rejected arguments that an 8-hour primary standard of 0.075 ppm failed to provide an adequate margin of safety, noting that margin of

⁷ This rulemaking concludes the reconsideration process. Under CAA section 109, the EPA is required to base its review of the NAAQS on the current air quality criteria, and thus the record and decision for this review also serve for the reconsideration.

⁸ The court cautioned, however, that "perhaps more [clinical] studies like the Adams studies will yet reveal that the 0.060 ppm level produces significant adverse decrements that simply cannot be attributed to normal variation in lung function," and further cautioned that "agencies may not merely recite the terms 'substantial uncertainty' as a justification for their actions." *Id.* at 1350, 1357 (internal citations omitted).

safety considerations involved policy judgments by the agency, and that by setting a standard "appreciably below" the level of the current standard (0.08 ppm), the agency had made a reasonable policy choice (*Mississippi v. EPA*, 744 F. 3d at 1351–52). Finally, the court rejected arguments that the EPA's decision was inconsistent with the CASAC's scientific recommendations because the CASAC had been insufficiently clear in its recommendations whether it was providing scientific or policy recommendations, and the EPA had reasonably addressed the CASAC's policy recommendations (*Mississippi v. EPA*, 744 F. 3d at 1357–58).

With respect to the secondary standard, the court held that the EPA's justification for setting the secondary standard identical to the revised 8-hour primary standard violated the CAA because the EPA had not adequately explained how that standard provided the required public welfare protection. The court thus remanded the secondary standard to the EPA (*Mississippi v. EPA*, 744 F. 3d at 1360–62).

At the time of the court's decision, the EPA had already completed significant portions of its next statutorily required periodic review of the O₃ NAAQS. This review was formally initiated in 2008 with a call for information in the **Federal Register** (73 FR 56581, Sept. 29, 2008). On October 28–29, 2008, the EPA held a public workshop to discuss the policy-relevant science, which informed identification of key policy issues and questions to frame the review. Based in part on the workshop discussions, the EPA developed a draft Integrated Review Plan (IRP) outlining the schedule, process,⁹ and key policy-relevant questions that would guide the evaluation of the air quality criteria for O₃ and the review of the primary and secondary O₃ NAAQS. A draft of the IRP was released for public review and comment in September 2009 and was the subject of a consultation with the CASAC on November 13, 2009 (74 FR 54562; October 22, 2009).¹⁰ After considering the comments received from that consultation and from the public, the EPA completed and released the IRP for the review in 2011 (U.S. EPA, 2011a).

⁹ As of this review, the document developed in NAAQS reviews to document the air quality criteria, previously the AQCD, is the ISA, and the document describing the OAQPS staff evaluation, previously the Staff Paper, is the PA. These documents are described in the IRP.

¹⁰ See <http://yosemite.epa.gov/sab/sabproduct.nsf/WebProjectsbyTopicCASAC!OpenView> for more information on CASAC activities related to the current O₃ NAAQS review.

In preparing the first draft ISA, the EPA's National Center for Environmental Assessment (NCEA) considered CASAC and public comments on the IRP, and also comments received from a workshop held on August 6, 2010, to review and discuss preliminary drafts of key ISA sections (75 FR 42085, July 20, 2010). In 2011, the first draft ISA was released for public comment and for review by CASAC at a public meeting on May 19–20, 2011 (U.S. EPA, 2011b; 76 FR 10893, February 28, 2011; 76 FR 23809, April 28, 2011). Based on CASAC and public comments, NCEA prepared a second draft ISA, which was released for public comment and CASAC review (U.S. EPA, 2011c; 76 FR 60820, September 30, 2011). The CASAC reviewed this draft at a January 9–10, 2012, public meeting (76 FR 236, December 8, 2011). Based on CASAC and public comments, NCEA prepared a third draft ISA (U.S. EPA, 2012; 77 FR 36534, June 19, 2012), which was reviewed at a CASAC meeting in September 2012. The EPA released the final ISA in February 2013 (U.S. EPA, 2013).

The EPA presented its plans for conducting Risk and Exposure Assessments (REAs) for health risk and exposure (HREA) and welfare risk and exposure (WREA) in two documents that outlined the scope and approaches for use in conducting quantitative assessments, as well as key issues to be addressed as part of the assessments (U.S. EPA, 2011d, e). The EPA released these documents for public comment in April 2011, and consulted with CASAC on May 19–20, 2011 (76 FR 23809, April 28, 2011). The EPA considered CASAC advice and public comments in further planning for the assessments, issuing a memo that described changes to elements of the REA plans and brief explanations regarding them (Samet, 2011; Wegman, 2012).

In July 2012, the EPA made the first drafts of the Health and Welfare REAs available for CASAC review and public comment (77 FR 42495, July 19, 2012; 77 FR 51798, August 27, 2012). The first draft PA was made available for CASAC review and public comment in August 2012 (77 FR 42495, July 19, 2012; 77 FR 51798, August 27, 2012).¹¹ The first

¹¹ The PA is prepared by the OAQPS staff. Formerly known as the Staff Paper, it presents a staff evaluation of the policy implications of the key scientific and technical information in the ISA and REAs for the EPA's consideration. The PA provides a transparent evaluation, and staff conclusions, regarding policy considerations related to reaching judgments about the adequacy of the current standards, and if revision is considered, what revisions may be appropriate to consider. The PA is intended to help "bridge the gap" between the agency's scientific assessments presented in the ISA

draft REAs and PA were the focus of a CASAC public meeting in September 2012 (Frey and Samet, 2012a, 2012b). The second draft REAs and PA, prepared with consideration of CASAC advice and public comments, were made available for public comment and CASAC review in January 2014 (79 FR 4694, January 29, 2014). These documents were the focus of a CASAC public meeting on March 25–27, 2014 (Frey, 2014a; Frey, 2014b; Frey, 2014c). The final versions of these documents were developed with consideration of the comments and recommendations from CASAC, as well as comments from the public on the draft documents, and were released in August 2014 (U.S. EPA 2014a; U.S. EPA, 2014b; U.S. EPA, 2014c).

The proposed decision (henceforth “proposal”) on this review of the O₃ NAAQS was signed on November 25, 2014, and published in the **Federal Register** on December 17, 2014. The EPA held three public hearings to provide direct opportunity for oral testimony by the public on the proposal. The hearings were held on January 29, 2015, in Arlington, Texas, and Washington, DC, and on February 2, 2015, in Sacramento, California. At these public hearings, the EPA heard testimony from nearly 500 individuals representing themselves or specific interested organizations. Transcripts from these hearings and written testimony provided at the hearings are in the docket for this review. Additionally, approximately 430,000 written comments were received from various commenters during the public comment period on the proposal, approximately 428,000 as part of mass mail campaigns. Significant issues raised in the public comments are discussed in the preamble of this final action. A summary of all other significant comments, along with the EPA’s responses, can be found in a separate document (henceforth “Response to Comments”) in the docket for this review.

The schedule for completion of this review is governed by a court order resolving a lawsuit filed in January 2014 by a group of plaintiffs who alleged that the EPA had failed to perform its mandatory duty, under section 109(d)(1), to complete a review of the O₃ NAAQS within the period provided by statute. The court order that governs this review, entered by the court on April 30, 2014, provides that the EPA will sign for publication a notice of final

rulemaking concerning its review of the O₃ NAAQS no later than October 1, 2015.

As in prior NAAQS reviews, the EPA is basing its decision in this review on studies and related information included in the ISA, REAs and PA, which have undergone CASAC and public review. The studies assessed in the ISA and PA, and the integration of the scientific evidence presented in them, have undergone extensive critical review by the EPA, the CASAC, and the public. The rigor of that review makes these studies, and their integrative assessment, the most reliable source of scientific information on which to base decisions on the NAAQS, decisions that all parties recognize as of great import. NAAQS decisions can have profound impacts on public health and welfare, and NAAQS decisions should be based on studies that have been rigorously assessed in an integrative manner not only by the EPA but also by the statutorily mandated independent advisory committee, as well as the public review that accompanies this process. Some commenters have referred to and discussed individual scientific studies on the health and welfare effects of O₃ that were not included in the ISA (USEPA, 2013) (“new” studies”). In considering and responding to comments for which such “new” studies were cited in support, the EPA has provisionally considered the cited studies in the context of the findings of the ISA. The EPA’s provisional consideration of these studies did not and could not provide the kind of in-depth critical review described above.

The decision to rely on studies and related information included in the ISA, REAs and PA, which have undergone CASAC and public review, is consistent with the EPA’s practice in prior NAAQS reviews and its interpretation of the requirements of the CAA. Since the 1970 amendments, the EPA has taken the view that NAAQS decisions are to be based on scientific studies and related information that have been assessed as a part of the pertinent air quality criteria, and the EPA has consistently followed this approach. This longstanding interpretation was strengthened by new legislative requirements enacted in 1977, which added section 109(d)(2) of the Act concerning CASAC review of air quality criteria. See 71 FR 61144, 61148 (October 17, 2006) (final decision on review of NAAQS for particulate matter) for a detailed discussion of this issue and the EPA’s past practice.

As discussed in the EPA’s 1993 decision not to revise the NAAQS for

O₃, “new” studies may sometimes be of such significance that it is appropriate to delay a decision on revision of a NAAQS and to supplement the pertinent air quality criteria so the studies can be taken into account (58 FR at 13013–13014, March 9, 1993). In the present case, the EPA’s provisional consideration of “new” studies concludes that, taken in context, the “new” information and findings do not materially change any of the broad scientific conclusions regarding the health and welfare effects and exposure pathways of ambient O₃ made in the air quality criteria. For this reason, reopening the air quality criteria review would not be warranted even if there were time to do so under the court order governing the schedule for this rulemaking.

Accordingly, the EPA is basing the final decisions in this review on the studies and related information included in the O₃ air quality criteria that have undergone CASAC and public review. The EPA will consider the “new” studies for purposes of decision making in the next periodic review of the O₃ NAAQS, which the EPA expects to begin soon after the conclusion of this review and which will provide the opportunity to fully assess these studies through a more rigorous review process involving the EPA, CASAC, and the public. Further discussion of these “new” studies can be found in the Response to Comments document, which is in the docket for this rulemaking and also available on the web (http://www.epa.gov/ttn/naaqs/standards/ozone/s_o3_index.html).

D. Ozone Air Quality

Ozone is formed near the earth’s surface due to chemical interactions involving solar radiation and precursor pollutants including volatile organic compounds (VOCs) and NO_x. Over longer time periods, methane (CH₄) and carbon monoxide (CO) can also lead to O₃ formation at the global scale. The precursor emissions leading to O₃ formation can result from both man-made sources (e.g., motor vehicles and electric power generation) and natural sources (e.g., vegetation and wildfires). Occasionally, O₃ that is created naturally in the stratosphere can also contribute to O₃ levels near the surface. Once formed, O₃ near the surface can be transported by winds before eventually being removed from the atmosphere via chemical reactions or deposition to surfaces. In sum, O₃ concentrations are influenced by complex interactions between precursor emissions, meteorological conditions, and surface characteristics (U.S. EPA, 2014a).

and REAs, and the judgments required of the EPA Administrator in determining whether it is appropriate to retain or revise the NAAQS.

In order to continuously assess O₃ air pollution levels, state and local environmental agencies operate O₃ monitors at various locations and subsequently submit the data to the EPA. At present, there are approximately 1,400 monitors across the U.S. reporting hourly O₃ averages during the times of the year when local O₃ pollution can be important (U.S. EPA, 2014c, Section 2.1). Much of this monitoring is focused on urban areas where precursor emissions tend to be largest, as well as locations directly downwind of these areas, but there are also over 100 sites in rural areas where high levels of O₃ can also be measured. Based on data from this national network, the EPA estimates that, in 2013, approximately 99 million Americans lived in counties where O₃ design values¹² were above the level of the existing health-based (primary) NAAQS of 0.075 ppm. High O₃ values can occur almost anywhere within the contiguous 48 states, although the poorest O₃ air quality in the U.S. is typically observed in California, Texas, and the Northeast Corridor, locations with some of the most densely populated areas in the country. From a temporal perspective, the highest daily peak O₃ concentrations generally tend to occur during the afternoon within the warmer months due to higher solar radiation and other conducive meteorological conditions during these times. The exceptions to this general rule include 1) some rural sites where transport of O₃ from upwind areas of regional production can occasionally result in high nighttime levels of O₃, 2) high-elevation sites episodically influenced by stratospheric intrusions which can occur in other months, and 3) certain locations in the western U.S. where large quantities of O₃ precursors emissions associated with oil and gas development can be trapped by strong inversions associated with snow cover during the colder months and efficiently converted to O₃ (U.S. EPA, 2014c, Section 2.3).

One of the challenging aspects of developing plans to address high O₃ concentrations is that the response of O₃ to precursor reductions is nonlinear. In particular, NO_x emissions can lead to both increases and decreases of O₃. The net impact of NO_x emissions on O₃ concentrations depends on the local quantities of NO_x, VOC, and sunlight which interact in a set of complex chemical reactions. In some areas, such as certain urban centers where NO_x

emissions typically are high compared to local VOC emissions, NO_x can suppress O₃ locally. This phenomenon is particularly pronounced under conditions associated with low O₃ concentrations (*i.e.*, during cool, cloudy weather and at night when photochemical activity is limited or nonexistent). However, while NO_x emissions can initially suppress O₃ levels near the emission sources, these same NO_x emissions ultimately react to form higher O₃ levels downwind when conditions are favorable. Photochemical model simulations suggest that, in general, reductions in NO_x emissions in the U.S. will slightly increase O₃ concentrations on days with lower O₃ concentrations in close proximity to NO_x sources (*e.g.*, in urban core areas), while at the same time decreasing the highest O₃ concentrations in downwind areas. See generally, U.S. EPA, 2014a (section 2.2.1).

At present, both the primary and secondary NAAQS use the annual fourth-highest daily maximum 8-hour concentration, averaged over 3 years, as the form of the standard. An additional metric, the W126 exposure index, is often used to assess impacts of O₃ exposure on ecosystems and vegetation. W126 is a cumulative seasonal aggregate of weighted hourly O₃ values observed between 8 a.m. and 8 p.m. As O₃ precursor emissions have decreased across the U.S., annual fourth-highest 8-hour O₃ maxima have concurrently shown a modest downward trend. The national average change in annual fourth-highest daily maximum 8-hour O₃ concentrations between 2000 and 2013 was an 18% decrease. The national average change in the annual W126 exposure index over the same period was a 52% decrease. Air quality model simulations estimate that O₃ air quality will continue to improve over the next decade as additional reductions in O₃ precursors from power plants, motor vehicles, and other sources are realized.

In addition to being affected by changing emissions, future O₃ concentrations may also be affected by climate change. Modeling studies in the EPA's Interim Assessment (U.S. EPA, 2009a) that are cited in support of the 2009 Endangerment Finding under CAA section 202(a) (74 FR 66496, Dec. 15, 2009) as well as a recent assessment of potential climate change impacts (Fann et al., 2015) project that climate change may lead to future increases in summer O₃ concentrations across the contiguous U.S.¹³ While the projected impact is not

uniform, climate change has the potential to increase average summertime O₃ concentrations by as much as 1–5 ppb by 2030, if greenhouse gas emissions are not mitigated. Increases in temperature are expected to be the principal factor in driving any O₃ increases, although increases in stagnation frequency may also contribute (Jacob and Winner, 2009). If unchecked, climate change has the potential to offset some of the improvements in O₃ air quality, and therefore some of the improvements in public health, that are expected from reductions in emissions of O₃ precursors.

Another challenging aspect of this air quality issue is the impact from sources of O₃ and its precursors beyond those from domestic, anthropogenic sources. Modeling analyses indicate that nationally the majority of O₃ exceedances are predominantly caused by anthropogenic emissions from within the U.S. However, observational and modeling analyses have concluded that O₃ concentrations in some locations in the U.S. on some days can be substantially influenced by sources that cannot be addressed by domestic control measures. In particular, certain high-elevation sites in the western U.S. are impacted by a combination of non-U.S. sources like international transport, or natural sources such as stratospheric O₃, and O₃ originating from wildfire emissions.¹⁴ Ambient O₃ from these non-U.S. and natural sources is collectively referred to as background O₃. See generally section 2.4 of the PA (U.S. EPA, 2014c). The analyses suggest that, at these locations, there can be episodic events with substantial background contributions where O₃ concentrations approach or exceed the level of the current NAAQS (*i.e.*, 75 ppb). These events are relatively infrequent, and the EPA has policies that allow for the exclusion of air quality monitoring data from design value calculations when they are substantially affected by certain background influences.

E. Summary of Proposed Revisions to the O₃ Standards

For reasons discussed in the proposal, the Administrator proposed to revise the

quality to climate change. A wide range of future climate scenarios and future years have been modeled and there can be variations in the expected response in U.S. O₃ by scenario and across models and years, within the overall signal of higher summer O₃ concentrations in a warmer climate.

¹⁴ Without global greenhouse gas mitigation efforts, climate change is projected to dramatically increase the area burned by wildfires across most of the contiguous U.S., especially in the West (U.S. EPA, 2015 p. 72).

¹² A design value is a statistic that describes the air quality status of a given location relative to the level of the NAAQS.

¹³ These modeling studies are based on coupled global climate and regional air quality models and are designed to assess the sensitivity of U.S. air

current primary and secondary standards for O₃. With regard to the primary standard, the Administrator proposed to revise the level from 75 ppb to a level within a range from 65 to 70 ppb. The EPA proposed to revise the AQI for O₃, consistent with revision to the primary standard.

With regard to the secondary standard, the Administrator proposed to revise the level of the current secondary standard to within the range of 0.065 to 0.070 ppm, which air quality analyses indicate would provide cumulative, seasonal air quality or exposure values, in terms of 3-year average W126 index values, at or below a range of 13–17 ppm-hours.

The EPA also proposed to make corresponding revisions in data handling conventions for O₃; to revise regulations for the PSD permitting program to add a provision grandfathering certain pending permits from certain requirements with respect to the proposed revisions to the standards; and to convey schedules and information related to implementing any revised standards. In conjunction with proposing exceptional event schedules related to implementing any revised O₃ standards, the EPA also proposed to extend the new schedule approach to other future NAAQS revisions and to remove obsolete regulatory language associated with expired exceptional event deadlines for historical standards for both O₃ and other pollutants for which NAAQS have been established. The EPA also proposed to make minor changes to the procedures and time periods for evaluating potential FRMs and equivalent methods, including making the requirements for NO₂ consistent with the requirements for O₃, and removing an obsolete requirement for the annual submission of documentation by manufacturers of certain particulate matter monitors.

F. Organization and Approach to Decisions in This O₃ NAAQS Review

This action presents the Administrator's final decisions in the current review of the primary and secondary O₃ standards. The final decisions addressing standards for O₃ are based on a thorough review in the ISA of scientific information on known and potential human health and welfare effects associated with exposure to O₃ at levels typically found in the ambient air. These final decisions also take into account the following: (1) Staff assessments in the PA of the most policy-relevant information in the ISA as well as a quantitative health and welfare exposure and risk assessments

based on that information; (2) CASAC advice and recommendations, as reflected in its letters to the Administrator and its discussions of drafts of the ISA, REAs, and PA at public meetings; (3) public comments received during the development of these documents, both in connection with CASAC meetings and separately; and (4) extensive public comments received on the proposed rulemaking.

The primary standard is addressed in section II. Corresponding changes to the AQI are addressed in section III. The secondary standard is addressed in section IV. Related data handling conventions and exceptional events are addressed in section V. Updates to the monitoring regulations are addressed in section VI. Implementation activities, including PSD-related actions, are addressed in sections VII and VIII. Section IX addresses applicable statutory and executive order reviews.

II. Rationale for Decision on the Primary Standard

This section presents the Administrator's final decisions regarding the need to revise the existing primary O₃ standard and the appropriate revision to the level of that standard. Based on her consideration of the full body of health effects evidence and exposure/risk analyses, the Administrator concludes that the current primary standard for O₃ is not requisite to protect public health with an adequate margin of safety. In order to increase public health protection, she is revising the level of the primary standard to 70 ppb, in conjunction with retaining the current indicator, averaging time and form. The Administrator concludes that such a revised standard will be requisite to protect public health with an adequate margin of safety. As discussed more fully below, the rationale for these final decisions draws from the thorough review in the ISA (U.S. EPA, 2013) of the available scientific evidence, generally published through July 2011, on human health effects associated with the presence of O₃ in the ambient air. This rationale also takes into account: (1) Analyses of O₃ air quality, human exposures to O₃, and O₃-associated health risks, as presented and assessed in the HREA (U.S. EPA, 2014a); (2) the EPA staff assessment of the most policy-relevant scientific evidence and exposure/risk information in the PA (U.S. EPA, 2014c); (3) CASAC advice and recommendations, as reflected in discussions of drafts of the ISA, REA, and PA at public meetings, in separate written comments, and in CASAC's letters to the Administrator; (4) public

input received during the development of these documents, either in connection with CASAC meetings or separately; and (5) public comments on the proposal notice.

Section II.A below summarizes the information presented in the proposal regarding O₃-associated health effects, O₃ exposures, and O₃-attributable health risks. Section II.B presents information related to the adequacy of the current primary O₃ standard, including a summary of the basis for the Administrator's proposed decision to revise the current standard, public comments received on the adequacy of the current standard, and the Administrator's final conclusions regarding the adequacy of the current standard. Section II.C presents information related to the elements of a revised primary O₃ standard, including information related to each of the major elements of the standard (*i.e.*, indicator, averaging time, form, level). Section II.D summarizes the Administrator's final decisions on the primary O₃ standard.

A. Introduction

As discussed in section II.A of the proposal (79 FR 75243–75246, December 17, 2014), the EPA's approach to informing decisions on the primary O₃ standard in the current review builds upon the general approaches used in previous reviews and reflects the broader body of scientific evidence, updated exposure/risk information, and advances in O₃ air quality modeling now available. This approach is based most fundamentally on using the EPA's assessment of the available scientific evidence and associated quantitative analyses to inform the Administrator's judgments regarding a primary standard for O₃ that is "requisite" (*i.e.*, neither more nor less stringent than necessary) to protect public health with an adequate margin of safety. Specifically, it is based on consideration of the available body of scientific evidence assessed in the ISA (U.S. EPA, 2013), exposure and risk analyses presented in the HREA (U.S. EPA, 2014a), evidence- and exposure-/risk-based considerations and conclusions presented in the PA (U.S. EPA, 2014c), advice and recommendations received from CASAC (Frey, 2014a, c), and public comments.

Section II.A.1 below summarizes the information presented in the proposal regarding O₃-associated health effects. Section II.A.2 summarizes the information presented in the proposal regarding O₃ exposures and O₃-attributable health risks.

1. Overview of Health Effects Evidence

The health effects of O₃ are described in detail in the ISA (U.S. EPA, 2013). Based on its assessment of the health effects evidence, the ISA determined that a “causal” relationship exists between short-term exposure to O₃ in ambient air and effects on the respiratory system¹⁵ and that a “likely to be causal” relationship exists between long-term exposure to O₃ in ambient air and respiratory effects¹⁶ (U.S. EPA, 2013, pp. 1–6 to 1–7). The ISA summarizes the longstanding body of evidence for O₃ respiratory effects as follows (U.S. EPA, 2013, p. 1–5):

The clearest evidence for health effects associated with exposure to O₃ is provided by studies of respiratory effects. Collectively, a very large amount of evidence spanning several decades supports a relationship between exposure to O₃ and a broad range of respiratory effects (see Section 6.2.9 and Section 7.2.8). The majority of this evidence is derived from studies investigating short-term exposures (*i.e.*, hours to weeks) to O₃, although animal toxicological studies and recent epidemiologic evidence demonstrate that long-term exposure (*i.e.*, months to years) may also harm the respiratory system.

Additionally, the ISA determined that the relationships between short-term exposures to O₃ in ambient air and both total mortality and cardiovascular effects are likely to be causal, based on expanded evidence bases in the current review (U.S. EPA, 2013, pp. 1–7 to 1–8). The ISA determined that the currently available evidence for additional endpoints is “suggestive” of causal relationships with short-term (central nervous system effects) and long-term exposures (cardiovascular effects, reproductive and developmental effects, central nervous system effects and total mortality) to ambient O₃.

Consistent with emphasis in past reviews on O₃ health effects for which the evidence is strongest, in this review the EPA places the greatest emphasis on studies of health effects that have been determined in the ISA to be caused by, or likely to be caused by, O₃ exposures (U.S. EPA, 2013, section 2.5.2). This preamble section summarizes the evidence for health effects attributable to O₃ exposures, with a focus on respiratory morbidity and mortality

¹⁵ In determining that a causal relationship exists for O₃ with specific health effects, the EPA has concluded that “[e]vidence is sufficient to conclude that there is a causal relationship with relevant pollutant exposures” (U.S. EPA, 2013, p. lxiv).

¹⁶ In determining a “likely to be causal” relationship exists for O₃ with specific health effects, the EPA has concluded that “[e]vidence is sufficient to conclude that a causal relationship is likely to exist with relevant pollutant exposures, but important uncertainties remain” (U.S. EPA, 2013, p. lxiv).

effects attributable to short- and long-term exposures, and cardiovascular system effects (including mortality) and total mortality attributable to short-term exposures (from section II.B in the proposal, 79 FR 75246–75271).

The information highlighted here is based on the assessment of the evidence in the ISA (U.S. EPA, 2013, Chapters 4 to 8) and consideration of that evidence in the PA (U.S. EPA, 2014c, Chapters 3 and 4) on the known or potential effects on public health which may be expected from the presence of O₃ in the ambient air. This section summarizes: (1) Information available on potential mechanisms for health effects associated with exposure to O₃ (II.A.1.a); (2) the nature of effects that have been associated directly with both short- and long-term exposure to O₃ and indirectly with the presence of O₃ in ambient air (II.A.1.b); (3) considerations related to the adversity of O₃-attributable health effects (II.A.1.c); and (4) considerations in characterizing the public health impact of O₃, including the identification of “at risk” populations (II.A.1.d).

a. Overview of Mechanisms

This section briefly summarizes the characterization of the key events and pathways that contribute to health effects resulting from O₃ exposures, as discussed in the proposal (79 FR 75247, section II.B.1) and in the ISA (U.S. EPA, 2013, section 5.3).

Experimental evidence elucidating modes of action and/or mechanisms contributes to our understanding of the biological plausibility of adverse O₃-related health effects, including respiratory effects and effects outside the respiratory system (U.S. EPA, 2013, Chapters 6 and 7). Evidence indicates that the initial key event is the formation of secondary oxidation products in the respiratory tract (U.S. EPA, 2013, section 5.3). This mainly involves direct reactions with components of the extracellular lining fluid (ELF). Although the ELF has inherent capacity to quench (based on individual antioxidant capacity), this capacity can be overwhelmed, especially with exposure to elevated concentrations of O₃ (U.S. EPA 2014c, at 3–3, 3–9). The resulting secondary oxidation products transmit signals to the epithelium, pain receptive nerve fibers and, if present, immune cells involved in allergic responses. The available evidence indicates that the effects of O₃ are mediated by components of ELF and by the multiple cell types in the respiratory tract. Oxidative stress is an implicit part of this initial key event.

Secondary oxidation products initiate numerous responses at the cellular, tissue, and whole organ level of the respiratory system. These responses include the activation of neural reflexes which leads to lung function decrements; initiation of pulmonary inflammation; alteration of barrier epithelial function; sensitization of bronchial smooth muscle; modification of lung host defenses; airways remodeling; and modulation of autonomic nervous function which may alter cardiac function (U.S. EPA, 2013, section 5.3, Figure 5–8).

Persistent inflammation and injury, which are observed in animal models of chronic and quasi-continuous exposure to O₃, are associated with airways remodeling (see section 7.2.3 of the ISA, U.S. EPA, 2013). Chronic quasi-continuous exposure to O₃ has also been shown to result in effects on the developing lung and immune system. Systemic inflammation and vascular oxidative/nitrosative stress are also key events in the toxicity pathway of O₃ (U.S. EPA, 2013, section 5.3.8). Extrapulmonary effects of O₃ occur in numerous organ systems, including the cardiovascular, central nervous, reproductive, and hepatic systems (U.S. EPA, 2013, sections 6.3 to 6.5 and sections 7.3 to 7.5).

Responses to O₃ exposure are variable within the population. Studies have shown a large range of pulmonary function (*i.e.*, spirometric) responses to O₃ among healthy young adults, while responses within an individual are relatively consistent over time. Other responses to O₃ have also been characterized by a large degree of interindividual variability, including airways inflammation. The mechanisms that may underlie the variability in responses seen among individuals are discussed in the ISA (U.S. EPA, 2013, section 5.4.2). Certain functional genetic polymorphisms, pre-existing conditions or diseases, nutritional status, lifestages, and co-exposures can contribute to altered risk of O₃-induced effects. Experimental evidence for such O₃-induced changes contributes to our understanding of the biological plausibility of adverse O₃-related health effects, including a range of respiratory effects as well as effects outside the respiratory system (*e.g.*, cardiovascular effects) (U.S. EPA, 2013, Chapters 6 and 7).

b. Nature of Effects

This section briefly summarizes the information presented in the proposal on respiratory effects attributable to short-term exposures (II.A.1.b.i), respiratory effects attributable to long-

term exposures (II.A.1.b.ii), cardiovascular effects attributable to short-term exposures (II.A.1.b.iii), and premature mortality attributable to short-term exposures (II.A.1.b.iv) (79 FR 75247, section II.B.2).

i. Respiratory Effects—Short-term Exposure

Controlled human exposure, animal toxicological, and epidemiologic studies available in the last review provided clear, consistent evidence of a causal relationship between short-term O₃ exposure and respiratory effects (U.S. EPA, 2006a). Recent studies evaluated since the completion of the 2006 AQCD support and expand upon the strong body of evidence available in the last review (U.S. EPA, 2013, section 6.2.9).

Key aspects of this evidence are discussed below with regard to (1) lung function decrements; (2) pulmonary inflammation, injury, and oxidative stress; (3) airway hyperresponsiveness; (4) respiratory symptoms and medication use; (5) lung host defense; (6) allergic and asthma-related responses; (7) hospital admissions and emergency department visits; and (8) respiratory mortality.¹⁷

Lung Function Decrements

Lung function decrements are typically measured by spirometry and refer to reductions in the maximal amount of air that can be forcefully exhaled. Forced expiratory volume in 1 second (FEV₁) is a common index used to assess the effect of O₃ on lung function. The ISA summarizes the currently available evidence from multiple controlled human exposure studies evaluating changes in FEV₁ following 6.6-hour O₃ exposures in young, healthy adults engaged in moderate levels of physical activity¹⁸ (U.S. EPA, 2013, section 6.2.1.1, Figure 6–1). Exposures to an average O₃ concentration of 60 ppb results in group mean decrements in FEV₁ ranging from 1.8% to 3.6% (Adams, 2002; Adams, 2006;¹⁹ Schelegle et al., 2009;²⁰ Kim et

al., 2011). The weighted average group mean decrement was 2.7% from these studies. In some analyses, these group mean decrements in lung function were statistically significant (Brown et al., 2008; Kim et al., 2011), while in other analyses they were not (Adams, 2006; Schelegle et al., 2009).²¹ Prolonged exposure to an average O₃ concentration of 72 ppb results in a statistically significant group mean decrement in FEV₁ of about 6% (Schelegle et al., 2009).²² There is a smooth dose-response curve without evidence of a threshold for exposures between 40 and 120 ppb O₃ (U.S. EPA, 2013, Figure 6–1). When these data are taken together, the ISA concludes that “mean FEV₁ is clearly decreased by 6.6-hour exposures to 60 ppb O₃ and higher concentrations in [healthy, young adult] subjects performing moderate exercise” (U.S. EPA, 2013, p. 6–9).

As described in the proposal (79 FR 75250), the ISA focuses on individuals with >10% decrements in FEV₁ because (1) it is accepted by the American Thoracic Society (ATS) as an abnormal response and a reasonable criterion for assessing exercise-induced bronchoconstriction, and (2) some individuals in the Schelegle et al. (2009) study experienced 5–10% FEV₁ decrements following exposure to filtered air. The proportion of healthy adults experiencing FEV₁ decrements >10% following prolonged exposures to 80 ppb O₃ while at moderate exertion ranged from 17% to 29% and following exposures to 60 ppb O₃ ranged from 3% to 20%. The weighted average proportion (*i.e.*, based on numbers of subjects in each study) of young, healthy adults with >10% FEV₁ decrements is 25% following exposure to 80 ppb O₃ and 10% following exposure to 60 ppb O₃, for 6.6 hours at moderate exertion (U.S. EPA, 2013, page 6–18 and 6–19).²³ Responses within an

²¹ Adams (2006) did not find effects on FEV₁ at 60 ppb to be statistically significant. In an analysis of the Adams (2006) data, Brown et al. (2008) addressed the more fundamental question of whether there were statistically significant differences in responses before and after the 6.6 hour exposure period and found the average effect on FEV₁ at 60 ppb to be small, but highly statistically significant using several common statistical tests, even after removal of potential outliers. Schelegle et al. (2009) reported that, compared to filtered air, the largest change in FEV₁ for the 60 ppb protocol occurred after the sixth (and final) exercise period.

²² As noted above, for the 70 ppb exposure group, Schelegle et al. (2009) reported that the actual mean exposure concentration was 72 ppb.

²³ The ISA notes that by considering responses uncorrected for filtered air exposures, during which lung function typically improves (which would increase the size of the change, pre-and post-exposure), 10% is an underestimate of the proportion of healthy individuals that are likely to

individual tend to be reproducible over a period of several months, reflecting differences in intrinsic responsiveness. Given this, the ISA concludes that “[t]hrough group mean decrements are biologically small and generally do not attain statistical significance, a considerable fraction of exposed individuals [in the clinical studies] experience clinically meaningful decrements in lung function” when exposed for 6.6 hours to 60 ppb O₃ during quasi-continuous, moderate exertion (U.S. EPA, 2013, section 6.2.1.1, p. 6–20).

This review has marked an advance in the ability to make reliable quantitative predictions of the potential lung function response to O₃ exposure, and, thus, to reasonably predict the degree of interindividual response of lung function to that exposure. McDonnell et al. (2012) and Schelegle et al. (2012) developed models, described in more detail in the proposal (79 FR 75250), that included mathematical approaches to simulate the potential protective effect of antioxidants in the ELF at lower ambient O₃ concentrations, and that included a dose threshold below which changes in lung function do not occur. The resulting empirical models can estimate the frequency distribution of individual responses and summary measures of the distribution such as the mean or median response and the proportions of individuals with FEV₁ decrements >10%, 15%, and 20%.²⁴ The predictions of the models are consistent with the observed results from the individual controlled human exposure studies of O₃-induced FEV₁ decrements (79 FR 75250–51, see also U.S. EPA, 2013, Figures 6–1 and 6–3). CASAC agreed that these models mark a significant technical advance over the exposure-response modeling approach used for the lung function risk assessment in the last review and explicitly found that “[t]he MSS model to be scientifically and biologically defensible” (Frey, 2014a, pp. 8, 2). CASAC also stated that “the comparison of the MSS model results to those obtained with the exposure-response model is of tremendous importance. Typically, the MSS model gives a result about a factor of three higher . . . for school-age children, which is expected because the MSS model includes

experience clinically meaningful changes in lung function following exposure for 6.6 hours to 60 ppb O₃ during quasi-continuous moderate exertion (U.S. EPA, 2012, section 6.2.1.1).

²⁴ One of these models, the McDonnell-Stewart-Smith (MSS) model (McDonnell et al. 2012) was used to estimate the occurrences of lung function decrements in the HREA.

¹⁷ CASAC concurred that these were “the kinds of identifiable effects on public health that are expected from the presence of ozone in the ambient air” (Frey 2014c, p. 3).

¹⁸ Table 6–1 of the ISA includes descriptions of the activity levels evaluated in controlled human exposure studies (U.S. EPA, 2013).

¹⁹ Adams (2006); (2002) both provide data for an additional group of 30 healthy subjects that were exposed via facemask to 60 ppb O₃ for 6.6 hours with moderate exercise. These subjects are described on page 133 of Adams (2006) and pages 747 and 761 of Adams (2002). The facemask exposure is not expected to affect the FEV₁ responses relative to a chamber exposure.

²⁰ For the 60 ppb target exposure concentration, Schelegle et al. (2009) reported that the actual mean exposure concentration was 63 ppb.

responses for a wider range of exposure protocols” (Frey, 2014a, pp. 8, 2).

Epidemiologic studies have consistently linked short-term increases in ambient O₃ concentrations with lung function decrements in diverse populations and lifestyles, including children attending summer camps, adults exercising or working outdoors, and groups with pre-existing respiratory diseases such as asthmatic children (U.S. EPA, 2013, section 6.2.1.2). Some of these studies reported O₃-associated lung function decrements accompanied by respiratory symptoms²⁵ in asthmatic children. In contrast, studies of children in the general population have reported similar O₃-associated lung function decrements but without accompanying respiratory symptoms (79 FR 75251; U.S. EPA, 2013, section 6.2.1.2). As noted in the PA (EPA, 2014c, pp. 4–70 to 4–71), additional research is needed to evaluate responses of people with asthma and healthy people in the 40 to 70 ppb range. Further epidemiologic studies and meta-analyses of the effects of O₃ exposure on children will help elucidate the concentration-response functions for lung function and respiratory symptom effects at lower O₃ concentrations.

Several epidemiologic panel studies²⁶ reported statistically significant associations with lung function decrements at relatively low ambient O₃ concentrations. For outdoor recreation or exercise, associations were reported in analyses restricted to 1-hour average O₃ concentrations less than 80 ppb, down to less than 50 ppb. Among outdoor workers, Brauer et al. (1996) found a robust association with daily 1-hour max O₃ concentrations less than 40 ppb. Ulmer et al. (1997) found a robust association in schoolchildren with 30-minute maximum O₃ concentrations less than 60 ppb. For 8-hour average O₃ concentrations, associations with lung function decrements in children with asthma were found to persist at concentrations less than 80 ppb in a U.S. multicity study (Mortimer et al., 2002) and less than 51 ppb in a study conducted in the Netherlands (Gielen et al., 1997).

As described in the proposal (79 FR 75251), several epidemiologic panel studies provided information on potential confounding by copollutants and most O₃ effect estimates for lung function were robust to adjustment for temperature, humidity, and copollutants

such as particulate matter with mass median aerodynamic diameter less than or equal to 2.5 micrometers (PM_{2.5}), particulate matter with mass median aerodynamic diameter less than or equal to 10 micrometers (PM₁₀), NO₂, or sulfur dioxide (SO₂) (Hoppe et al., 2003; Brunekreef et al., 1994; Hoek et al. 1993; U.S. EPA, 2013, pp. 6–67 to 6–69). Although examined in only a few epidemiologic studies, O₃ also remained associated with decreases in lung function with adjustment for pollen or acid aerosols (79 F 75251; U.S. EPA, 2013, section 6.2.1.2).

Pulmonary Inflammation, Injury and Oxidative Stress

As described in detail in section II.B.2.a.ii of the proposal (79 FR 75252), O₃ exposures can result in increased respiratory tract inflammation and epithelial permeability. Inflammation is a host response to injury, and the induction of inflammation is evidence that injury has occurred. Oxidative stress has been shown to play a key role in initiating and sustaining O₃-induced inflammation. As noted in the ISA (U.S. EPA, 2013, section 6.2.3), O₃ exposures can initiate an acute inflammatory response throughout the respiratory tract that has been reported to persist for at least 18–24 hours after exposure.

Inflammation induced by exposure of humans to O₃ can have several potential outcomes, ranging from resolving entirely following a single exposure to becoming a chronic inflammatory state, as described in detail in section II.B.2.a.ii of the proposal (79 FR 75252) and in the ISA (U.S. EPA, 2013, section 6.2.3). Continued cellular damage due to chronic inflammation “may alter the structure and function of pulmonary tissues” (U.S. EPA, 2013, p. 6–161). Lung injury and the resulting inflammation provide a mechanism by which O₃ may cause other more serious morbidity effects (e.g., asthma exacerbations) (U.S. EPA, 2013, section 6.2.3).²⁷

Building on the last review, recent studies continue to support the evidence for airway inflammation and injury with new evidence for such effects following exposures to lower concentrations than had been evaluated previously. These studies include recent controlled human exposure and epidemiologic studies and are discussed more below.

²⁷ CASAC also addressed this issue: “The CASAC believes that these modest changes in FEV₁ are usually associated with inflammatory changes, such as more neutrophils in the bronchoalveolar lavage fluid. Such changes may be linked to the pathogenesis of chronic lung disease” (Frey, 2014a p. 2).

An extensive body of evidence from controlled human exposure studies, described in section II.B.2.a.ii of the proposal, indicates that short-term exposures to O₃ can cause pulmonary inflammation and increases in polymorphonuclear leukocyte (PMN) influx and permeability following 80–600 O₃ ppb exposures, eosinophilic inflammation following exposures at or above 160 ppb, and O₃-induced PMN influx following exposures of healthy adults to 60 ppb O₃, the lowest concentration that has been evaluated for inflammation. A meta-analysis of 21 controlled human exposure studies (Mudway and Kelly, 2004) using varied experimental protocols (80–600 ppb O₃ exposures; 1–6.6 hours exposure duration; light to heavy exercise; bronchoscopy at 0–24 hours post-O₃ exposure) reported that PMN influx in healthy subjects is linearly associated with total O₃ dose.

As with FEV₁ responses to O₃, inflammatory responses to O₃ are generally reproducible within individuals, with some individuals experiencing more severe O₃-induced airway inflammation than indicated by group averages. Unlike O₃-induced decrements in lung function, which are attenuated following repeated exposures over several days, some markers of O₃-induced inflammation and tissue damage remain elevated during repeated exposures, indicating ongoing damage to the respiratory system (79 FR 75252). Most controlled human exposure studies have reported that asthmatics experience larger O₃-induced inflammatory responses than non-asthmatics.²⁸

In the previous review (U.S. EPA, 2006a), the epidemiologic evidence of O₃-associated changes in airway inflammation and oxidative stress was limited (79 FR 75253). Since then, as a result of the development of less invasive test methods, there has been a large increase in the number of studies assessing ambient O₃-associated changes in airway inflammation and oxidative stress, the types of biological samples collected, and the types of indicators. Most of these recent studies have evaluated biomarkers of inflammation or oxidative stress in exhaled breath, nasal lavage fluid, or induced sputum (U.S. EPA, 2013, section 6.2.3.2). These recent studies form a larger database to establish coherence with findings from controlled human exposure and animal

²⁸ When evaluated, these studies have also reported O₃-induced respiratory symptoms in asthmatics. Specifically, Scannell et al. (1996), Basha et al. (1994), and Vagaggini et al. (2001, 2007) reported increased symptoms in addition to inflammation.

studies that have measured the same or related biological markers. Additionally, results from these studies provide further biological plausibility for the associations observed between ambient O₃ concentrations and respiratory symptoms and asthma exacerbations.

Airway Hyperresponsiveness (AHR)

A strong body of controlled human exposure and animal toxicological studies, most of which were available in the last review of the O₃ NAAQS, report O₃-induced AHR after either acute or repeated exposures (U.S. EPA, 2013, section 6.2.2.2). People with asthma often exhibit increased airway responsiveness at baseline relative to healthy control subjects, and asthmatics can experience further increases in responsiveness following exposures to O₃. Studies reporting increased airway responsiveness after O₃ exposure contribute to a plausible link between ambient O₃ exposures and increased respiratory symptoms in asthmatics, and increased hospital admissions and emergency department visits for asthma (section II.B.2.a.iii, 79 FR 75254; U.S. EPA, 2013, section 6.2.2.2).

Respiratory Symptoms and Medication Use

Respiratory symptoms are associated with adverse outcomes such as limitations in activity, and are the primary reason for people with asthma to use quick relief medication and to seek medical care. Studies evaluating the link between O₃ exposures and such symptoms allow a direct characterization of the clinical and public health significance of ambient O₃ exposure. Controlled human exposure and toxicological studies have described modes of action through which short-term O₃ exposures may increase respiratory symptoms by demonstrating O₃-induced AHR (U.S. EPA, 2013, section 6.2.2) and pulmonary inflammation (U.S. EPA, 2013, section 6.2.3).

The link between subjective respiratory symptoms and O₃ exposures has been evaluated in both controlled human exposure and epidemiologic studies, and the link with medication use has been evaluated in epidemiologic studies. In the last review, several controlled human exposure studies reported respiratory symptoms following exposures to O₃ concentrations at or above 80 ppb. In addition, one study reported such symptoms following exposures to 60 ppb O₃, though the increase was not statistically different from filtered air controls. Epidemiologic studies reported associations between ambient O₃ and

respiratory symptoms and medication use in a variety of locations and populations, including asthmatic children living in U.S. cities (U.S. EPA, 2013, pp. 6–1 to 6–2). In the current review, additional controlled human exposure studies have evaluated respiratory symptoms following exposures to O₃ concentrations below 80 ppb and recent epidemiologic studies have evaluated associations with respiratory symptoms and medication use (U.S. EPA, 2013, sections 6.2.1, 6.2.4).

As noted in section II.B.2.a.iv in the proposal (79 FR 75255), the findings for O₃-induced respiratory symptoms in controlled human exposure studies, and the evidence integrated across disciplines describing underlying modes of action, provide biological plausibility for epidemiologic associations observed between short-term increases in ambient O₃ concentration and increases in respiratory symptoms (U.S. EPA, 2013, section 6.2.4).

Most epidemiologic studies of O₃ and respiratory symptoms and medication use have been conducted in children and/or adults with asthma, with fewer studies, and less consistent results, in non-asthmatic populations (U.S. EPA, 2013, section 6.2.4). The 2006 AQCD (U.S. EPA, 2006a; U.S. EPA, 2013, section 6.2.4) concluded that the collective body of epidemiologic evidence indicated that short-term increases in ambient O₃ concentrations are associated with increases in respiratory symptoms in children with asthma. A large body of single-city and single-region studies of asthmatic children provides consistent evidence for associations between short-term increases in ambient O₃ concentrations and increased respiratory symptoms and asthma medication use in children with asthma (U.S. EPA, 2013, Figure 6–12, Table 6–20, section 6.2.4.1). Methodological differences, described in section II.B.2.a.iv of the proposal, among studies make comparisons across recent multicity studies of respiratory symptoms difficult.

Available evidence indicates that O₃-associated increases in respiratory symptoms are not confounded by temperature, pollen, or copollutants (primarily PM) (U.S. EPA, 2013, section 6.2.4.5; Table 6–25). However, identifying the independent effects of O₃ in some studies was complicated due to the high correlations observed between O₃ and PM or different lags and averaging times examined for copollutants. Nonetheless, the ISA noted that the robustness of associations in some studies of individuals with

asthma, combined with findings from controlled human exposure studies for the direct effects of O₃ exposure, provide substantial evidence supporting the independent effects of short-term ambient O₃ exposure on respiratory symptoms (U.S. EPA, 2013, section 6.2.4.5).

In summary, both controlled human exposure and epidemiologic studies have reported respiratory symptoms attributable to short-term O₃ exposures. In the last review, the majority of the evidence from controlled human exposure studies in young, healthy adults was for symptoms following exposures to O₃ concentrations at or above 80 ppb. Although studies that have become available since the last review have not reported increased respiratory symptoms in young, healthy adults following exposures with moderate exertion to 60 ppb, one recent study did report increased symptoms following exposure to 72 ppb O₃. As was concluded in the last review, the collective body of epidemiologic evidence indicates that short-term increases in ambient O₃ concentration are associated with increases in respiratory symptoms in children with asthma (U.S. EPA, 2013, section 6.2.4). Recent studies of respiratory symptoms and medication use, primarily in asthmatic children, add to this evidence. In a smaller body of studies, increases in ambient O₃ concentration were associated with increases in respiratory symptoms in adults with asthma.

Lung Host Defense

The mammalian respiratory tract has a number of closely integrated defense mechanisms that, when functioning normally, provide protection from the potential health effects of exposures to a wide variety of inhaled particles and microbes. Based on toxicological and human exposure studies, in the last review EPA concluded that available evidence indicates that short-term O₃ exposures have the potential to impair host defenses in humans, primarily by interfering with alveolar macrophage function. Any impairment in alveolar macrophage function may lead to decreased clearance of microorganisms or nonviable particles. Compromised alveolar macrophage functions in asthmatics may increase their susceptibility to other O₃ effects, the effects of particles, and respiratory infections (U.S. EPA, 2006a).

Relatively few studies conducted since the last review have evaluated the effects of O₃ exposures on lung host defense. As presented in section II.B.2.a.v of the proposal (79 FR 75256),

when the available evidence is taken as a whole, the ISA concludes that acute O₃ exposures impair the host defense capability of animals, primarily by depressing alveolar macrophage function and perhaps also by decreasing mucociliary clearance of inhaled particles and microorganisms. Coupled with limited evidence from controlled human exposure studies, this suggests that humans exposed to O₃ could be predisposed to bacterial infections in the lower respiratory tract.

Allergic and Asthma Related Responses

Evidence from controlled human exposure and epidemiologic studies available in the last review indicates that O₃ exposure skews immune responses toward an allergic phenotype and could also make airborne allergens more allergenic, as discussed in more detail in the proposal (79 FR 75257). Evidence from controlled human exposure and animal toxicology studies available in the last review indicates that O₃ may also increase AHR to specific allergen triggers (75 FR 2970, January 19, 2010). When combined with NO₂, O₃ has been shown to enhance nitration of common protein allergens, which may increase their allergenicity (Franze et al., 2005).

Hospital Admissions and Emergency Department Visits

The 2006 AQCD concluded that “the overall evidence supports a causal relationship between acute ambient O₃ exposures and increased respiratory morbidity resulting in increased emergency department visits and [hospital admissions] during the warm season”²⁹ (U.S. EPA, 2006a). This conclusion was “strongly supported by the human clinical, animal toxicologic[al], and epidemiologic evidence for [O₃-induced] lung function decrements, increased respiratory symptoms, airway inflammation, and airway hyperreactivity” (U.S. EPA, 2006a).

The results of recent studies largely support the conclusions of the 2006 AQCD (U.S. EPA, 2013, section 6.2.7). Since the completion of the 2006 AQCD, relatively fewer studies, conducted in the U.S., Canada, and Europe, have evaluated associations between short-term O₃ concentrations and respiratory hospital admissions and emergency department visits, with a growing

²⁹ Epidemiologic associations for O₃ are more robust during the warm season than during cooler months (e.g., smaller measurement error, less potential confounding by copollutants). The rationale for focusing on warm season epidemiologic studies for O₃ can be found at 72 FR 37838–37840.

number of studies conducted in Asia. This epidemiologic evidence is discussed in detail in the proposal (79 FR 75258) and in the ISA (U.S. EPA, 2013, section 6.2.7).³⁰

In considering this body of evidence, the ISA focused primarily on multicity studies because they examine associations with respiratory-related hospital admissions and emergency department visits over large geographic areas using consistent statistical methodologies (U.S. EPA, 2013, section 6.2.7.1). The ISA also focused on single-city studies that encompassed a large number of daily hospital admissions or emergency department visits, included long study-durations, were conducted in locations not represented by the larger studies, or examined population-specific characteristics that may impact the risk of O₃-related health effects but were not evaluated in the larger studies (U.S. EPA, 2013, section 6.2.7.1). When examining the association between short-term O₃ exposure and respiratory health effects that require medical attention, the ISA distinguishes between hospital admissions and emergency department visits because it is likely that a small percentage of respiratory emergency department visits will be admitted to the hospital; therefore, respiratory emergency department visits may represent potentially less serious, but more common outcomes (U.S. EPA, 2013, section 6.2.7.1).

The collective evidence across studies indicates a mostly consistent positive association between O₃ exposure and respiratory-related hospital admissions and emergency department visits. Moreover, the magnitude of these associations may be underestimated to the extent members of study populations modify their behavior in response to air quality forecasts, and to the extent such behavior modification increases exposure misclassification (U.S. EPA, 2013, Section 4.6.6). Studies examining the potential confounding effects of copollutants have reported that O₃ effect estimates remained relatively robust upon the inclusion of PM and gaseous pollutants in two-pollutant models (U.S. EPA, 2013, Figure 6–20, Table 6–29). Additional studies that conducted copollutant analyses, but did not present quantitative results, also support these conclusions (Strickland et al., 2010; Tolbert et al., 2007; Medina-Ramon et

³⁰ The consideration of ambient O₃ concentrations in the locations of these epidemiologic studies are discussed in sections II.D.1.b and II.E.4.a below, for the current standard and for alternative standards, respectively.

al., 2006; U.S. EPA, 2013, section 6.2.7.5).³¹

In the last review, studies had not evaluated the concentration-response relationship between short-term O₃ exposure and respiratory-related hospital admissions and emergency department visits. As described in the proposal in section II.B.2.a.vii (79 FR 75257) and in the ISA (U.S. EPA, 2013, section 6.2.7.2), a preliminary examination of this relationship in studies that have become available since the last review found no evidence of a deviation from linearity when examining the association between short-term O₃ exposure and asthma hospital admissions (Silverman and Ito, 2010; Strickland et al., 2010). In addition, an examination of the concentration-response relationship for O₃ exposure and pediatric asthma emergency department visits found no evidence of a threshold at O₃ concentrations as low as 30 ppb (for daily maximum 8-hour concentrations) (U.S. EPA, 2013, section 6.2.7.3). However, in these studies there is uncertainty in the shape of the concentration-response curve at the lower end of the distribution of O₃ concentrations due to the low density of data in this range. Further studies at low-level O₃ exposures might reduce this uncertainty.

Respiratory Mortality

Evidence from experimental studies indicates multiple potential pathways of respiratory effects from short-term O₃ exposures, which support the continuum of respiratory effects that could potentially result in respiratory-related mortality in adults (U.S. EPA, 2013, section 6.2.8).³² The evidence in the last review was inconsistent for associations between short-term O₃ concentrations and respiratory mortality (U.S. EPA, 2006a). New epidemiologic evidence for respiratory mortality is discussed in detail in the ISA (U.S. EPA, 2013, section 6.6) and summarized below. The majority of recent multicity studies have reported positive associations between short-term O₃ exposures and respiratory mortality, particularly during the summer months (U.S. EPA, 2013, Figure 6–36).

³¹ The ISA concluded that, “[o]verall, recent studies provide copollutant results that are consistent with those from the studies evaluated in the 2006 O₃ AQCD [(U.S. EPA, 2006[a]), Figure 7–12, page 7–80 of the 2006 O₃ AQCD], which found that O₃ respiratory hospital admissions risk estimates remained robust to the inclusion of PM in copollutant models (U.S. EPA, 2013, pp. 6–152 to 6–153).

³² Premature mortality is discussed in more detail below in section II.A.1.b.iv.

Recent multicity studies from the U.S. (Zanobetti and Schwartz, 2008), Europe (Samoli et al., 2009), Italy (Stafoggia et al., 2010), and Asia (Wong et al., 2010), as well as a multi-continent study (Katsouyanni et al., 2009), reported associations between short-term O₃ concentrations and respiratory mortality (U.S. EPA, 2013, Figure 6–37, page 6–259). With respect to respiratory mortality, summer-only analyses were consistently positive and most were statistically significant. All-year analyses had more mixed results, but most were positive.

Of the studies evaluated, only two studies analyzed the potential for copollutant confounding of the O₃-respiratory mortality relationship (Katsouyanni et al., (2009); Stafoggia et al., (2010)). Based on the results of these analyses, the O₃ respiratory mortality risk estimates appear to be moderately to substantially sensitive (e.g., increased or attenuated) to inclusion of PM₁₀. However, in the APHENA study (Katsouyanni et al., 2009), the mostly every-6th-day sampling schedule for PM₁₀ in the Canadian and U.S. datasets greatly reduced their sample size and limits the interpretation of these results (U.S. EPA, 2013, sections 6.2.8 and 6.2.9).

The evidence for associations between short-term O₃ concentrations and respiratory mortality has been strengthened since the last review, with the addition of several large multicity studies. The biological plausibility of the associations reported in these studies is supported by the experimental evidence for respiratory effects.

ii. Respiratory Effects—Long-Term Exposure

Since the last review, the body of evidence indicating the occurrence of respiratory effects due to long-term O₃ exposure has been strengthened. This evidence is discussed in detail in the ISA (U.S. EPA, 2013, Chapter 7) and summarized below for new-onset asthma and asthma prevalence, asthma hospital admissions, pulmonary structure and function, and respiratory mortality.

Asthma is a heterogeneous disease with a high degree of temporal variability. The onset, progression, and symptoms can vary within an individual's lifetime, and the course of asthma may vary markedly in young children, older children, adolescents, and adults. In the previous review, longitudinal cohort studies that examined associations between long-term O₃ exposures and the onset of asthma in adults and children indicated

a direct effect of long-term O₃ exposures on asthma risk in adults and effect modification by O₃ in children. Since then, additional studies have evaluated associations with new onset asthma, further informing our understanding of the potential gene-environment interactions, mechanisms, and biological pathways associated with incident asthma.

In children, the relationship between long-term O₃ exposure and new-onset asthma has been extensively studied in the Children's Health Study (CHS), a long-term study that was initiated in the early 1990's which has evaluated effects in several cohorts of children. For this review, recent studies from the CHS provide evidence for gene-environment interactions in effects on new-onset asthma by indicating that the lower risks associated with specific genetic variants are found in children who live in lower O₃ communities. Described in detail in the proposal (79 FR 75259) and in the ISA (U.S. EPA, 2013, section 7.2.1), these studies indicate that the risk for new-onset asthma is related in part to genetic susceptibility, as well as behavioral factors and environmental exposure. Cross-sectional studies by Akinbami et al. (2010) and Hwang et al. (2005) provide further evidence relating O₃ exposures with asthma prevalence. Gene-environment interactions are discussed in detail in Section 5.4.2.1 in the ISA (U.S. EPA, 2013).

In the 2006 AQCD (U.S. EPA, 2006a), studies on O₃-related hospital discharges and emergency department visits for asthma and respiratory disease mainly looked at short-term (daily) metrics. Recent studies continue to indicate that there is evidence for increases in both hospital admissions and emergency department visits in children and adults related to all respiratory outcomes, including asthma, with stronger associations in the warm months.

In the 2006 AQCD (U.S. EPA, 2006a), few epidemiologic studies had investigated the effect of chronic O₃ exposure on pulmonary function. As discussed in the proposal, epidemiologic studies of long-term exposures in both children and adults provide mixed results about the effects of long-term O₃ exposure on pulmonary function and the growth rate of lung function.

Long-term studies in animals allow for greater insight into the potential effects of prolonged exposure to O₃ that may not be easily measured in humans, such as structural changes in the respiratory tract. Despite uncertainties, epidemiologic studies observing associations of O₃ exposure with

functional changes in humans can attain biological plausibility in conjunction with long-term toxicological studies, particularly O₃-inhalation studies performed in non-human primates whose respiratory systems most closely resemble that of the human. An important series of studies, discussed in section 7.2.3.2 of the ISA (U.S. EPA, 2013), have used nonhuman primates to examine the effect of O₃ alone, or in combination with an inhaled allergen, house dust mite antigen, on morphology and lung function. Animals exhibit the hallmarks of allergic asthma defined for humans (NHLBI, 2007). These studies and others have demonstrated changes in pulmonary function and airway morphology in adult and infant nonhuman primates repeatedly exposed to environmentally relevant concentrations of O₃ (U.S. EPA, 2013, section 7.2.3.2). As discussed in more detail in the proposal, the studies provide evidence of an O₃-induced change in airway resistance and responsiveness and provide biological plausibility of long-term exposure, or repeated short-term exposures, to O₃ contributing to the effects of asthma in children.

Collectively, evidence from animal studies strongly suggests that chronic O₃ exposure is capable of damaging the distal airways and proximal alveoli, resulting in lung tissue remodeling and leading to apparent irreversible changes. Potentially, persistent inflammation and interstitial remodeling play an important role in the progression and development of chronic lung disease. Further discussion of the modes of action that lead to O₃-induced morphological changes and the mechanisms involved in lifestage susceptibility and developmental effects can be found in the ISA (U.S. EPA, 2013, section 5.3.7, section 5.4.2.4). The findings reported in chronic animal studies offer insight into potential biological mechanisms for the suggested association between seasonal O₃ exposure and reduced lung function development in children as observed in epidemiologic studies (U.S. EPA, 2013, section 7.2.3.1). Further research could help fill in the gaps in our understanding of the mechanisms involved in lifestage susceptibility and developmental effects in children of seasonal or long-term exposure to O₃.

A limited number of epidemiologic studies have assessed the relationship between long-term exposure to O₃ and mortality in adults. The 2006 AQCD concluded that an insufficient amount of evidence existed "to suggest a causal relationship between chronic O₃ exposure and increased risk for

mortality in humans” (U.S. EPA, 2006a). Though total and cardio-pulmonary mortality were considered in these studies, respiratory mortality was not specifically considered.

In a recent follow-up analysis of the American Cancer Society cohort (Jerrett et al., 2009), cardiopulmonary deaths were separately subdivided into respiratory and cardiovascular deaths, rather than combined as in the Pope et al. (2002) work. Increased O₃ exposure was associated with the risk of death from respiratory causes, and this effect was robust to the inclusion of PM_{2.5}. Additionally, a recent multicity time series study (Zanobetti and Schwartz, 2011), which followed (from 1985 to 2006) four cohorts of Medicare enrollees with chronic conditions that might predispose to O₃-related effects, observed an association between long-term (warm season) exposure to O₃ and elevated risk of mortality in the cohort that had previously experienced an emergency hospital admission due to chronic obstructive pulmonary disease (COPD). A key limitation of this study is the inability to control for PM_{2.5}, because data were not available in these cities until 1999.

iii. Cardiovascular Effects—Short-Term Exposure

A relatively small number of studies have examined the potential effect of short-term O₃ exposure on the cardiovascular system. The 2006 AQCD (U.S. EPA, 2006a, p. 8–77) concluded that “O₃ directly and/or indirectly contributes to cardiovascular-related morbidity,” but added that the body of evidence was limited. This conclusion was based on a controlled human exposure study that included hypertensive adult males; a few epidemiologic studies of physiologic effects, heart rate variability, arrhythmias, myocardial infarctions, and hospital admissions; and toxicological studies of heart rate, heart rhythm, and blood pressure.

More recently, the body of scientific evidence available that has examined the effect of O₃ on the cardiovascular system has expanded. There is an emerging body of animal toxicological evidence demonstrating that short-term exposure to O₃ can lead to autonomic nervous system alterations (in heart rate and/or heart rate variability) and suggesting that proinflammatory signals may mediate cardiovascular effects. Interactions of O₃ with respiratory tract components result in secondary oxidation product formation and subsequent production of inflammatory mediators, which have the potential to penetrate the epithelial barrier and to

initiate toxic effects systemically. In addition, animal toxicological studies of long-term exposure to O₃ provide evidence of enhanced atherosclerosis and ischemia/reperfusion (I/R) injury, corresponding with development of a systemic oxidative, proinflammatory environment. Recent experimental and epidemiologic studies have investigated O₃-related cardiovascular events and are summarized in the ISA (U.S. EPA, 2013, section 6.3).

Controlled human exposure studies discussed in previous reviews have not demonstrated any consistent extrapulmonary effects. In this review, evidence from controlled human exposure studies suggests cardiovascular effects in response to short-term O₃ exposure (U.S. EPA, 2013, section 6.3.1) and provides some coherence with evidence from animal toxicology studies. Controlled human exposure studies also support the animal toxicological studies by demonstrating O₃-induced effects on blood biomarkers of systemic inflammation and oxidative stress, as well as changes in biomarkers that can indicate the potential for increased clotting following O₃ exposures. Increases and decreases in high frequency heart rate variability (HRV) have been reported. These changes in cardiac function observed in animal and human studies provide preliminary evidence for O₃-induced modulation of the autonomic nervous system through the activation of neural reflexes in the lung (U.S. EPA, 2013, section 5.3.2).

Overall, the ISA concludes that the available body of epidemiologic evidence examining the relationship between short-term exposures to O₃ concentrations and cardiovascular morbidity is inconsistent (U.S. EPA, 2013, section 6.3.2.9).

Despite the inconsistent evidence for an association between O₃ concentration and cardiovascular disease (CVD) morbidity, mortality studies indicate a consistent positive association between short-term O₃ exposure and cardiovascular mortality in multicity studies and in a multi-continent study. When examining mortality due to CVD, epidemiologic studies consistently observe positive associations with short-term exposure to O₃. Additionally, there is some evidence for an association between long-term exposure to O₃ and mortality, although the association between long-term ambient O₃ concentrations and cardiovascular mortality can be confounded by other pollutants (U.S. EPA, 2013). The ISA (U.S. EPA, 2013, section 6.3.4) states that taken together, the overall body of evidence across the animal and human

studies is sufficient to conclude that there is likely to be a causal relationship between relevant short-term exposures to O₃ and cardiovascular system effects.

iv. Premature Mortality—Short-Term Exposure

The 2006 AQCD concluded that the overall body of evidence was highly suggestive that short-term exposure to O₃ directly or indirectly contributes to nonaccidental and cardiopulmonary-related mortality in adults, but additional research was needed to more fully establish underlying mechanisms by which such effects occur (U.S. EPA, 2006a; U.S. EPA, 2013, p. 2–18). In building on the evidence for mortality from the last review, the ISA states (U.S. EPA, 2013, p. 6–261):

The evaluation of new multicity studies that examined the association between short-term O₃ exposures and mortality found evidence that supports the conclusions of the 2006 AQCD. These new studies reported consistent positive associations between short-term O₃ exposure and all-cause (nonaccidental) mortality, with associations persisting or increasing in magnitude during the warm season, and provide additional support for associations between O₃ exposure and cardiovascular and respiratory mortality.

The 2006 AQCD reviewed a large number of time-series studies of associations between short-term O₃ exposures and total mortality including single- and multicity studies, and meta-analyses. Available studies reported some evidence for heterogeneity in O₃ mortality risk estimates across cities and across studies. Studies that conducted seasonal analyses reported larger O₃ mortality risk estimates during the warm or summer season. Overall, the 2006 AQCD identified robust associations between various measures of daily ambient O₃ concentrations and all-cause mortality, which could not be readily explained by confounding due to time, weather, or copollutants. With regard to cause-specific mortality, consistent positive associations were reported between short-term O₃ exposure and cardiovascular mortality, with less consistent evidence for associations with respiratory mortality. The majority of the evidence for associations between O₃ and cause-specific mortality were from single-city studies, which had small daily mortality counts and subsequently limited statistical power to detect associations. The 2006 AQCD concluded that “the overall body of evidence is highly suggestive that O₃ directly or indirectly contributes to nonaccidental and cardiopulmonary-related mortality” (U.S. EPA, 2013, section 6.6.1).

Recent studies have strengthened the body of evidence that supports the association between short-term O₃ concentrations and mortality in adults. This evidence includes a number of studies reporting associations with nonaccidental as well as cause-specific mortality. Multi-continent and multicity studies have consistently reported positive and statistically significant associations between short-term O₃ concentrations and all-cause mortality, with evidence for larger mortality risk estimates during the warm or summer months (79 FR 75262; U.S. EPA, 2013 Figure 6–27; Table 6–42). Similarly, evaluations of cause-specific mortality have reported consistently positive associations with O₃, particularly in analyses restricted to the warm season (79 FR 75262; U.S. EPA, 2013 Fig. 6–37; Table 6–53).

In the previous review, multiple uncertainties remained regarding the relationship between short-term O₃ concentrations and mortality, including the extent of residual confounding by copollutants; characterization of the factors that modify the O₃-mortality association; the appropriate lag structure for identifying O₃-mortality effects; and the shape of the O₃-mortality concentration-response function and whether a threshold exists. Many of the studies, published since the last review, have attempted to address one or more of these uncertainties and are described in more detail in the proposal (79 FR 75262 and in the ISA (U.S. EPA, 2013, section 6.6.2).

In particular, recent studies have evaluated different statistical approaches to examine the shape of the O₃-mortality concentration-response relationship and to evaluate whether a threshold exists for O₃-related mortality. These studies are detailed in the proposal (79 FR 75262) and in the ISA (U.S. EPA, 2013, p. 2–32). The ISA reaches the following overall conclusions that the epidemiologic studies identified in the ISA indicated a generally linear C–R function with no indication of a threshold but that there is a lack of data at lower O₃ concentrations and therefore, less certainty in the shape of the C–R curve at the lower end of the distribution (U.S. EPA, 2013, p. 2–32).

c. Adversity of Effects

In making judgments as to when various O₃-related effects become regarded as adverse to the health of individuals, in previous NAAQS reviews, the EPA has relied upon the guidelines published by the ATS and the advice of CASAC. In 2000, the ATS published an official statement on

“What Constitutes an Adverse Health Effect of Air Pollution?” (ATS, 2000a), which updated and built upon its earlier guidance (ATS, 1985). The earlier guidance defined adverse respiratory health effects as “medically significant physiologic changes generally evidenced by one or more of the following: (1) Interference with the normal activity of the affected person or persons, (2) episodic respiratory illness, (3) incapacitating illness, (4) permanent respiratory injury, and/or (5) progressive respiratory dysfunction,” while recognizing that perceptions of “medical significance” and “normal activity” may differ among physicians, lung physiologists and experimental subjects (ATS, 1985). The more recent guidance concludes that transient, reversible loss of lung function in combination with respiratory symptoms should be considered adverse.³³ However, the committee also recommended “that a small, transient loss of lung function, by itself, should not automatically be designated as adverse” (ATS, 2000a, p. 670).

There is also a more specific consideration of population risk in the 2000 guidance. Specifically, the committee considered that a shift in the risk factor distribution, and hence the risk profile of the exposed population, should be considered adverse, even in the absence of the immediate occurrence of frank illness (ATS, 2000a, p. 668). For example, a population of asthmatics could have a distribution of lung function such that no individual has a level associated with clinically important impairment. Exposure to air pollution could shift the distribution to lower levels of lung function that still do not bring any individual to a level that is associated with clinically relevant effects. However, this would be considered to be adverse because individuals within the population would already have diminished reserve function, and therefore would be at increased risk to further environmental insult (ATS, 2000a, p. 668).

The ATS also concluded in its guidance that elevations of biomarkers such as cell numbers and types, cytokines, and reactive oxygen species may signal risk for ongoing injury and more serious effects or may simply represent transient responses, illustrating the lack of clear boundaries that separate adverse from nonadverse events. More subtle health outcomes also may be connected mechanistically

³³ “In drawing the distinction between adverse and nonadverse reversible effects, this committee recommended that reversible loss of lung function in combination with the presence of symptoms should be considered as adverse” (ATS, 2000a).

to health effects that are clearly adverse, so that small changes in physiological measures may not appear clearly adverse when considered alone, but may be part of a coherent and biologically plausible chain of related health outcomes that include responses that are clearly adverse, such as mortality (U.S. EPA, 2014c, section 3.1.2.1).

Application of the ATS guidelines to the least serious category of effects³⁴ related to ambient O₃ exposures, which are also the most numerous and, therefore, are also important from a public health perspective, involves judgments about which medical experts on CASAC panels and public commenters have in the past expressed diverse views. To help frame such judgments, in past reviews, the EPA has defined gradations of individual functional responses (*e.g.*, decrements in FEV₁ and airway responsiveness) and symptomatic responses (*e.g.*, cough, chest pain, wheeze), together with judgments as to the potential impact on individuals experiencing varying degrees of severity of these responses. These gradations were used by the EPA in the 1997 O₃ NAAQS review and slightly revised in the 2008 review (U.S. EPA, 1996b, p. 59; U.S. EPA, 2007, p. 3–72; 72 FR 37849, July 11, 2007). These gradations and impacts are summarized in Tables 3–2 and 3–3 in the 2007 O₃ Staff Paper (U.S. EPA, 2007, pp. 3–74 to 3–75).

For the purpose of estimating potentially adverse lung function decrements in active healthy people, the CASAC panel in the 2008 O₃ NAAQS review indicated that a focus on the mid to upper end of the range of moderate levels of functional responses is most appropriate (*e.g.*, FEV₁ decrements ≥15% but <20%) (Henderson, 2006; U.S. EPA, 2007, p. 3–76). In this review, CASAC reiterated that the “[e]stimation of FEV₁ decrements of ≥15% is appropriate as a scientifically relevant surrogate for adverse health outcomes in active healthy adults” (Frey, 2014c, p. 3).

For the purpose of estimating potentially adverse lung function decrements in people with lung disease, the CASAC panel in the 2008 O₃ NAAQS review indicated that a focus on the lower end of the range of moderate levels of functional responses is most appropriate (*e.g.*, FEV₁ decrements ≥10%) (Henderson, 2006; U.S. EPA, 2007, p. 3–76). In their letter

³⁴ These include, for example, the transient and reversible effects demonstrated in controlled human exposure studies, such as lung function decrements or respiratory symptoms.

advising the Administrator on the reconsideration of the 2008 final decision, CASAC stated that “[a] 10% decrement in FEV₁ can lead to respiratory symptoms, especially in individuals with pre-existing pulmonary or cardiac disease. For example, people with chronic obstructive pulmonary disease have decreased ventilatory reserve (*i.e.*, decreased baseline FEV₁) such that a ≥ 10% decrement could lead to moderate to severe respiratory symptoms” (Samet, 2011). In this review, CASAC provided similar advice, stating that “[a]n FEV₁ decrement of ≥ 10% is a scientifically relevant surrogate for adverse health outcomes for people with asthma and lung disease”, and that such decrements “could be adverse for people with lung disease” (Frey, 2014c, pp. 3, 7).

In judging the extent to which these impacts represent effects that should be regarded as adverse to the health status of individuals, in previous NAAQS reviews, the EPA has also considered whether effects were experienced repeatedly during the course of a year or only on a single occasion (U.S. EPA, 2007). While some experts would judge single occurrences of moderate responses to be a “nuisance,” especially for healthy individuals, a more general consensus view of the adversity of such moderate responses emerges as the frequency of occurrence increases. In particular, not every estimated occurrence of an O₃-induced FEV₁ decrement will be adverse.³⁵ However, repeated occurrences of moderate responses, even in otherwise healthy individuals, may be considered to be adverse since they could set the stage for more serious illness (61 FR 65723). The CASAC panel in the 1997 NAAQS review expressed a consensus view that these “criteria for the determination of an adverse physiological response were reasonable” (Wolff, 1995). In the review completed in 2008, as in the current review (II.B, II.C below), estimates of repeated occurrences continued to be an important public health policy factor in judging the adversity of moderate lung function decrements in healthy and asthmatic people (72 FR 37850, July 11, 2007).

d. Ozone-Related Impacts on Public Health

The currently available evidence expands the understanding of populations that were identified to be at greater risk of O₃-related health effects

at the time of the last review (*i.e.*, people who are active outdoors, people with lung disease, children and older adults and people with increased responsiveness to O₃) and supports the identification of additional factors that may lead to increased risk (U.S. EPA, 2006a, section 6.3; U.S. EPA, 2013, Chapter 8). Populations and lifestages may be at greater risk for O₃-related health effects due to factors that contribute to their susceptibility and/or vulnerability to O₃. The definitions of susceptibility and vulnerability have been found to vary across studies, but in most instances “susceptibility” refers to biological or intrinsic factors (*e.g.*, lifestage, sex, preexisting disease/conditions) while “vulnerability” refers to non-biological or extrinsic factors (*e.g.*, socioeconomic status [SES]) (U.S. EPA, 2013, p. 8–1; U.S. EPA, 2010, 2009b). In some cases, the terms “at-risk” and “sensitive” have been used to encompass these concepts more generally. In the ISA, PA, and proposal, “at-risk” is the all-encompassing term used to define groups with specific factors that increase their risk of O₃-related health effects.

There are multiple avenues by which groups may experience increased risk for O₃-induced health effects. A population or lifestage³⁶ may exhibit greater effects than other populations or lifestages exposed to the same concentration or dose, or they may be at greater risk due to increased exposure to an air pollutant (*e.g.*, time spent outdoors). A group with intrinsically increased risk would have some factor(s) that increases risk through a biological mechanism and, in general, would have a steeper concentration-risk relationship, compared to those not in the group. Factors that are often considered intrinsic include pre-existing asthma, genetic background, and lifestage. A group of people could also have extrinsically increased risk, which would be through an external, non-biological factor, such as socioeconomic status (SES) and diet. Some groups are at risk of increased internal dose at a given exposure concentration, for example, because of breathing patterns. This category would include people who work or exercise outdoors. Finally, there are those who might be placed at increased risk for experiencing greater exposures by being exposed to higher O₃ concentrations. This would include, for example, groups of people with greater exposure

to ambient O₃ due to less availability or use of home air conditioners such that they are more likely to be in locations with open windows on high O₃ days. Some groups may be at increased risk of O₃-related health effects through a combination of factors. For example, children tend to spend more time outdoors when O₃ levels are high, and at higher levels of activity than adults, which leads to increased exposure and dose, and they also have biological, or intrinsic, risk factors (*e.g.*, their lungs are still developing) (U.S. EPA, 2013, Chapter 8). An at-risk population or lifestage is more likely to experience adverse health effects related to O₃ exposures and/or, develop more severe effects from exposure than the general population. The populations and lifestages identified by the ISA (U.S. EPA, 2013, section 8.5) identified that have “adequate” evidence for increased O₃-related health effects are people with certain genotypes, people with asthma, younger and older age groups, people with reduced intake of certain nutrients, and outdoor workers. These at-risk populations and lifestages are described in more detail in section II.B.4 of the proposal (79 FR 75264–269).

One consideration in the assessment of potential public health impacts is the size of various population groups for which there is adequate evidence of increased risk for health effects associated with O₃-related air pollution exposure (U.S. EPA, 2014c, section 3.1.5.2). The factors for which the ISA judged the evidence to be “adequate” with respect to contributing to increased risk of O₃-related effects among various populations and lifestages included: Asthma; childhood and older adulthood; diets lower in vitamins C and E; certain genetic variants; and working outdoors (U.S. EPA, 2013, section 8.5). No statistics are available to estimate the size of an at-risk population based on nutritional status or genetic variability.

With regard to asthma, Table 3–7 in the PA (U.S. EPA, 2014c, section 3.1.5.2) summarizes information on the prevalence of current asthma by age in the U.S. adult population in 2010 (Schiller et al. 2012; children—Bloom et al., 2011). Individuals with current asthma constitute a fairly large proportion of the population, including more than 25 million people. Asthma prevalence tends to be higher in children than adults. Within the U.S., approximately 8.2% of adults have reported currently having asthma (Schiller et al., 2012) and 9.5% of

³⁵ As noted above, the ATS recommended “that a small, transient loss of lung function, by itself, should not automatically be designated as adverse” (ATS, 2000a, p. 670).

³⁶ Lifestages, which in this case includes childhood and older adulthood, are experienced by most people over the course of a lifetime, unlike other factors associated with at-risk populations.

children have reported currently having asthma (Bloom et al., 2011).³⁷

With regard to lifestyles, based on U.S. census data from 2010 (Howden and Meyer, 2011), about 74 million people, or 24% of the U.S. population, are under 18 years of age and more than 40 million people, or about 13% of the U.S. population, are 65 years of age or older. Hence, a large proportion of the U.S. population (*i.e.*, more than a third) is included in age groups that are considered likely to be at increased risk for health effects from ambient O₃ exposure.

With regard to outdoor workers, in 2010, approximately 11.7% of the total number of people (143 million people) employed, or about 16.8 million people, worked outdoors one or more days per week (based on worker surveys).³⁸ Of these, approximately 7.4% of the workforce, or about 7.8 million people, worked outdoors three or more days per week.

While it is difficult to estimate the total number of people in groups that are at greater risk from exposure to O₃, due to the overlap in members of the different at-risk population groups, the proportion of the total population at greater risk is large. The size of the at-risk population combined with the estimates of risk of different health outcomes associated with exposure to O₃ can give an indication of the magnitude of O₃ impacts on public health.

2. Overview of Human Exposure and Health Risk Assessments

To put judgments about health effects into a broader public health context, the EPA has developed and applied models to estimate human exposures to O₃ and O₃-associated health risks. Exposure and risk estimates that are output from such models are presented and assessed in the HREA (U.S. EPA, 2014a). Section II.C of the proposal discusses the quantitative assessments of O₃ exposures and O₃-related health risks that are presented in the HREA (79 FR

75270). Summaries of these discussions are provided below for the approach used to adjust air quality for quantitative exposure and risk analyses in the HREA (II.A.2.a), the HREA assessment of exposures to ambient O₃ (II.A.2.b), and the HREA assessments of O₃-related health risks (II.A.2.c).

a. Air Quality Adjustment

As discussed in section II.C.1 of the proposal (79 FR 75270), the HREA uses a photochemical model to estimate sensitivities of O₃ to changes in precursor emissions in order to estimate ambient O₃ concentrations that would just meet the current and alternative standards (U.S. EPA, 2014a, Chapter 4).³⁹ For the 15 urban study areas evaluated in the HREA,⁴⁰ this model-based adjustment approach estimates hourly O₃ concentrations at each monitor location when modeled U.S. anthropogenic precursor emissions (*i.e.*, NO_x, VOC)⁴¹ are reduced. The HREA estimates air quality that just meets the current and alternative standards for the 2006–2008 and 2008–2010 periods.⁴²

As discussed in Chapter 4 of the HREA (U.S. EPA, 2014a), this approach to adjusting air quality models the physical and chemical atmospheric processes that influence ambient O₃ concentrations. Compared to the quadratic rollback approach used in previous reviews, it provides more realistic estimates of the spatial and temporal responses of O₃ to reductions in precursor emissions. Because ambient NO_x can contribute both to the formation and destruction of O₃ (U.S. EPA, 2014a, Chapter 4), the response of ambient O₃ concentrations to reductions in NO_x emissions is more variable than

indicated by the quadratic rollback approach. This improved approach to adjusting O₃ air quality is consistent with recommendations from the National Research Council of the National Academies (NRC, 2008). In addition, CASAC strongly supported the new approach as an improvement and endorsed the way it was utilized in the HREA, stating that “the quadratic rollback approach has been replaced by a scientifically more valid Higher-order Decoupled Direct Method (HDDM)” and that “[t]he replacement of the quadratic rollback procedure by the HDDM procedure is important and supported by the CASAC” (Frey, 2014a, pp. 1 and 3).

Within urban study areas, the model-based air quality adjustments show reductions in the O₃ levels at the upper ends of ambient concentrations and increases in the O₃ levels at the lower ends of those distributions (U.S. EPA, 2014a, section 4.3.3.2, Figures 4–9 and 4–10).⁴³ Seasonal means of daily O₃ concentrations generally exhibit only modest changes upon model adjustment, reflecting the seasonal balance between daily decreases in relatively higher concentrations and increases in relatively lower concentrations (U.S. EPA, 2014a, Figures 4–9 and 4–10). The resulting compression in the seasonal distributions of ambient O₃ concentrations is evident in all of the urban study areas evaluated, though the degree of compression varies considerably across areas (U.S. EPA, 2014a, Figures 4–9 and 4–10).

As discussed in the PA (U.S. EPA, 2014c, section 3.2.1), adjusted patterns of O₃ air quality have important implications for exposure and risk estimates in urban case study areas. Estimates influenced largely by the upper ends of the distribution of ambient concentrations (*i.e.*, exposures of concern and lung function risk estimates, as discussed in sections 3.2.2 and 3.2.3.1 of the PA) will decrease with model-adjustment to the current and alternative standards. In contrast, seasonal risk estimates influenced by the full distribution of ambient O₃ concentrations (*i.e.*, epidemiology-based risk estimates, as discussed in section 3.2.3.2 of the PA) either increase or decrease in response to air quality adjustment, depending on the balance between the daily decreases in high O₃

³⁷ As noted below (II.C.3.a.ii), asthmatics can experience larger O₃-induced respiratory effects than non-asthmatic, healthy adults. The responsiveness of asthmatics to O₃ exposures could depend on factors that have not been well-evaluated such as asthma severity, the effectiveness of asthma control, or the prevalence of medication use.

³⁸ The O*NET program is the nation's primary source of occupational information. Central to the project is the O*NET database, containing information on hundreds of standardized and occupation-specific descriptors. The database, which is available to the public at no cost, is continually updated by surveying a broad range of workers from each occupation. <http://www.onetcenter.org/overview.html>. http://www.onetonline.org/find/descriptor/browse/Work_Context/4.C.2/.

³⁹ The HREA uses the Community Multi-scale Air Quality (CMAQ) photochemical model instrumented with the higher order direct decoupled method (HDDM) to estimate O₃ concentrations that would occur with the achievement of the current and alternative O₃ standards (U.S. EPA, 2014a, Chapter 4).

⁴⁰ The urban study areas assessed are Atlanta, Baltimore, Boston, Chicago, Cleveland, Dallas, Denver, Detroit, Houston, Los Angeles, New York, Philadelphia, Sacramento, St. Louis, and Washington, DC.

⁴¹ Exposure and risk analyses for most of the urban study areas focus on reducing U.S. anthropogenic NO_x emissions alone. The exceptions are Chicago and Denver. Exposure and risk analyses for Chicago and Denver are based on reductions in emissions of both NO_x and VOC (U.S. EPA, 2014a, section 4.3.3.1; Appendix 4D).

⁴² These estimates thus reflect design values—8 hour values using the form of the NAAQS that meet the level of the current or alternative standards. These simulations are illustrative and do not reflect any consideration of specific control programs designed to achieve the reductions in emissions required to meet the specified standards. Further, these simulations do not represent predictions of when, whether, or how areas might meet the specified standards.

⁴³ It is important to note that sensitivity analyses in the HREA indicate that the increases in low O₃ concentrations are smaller when NO_x and VOC emissions are reduced than when only NO_x emissions are reduced (U.S. EPA, 2014a, Appendix 4–D, section 4.7).

concentrations and increases in low O₃ concentrations.⁴⁴

To evaluate uncertainties in air quality adjustments, the HREA assessed the extent to which the modeled O₃ response to reductions in NO_x emissions appropriately represent the trends observed in monitored ambient O₃ following actual reductions in NO_x emissions, and the extent to which the O₃ response to reductions in precursor emissions could differ with emissions reduction strategies that are different from those used in HREA to generate risk estimates.

To evaluate the first issue, the HREA conducted a national analysis evaluating trends in monitored ambient O₃ concentrations during a time period when the U.S. experienced large-scale reductions in NO_x emissions (*i.e.*, 2001 to 2010). Analyses of trends in monitored O₃ indicate that over such a time period, the upper end of the distribution of monitored O₃ concentrations (*i.e.*, indicated by the 95th percentile) generally decreased in urban and non-urban locations across the U.S. (U.S. EPA, 2014a, Figure 8–29). During this same time period, median O₃ concentrations decreased in suburban and rural locations, and in some urban locations. However, median concentrations increased in some large urban centers (U.S. EPA, 2014a, Figure 8–28). As discussed in the HREA, these increases in median concentrations likely reflect the increases in relatively low O₃ concentrations that can occur near important sources of NO_x upon reductions in NO_x emissions (U.S. EPA, 2014a, section 8.2.3.1). These patterns of monitored O₃ during a period when the U.S. experienced large reductions in NO_x emissions are qualitatively consistent with the modeled responses of O₃ to reductions in NO_x emissions.

To evaluate the second issue, the HREA assessed the O₃ air quality response to reducing both NO_x and VOC emissions (*i.e.*, in addition to assessing reductions in NO_x emissions alone) for a subset of seven urban study areas. As discussed in the PA (U.S. EPA, 2014c, section 3.2.1), the addition of VOC reductions generally resulted in larger decreases in mid-range O₃ concentrations (25th to 75th percentiles) (U.S. EPA, 2014a, Appendix 4D, section 4.7).⁴⁵ In addition, in all seven of the

⁴⁴In addition, because epidemiology-based risk estimates use “area-wide” average O₃ concentrations, calculated by averaging concentrations across multiple monitors in urban case study areas (section 3.2.3.2 below), risk estimates on a given day depend on the daily balance between increasing and decreasing O₃ concentrations at individual monitors.

⁴⁵This was the case for all of the urban study areas evaluated, with the exception of New York

urban study areas evaluated, the increases in low O₃ concentrations were smaller for the NO_x/VOC scenarios than the NO_x alone scenarios (U.S. EPA, 2014a, Appendix 4D, section 4.7). This was most apparent for Denver, Houston, Los Angeles, New York, and Philadelphia. Given the impacts on total risk estimates of increases in low O₃ concentrations (discussed below), these results suggest that in some locations optimized emissions reduction strategies could result in larger reductions in O₃-associated mortality and morbidity than indicated by HREA estimates.

b. Exposure Assessment

As discussed in section II.C.2 of the proposal, the O₃ exposure assessment presented in the HREA (U.S. EPA, 2014a, Chapter 5) provides estimates of the number and percent of people exposed to various concentrations of ambient O₃ while at specified exertion levels. The HREA estimates exposures in the 15 urban study areas for four study groups, all school-age children (ages 5 to 18), asthmatic school-age children, asthmatic adults (ages 19 to 95), and all older adults (ages 65 to 95), reflecting the evidence indicating that these populations are at increased risk for O₃-attributable effects (U.S. EPA, 2013, Chapter 8; II.A.1.d, above). An important purpose of these exposure estimates is to provide perspective on the extent to which air quality adjusted to just meet the current O₃ NAAQS could be associated with exposures to O₃ concentrations reported to result in respiratory effects.⁴⁶ These analyses of exposure assessment incorporate behavior patterns, including estimates of physical exertion, which are critical in assessing whether ambient concentrations of O₃ may pose a public health risk.⁴⁷ In particular, exposures to

(U.S. EPA, 2014a, Appendix 4–D, section 4.7). In this analysis, emissions of NO_x and VOC were reduced by equal percentages, a scenario not likely to reflect the optimal combination for reducing risks. In most of the urban study areas the inclusion of VOC emissions reductions did not alter the NO_x emissions reductions required to meet the current or alternative standards. The exceptions are Chicago and Denver, for which the HREA risk estimates are based on reductions in both NO_x and VOC (U.S. EPA, 2014a, section 4.3.3.1).

⁴⁶In addition, the range of modeled personal exposures to ambient O₃ provide an essential input to the portion of the health risk assessment based on exposure-response functions (for lung function decrements) from controlled human exposure studies. The health risk assessment based on exposure-response information is discussed below (II.C.3).

⁴⁷See 79 FR 75269 “The activity pattern of individuals is an important determinant of their exposure. Variation in O₃ concentrations among various microenvironments means that the amount of time spent in each location, as well as the level

ambient or near-ambient O₃ concentrations have only been shown to result in potentially adverse effects if the ventilation rates of people in the exposed populations are raised to a sufficient degree (*e.g.*, through physical exertion) (U.S. EPA, 2013, section 6.2.1.1). Estimates of such “exposures of concern” provide perspective on the potential public health impacts of O₃-related effects, including effects that cannot currently be evaluated in a quantitative risk assessment.⁴⁸

The HREA estimates 8-hour exposures at or above benchmark concentrations of 60, 70, and 80 ppb for individuals engaged in moderate or greater exertion (*i.e.*, to approximate conditions in the controlled human exposure studies on which benchmarks are based). Benchmarks reflect exposure concentrations at which O₃-induced respiratory effects are known to occur in some healthy adults engaged in moderate, quasi-continuous exertion, based on evidence from controlled human exposure studies (U.S. EPA, 2013, section 6.2; U.S. EPA, 2014c, section 3.1.2.1). The amount of weight to place on the estimates of exposures at or above specific benchmark concentrations depends in part on the weight of the scientific evidence concerning health effects associated with O₃ exposures at those benchmark concentrations. It also depends on judgments about the importance, from a public health perspective, of the health effects that are known or can reasonably be inferred to occur as a result of exposures at benchmark concentrations (U.S. EPA, 2014c, sections 3.1.3, 3.1.5).

In considering estimates of O₃ exposures of concern at or above benchmarks of 60, 70, and 80 ppb, the PA focuses on modeled exposures for school-age children (ages 5–18), including asthmatic school-age children, which are key at-risk populations identified in the ISA (U.S. EPA, 2014c, section 3.1.5). The percentages of children estimated to experience exposures of concern are considerably larger than the percentages estimated for adult populations (*i.e.*, approximately 3-fold larger across urban

of activity, will influence an individual’s exposure to ambient O₃. Activity patterns vary both among and within individuals, resulting in corresponding variations in exposure across a population and over time” (internal citations omitted).

⁴⁸In this review, the term “exposure of concern” is defined as a personal exposure, while at moderate or greater exertion, to 8-hour average ambient O₃ concentrations at and above specific benchmarks levels. As discussed below, these benchmark levels represent exposure concentrations at which O₃-induced health effects are known to occur, or can reasonably be anticipated to occur, in some individuals.

study areas)⁴⁹ (U.S. EPA, 2014a, section 5.3.2 and Figures 5–5 to 5–8). The larger exposure estimates for children are due primarily to the larger percentage of children estimated to spend an extended period of time being physically active outdoors when O₃ concentrations are elevated (U.S. EPA, 2014a, sections 5.3.2 and 5.4.1).

Although exposure estimates differ between children and adults, the patterns of results across the urban study areas and years are similar among all of the populations evaluated (U.S. EPA, 2014a, Figures 5–5 to 5–8). Therefore, while the PA highlights estimates in children, including asthmatic school-age children, it also

notes that the patterns of exposures estimated for children represent the patterns estimated for adult asthmatics and older adults.

Table 1 of the proposal (79 FR 75272 to 75273) summarizes key results from the exposure assessment. This table is reprinted below.

TABLE 1—SUMMARY OF ESTIMATED EXPOSURES OF CONCERN IN ALL SCHOOL-AGE CHILDREN FOR THE CURRENT AND ALTERNATIVE O₃ STANDARDS IN URBAN STUDY AREAS

Benchmark concentration	Standard level (ppb)	Average % children exposed ⁵⁰	Average number of children exposed [average number of asthmatic children] ⁵¹	% Children—worst year and worst area
One or more exposures of concern per season				
≥ 80 ppb	75	0–0.3 (0.1)	27,000 [3,000]	1.1
	70	0–0.1 (0)	3,700 [300]	0.2
	65	0 (0)	300 [0]	0
	60	0 (0)	100 ⁵² [0]	0
≥ 70 ppb	75	0.6–3.3 (1.9)	362,000 [40,000]	8.1
	70	0.1–1.2 (0.5)	94,000 [10,000]	3.2
	65	0–0.2 (0.1)	14,000 [2,000]	0.5
	60	0 (0)	1,400 [200]	0.1
≥ 60 ppb	75	9.5–17 (12.2)	2,316,000 [246,000]	25.8
	70	3.3–10.2 (6.2)	1,176,000 [126,000]	18.9
	65	0–4.2 (2.1)	392,000 [42,000]	9.5
	60	0–1.2 (0.4)	70,000 [8,000]	2.2
Two or more exposures of concern per season				
≥ 80 ppb	75	0 (0)	600 [100]	0.1
	70	0 (0)	0 [0]	0
	65	0 (0)	0 [0]	0
	60	0 (0)	0 [0]	0
≥ 70 ppb	75	0.1–0.6 (0.2)	46,000 [5,000]	2.2
	70	0–0.1 (0)	5,400 [600]	0.4
	65	0 (0)	300 [100]	0
	60	0 (0)	0 [0]	0
≥ 60 ppb	75	3.1–7.6 (4.5)	865,000 [93,000]	14.4
	70	0.5–3.5 (1.7)	320,000 [35,000]	9.2
	65	0–0.8 (0.3)	67,000 [7,500]	2.8
	60	0–0.2 (0)	5,100 [700]	0.3

Uncertainties in exposure estimates are summarized in section II.C.2.b of the proposal (79 FR 75273). For example, due to variability in responsiveness, only a subset of individuals who experience exposures at or above a benchmark concentration can be expected to experience health effects.⁵³ In addition, not all of these effects will

be adverse. Given the lack of sufficient exposure-response information for most of the health effects that informed benchmark concentrations, estimates of the number of people likely to experience exposures at or above benchmark concentrations generally cannot be translated into quantitative estimates of the number of people likely

to experience specific health effects.⁵⁴ The PA views health-relevant exposures as a continuum with greater confidence and less uncertainty about the existence of adverse health effects at higher O₃ exposure concentrations, and less confidence and greater uncertainty as one considers lower exposure concentrations (e.g., U.S. EPA, 2014c,

⁴⁹ HREA exposure estimates for all children and asthmatic children are virtually indistinguishable, in terms of the percent estimated to experience exposures of concern (U.S. EPA, 2014a, Chapter 5). Consistent with this, HREA analyses indicate that activity data for people with asthma is generally similar to non-asthmatic populations (U.S. EPA, 2014a, Appendix 5G, Tables 5G2-to 5G–5).

⁵⁰ Estimates for each urban case study area were averaged for the years evaluated in the HREA (2006 to 2010). Ranges reflect the ranges across urban study areas. Estimates smaller than 0.05% were rounded downward to zero (from U.S. EPA, 2014a, Tables 5–11 and 5–12). Numbers in parentheses

reflect averages across urban study areas, as well as over the years evaluated in the HREA.

⁵¹ Numbers of children exposed in each urban case study area were averaged over the years 2006 to 2010. These averages were then summed across urban study areas. Numbers were rounded to nearest thousand unless otherwise indicated. Estimates smaller than 50 were rounded downward to zero (from U.S. EPA, 2014a, Appendix 5F Table 5F–5).

⁵² As discussed in section 4.3.3 of the HREA, the model-based air quality adjustment approach used to estimate exposures and lung function decrements associated with the current and alternative standards was unable to estimate the distribution of

ambient O₃ concentrations in New York City upon just meeting an alternative standard with a level of 60 ppb. Therefore, for the 60 ppb standard level, the numbers of children and asthmatic children, and the ranges of percentages, reflect all of the urban study areas except New York.

⁵³ As noted below (II.C.3.a.ii), in the case of asthmatics, responsiveness to O₃ could depend on factors that have not been well-evaluated, such as asthma severity, the effectiveness of asthma control, or the prevalence of medication use.

⁵⁴ The exception to this is lung function decrements, as discussed below (and in U.S. EPA, 2014c, section 3.2.3.1).

sections 3.1 and 4.6). This view draws from the overall body of available health evidence, which indicates that as exposure concentrations increase, the incidence, magnitude, and severity of effects increases.

Another important uncertainty is that there is very limited evidence from controlled human exposure studies, which provided the basis for health benchmark concentrations for both exposures of concern and lung function decrements, related to clinical responses in at-risk populations. Compared to the healthy young adults included in the controlled human exposure studies, members of at-risk populations could be more likely to experience adverse effects, could experience larger and/or more serious effects, and/or could experience effects following exposures to lower O₃ concentrations.⁵⁵

There are also uncertainties associated with the exposure modelling. These are described most fully, and their potential impact characterized, in section 5.5.2 of the HREA (U.S. EPA, 2013, pp. 5–72 to 5–79). These include interpretation of activity patterns set forth in diaries which do not typically distinguish the basis for activity patterns and so may reflect averting behavior,⁵⁶ and whether the HREA underestimates exposures for groups spending especially large proportion of time being active outdoors during the O₃ season (outdoor workers and especially active children).

c. Quantitative Health Risk Assessments

As discussed in section II.C.3 of the proposal (79 FR 75274), for some health endpoints, there is sufficient scientific evidence and information available to support the development of quantitative estimates of O₃-related health risks. In the current review, for short-term O₃ concentrations, the HREA estimates lung function decrements; respiratory symptoms in asthmatics; hospital admissions and emergency department visits for respiratory causes; and all-cause mortality (U.S. EPA, 2014a). For long-term O₃ concentrations, the HREA estimates respiratory mortality (U.S. EPA, 2014a).⁵⁷ Estimates of O₃-induced lung function decrements are based on exposure modeling using the MSS model (see section II.1.b.i.(1) above, and 79 FR 75250), combined with exposure-response relationships from controlled human exposure studies (U.S. EPA, 2014a, Chapter 6). Estimates of O₃-associated respiratory symptoms, hospital admissions and emergency department visits, and mortality are based on concentration-response relationships from epidemiologic studies (U.S. EPA, 2014a, Chapter 7). As with the exposure assessment discussed above, O₃-associated health risks are estimated for recent air quality and for ambient concentrations adjusted to just meet the current and alternative O₃ standards, based on 2006–2010 air quality and adjusted precursor emissions. The following sections summarize the discussions from the

proposal on the lung function risk assessment (II.A.2.c.i) and the epidemiology-based morbidity and mortality risk assessments (II.A.2.c.ii).

i. Lung Function Risk Assessment

The HREA estimates risks of lung function decrements in school-aged children (ages 5 to 18), asthmatic school-aged children, and the general adult population for the 15 urban study areas. The results presented in the HREA are based on an updated dose-threshold model that estimates FEV₁ responses for individuals following short-term exposures to O₃ (McDonnell et al., 2012), reflecting methodological improvements since the last review (II.B.2.a.i (1), above; U.S. EPA, 2014a, section 6.2.4). The impact of the dose threshold is that O₃-induced FEV₁ decrements result primarily from exposures on days with average ambient O₃ concentrations above about 40 ppb (U.S. EPA, 2014a, section 6.3.1, Figure 6–9).⁵⁸

Table 2 in the proposal (79 FR 75275), and reprinted below, summarizes key results from the lung function risk assessment. Table 2 presents estimates of the percentages of school-aged children estimated to experience O₃-induced FEV₁ decrements >10, 15, or 20% when air quality was adjusted to just meet the current and alternative 8-hour O₃ standards. Table 2 also presents the numbers of children, including children with asthma, estimated to experience such decrements.

TABLE 2—SUMMARY OF ESTIMATED O₃-INDUCED LUNG FUNCTION DECREMENTS FOR THE CURRENT AND POTENTIAL ALTERNATIVE O₃ STANDARDS IN URBAN CASE STUDY AREAS

Lung function decrement	Alternative standard level	Average % children ⁵⁹	Number of children (5 to 18 years) [number of asthmatic children] ⁶⁰	% Children worst year and area
One or more decrements per season				
≥10%	75	14–19	3,007,000 [312,000]	22
	70	11–17	2,527,000 [261,000]	20
	65	3–15	1,896,000 [191,000]	18
	60	5–11	⁶¹ 1,404,000 [139,000]	13
≥15%	75	3–5	766,000 [80,000]	7
	70	2–4	562,000 [58,000]	5
	65	0–3	356,000 [36,000]	4
	60	1–2	225,000 [22,000]	3
≥20%	75	1–2	285,000 [30,000]	2.8
	70	1–2	189,000 [20,000]	2.1
	65	0–1	106,000 [11,000]	1.4
	60	0–1	57,000 [6,000]	0.9

⁵⁵ “The CASAC further notes that clinical studies do not address sensitive subgroups, such as children with asthma, and that there is a scientific basis to anticipate that the adverse effects for such subgroups are likely to be more significant at 60 ppb than for healthy adults” (Frey 2014a, p. 7).

⁵⁶ See EPA 2014a pp. 5–53 to 54 describing EPA’s sensitivity analysis regarding impacts of potential averting behavior for school-age children on the

exposure and lung function decrement estimate, and see also section B.2.a.i below.

⁵⁷ Estimates of O₃-associated respiratory mortality are based on the study by Jerrett *et al.* (2009). This study used seasonal averages of 1-hour daily maximum O₃ concentrations to estimate long-term concentrations.

⁵⁸ Analysis of this issue in the HREA is based on risk estimates in Los Angeles for 2006 unadjusted air quality. The HREA shows that more than 90% of daily instances of FEV₁ decrements ≥10% occur when 8-hr average ambient concentrations are above 40 ppb for this modeled scenario. The HREA notes that the distribution of responses will be different for different study areas, years, and air quality scenarios (U.S. EPA, 2014c, Chapter 6).

TABLE 2—SUMMARY OF ESTIMATED O₃-INDUCED LUNG FUNCTION DECREMENTS FOR THE CURRENT AND POTENTIAL ALTERNATIVE O₃ STANDARDS IN URBAN CASE STUDY AREAS—Continued

Lung function decrement	Alternative standard level	Average % children ⁵⁹	Number of children (5 to 18 years) [number of asthmatic children] ⁶⁰	% Children worst year and area
Two or more decrements per season				
≥10%	75	7.5–12	1,730,000 [179,000]	14
	70	5.5–11	1,414,000 [145,000]	13
	65	1.3–8.8	1,023,000 [102,000]	11
≥15%	60	2.1–6.4	741,000 [73,000]	7.3
	75	1.7–2.9	391,000 [40,000]	3.8
	70	0.9–2.4	276,000 [28,000]	3.1
≥20%	65	0.1–1.8	168,000 [17,000]	2.3
	60	0.2–1.0	101,000 [10,000]	1.4
	75	0.5–1.1	128,000 [13,000]	1.5
	70	0.3–0.8	81,000 [8,000]	1.1
	65	0–0.5	43,000 [4,000]	0.8
	60	0–0.2	21,000 [2,000]	0.4

Uncertainties in estimates of lung function risks are summarized in section II.C.3.a.ii of the proposal (79 FR 75275). In addition to the uncertainties noted for exposure estimates, an uncertainty which impacts lung function risk estimates stems from the lack of exposure-response information in children. In the near absence of controlled human exposure data for children, risk estimates are based on the assumption that children exhibit the same lung function response following O₃ exposures as healthy 18 year olds (*i.e.*, the youngest age for which controlled human exposure data is generally available) (U.S. EPA, 2014a, section 6.5.3). This assumption is justified in part by the findings of McDonnell et al. (1985), who reported that children (8–11 years old) experienced FEV₁ responses similar to those observed in adults (18–35 years old) (U.S. EPA, 2014a, p. 3–10). In

⁵⁹ Estimates in each urban case study area were averaged for the years evaluated in the HREA (2006 to 2010). Ranges reflect the ranges across urban study areas.

⁶⁰ Numbers of children estimated to experience decrements in each study urban case study area were averaged over 2006 to 2010. These averages were then summed across urban study areas. Numbers are rounded to nearest thousand unless otherwise indicated.

⁶¹ As discussed in section 4.3.3 of the HREA, the model-based air quality adjustment approach used to estimate risks associated with the current and alternative standards was unable to estimate the distribution of ambient O₃ concentrations in New York City upon just meeting an alternative standard with a level of 60 ppb. Therefore, for the 60 ppb standard level, the numbers of children and asthmatic children experiencing decrements, and the ranges of percentages of such children across study areas, reflect all of the urban study areas except New York City. Because of this, in some cases (*i.e.*, when New York City provided the smallest risk estimate), the lower end of the ranges in Table 2 are higher for a standard level of 60 ppb than for a level of 65 ppb.

addition, as discussed in the ISA (U.S. EPA, 2013, section 6.2.1), summer camp studies of school-aged children reported O₃-induced lung function decrements similar in magnitude to those observed in controlled human exposure studies using adults. In extending the risk model to children, the HREA thus fixes the age term in the model at its highest value, the value for age 18. Notwithstanding the information just summarized supporting this approach, EPA acknowledges the uncertainty involved, and notes that the approach could result in either over- or underestimates of O₃-induced lung function decrements in children, depending on how children compare to the adults used in controlled human exposure studies (U.S. EPA, 2014a, section 6.5.3).

A related source of uncertainty is that the risk assessment estimates of O₃-induced decrements in asthmatics used the exposure-response relationship developed from data collected from healthy individuals. Although the evidence has been mixed (U.S. EPA, 2013, section 6.2.1.1), several studies have reported statistically larger, or a tendency toward larger, O₃-induced lung function decrements in asthmatics than in non-asthmatics (Kreit et al., 1989; Horstman et al., 1995; Jorres et al., 1996; Alexis et al., 2000). On this issue, CASAC noted that “[a]sthmatic subjects appear to be at least as sensitive, if not more sensitive, than non-asthmatic subjects in manifesting O₃-induced pulmonary function decrements” (Frey, 2014c, p. 4). To the extent asthmatics experience larger O₃-induced lung function decrements than the healthy adults used to develop exposure-response relationships, the HREA could underestimate the impacts of O₃ exposures on lung function in

asthmatics, including asthmatic children. The implications of this uncertainty for risk estimates remain unknown at this time (U.S. EPA, 2014a, section 6.5.4), and could depend on a variety of factors that have not been well-evaluated, including the severity of asthma and the prevalence of medication use. However, the available evidence shows responses to O₃ increase with severity of asthma (Horstman et al., 1995) and corticosteroid usage does not prevent O₃ effects on lung function decrements or respiratory symptoms in people with asthma (Vagaggini et al., 2001, 2007).

ii. Mortality and Morbidity Risk Assessments

As discussed in section II.C.3.b of the proposal (79 FR 75276), the HREA estimates O₃-associated risks in 12 urban study areas⁶² using concentration-response relationships drawn from epidemiologic studies. These concentration-response relationships are based on “area-wide” average O₃ concentrations.⁶³ The HREA estimates risks for the years 2007 and 2009 in order to provide estimates of risk for a year with generally higher O₃

⁶² The 12 urban areas evaluated are Atlanta, Baltimore, Boston, Cleveland, Denver, Detroit, Houston, Los Angeles, New York, Philadelphia, Sacramento, and St. Louis.

⁶³ In the epidemiologic studies that provide the health basis for HREA risk assessments, concentration-response relationships are based on daytime O₃ concentrations, averaged across multiple monitors within study areas. These daily averages are used as surrogates for the spatial and temporal patterns of exposures in study populations. Consistent with this approach, the HREA epidemiologic-based risk estimates also utilize daytime O₃ concentrations, averaged across monitors, as surrogates for population exposures. In this notice, we refer to these averaged concentrations as “area-wide” O₃ concentrations. Area-wide concentrations are discussed in more detail in section 3.1.4 of the PA (U.S. EPA, 2014c).

concentrations (2007) and a year with generally lower O₃ concentrations (2009) (U.S. EPA, 2014a, section 7.1.1).

In considering the epidemiology-based risk estimates, the proposal focuses on mortality risks associated with short-term O₃ concentrations. The proposal considers estimates of total risk (*i.e.*, based on the full distributions of ambient O₃ concentrations) and estimates of risk associated with O₃ concentrations in the upper portions of ambient distributions. Both estimates are discussed to provide information that considers risk estimates based on concentration-response relationships being linear over the entire distribution of ambient O₃ concentrations, and thus have the greater potential for morbidity and mortality to be affected by changes in relatively low O₃ concentrations, as well as risk estimates that are associated with O₃ concentrations in the upper portions of the ambient distribution, thus focusing on risk from higher O₃ concentrations and placing greater weight on the uncertainty associated with the shapes of concentration-response curves for O₃ concentrations in the lower portions of the distribution. These results for O₃-associated mortality risk are summarized in Table 3 in the proposal (79 FR 75277).

Important uncertainties in epidemiology-based risk estimates, based on their consideration in the HREA and PA, are discussed in section II.C.3.b.ii of the proposal (79 FR 75277). Compared to estimates of O₃ exposures of concern and estimates of O₃-induced lung function decrements (discussed above), the HREA conclusions reflect lower confidence in epidemiologic-based risk estimates (U.S. EPA, 2014a, section 9.6). In particular, the HREA highlights the heterogeneity in effect estimates between locations, the potential for exposure measurement errors, and uncertainty in the interpretation of the shape of concentration-response functions at lower O₃ concentrations (U.S. EPA, 2014a, section 9.6). The HREA also concludes that lower confidence should be placed in the results of the assessment of respiratory mortality risks associated with long-term O₃, primarily because that analysis is based on only one study, though that study is well-designed, and because of the uncertainty in that study about the existence and identification of a potential threshold in the concentration-response function (U.S. EPA, 2014a, section 9.6).^{64,65} This section further

discusses some of the key uncertainties in epidemiologic-based risk estimates, as summarized in the PA (U.S. EPA, 2014c, section 3.2.3.2), with a focus on uncertainties that can have particularly important implications for the Administrator's consideration of epidemiology-based risk estimates.

The PA notes that reducing NO_x emissions generally reduces O₃-associated mortality and morbidity risk estimates in locations and time periods with relatively high ambient O₃ concentrations and increases risk estimates in locations and time periods with relatively low concentrations (II.A. above). When evaluating uncertainties in epidemiologic risk estimates, the PA considered (1) the extent to which the modeled O₃ response to reductions in NO_x emissions appropriately represents the trends observed in monitored ambient O₃ following actual reductions in NO_x emissions, (2) the extent to which the O₃ response to reductions in precursor emissions could differ with emissions reduction strategies that are different from those used in HREA to generate risk estimates, and (3) the extent to which estimated changes in risks in urban study areas are representative of the changes that would be experienced broadly across the U.S. population. The first two of these issues are discussed in section II.A.2.c above. The third issue is discussed below.

The HREA conducted national air quality modeling analyses that estimated the proportion of the U.S. population living in locations where seasonal averages of daily O₃ concentrations are estimated to decrease in response to reductions in NO_x emissions, and the proportion living in locations where such seasonal averages are estimated to increase. Given the close relationship between changes in seasonal averages of daily O₃ concentrations and changes in seasonal mortality and morbidity risk estimates, this analysis informs consideration of the extent to which the risk results in urban study areas represent the U.S. population as a whole. This "representativeness analysis" indicates that the majority of the U.S. population lives in locations where reducing NO_x emissions would be expected to result in decreases in warm season averages of

mortality response, the estimated number of premature deaths avoidable for long-term exposure reductions for several levels need to be viewed with caution" (Frey, 2014a, p. 3).

⁶⁵ There is also uncertainty about the extent to which mortality estimates based on the long-term metric used in the study by Jerrett et al. (2009) (*i.e.*, seasonal average of 1-hour daily maximum concentrations) reflects associations with long-term average O₃ versus repeated occurrences of elevated short-term concentrations.

daily maximum 8-hour ambient O₃ concentrations. Because the HREA urban study areas tend to underrepresent the populations living in such areas (*e.g.*, suburban, smaller urban, and rural areas), risk estimates for the urban study areas are likely to understate the average reductions in O₃-associated mortality and morbidity risks that would be experienced across the U.S. population as a whole upon reducing NO_x emissions (U.S. EPA, 2014a, section 8.2.3.2).

Section 7.4 of the HREA also highlights some additional uncertainties associated with epidemiologic-based risk estimates (U.S. EPA, 2014a). This section of the HREA identifies and discusses sources of uncertainty and presents a qualitative evaluation of key parameters that can introduce uncertainty into risk estimates (U.S. EPA, 2014a, Table 7-4). For several of these parameters, the HREA also presents quantitative sensitivity analyses (U.S. EPA, 2014a, sections 7.4.2 and 7.5.3). Of the uncertainties discussed in Chapter 7 of the HREA, those related to the application of concentration-response functions from epidemiologic studies can have particularly important implications for consideration of epidemiology-based risk estimates, as discussed below.

An important uncertainty is the shape of concentration-response functions at low ambient O₃ concentrations (U.S. EPA, 2014a, Table 7-4).⁶⁶ In recognition of the ISA's conclusion that certainty in the shape of O₃ concentration-response functions decreases at low ambient concentrations, the HREA provides estimates of epidemiology-based mortality risks for entire distributions of ambient O₃ concentrations, as well as estimates of total mortality associated with various ambient O₃ concentrations. The PA considers both types of risk estimates, recognizing greater public health concern for adverse O₃-attributable effects at higher ambient O₃ concentrations (which drive higher exposure concentrations, section 3.2.2 of the PA (U.S. EPA, 2014c)), as compared to lower concentrations.

A related consideration is associated with the public health importance of the increases in relatively low O₃ concentrations following air quality adjustment. There is uncertainty that relates to the assumption that the concentration response function for O₃ is linear, such that total risk estimates are equally influenced by decreasing

⁶⁶ A related uncertainty is the existence, or not, of a threshold. The HREA addresses this issue for long-term O₃ by evaluating risks in models that include potential thresholds (II.D.2.c).

high concentrations and increasing low concentrations, when the increases and decreases are of equal magnitude. Even on days with increases in relatively low area-wide average concentrations, resulting in increases in estimated risks, some portions of the urban study areas could experience decreases in high O₃ concentrations. To the extent adverse O₃-attributable effects are more strongly supported for higher ambient concentrations (which, as noted above, are consistently reduced upon air quality adjustment), the impacts on risk estimates of increasing low O₃ concentrations reflect an important source of uncertainty. In addition to the uncertainties discussed above, the proposal also notes uncertainties related to (1) using concentration-response relationships developed for a particular population in a particular location to estimate health risks in different populations and locations; (2) using concentration-response functions from epidemiologic studies reflecting a particular air quality distribution to adjusted air quality necessarily reflecting a different (simulated) air quality distribution; (3) using a national concentration-response function to estimate respiratory mortality associated with long-term O₃; and (4) unquantified reductions in risk that could be associated with reductions in the ambient concentrations of pollutants other than O₃, resulting from control of NO_x (79 FR 75277 to 75279).

B. Need for Revision of the Primary Standard

The initial issue to be addressed in the current review of the primary O₃ standard is whether, in view of the advances in scientific knowledge and additional information, it is appropriate to revise the existing standard. This section presents the Administrator's final decision on whether it is "appropriate" to revise the current standard within the meaning of section 109 (d)(1) of the CAA. Section II.B.1 contains a summary discussion of the basis for the proposed conclusions on the adequacy of the primary standard. Section II.B.2 discusses comments received on the adequacy of the primary standard. Section II.B.3 presents the Administrator's final conclusions on the adequacy of the current primary standard.

1. Basis for Proposed Decision

In evaluating whether it is appropriate to retain or revise the current standard, the Administrator's considerations build upon those in the 2008 review, including consideration of the broader body of scientific evidence and

exposure and health risk information now available, as summarized in sections II.A to II.C (79 FR 75246–75279) of the proposal and section II.A above.

In developing conclusions on the adequacy of the current primary O₃ standard, the Administrator takes into account both evidence-based and quantitative exposure- and risk-based considerations. Evidence-based considerations include the assessment of evidence from controlled human exposure, animal toxicological, and epidemiologic studies for a variety of health endpoints. The Administrator focuses on health endpoints for which the evidence is strong enough to support a "causal" or a "likely to be causal" relationship, based on the ISA's integrative synthesis of the entire body of evidence. The Administrator's consideration of quantitative exposure and risk information draws from the results of the exposure and risk assessments presented in the HREA.

The Administrator's consideration of the evidence and exposure/risk information is informed by the considerations and conclusions presented in the PA (U.S. EPA, 2014c). The purpose of the PA is to help "bridge the gap" between the scientific and technical information assessed in the ISA and HREA, and the policy decisions that are required of the Administrator (U.S. EPA, 2014c, Chapter 1); see also *American Farm Bureau Federation*, 559 F. 3d at 516, 521 ("[a]lthough not required by the statute, in practice EPA staff also develop a Staff Paper, which discusses the information in the Criteria Document that is most relevant to the policy judgments the EPA makes when it sets the NAAQS"). The PA's evidence-based and exposure-/risk-based considerations and conclusions are briefly summarized below in sections II.B.1.a (evidence-based considerations), II.B.1.b (exposure- and risk-based considerations), and II.B.1.c (PA conclusions on the current standard). Section II.B.1.d summarizes CASAC advice to the Administrator and public commenter views on the current standard. Section II.B.1.e presents a summary of the Administrator's proposed conclusions concerning the adequacy of the public health protection provided by the current standard, and her proposed decision to revise that standard.

a. Evidence-Based Considerations From the PA

In considering the available scientific evidence, the PA evaluates the O₃ concentrations in health effects studies (U.S. EPA, 2014c, section 3.1.4).

Specifically, the PA characterizes the extent to which health effects have been reported for the O₃ exposure concentrations evaluated in controlled human exposure studies, and effects occurring over the distributions of ambient O₃ concentrations in locations where epidemiologic studies have been conducted. These considerations, as they relate to the adequacy of the current standard, are presented in detail in section 3.1.4 of the PA (U.S. EPA, 2014c) and are summarized in the proposal (79 FR 75279–75287). The PA's considerations are summarized briefly below for controlled human exposure, epidemiologic panel studies, and epidemiologic population-based studies.

Section II.D.1.a of the proposal discusses the PA's consideration of the evidence from controlled human exposure and panel studies. This evidence is assessed in section 6.2 of the ISA (U.S. EPA, 2013) and is summarized in section 3.1.2 of the PA (U.S. EPA, 2014c). A large number of controlled human exposure studies have reported lung function decrements, respiratory symptoms, air inflammation, airway hyperresponsiveness, and/or impaired lung host defense in young, healthy adults engaged in moderate quasi-continuous exertion, following 6.6-hour O₃ exposures. These studies have consistently reported such effects following exposures to O₃ concentrations of 80 ppb or greater. In addition to lung function decrements, available studies have evaluated respiratory symptoms or airway inflammation following exposures to O₃ concentrations below 75 ppb. Table 3–1 in the PA highlights the group mean results of individual controlled human exposure studies that evaluated exposures to O₃ concentrations below 75 ppb. These studies observe the combination of lung function decrements and respiratory symptoms following exposures to O₃ concentrations as low as 72 ppb, and lung function decrements and airway inflammation following exposures to O₃ concentrations as low as 60 ppb (based on group means).

Based on this evidence, the PA notes that controlled human exposure studies have reported a variety of respiratory effects in young, healthy adults following exposures to a wide range of O₃ concentrations for 6.6 hours, including exposures to concentrations below 75 ppb. In particular, the PA further notes that a recent controlled human exposure study reported the combination of lung function decrements and respiratory symptoms in healthy adults engaged in quasi-

continuous, moderate exertion following 6.6 hour exposures to 72 ppb O₃, a combination of effects that have been classified as adverse based on ATS guidelines for adversity (ATS, 2000a). In addition, a recent study has also reported lung function decrements and pulmonary inflammation following exposure to 60 ppb O₃. Sixty ppb is the lowest exposure concentration for which inflammation has been evaluated and reported to occur, and corresponds to the lowest exposure concentration demonstrated to result in lung function decrements large enough to be judged an abnormal response by ATS (ATS, 2000b). The PA also notes, and CASAC agreed, that these controlled human exposure studies were conducted in healthy adults, while at-risk groups (e.g., children, people with asthma) could experience larger and/or more serious effects. Therefore, the PA concludes that the evidence from controlled human exposure studies provide support that the respiratory effects experienced following exposures to O₃ concentrations lower than 75 ppb would be adverse in some individuals, particularly if experienced by members of at-risk populations (e.g., people with asthma, children).

The PA also notes consistent results in some panel studies of O₃-associated lung function decrements. In particular, the PA notes that epidemiologic panel studies in children and adults consistently indicate O₃-associated lung function decrements when on-site, ambient monitored concentrations were below 75 ppb (although the evidence becomes less consistent at low O₃ concentrations, and the averaging periods involved ranged from 10 minutes to 12 hours (U.S. EPA, 2014c, section 3.2.4.2)).

Section II.D.1.b of the proposal summarizes the PA's analyses of monitored O₃ concentrations in locations of epidemiologic studies. While the majority of the epidemiologic study areas evaluated would have violated the current standard during study periods, the PA makes the following observations with regard to health effect associations at O₃ concentrations likely to have met the current standard:

(1) A single-city study reported positive and statistically significant associations with asthma emergency department visits in children and adults in Seattle, a location that would have met the current standard over the entire study period (Mar and Koenig, 2009).

(2) Additional single-city studies support associations with respiratory morbidity at relatively low ambient O₃ concentrations, including when

virtually all monitored concentrations were below the level of the current standard (Silverman and Ito, 2010; Strickland et al., 2010).

(3) Canadian multicity studies reported positive and statistically significant associations with respiratory morbidity or mortality when the majority of study cities, though not all study cities, would have met the current standard over the study period in each of these studies (Cakmak et al., 2006; Dales et al., 2006; Katsouyanni et al., 2009; Stieb et al., 2009).

(4) A U.S. multicity study reported positive and statistically significant associations with mortality when ambient O₃ concentrations were restricted to those likely to have met the current O₃ standard (Bell et al., 2006).

The PA also takes into account important uncertainties in these analyses of air quality in locations of epidemiologic study areas. These uncertainties are summarized in section II.D.1.b.iii of the proposal. Briefly, they include the following: (1) Uncertainty in conclusions about the extent to which multicity effect estimates reflect associations with air quality meeting the current standard, versus air quality violating that standard; (2) uncertainty regarding the potential for thresholds to exist, given that regional heterogeneity in O₃ health effect associations could obscure the presence of thresholds, should they exist; (3) uncertainty in the extent to which the PA appropriately recreated the air quality analyses in the published study by Bell et al. (2006); and (4) uncertainty in the extent to which reported health effects are caused by exposures to O₃ itself, as opposed to other factors such as co-occurring pollutants or pollutant mixtures, particularly at low ambient O₃ concentrations.⁶⁷

In considering the analyses of monitored O₃ air quality in locations of epidemiologic studies, as well as the important uncertainties in these analyses, the PA concludes that these analyses provide support for the occurrence of morbidity and mortality associated with short-term ambient O₃ concentrations likely to meet the current O₃ standard.⁶⁸ In considering the

⁶⁷ As noted above (section II.A.1.B.i), the ISA concludes that studies that examined the potential confounding effects of copollutants found that O₃ effect estimates remained relatively robust upon the inclusion of PM and gaseous pollutants in two-pollutant models (U.S. EPA, 2013, section 6.2.7.5).

⁶⁸ Unlike for the studies of short-term O₃, the available U.S. and Canadian epidemiologic studies evaluating long-term ambient O₃ concentration metrics have not been conducted in locations likely to have met the current 8-hour O₃ standard during the study period, and have not reported concentration-response functions that indicate

evidence as a whole, the PA concludes that (1) controlled human exposure studies provide strong support for the occurrence of adverse respiratory effects following exposures to O₃ concentrations below the level of the current standard and (2) epidemiologic studies provide support for the occurrence of adverse respiratory effects and mortality under air quality conditions that would meet the current standard.

b. Exposure- and Risk-Based Considerations in the PA

In order to further inform judgments about the potential public health implications of the current O₃ NAAQS, the PA considers the exposure and risk assessments presented in the HREA (U.S. EPA, 2014c, section 3.2). Overviews of these exposure and risk assessments, including brief summaries of key results and uncertainties, are provided in section II.A.2 above. Section II.D.2 of the proposal summarizes key observations from the PA related to the adequacy of the current O₃ NAAQS, based on consideration of the HREA exposure assessment, lung function risk assessment, and mortality/morbidity risk assessments (79 FR 75283).

Section II.D.2.a of the proposal summarizes key observations from the PA regarding estimates of O₃ exposures of concern (79 FR 75283). Given the evidence for respiratory effects from controlled human exposure studies, the PA considers the extent to which the current standard would be estimated to protect at-risk populations against exposures of concern to O₃ concentrations at or above the health benchmark concentrations of 60, 70, and 80 ppb (*i.e.*, based on HREA estimates of one or more and two or more exposures of concern). In doing so, the PA notes the CASAC conclusion that (Frey, 2014c, p. 6):

The 80 ppb-8hr benchmark level represents an exposure level for which there is substantial clinical evidence demonstrating a range of ozone-related effects including lung inflammation and airway responsiveness in healthy individuals. The 70 ppb-8hr benchmark level reflects the fact that in healthy subjects, decreases in lung function and respiratory symptoms occur at concentrations as low as 72 ppb and that these effects almost certainly occur in some people, including asthmatics and others with low lung function who are less tolerant of such effects, at levels of 70 ppb and below. The 60 ppb-8hr benchmark level represents the lowest exposure level at which ozone-

confidence in health effect associations at O₃ concentrations meeting the current standard (U.S. EPA, 2014c, section 3.1.4.3).

related effects have been observed in clinical studies of healthy individuals.

For exposures of concern at or above 60 ppb, the proposal highlights the following key observations for air quality adjusted to just meet the current standard:

(1) On average over the years 2006 to 2010, the current standard is estimated to allow approximately 10 to 18% of children in urban study areas to experience one or more exposures of concern at or above 60 ppb. Summing across urban study areas, these percentages correspond to almost 2.5 million children experiencing approximately 4 million exposures of concern at or above 60 ppb during a single O₃ season. Of these children, almost 250,000 are asthmatics.⁶⁹

(2) On average over the years 2006 to 2010, the current standard is estimated to allow approximately 3 to 8% of children in urban study areas to experience two or more exposures of concern to O₃ concentrations at or above 60 ppb. Summing across the urban study areas, these percentages correspond to almost 900,000 children (including almost 90,000 asthmatic children).

(3) In the worst-case years (*i.e.*, those with the largest exposure estimates), the current standard is estimated to allow approximately 10 to 25% of children to experience one or more exposures of concern at or above 60 ppb, and approximately 4 to 14% to experience two or more exposures of concern at or above 60 ppb.

For exposures of concern at or above 70 ppb, the PA highlights the following key observations for air quality adjusted to just meet the current standard:

(1) On average over the years 2006 to 2010, the current standard is estimated to allow up to approximately 3% of children in urban study areas to experience one or more exposures of concern at or above 70 ppb. Summing across urban study areas, almost 400,000 children (including almost 40,000 asthmatic children) are estimated to experience O₃ exposure concentrations at or above 70 ppb during a single O₃ season.

(2) On average over the years 2006 to 2010, the current standard is estimated to allow less than 1% of children in urban study areas to experience two or more exposures of concern to O₃ concentrations at or above 70 ppb.

(3) In the worst-case location and year, the current standard is estimated to allow approximately 8% of children to experience one or more exposures of concern at or above 70 ppb, and approximately 2% to experience two or more exposures of concern, at or above 70 ppb.

For exposures of concern at or above 80 ppb, the PA highlights the observation that the current standard is estimated to allow about 1% or fewer children in urban study areas to experience exposures of concern at or above 80 ppb, even in years with the highest exposure estimates.

Uncertainties in exposure estimates are summarized in section II.C.2.b of the proposal (79 FR 75273), and discussed more fully in the HREA (U.S. EPA, 2014a, section 5.5.2) and the PA (U.S. EPA, 2014c, section 3.2.2). Key uncertainties include the variability in responsiveness following O₃ exposures, resulting in only a subset of exposed individuals experiencing health effects, adverse or otherwise, and the limited evidence from controlled human exposure studies conducted in at-risk populations. In addition, there are a number of uncertainties in the exposure modelling approach used in the HREA, contributing to overall uncertainty in exposure estimates.

Section II.D.2.b of the proposal summarizes key observations from the PA regarding the estimated risk of O₃-induced lung function decrements (79 FR 75283 to 75284). With respect to the lung function decrements that have been evaluated in controlled human exposure studies, the PA considers the extent to which standards with revised levels would be estimated to protect healthy and at-risk populations against one or more, and two or more, moderate (*i.e.*, FEV₁ decrements ≥10% and ≥15%) and large (*i.e.*, FEV₁ decrements ≥20%) lung function decrements. As discussed in section 3.1.3 of the PA (U.S. EPA, 2014c), although some experts would judge single occurrences of moderate responses to be a nuisance, especially for healthy individuals, a more general consensus view of the adversity of moderate lung function decrements emerges as the frequency of occurrence increases.

With regard to decrements ≥10%, the PA highlights the following key observations for air quality adjusted to just meet the current standard:

(1) On average over the years 2006 to 2010, the current standard is estimated to allow approximately 14 to 19% of children in urban study areas to experience one or more lung function decrements ≥10%. Summing across

urban study areas, this corresponds to approximately 3 million children experiencing 15 million O₃-induced lung function decrements ≥10% during a single O₃ season. Of these children, about 300,000 are asthmatics.

(2) On average over the years 2006 to 2010, the current standard is estimated to allow approximately 7 to 12% of children in urban study areas to experience two or more O₃-induced lung function decrements ≥10%. Summing across the urban study areas, this corresponds to almost 2 million children (including almost 200,000 asthmatic children) estimated to experience two or more O₃-induced lung function decrements greater than 10% during a single O₃ season.

(3) In the worst-case years, the current standard is estimated to allow approximately 17 to 23% of children in urban study areas to experience one or more lung function decrements ≥10%, and approximately 10 to 14% to experience two or more O₃-induced lung function decrements ≥10%.

With regard to decrements ≥15%, the PA highlights the following key observations for air quality adjusted to just meet the current standard:

(1) On average over the years 2006 to 2010, the current standard is estimated to allow approximately 3 to 5% of children in urban study areas to experience one or more lung function decrements ≤15%. Summing across urban study areas, this corresponds to approximately 800,000 children (including approximately 80,000 asthmatic children) estimated to experience at least one O₃-induced lung function decrement ≤15% during a single O₃ season.

(2) On average over the years 2006 to 2010, the current standard is estimated to allow approximately 2 to 3% of children in urban study areas to experience two or more O₃-induced lung function decrements ≤15%.

(3) In the worst-case years, the current standard is estimated to allow approximately 4 to 6% of children in urban study areas to experience one or more lung function decrements ≤15%, and approximately 2 to 4% to experience two or more O₃-induced lung function decrements ≤15%.

With regard to decrements ≤20%, the PA highlights the following key observations for air quality adjusted to just meet the current standard:

(1) On average over the years 2006 to 2010, the current standard is estimated to allow approximately 1 to 2% of children in urban study areas to experience one or more lung function decrements ≥20%. Summing across

⁶⁹ As discussed in section II.C.2.b of the proposal, due to variability in responsiveness, only a subset of individuals who experience exposures at or above a benchmark concentration can be expected to experience adverse health effects.

urban study areas, this corresponds to approximately 300,000 children (including approximately 30,000 asthmatic children) estimated to experience at least one O₃-induced lung function decrement $\geq 20\%$ during a single O₃ season.

(2) On average over the years 2006 to 2010, the current standard is estimated to allow less than 1% of children in urban study areas to experience two or more O₃-induced lung function decrements $\geq 20\%$.

(3) In the worst-case years, the current standard is estimated to allow approximately 2 to 3% of children to experience one or more lung function decrements $\geq 20\%$, and less than 2% to experience two or more O₃-induced lung function decrements $\geq 20\%$.

Uncertainties in lung function risk estimates are summarized in section II.C.3.a of the proposal, and discussed more fully in the HREA (U.S. EPA, 2014a, section 6.5) and the PA (U.S. EPA, 2014c, section 3.2.3.1). In addition to the uncertainties noted above for exposure estimates, the key uncertainties associated with estimates of O₃-induced lung function decrements include the paucity of exposure-response information in children and in people with asthma.

Section II.D.2.c of the proposal summarizes key observations from the PA regarding risk estimates of O₃-associated mortality and morbidity (79 FR 75284 to 75285). With regard to total mortality or morbidity associated with short-term O₃, the PA notes the following for air quality adjusted to just meet the current standard:

(1) When air quality was adjusted to the current standard for the 2007 model year (the year with generally “higher” O₃-associated risks), 10 of 12 urban study areas exhibited either decreases or virtually no change in estimates of the number of O₃-associated deaths (U.S. EPA, 2014a, Appendix 7B). Increases were estimated in two of the urban

study areas (Houston, Los Angeles)⁷⁰ (U.S. EPA, 2014a, Appendix 7B).⁷¹

(2) In focusing on total risk, the current standard is estimated to allow thousands of O₃-associated deaths per year in the urban study areas. In focusing on the risks associated with the upper portions of distributions of ambient concentrations (area-wide concentrations $\leq 40, 60$ ppb), the current standard is estimated to allow hundreds to thousands of O₃-associated deaths per year in the urban study areas.

(3) The current standard is estimated to allow tens to thousands of O₃-associated morbidity events per year (*i.e.*, respiratory-related hospital admissions, emergency department visits, and asthma exacerbations). With regard to respiratory mortality associated with long-term O₃, the PA notes the following for air quality adjusted to just meet the current standard:

(1) Based on a linear concentration-response function, the current standard is estimated to allow thousands of O₃-associated respiratory deaths per year in the urban study areas.

(2) Based on threshold models, HREA sensitivity analyses indicate that the number of respiratory deaths associated with long-term O₃ concentrations could potentially be considerably lower (*i.e.*,

⁷⁰ As discussed above (II.C.1), in locations and time periods when NO_x is predominantly contributing to O₃ formation (*e.g.*, downwind of important NO_x sources, where the highest O₃ concentrations often occur), model-based adjustment to the current and alternative standards decreases estimated ambient O₃ concentrations compared to recent monitored concentrations (U.S. EPA, 2014a, section 4.3.3.2). In contrast, in locations and time periods when NO_x is predominantly contributing to O₃ titration (*e.g.*, in urban centers with high concentrations of NO_x emissions, where ambient O₃ concentrations are often suppressed and are thus relatively low), model-based adjustment increases ambient O₃ concentrations compared to recent monitored concentrations (U.S. EPA, 2014a, section 4.3.3.2). Changes in epidemiology-based risk estimates depend on the balance between the daily decreases in high O₃ concentrations and increases in low O₃ concentrations following the model-based air quality adjustment. Commenting on this issue, CASAC noted that “controls designed to reduce the peak levels of ozone (*e.g.*, the fourth-highest annual MDA8) may not be effective at reducing lower levels of ozone on more typical days and may actually increase ozone levels on days where ozone concentrations are low” (Frey 2014a, p. 2). CASAC further noted that risk results “suggest that the ozone-related health risks in the urban cores can increase for some of the cities as ozone NAAQS alternatives become more stringent. This is because reductions in nitrogen oxides emissions can lead to less scavenging of ozone and free radicals, resulting in locally higher levels of ozone” (Frey 2014c, p. 10).

⁷¹ For the 2009 adjusted year (*i.e.*, the year with generally lower O₃ concentrations), changes in risk were generally smaller than in 2007 (*i.e.*, most changes about 2% or smaller). Increases were estimated for Houston, Los Angeles, and New York City.

by more than 75% if a threshold exists at 40 ppb, and by about 98% if a threshold exists at 56 ppb) (U.S. EPA, 2014a, Figure 7–9).⁷²

Compared to the weight given to HREA estimates of exposures of concern and lung function risks, and the weight given to the evidence, the PA places relatively less weight on epidemiologic-based risk estimates. In doing so, the PA notes that the overall conclusions from the HREA likewise reflect less confidence in estimates of epidemiologic-based risks than in estimates of exposures and lung function risks. The determination to attach less weight to the epidemiologic-based estimates reflects the uncertainties associated with mortality and morbidity risk estimates, including the heterogeneity in effect estimates between locations, the potential for exposure measurement errors, and uncertainty in the interpretation of the shape of concentration-response functions at lower O₃ concentrations (U.S. EPA, 2014a, section 9.6).

Uncertainty in the shape of concentration-response functions at lower O₃ concentrations is particularly important to interpreting risk estimates given the approach used to adjust air quality to just meet the current standard, and potential alternative standards, and the resulting compression in the air quality distributions (*i.e.*, decreasing high concentrations and increasing low concentrations) (II.A.2.a, above). Total risk estimates in the HREA are based on the assumption that the concentration response function for O₃ is linear, such that total risk estimates are equally influenced by decreasing high concentrations and increasing low concentrations, when the increases and decreases are of equal magnitude. However, consistent with the PA’s consideration of risk estimates, in the proposal the Administrator notes that the overall body of evidence provides stronger support for the occurrence of

⁷² Risk estimates for respiratory mortality associated with long-term O₃ exposures are based on the study by Jerrett et al. (2009) (U.S. EPA, 2014a, Chapter 7). As discussed above (II.B.2.b.iv) and in the PA (U.S. EPA, 2014c, section 3.1.4.3), Jerrett et al. (2009) reported that when seasonal averages of 1-hour daily maximum O₃ concentrations ranged from 33 to 104 ppb, there was no statistical deviation from a linear concentration-response relationship between O₃ and respiratory mortality across 96 U.S. cities (U.S. EPA, 2013, section 7.7). However, the authors reported “limited evidence” for an effect threshold at an O₃ concentration of 56 ppb ($p=0.06$). In communications with EPA staff (Sasser, 2014), the study authors indicated that it is not clear whether a threshold model is a better predictor of respiratory mortality than the linear model, and that “considerable caution should be exercised in accepting any specific threshold.”

O₃-attributable health effects following exposures to O₃ concentrations corresponding to the upper ends of typical ambient distributions (II.E.4.d of the proposal). In addition, even on days with increases in relatively low area-wide average concentrations, resulting in increases in estimated risks, some portions of the urban study areas could experience decreases in high O₃ concentrations. Therefore, to the extent adverse O₃-attributable effects are more strongly supported for higher ambient concentrations (which, as noted above, are consistently reduced upon air quality adjustment), the PA notes that the impacts on risk estimates of increasing low O₃ concentrations reflect an important source of uncertainty.

c. PA Conclusions on the Current Standard

Section II.D.3 of the proposal summarizes the PA conclusions on the adequacy of the existing primary O₃ standard (79 FR 75285). As an initial matter, the PA concludes that reducing precursor emissions to achieve O₃ concentrations that meet the current standard will provide important improvements in public health protection. This initial conclusion is based on (1) the strong body of scientific evidence indicating a wide range of adverse health outcomes attributable to exposures to O₃ concentrations commonly found in the ambient air and (2) estimates indicating decreased occurrences of O₃ exposures of concern and decreased health risks upon meeting the current standard, compared to recent air quality.

In particular, the PA concludes that strong support for this initial conclusion is provided by controlled human exposure studies of respiratory effects, and by quantitative estimates of exposures of concern and lung function decrements based on information in these studies. Analyses in the HREA estimate that the percentages of children (*i.e.*, all children and children with asthma) in urban study areas experiencing exposures of concern, or experiencing abnormal and potentially adverse lung function decrements, are consistently lower for air quality that just meets the current O₃ standard than for recent air quality. The HREA estimates such reductions consistently across the urban study areas evaluated and throughout various portions of individual urban study areas, including in urban cores and the portions of urban study areas surrounding urban cores. These reductions in exposures of concern and O₃-induced lung function decrements reflect the consistent decreases in the highest O₃

concentrations following reductions in precursor emissions to meet the current standard. Thus, populations in both urban and non-urban areas would be expected to experience important reductions in O₃ exposures and O₃-induced lung function risks upon meeting the current standard.

The PA further concludes that support for this initial conclusion is also provided by estimates of O₃-associated mortality and morbidity based on application of concentration-response relationships from epidemiologic studies to air quality adjusted to just meet the current standard. These estimates are based on the assumption that concentration-response relationships are linear over entire distributions of ambient O₃ concentrations, an assumption which has uncertainties that complicate interpretation of these estimates (II.A.2.c.ii). However, risk estimates for effects associated with short- and long-term O₃ exposures, combined with the HREA's national analysis of O₃ responsiveness to reductions in precursor emissions and the consistent reductions estimated for the highest ambient O₃ concentrations, suggest that O₃-associated mortality and morbidity would be expected to decrease nationwide following reductions in precursor emissions to meet the current O₃ standard.

After reaching the initial conclusion that meeting the current primary O₃ standard will provide important improvements in public health protection, and that it is not appropriate to consider a standard that is less protective than the current standard, the PA considers the adequacy of the public health protection that is provided by the current standard. In considering the available scientific evidence, exposure/risk information, advice from CASAC (II.B.1.d, below), and input from the public, the PA reaches the conclusion that the available evidence and information clearly call into question the adequacy of public health protection provided by the current primary standard. In reaching this conclusion, the PA notes that evidence from controlled human exposure studies provides strong support for the occurrence of adverse respiratory effects following exposures to O₃ concentrations below the level of the current standard. Epidemiologic studies provide support for the occurrence of adverse respiratory effects and mortality under air quality conditions that would likely meet the current standard. In addition, based on the analyses in the HREA, the PA concludes that the exposures and risks projected to remain

upon meeting the current standard are indicative of risks that can reasonably be judged to be important from a public health perspective. Thus, the PA concludes that the evidence and information provide strong support for giving consideration to revising the current primary standard in order to provide increased public health protection against an array of adverse health effects that range from decreased lung function and respiratory symptoms to more serious indicators of morbidity (*e.g.*, including emergency department visits and hospital admissions), and mortality. In consideration of all of the above, the PA draws the conclusion that it is appropriate for the Administrator to consider revision of the current primary O₃ standard to provide increased public health protection.

d. CASAC Advice

Section II.D.4 of the proposal summarizes CASAC advice regarding the adequacy of the existing primary O₃ standard. Following the 2008 decision to revise the primary O₃ standard by setting the level at 0.075 ppm (75 ppb), CASAC strongly questioned whether the standard met the requirements of the CAA. In September 2009, the EPA announced its intention to reconsider the 2008 standards, issuing a notice of proposed rulemaking in January 2010 (75 FR 2938). Soon after, the EPA solicited CASAC review of that proposed rule and in January 2011, solicited additional advice. This proposal was based on the scientific and technical record from the 2008 rulemaking, including public comments and CASAC advice and recommendations. As further described above (I.D), in the fall of 2011, the EPA did not revise the standard as part of the reconsideration process but decided to defer decisions on revisions to the O₃ standards to the next periodic review, which was already underway. Accordingly, in this section we describe CASAC's advice related to the 2008 final decision and the subsequent reconsideration, as well as its advice on this current review of the O₃ NAAQS that was initiated in September 2008.

In April 2008, the members of the CASAC Ozone Review Panel sent a letter to EPA stating “[I]n our most-recent letters to you on this subject—dated October 2006 and March 2007—the CASAC unanimously recommended selection of an 8-hour average Ozone NAAQS within the range of 0.060 to 0.070 parts per million [60 to 70 ppb] for the primary (human health-based) Ozone NAAQS” (Henderson, 2008). In 2010, in response to the EPA's solicitation of advice on the EPA's

proposed rulemaking as part of the reconsideration, CASAC again stated that the current standard should be revised to provide additional protection to the public health (Samet, 2010):

CASAC fully supports EPA's proposed range of 0.060–0.070 parts per million (ppm) for the 8-hour primary ozone standard. CASAC considers this range to be justified by the scientific evidence as presented in the Air Quality Criteria for Ozone and Related Photochemical Oxidants (March 2006) and Review of the National Ambient Air Quality Standards for Ozone: Policy Assessment of Scientific and Technical Information, OAQPS Staff Paper (July 2007). As stated in our letters of October 24, 2006, March 26, 2007 and April 7, 2008 to former Administrator Stephen L. Johnson, CASAC unanimously recommended selection of an 8-hour average ozone NAAQS within the range proposed by EPA (0.060 to 0.070 ppm). In proposing this range, EPA has recognized the large body of data and risk analyses demonstrating that retention of the current standard would leave large numbers of individuals at risk for respiratory effects and/or other significant health impacts including asthma exacerbations, emergency room visits, hospital admissions and mortality.

In response to the EPA's request for additional advice on the reconsideration in 2011, CASAC reaffirmed their conclusion that "the evidence from controlled human and epidemiological studies strongly supports the selection of a new primary ozone standard within the 60–70 ppb range for an 8-hour averaging time" (Samet, 2011, p. ii). As requested by the EPA, CASAC's advice and recommendations were based on the scientific and technical record from the 2008 rulemaking. In considering the record for the 2008 rulemaking, CASAC stated the following to summarize the basis for their conclusions (Samet, 2011, p. ii to iii):

(1) The evidence available on dose-response for effects of O₃ shows associations extending to levels within the range of concentrations currently experienced in the United States.

(2) There is scientific certainty that 6.6-hour exposures with exercise of young, healthy, non-smoking adult volunteers to concentrations ≥80 ppb cause clinically relevant decrements of lung function.

(3) Some healthy individuals have been shown to have clinically relevant responses, even at 60 ppb.

(4) Since the majority of clinical studies involve young, healthy adult populations, less is known about health effects in such potentially ozone sensitive populations as the elderly, children and those with cardiopulmonary disease. For these susceptible groups, decrements in lung function may be greater than in healthy

volunteers and are likely to have a greater clinical significance.

(5) Children and adults with asthma are at increased risk of acute exacerbations on or shortly after days when elevated O₃ concentrations occur, even when exposures do not exceed the NAAQS concentration of 75 ppb.

(6) Large segments of the population fall into what the EPA terms a "sensitive population group," *i.e.*, those at increased risk because they are more intrinsically susceptible (children, the elderly, and individuals with chronic lung disease) and those who are more vulnerable due to increased exposure because they work outside or live in areas that are more polluted than the mean levels in their communities.

With respect to evidence from epidemiologic studies, CASAC stated "while epidemiological studies are inherently more uncertain as exposures and risk estimates decrease (due to the greater potential for biases to dominate small effect estimates), specific evidence in the literature does not suggest that our confidence on the specific attribution of the estimated effects of ozone on health outcomes differs over the proposed range of 60–70 ppb" (Samet, 2011, p. 10).

Following its review of the second draft PA in the current review, which considers an updated scientific and technical record since the 2008 rulemaking, CASAC concluded that "there is clear scientific support for the need to revise the standard" (Frey, 2014c, p. ii). In particular, CASAC noted the following (Frey, 2014c, p. 5):

[T]he scientific evidence provides strong support for the occurrence of a range of adverse respiratory effects and mortality under air quality conditions that would meet the current standard. Therefore, CASAC unanimously recommends that the Administrator revise the current primary ozone standard to protect public health.⁷³

In supporting these conclusions, CASAC judged that the strongest evidence comes from controlled human exposure studies of respiratory effects. The Committee specifically noted that "the combination of decrements in FEV₁ together with the statistically significant alterations in symptoms in human subjects exposed to 72 ppb ozone meets the American Thoracic Society's definition of an adverse health effect" (Frey, 2014c, p. 5). CASAC further judged that "if subjects had been exposed to ozone using the 8-hour

averaging period used in the standard, adverse effects could have occurred at lower concentration" and that "the level at which adverse effects might be observed would likely be lower for more sensitive subgroups, such as those with asthma" (Frey, 2014c, p. 5). With regard to 60 ppb exposures, CASAC noted that "a level of 60 ppb corresponds to the lowest exposure concentration demonstrated to result in lung function decrements large enough to be judged an abnormal response by ATS and that could be adverse in individuals with lung disease" (Frey, 2014c, p. 7). The CASAC further noted that "a level of 60 ppb also corresponds to the lowest exposure concentration at which pulmonary inflammation has been reported" (Frey, 2014c, p. 7).

In their advice, CASAC also took note of estimates of O₃ exposures of concern and the risk of O₃-induced lung function decrements. With regard to the benchmark concentrations used in estimating exposures of concern, CASAC stated the following (Frey, 2014c, p. 6):

The 80 ppb-8hr benchmark level represents an exposure level for which there is substantial clinical evidence demonstrating a range of ozone-related effects including lung inflammation and airway responsiveness in healthy individuals. The 70 ppb-8hr benchmark level reflects the fact that in healthy subjects, decreases in lung function and respiratory symptoms occur at concentrations as low as 72 ppb and that these effects almost certainly occur in some people, including asthmatics and others with low lung function who are less tolerant of such effects, at levels of 70 ppb and below. The 60 ppb-8hr benchmark level represents the lowest exposure level at which ozone-related effects have been observed in clinical studies of healthy individuals. Based on its scientific judgment, the CASAC finds that the 60 ppb-8hr exposure benchmark is relevant for consideration with respect to adverse effects on asthmatics.

With regard to lung function risk estimates, CASAC concluded that "estimation of FEV₁ decrements of ≥15% is appropriate as a scientifically relevant surrogate for adverse health outcomes in active healthy adults, whereas an FEV₁ decrement of ≥10% is a scientifically relevant surrogate for adverse health outcomes for people with asthma and lung disease" (Frey, 2014c, p. 3). The Committee further concluded that "[a]sthmatic subjects appear to be at least as sensitive, if not more sensitive, than non-asthmatic subjects in manifesting O₃-induced pulmonary function decrements" (Frey, 2014c, p. 4).

Although CASAC judged that controlled human exposure studies of respiratory effects provide the strongest

⁷³ CASAC provided similar advice in their letter to the Administrator on the HREA, stating that "The CASAC finds that the current primary NAAQS for ozone is not protective of human health and needs to be revised" (Frey, 2014a, p. 15).

evidence supporting their conclusion on the current standard, the Committee judged that there is also “sufficient scientific evidence based on epidemiologic studies for mortality and morbidity associated with short-term exposure to ozone at the level of the current standard” (Frey, 2014c, p. 5) and noted that “[r]ecent animal toxicological studies support identification of modes of action and, therefore, the biological plausibility associated with the epidemiological findings” (Frey, 2014c, p. 5).

e. Administrator’s Proposed Decision

Section II.D.5 in the proposal (79 FR 75287–75291) discusses the Administrator’s proposed conclusions related to the adequacy of the public health protection provided by the current primary O₃ standard, resulting in her proposed decision to revise that standard. These proposed conclusions and her proposed decision, summarized below, were based on the Administrator’s consideration of the available scientific evidence, exposure/risk information, the comments and advice of CASAC, and public input that had been received by the time of proposal.

As an initial matter, the Administrator concluded that reducing precursor emissions to achieve O₃ concentrations that meet the current primary O₃ standard will provide important improvements in public health protection, compared to recent air quality. In reaching this initial conclusion, she noted the discussion in section 3.4 of the PA (U.S. EPA, 2014c). In particular, the Administrator noted that this initial conclusion is supported by (1) the strong body of scientific evidence indicating a wide range of adverse health outcomes attributable to exposures to O₃ concentrations commonly measured in the ambient air and (2) estimates indicating decreased occurrences of O₃ exposures of concern and decreased O₃-associated health risks upon meeting the current standard, compared to recent air quality. Thus, she concluded that it would not be appropriate in this review to consider a standard that is less protective than the current standard.⁷⁴

⁷⁴ Although the Administrator noted that reductions in O₃ precursor emissions (e.g., NO_x; VOC) to achieve O₃ concentrations that meet the current standard could also increase public health protection by reducing the ambient concentrations of pollutants other than O₃ (e.g., PM_{2.5}, NO₂), we did not quantitatively analyze these effects, consistent with CASAC advice (Frey, 2014a, p.10). However, the Administrator is not setting the standard to address risks from pollutants other than O₃.

After reaching the initial conclusion that meeting the current primary O₃ standard will provide important improvements in public health protection, and that it is not appropriate to consider a standard that is less protective than the current standard, the Administrator next considered the adequacy of the public health protection that is provided by the current standard. In doing so, the Administrator first noted that studies evaluated since the completion of the 2006 AQCD support and expand upon the strong body of evidence that, in the last review, indicated a causal relationship between short-term O₃ exposures and respiratory health effects, the strongest determination under the ISA’s hierarchical system for classifying weight of evidence for causation. Together, experimental and epidemiologic studies support conclusions regarding a continuum of O₃ respiratory effects ranging from small reversible changes in pulmonary function, and pulmonary inflammation, to more serious effects that can result in respiratory-related emergency department visits, hospital admissions, and premature mortality. The Administrator further noted that recent animal toxicology studies support descriptions of modes of action for these respiratory effects and provide support for biological plausibility for the role of O₃ in reported effects. With regard to mode of action, evidence indicates that antioxidant capacity may modify the risk of respiratory morbidity associated with O₃ exposure, and that the inherent capacity to quench (based on individual antioxidant capacity) can be overwhelmed, especially with exposure to elevated concentrations of O₃. In addition, based on the consistency of findings across studies and evidence for the coherence of results from different scientific disciplines, evidence indicates that certain populations are at increased risk of experiencing O₃-related effects, including the most severe effects. These include populations and lifestages identified in previous reviews (*i.e.*, people with asthma, children, older adults, outdoor workers) and populations identified since the last review (*i.e.*, people with certain genotypes related to antioxidant and/or anti-inflammatory status; people with reduced intake of certain antioxidant nutrients, such as Vitamins C and E).

The Administrator further noted that evidence for adverse respiratory health effects attributable to long-term⁷⁵ O₃

⁷⁵ Based on the exposure surrogates used in recent epidemiologic studies of long-term O₃ exposure, it is not possible to distinguish between

exposures is much stronger than in previous reviews, and noted the ISA’s conclusion that there is “likely to be” a causal relationship between such O₃ exposures and adverse respiratory health effects (the second strongest causality determination). She noted that the evidence available in this review includes new epidemiologic studies using a variety of designs and analysis methods, conducted by different research groups in different locations, evaluating the relationships between long-term O₃ exposures and measures of respiratory morbidity and mortality. New evidence supports associations between long-term O₃ exposures and the development of asthma in children, with several studies reporting interactions between genetic variants and such O₃ exposures. Studies also report associations between long-term O₃ exposures and asthma prevalence, asthma severity and control, respiratory symptoms among asthmatics, and respiratory mortality.

In considering the O₃ exposure concentrations reported to elicit respiratory effects, the Administrator agreed with the conclusions of the PA and with the advice of CASAC (Frey, 2014c) that controlled human exposure studies provide the most certain evidence indicating the occurrence of health effects in humans following exposures to specific O₃ concentrations. In particular, she noted that the effects reported in controlled human exposure studies are due solely to O₃ exposures, and interpretation of study results is not complicated by the presence of co-occurring pollutants or pollutant mixtures.

In considering the evidence from controlled human exposure studies, the Administrator first noted that these studies have reported a variety of respiratory effects in healthy adults following exposures to O₃ concentrations of 60, 72, or 80 ppb, and higher. The largest respiratory effects, and the broadest range of effects, have been studied and reported following exposures of healthy adults to 80 ppb O₃ or higher, with most exposure studies conducted at these higher concentrations. She further noted that recent evidence includes controlled human exposure studies reporting the combination of lung function decrements and respiratory symptoms in healthy adults engaged in quasi-continuous, moderate exertion following 6.6 hour exposures to concentrations as low as 72 ppb, and lung function decrements and

the impacts of long-term O₃ exposure and exposure to repeated short-term peaks over an O₃ season.

pulmonary inflammation following exposures to O₃ concentrations as low as 60 ppb. As discussed below, compared to the evidence available in the last review, the Administrator viewed these studies as having strengthened support for the occurrence of abnormal and adverse respiratory effects attributable to short-term exposures to O₃ concentrations below the level of the current standard. The Administrator stated that such exposures to O₃ concentrations below the level of the current standard are potentially important from a public health perspective, given the following:

(1) The combination of lung function decrements and respiratory symptoms reported to occur in healthy adults following exposures to 72 ppb O₃ or higher, while at moderate exertion, meet ATS criteria for an adverse response. In specifically considering the 72 ppb exposure concentration, CASAC noted that “the combination of decrements in FEV₁ together with the statistically significant alterations in symptoms in human subjects exposed to 72 ppb ozone meets the American Thoracic Society’s definition of an adverse health effect” (Frey, 2014c, p. 5).

(2) With regard to 60 ppb O₃, CASAC agreed that “a level of 60 ppb corresponds to the lowest exposure concentration demonstrated to result in lung function decrements large enough to be judged an abnormal response by ATS and that could be adverse in individuals with lung disease” (Frey, 2014c, p. 7). CASAC further noted that “a level of 60 ppb also corresponds to the lowest exposure concentration at which pulmonary inflammation has been reported” (Frey, 2014c, p. 7).

(3) The controlled human exposure studies reporting these respiratory effects were conducted in healthy adults, while at-risk groups (*e.g.*, children, people with asthma) could experience larger and/or more serious effects. In their advice to the Administrator, CASAC concurred with this reasoning (Frey, 2014a, p. 14; Frey, 2014c, p. 5).

(4) These respiratory effects are coherent with the serious health outcomes that have been reported in epidemiologic studies evaluating exposure to O₃ (*e.g.*, respiratory-related hospital admissions, emergency department visits, and mortality).

As noted above, the Administrator’s proposed conclusions regarding the adequacy of the current primary O₃ standard placed a large amount of weight on the results of controlled human exposure studies. In particular, given the combination of lung function decrements and respiratory symptoms

following 6.6-hour exposures to O₃ concentrations as low as 72 ppb, and given CASAC advice regarding effects at 72 ppb, along with ATS adversity criteria, she concluded that the evidence in this review supports the occurrence of adverse respiratory effects following exposures to O₃ concentrations lower than the level of the current standard.⁷⁶ As discussed below, the Administrator further considered information from the broader body of controlled human exposure studies within the context of quantitative estimates of exposures of concern and O₃-induced FEV₁ decrements.

While putting less weight on information from epidemiologic studies than on information from controlled human exposure studies, the Administrator also considered what the available epidemiologic evidence indicates with regard to the adequacy of the public health protection provided by the current primary O₃ standard. She noted that recent epidemiologic studies provide support, beyond that available in the last review, for associations between short-term O₃ exposures and a wide range of adverse respiratory outcomes (including respiratory-related hospital admissions, emergency department visits, and mortality) and with total mortality. Associations with morbidity and mortality are stronger during the warm or summer months, and remain robust after adjustment for copollutants.

In considering information from epidemiologic studies within the context of her conclusions on the adequacy of the current standard, the Administrator considered the extent to which available studies support the occurrence of O₃ health effect associations with air quality likely to be allowed by the current standard. Most of the epidemiologic studies considered by the Administrator were conducted in locations likely to have violated the current standard over at least part of the study period. However, she noted three U.S. single-city studies that support the occurrence of O₃-associated hospital admissions or emergency department visits at ambient O₃ concentrations below the level of the current standard, or when virtually all monitored concentrations were below the level of the current standard (Mar and Koenig, 2009; Silverman and Ito, 2010; Strickland et al., 2010) (section II.D.1 of the proposal). While the Administrator acknowledged greater uncertainty in interpreting air quality for multicity

studies, she noted that O₃ associations with respiratory morbidity or mortality have been reported when the majority of study locations (though not all study locations) would likely have met the current O₃ standard. When taken together, the Administrator reached the initial conclusion at proposal that single-city epidemiologic studies and associated air quality information support the occurrence of O₃-associated hospital admissions and emergency department visits for ambient O₃ concentrations likely to have met the current standard, and that air quality analyses in locations of multicity studies provide some support for this conclusion for a broader range of effects, including mortality.

Beyond her consideration of the scientific evidence, the Administrator also considered the results of the HREA exposure and risk analyses in reaching initial conclusions regarding the adequacy of the current primary O₃ standard. In doing so, as noted above, she focused primarily on exposure and risk estimates based on information from controlled human exposure studies (*i.e.*, exposures of concern and O₃-induced lung function decrements) and placed relatively less weight on epidemiologic-based risk estimates.

With regard to estimates of exposures of concern, the Administrator considered the extent to which the current standard provides protection against exposures to O₃ concentrations at or above 60, 70, and 80 ppb. Consistent with CASAC advice (Frey, 2014c), the Administrator focused on children in these analyses of O₃ exposures, noting that estimates for all children and asthmatic children are virtually indistinguishable, in terms of the percent estimated to experience exposures of concern.⁷⁷ Though she focused on children, she also recognized that exposures to O₃ concentrations at or above 60 or 70 ppb could be of concern for adults. As discussed in the HREA and PA (and II.C.2.a of the proposal), the patterns of exposure estimates across urban study areas, across years, and across air quality scenarios are similar in adults with asthma, older adults, all children, and children with asthma, though smaller percentages of adult populations are estimated to experience exposures of concern than children and children with asthma. Thus, the Administrator recognized that the exposure patterns for children across years, urban study areas, and air

⁷⁶ This CASAC advice and ATS recommendations are discussed in more detail in section II.C.4 below (see also II.A.1.c, above).

⁷⁷ As noted above, HREA analyses indicate that activity data for asthmatics is generally similar to non-asthmatics (U.S. EPA, 2014a, Appendix 5G, Tables 5G2-to 5G-5).

quality scenarios are indicative of the exposure patterns in a broader group of at-risk populations that also includes asthmatic adults and older adults.

She further noted that while single exposures of concern could be adverse for some people, particularly for the higher benchmark concentrations (70, 80 ppb) where there is stronger evidence for the occurrence of adverse effects, she became increasingly concerned about the potential for adverse responses as the number of occurrences increases (61 FR 75122).⁷⁸ In particular, she noted that repeated occurrences of the types of effects shown to occur following exposures of concern can have potentially adverse outcomes. For example, repeated occurrences of airway inflammation could potentially result in the induction of a chronic inflammatory state; altered pulmonary structure and function, leading to diseases such as asthma; altered lung host defense response to inhaled microorganisms; and altered lung response to other agents such as allergens or toxins (U.S. EPA, 2013, section 6.2.3). Thus, the Administrator noted that the types of respiratory effects shown to occur in some individuals following exposures to O₃ concentrations from 60 to 80 ppb, particularly if experienced repeatedly, provide a mode of action by which O₃ may cause other more serious effects (e.g., asthma exacerbations). Therefore, the Administrator placed the most weight on estimates of two or more exposures of concern (i.e., as a surrogate for the occurrence of repeated exposures), though she also considered estimates of one or more, particularly for the 70 and 80 ppb benchmarks.⁷⁹

As illustrated in Table 1 (above), the Administrator noted that if the 15 urban study areas evaluated in the HREA were to just meet the current O₃ standard, fewer than 1% of children in those areas would be estimated to experience two or more exposures of concern at or above 70 ppb, though approximately 3 to 8% of children, including approximately 3 to 8% of asthmatic children, would be

estimated to experience two or more exposures of concern to O₃ concentrations at or above 60 ppb⁸⁰ (based on estimates averaged over the years of analysis). To provide some perspective on these percentages, the Administrator noted that they correspond to almost 900,000 children in urban study areas, including about 90,000 asthmatic children, estimated to experience two or more exposures of concern at or above 60 ppb. Nationally, if the current standard were to be just met, the number of children experiencing such exposures would be larger. In the worst-case year and location (i.e., year and location with the largest exposure estimates), the Administrator noted that over 2% of children are estimated to experience two or more exposures of concern at or above 70 ppb and over 14% are estimated to experience two or more exposures of concern at or above 60 ppb.

Although, as discussed above and in section II.E.4.d of the proposal, the Administrator was less concerned about single occurrences of exposures of concern, she noted that even single occurrences can cause adverse effects in some people, particularly for the 70 and 80 ppb benchmarks. Therefore, she also considered estimates of one or more exposures of concern. As illustrated in Table 1 (above), if the 15 urban study areas evaluated in the HREA were to just meet the current O₃ standard, fewer than 1% of children in those areas would be estimated to experience one or more exposures of concern at or above 80 ppb (based on estimates averaged over the years of analysis). However, approximately 1 to 3% of children, including 1 to 3% of asthmatic children, would be estimated to experience one or more exposures of concern to O₃ concentrations at or above 70 ppb and approximately 10 to 17% would be estimated to experience one or more exposures of concern to O₃ concentrations at or above 60 ppb. In the worst-case year and location, the Administrator noted that over 1% of children are estimated to experience one or more exposures of concern at or above 80 ppb, over 8% are estimated to experience one or more exposures of concern at or above 70 ppb, and about 26% are estimated to experience one or more exposures of concern at or above 60 ppb.

In addition to estimated exposures of concern, the Administrator also considered HREA estimates of the

occurrence of O₃-induced lung function decrements. In doing so, she particularly noted CASAC advice that “estimation of FEV₁ decrements of ≥15% is appropriate as a scientifically relevant surrogate for adverse health outcomes in active healthy adults, whereas an FEV₁ decrement of ≥10% is a scientifically relevant surrogate for adverse health outcomes for people with asthma and lung disease” (Frey, 2014c, p. 3). While these surrogates provide perspective on the potential for the occurrence of adverse respiratory effects following O₃ exposures, the Administrator agreed with the conclusion in past reviews that a more general consensus view of the adversity of moderate responses emerges as the frequency of occurrence increases (citing to 61 FR 65722–3) (Dec, 13, 1996). Therefore, in the proposal the Administrator expressed increasing concern about the potential for adversity as the frequency of occurrences increased and, as a result, she focused primarily on estimates of two or more O₃-induced FEV₁ decrements (i.e., as a surrogate for repeated exposures).

When averaged over the years evaluated in the HREA, the Administrator noted that the current standard is estimated to allow about 1 to 3% of children in the 15 urban study areas (corresponding to almost 400,000 children) to experience two or more O₃-induced lung function decrements ≥15%, and to allow about 8 to 12% of children (corresponding to about 180,000 asthmatic children) to experience two or more O₃-induced lung function decrements ≥10%. Nationally, larger numbers of children would be expected to experience such O₃-induced decrements if the current standard were to be just met. The current standard is also estimated to allow about 3 to 5% of children in the urban study areas to experience one or more decrements ≥15% and about 14 to 19% of children to experience one or more decrements ≥10%. In the worst-case year and location, the current standard is estimated to allow 4% of children in the urban study areas to experience two or more decrements ≥15% (and 7% to experience one or more such decrements) and 14% of children to experience two or more decrements ≥10% (and 22% to experience one or more such decrements).⁸¹

⁸¹ As discussed below (II.C.4), in her consideration of potential alternative standard levels, the Administrator placed less weight on estimates of the risk of O₃-induced FEV₁ decrements. In doing so, she particularly noted that, unlike exposures of concern, the variability in lung

Continued

⁷⁸ The Administrator noted that not all people who experience an exposure of concern will experience an adverse effect (even members of at-risk populations). For most of the endpoints evaluated in controlled human exposure studies (with the exception of O₃-induced FEV₁ decrements, as discussed below), the number of those experiencing exposures of concern who will experience adverse effects cannot be reliably quantified.

⁷⁹ The Administrator’s considerations related to estimated O₃ exposures of concern, including her views on estimates of two or more and one or more such exposures, are discussed in more detail within the context of her consideration of public comments on the level of the revised standard and her final decision on level (II.C.4.b and II.C.4.c, below).

⁸⁰ Almost no children in those areas would be estimated to experience two or more exposures of concern at or above 80 ppb.

In further considering the HREA results, the Administrator considered the epidemiology-based risk estimates. Compared to the weight given to HREA estimates of exposures of concern and lung function risks, she placed relatively less weight on epidemiology-based risk estimates. Consistent with the conclusions in the PA, her determination to attach less weight to the epidemiologic-based risk estimates reflected her consideration of key uncertainties, including the heterogeneity in effect estimates between locations, the potential for exposure measurement errors, and uncertainty in the interpretation of the shape of concentration-response functions for O₃ concentrations in the lower portions of ambient distributions (U.S. EPA, 2014a, section 9.6) (section II.D.2 of the proposal).

The Administrator focused on estimates of total mortality risk associated with short-term O₃ exposures.⁸² Given the decreasing certainty in the shape of concentration-response functions for area-wide O₃ concentrations at the lower ends of warm season distributions (U.S. EPA, 2013, section 2.5.4.4), the Administrator focused on estimates of risk associated with O₃ concentrations in the upper portions of ambient distributions. Even when considering only area-wide O₃ concentrations from these upper portions of seasonal distributions, the Administrator noted that the current standard is estimated to allow hundreds to thousands of O₃-associated deaths per year in urban study areas (79 FR 75291 citing to section II.C.3 of the proposal).

In addition to the evidence and exposure/risk information discussed above, the Administrator took note of the CASAC advice in the current review and in the 2010 proposed

function risk estimates across urban study areas is often greater than the differences in risk estimates between various standard levels (Table 2, above). Given this, and the resulting considerable overlap between the ranges of lung function risk estimates for different standard levels, although the Administrator noted her confidence in the lung function risk estimates themselves, she viewed them as providing a more limited basis than exposures of concern for distinguishing between the degree of public health protection provided by alternative standard levels.

⁸² In doing so, she concluded that lower confidence should be placed in the results of the assessment of respiratory mortality risks associated with long-term O₃ exposures, primarily because that analysis is based on only one study (even though that study is well-designed) and because of the uncertainty in that study about the existence and identification of a potential threshold in the concentration-response function (U.S. EPA, 2014a, section 9.6) (section II.D.2 of the proposal). CASAC also called into question the extent to which it is appropriate to place confidence in risk estimates for respiratory mortality (Frey, 2014a, p. 11).

reconsideration of the 2008 decision establishing the current standard. As discussed in more detail above, the current CASAC “finds that the current NAAQS for ozone is not protective of human health” and “unanimously recommends that the Administrator revise the current primary ozone standard to protect public health” (Frey, 2014c, p. 5).

In consideration of all of the above, the Administrator proposed that the current primary O₃ standard is not adequate to protect public health, and that it should be revised to provide increased public health protection. This proposed decision was based on the Administrator’s initial conclusions that the available evidence and exposure and risk information clearly call into question the adequacy of public health protection provided by the current primary standard and, therefore, that the current standard is not requisite to protect public health with an adequate margin of safety. With regard to the evidence, she specifically noted that (1) controlled human exposure studies provide support for the occurrence of adverse respiratory effects following exposures to O₃ concentrations below the level of the current standard (*i.e.*, as low as 72 ppb), and that (2) single-city epidemiologic studies provide support for the occurrence of adverse respiratory effects under air quality conditions that would likely meet the current standard, with multicity studies providing limited support for this conclusion for a broader range of effects (*i.e.*, including mortality). In addition, based on the analyses in the HREA, the Administrator concluded that the exposures and risks projected to remain upon meeting the current standard can reasonably be judged to be important from a public health perspective. Thus, she reached the proposed conclusion that the evidence and information, together with CASAC advice based on their consideration of that evidence and information, provide strong support for revising the current primary standard in order to increase public health protection against an array of adverse effects that range from decreased lung function and respiratory symptoms to more serious indicators of morbidity (*e.g.*, including emergency department visits and hospital admissions), and mortality.

2. Comments on the Need for Revision

The EPA received a large number of comments, more than 430,000 comments, on the proposed decision to revise the current primary O₃ standard. These comments generally fell into one

of two broad groups that expressed sharply divergent views.

Many commenters asserted that the current primary O₃ standard is not sufficient to protect public health, especially the health of sensitive groups, with an adequate margin of safety. These commenters agreed with the EPA’s proposed decision to revise the current standard to increase public health protection. Among those calling for revisions to the current primary standard were medical groups (*e.g.*, American Academy of Pediatrics (AAP), American Medical Association, American Lung Association (ALA), American Thoracic Society, American Heart Association, and the American College of Occupational and Environmental Medicine); national, state, and local public health and environmental organizations (*e.g.*, the National Association of County and City Health Officials, American Public Health Association, Physicians for Social Responsibility, Sierra Club, Natural Resources Defense Council, Environmental Defense Fund, Center for Biological Diversity, and Earthjustice); the majority of state and local air pollution control authorities that submitted comments (*e.g.*, agencies from California Air Resources Board and Office of Environmental Health Hazard Assessment, Connecticut, Delaware, Iowa, Illinois, Maryland, Minnesota, New Hampshire, New York, North Dakota, Oregon, Pennsylvania, Tennessee, and Wisconsin); the National Tribal Air Association; State organizations (*e.g.*, National Association of Clean Air Agencies (NACAA), Northeast States for Coordinated Air Use Management, Ozone Transport Commission). While all of these commenters agreed with the EPA that the current O₃ standard needs to be revised, many supported a more protective standard than proposed by EPA, as discussed in more detail below (II.C.4). Many individual commenters also expressed similar views.

A second group of commenters, representing industry associations, businesses and some state agencies, opposed the proposed decision to revise the current primary O₃ standard, expressing the view that the current standard is adequate to protect public health, including the health of sensitive groups, and to do so with an adequate margin of safety. Industry and business groups expressing this view included the American Petroleum Institute (API), the Alliance of Automobile Manufacturers (AAM), the American Forest and Paper Association, the Dow Chemical Company, the National Association of Manufacturers, the

National Mining Association, the U.S. Chamber of Commerce (in a joint comment with other industry groups), and the Utility Air Regulatory Group (UARG). State environmental agencies opposed to revising the current primary O₃ standard included agencies from Arkansas, Georgia, Louisiana, Kansas, Michigan, Mississippi, Nebraska, North Carolina, Ohio, Texas, Virginia, and West Virginia.

The following sections discuss comments submitted by these and other groups, and the EPA's responses to those comments. Comments dealing with overarching issues that are fundamental to EPA's decision-making methodology are addressed in section II.B.2.a. Comments on the health effects evidence, including evidence from controlled human exposure and epidemiologic studies, are addressed in section II.B.2.b. Comments on human exposure and health risk assessments are addressed in section II.B.2.c. Comments on the appropriate indicator, averaging time, form, or level of a revised primary O₃ standard are addressed below in section II.C. In addition to the comments addressed in this preamble, the EPA has prepared a Response to Comments document that addresses other specific comments related to standard setting, as well as comments on implementation- and/or cost-related factors that the EPA may not consider as part of the basis for decisions on the NAAQS. This document is available for review in the docket for this rulemaking and through the EPA's OAQPS TTN Web site (http://www.epa.gov/ttn/naqs/standards/ozone/s_o3_index.html).

a. Overarching Comments

Some commenters maintained that the proposed rule (and by extension the final rule) is fundamentally flawed because it does not quantify, or otherwise define, what level of protection is "requisite" to protect the public health. These commenters asserted that "EPA has not explained how far above zero-risk it believes is appropriate or how close to background is acceptable. EPA has failed to explain how the current standard is inadequate on this specific basis" (e.g., UARG, p. 10). These commenters further maintained that the failure to quantify a requisite level of protection "drastically reduces the value of public participation" since "the public does not understand what is driving EPA's decision" (e.g., UARG, p. 11).

The EPA disagrees with these comments and notes that industry petitioners made virtually the same argument before the D.C. Circuit in *ATA*

III, on remand from the Supreme Court, arguing that unless EPA identifies and quantifies a degree of acceptable risk, it is impossible to determine if a NAAQS is requisite (i.e., neither too stringent or insufficiently stringent to protect the public health). The D.C. Circuit rejected petitioners' argument, holding that "[a]lthough we recognize that the Clean Air Act and circuit precedent require EPA qualitatively to describe the standard governing its selection of particular NAAQS, we have expressly rejected the notion that the Agency must 'establish a measure of the risk to safety it considers adequate to protect public health every time it establish a [NAAQS]'" *ATA III*, 283 F. 3d at 369 (quoting *NRDC v. EPA*, 902 F.2d 962, 973 (D.C. Cir. 1990)). The court went on to explain that the requirement is only for EPA to engage in reasoned decision-making, "not that it definitively identify pollutant levels below which risks to public health are negligible." *ATA III*, 283 F. 3d at 370.

Thus, the Administrator is required to exercise her judgment in the face of scientific uncertainty to establish the NAAQS to provide appropriate protection against risks to public health, both known and unknown. As discussed below, in the current review, the Administrator judges that the existing primary O₃ standard is not requisite to protect public health with an adequate margin of safety, a judgment that is consistent with CASAC's conclusion that "there is clear scientific support for the need to revise the standard" (Frey, 2014c, p. ii). Further, in section II.C.4 below, the Administrator has provided a thorough explanation of her rationale for concluding that a standard with a level of 70 ppb is requisite to protect public health with an adequate margin of safety, explaining the various scientific uncertainties which circumscribe the range of potential alternative standards, and how she exercised her "judgment" (per section 109 (b)(1) of the CAA) in selecting a standard from within that range of scientifically reasonable choices. This "reasoned decision making" is what the Act requires, 283 F. 3d at 370, not the quantification advocated by these commenters.

The EPA further disagrees with the comment that a failure to quantify a requisite level of protection impaired or impeded public notice and comment opportunities. In fact, the EPA clearly gave adequate notice of the bases both for determining that the current standard does not afford requisite

protection,⁸³ and for determining how the standard should be revised. In particular, the EPA explained in detail which evidence it considered critical, and the scientific uncertainties that could cause the Administrator to weight that evidence in various ways (79 FR 75308–75310). There were robust comments submitted by commenters from a range of viewpoints on all of these issues, an indication of the adequacy of notice. The public was also afforded multiple opportunities to comment to the EPA and to CASAC during the development of the ISA, REA, and PA. Thus, the EPA does not agree that lack of quantification of a risk level that is "requisite" has deprived commenters of adequate notice and opportunity to comment in this proceeding.

Various commenters maintained that it was inappropriate to revise the current NAAQS based on their view that natural background concentrations in several states are at or above O₃ concentrations associated with meeting a NAAQS set at a level less than 75 ppb (presumably retaining the same indicator, form, and averaging time), making the NAAQS impossible for those states to attain and maintain, a result they claim is legally impermissible. In support for their argument, the commenters cite monitoring and modelling results from various areas in the intermountain west, state that EPA analyses provide underestimates of background O₃ and conclude that high concentrations of background O₃⁸⁴ exist

⁸³ See 79 FR 75287–91 (noting, among other things, that exposure to ambient O₃ concentrations below the level of the current standard has been associated with diminished lung function capacity, respiratory symptoms, and respiratory health effects resulting in emergency room visits or hospital admissions, and that a single-city epidemiologic study showed associations with asthma emergency department visits in an area that would have met the current standard over the entire study period). See also Frey 2014c, p. 5 (CASAC reiterated its conclusion, after multiple public comment opportunities, that as a matter of science the current standard "is not protective of public health" and provided the bases for that conclusion).

⁸⁴ Background O₃ can be generically defined as the portion of O₃ in ambient air that comes from sources outside the jurisdiction of an area and can include natural sources as well as transported O₃ of anthropogenic origin. EPA has identified two specific definitions of background O₃ relevant to this discussion: natural background (NB) and United States background (USB). NB is defined as the O₃ that would exist in the absence of any manmade precursor emissions. USB is defined as that O₃ that would exist in the absence of any manmade emissions inside the U.S. This includes anthropogenic emissions outside the U.S. as well as naturally occurring ozone. In many cases, the comments reference background O₃ only in the generic sense. Unless explicitly noted otherwise, we have assumed all references to background in the comments are intended to refer to USB.

in many parts of the United States that will “prevent attainment” of a revised standard (NMA, p. 5).

The courts have clearly established that “[a]ttainability and technological feasibility are not relevant considerations in the promulgation of [NAAQS].” *API v. EPA*, 665 F. 2d 1176, 1185 (D.C. Cir. 1981). Further, the courts have clarified that the EPA may consider proximity to background concentrations as a factor in the decision whether and how to revise the NAAQS only in the context of considering standard levels within the range of reasonable values supported by the air quality criteria and judgments of the Administrator. 79 FR 75242–43 (citing *ATA III*, 283 F. 3d at 379). In this review, the overall body of scientific evidence and exposure/risk information, as discussed in Section II.B of this notice, is clear and convincing: The existing standard is not adequate to protect public health with an adequate margin of safety and that the standard needs to be revised to reflect a lower level to provide that protection. The EPA analyses indicate that there may be infrequent instances in a limited number of rural areas where background O₃ would be appreciable but not the sole contributor to an exceedance of the revised NAAQS, but do not indicate U.S. background (USB) O₃ concentrations will prevent attainment of a revised O₃ standard with a level of 70 ppb. USB is defined as that O₃ that would exist even in the absence of any manmade emissions within the United States.

The EPA’s estimates of U.S. background ozone concentrations are based on frequently-utilized, state-of-the-science air quality models and are considered reasonable and reliable, not underestimates. In support of their view, the commenters state that monitored (not modelled) ozone concentrations in remote rural locations include instances of 8-hour average concentrations very occasionally higher than 70 ppb. Monitoring data from places like the Grand Canyon and Yellowstone National Parks, are examples cited in comments. It is inappropriate to assume that monitored O₃ concentrations at remote sites can be used as a proxy for background O₃. Even at the most remote locations, local O₃ concentrations are impacted by anthropogenic emissions from within the U.S. The EPA modeling analyses (U.S. EPA, 2014c, Figure 2–18) estimate that, on a seasonal basis, 10–20% of the O₃ at even the most remote locations in the intermountain western U.S. originates from manmade emissions from the U.S., and thus is not part of

USB. This conclusion is supported by commenter-submitted recent data analyses of rural O₃ observations in Nevada and Utah (NMA, Appendices D and H). These analyses conclude that natural sources, international O₃ transport, O₃ transported from upwind states, and O₃ transported from urban areas within a state all contributed to O₃ concentrations at rural sites.⁸⁵ Thus, while O₃ in high-altitude, rural portions of the intermountain western U.S. can, at times, be substantially influenced by background sources such as wildfires, international transport or the stratosphere, measured O₃ in rural locations are also influenced by domestic emissions and so cannot, by themselves, be used to estimate USB concentrations. Accordingly, the fact that 2011–2013 design values in locations like Yellowstone National Park (66 ppb) or Grand Canyon National Park (72 ppb) approach or exceed 70 ppb, does not support the conclusion that a standard with a level of 70 ppb is impossible to attain.

To accurately estimate USB concentrations, it is necessary to use air quality models which can estimate how much of the O₃ at any given location originates from sources other than manmade emissions within the U.S. As part of the rulemaking, the EPA has summarized a variety of modeling-based analyses of background O₃ (U.S. EPA, 2013, Chapter 3) and conducted our own multi-model assessment of USB concentrations across the U.S. (U.S. EPA, 2014c, Chapter 2). The EPA analyses, which are consistent with the previously-summarized studies highlighted by commenters, concluded that seasonal mean daily maximum 8-hour average concentrations of USB O₃ range from 25–50 ppb, with the highest estimates located across the intermountain western U.S.

Importantly, the modeling analyses also indicate that the highest O₃ days (*i.e.*, the days most relevant to the form of the NAAQS) generally have similar daily maximum 8-hour average USB concentrations as the seasonal means of this metric, but have larger contributions from U.S. anthropogenic sources. As summarized in the PA, “the highest modeled O₃ site-days tend to have background O₃ levels similar to mid-range O₃ days . . . [T]he days with

⁸⁵ The analysis of observations in Utah notes the influence of domestic emissions—either from Salt Lake City (for two of the areas) or from Los Angeles and California (for the third of the areas)—on O₃ concentrations at each of the locations included (NMA comments, Appendix E). Additionally, the analysis of monitoring data for Nevada also describes the influence of the monitoring sites by domestic emissions from other western states (NMA, Appendix H).

highest O₃ levels have similar distributions (*i.e.* means, inter-quartile ranges) of background levels as days with lower values, down to approximately 40 ppb. As a result, the proportion of total O₃ that has background origins is smaller on high O₃ days (*e.g.* greater than 60 ppb) than on the more common lower O₃ days that tend to drive seasonal means” (U.S. EPA, 2014c, p. 2–21, emphasis added). When averaged over the entire U.S., the models estimate that the mean USB fractional contribution to daily maximum 8-hour average O₃ concentrations above 70 ppb is less than 35 percent. U.S. anthropogenic emission sources are thus the dominant contributor to the majority of modeled O₃ exceedances across the U.S. (U.S. EPA, 2014c, Figures 2–14 and 2–15).

As noted in the PA, and as highlighted by the commenters based on existing modeling, there can be infrequent events where daily maximum 8-hour O₃ concentrations approach or exceed 70 ppb largely due to the influence of USB sources like a wildfire or stratospheric intrusion. As discussed below in Section V, the statute and EPA implementing regulations allow for the exclusion of air quality monitoring data from design value calculations when there are exceedances caused by certain event-related U.S. background influences (*e.g.*, wildfires or stratospheric intrusions). As a result, these “exceptional events” will not factor into attainability concerns.

In sum, the EPA believes that the commenters have failed to establish the predicate for their argument. Uncontrollable background concentrations of O₃ are not expected to preclude attainment of a revised O₃ standard with a level of 70 ppb. The EPA also disagrees with aspects of the specific statements made by the commenters as support for their view that the EPA analyses have underestimated background O₃.⁸⁶ Thus, even assuming the commenters are correct that the EPA may use proximity to background as a justification for not revising a standard that, in the judgment of the Administrator, is inadequate to protect public health, the commenters’ arguments for the justification and need to do so for this review are based on a flawed premise.

b. Comments on the Health Effects Evidence

As noted above, comments on the adequacy of the current standard fell into two broad categories reflecting very

⁸⁶ Specific aspects of the comments on the EPA analyses are addressed in more detail in the RTC.

different views of the available scientific evidence. Commenters who expressed support for the EPA's proposed decision to revise the current primary O₃ standard generally concluded that the body of scientific evidence assessed in the ISA is much stronger and more compelling than in the last review. These commenters also generally emphasized CASAC's interpretation of the body of available evidence, which formed an important part of the basis for CASAC's reiterated recommendations to revise the O₃ standard to provide increased public health protection. In some cases, these commenters supported their positions by citing studies published since the completion of the ISA.

The EPA generally agrees with these commenters regarding the need to revise the current primary O₃ standard in order to increase public health protection though, in many cases, not with their conclusions about the degree of protection that is appropriate (II.C.4.b and II.C.4.c, below). The scientific evidence noted by these commenters was generally the same as that assessed in the ISA (U.S. EPA, 2013) and the proposal,⁸⁷ and their interpretation of the evidence was often, though not always, consistent with the conclusions of the ISA and CASAC. The EPA agrees that the evidence available in this review provides a strong basis for the conclusion that the current O₃ standard is not adequately protective of public health. In reaching this conclusion, the EPA places a large amount of weight on the scientific advice of CASAC, and on CASAC's endorsement of the assessment of the evidence in the ISA (Frey and Samet, 2012).

In contrast, while commenters who opposed the proposed decision to revise the primary O₃ standard generally focused on many of the same studies assessed in the ISA, these commenters highlighted different aspects of these studies and reached substantially different conclusions about their strength and the extent to which progress has been made in reducing uncertainties in the evidence since the last review. These commenters generally concluded that information about the health effects of concern has not changed significantly since 2008 and that the uncertainties in the underlying health science have not been reduced

⁸⁷ As discussed in section I.C above, the EPA has provisionally considered studies that were highlighted by commenters and that were published after the ISA. These studies are generally consistent with the evidence assessed in the ISA, and they do not materially alter our understanding of the scientific evidence or the Agency's conclusions based on that evidence.

since the 2008 review. In some cases, these commenters specifically questioned the EPA's approach to assessing the scientific evidence and to reaching conclusions on the strength of that evidence in the ISA. For example, several commenters asserted that the EPA's causal framework, discussed in detail in the ISA, is flawed and that it has not been applied consistently across health endpoints. Commenters also noted departures from other published causality frameworks (Samet and Bodurow, 2008) and from the criteria for judging causality put forward by Sir Austin Bradford Hill (Hill, 1965).

The EPA disagrees with comments questioning the ISA's approach to assessing the evidence, the causal framework established in the ISA, or the consistent application of that framework across health endpoints. While the EPA acknowledges the ISA's approach departs from assessment and causality frameworks that have been developed for other purposes, such departures reflect appropriate adaptations for the NAAQS. As with other ISAs, the O₃ ISA uses a five-level hierarchy that classifies the weight of evidence for causation. In developing this hierarchy, the EPA has drawn on the work of previous evaluations, most prominently the IOM's *Improving the Presumptive Disability Decision-Making Process for Veterans* (Samet and Bodurow, 2008), EPA's Guidelines for Carcinogen Risk Assessment (U.S. EPA, 2005), and the U.S. Surgeon General's smoking report (CDC, 2004). The ISA's weight of evidence evaluation is based on the integration of findings from various lines of evidence from across the health and environmental effects disciplines. These separate judgments are integrated into a qualitative statement about the overall weight of the evidence and causality. The ISA's causal framework has been developed over multiple NAAQS reviews, based on extensive interactions with CASAC and based on the public input received as part of the CASAC review process. In the current review, the causality framework, and the application of that framework to causality determinations in the O₃ ISA, have been reviewed and endorsed by CASAC (Frey and Samet, 2012).

Given these views on the assessment of the evidence in the ISA, it is relevant to note that many of the issues and concerns raised by commenters on the EPA's interpretation of the evidence, and on the EPA's conclusions regarding the extent to which uncertainties have been reduced since the 2008 review, are essentially restatements of issues raised during the development of the ISA, HREA, and/or PA. The CASAC O₃ Panel

reviewed the interpretation of the evidence, and the EPA's use of information from specific studies, in drafts of these documents. In CASAC's advice to the Administrator, which incorporates its consideration of many of the issues raised by commenters, CASAC approved of the scientific content, assessments, and accuracy of the ISA, REA, and PA, and indicated that these documents provide an appropriate basis for use in regulatory decision making for the O₃ NAAQS (Frey and Samet, 2012, Frey, 2014a, Frey, 2014c). Therefore, the EPA's responses to many of the comments on the evidence rely heavily on the process established in the ISA for assessing the evidence, which is the product of extensive interactions with CASAC over a number of different reviews, and on CASAC advice received as part of this review of the O₃ NAAQS.

The remainder of this section discusses public comments and the EPA's responses, on controlled human exposure studies (II.B.2.b.i); epidemiologic studies (II.B.2.b.ii); and at-risk populations (II.B.2.b.iii).

i. Evidence From Controlled Human Exposure Studies

This section discusses major comments on the evidence from controlled human exposure studies and provides the Agency's responses to those comments. To support their views on the adequacy of the current standard, commenters often highlighted specific aspects of the scientific evidence from controlled human exposure studies. Key themes discussed by these commenters included the following: (1) The adversity of effects demonstrated in controlled human exposure studies, especially studies conducted at exposure concentrations below 80 ppb; (2) representativeness of different aspects of the controlled human exposure studies for making inferences to the general population and at-risk populations; (3) results of additional analyses of the data from controlled human exposure studies; (4) evaluation of a threshold for effects; and (5) importance of demonstration of inflammation at 60 ppb. This section discusses these key comment themes, and provides the EPA's responses. More detailed discussion of individual comments, and the EPA's responses, is provided in the Response to Comments document.

Adversity

Some commenters who disagreed with the EPA's proposed decision to revise the current primary O₃ standard disputed the Agency's characterization

of the adversity of the O₃-induced health effects shown to occur in controlled human exposure studies. Some of these commenters contended that the proposal does not provide a clear definition of adversity or that there is confusion concerning what responses the Administrator considers adverse. The EPA disagrees with these comments, and notes that section II.E.4.d of the proposal describes the Administrator's proposed approach to considering the adversity of effects observed in controlled human exposure studies. Her final approach to considering the adversity of these effects, and her conclusions on adversity, are described in detail below (II.C.4.b, II.C.4.c).

Other commenters disagreed with the EPA's judgments regarding adversity and expressed the view that the effects observed in controlled human exposure studies following 6.6-hour exposures to O₃ concentrations below the level of the current standard (*i.e.*, 75 ppb) are not adverse.⁸⁸ This group of commenters cited several reasons to support their views, including that: (1) The lung function decrements and respiratory symptoms observed at 72 ppb in the study by Schelegle et al. (2009) were not correlated with each other, and therefore were not adverse; and (2) group mean FEV₁ decrements observed following exposures below 75 ppb are small (*e.g.*, <10%, as highlighted by some commenters), transient and reversible, do not interfere with daily activities, and do not result in permanent respiratory injury or progressive respiratory dysfunction.

While the EPA agrees that not all effects reported in controlled human exposure studies following exposures below 75 ppb can reasonably be considered to be adverse, the Agency strongly disagrees with comments asserting that none of these effects can be adverse. As an initial matter, the Administrator notes that, when considering the extent to which the current or a revised standard could allow adverse respiratory effects, based on information from controlled human exposure studies, she considers not only the effects themselves, but also quantitative estimates of the extent to which the current or a revised standard could allow such effects. Quantitative

⁸⁸ Commenters who supported revising the primary O₃ standard often concluded that there is clear evidence for adverse effects following exposures to O₃ concentrations at least as low as 60 ppb, and that such adverse effects support setting the level of a revised primary O₃ standard at 60 ppb. These comments, and the EPA's responses, are discussed below within the context of the Administrator's decision on a revised level (II.C.4.b).

exposure and risk estimates provide perspective on the extent to which various standards could allow populations, including at-risk populations such as children and children with asthma, to experience the types of O₃ exposures that have been shown in controlled human exposure studies to cause respiratory effects. As discussed further below (II.B.3, II.C.4.b, II.C.4.c), to the extent at-risk populations are estimated to experience such exposures repeatedly, the Administrator becomes increasingly concerned about the potential for adverse responses in the exposed population. Repeated exposures provide a plausible mode of action by which O₃ may cause other more serious effects. Thus, even though the Administrator concludes there is important uncertainty in the adversity of some of the effects observed in controlled human exposure studies based on the single exposure periods evaluated in these studies (*e.g.*, FEV₁ decrements observed following exposures to 60 ppb O₃, as discussed in sections II.C.4.b and II.C.4.c below), she judges that the potential for adverse effects increases as the number of exposures increases. Contrary to the commenters' views noted above, the Administrator considers the broader body of available information (*i.e.*, including quantitative exposure and risk estimates) when considering the extent to which the current or a revised standard could allow adverse respiratory effects (II.B.3, II.C.4.b, II.C.4.c, below).

In further considering commenters' views on the potential adversity of the respiratory effects themselves (*i.e.*, without considering quantitative estimates), the EPA notes that although the results of controlled human exposure studies provide a high degree of confidence regarding the occurrence of health effects following exposures to O₃ concentrations from 60 to 80 ppb, there are no universally accepted criteria by which to judge the adversity of the observed effects. Therefore, as in the proposal, the Administrator relies upon recommendations from the ATS and advice from CASAC to inform her judgments on adversity.

In particular, the Administrator focuses on the ATS recommendation that "reversible loss of lung function in combination with the presence of symptoms should be considered adverse" (ATS, 2000a). The study by Schelegle et al. (2009) reported a statistically significant decrease in group mean FEV₁ and a statistically significant increase in respiratory symptoms in healthy adults following 6.6-hour exposures to average O₃

concentrations of 72 ppb. In considering these effects, CASAC noted that "the combination of decrements in FEV₁ together with the statistically significant alterations in symptoms in human subjects exposed to 72 ppb ozone meets the American Thoracic Society's definition of an adverse health effect" (Frey, 2014c, p. 5).

As mentioned above, some commenters nonetheless maintained that the effects observed in Schelegle et al. (2009) following exposure to 72 ppb O₃ (average concentration) were not adverse because the magnitudes of the FEV₁ decrements and the increases in respiratory symptoms (as measured by the total subjective symptoms score, TSS) were not correlated across individual study subjects. A commenter submitted an analysis of the individual-level data from the study by Schelegle et al. (2009) to support their position. This analysis indicated that, while the majority of study volunteers (66%) did experience both lung function decrements and increased respiratory symptoms following 6.6-hour exposures to 72 ppb O₃, some (33%) did not (*e.g.*, Figure 3 in comments from Gradient).⁸⁹ In addition, the study subjects who experienced relatively large lung function decrements did not always also experience relatively large increases in respiratory symptoms. These commenters interpreted the lack of a statistically significant correlation between the magnitudes of decrements and symptoms as meaning that the effects reported by Schelegle et al. (2009) at 72 ppb did not meet the ATS criteria for an adverse response.

However, the ATS recommendation that the combination of lung function decrements and symptomatic responses be considered adverse is not restricted to effects of a particular magnitude nor a requirement that individual responses be correlated. Similarly, CASAC made no such qualifications in its advice on the combination of respiratory symptoms and lung function decrements (See *e.g.*, Frey, 2014c, p. 5). Therefore, as in the proposal and consistent with both CASAC advice and ATS recommendations, the EPA continues to conclude that the finding of both statistically significant decrements in lung function and significant increases in respiratory symptoms following 6.6-hour exposures to an average O₃ concentration of 72 ppb provides a strong indication of the

⁸⁹ The figure provided in comments by Gradient only clearly illustrated the responses of 30 out of 31 subjects.

potential for exposed individuals to experience this combination of effects.⁹⁰

In particular, the Administrator notes that lung function provides an objective measure of the respiratory response to O₃ exposure while respiratory symptoms are subjective, and as evaluated by Schelegle et al. (2009) were based on a TSS score. If an O₃ exposure causes increases in both objectively measured lung function decrements and subjective respiratory symptoms, which indicate that people may modify their behavior in response to the exposure, then the effect is properly viewed as adverse. As noted above, the commenter's analysis shows that the majority of study volunteers exposed to 72 ppb O₃ in the study by Schelegle et al. (2009) did, in fact, experience both a decrease in lung function and an increase in respiratory symptoms.

In further considering this comment, the EPA recognizes that, consistent with commenter's analysis, some individuals may experience large decrements in lung function with minimal to no respiratory symptoms (McDonnell et al., 1999), and vice versa. As indicated above and discussed in the proposal (79 FR 75289), the Administrator acknowledges such interindividual variability in responsiveness in her interpretation of estimated exposures of concern. Specifically, she notes that not everyone who experiences an exposure of concern, including for the 70 ppb benchmark, is expected to experience an adverse response. However, she further judges that the likelihood of adverse effects increases as the number of occurrences of O₃ exposures of concern increases. In making this judgment, she notes that the types of respiratory effects that can occur following exposures of concern, particularly if experienced repeatedly, provide a plausible mode of action by which O₃ may cause other more serious effects.⁹¹ Therefore, her decisions on the primary standard emphasize the public health importance of limiting the occurrence of repeated exposures to O₃ concentrations at or above those shown to cause adverse

⁹⁰ Indeed, the finding of statistically significant decreases in lung function and increases in respiratory symptoms in the same study population indicates that, on average, study volunteers did experience both effects.

⁹¹ For example, as discussed in the proposal (79 FR 75252) and the ISA (p. 6–76), inflammation induced by a single exposure (or several exposures over the course of a summer) can resolve entirely. However, repeated occurrences of airway inflammation could potentially result in the induction of a chronic inflammatory state; altered pulmonary structure and function, leading to diseases such as asthma; altered lung host defense response to inhaled microorganisms; and altered lung response to other agents such as allergens or toxins (ISA, section 6.2.3).

effects in controlled human exposure studies (II.B.3, II.C.4.b, II.C.4.c). The Administrator views this approach to considering the evidence from controlled human exposure studies as being consistent with commenter's analysis indicating that, while the majority did, not all study volunteers exposed to 72 ppb O₃ experienced the adverse combination of lung function decrements and respiratory symptoms following the single exposure period evaluated by Schelegle et al. (2009).

Representativeness

A number of commenters raised issues concerning the representativeness of controlled human exposure studies considered by the Administrator in this review, based on different aspects of these studies. These commenters asserted that since the controlled human exposure studies were not representative of real-world exposures, they should not be relied upon as a basis for finding that the current standard is not adequate to protect public health. Some issues highlighted by commenters include: Small size of the study populations; unrealistic activity levels used in the studies; unrealistic exposure scenarios (*i.e.*, triangular exposure protocol) used in some studies, including Schelegle et al. (2009); and differences in study design that limit comparability across studies.

Some commenters noted that the controlled human exposure studies were not designed to have individuals represent portions of any larger group and that the impacts on a small number of people do not implicate the health of an entire subpopulation, particularly when the FEV₁ decrements are small, temporary, and reversible. These commenters also noted that the Administrator failed to provide an explanation or justification for why the individuals in these studies can be viewed as representatives of a subpopulation. Further, they asserted that EPA's use of results from individuals, rather than the group mean responses, contradicts the intent of CAA section 109 to protect groups of people, not just the most sensitive individuals in any group (79 FR 75237).

Consistent with CASAC advice (Frey, 2014c, p. 5), the EPA concludes that the body of controlled human exposure studies are sufficiently representative to be relied upon as a basis for finding that the current standard is not adequate to protect public health. These studies generally recruit healthy young adult volunteers, and often expose them to O₃ concentrations found in the ambient air under real-world exposure conditions. As described in more detail above in

section II.A.1.b, the evidence from controlled human exposure studies to date makes it clear that there is considerable variability in responses across individuals, even in young healthy adult volunteers, and that group mean responses are not representative of more responsive individuals. It is important to look beyond group mean responses to the responses of these individuals to evaluate the potential impact on more responsive members of the population. Moreover, relying on group mean changes to evaluate lung function responses to O₃ exposures would mask the responses of the most sensitive groups, particularly where, as here, the group mean reflects responses solely among the healthy young adults who were the study participants. Thus, the studies of exposures below 80 ppb O₃ show that 10% of young healthy adults experienced FEV₁ decrements >10% following exposures to 60 ppb O₃, and 19% experienced such decrements following exposures to 72 ppb (under the controlled test conditions involving moderate exertion for 6.6 hours). These percentages would likely have been higher had people with asthma or other at-risk populations been exposed (U.S. EPA, 2013, pp. 6–17 and 6–18; Frey 2014c, p. 7; Frey, 2014a, p. 14).⁹²

Moreover, the EPA may legitimately view the individuals in these studies as representatives of the larger subpopulation of at-risk or sensitive groups. As stated in the Senate Report to the 1970 legislation establishing the NAAQS statutory provisions, “the Committee emphasizes that included among these persons whose health should be protected by the ambient standard are particularly sensitive citizens such as bronchial asthmatics and emphysematics who in the normal course of daily activity are exposed to the ambient environment. In establishing an ambient standard necessary to protect the health of these persons, reference should be made to a representative sample of persons comprising the sensitive group rather than to a single person in such a group. . . . For purposes of this description, a statistically related sample is the number of persons necessary to test in order to detect a deviation in the health of any person within such sensitive group which is attributable to the condition of the ambient air.” S. Rep. No. 11–1196, 91st

⁹² See also *National Environmental Development Associations Clean Action Project v. EPA*, 686 F. 3d 803, 811 (D.C. Cir. 2012) (EPA drew legitimate inference that serious asthmatics would experience more serious health effects than clinical test subjects who did not have this degree of lung function impairment).

Cong. 2d sess. at 10. As just noted above, 10% of healthy young adults in these studies experienced >10% FEV₁ decrements following exposure to 60 ppb O₃, and the proportion of individuals experiencing such decrements increases with increasing O₃ exposure concentrations. This substantial percentage certainly can be viewed as “a representative sample of persons” and as a sufficient number to “detect a deviation in the health of any person within such sensitive group,” especially given that it reflects the percentage of *healthy adults* who experienced decrements >10%.

These results are consistent with estimates from the MSS model, which makes reliable quantitative predictions of the lung function response to O₃ exposures, and reasonably predicts the magnitude of individual lung function responses following such exposures. As described in section II.A.2.c above, and documented in the HREA, when the MSS model was used to quantify the risk of O₃-induced FEV₁ decrements in 15 urban study areas, the current standard was estimated to allow about 8 to 12% of children to experience two or more O₃-induced FEV₁ decrements ≥10%, and about 2 to 3% to experience two or more decrements ≥15% (Table 2, above). These percentages correspond to hundreds of thousands of children in urban study areas, and tens of thousands of asthmatic children. While the Administrator judges that there is uncertainty with regard to the adversity of these O₃-induced lung function decrements (see II.C.4.b, II.C.4.c, below), such risk estimates clearly indicate that they are a matter of public health importance on a broad scale, not isolated effects on idiosyncratically responding individuals.

Other commenters considered the ventilation rates used in controlled human exposure studies to be unreasonably high and at the extreme of prolonged daily activity. Some of these commenters noted that these scenarios are unrealistic for sensitive populations, such as asthmatics and people with COPD, whose conditions would likely prevent them from performing the intensity of exercise, and therefore experiencing the ventilation rates, required to produce decrements in lung function observed in experimental settings.

The EPA disagrees with these commenters. The activity levels used in controlled human exposure studies were summarized in Table 6–1 of the ISA (U.S. EPA, 2013). The exercise level in the 6.6-hour exposure studies by Adams (2006), Schelegle et al. (2009), and Kim et al. (2011) of young healthy

adults was moderate and ventilation rates are typically targeted for 20 L/min-m² BSA.⁹³ Following the exposures to 60 ppb at this activity level, 10% of the individuals had greater than a 10% decrement in FEV₁ (U.S. EPA, 2013, p. 6–18). Similar 6.6-hour exposure studies of individuals with asthma are not available to assess either the effects of O₃ on their lung function or their ability to perform the required level of moderate exercise.

However, referring to Tables 6–9 and 6–10 of the HREA (U.S. EPA, 2014a), between 42% and 45% of FEV₁ decrements ≥ 10% were estimated to occur at exercise levels of <13 L/min-m² BSA. This corresponds to light exercise, and this level of exercise has been used in a 7.6-hour study of healthy people and people with asthma exposed to 160 ppb O₃ (Horstman et al., 1995). In that study, people with asthma exercised with an average minute ventilation of 14.2 L/min-m² BSA. Adjusted for filtered air responses, an average 19% FEV₁ decrement was seen in the people with asthma versus an average 10% FEV₁ decrement in the healthy people. In addition, the EPA noted in the HREA that the data underlying the exposure assessment indicate that “activity data for asthmatics [is] generally similar to [that for] non-asthmatics” (U.S. EPA, 2014a, p. 5–75, Tables 5G–2 and 5G–3). Thus, contrary to the commenters’ assertion, based on both the HREA and the Horstman et al. (1995) study, people with respiratory disease such as asthma can exercise for a prolonged period under conditions where they would experience >10% FEV₁ decrements in response to O₃ exposure.

Additionally, a number of commenters asserted that the exposure scenarios in Schelegle et al. (2009), which are based on a so-called triangular study protocol, where O₃ concentrations ramp up and down as the study is conducted, are not directly generalizable to most healthy or sensitive populations because of large changes in the O₃ concentrations from one hour to the next. Commenters stated that although large fluctuations in O₃ are possible in certain locations due to meteorological conditions (e.g., in valleys on very hot, summer days), they believe that, in general, concentrations of O₃ do not fluctuate by more than 20–30 ppb from one hour to the next. Thus, commenters suggested the Schelegle et

⁹³ Exercise consisted of alternating periods walking on a treadmill at a pace of 17–18 minutes per mile inclined to a grade of 4–5% or cycling at a load of about 72 watts. Typical heart rates during the exercise periods were between 115–130 beats per minute. This activity level is considered moderate (Table 6–1, U.S. EPA, 2013, p. 6–18).

al. (2009) study design could happen in a “worst-case” exposure scenario, but that the exposure protocol was not reflective of conditions in most cities and thus not informative with regard to the adequacy of the current standard.

The EPA disagrees with the comment that these triangular exposure scenarios are not generalizable because of hour-to-hour fluctuations. Adams (2002, 2006) showed that FEV₁ responses following 6.6 hours of exposure to 60 and 80 ppb average O₃ exposures do not differ between triangular (*i.e.* ramping concentration up and down) and square-wave (*i.e.* constant concentration). Schelegle et al. (2009) used the 80 ppb triangular protocol and a slightly modified 60 ppb triangular protocol (concentrations during the third and fourth hours were reversed) from Adams (2006). Therefore, in considering pre- to post-exposure changes in lung function, concerns about the hour-by-hour changes in O₃ concentrations at 60 and 80 ppb in the Schelegle et al. (2009) study are unfounded.

Finally, some commenters also stated that the Kim et al. (2011) study is missing critical information and its study design makes comparison to the other studies difficult. That is, the commenter suggests that data at times other than pre- and post-exposure should have been provided.

The EPA disagrees with this comment. With regard to providing data at other time points besides pre- and post-exposure, there is no standard that suggests an appropriate frequency at which lung function should be measured in prolonged 6.6-hour exposure studies. The Adams (2006) study showed that lung function decrements during O₃ exposures with moderate exercise become most apparent following the third hour of exposure. As such, it makes little sense to measure lung function during the first couple hours of exposure. However, having data at multiple time points toward the end of an exposure can provide evidence that the mean post-exposure FEV₁ response is not a single anomalous data point. The FEV₁ response data for the 3-, 4.6-, 5.6-, and 6.6-hour time points of the Kim et al. (2011) study are available in Figure 6 of the McDonnell et al. (2012) paper where they are plotted with the Adams (2006) data for 60 ppb. Similar to the Adams (2006) study, the responses at 5.6 hours are only marginally smaller than the response at 6.6 hours in the Kim et al. (2011) study. This indicates that the post-exposure FEV₁ responses in both studies are consistent with responses at an earlier time point and thus not likely to be anomalous data.

Additional Studies

Several commenters analyzed the data from controlled human exposure studies, or they commented on the EPA's analysis of the data from some of these studies (Brown et al., 2008), to come to a different conclusion than the EPA's interpretation of these studies thereby questioning the proposed decision that the current standard is not adequate to protect public health. One commenter submitted an independent assessment of the scientific evidence and risk, and used this analysis to assert that there are multiple flaws in the underlying studies and their interpretation by the EPA. This commenter stated that the EPA's discussion of the spirometric responses of children and adolescents and older adults to O₃ was misleading. They claimed that the EPA did not mention that "the responses of children and adolescents are equivalent to those of young adults (18–35 years old; McDonnell et al., 1985) and that this response diminishes in middle-aged and older adults (Hazucha 1985)." The EPA notes that the commenter misrepresented our characterization of the effect of age on FEV₁ responses to O₃ and asserted mistakenly that EPA did not mention diminished responses on older adults. In fact, the proposal clearly states that, "Respiratory symptom responses to O₃ exposure appears to increase with age until early adulthood and then gradually decrease with increasing age (U.S. EPA, 1996b); lung function responses to O₃ exposure also decline from early adulthood (U.S. EPA, 1996b)" (79 FR 75267) (see also U.S. EPA, 2014c p. 3–82). With regard to differences between children and adults, it was clearly stated in the ISA (U.S. EPA, 2013, p. 6–21) that healthy children exposed to filtered air and 120 ppb O₃ experienced similar spirometric responses, but lesser symptoms than similarly exposed young healthy adults (McDonnell et al., 1985). In addition, the EPA's approach to modeling the effect of age on responses to O₃ is clearly provided in the HREA (U.S. EPA, 2014a, Table 6–2).

The commenter also stated that the EPA's treatment of filtered air responses in the dose-response curve was incorrect. They claimed that when creating a dose-response curve, it is most appropriate to include a zero-dose point and not to subtract the filtered air response from responses to O₃. Contrary to this assertion, EPA correctly adjusted FEV₁ responses to O₃ by responses following filtered air, as was also done in the McDonnell et al. (2012) model. As indicated in the ISA (U.S. EPA, 2013, p.

6–4), the majority of controlled human exposure studies investigating the effects O₃ are of a randomized, controlled, crossover design in which subjects were exposed, without knowledge of the exposure condition and in random order, to clean filtered air and, depending on the study, to one or more O₃ concentrations. The filtered air control exposure provides an unbiased estimate of the effects of the experimental procedures on the outcome(s) of interest. Comparison of responses following this filtered air exposure to those following an O₃ exposure allows for estimation of the effects of O₃ itself on an outcome measurement while controlling for independent effects of the experimental procedures, such as ventilation rate. Thus, the commenter's approach does not provide an estimate of the effects of O₃ alone. Furthermore, as illustrated in these comments, following "long" filtered air exposures, there is about a 1% improvement in FEV₁. By not accounting for this increase in FEV₁, the commenter underestimated the FEV₁ decrement due to O₃ exposure. The commenter's approach thus is fundamentally flawed.

The commenter also asserted that the McDonnell et al. (2012) model and exposure-response (E–R) models incorrectly used only the most responsive people and that EPA's reliance on data from clinical trials that use only the most responsive people irrationally ignores large portions of relevant data. The EPA rejects this assertion that the McDonnell et al. (2012) model and the E–R analysis ignored large portions of relevant data. The McDonnell et al. (2012) model was fit to the FEV₁ responses of 741 individuals to O₃ and filtered air (*i.e.*, reflecting all available data for O₃-induced changes in FEV₁). The filtered air responses were subtracted from responses measured during O₃ exposures. Subsequently, as illustrated by the figures in the McDonnell et al. (2012) paper and described in the text of paper, the model was fit to all available FEV₁ data measured during the course of O₃ exposures, including exposures shorter than 6.6 hours. Thus, the model predicts temporal dynamics of FEV₁ response to any set of O₃ exposure conditions that might reasonably be experienced in the ambient environment, predicting the mean responses and the distribution of responses around the mean. For the HREA (EPA, 2014a), the proportion of individuals, under variable exposure conditions, predicted to have FEV₁

decrements ≥ 10 , 15 and 20% was estimated.

Finally, the commenter referenced the exposure-response model on p. 6–18 of the HREA. However, they neglected to note that this was in a section describing the exposure-response function approach used in prior reviews (U.S. EPA, 2014a, starting on p. 6–17). Thus, the commenter confused the exposure-response model used in the last review with the updated approach used in this review.

The commenter also stated that EPA did not properly consider O₃ dose when interpreting the human clinical data. Ozone total dose includes three factors: duration of exposure, concentration, and ventilation rate. The commenter claimed the EPA emphasized only concentration without properly considering and communicating duration of exposure and ventilation rate. Further, they asserted that because people are not exposed to the same dose, they cannot be judged to have the same exposure and would therefore not be expected to respond consistently. The EPA rejects the claim that we emphasized only concentration without properly incorporating the other two factors. As noted in the ISA, total O₃ dose does not describe the temporal dynamics of FEV₁ responses as a function of concentration, ventilation rate, time and age of the exposed individuals (U.S. EPA, 2013, p. 6–5). Thus, the use of total O₃ dose is antiquated and the EPA therefore conducted a more sophisticated analysis of FEV₁ response to O₃ in the HREA. In this review, the HREA estimates risks of lung function decrements in school-aged children (ages 5 to 18), asthmatic school-aged children, and the general adult population for 15 urban study areas. A probabilistic model designed to account for the numerous sources of variability that affect people's exposures was used to simulate the movement of individuals through time and space and to estimate their exposure to O₃ while occupying indoor, outdoor, and in-vehicle locations. That information was linked with the McDonnell et al. (2012) model to estimate FEV₁ responses over time as O₃ exposure concentrations and ventilation rates changed. As noted earlier, CASAC agreed that this approach is both scientifically valid and a significant improvement over approaches used in past O₃ reviews (Frey, 2014a, p. 2).

Several commenters criticized the EPA analysis published by Brown et al. (2008). One commenter suggested that the EPA needed to state why the Brown et al. (2008) analysis was relied on rather than Nicolich (2007) or Lefohn et

al. (2010). Further, commenters stated that the analysis of the Adams (2006) data in Brown et al. (2008) was flawed. Among other reasons, one commenter expressed the opinion that it was not appropriate for Brown et al. (2008) to only examine a portion of the Adams (2006) data, citing comments submitted by Gradient.

The EPA disagrees with these commenters.⁹⁴ As an initial matter, Nicolich (2007) was a public comment and is not a peer-reviewed publication that would be used to assess the scientific evidence for effects of O₃ on lung function in the ISA (U.S. EPA, 2013). The Nicolich (2007) comments were specifically addressed by the EPA on pp. 24–25 in the Response to Comments Document for the 2007 proposed rule (U.S. EPA, 2008). On page A–3 of his comments, Dr. Nicolich stated “that the residuals are not normally distributed and the observations do not meet the assumptions required for the model” and that “the subject-based errors are not independently, identically and normally distributed and the subjects do not meet the assumptions required for the model.” The EPA reasonably chose not to rely on this analysis: “Therefore, given that the underlying statistical assumptions required for his analyses were not met and that significance levels are questionable, in EPA’s judgment the analyses presented by Dr. Nicolich are ambiguous” (U.S. EPA, 2008). It is likely that the Lefohn et al. (2010) analysis of the Adams (2006) data would similarly not meet the statistical assumptions of the model (*e.g.*, homoscedasticity). In contrast, recognizing the concerns related to the distribution of responses, Brown et al. (2008) conservatively used a nonparametric sign test to obtain a *p*-value of 0.002 for the comparison responses following 60 ppb O₃ versus filter air. Other common statistical tests also showed significant effects on lung function. In addition, the effects of 60 ppb O₃ on FEV₁ responses in Brown et al. (2008) remained statistically significant even following the exclusion of three potential outliers.

EPA disagrees with the comment stating that it was not appropriate for Brown et al. (2008) to only examine a portion of the Adams (2006) data. In

fact, there is no established single manner or protocol decreeing that data throughout the protocol must be analyzed and included. Furthermore, Brown et al. (2008) was a peer-reviewed journal publication. CASAC also expressed favorable comments in their March 30, 2011, letter to Administrator Jackson. With reference to a memorandum (Brown, 2007) that preceded the Brown et al. (2008) publication, on p. 6 of the CASAC Consensus Responses to Charge Questions CASAC stated, “The results of the Adams et al. study also have been carefully reanalyzed by EPA investigators (Brown et al., [2008]), and this reanalysis showed a statistically significant group effect on FEV₁ after 60 ppb ozone exposure.” On p. A–13, a CASAC panelist and biostatistician stated, “Thus, from my understanding of the statistical analyses that have been conducted, I would argue that the analysis by EPA should be preferred to that of Adams for the specific comparison of the FEV₁ effects of 0.06 ppm exposure relative to filtered air exposure.” (Samet 2011, p. a-13)

Threshold

Several commenters used the new McDonnell et al. (2012) and Schelegle et al. (2012) models to support their views about the O₃ concentrations associated with a threshold for adverse lung function decrements. For example, one commenter who supported retaining the current standard noted that McDonnell et al. (2012) found that the threshold model fit the observed data better than the original (no-threshold) model, especially at earlier time points and at the lowest exposure concentrations. The commenter expressed the view that the threshold model showed that the population mean FEV₁ decrement did not reach 10% until exposures were at least 80 ppb, indicating that O₃ exposures of 80 ppb or higher may cause lung function decrements and other respiratory effects.⁹⁵

As described above in section II.A.1.b, the McDonnell et al. (2012) and Schelegle et al. (2012) models represent a significant technological advance in the exposure-response modeling approach since the last review, and these models indicate that a dose-threshold model fits the data better than a non-threshold model. However, the

EPA disagrees that using the predicted group mean response from the McDonnell model provides support for retaining the current standard. As discussed above, the group mean responses do not convey information about interindividual variability, or the proportion of the population estimated to experience the larger lung function decrements (*e.g.*, 10 or 15% FEV₁ decrements) that could be adverse. In fact, it masks this variability. These variable effects in individuals have been found to be reproducible. In other words, a person who has a large lung function response after exposure to O₃ will likely have about the same response if exposed again in a similar manner (raising health concerns, as noted above). Group mean responses are not representative of this segment of the population that has much larger than average responses to O₃.

Inflammation

Some commenters asserted that the pulmonary inflammation observed following exposure to 60 ppb in the controlled human exposure study by Kim et al. (2011) was small and unlikely to result in airway damage. It was also suggested that this inflammation is a normal physiological response in all living organisms to stimuli to which people are normally exposed.

The EPA recognized in the proposal (79 FR 75252) and the ISA (U.S. EPA, 2013, p. 6–76) that inflammation induced by a single exposure (or several exposures over the course of a summer) can resolve entirely. Thus, the inflammatory response observed following the single exposure to 60 ppb in the study by Kim et al. (2011) is not necessarily a concern. However, the EPA notes that it is also important to consider the potential for continued acute inflammatory responses to evolve into a chronic inflammatory state and to affect the structure and function of the lung.⁹⁶ The Administrator considers this possibility through her consideration of estimated exposures of concern for the 60 ppb benchmark (II.B.3, II.C.4). As discussed in detail below (II.C.4.b), while she judges that there is uncertainty in the adversity of the effects shown to occur following exposures to 60 ppb O₃, including the inflammation reported by Kim et al.

⁹⁴ The DC Circuit has held that EPA reasonably used and interpreted the Brown (2007) study in the last review. *Mississippi*, 744 F. 3d at 1347. In this review, there is now additional corroborative evidence supporting the Brown (2007) analysis, in the form of further controlled human clinical studies finding health effects in young, healthy adults at moderate exercise at O₃ concentrations of 60 ppb over a 6.6 hour exposure period.

⁹⁵ Conversely, another group of commenters who supported revising the standard to a level of 60 ppb noted that the results of these models are consistent with the results of controlled human exposure studies finding adverse health effects at 60 ppb. These comments are discussed below (II.C.4.b), within the context of the Administrator’s decision on a revised standard level.

⁹⁶ Inflammation induced by exposure of humans to O₃ can have several potential outcomes, ranging from resolving entirely following a single exposure to becoming a chronic inflammatory state (U.S. EPA, 2013, section 6.2.3). Lung injury and the resulting inflammation provide a mechanism by which O₃ may cause other more serious morbidity effects (*e.g.*, asthma exacerbations) (U.S. EPA, 2013, section 6.2.3). See generally section II.A.1.a above.

(2011), she gives some consideration to estimates of two or more exposures of concern for the 60 ppb benchmark (*i.e.*, as a health-protective surrogate for repeated exposures of concern at or above 60 ppb), particularly when considering the extent to which the current and revised standards incorporate a margin of safety.

ii. Evidence From epidemiologic studies

This section discusses key comments on the EPA's assessment of the epidemiologic evidence and provides the Agency's responses to those comments. The focus in this section is on overarching comments related to the EPA's approach to assessing and interpreting the epidemiologic evidence as a whole. Detailed comments on specific studies, or specific methodological or technical issues, are addressed in the Response to Comments document. As discussed above, many of the issues and concerns raised by commenters on the interpretation of the epidemiologic evidence are essentially restatements of issues raised during the development of the ISA, HREA, and/or PA, and in many instances were considered by CASAC in the development of its advice on the current standard. The EPA's responses to these comments rely heavily on the process established in the ISA for assessing the evidence, and on CASAC advice received as part of this review of the O₃ NAAQS.

As with evidence from controlled human exposure studies, commenters expressed sharply divergent views on the evidence from epidemiologic studies, and on the EPA's interpretation of that evidence. One group of commenters, representing medical, public health and environmental organizations, and some states, generally supported the EPA's interpretation of the epidemiologic evidence with regard to the consistency of associations, the coherence with other lines of evidence, and the support provided by epidemiologic studies for the causality determinations in the ISA. These commenters asserted that the epidemiologic studies evaluated in the ISA provide valuable information supporting the need to revise the level of the current primary O₃ standard in order to increase public health protection. In reaching this conclusion, commenters often cited studies (including a number from the past review) which they interpreted as showing health effect associations in locations with O₃ air quality concentrations below the level of the current standard. A second group of commenters, mostly representing

industry associations, businesses, and states opposed to revising the primary O₃ standard, expressed the general view that while many new epidemiologic studies have been published since the last review of the O₃ NAAQS, inconsistencies and uncertainties inherent in these studies as a whole, and in the EPA's assessment of study results, should preclude any reliance on them as justification for a more stringent primary O₃ standard. To support their views, these commenters often focused on specific technical or methodological issues that contribute to uncertainty in epidemiologic studies, including the potential for exposure error, confounding by copollutants and by other factors (*e.g.*, weather, season, disease, day of week, etc.), and heterogeneity in results across locations.

The EPA agrees with certain aspects of each of these views. Specifically, while the EPA agrees that epidemiologic studies are an important part of the broader body of evidence that supports the ISA's causality determinations, and that these studies provide support for the decision to revise the current primary O₃ standard, the Agency also acknowledges that there are important uncertainties and limitations associated with these epidemiologic studies that should be considered when reaching decisions on the current standard. Thus, although these studies show consistent associations between O₃ exposures and serious health effects, including morbidity and mortality, and some of these studies reported such associations with ambient O₃ concentrations below the level of the current standard, there are also uncertainties regarding the ambient O₃ concentrations in critical studies, such that they lend only limited support to establishing a specific level for a revised standard. (See generally, *Mississippi*, 744 F. 3d at 1351 (noting that in prior review, EPA reasonably relied on epidemiologic information in determining to revise the standard but appropriately gave the information limited weight in determining a level of a revised standard); see also *ATA III*, 283 F. 3d at 370 (EPA justified in revising NAAQS when health effect associations are observed in epidemiologic studies at levels allowed by the current NAAQS); *Mississippi*, 744 F. 3d at 1345 (same)).

Uncertainties in the evidence were considered by the Administrator in the proposal, and contributed to her decision to place less weight on information from epidemiologic studies than on information from controlled human exposure studies when considering the adequacy of the current primary O₃ standard (see 79 FR 75281–

83). Despite receiving less weight in the proposal, the EPA does not agree with commenters who asserted that uncertainties in the epidemiologic evidence provide a basis for concluding that the current primary standard does not need revision. The Administrator specifically considered the extent to which available studies support the occurrence of O₃ health effect associations with air quality likely to be allowed by the current standard, while also considering the implications of important uncertainties, as assessed in the ISA and discussed in the PA. This consideration is consistent with CASAC comments on consideration of these studies in the draft PA (Frey, 2014c, p. 5).

Based on analyses of study area air quality in the PA, the EPA notes that most of the U.S. and Canadian epidemiologic studies evaluated were conducted in locations likely to have violated the current standard over at least part of the study period. Although these studies support the ISA's causality determinations, they provide limited insight into the adequacy of the public health protection provided by the current primary O₃ standard. However, as discussed in the proposal, air quality analyses in the locations of three U.S. single-city studies provide support for the occurrence of O₃-associated hospital admissions or emergency department visits at ambient O₃ concentrations below the level of the current standard.⁹⁷ Specifically, a U.S. single-city study reported associations with respiratory emergency department visits in children and adults in a location that would have met the current O₃ standard over the entire study period (Mar and Koenig, 2009). In addition, for two studies conducted in locations where the current standard was likely not met (*i.e.*, Silverman and Ito, 2010; Strickland et al., 2010), PA analyses indicate that reported concentration-response functions and available air quality data support the occurrence of O₃-health effect associations on subsets of days with virtually all monitored ambient O₃ concentrations below the level of the current standard (U.S. EPA, 2014c,

⁹⁷ As discussed in section II.E.4.d of the proposal, is the Administrator noted the greater uncertainty in using analyses of short-term O₃ air quality in locations of the multicity studies in this review to inform decisions on the primary O₃ standard. This is because the health information in these studies cannot be disaggregated by individual city. Thus, the multicity effect estimates reported in these studies do not provide clear indication of the extent to which health effects are associated with the ambient O₃ concentrations in the study locations that met the current O₃ standard, versus the ambient O₃ concentrations in the study locations that violated the standard.

section 3.1.4.2, pp. 3–66 to 67).⁹⁸ Thus, the EPA notes that a small number of O₃ epidemiologic studies provide support for the conclusion that the current primary standard is not requisite, and that it should be revised to increase public health protection.

As part of a larger set of comments criticizing the EPA's interpretation of the evidence from time series epidemiologic studies, some commenters objected to the EPA's reliance on the studies by Strickland et al. (2010), Silverman and Ito (2010), and Mar and Koenig (2009). These commenters highlighted what they considered to be key uncertainties in interpreting these studies, including uncertainties due to the potential for confounding by co-pollutants, aeroallergens, or the presence of upper respiratory infections; and uncertainties in the interpretation of zero-day lag models (*i.e.*, specifically for Mar and Koenig, 2009).

While the EPA agrees that there are uncertainties associated with interpreting the O₃ epidemiologic evidence, as discussed above and elsewhere in this preamble, we disagree with commenters' assertion that these uncertainties should preclude the use of the O₃ epidemiologic evidence in general, or the studies by Silverman and Ito, Strickland, or Mar and Koenig in particular, as part of the basis for the Administrator's decision to revise the current primary standard. As a general point, when considering the potential importance of uncertainties in epidemiologic studies, we rely on the broader body of evidence, not restricted to these three studies, and the ISA conclusions based on this evidence. The evidence, the ISA's interpretation of specific studies, and the use of information from these studies in the HREA and PA, was considered by CASAC in its review of drafts of the ISA, HREA, and PA. Based on the assessment of the evidence in the ISA, and CASAC's endorsement of the ISA conclusions, as well as CASAC's endorsement of the approaches to using and considering information from epidemiologic studies in the HREA and

⁹⁸ Air quality analyses in locations of the studies by Silverman and Ito (2010) and Strickland et al. (2010) were used in the PA to inform staff conclusions on the adequacy of the current primary O₃ standard. However, the appropriate interpretation of these analyses became less clear for standard levels below 75 ppb, as the number of days increased with monitored concentrations exceeding the level being evaluated (U.S. EPA, 2014c, Appendix 3B, Tables 3B–6 and 3B–7). Therefore, these analyses were not used in the PA to inform conclusions on potential alternative standard levels lower than 75 ppb (U.S. EPA, 2014c, Chapters 3 and 4).

PA (Frey, 2014c, p. 5), we do not agree with these commenters' conclusions regarding the usefulness of the epidemiologic studies by Strickland et al. (2010), Silverman and Ito (2010), and Mar and Koenig (2009).

More specifically, with regard to confounding by co-pollutants, we note the ISA conclusion that, in studies of O₃-associated hospital admissions and emergency department visits “O₃ effect estimates remained relatively robust upon the inclusion of PM₁₀ and gaseous pollutants in two-pollutant models” (U.S. EPA, 2013, pp. 6–152 and 6–153). This conclusion was supported by several studies that evaluated co-pollutant models including, but not limited to, two of the studies specifically highlighted by commenters (*i.e.*, Silverman and Ito, 2010; Strickland et al., 2010) (U.S. EPA, 2013, section 6.2.7.5; Figure 6–20 and Table 6–29).

Other potential uncertainties highlighted by commenters have been evaluated less frequently (*e.g.*, confounding by allergen exposure, respiratory infections). However, we note that Strickland et al. (2010) did consider the potential for pollen (a common airborne allergen) to confound the association between ambient O₃ and emergency department visits. While quantitative results were not presented, the authors reported that “estimates for associations between ambient air pollutant concentrations and pediatric asthma emergency department visits were similar regardless of whether pollen concentrations were included in the model as covariates” (Strickland et al., 2010, p. 309). This suggests a limited impact of aeroallergens on O₃ associations with asthma-related emergency department visits and hospital admissions.

With respect to the comment about epidemiologic studies not controlling for respiratory infections in the model, the EPA disagrees with the commenter's assertion. We recognize that asthma is a multi-etiological disease and that air pollutants, including O₃, represent only one potential avenue to trigger an asthma exacerbation. Strickland et al. attempted to further clarify the relationship between short-term O₃ exposures and asthma emergency department visits by controlling for the possibility that respiratory infections may lead to an asthma exacerbation. By including the daily count of upper respiratory visits as a covariate in the model, Strickland et al. were able to account for the possibility that respiratory infections contribute to the daily counts of asthma emergency department visits, and to identify the O₃ effect on asthma emergency department

visits. In models that controlled for upper respiratory infection visits, associations between O₃ and emergency department visits remained statistically significant (Strickland et al., Table 4 in published study), demonstrating a relatively limited influence of respiratory infections on the association observed between short-term O₃ exposures and asthma emergency department visits, contrary to the commenter's claim.

In addition, with regard to the criticism of the results reported by Mar and Koenig, the EPA disagrees with commenters who questioned the appropriateness of a zero-day lag. These commenters specifically noted uncertainty in the relative timing of the O₃ exposure and the emergency department visit when they occurred on the same day. However, based on the broader body of evidence the ISA concludes that the strongest support is for a relatively immediate respiratory response following O₃ exposures. Specifically, the ISA states that “[t]he collective evidence indicates a rather immediate response within the first few days of O₃ exposure (*i.e.*, for lags days averaged at 0–1, 0–2, and 0–3 days) for hospital admissions and [emergency department] visits for all respiratory outcomes, asthma, and chronic obstructive pulmonary disease in all-year and seasonal analyses” (U.S. EPA, 2013, p. 2–32). Thus, the use of a zero-day lag is consistent with the broader body of evidence supporting the occurrence of O₃-associated health effects. In addition, while Mar and Koenig reported the strongest associations for zero-day lags, they also reported positive associations for lags ranging from zero to five days (Mar and Koenig, 2009, Table 5 in the published study). In considering this study, the ISA stated that Mar and Koenig (2009) “found consistent positive associations across individual lag days” and that “[f]or children, consistent positive associations were observed across all lags . . . with the strongest associations observed at lag 0 (33.1% [95% CI: 3.0, 68.5]) and lag 3 (36.8% [95% CI: 6.1, 77.2])” (U.S. EPA, 2013, p. 6–150). Given support for a relatively immediate response to O₃ and given the generally consistent results in analyses using various lags, we disagree with commenters who asserted that the use of a zero-day lag represents an important uncertainty in the interpretation of the study by Mar and Koenig (2009).

Given all of the above, we do not agree with commenters who asserted that uncertainties in the epidemiologic evidence in general, or in specific key studies, should preclude the

Administrator from relying on those studies to inform her decisions on the primary O₃ standard.

Some commenters also objected to the characterization in the ISA and the proposal that the results of epidemiologic studies are consistent. These commenters contended that the purported consistency of results across epidemiologic studies is the result of inappropriate selectivity on the part of the EPA in focusing on specific studies and specific results within those studies. In particular, commenters contend that EPA favors studies that show positive associations and selectively ignores certain studies that report null results. They also cite a study published after the completion of the ISA (Goodman et al., 2013) suggesting that, in papers where the results of more than one statistical model are reported, the EPA tends to report the results with the strongest associations.

The EPA disagrees that it has inappropriately focused on specific positive studies or specific positive results within individual studies. The ISA appropriately builds upon the assessment of the scientific evidence presented in previous AQCDs and ISAs.⁹⁹ When evaluating new literature, “[s]election of studies for inclusion in the ISA is based on the general scientific quality of the study, and consideration of the extent to which the study is informative and policy-relevant” (U.S. EPA, 2013, p. liii). In addition, “the intent of the ISA is to provide a concise review, synthesis, and evaluation of the most policy-relevant science to serve as a scientific foundation for the review of the NAAQS, not extensive summaries of all health, ecological and welfare effects studies for a pollutant” (U.S. EPA, 2013, p. lv). Therefore, not all studies published since the previous review would be appropriate for inclusion in the ISA.¹⁰⁰ With regard to the specific

studies that are included in the ISA, and the analyses focused upon within given studies, the EPA notes that the ISA undergoes extensive peer review in a public setting by the CASAC. This process provides ample opportunity for CASAC and the public to comment on studies not included in the ISA, and on the specific analyses focused upon within individual studies. In endorsing the final O₃ ISA as adequate for rule-making purposes, CASAC agreed with the selection and presentation of analyses on which to base the ISA’s key conclusions.

iii. Evidence Pertaining to At-Risk Populations and Lifestyles

A number of groups submitted comments on the EPA’s identification of at-risk populations and lifestyles. Some industry commenters who opposed revising the current standard disagreed with the EPA’s identification of people with asthma or other respiratory diseases as an at-risk population for O₃-attributable effects, citing controlled human exposure studies that did not report larger O₃-induced FEV₁ decrements in people with asthma than in people without asthma. In contrast, comments from medical, environmental, and public health groups generally agreed with the at-risk populations identified by EPA, and also identified other populations that they stated should be considered at risk, including people of lower socio-economic status, people with diabetes or who are obese, pregnant women (due to reproductive and developmental effects, and African American, Asian, Hispanic/Latino or tribal communities. As support for the additional populations, these commenters cited various studies, including some that were not included in the ISA (which we have provisionally considered, as described in section I.C above).

With regard to the former group of comments stating that the evidence does not support the identification of asthmatics as an at-risk population, we disagree. As summarized in the proposal, the EPA’s identification of populations at risk of O₃ effects is based on a systematic approach that assesses the current scientific evidence across the relevant scientific disciplines (*i.e.*, exposure sciences, dosimetry, controlled human exposure, toxicology, and epidemiology), with a focus on studies that conducted stratified analyses allowing for an evaluation of different populations exposed to similar

epidemiologic evidence, and most certainly did not select merely studies favorable to the point of view of revising the current standard.

O₃ concentrations within the same study design (U.S. EPA, 2013, pp. 8–1 to 8–3). Based on this established process and framework, the ISA identifies individuals with asthma among the populations and lifestyles for which there is “adequate” evidence to support the conclusion of increased risk of O₃-related health effects. Other populations for which the evidence is adequate are individuals with certain genotypes, younger and older age groups, individuals with reduced intake of certain nutrients, and outdoor workers. These conclusions are based on consistency in findings across studies and evidence of coherence in results from different scientific disciplines.

For example, with regard to people with asthma, the ISA notes a number of epidemiologic and controlled human exposure studies reporting larger and/or more serious effects in people with asthma than in people without asthma or other respiratory diseases. These include epidemiologic studies of lung function, respiratory symptoms, and medication use, as well as controlled human exposure studies showing larger inflammatory responses and markers indicating altered immune functioning in people with asthma, and also includes evidence from animal models of asthma that informs the EPA’s interpretation of the other studies. We disagree with the industry commenters’ focus solely on the results of certain studies without an integrated consideration of the broader body of evidence, and wider range of respiratory endpoints. It is such an integrated approach that supports EPA’s conclusion that “there is adequate evidence for asthmatics to be an at-risk population” (U.S. EPA, 2013, section 8.2.2).

We also disagree with commenters’ misleading reference to various studies cited to support the claim that asthmatics are not at increased risk of O₃-related health effects. One of the controlled human studies cited in those comments (Mudway et al. 2001) involved asthmatic adults who were older than the healthy controls, and it is well-recognized that responses to O₃ decrease with age (U.S. EPA, 2014c, p. 3–80). Another study (Alexis et al. 2000) used subjects with mild asthma who are unlikely to be as responsive as people with more severe disease (Horstman et al., 1995) (EPA 2014c, p. 3–80). Controlled human exposure studies and epidemiologic studies of adults and children amply confirm that “there is adequate evidence for asthmatics to be an at-risk population” (U.S. EPA, 2014c, p. 3–81).

⁹⁹Cf. *Coalition for Responsible Regulation v. EPA*, 684 F. 3d 102, 119 (D.C. Cir. 2012) (aff’d in part and rev’d in part on other grounds sub. nom. *UARG v. EPA*, S Ct. (2014)) (“EPA simply did here what it and other decision-makers often must do to make a science-based judgment: it sought out and reviewed existing scientific evidence to determine whether a particular finding was warranted. It makes no difference that much of the scientific evidence in large part consisted of ‘syntheses’ of individual studies and research. Even individual studies and research papers often synthesize past work in an area and then build upon it. That is how science works”).

¹⁰⁰See also section II.C.4.b below responding to comments from environmental interests that EPA inappropriately omitted many studies which (in their view) support establishing a revised standard at a level of 60 ppb or lower. Although, as explained there, the EPA disagrees with these comments, the comments illustrate that the EPA was even-handed in its consideration of the

We also do not agree with the latter group of commenters that there is sufficient evidence to support the identification of additional populations as at risk of O₃-attributable health effects. Specifically with regard to pregnant women, the ISA concluded that the “evidence is suggestive of a causal relationship between exposures to O₃ and reproductive and developmental effects” including birth outcomes, noting that “the collective evidence for many of the birth outcomes examined is generally inconsistent” (U.S. EPA, 2013, pp. 7–74 and 7–75). At the time of the completion of the ISA, no studies had been identified that examined the relationship between exposure to O₃ and the health of pregnant women (*e.g.*, studies on pre-eclampsia, gestational hypertension). Due to the generally inconsistent epidemiologic evidence for effects on birth outcomes, the lack of studies on the health of pregnant women, and the lack of studies from other disciplines to provide biological plausibility for the effects examined in epidemiologic studies, pregnant women were not considered an at-risk population. Based on the EPA’s provisional consideration of studies published since the completion of the ISA (I.C, above), recent studies that examine exposure to O₃ and pre-eclampsia and other health effects experienced by pregnant women are not sufficient to materially change the ISA’s conclusions on at-risk populations (I.C, above). In addition, as summarized in the proposal, the ISA concluded that the evidence for other populations was either suggestive of increased risk, with further investigation needed (*e.g.*, other genetic variants, obesity, sex, and socioeconomic status), or was inadequate to determine if they were of increased risk of O₃-related health effects (influenza/infection, COPD, CVD, diabetes, hyperthyroidism, smoking, race/ethnicity, and air conditioning use) (U.S. EPA, 2013, section 2.5.4.1). The CASAC has concurred with the ISA conclusions (Frey, 2014c).

c. Comments on Exposure and Risk Assessments

This section discusses major comments on the EPA’s quantitative assessments of O₃ exposures and health risks, presented in the HREA and considered in the PA, and the EPA’s responses to those comments. The focus in this section is on overarching comments related to the EPA’s approach to assessing exposures and risks, and to interpreting the exposure/risk results within the context of the adequacy of the current primary O₃ standard. More

detailed discussion of comments and Agency responses is provided in the Response to Comments document. Section II.B.2.c.i discusses comments on estimates of O₃ exposures of concern, section II.B.2.c.ii discusses comments on estimates of the risk of O₃-induced lung function decrements, and section II.B.2.b.iii discusses comments on estimates of the risk of O₃-associated mortality and morbidity.

i. O₃ Exposures of Concern

The EPA received a number of comments expressing divergent views on the estimation of, and interpretation of, O₃ exposures of concern. In general, comments from industry, business, and some state groups opposed to revising the current primary O₃ standard asserted that the approaches and assumptions that went into the HREA assessment result in overestimates of O₃ exposures. These commenters highlighted several aspects of the assessment, asserting that the HREA overestimates the proportion of the population expected to achieve ventilation rates high enough to experience an exposure of concern; that the use of out-of-date information on activity patterns results in overestimates of the amount of time people spend being active outdoors; and that exposure estimates do not account for the fact that people spend more time indoors on days with bad air quality (*i.e.*, they engage in averting behavior). In contrast, comments from medical, public health, and environmental groups that supported revision of the current standard asserted that the HREA assessment of exposures of concern, and the EPA’s interpretation of exposure estimates, understates the potential for O₃ exposures that could cause adverse health effects. These commenters claimed that the EPA’s focus on 8-hour exposures understates the O₃ impacts on public health since effects in controlled human exposure studies were shown following 6.6-hour exposures; that the HREA exposure estimates do not capture the most highly exposed populations, such as highly active children and outdoor workers; and that the EPA’s interpretation of estimated exposures of concern impermissibly relies on the assumption that people stay indoors to avoid dangerous air pollution (*i.e.*, that they engage in averting behavior).

In considering these comments, the EPA first notes that as discussed in the HREA, PA, and the proposal, there are aspects of the exposure assessment that, considered by themselves, can result in either overestimates or underestimates of the occurrence of O₃ exposures of

concern. Commenters tended to highlight the aspects of the assessment that supported their positions, including aspects that were discussed in the HREA and/or the PA and that were considered by CASAC. In contrast, commenters tended to ignore the aspects of the assessment that did not support their positions. The EPA has carefully described and assessed the significance of the various uncertainties in the exposure analysis (U.S. EPA, 2014a, Table 5–10), noting that, in most instances, the uncertainties could result in either overestimates or underestimates of exposures and that the magnitudes of the impacts on exposure results were either “low,” “low to moderate,” or “moderate” (U.S. EPA, 2014a, Table 5–10).

Consistent with the characterization of uncertainties in the HREA, PA, and the proposal, the EPA agrees with some, though not all, aspects of these commenters’ views. For example, the EPA agrees with the comment by groups opposed to revision that the equivalent ventilation rate (EVR) used to characterize individuals as at moderate or greater exertion in the HREA likely leads to overestimates of the number of individuals experiencing exposures of concern (U.S. EPA, 2014a, Table 5–10, p. 5–79). In addition, we note that other physiological processes that are incorporated into exposure estimates are also identified in the HREA as likely leading to overestimates of O₃ exposures, based on comparisons with the available scientific literature (U.S. EPA, 2014a, Table 5–10, p. 5–79). These aspects of the exposure assessment are estimated to have either a “moderate” (*i.e.*, EVR) or a “low to moderate” (*i.e.*, physiological processes) impact on exposure estimates (U.S. EPA, 2014a, Table 5–10, p. 5–79). Focusing on these aspects of the assessment, by themselves, could lead to the conclusion that the HREA overstates the occurrence of O₃ exposures of concern.

However, the EPA notes that there are also aspects of the HREA exposure assessment that, taken by themselves, could lead to the conclusion that the HREA understates the occurrence of O₃ exposures of concern. For example, as noted above, some medical, public health, and environmental groups asserted that the exposure assessment could underestimate O₃ exposures for highly active populations, including outdoor workers and children who spend a large portion of time outdoors during summer. In support of these assertions, commenters highlighted sensitivity analyses conducted in the HREA. However, as noted in the HREA (U.S. EPA, 2014a, Table 5–10), this

aspect of the assessment is likely to have a “low to moderate” impact on exposure estimates (*i.e.*, a smaller impact than uncertainty associated with the EVR, and similar in magnitude to uncertainties related to physiological processes, as noted above). Therefore, when considered in the context of all of the uncertainties in exposure estimates, it is unlikely that the HREA’s approach to using data on activity patterns leads to overall underestimates of O₃ exposures. The implications of this uncertainty are discussed in more detail below (II.C.4.b), within the context of the Administrator’s decision on a revised standard level.

In addition, medical, public health, and environmental groups also pointed out that the controlled human exposures studies that provided the basis for health effect benchmarks were conducted in healthy adults, rather than at-risk populations, and these studies evaluated 6.6 hour exposures, rather than the 8-hour exposures evaluated in the HREA exposure analyses. They concluded that adverse effects would occur at lower exposure concentrations in at-risk populations, such as people with asthma, and if people were exposed for 8 hours, rather than 6.6 hours. In its review of the PA, CASAC clearly recognized these uncertainties, which provided part of the basis for CASAC’s advice to consider exposures of concern for the 60 ppb benchmark. For example, when considering the results of the study by Schelegle et al. (2009) for 6.6-hour exposures to an average O₃ concentration of 72 ppb, CASAC judged that if subjects had been exposed for eight hours, the adverse combination of lung function decrements and respiratory symptoms “could have occurred” at lower O₃ exposure concentrations (Frey, 2014c, p. 5). With regard to at-risk populations, CASAC concluded that “based on results for clinical studies of healthy adults, and scientific considerations of differences in responsiveness of asthmatic children compared to healthy adults, there is scientific support that 60 ppb is an appropriate exposure of concern for asthmatic children” (Frey, 2014c, p. 8). As discussed below (II.B.3, II.C.4.b, II.C.4.c), based in large part on CASAC advice, the Administrator does consider exposure results for the 60 ppb benchmark.

Thus, rather than viewing the potential implications of various aspects of the HREA exposure assessment in isolation, as was done by many commenters, the EPA considers them together, along with other issues and uncertainties related to the interpretation of exposure estimates. As

discussed above, CASAC recognized the key uncertainties in exposure estimates, as well as in the interpretation of those estimates in the HREA and PA (Frey, 2014a, c). In its review of the 2nd draft REA, CASAC concluded that “[t]he discussion of uncertainty and variability is comprehensive, appropriately listing the major sources of uncertainty and their potential impacts on the APEX exposure estimates” (Frey, 2014a, p. 6). Even considering these and other uncertainties, CASAC emphasized estimates of O₃ exposures of concern as part of the basis for their recommendations on the primary O₃ NAAQS. In weighing these uncertainties, which can bias exposure results in different directions but tend to have impacts that are similar in magnitude (U.S. EPA, 2014a, Table 5–10), and in light of CASAC’s advice based on its review of the HREA and the PA, the EPA continues to conclude that the approach to considering estimated exposures of concern in the HREA, PA, and the proposal reflects an appropriate balance, and provides an appropriate basis for considering the public health protectiveness of the primary O₃ standard.

The EPA disagrees with other aspects of commenters’ views on HREA estimates of exposures of concern. For example, commenters on both sides of the issue objected to the EPA’s handling of averting behavior in exposure estimates. Some commenters who supported retaining the current standard claimed that the HREA overstates exposures of concern because available time-location-activity data do not account for averting behavior. These commenters noted sensitivity analyses in the HREA that estimated fewer exposures of concern when averting behavior was considered. In contrast, commenters supporting revision of the standard criticized the EPA’s estimates of exposures of concern, claiming that the EPA “emphasizes the role of averting behavior, noting that it may result in an overestimation of exposures of concern, and cites this behavior (essentially staying indoors or not exercising) in order to reach what it deems an acceptable level of risk” (*e.g.*, ALA et al., p. 120).

The EPA disagrees with both of these comments. In brief, the NAAQS must “be established at a level necessary to protect the health of persons,” not the health of persons refraining from normal activity or resorting to medical interventions to ward off adverse effects of poor air quality (S. Rep. No. 11–1196, 91st Cong. 2d Sess. at 10). On the other hand, ignoring normal activity patterns for a pollutant like O₃, where adverse

responses are critically dependent on ventilation rates, will result in a standard which provides more protection than is requisite. This issue is discussed in more detail below (II.C.4.b), within the context of the Administrator’s decision on a revised standard level.

These commenters also misconstrue the EPA’s limited sensitivity analyses on impacts of averting behavior in the HREA. The purpose of the HREA sensitivity analyses was to provide perspective on the potential role of averting behavior in modifying O₃ exposures. These sensitivity analyses were limited to a single urban study area, a 2-day period, and a single air quality adjustment scenario (U.S. EPA, 2014a, section 5.4.3.3). In addition, the approach used in the HREA to simulate averting behavior was itself uncertain, given the lack of actual activity pattern data that explicitly incorporated this type of behavioral response. In light of these important limitations, sensitivity analyses focused on averting behavior were discussed in the proposal within the context of the discussion of uncertainties in the HREA assessment of exposures of concern (II.C.2.b in the proposal) and, contrary to the claims of some commenters, they were not used to support the proposed decision.

Some industry groups also claimed that the time-location-activity diaries used by APEX to estimate exposures are out-of-date, and do not represent activity patterns in the current population. These commenters asserted that the use of out-of-date diary information leads to overestimates in exposures of concern. This issue was explicitly addressed in the HREA and the EPA disagrees with commenters’ conclusions. In particular, diary data was updated in this review to include data from studies published as late as 2010, directly in response to CASAC concerns. In their review of this data, CASAC stated that “[t]he addition of more recent time activity pattern data addresses a concern raised previously by the CASAC concerning how activity pattern information should be brought up to date” (Frey, 2014a, p. 8). As indicated in the HREA (U.S. EPA, 2014a, Appendix 5G, Figures 5G–7 and Figure 5G–8), the majority of diary days used in exposure simulations of children originate from the most recently conducted activity pattern studies (U.S. EPA, 2014a, Table 5–3). In addition, evaluations included in the HREA indicated that there were not major systematic differences in time-location-activity patterns based on information from older diaries versus those collected more recently (U.S. EPA,

2014a, Appendix 5G, Figures 5G–1 and 5G–2). Given all of the above, the EPA does not agree with commenters who claimed that the time-location-activity diaries used by APEX are out-of-date, and result in overestimates of exposures of concern.

ii. Risk of O₃-Induced FEV₁ Decrements

The EPA also received a large number of comments on the FEV₁ risk assessment presented in chapter 6 of the HREA (U.S. EPA, 2014a) and summarized in the proposal (II.C.3.a in the proposal). Commenters representing medical, public health, and environmental groups generally expressed the view that these risk estimates support the need to revise the current primary O₃ standard in order to increase public health protection, though these groups also questioned some of the assumptions inherent in the EPA’s interpretation of those risk estimates. For example, ALA et al. (p. 127) stated that “[t]he HREA uses a risk function derived from a controlled human exposure study of healthy young adults to estimate lung function decrements in children, including children with asthma. This assumption could result in an underestimate of risk.” On this same issue, commenters representing industry groups opposed to revising the standard also asserted that assumptions about children’s responses to O₃ exposures are highly uncertain. In contrast to medical and public health groups, these commenters concluded that this uncertainty, along with others discussed below, call into question the use of FEV₁ risk estimates to support a decision to revise the current primary O₃ standard.

The EPA agrees that an important source of uncertainty is the approach to estimating the risk of FEV₁ decrements in children and in children with asthma based on data from healthy adults. However, this issue is discussed at length in the HREA and the PA, and was considered carefully by CASAC in its review of draft versions of these documents. The conclusions of the HREA and PA, and the advice of CASAC, were reflected in the Administrator’s interpretation of FEV₁ risk estimates in the proposal, as described below. Commenters have not provided additional information that changes the EPA’s views on this issue.

As discussed in the proposal (II.C.3.a.ii in the proposal), in the near absence of controlled human exposure data for children, risk estimates are based on the assumption that children exhibit the same lung function response following O₃ exposures as healthy 18-year olds (*i.e.*, the youngest age for

which sufficient controlled human exposure data is available) (U.S. EPA, 2014a, section 6.5.3). As noted by CASAC (Frey, 2014a, p. 8), this assumption is justified in part by the findings of McDonnell et al. (1985), who reported that children (8–11 years old) experienced FEV₁ responses similar to those observed in adults (18–35 years old). The HREA concludes that this approach could result in either over- or underestimates of O₃-induced lung function decrements in children, depending on how children compare to the adults used in controlled human exposure studies (U.S. EPA, 2014a, section 6.5.3). With regard to people with asthma, although the evidence has been mixed (U.S. EPA, 2013, section 6.2.1.1), several studies have reported statistically larger, or a tendency for larger, O₃-induced lung function decrements in asthmatics than in non-asthmatics (Kreit et al., 1989; Horstman et al., 1995; Jorres et al., 1996; Alexis et al., 2000). On this issue, CASAC noted that “[a]sthmatic subjects appear to be at least as sensitive, if not more sensitive, than non-asthmatic subjects in manifesting O₃-induced pulmonary function decrements” (Frey, 2014c, p. 4). To the extent asthmatics experience larger O₃-induced lung function decrements than the healthy adults used to develop exposure-response relationships, the HREA could underestimate the impacts of O₃ exposures on lung function in asthmatics, including asthmatic children (U.S. EPA, 2014a, section 6.5.4). As noted above, these uncertainties have been considered carefully by the EPA and by CASAC during the development of the HREA and PA. In addition, the Administrator has appropriately considered these and other uncertainties in her interpretation of risk estimates, as discussed further below (II.B.3, II.C.4.b, II.C.4.c).

Some commenters additionally asserted that the HREA does not appropriately characterize the uncertainty in risk estimates for O₃-induced lung function decrements. Commenters pointed out that there is statistical uncertainty in model coefficients that is not accounted for in risk estimates. One commenter presented an analysis of this uncertainty, and concluded that there is considerable overlap between risk estimates for standard levels of 75, 70, and 65 ppb, undercutting the confidence in estimated risk reductions for standard levels below 75 ppb.

The Agency recognizes that there are important sources of uncertainty in the FEV₁ risk assessment. In some cases, these sources of uncertainty can

contribute to substantial variability in risk estimates, complicating the interpretation of those estimates. For example, as discussed in the proposal, the variability in FEV₁ risk estimates across urban study areas is often greater than the differences in risk estimates between various standard levels (Table 2, above and 79 FR 75306 n. 164). Given this, and the resulting considerable overlap between the ranges of FEV₁ risk estimates for different standard levels, in the proposal the Administrator viewed these risk estimates as providing a more limited basis than exposures of concern for distinguishing between the degree of public health protection provided by alternative standard levels. Thus, although the EPA does not agree with the overall conclusions of industry commenters, their analysis of statistical uncertainty in risk estimates, and the resulting overlap between risk estimates for standard levels of 75, 70, and 65 ppb, tends to reinforce the Administrator’s approach, which places greater weight on estimates of O₃ exposures of concern than on risk estimates for O₃-induced FEV₁ decrements.

iii. Risk of O₃-Associated Mortality and Morbidity

In the proposal, the Administrator placed the greatest emphasis on the results of controlled human exposure studies and on quantitative analyses based on information from these studies, and less weight on mortality and morbidity risk assessments based on information from epidemiology studies. The EPA received a number of comments on its consideration of epidemiology-based risks, with some commenters expressing support for the Agency’s approach and others expressing opposition.

In general, commenters representing industry organizations or states opposed to revising the current primary O₃ standard agreed with the Administrator’s approach in the proposal to viewing epidemiology-based risk estimates, though these commenters reached a different conclusion than the EPA regarding the adequacy of the current standard. In supporting their views, these commenters highlighted a number of uncertainties in the underlying epidemiologic studies, and concluded that risk estimates based on information from such studies do not provide an appropriate basis for revising the current standard. For example, commenters noted considerable spatial heterogeneity in health effect associations; the potential for co-occurring pollutants (*e.g.*, PM_{2.5}) to confound O₃ health effect associations;

and the lack of statistically significant O₃ health effect associations in many of the individual cities evaluated as part of multicity analyses. In contrast, some commenters representing medical, public health, or environmental organizations placed greater emphasis than the EPA on epidemiology-based risk estimates. These commenters asserted that risk estimates provide strong support for a lower standard level, and pointed to CASAC advice to support their position.

As in the proposal, the EPA continues to place the greatest weight on the results of controlled human exposure studies and on quantitative analyses based on information from these studies (particularly exposures of concern, as discussed below in II.B.3 and II.C.4), and less weight on risk analyses based on information from epidemiologic studies. In doing so, the Agency continues to note that controlled human exposure studies provide the most certain evidence indicating the occurrence of health effects in humans following specific O₃ exposures. In addition, the effects reported in these studies are due solely to O₃ exposures, and interpretation of study results is not complicated by the presence of co-occurring pollutants or pollutant mixtures (as is the case in epidemiologic studies). The Agency further notes the CASAC judgment that “the scientific evidence supporting the finding that the current standard is inadequate to protect public health is strongest based on the controlled human exposure studies of respiratory effects” (Frey, 2014c, p. 5). Consistent with this emphasis, the HREA conclusions reflect relatively greater confidence in the results of the exposure and risk analyses based on information from controlled human exposure studies than the results of epidemiology-based risk analyses. As discussed in the HREA (U.S. EPA, 2014a, section 9.6), several key uncertainties complicate the interpretation of these epidemiology-based risk estimates, including the heterogeneity in O₃ effect estimates between locations, the potential for exposure measurement errors in these epidemiologic studies, and uncertainty in the interpretation of the shape of concentration-response functions at lower O₃ concentrations. Commenters who opposed the EPA’s approach in the proposal to viewing the results of quantitative analyses tended to highlight aspects of the evidence and CASAC advice that were considered by the EPA at the time of proposal and nothing in these commenters’ views has changed those considerations.

Therefore, the EPA continues to place the most emphasis on using the information from controlled human exposure studies to inform consideration of the adequacy of the primary O₃ standard.

However, while the EPA agrees that there are important uncertainties in the O₃ epidemiology-based risk estimates, the Agency disagrees with industry commenters that these uncertainties support a conclusion to retain the current standard. As discussed below, the decision to revise the current primary O₃ standard is based on the EPA’s consideration of the broad body of scientific evidence, quantitative analyses of O₃ exposures and risks, CASAC advice, and public comments. While recognizing uncertainties in the epidemiology-based risk estimates here, and giving these uncertainties appropriate consideration, the Agency continues to conclude that these risk estimates contribute to the broader body of evidence and information supporting the need to revise the primary O₃ standard.

Some commenters opposed to revising the current O₃ standard highlighted the fact that, in a few urban study locations, larger risks are estimated for standard levels below 75 ppb than for the current standard with its level of 75 ppb. For example, TCEQ (p. 3) states that “differential effects on ozone in urban areas also lead to the EPA’s modeled increases in mortality in Houston and Los Angeles with decreasing ozone standards.” These commenters cited such increases in estimated risk as part of the basis for their conclusion that the current standard should be retained.

For communities across the U.S. (including in the Houston and Los Angeles areas), exposure and risk analyses indicate that reducing emissions of O₃ precursors (NO_x, VOCs) to meet a revised standard with a level of 70 ppb will substantially reduce the occurrence of adverse respiratory effects and mortality risk attributable to high O₃ concentrations (U.S. EPA, 2014a, Appendix 9A; U.S. EPA, 2014c, sections 4.4.2.1 to 4.4.2.3). However, because of the complex chemistry governing the formation and destruction of O₃, some NO_x control strategies designed to reduce the highest ambient O₃ concentrations can also result in increases in relatively low ambient O₃ concentrations. As a result of the way the EPA’s epidemiology-based risk assessments were conducted (U.S. EPA, 2014a, Chapter 7), increases estimated in low O₃ concentrations impacted mortality and morbidity risks, leading to the estimated risk increases highlighted

by some commenters. However, while the EPA is confident that reducing the highest ambient O₃ concentrations will result in substantial improvements in public health, including reducing the risk of O₃-associated mortality, the Agency is far less certain about the public health implications of the changes in relatively low ambient O₃ concentrations (79 FR at 75278/3, 75291/1, and 75308/2). Therefore, reducing precursor emissions to meet a lower O₃ standard is expected to result in important reductions in O₃ concentrations from the part of the air quality distribution where the evidence provides the strongest support for adverse health effects.

Specifically, for area-wide O₃ concentrations at or above 40 ppb,¹⁰¹ a revised standard with a level of 70 ppb is estimated to reduce the number of premature deaths associated with short-term O₃ concentrations by about 10%, compared to the current standard. In addition, for area-wide concentrations at or above 60 ppb, a revised standard with a level of 70 ppb is estimated to reduce O₃-associated premature deaths by about 50% to 70%.¹⁰² The EPA views these results, which focus on the portion of the air quality distribution where the evidence indicates the most certainty regarding the occurrence of adverse O₃-attributable health effects, not only as supportive of the need to revise the current standard (II.B.3, below), but also as showing the benefits of reducing the peak O₃ concentrations associated with air quality distributions meeting the current standard (II.C.4, below).

In addition, even considering risk estimates based on the full distribution of ambient O₃ concentrations (*i.e.*, estimates influenced by decreases in higher concentrations and increases in lower concentrations), the EPA notes that, compared to the current standard, standards with lower levels are estimated to result in overall reductions in mortality risk across the urban study areas evaluated (U.S. EPA, 2014c, Figure 4–10). As discussed above (II.A.2.a, II.A.2.c), analyses in the HREA indicate that these overall risk reductions could understate the actual reductions that

¹⁰¹ The ISA concludes that there is less certainty in the shape of concentration-response functions for area-wide O₃ concentrations at the lower ends of warm season distributions (*i.e.*, below about 20 to 40 ppb) (U.S. EPA, 2013, section 2.5.4.4).

¹⁰² Available experimental studies provide the strongest evidence for O₃-induced effects following exposures to O₃ concentrations corresponding to the upper portions of typical ambient distributions. In particular, as discussed above, controlled human exposure studies showing respiratory effects following exposures to O₃ concentrations at or above 60 ppb.

would be experienced by the U.S. population as a whole.

For example, the HREA's national air quality modeling analyses indicate that the HREA urban study areas tend to underrepresent the populations living in areas where reducing NO_x emissions would be expected to result in decreases in warm season averages of daily maximum 8-hour ambient O₃ concentrations.¹⁰³ Given the strong connection between these warm season average O₃ concentrations and risk, risk estimates for the urban study areas are likely to understate the average reductions in O₃-associated mortality and morbidity risks that would be experienced across the U.S. population as a whole upon reducing NO_x emissions (U.S. EPA, 2014a, section 8.2.3.2).

In addition, in recognizing that the reductions in modeled NO_x emissions used in the HREA's core analyses are meant to be illustrative, rather than to imply a particular control strategy for meeting a revised O₃ NAAQS, the HREA also conducted sensitivity analyses in which both NO_x and VOC emissions reductions were evaluated. In all of the urban study areas evaluated in these analyses, the increases in low O₃ concentrations were smaller for the NO_x/VOC emission reduction scenarios than the NO_x only emission reduction scenario (U.S. EPA, 2014a, Appendix 4D, section 4.7). This was most apparent for Denver, Houston, Los Angeles, New York, and Philadelphia. These results suggest that in some locations, optimized emissions reduction strategies could result in larger reductions in O₃-associated mortality and morbidity than indicated by HREA's core estimates.

Thus, the patterns of estimated mortality and morbidity risks across various air quality scenarios and locations have been evaluated and considered extensively in the HREA and the PA, as well as in the proposal. Epidemiology-based risk estimates have also been considered by CASAC, and those considerations are reflected in CASAC's advice. Specifically, in considering epidemiology-based risk estimates in its review of the REA, CASAC stated that "[a]lthough these estimates for short-term exposure impacts are subject to uncertainty, the CASAC is confident that that the evidence of health effects of O₃

¹⁰³ Specifically, the HREA urban study areas tend to underrepresent populations living in suburban, smaller urban, and rural areas, where reducing NO_x emissions would be expected to result in decreases in warm season averages of daily maximum 8-hour ambient O₃ concentrations (U.S. EPA, 2014a, section 8.2.3.2).

presented in the ISA and Second Draft HREA in its totality, indicates that there are meaningful reductions in mean, absolute, and relative premature mortality associated with short-term exposures to O₃ levels lower than the current standard" (Frey, 2014a, p. 3). Commenters' views on this issue are not based on new information, but on an interpretation of the analyses presented in the HREA that is different from the EPA's, and CASAC's, interpretation. Given this, the EPA's considerations and conclusions related to this issue, as described in the proposal and as summarized briefly above, remain valid. Therefore, the EPA does not agree with commenters who cited increases in estimated risk in some locations as supporting a conclusion that the current standard should be retained.

For risk estimates of respiratory mortality associated with long-term O₃, several industry commenters supported placing more emphasis on threshold models, and including these models as part of the core analyses rather than as sensitivity analyses. The EPA agrees with these commenters that an important uncertainty in risk estimates of respiratory mortality associated with long-term O₃ stems from the potential for the existence of a threshold. Based on sensitivity analyses included in the HREA in response to CASAC advice, the existence of a threshold could substantially reduce estimated risks. CASAC discussed this issue at length during its review of the REA and supported the EPA's approach to including a range of threshold models as sensitivity analyses (Frey, 2014a p. 3). Based in part on uncertainty in the existence and identification of a threshold, the HREA concluded that lower confidence should be placed in risk estimates for respiratory mortality associated with long-term O₃ exposures (U.S. EPA, 2014a, section 9.6). This uncertainty was also a key part of the Administrator's rationale for placing only limited emphasis on risk estimates for long-term O₃ exposures. In her final decisions, discussed below (II.B.3, II.C.4.b, II.C.4.c), the Administrator continues to place only limited emphasis on these estimates. The EPA views this approach to considering risk estimates for respiratory mortality as generally consistent with the approach supported by the commenters noted above.

3. Administrator's Conclusions on the Need for Revision

This section discusses the Administrator's conclusions related to the adequacy of the public health protection provided by the current

primary O₃ standard, and her final decision that the current standard is not requisite to protect public health with an adequate margin of safety. These conclusions, and her final decision, are based on the Administrator's consideration of the available scientific evidence assessed in the ISA (U.S. EPA, 2013), the exposure/risk information presented and assessed in the HREA (U.S. EPA, 2014a), the consideration of that evidence and information in the PA (U.S. EPA, 2014c), the advice of CASAC, and public comments received on the proposal.

As an initial matter, the Administrator concludes that reducing precursor emissions to achieve O₃ concentrations that meet the current primary O₃ standard will provide important improvements in public health protection, compared to recent air quality. In reaching this conclusion, she notes the discussion in section 3.4 of the PA (U.S. EPA, 2014c). In particular, the Administrator notes that this conclusion is supported by (1) the strong body of scientific evidence indicating a wide range of adverse health outcomes attributable to exposures to O₃ at concentrations commonly found in the ambient air and (2) estimates indicating decreased occurrences of O₃ exposures of concern and decreased O₃-associated health risks upon meeting the current standard, compared to recent air quality. Thus, she concludes that it would not be appropriate in this review to consider a standard that is less protective than the current standard.

After reaching the conclusion that meeting the current primary O₃ standard will provide important improvements in public health protection, and that it is not appropriate to consider a standard that is less protective than the current standard, the Administrator next considers the adequacy of the public health protection that is provided by the current standard. In doing so, the Administrator first notes that studies evaluated since the completion of the 2006 AQCD support and expand upon the strong body of evidence that, in the last review, indicated a causal relationship between short-term O₃ exposures and respiratory morbidity outcomes (U.S. EPA, 2013, section 2.5). This is the strongest causality finding possible under the ISA's hierarchical system for classifying weight of evidence for causation. In addition, the Administrator notes that the evidence for respiratory health effects attributable to long-term O₃ exposures, including the development of asthma in children, is much stronger than in previous reviews, and the ISA concludes that there is "likely to be" a causal relationship

between such O₃ exposures and adverse respiratory health effects (the second strongest causality finding).

Together, experimental and epidemiologic studies support conclusions regarding a continuum of O₃ respiratory effects ranging from small, reversible changes in pulmonary function, and pulmonary inflammation, to more serious effects that can result in respiratory-related emergency department visits, hospital admissions, and premature mortality. Recent animal toxicology studies support descriptions of modes of action for these respiratory effects and augment support for biological plausibility for the role of O₃ in reported effects. With regard to mode of action, evidence indicates that the initial key event is the formation of secondary oxidation products in the respiratory tract, that antioxidant capacity may modify the risk of respiratory morbidity associated with O₃ exposure, and that the inherent capacity to quench (based on individual antioxidant capacity) can be overwhelmed, especially with exposure to elevated concentrations of O₃.

In addition, based on the consistency of findings across studies and the coherence of results from different scientific disciplines, the available evidence indicates that certain populations are at increased risk of experiencing O₃-related effects, including the most severe effects. These include populations and lifestyles identified in previous reviews (*i.e.*, people with asthma, children, older adults, outdoor workers) and populations identified since the last review (*i.e.*, people with certain genotypes related to antioxidant and/or anti-inflammatory status; people with reduced intake of certain antioxidant nutrients, such as Vitamins C and E).

In considering the O₃ exposure concentrations reported to elicit respiratory effects, as in the proposal, the Administrator agrees with the conclusions of the PA that controlled human exposure studies provide the most certain evidence indicating the occurrence of health effects in humans following specific O₃ exposures. In particular, she notes that the effects reported in controlled human exposure studies are due solely to O₃ exposures, and interpretation of study results is not complicated by the presence of co-occurring pollutants or pollutant mixtures (as is the case in epidemiologic studies). Therefore, consistent with CASAC advice (Frey, 2014c), she places the most weight on information from controlled human exposure studies in reaching conclusions on the adequacy of the current primary O₃ standard.

In considering the evidence from controlled human exposure studies, the Administrator first notes that these studies have reported a variety of respiratory effects in healthy adults following exposures to O₃ concentrations of 60, 63,¹⁰⁴ 72,¹⁰⁵ or 80 ppb, and higher. The largest respiratory effects, and the broadest range of effects, have been studied and reported following exposures of healthy adults to 80 ppb O₃ or higher, with most exposure studies conducted at these higher concentrations. As discussed above (II.A.1), the Administrator further notes that recent evidence includes controlled human exposure studies reporting the combination of lung function decrements and respiratory symptoms in healthy adults engaged in moderate exertion following 6.6-hour exposures to concentrations as low as 72 ppb, and lung function decrements and pulmonary inflammation following exposures to O₃ concentrations as low as 60 ppb.

As discussed in her response to public comments above (II.B.2.b.i), and in detail below (II.C.4.b, II.C.4.c), the Administrator concludes that these controlled human exposure studies indicate that adverse effects are likely to occur following exposures to O₃ concentrations below the level of the current standard. The effects observed following such exposures are coherent with the serious health outcomes that have been reported in O₃ epidemiologic studies (*e.g.*, respiratory-related hospital admissions, emergency department visits), and the Administrator judges that such effects have the potential to be important from a public health perspective.

In reaching these conclusions, she particularly notes that the combination of lung function decrements and respiratory symptoms reported to occur in healthy adults following exposures to 72 ppb O₃ meets ATS criteria for an adverse response (II.B.2.b.i, above). In specifically considering the 72 ppb exposure concentration, CASAC noted that “the combination of decrements in FEV₁ together with the statistically significant alterations in symptoms in human subjects exposed to 72 ppb ozone meets the American Thoracic Society’s definition of an adverse health effect” (Frey, 2014c, p. 5). In addition, given that the controlled human exposure study reporting these results was conducted in healthy adults,

¹⁰⁴ For a 60 ppb target exposure concentration, Schelegle et al. (2009) reported that the actual 6.6-hour mean exposure concentration was 63 ppb.

¹⁰⁵ For a 70 ppb target exposure concentration, Schelegle et al. (2009) reported that the actual 6.6-hour mean exposure concentration was 72 ppb.

CASAC judged that the adverse combination of lung function decrements and respiratory symptoms “almost certainly occur in some people” (*e.g.*, people with asthma) following exposures to lower O₃ concentrations (Frey, 2014c, p. 6).

While the Administrator is less certain regarding the adversity of the lung function decrements and airway inflammation that have been observed following exposures as low as 60 ppb, as discussed in more detail elsewhere in this preamble (II.B.2.b.i, II.C.4.b, II.C.4.c), she judges that these effects also have the potential to be adverse, and to be of public health importance, particularly if they are experienced repeatedly. With regard to this judgment, she specifically notes the ISA conclusion that, while the airway inflammation induced by a single exposure (or several exposures over the course of a summer) can resolve entirely, continued inflammation could potentially result in adverse effects, including the induction of a chronic inflammatory state; altered pulmonary structure and function, leading to diseases such as asthma; altered lung host defense response to inhaled microorganisms; and altered lung response to other agents such as allergens or toxins (U.S. EPA, 2013, section 6.2.3). Thus, the Administrator becomes increasingly concerned about the potential for adverse effects at 60 ppb O₃ as the number of exposures increases, though she notes that the available evidence does not indicate a particular number of occurrences of such exposures that would be required to achieve an adverse respiratory effect, and that this number is likely to vary across the population.

In addition to controlled human exposure studies, the Administrator also considers what the available epidemiologic evidence indicates with regard to the adequacy of the public health protection provided by the current primary O₃ standard. She notes that recent epidemiologic studies provide support, beyond that available in the last review, for associations between short-term O₃ exposures and a wide range of adverse respiratory outcomes (including respiratory-related hospital admissions, emergency department visits, and mortality) and with total mortality. As discussed above in the EPA responses to public comments (II.B.2.b.ii), associations with morbidity and mortality are stronger during the warm or summer months, and remain robust after adjustment for copollutants (U.S. EPA, 2013, Chapter 6).

In considering information from epidemiologic studies within the context of her conclusions on the adequacy of the current standard, the Administrator specifically considers analyses in the PA that evaluate the extent to which O₃ health effect associations have been reported for air quality concentrations likely to be allowed by the current standard. She notes that such analyses can provide insight into the extent to which the current standard would allow the distributions of ambient O₃ concentrations that provided the basis for these health effect associations. While the majority of O₃ epidemiologic studies evaluated in the PA were conducted in areas that would have violated the current standard during study periods, as discussed above (II.B.2.b.ii), the Administrator observes that the study by Mar and Koenig (2009) reported associations between short-term O₃ concentrations and asthma emergency department visits in children and adults in a U.S. location that would have met the current O₃ standard over the entire study period.¹⁰⁶ Based on this, she notes the conclusion from the PA that the current primary O₃ standard would have allowed the distribution of ambient O₃ concentrations that provided the basis for the associations with asthma emergency department visits reported by Mar and Koenig (2009) (U.S. EPA, 2014c, section 3.1.4.2).

In addition, even in some single-city study locations where the current standard was violated (*i.e.*, those evaluated in Silverman and Ito, 2010; Strickland et al., 2010), the Administrator notes that PA analyses of reported concentration-response functions and available air quality data support the occurrence of O₃-attributable hospital admissions and emergency department visits on subsets of days with virtually all ambient O₃ concentrations below the level of the current standard. PA analyses of study area air quality further support the conclusion that exposures to the ambient O₃ concentrations present in the locations evaluated by Strickland et al. (2010) and Silverman and Ito (2010) could have plausibly resulted in the respiratory-related emergency department visits and hospital admissions reported in these studies (U.S. EPA, 2014c, section 3.1.4.2). The Administrator agrees with the PA

¹⁰⁶ The large majority of locations evaluated in U.S. epidemiologic studies of long-term O₃ would have violated the current standard during study periods, thus providing limited insight into the adequacy of the current standard (U.S. EPA, 2014c, section 3.1.4.3).

conclusion that these analyses indicate a relatively high degree of confidence in reported statistical associations with respiratory health outcomes on days when virtually all monitored 8-hour O₃ concentrations were 75 ppb or below. She further agrees with the PA conclusion that although these analyses do not identify true design values, the presence of O₃-associated respiratory effects on such days provides insight into the types of health effects that could occur in locations with maximum ambient O₃ concentrations below the level of the current standard.

Compared to the single-city epidemiologic studies discussed above, the Administrator notes additional uncertainty in interpreting the relationships between short-term O₃ air quality in individual study cities and reported O₃ multicity effect estimates. In particular, she judges that the available multicity effect estimates in studies of short-term O₃ do not provide a basis for considering the extent to which reported O₃ health effect associations are influenced by individual locations with ambient O₃ concentrations low enough to meet the current O₃ standard, versus locations with O₃ concentrations that violate this standard.¹⁰⁷ While such uncertainties limit the extent to which the Administrator bases her conclusions on air quality in locations of multicity epidemiologic studies, she does note that O₃ associations with respiratory morbidity or premature mortality have been reported in several multicity studies when the majority of study locations (though not all study locations) would have met the current O₃ standard (U.S. EPA, 2014c, section 3.1.4.2).

Looking across the body of epidemiologic evidence, the Administrator thus reaches the conclusion that analyses of air quality in study locations support the occurrence of adverse O₃-associated effects at ambient O₃ concentrations that met, or are likely to have met, the current standard. She further concludes that the strongest support for this conclusion comes from single-city studies of

¹⁰⁷ As noted in the proposal (II.E.4.d), this uncertainty applies specifically to interpreting air quality analyses within the context of multicity effect estimates for short-term O₃ concentrations, where effect estimates for individual study cities are not presented (as is the case for the key O₃ studies analyzed in the PA, with the exception of the study by Stieb et al. (2009) where none of the city-specific effect estimates for asthma emergency department visits were statistically significant). This specific uncertainty does not apply to multicity epidemiologic studies of long-term O₃ concentrations, where multicity effect estimates are based on comparisons across cities. For example, see discussion of study by Jerrett et al. (2009) in the PA (U.S. EPA, 2014c, section 3.1.4.3).

respiratory-related hospital admissions and emergency department visits associated with short-term O₃ concentrations, with some support also from multicity studies of morbidity or mortality.

Taken together, the Administrator concludes that the scientific evidence from controlled human exposure and epidemiologic studies calls into question the adequacy of the public health protection provided by the current standard. In reaching this conclusion, she particularly notes that the current standard level is higher than the lowest O₃ exposure concentration shown to result in the adverse combination of lung function decrements and respiratory symptoms (*i.e.*, 72 ppb), and that CASAC concluded that such effects “almost certainly occur in some people” following exposures to O₃ concentrations below 72 ppb (Frey, 2014c, p. 6). While she also notes that the current standard level is well-above the lowest O₃ exposure concentration shown to cause respiratory effects (*i.e.*, 60 ppb), she has less confidence that the effects observed at 60 ppb are adverse (discussed in II.B.2.b.i, II.C.4.b, II.C.4.c). She further considers these effects, and the extent to which the current primary O₃ standard could protect against them, within the context of quantitative analyses of O₃ exposures (discussed below). With regard to the available epidemiologic evidence, the Administrator notes PA analyses of O₃ air quality indicating that, while most O₃ epidemiologic studies reported health effect associations with ambient O₃ concentrations that violated the current standard, a small number of single-city U.S. studies support the occurrence of asthma-related hospital admissions and emergency department visits at ambient O₃ concentrations below the level of the current standard, including one study with air quality that would have met the current standard during the study period. Some support for such O₃ associations is also provided by multicity studies of morbidity or mortality. The Administrator further judges that the biological plausibility of associations with clearly adverse morbidity effects is supported by the evidence noted above from controlled human exposure studies conducted at, or in some cases below, typical warm-season ambient O₃ concentrations.

Beyond her consideration of the scientific evidence, the Administrator also considers the results of the HREA exposure and risk analyses in reaching final conclusions regarding the adequacy of the current primary O₃ standard. In doing so, consistent with

her consideration of the evidence, she focuses primarily on quantitative analyses based on information from controlled human exposure studies (*i.e.*, exposures of concern and risk of O₃-induced FEV₁ decrements). Consistent with the considerations in the PA, and with CASAC advice (Frey, 2014c), she particularly focuses on exposure and risk estimates in children.¹⁰⁸ As discussed in the HREA and PA (and II.B, above), the patterns of exposure and risk estimates across urban study areas, across years, and across air quality scenarios are similar in children and adults though, because children spend more time being physically active outdoors and are more likely to experience the types of O₃ exposures shown to cause respiratory effects, larger percentages of children are estimated to experience exposures of concern and O₃-induced FEV₁ decrements. Children also have intrinsic risk factors that make them particularly susceptible to O₃-related effects (*e.g.*, higher ventilation rates relative to lung volume) (U.S. EPA, 2013, section 8.3.1.1; see section II.A.1.d above). In focusing on exposure and risk estimates in children, the Administrator recognizes that the exposure patterns for children across years, urban study areas, and air quality scenarios are indicative of the exposure patterns in a broader group of at-risk populations that also includes asthmatic adults and older adults. She judges that, to the extent the primary O₃ standard provides appropriate protection for children, it will also do so for adult populations,¹⁰⁹ given the larger exposures and intrinsic risk factors in children.

In first considering estimates of exposures of concern, the Administrator considers the extent to which estimates indicate that the current standard limits population exposures to the broader range of O₃ concentrations shown in controlled human exposure studies to cause respiratory effects. In doing so, she focuses on estimates of O₃

¹⁰⁸ She focuses on estimates for all children and estimates for children with asthma, noting that exposure and risk estimates for these groups are virtually indistinguishable in terms of the percent estimated to experience exposures of concern or O₃-induced FEV₁ decrements (U.S. EPA, 2014c, sections 3.2 and 4.4.2).

¹⁰⁹ As noted below (II.C.4.2), this includes populations of highly active adults, such as outdoor workers. Limited sensitivity analyses in the HREA indicate that when diaries were selected to mimic exposures that could be experienced by outdoor workers, the percentages of modeled individuals estimated to experience exposures of concern were generally similar to the percentages estimated for children (*i.e.*, using the full database of diary profiles) in the urban study areas and years with the largest exposure estimates (U.S. EPA, 2014, section 5.4.3.2, Figure 5–14).

exposures of concern at or above the benchmark concentrations of 60, 70, and 80 ppb. She notes that the current O₃ standard can provide some protection against exposures of concern to a range of O₃ concentrations, including concentrations below the standard level, given that (1) with the current fourth-high form, most days will have concentrations below the standard level and that (2) exposures of concern depend on both the presence of relatively high ambient O₃ concentrations and on activity patterns in the population that result in exposures to such high concentrations while at an elevated ventilation rate (discussed in detail below, II.C.4.b and II.C.4.c).

In considering estimates of O₃ exposures of concern allowed by the current standard, she notes that while single exposures of concern could be adverse for some people, particularly for the higher benchmark concentrations (70, 80 ppb) where there is stronger evidence for the occurrence of adverse effects (II.B.2.b.i, II.C.4.b, II.C.4.c, below), she becomes increasingly concerned about the potential for adverse responses as the number of occurrences increases.¹¹⁰ In particular, as discussed above with regard to inflammation, she notes that the types of lung injury shown to occur following exposures to O₃ concentrations from 60 to 80 ppb, particularly if experienced repeatedly, provide a mode of action by which O₃ may cause other more serious effects (*e.g.*, asthma exacerbations). Therefore, the Administrator places the most weight on estimates of two or more exposures of concern (*i.e.*, as a surrogate for the occurrence of repeated exposures), though she also considers estimates of one or more exposures for the 70 and 80 ppb benchmarks.

In considering estimates of exposures of concern, the Administrator first notes that if the 15 urban study areas evaluated in the HREA were to just meet the current O₃ standard, fewer than 1% of children in those areas would be estimated to experience two or more exposures of concern at or above 70 ppb, based on exposure estimates averaged over the years of analysis, though up to about 2% would be estimated to experience such exposures in the worst-case year and location (*i.e.*, year and location with the largest

¹¹⁰ Not all people who experience an exposure of concern will experience an adverse effect (even members of at-risk populations). For the endpoints evaluated in controlled human exposure studies, the number of those experiencing exposures of concern who will experience adverse effects cannot be reliably quantified.

exposure estimates).¹¹¹ Although the Administrator is less concerned about single occurrences of exposures of concern, she notes that even single occurrences could cause adverse effects in some people, particularly for the 70 and 80 ppb benchmarks.¹¹² As illustrated in Table 1 (above), the current standard could allow up to about 3% of children to experience one or more exposures of concern at or above 70 ppb, averaged over the years of analysis, and up to about 8% in the worst-case year and location. In addition, in the worst-case year and location, the current standard could allow about 1% of children to experience at least one exposure of concern at or above 80 ppb, the highest benchmark evaluated.

While the Administrator has less confidence in the adversity of the effects observed following exposures to 60 ppb O₃ (II.B.2.b.i, II.C.4.b, II.C.4.c), particularly for single exposures, she judges that the potential for adverse effects increases as the number of exposures of concern increases. With regard to the 60 ppb benchmark, she particularly notes that the current standard is estimated to allow approximately 3 to 8% of children in urban study areas, including approximately 3 to 8% of asthmatic children, to experience two or more exposures of concern to O₃ concentrations at or above 60 ppb, based on estimates averaged over the years of analysis. To provide some perspective on the average percentages estimated, the Administrator notes that they correspond to almost 900,000 children in urban study areas, including about 90,000 asthmatic children. Nationally, if the current standard were to be just met, the number of children experiencing such exposures would be larger.

Based on her consideration of these estimates within the context of her judgments on adversity, as discussed in her responses to public comments (II.B.2.b.i, II.C.4.b), the Administrator concludes that the exposures projected to remain upon meeting the current standard can reasonably be judged to be important from a public health perspective. In particular, given that the average percent of children estimated to experience two or more exposures of concern for the 60 ppb benchmark approaches 10% in some areas, even based on estimates averaged over the

¹¹¹ Virtually no children in those areas would be estimated to experience two or more exposures of concern at or above 80 ppb.

¹¹² That is, adverse effects are a possible outcome of single exposures of concern at/above 70 or 80 ppb, though the available information is not sufficient to estimate the likelihood of such effects.

years of the analysis, she concludes that the current standard does not incorporate an adequate margin of safety against the potentially adverse effects that can occur following repeated exposures at or above 60 ppb. Although she has less confidence that the effects observed at 60 ppb are adverse, compared to the effects at and above 72 ppb, she judges that this approach to considering the results for the 60 ppb benchmark is appropriate given CASAC advice, which clearly focuses the EPA on considering the effects observed at 60 ppb (Frey, 2014c) (II.C.4.b, II.C.4.c below).¹¹³ This approach to considering estimated exposures of concern is consistent with setting standards that provide some safeguard against dangers to human health that are not fully certain (*i.e.*, standards that incorporate an adequate margin of safety) (See, *e.g.*, *State of Mississippi*, 744 F. 3d at 1353).

In addition to estimated exposures of concern, the Administrator also considers HREA estimates of the risk of O₃-induced FEV₁ decrements ≥ 10 and 15%. In doing so, she particularly notes CASAC advice that “estimation of FEV₁ decrements of $\geq 15\%$ is appropriate as a scientifically relevant surrogate for adverse health outcomes in active healthy adults, whereas an FEV₁ decrement of $\geq 10\%$ is a scientifically relevant surrogate for adverse health outcomes for people with asthma and lung disease” (Frey, 2014c, p. 3). The Administrator notes that while single occurrences of O₃-induced lung function decrements could be adverse for some people, as discussed above (II.B.1), she agrees with the judgment in past reviews that a more general consensus view of the potential adversity of such decrements emerges as the frequency of occurrences increases. Therefore, as in the proposal, the Administrator focuses primarily on the estimates of two or more O₃-induced lung function decrements. When averaged over the years evaluated in the HREA, the Administrator notes that the current standard is estimated to allow about 1 to 3% of children in the 15 urban study areas (corresponding to almost 400,000 children) to experience two or more O₃-induced lung function decrements $\geq 15\%$, and to allow about 8 to 12% of children (corresponding to about 180,000 asthmatic children) to experience two or more O₃-induced lung function decrements $\geq 10\%$.

In further considering the HREA results, the Administrator considers the

¹¹³ Though this advice is less clear regarding the adversity of effects at 60 ppb than CASAC’s advice regarding the adversity of effects at 72 ppb (II.C.4.b, II.C.4.c).

epidemiology-based risk estimates. As discussed in the proposal, compared to the weight given to HREA estimates of exposures of concern and lung function risks, she places relatively less weight on epidemiology-based risk estimates. In giving some consideration to these risk estimates, as discussed in the proposal and above in the EPA’s responses to public comments (II.B.2.b.iii), the Administrator focuses on the risks associated with O₃ concentrations in the upper portions of ambient distributions. In doing so, she notes the increasing uncertainty associated with the shapes of concentration-response curves for O₃ concentrations in the lower portions of ambient distributions and the evidence from controlled human exposure studies, which provide the strongest support for O₃-induced effects following exposures to O₃ concentrations corresponding to the upper portions of typical ambient distributions (*i.e.*, 60 ppb and above). Even when considering only area-wide O₃ concentrations from the upper portions of seasonal distributions (*i.e.*, ≥ 40 , 60 ppb, Table 3 in the proposal), the Administrator notes that the general magnitude of mortality risk estimates suggests the potential for a substantial number of O₃-associated deaths and adverse respiratory events to occur nationally, even when the current standard is met (79 FR 75277 and II.B.2.c.iii above).

In addition to the evidence and exposure/risk information discussed above, the Administrator also takes note of the CASAC advice in the current review, in the 2008 review and decision establishing the current standard, and in the 2010 reconsideration of the 2008 decision. As discussed in more detail above, the current CASAC “finds that the current NAAQS for ozone is not protective of human health” and “unanimously recommends that the Administrator revise the current primary ozone standard to protect public health” (Frey, 2014c, p. 5). The prior CASAC O₃ Panel likewise recommended revision of the current standard to one with a lower level due to the lack of protectiveness of the current standard. This earlier recommendation was based entirely on the evidence and information in the record for the 2008 standard decision, which, as discussed above, has been substantially strengthened in the current review (Samet, 2011; Frey and Samet, 2012).

In consideration of all of the above, the Administrator concludes that the current primary O₃ standard is not requisite to protect public health with an adequate margin of safety, and that

it should be revised to provide increased public health protection. This decision is based on the Administrator’s conclusions that the available evidence and exposure and risk information clearly call into question the adequacy of public health protection provided by the current primary standard such that it is not appropriate, within the meaning of section 109(d)(1) of the CAA, to retain the current standard. With regard to the evidence, she particularly notes that the current standard level is higher than the lowest O₃ exposure concentration shown to result in the adverse combination of lung function decrements and respiratory symptoms (*i.e.*, 72 ppb), and also notes CASAC’s advice that at-risk groups (*e.g.*, people with asthma) could experience adverse effects following exposure to lower concentrations. In addition, while the Administrator is less certain about the adversity of the effects that occur following lower exposure concentrations, she judges that recent controlled human exposure studies at 60 ppb provide support for a level below 75 ppb in order to provide an increased margin of safety, compared to the current standard, against effects with the potential to be adverse, particularly if they are experienced repeatedly. With regard to O₃ epidemiologic studies, she notes that while most available studies reported health effect associations with ambient O₃ concentrations that violated the current standard, a small number provide support for the occurrence of adverse respiratory effects at ambient O₃ concentrations below the level of the current standard.¹¹⁴

Based on the analyses in the HREA, the Administrator concludes that the exposures and risks projected to remain upon meeting the current standard can reasonably be judged to be important from a public health perspective. In particular, this conclusion is based on her judgment that it is appropriate to set a standard that would be expected to eliminate, or almost eliminate, exposures of concern at or above 70 and 80 ppb. In addition, given that the average percent of children estimated to experience two or more exposures of concern for the 60 ppb benchmark approaches 10% in some urban study areas, the Administrator concludes that the current standard does not incorporate an adequate margin of safety

¹¹⁴ Courts have repeatedly held that this type of evidence justifies an Administrator’s conclusion that it is “appropriate” (within the meaning of section 109 (d)(1) of the CAA) to revise a primary NAAQS to provide further protection of public health. See *e.g. Mississippi*, 744 F. 3d at 1345; *American Farm Bureau*, 559 F. 3d at 525–26.

against the potentially adverse effects that could occur following repeated exposures at or above 60 ppb. Beyond estimated exposures of concern, the Administrator concludes that the HREA risk estimates (FEV₁ risk estimates, mortality risk estimates) further support a conclusion that the O₃-associated health effects estimated to remain upon just meeting the current standard are an issue of public health importance on a broad national scale. Thus, she concludes that O₃ exposure and risk estimates, when taken together, support a conclusion that the exposures and health risks associated with just meeting the current standard can reasonably be judged important from a public health perspective, such that the current standard is not sufficiently protective and does not incorporate an adequate margin of safety.

In the next section, the Administrator considers what revisions are appropriate in order to set a standard that is requisite to protect public health with an adequate margin of safety.

C. Conclusions on the Elements of a Revised Primary Standard

Having reached the conclusion that the current O₃ standard is not requisite to protect public health with an adequate margin of safety, based on the currently available scientific evidence and exposure/risk information, the Administrator next considers the range of alternative standards supported by that evidence and information. Consistent with her consideration of the adequacy of the current standard, the Administrator's conclusions on the elements of the primary standard are informed by the available scientific evidence assessed in the ISA, exposure/risk information presented and assessed in the HREA, the evidence-based and exposure-/risk-based considerations and conclusions in the PA, CASAC advice, and public comments. The sections below discuss the evidence and exposure/risk information, CASAC advice and public input, and the Administrator's proposed conclusions, for the major elements of the NAAQS: Indicator (II.C.1), averaging time (II.C.2), form (II.C.3), and level (II.C.4).

1. Indicator

In the 2008 review, the EPA focused on O₃ as the most appropriate indicator for a standard meant to provide protection against ambient photochemical oxidants. In this review, while the complex atmospheric chemistry in which O₃ plays a key role has been highlighted, no alternatives to O₃ have been advanced as being a more appropriate indicator for ambient

photochemical oxidants. More specifically, the ISA noted that O₃ is the only photochemical oxidant (other than NO₂) that is routinely monitored and for which a comprehensive database exists (U.S. EPA, 2013, section 3.6). Data for other photochemical oxidants (e.g., peroxyacetyl nitrate, hydrogen peroxide, etc.) typically have been obtained only as part of special field studies. Consequently, no data on nationwide patterns of occurrence are available for these other oxidants; nor are extensive data available on the relationships of concentrations and patterns of these oxidants to those of O₃ (U.S. EPA, 2013, section 3.6). In its review of the second draft PA, CASAC stated "The indicator of ozone is appropriate based on its causal or likely causal associations with multiple adverse health outcomes and its representation of a class of pollutants known as photochemical oxidants" (Frey, 2014c, p. ii).

In addition, the PA notes that meeting an O₃ standard can be expected to provide some degree of protection against potential health effects that may be independently associated with other photochemical oxidants, even though such effects are not discernible from currently available studies indexed by O₃ alone (U.S. EPA, 2014c, section 4.1). That is, since the precursor emissions that lead to the formation of O₃ generally also lead to the formation of other photochemical oxidants, measures leading to reductions in population exposures to O₃ can generally be expected to lead to reductions in population exposures to other photochemical oxidants. In considering this information, and CASAC's advice, the Administrator reached the proposed conclusion that O₃ remains the most appropriate indicator for a standard meant to provide protection against photochemical oxidants.¹¹⁵

The EPA received very few comments on the indicator of the primary standard. Those who did comment supported the proposed decision to retain O₃ as the indicator, noting the rationale put forward in the preamble to the proposed rule. These commenters generally expressed support for retaining the current indicator in conjunction with retaining other elements of the current standard, such as the averaging time and form. After considering the available evidence, CASAC advice, and public comments, the Administrator concludes that O₃ remains the most appropriate indicator

¹¹⁵ The DC Circuit upheld the use of O₃ as the indicator for photochemical oxidants based on these same considerations. *American Petroleum Inst. v. Costle*, 665 F. 2d 1176, 1186 (D.C. Cir. 1981).

for a standard meant to provide protection against photochemical oxidants. Therefore, she is retaining O₃ as the indicator for the primary standard in this final rule.

2. Averaging Time

The EPA established the current 8-hour averaging time¹¹⁶ for the primary O₃ NAAQS in 1997 (62 FR 38856). The decision on averaging time in that review was based on numerous controlled human exposure and epidemiologic studies reporting associations between adverse respiratory effects and 6- to 8-hour O₃ concentrations (62 FR 38861). The EPA also noted that a standard with a maximum 8-hour averaging time is likely to provide substantial protection against respiratory effects associated with 1-hour peak O₃ concentrations. The EPA reached similar conclusions in the last O₃ NAAQS review and thus, the EPA retained the 8-hour averaging time in 2008.

In reaching a proposed conclusion on averaging time in the current review, the Administrator considered the extent to which the available evidence continues to support the appropriateness of a standard with an 8-hour averaging time (79 FR 75292). Specifically, the Administrator considered the extent to which the available information indicates that a standard with the current 8-hour averaging time provides appropriate protection against short- and long-term O₃ exposures. These considerations from the proposal are summarized below in sections II.C.2.a (short-term) and II.C.2.b (long-term). Section II.C.2.c summarizes the Administrator's proposed decision on averaging time. Section II.C.2.d discusses comments received on averaging time. Section II.C.2.e presents the Administrator's final decision regarding averaging time.

a. Short-Term

As an initial consideration with respect to the most appropriate averaging time for the O₃ NAAQS, in the proposal the Administrator noted that the strongest evidence for O₃-associated health effects is for respiratory effects following short-term exposures. More specifically, the Administrator noted the ISA conclusion that the evidence is "sufficient to infer a causal relationship" between short-term O₃ exposures and respiratory effects. The ISA also judges that for short-term O₃ exposures, the evidence indicates "likely to be causal" relationships with

¹¹⁶ This 8-hour averaging time reflects daily maximum 8-hour average O₃ concentrations.

both cardiovascular effects and mortality (U.S. EPA, 2013, section 2.5.2). Therefore, as in past reviews, the Administrator noted that the strength of the available scientific evidence provides strong support for a standard that protects the public health against short-term exposures to O₃.

In first considering the level of support available for specific short-term averaging times, the Administrator noted in the proposal the evidence available from controlled human exposure studies. As discussed in more detail in Chapter 3 of the PA, substantial health effects evidence from controlled human exposure studies demonstrates that a wide range of respiratory effects (e.g., pulmonary function decrements, increases in respiratory symptoms, lung inflammation, lung permeability, decreased lung host defense, and airway hyperresponsiveness) occur in healthy adults following 6.6-hour exposures to O₃ (U.S. EPA, 2013, section 6.2.1.1). Compared to studies evaluating shorter exposure durations (e.g., 1-hour), studies evaluating 6.6-hour exposures in healthy adults have reported respiratory effects at lower O₃ exposure concentrations and at more moderate levels of exertion.

The Administrator also noted in the proposal the strength of evidence from epidemiologic studies that evaluated a wide variety of populations (e.g., including at-risk lifestyles and populations, such as children and people with asthma, respectively). A number of different averaging times have been used in O₃ epidemiologic studies, with the most common being the max 1-hour concentration within a 24-hour period (1-hour max), the max 8-hour average concentration within a 24-hour period (8-hour max), and the 24-hour average. These studies are assessed in detail in Chapter 6 of the ISA (U.S. EPA, 2013). Limited evidence from time-series and panel epidemiologic studies comparing risk estimates across averaging times does not indicate that one exposure metric is more consistently or strongly associated with respiratory health effects or mortality, though the ISA notes some evidence for “smaller O₃ risk estimates when using a 24-hour average exposure metric” (U.S. EPA, 2013, section 2.5.4.2; p. 2–31). For single- and multi-day average O₃ concentrations, lung function decrements were associated with 1-hour max, 8-hour max, and 24-hour average ambient O₃ concentrations, with no strong difference in the consistency or magnitude of association among the averaging times (U.S. EPA, 2013, p. 6–71). Similarly, in studies of short-term exposure to O₃ and mortality, Smith et

al. (2009) and Darrow *et al.* (2011) have reported high correlations between risk estimates calculated using 24-hour average, 8-hour max, and 1-hour max averaging times (U.S. EPA, 2013, p. 6–253). Thus, the Administrator noted that the epidemiologic evidence alone does not provide a strong basis for distinguishing between the appropriateness of 1-hour, 8-hour, and 24-hour averaging times.

Considering the health information discussed above, in the proposal the Administrator concluded that an 8-hour averaging time remains appropriate for addressing health effects associated with short-term exposures to ambient O₃. An 8-hour averaging time is similar to the exposure periods evaluated in controlled human exposure studies, including recent studies that provide evidence for respiratory effects following exposures to O₃ concentrations below the level of the current standard. In addition, epidemiologic studies provide evidence for health effect associations with 8-hour O₃ concentrations, as well as with 1-hour and 24-hour concentrations. As in previous reviews, the Administrator noted that a standard with an 8-hour averaging time (combined with an appropriate standard form and level) would also be expected to provide substantial protection against health effects attributable to 1-hour and 24-hour exposures (e.g., 62 FR 38861, July 18, 1997). This conclusion is consistent with the advice received from CASAC that “the current 8-hour averaging time is justified by the combined evidence from epidemiologic and clinical studies” (Frey, 2014c, p. 6).

b. Long-Term

The ISA concludes that the evidence for long-term O₃ exposures indicates that there is “likely to be a causal relationship” with respiratory effects (U.S. EPA, 2013, chapter 7). Thus, in this review the Administrator also considers the extent to which currently available evidence and exposure/risk information suggests that a standard with an 8-hour averaging time can provide protection against respiratory effects associated with longer term exposures to ambient O₃.

In considering this issue in the 2008 review of the O₃ NAAQS, the Staff Paper noted that “because long-term air quality patterns would be improved in areas coming into attainment with an 8-hr standard, the potential risk of health effects associated with long-term exposures would be reduced in any area meeting an 8-hr standard” (U.S. EPA, 2007, p. 6–57). In the current review, the PA further evaluates this issue, with

a focus on the long-term O₃ metrics reported to be associated with mortality or morbidity in recent epidemiologic studies. As discussed in section 3.1.3 of the PA (U.S. EPA, 2014c, section 4.2), much of the recent evidence for such associations is based on studies that defined long-term O₃ in terms of seasonal averages of daily maximum 1-hour or 8-hour concentrations.

As an initial consideration, in the proposal the Administrator noted the risk results from the HREA for respiratory mortality associated with long-term O₃ concentrations. These HREA analyses indicate that as air quality is adjusted to just meet the current 8-hour standard, most urban study areas are estimated to experience reductions in respiratory mortality associated with long-term O₃ concentrations based on the seasonal averages of 1-hour daily maximum O₃ concentrations evaluated in the study by Jerrett *et al.* (2009) (U.S. EPA, 2014a, chapter 7).¹¹⁷ As air quality is adjusted to meet lower alternative standard levels, for standards based on 3-year averages of the annual fourth-highest daily maximum 8-hour O₃ concentrations, respiratory mortality risks are estimated to be reduced further in urban study areas. This analysis indicates that an O₃ standard with an 8-hour averaging time, when coupled with an appropriate form and level, can reduce respiratory mortality reported to be associated with long-term O₃ concentrations.

In further considering the study by Jerrett *et al.* (2009), in the proposal the Administrator noted the PA comparison of long-term O₃ concentrations following model adjustment in urban study areas (*i.e.*, adjusted to meet the current and alternative 8-hour standards) to the concentrations present in study cities that provided the basis for the positive and statistically significant association with respiratory mortality. As indicated in Table 4–3 of the PA (U.S. EPA, 2014c, section 4.2), this comparison suggests that a standard with an 8-hour averaging time can decrease seasonal averages of 1-hour daily maximum O₃ concentrations, and can maintain those O₃ concentrations below the seasonal average concentration where the study indicates the most confidence in the reported concentration-response relationship with respiratory mortality (U.S. EPA, 2014c, sections 4.2 and 4.4.1).

¹¹⁷ Though the Administrator also notes important uncertainties associated with these risk estimates, as discussed in section II.C.3.b of the proposal.

The Administrator also noted in the proposal that the HREA conducted analyses evaluating the impacts of reducing regional NO_x emissions on the seasonal averages of daily maximum 8-hour O₃ concentrations. Seasonal averages of 8-hour daily max O₃ concentrations reflect long-term metrics that have been reported to be associated with respiratory morbidity effects in several recent O₃ epidemiologic studies (e.g., Islam *et al.*, 2008; Lin *et al.*, 2008a, 2008b; Salam *et al.*, 2009). The HREA analyses indicate that the large majority of the U.S. population lives in locations where reducing NO_x emissions would be expected to result in decreases in seasonal averages of daily max 8-hour ambient O₃ concentrations (U.S. EPA, 2014a, chapter 8). Thus, consistent with the respiratory mortality risk estimates noted above, these analyses suggest that reductions in O₃ precursor emissions in order to meet a standard with an 8-hour averaging time would also be expected to reduce the long-term O₃ concentrations that have been reported in recent epidemiologic studies to be associated with respiratory morbidity.

c. Administrator's Proposed Conclusion on Averaging Time

In the proposal the Administrator noted that, when taken together, the analyses summarized above indicate that a standard with an 8-hour averaging time, coupled with the current fourth-high form and an appropriate level, would be expected to provide appropriate protection against the short- and long-term O₃ concentrations that have been reported to be associated with respiratory morbidity and mortality. The CASAC agreed with this conclusion, stating that “[t]he current 8-hour averaging time is justified by the combined evidence from epidemiologic and clinical studies” and that “[t]he 8-hour averaging window also provides protection against the adverse impacts of long-term ozone exposures, which were found to be “likely causal” for respiratory effects and premature mortality” (Frey, 2014c, p. 6). Therefore, considering the available evidence and exposure risk information, and CASAC's advice, the Administrator proposed to retain the current 8-hour averaging time, and not to set an additional standard with a different averaging time.

d. Comments on Averaging Time

Most public commenters did not address the issue of whether the EPA should consider additional or alternative averaging times. Of those who did address this issue, some commenters representing state agencies or industry groups agreed with the

proposed decision to retain the current 8-hour averaging time, generally noting the supportive evidence discussed in the preamble to the proposed rule. In contrast, several medical organizations and environmental groups questioned the degree of health protection provided by a standard based on an 8-hour averaging time. For example, one group asserted that “[a]veraging over any time period, such as 8 hours, is capable of hiding peaks that may be very substantial if they are brief enough.”

The EPA agrees with these commenters that an important issue in the current review is the appropriateness of using a standard with an 8-hour averaging time to protect against adverse health effects that are attributable to a wide range of O₃ exposure durations, including those shorter and longer than 8 hours. This is an issue that has been thoroughly evaluated by the EPA in past reviews, as well as in the current review.

The 8-hour O₃ NAAQS was originally set in 1997, as part of revising the then-existing standard with its 1-hour averaging time, and was retained in the review completed in 2008 (73 FR 16472). In both of these reviews, several lines of evidence and information provided support for an 8-hour averaging time rather than a shorter averaging time. For example, substantial health evidence demonstrated associations between a wide range of respiratory effects and 6- to 8-hour exposures to relatively low O₃ concentrations (*i.e.*, below the level of the 1-hour O₃ NAAQS in place prior to the review completed in 1997). A standard with an 8-hour averaging time was determined to be more directly associated with health effects of concern at lower O₃ concentrations than a standard with a 1-hour averaging time. In addition, results of quantitative analyses showed that a standard with an 8-hour averaging time can effectively limit both 1- and 8-hour exposures of concern, and that an 8-hour averaging time results in a more uniformly protective national standard than a 1-hour averaging time. In past reviews, CASAC has agreed that an 8-hour averaging time is appropriate.

In reaching her proposed decision to retain the 8-hour averaging time in the current review, the Administrator again considered the body of evidence for adverse effects attributable to a wide range of O₃ exposure durations, including studies specifically referenced by public commenters who questioned the protectiveness of a standard with an 8-hour averaging time. For example, as noted above a substantial body of health effects evidence from controlled human

exposure studies demonstrates that a wide range of respiratory effects occur in healthy adults following 6.6-hour exposures to O₃ (U.S. EPA, 2013, section 6.2.1.1). Compared to studies evaluating shorter exposure durations (e.g., 1-hour), studies evaluating 6.6-hour exposures in healthy adults have reported respiratory effects at lower O₃ exposure concentrations and at more moderate levels of exertion. The Administrator also noted the strength of evidence from epidemiologic studies that evaluated a number of different averaging times, with the most common being the maximum 1-hour concentration within a 24-hour period (1-hour max), the maximum 8-hour average concentration within a 24-hour period (8-hour max), and the 24-hour average. Evidence from time-series and panel epidemiologic studies comparing risk estimates across averaging times does not indicate that one exposure metric is more consistently or strongly associated with respiratory health effects or mortality (U.S. EPA, 2013, section 2.5.4.2; p. 2–31). For single- and multi-day average O₃ concentrations, lung function decrements were associated with 1-hour max, 8-hour max, and 24-hour average ambient O₃ concentrations, with no strong difference in the consistency or magnitude of association among the averaging times (U.S. EPA, 2013, p. 6–71). Similarly, in studies of short-term exposure to O₃ and mortality, Smith *et al.* (2009) and Darrow *et al.* (2011) have reported high correlations between risk estimates calculated using 24-hour average, 8-hour max, and 1-hour max averaging times (U.S. EPA, 2013, p. 6–253). Thus, the epidemiologic evidence does not provide a strong basis for distinguishing between the appropriateness of 1-hour, 8-hour, and 24-hour averaging times.

In addition, quantitative exposure and risk analyses in the HREA are based on an air quality adjustment approach that estimates hourly O₃ concentrations, and on scientific studies that evaluated health effects attributable to a wide range of O₃ exposure durations. For example, the risk of lung function decrements is estimated using a model based on controlled human exposure studies with exposure durations ranging from 2 to 7.6 hours (U.S. EPA, 2013, section 6.2.1.1). Epidemiology-based risk estimates are based on studies that reported health effect associations with short-term ambient O₃ concentrations ranging from 1-hour to 24-hours and with long-term seasonal average concentrations (U.S. EPA, 2014a, Table 7–2). Thus, the HREA estimated health

risks associated with a wide range of O₃ exposure durations and the Administrator's conclusions on averaging time in the current review are based, in part, on consideration of these estimates.

When taken together, the evidence and analyses indicate that a standard with an 8-hour averaging time, coupled with the current fourth-high form and an appropriate level, would be expected to provide appropriate protection against the short- and long-term O₃ concentrations that have been reported to be associated with respiratory morbidity and mortality. The CASAC agreed with this, stating the following (Frey, 2014c, p. 6):

The current 8-hour averaging time is justified by the combined evidence from epidemiologic and clinical studies referenced in Chapter 4. Results from clinical studies, for example, show a wide range of respiratory effects in healthy adults following 6.6 hours of exposure to ozone, including pulmonary function decrements, increases in respiratory symptoms, lung inflammation, lung permeability, decreased lung host defense, and airway hyperresponsiveness. These findings are supported by evidence from epidemiological studies that show causal associations between short-term exposures of 1, 8 and 24-hours and respiratory effects and "likely to be causal" associations for cardiovascular effects and premature mortality. The 8-hour averaging window also provides protection against the adverse impacts of long-term ozone exposures, which were found to be "likely causal" for respiratory effects and premature mortality.

Given all of the above, the EPA disagrees with commenters who question the protectiveness of an O₃ standard with an 8-hour averaging time, particularly for an 8-hour standard with the revised level of 70 ppb that is being established in this review, as discussed below (II.C.4).

e. Administrator's Final Decision Regarding Averaging Time

In considering the evidence and information summarized in the proposal and discussed in detail in the ISA, HREA, and PA; CASAC's views; and public comments, the Administrator concludes that a standard with an 8-hour averaging time can effectively limit health effects attributable to both short- and long-term O₃ exposures. As was the case in the proposal, this final conclusion is based on (1) the strong evidence that continues to support the importance of protecting public health against short-term O₃ exposures (e.g., ≤ 1-hour to 24-hour) and (2) analyses in the HREA and PA supporting the conclusion that the current 8-hour averaging time can effectively limit long-term O₃ exposures. Furthermore,

the Administrator observes that the CASAC Panel agreed with the choice of averaging time (Frey, 2014c). Therefore, in the current review, the Administrator concludes that it is appropriate to retain the 8-hour averaging time and to not set a separate standard with a different averaging time in this final rule.

3. Form

The "form" of a standard defines the air quality statistic that is to be compared to the level of the standard in determining whether an area attains that standard. The foremost consideration in selecting a form is the adequacy of the public health protection provided by the combination of the form and the other elements of the standard. In this review, the Administrator considers the extent to which the available evidence and/or information continue to support the appropriateness of a standard with the current form, defined by the 3-year average of annual fourth-highest 8-hour daily maximum O₃ concentrations. Section II.C.3.a below summarizes the basis for the current form. Section II.C.3.b discusses the Administrator's proposed decision to retain the current form. Section II.C.3.c discusses public comments received on the form of the primary standard. Section II.C.3.d discusses the Administrator's final decision on form.

a. Basis for the Current Form

The EPA established the current form of the primary O₃ NAAQS in 1997 (62 FR 38856). Prior to that time, the standard had a "1-expected-exceedance" form.¹¹⁸ An advantage of the current concentration-based form recognized in the 1997 review is that such a form better reflects the continuum of health effects associated with increasing ambient O₃ concentrations. Unlike an expected exceedance form, a concentration-based form gives proportionally more weight to years when 8-hour O₃ concentrations are well above the level of the standard than years when 8-hour O₃ concentrations are just above the level of the standard.¹¹⁹ The EPA judged it

¹¹⁸ For a standard with a 1-expected-exceedance form to be met at an air quality monitoring site, the fourth-highest air quality value in 3 years, given adjustments for missing data, must be less than or equal to the level of the standard.

¹¹⁹ As discussed (61 FR 65731), this is because with an exceedance-based form, days on which the ambient O₃ concentration is well above the level of the standard are given equal weight to those days on which the O₃ concentration is just above the standard (i.e., each day is counted as one exceedance), even though the public health impact of such days would be very different. With a concentration-based form, days on which higher O₃ concentrations occur would weigh proportionally more than days with lower O₃ concentrations since

appropriate to give more weight to higher O₃ concentrations, given that available health evidence indicated a continuum of effects associated with exposures to varying concentrations of O₃, and given that the extent to which public health is affected by exposure to ambient O₃ is related to the actual magnitude of the O₃ concentration, not just whether the concentration is above a specified level.

During the 1997 review, the EPA considered a range of alternative "concentration-based" forms, including the second-, third-, fourth- and fifth-highest daily maximum 8-hour concentrations in an O₃ season. The fourth-highest daily maximum was selected, recognizing that a less restrictive form (e.g., fifth-highest) would allow a larger percentage of sites to experience O₃ peaks above the level of the standard, and would allow more days on which the level of the standard may be exceeded when the site attains the standard (62 FR 38856). The EPA also considered setting a standard with a form that would provide a margin of safety against possible but uncertain chronic effects, and would provide greater stability to ongoing control programs.¹²⁰ A more restrictive form was not selected, recognizing that the differences in the degree of protection afforded by the alternatives were not well enough understood to use any such differences as a basis for choosing the most restrictive forms (62 FR 38856).

In the 2008 review, the EPA additionally considered the potential value of a percentile-based form. In doing so, the EPA recognized that such a statistic is useful for comparing datasets of varying length because it samples approximately the same place in the distribution of air quality values, whether the dataset is several months or several years long. However, the EPA concluded that a percentile-based statistic would not be effective in ensuring the same degree of public health protection across the country. Specifically, a percentile-based form would allow more days with higher air quality values in locations with longer O₃ seasons relative to locations with shorter O₃ seasons. Thus, in the 2008 review, the EPA concluded that a form based on the nth-highest maximum O₃ concentration would more effectively ensure that people who live in areas

the actual concentrations are used directly to calculate whether the standard is met or violated.

¹²⁰ See *American Trucking Ass'n v. EPA*, 283 F. 3d at 374-75 (less stable implementation programs may be less effective and would thereby provide less public health protection; EPA may therefore legitimately consider programmatic stability in determining the form of a NAAQS).

with different length O₃ seasons receive the same degree of public health protection.

Based on analyses of forms specified in terms of an nth-highest concentration (n ranged from 3 to 5), advice from CASAC, and public comment, the Administrator concluded that a fourth-highest daily maximum should be retained (73 FR 16465, March 27, 2008). In reaching this decision, the Administrator recognized that “there is not a clear health-based threshold for selecting a particular nth-highest daily maximum form of the standard” and that “the adequacy of the public health protection provided by the combination of the level and form is a foremost consideration” (73 FR 16475, March 27, 2008). Based on this, the Administrator judged that the existing form (fourth-highest daily maximum 8-hour average concentration) should be retained, recognizing the increase in public health protection provided by combining this form with a lower standard level (*i.e.*, 75 ppb).

The Administrator also recognized that it is important to have a form that provides stability with regard to implementation of the standard. In the case of O₃, for example, he noted the importance of a form insulated from the impacts of extreme meteorological events that are conducive to O₃ formation. Such events could have the effect of reducing public health protection, to the extent they result in frequent shifts in and out of attainment due to meteorological conditions. The Administrator noted that such frequent shifting could disrupt an area’s ongoing implementation plans and associated control programs (73 FR 16474, March 27, 2008). In his final decision, the Administrator judged that a fourth-high form “provides a stable target for implementing programs to improve air quality” (*id.* at 16475).

b. Proposed Decision on Form

In the proposal for the current review, the Administrator considered the extent to which newly available information provides support for the current form (79 FR 75293). In so doing, she took note of the conclusions of prior reviews summarized above. She recognized the value of an nth-high statistic over that of an expected exceedance or percentile-based form in the case of the O₃ standard, for the reasons summarized above. The Administrator additionally took note of the importance of stability in implementation to achieving the level of protection specified by the NAAQS. Specifically, she noted that to the extent areas engaged in implementing the O₃ NAAQS frequently shift from meeting

the standard to violating the standard, it is possible that ongoing implementation plans and associated control programs could be disrupted, thereby reducing public health protection.

In light of this, while giving foremost consideration to the adequacy of public health protection provided by the combination of all elements of the standard, including the form, the Administrator considered particularly the findings from prior reviews with regard to the use of the nth-high metric. As noted above, the EPA selected the fourth-highest daily maximum, recognizing the public health protection provided by this form, when coupled with an appropriate averaging time and level, and recognizing that such a form can provide stability for implementation programs. In the proposal the Administrator concluded that the currently available evidence and information do not call into question these conclusions from previous reviews. In reaching this initial conclusion, the Administrator noted that CASAC concurred that the O₃ standard should be based on the fourth-highest, daily maximum 8-hour average value (averaged over 3 years), stating that this form “provides health protection while allowing for atypical meteorological conditions that can lead to abnormally high ambient ozone concentrations which, in turn, provides programmatic stability” (Frey, 2014c, p. 6). Thus, a standard with the current fourth-high form, coupled with a level lower than 75 ppb as discussed below, would be expected to increase public health protection relative to the current standard while continuing to provide stability for implementation programs. Therefore, the Administrator proposed to retain the current fourth-highest daily maximum form for an O₃ standard with an 8-hour averaging time and a revised level.

c. Public Comments on Form

Several commenters focused on the stability of the standard to support their positions regarding form. Some industry associations and state agencies support changing to a form that would allow a larger number of exceedances of the standard level than are allowed by the current fourth-high form. In some cases, these commenters argued that a standard allowing a greater number of exceedances would provide the same degree of public health protection as the current standard. Some commenters advocated a percentile-based form, such as the 98th percentile. These commenters cited a desire for consistency with short-term standards for other criteria pollutants (*e.g.*, PM_{2.5},

NO₂), as well as a desire to allow a greater number of exceedances of the standard level, thus making the standard less sensitive to fluctuations in background O₃ concentrations and to extreme meteorological events.

Other commenters submitted analyses purporting to indicate that a fourth-high form provides only a small increase in stability, relative to forms that allow fewer exceedances of the standard level (*i.e.*, first-high, second-high). These commenters also called into question the degree of health protection achieved by a standard with a fourth-high form and a level in the proposed range (*i.e.*, 65 to 70 ppb). They pointed out that a fourth-high form will, by definition, allow 3 days per year, on average, with 8-hour O₃ concentrations above the level of the standard. Commenters further stated that “[i]f ozone levels on these peak days are appreciably higher than on the fourth-highest day, given EPA’s acknowledged concerns regarding single or multiple (defined by EPA as 2 or more) exposures to elevated ozone concentrations, EPA must account for the degree of under-protection in setting the level of the NAAQS” (*e.g.*, ALA *et al.*, p. 138).

For the reasons discussed in the proposal, and summarized above, the EPA disagrees with commenters who supported a percentile-based form, such as the 98th percentile, for the O₃ NAAQS. As noted above, a percentile-based statistic would not be effective in ensuring the same degree of public health protection across the country. Rather, a percentile-based form would allow more days with higher air quality values in locations with longer O₃ seasons relative to locations with shorter O₃ seasons. Thus, as in the 2008 review, in the current review the EPA concludes that a form based on the nth-highest maximum O₃ concentration would more effectively ensure that people who live in areas with different length O₃ seasons receive the same degree of public health protection.

In considering various nth-high values, as in past reviews (*e.g.*, 73 FR 16475, March 27, 2008), the EPA recognizes that there is not a clear health-based threshold for selecting a particular nth-highest daily maximum form. Rather, the primary consideration is the adequacy of the public health protection provided by the combination of all of the elements of the standard, including the form. Environmental and public health commenters are correct that a standard with the current fourth-high form will allow 3 days per year, on average, with 8-hour O₃ concentrations higher than the standard level. However, the EPA disagrees with these

commenters' assertion that using a fourth-high form results in a standard that is under-protective. The O₃ exposure and risk estimates that informed the Administrator's consideration of the degree of public health protection provided by various standard levels were based on air quality that "just meets" various standards with the current 8-hour averaging time and fourth-high, 3-year average form (U.S. EPA, 2014a, section 4.3.3). Therefore, air quality adjusted to meet various levels of the standard with the current form and averaging time will include days with concentrations above the level of the standard, and these days contribute to exposure and risk estimates. In this way, the Administrator has reasonably considered the public health protection provided by the combination of all of the elements of the standard, including the fourth-high form.

In past reviews, EPA selected the fourth-highest daily maximum form in recognition of the public health protection provided by this form, when coupled with an appropriate averaging time and level, and recognizing that such a form can provide stability for ongoing implementation programs. As noted above, some commenters submitted analyses suggesting that a fourth-high form provides only a small increase in stability, relative to a first- or second-high form. The EPA has conducted analyses of ambient O₃ monitoring data to further consider these commenters' assertions regarding stability. The EPA's analyses of nth-high concentrations ranging from first-high to fifth-high have been summarized in a memo to the docket (Wells, 2015a). Consistent with commenters' analyses, Wells (2015a) indicates a progressive decrease in the variability of O₃ concentrations, and an increase in the stability of those concentrations, as "n" increases. Based on these analyses, there is no clear threshold for selecting a particular nth-high form based on stability alone. Rather, as in past reviews, the decision on form in this review focuses first and foremost on the Administrator's judgments on public health protection, with judgments regarding stability of the standard being a legitimate, but secondary consideration. The Administrator's final decision on form is discussed below.

d. Administrator's Final Decision Regarding Form

In reaching a final decision on the form of the primary O₃ standard, as described in the proposal and above, the Administrator recognizes that there is not a clear health-based rationale for

selecting a particular nth-highest daily maximum form. Her foremost consideration is the adequacy of the public health protection provided by the combination of all of the elements of the standard, including the form. In this regard, the Administrator recognizes the support from analyses in previous reviews, and from the CASAC in the current review, for the conclusion that the current fourth-high form of the standard, when combined with a revised level as discussed below, provides an appropriate balance between public health protection and a stable target for implementing programs to improve air quality. In particular, she notes that the CASAC concurred that the O₃ standard should be based on the fourth-highest, daily maximum 8-hour average value (averaged over 3 years), stating that this form "provides health protection while allowing for atypical meteorological conditions that can lead to abnormally high ambient ozone concentrations which, in turn, provides programmatic stability" (Frey, 2014c, p. 6). Based on these considerations, and on consideration of public comments on form as discussed above, the Administrator judges it appropriate to retain the current fourth-high form (fourth-highest daily maximum 8-hour O₃ concentration, averaged over 3 years) in this final rule.

4. Level

This section summarizes the basis for the Administrator's proposed decision to revise the current standard level (II.C.4.a); discusses public comments, and the EPA's responses, on that proposed decision (II.C.4.b); and presents the Administrator's final decision regarding the level of the primary O₃ standard (II.C.4.c).

a. Basis for the Administrator's Proposed Decision on Level

In conjunction with her proposed decisions to retain the current indicator, averaging time, and form (II.C.1 to II.C.3, above), the Administrator proposed to revise the level of the primary O₃ standard to within the range of 65 to 70 ppb. In proposing this range of standard levels, as discussed in section II.E.4 of the proposal, the Administrator carefully considered the scientific evidence assessed in the ISA (U.S. EPA, 2013); the results of the exposure and risk assessments in the HREA (U.S. EPA, 2014a); the evidence-based and exposure-/risk-based considerations and conclusions in the PA (U.S. EPA, 2014c); CASAC advice and recommendations, as reflected in CASAC's letters to the Administrator and in public discussions of drafts of

the ISA, HREA, and PA (Frey and Samet, 2012; Frey, 2014 a, c); and public input received during the development of these documents.

The Administrator's proposal to revise the standard level built upon her proposed conclusion that the overall body of scientific evidence and exposure/risk information calls into question the adequacy of public health protection afforded by the current primary O₃ standard, particularly for at-risk populations and lifestages. In reaching proposed conclusions on alternative levels for the primary O₃ standard, the Administrator considered the extent to which various alternatives would be expected to protect the public, including at-risk populations, against the wide range of adverse health effects that have been linked with short- or long-term O₃ exposures.

As was the case for her consideration of the adequacy of the current primary O₃ standard (II.B.3, above), the Administrator placed the greatest weight on the results of controlled human exposure studies and on exposure and risk analyses based on information from these studies. In doing so, she noted that controlled human exposure studies provide the most certain evidence indicating the occurrence of health effects in humans following exposures to specific O₃ concentrations. The effects reported in these studies are due solely to O₃ exposures, and interpretation of study results is not complicated by the presence of co-occurring pollutants or pollutant mixtures (as is the case in epidemiologic studies). She further noted the CASAC judgment that "the scientific evidence supporting the finding that the current standard is inadequate to protect public health is strongest based on the controlled human exposure studies of respiratory effects" (Frey, 2014c, p. 5).

In considering the evidence from controlled human exposure studies, the Administrator first noted that the largest respiratory effects, and the broadest range of effects, have been studied and reported following exposures to 80 ppb O₃ or higher, with most exposure studies conducted at these higher concentrations. Exposures of healthy adults to O₃ concentrations of 80 ppb or higher have been reported to decrease lung function, increase airway inflammation, increase respiratory symptoms, result in airway hyperresponsiveness, and decrease lung host defenses. The Administrator further noted that O₃ exposure concentrations as low as 72 ppb have been shown to both decrease lung function and increase respiratory

symptoms (Schelegle *et al.*, 2009),¹²¹ a combination that meets the ATS criteria for an adverse response, and that exposures as low as 60 ppb have been reported to decrease lung function and increase airway inflammation.

Based on this evidence, the Administrator reached the initial conclusion that the results of controlled human exposure studies strongly support setting the level of a revised O₃ standard no higher than 70 ppb. In reaching this conclusion, she placed a large amount of weight on the importance of setting the level of the standard well below 80 ppb, the exposure concentration at which the broadest range of effects have been studied and reported, and below 72 ppb, the lowest exposure concentration shown to result in the adverse combination of lung function decrements and respiratory symptoms. She placed significant weight on this *combination* of effects, as did CASAC, in making judgments regarding the potential for adverse responses.

In further considering the potential public health implications of a standard with a level of 70 ppb, the Administrator also considered quantitative estimates of the extent to which such a standard would be expected to limit population exposures to the broader range of O₃ concentrations shown in controlled human exposure studies to cause respiratory effects. In doing so, she focused on estimates of O₃ exposures of concern at or above the benchmark concentrations of 60, 70, and 80 ppb. The Administrator judged that the evidence supporting the occurrence of adverse respiratory effects is strongest for exposures at or above the 70 and 80 ppb benchmarks. Therefore, she placed a large amount of emphasis on the importance of setting a standard that limits exposures of concern at or above these benchmarks.

The Administrator expressed less confidence that adverse effects will occur following exposures to O₃ concentrations as low as 60 ppb. In reaching this conclusion, she highlighted the fact that statistically significant increases in respiratory symptoms, combined with lung function decrements, have not been reported following exposures to 60 or 63 ppb O₃, though several studies have evaluated the potential for such effects (Kim *et al.*, 2011; Schelegle *et al.*, 2009;

¹²¹ As noted above, for the 70 ppb target exposure concentration, Schelegle *et al.* (2009) reported that the actual mean exposure concentration was 72 ppb.

Adams, 2006).¹²² The proposal specifically stated that “[t]he Administrator has decreasing confidence that adverse effects will occur following exposures to O₃ concentrations below 72 ppb. In particular, compared to O₃ exposure concentrations at or above 72 ppb, she has less confidence that adverse effects will occur following exposures to O₃ concentrations as low as 60 ppb” (79 FR 73304–05).

However, she noted the possibility for adverse effects following such exposures given that: (1) CASAC judged the adverse combination of lung function decrements and respiratory symptoms “almost certainly occur in some people” following exposures to O₃ concentrations below 72 ppb (though CASAC did not specify or otherwise indicate how far below) (Frey, 2014c, p. 6); (2) CASAC indicated the moderate lung function decrements (*i.e.*, FEV₁ decrements $\geq 10\%$) that occur in some healthy adults following exposures to 60 ppb O₃ could be adverse to people with lung disease; and (3) airway inflammation has been reported following exposures as low as 60 ppb O₃. She also took note of CASAC advice that the occurrence of exposures of concern at or above 60 ppb is an appropriate consideration for people with asthma (Frey, 2014c, p. 6). Therefore, while the Administrator expressed less confidence that adverse effects will occur following exposures to O₃ concentrations as low as 60 ppb, compared to 70 ppb and above, based on the evidence and CASAC advice she also gave some consideration to exposures of concern for the 60 ppb benchmark.

Due to interindividual variability in responsiveness, the Administrator further noted that not every occurrence of an exposure of concern will result in an adverse effect, and that repeated occurrences of some of the effects demonstrated following exposures of concern could increase the likelihood of adversity (U.S. EPA, 2013, section 6.2.3). Therefore, the Administrator was most concerned about protecting at-risk populations against repeated occurrences of exposures of concern. Based on the above considerations, the Administrator focused on the extent to which a revised standard with a level of 70 ppb would be expected to protect populations from experiencing two or more O₃ exposures of concern (*i.e.*, as a surrogate for repeated exposures).

¹²² In the study by Schelegle, for the 60 ppb target exposure concentration, study authors reported that the actual mean exposure concentration was 63 ppb.

As illustrated in Table 1 in the proposal (and Table 1 above), the Administrator noted that, in urban study areas, a revised standard with a level of 70 ppb is estimated to eliminate the occurrence of two or more exposures of concern to O₃ concentrations at and above 80 ppb and to virtually eliminate the occurrence of two or more exposures of concern to O₃ concentrations at and above 70 ppb, even in the worst-case urban study area and year evaluated. Though the Administrator acknowledged greater uncertainty with regard to the occurrence of adverse effects following exposures to 60 ppb, she noted that a revised standard with a level of 70 ppb would also be expected to protect the large majority of children in the urban study areas (*i.e.*, about 96% to more than 99% of children in individual urban study areas) from experiencing two or more exposures of concern at or above the 60 ppb benchmark. Compared to the current standard, this represents a reduction of more than 60%.¹²³

In further evaluating the potential public health impacts of a standard with a level of 70 ppb, the Administrator also considered the HREA estimates of O₃-induced lung function decrements. To inform her consideration of these decrements, the Administrator took note of CASAC advice that “estimation of FEV₁ decrements of $\geq 15\%$ is appropriate as a scientifically relevant surrogate for adverse health outcomes in active healthy adults, whereas an FEV₁ decrement of $\geq 10\%$ is a scientifically relevant surrogate for adverse health outcomes for people with asthma and lung disease” (Frey, 2014c, p. 3).

Although these FEV₁ decrements provide perspective on the potential for the occurrence of adverse respiratory effects following O₃ exposures, the Administrator agreed with the conclusion in past reviews that a more general consensus view of the adversity of moderate responses emerges as the frequency of occurrence increases (61 FR 65722–3, Dec. 13, 1996). Specifically, she judged that not every estimated occurrence of an O₃-induced FEV₁ decrement will be adverse and

¹²³ The Administrator judged that the evidence is less compelling, and indicates greater uncertainty, with regard to the potential for adverse effects following single occurrences of O₃ exposures of concern. While acknowledging this greater uncertainty, she noted that a standard with a level of 70 ppb would also be expected to virtually eliminate all occurrences (including single occurrences) of exposures of concern at or above 80 ppb, even in the worst-case year and location. She also judged that such a standard will achieve important reductions, compared to the current standard, in the occurrence of one or more exposures of concern at or above 70 and 60 ppb.

that repeated occurrences of moderate responses could lead to more serious illness. Therefore, the Administrator noted increasing concern about the potential for adversity as the number of occurrences increases and, as a result, she focused primarily on estimates of two or more O₃-induced FEV₁ decrements (*i.e.*, as a surrogate for repeated exposures).¹²⁴

The Administrator noted that a revised O₃ standard with a level of 70 ppb is estimated to protect about 98 to 99% of children in urban study areas from experiencing two or more O₃-induced FEV₁ decrements $\geq 15\%$, and about 89 to 94% from experiencing two or more decrements $\geq 10\%$. She judged that these estimates reflect important risk reductions, compared to the current standard. Given these estimates, as well as estimates of one or more decrements per season (about which she was less concerned (79 FR 75290, December 17, 2014)), the Administrator concluded that a revised standard with a level of 70 ppb would be expected to provide substantial protection against the risk of O₃-induced lung function decrements, and would be expected to result in important reductions in such risks, compared to the current standard. The Administrator further noted, however, that the variability in lung function risk estimates across urban study areas is often greater than the differences in risk estimates between various standard levels (Table 2, above). Given this, and the resulting considerable overlap between the ranges of lung function risk estimates for different standard levels, in the proposal the Administrator viewed lung function risk estimates as providing a more limited basis than exposures of concern for distinguishing between the degrees of public health protection provided by alternative standard levels (79 FR 75306 n. 164).

In next considering the additional protection that would be expected from standard levels below 70 ppb, the Administrator evaluated the extent to which a standard with a level of 65 ppb would be expected to further limit O₃ exposures of concern and O₃-induced lung function decrements. In addition to eliminating almost all exposures of concern to O₃ concentrations at or above 80 and 70 ppb, even in the worst-case years and locations, the Administrator noted that a revised standard with a

¹²⁴ In the proposal, the Administrator further judged that it would not be appropriate to set a standard that is intended to eliminate all O₃-induced FEV₁ decrements. She noted that this is consistent with CASAC advice, which did not include a recommendation to set the standard level low enough to eliminate all O₃-induced FEV₁ decrements ≥ 10 or 15% (Frey, 2014c).

level of 65 ppb would be expected to protect more than 99% of children in urban study areas from experiencing two or more exposures of concern at or above 60 ppb and to substantially reduce the occurrence of one or more such exposures, compared to the current standard. With regard to O₃-induced lung function decrements, an O₃ standard with a level of 65 ppb is estimated to protect about 98% to more than 99% of children from experiencing two or more O₃-induced FEV₁ decrements $\geq 15\%$ and about 91 to 99% from experiencing two or more decrements $\geq 10\%$.¹²⁵

Taken together, the Administrator concluded that the evidence from controlled human exposure studies, and the information from quantitative analyses that draw upon these studies, provide strong support for standard levels from 65 to 70 ppb. In particular, she based this conclusion on the fact that such standard levels would be well below the O₃ exposure concentration shown to result in the widest range of respiratory effects (*i.e.*, 80 ppb),¹²⁶ and below the lowest O₃ exposure concentration shown to result in the adverse combination of lung function decrements and respiratory symptoms (*i.e.*, 72 ppb). A standard with a level from 65 to 70 ppb would also be expected to result in important reductions, compared to the current standard, in the occurrence of O₃ exposures of concern for all of the benchmarks evaluated (*i.e.*, 60, 70, and 80 ppb) and in the risk of O₃-induced lung function decrements ≥ 10 and 15%.

In further considering the evidence and exposure/risk information, the Administrator considered the extent to which the epidemiologic evidence also provides support for standard levels from 65 to 70 ppb. In particular, the Administrator noted analyses in the PA (U.S. EPA, 2014c, section 4.4.1) indicating that a revised standard with a level of 65 or 70 ppb would be expected to maintain distributions of short-term ambient O₃ concentrations below those present in the locations of all the single-city epidemiologic studies of hospital admissions or emergency department visits analyzed. She concluded that a revised standard with a level at least as low as 70 ppb would

¹²⁵ Although the Administrator was less concerned about the public health implications of single O₃-induced lung function decrements, she also noted that a revised standard with a level of 65 ppb is estimated to reduce the risk of one or more O₃-induced decrements per season, compared to the current standard.

¹²⁶ Although the widest range of effects have been evaluated following exposures to 80 ppb O₃, there is no evidence that 80 ppb is a threshold for these effects.

result in improvements in public health, beyond the protection provided by the current standard, in the locations of the single-city epidemiologic studies that reported significant health effect associations.¹²⁷

The Administrator noted additional uncertainty in interpreting air quality in locations of multicity epidemiologic studies of short-term O₃ for the purpose of evaluating alternative standard levels (II.D.1 and U.S. EPA, 2014c, section 4.4.1). While acknowledging this uncertainty, and therefore placing less emphasis on these analyses of study location air quality, she noted that PA analyses suggest that standard levels of 65 or 70 ppb would require reductions, beyond those required by the current standard, in ambient O₃ concentrations present in several of the locations that provided the basis for statistically significant O₃ health effect associations in multicity studies.

In further evaluating information from epidemiologic studies, the Administrator considered the HREA's epidemiology-based risk estimates for O₃-associated morbidity or mortality (U.S. EPA, 2014a, Chapter 7). Compared to the weight given to the evidence from controlled human exposure studies, and to HREA estimates of exposures of concern and lung function risks, she placed relatively less weight on epidemiology-based risk estimates. In doing so, she noted that the overall conclusions from the HREA likewise reflect relatively less confidence in estimates of epidemiology-based risks than in estimates of exposures of concern and lung function risks.

In considering epidemiology-based risk estimates, the Administrator focused on risks associated with O₃ concentrations in the upper portions of ambient distributions, given the greater uncertainty associated with the shapes of concentration-response curves for O₃ concentrations in the lower portions of ambient distributions (*i.e.*, below about 20 to 40 ppb depending on the O₃ metric, health endpoint, and study population) (U.S. EPA, 2013, section 2.5.4.4). The Administrator further noted that experimental studies provide the strongest evidence for O₃-induced effects following exposures to O₃ concentrations corresponding to the upper portions of typical ambient

¹²⁷ The Administrator also concluded that analyses in the HREA and PA indicate that a standard with an 8-hour averaging time, coupled with the current fourth-high form and a level from 65 to 70 ppb, would be expected to provide increased protection, compared to the current standard, against the long-term O₃ concentrations that have been reported to be associated with respiratory morbidity or mortality (79 FR 75293; 75308).

distributions. In particular, as discussed above, she noted controlled human exposure studies showing respiratory effects following exposures to O₃ concentrations at or above 60 ppb (79 FR 75308, December 17, 2014).

Therefore, in considering risks associated with O₃ concentrations in the upper portions of ambient distributions, the Administrator focused on the extent to which revised standards with levels of 70 or 65 ppb are estimated to reduce the risk of premature deaths associated with area-wide O₃ concentrations at or above 40 ppb and 60 ppb.

Given all of the above evidence, exposure/risk information, and advice from CASAC, the Administrator proposed to revise the level of the current primary O₃ standard to within the range of 65 to 70 ppb. In considering CASAC advice on the range of standard levels, the Administrator placed a large amount of weight on CASAC's conclusion that there is adequate scientific evidence to consider a range of levels for a primary standard that includes an upper end at 70 ppb. She also noted that although CASAC expressed concern about the margin of safety at a level of 70 ppb, it further acknowledged that the choice of a level within the range recommended based on scientific evidence is a policy judgment (Frey, 2014c, p. ii). While she agreed with CASAC that it is appropriate to consider levels below 70 ppb, as reflected in her range of proposed levels from 65 to 70 ppb, for the reasons discussed above she also concluded that a standard level as high as 70 ppb, which CASAC concluded could be supported by the scientific evidence, could reasonably be judged to be requisite to protect public health with an adequate margin of safety.

In considering the appropriateness of standard levels below 65 ppb, the Administrator noted the conclusions of the PA and the advice of CASAC that it would be appropriate for her to consider standard levels as low as 60 ppb. In making the decision to not propose levels below 65 ppb, she focused on CASAC's rationale for a level of 60 ppb, which focused on the importance of limiting exposures to O₃ concentrations as low as 60 ppb (Frey, 2014c, p. 7). As discussed above, the Administrator agreed that it is appropriate to consider the implications of a revised standard level for estimated exposures of concern at or above 60 ppb. She noted that standards within the proposed range of 65 to 70 ppb would be expected to substantially limit the occurrence of exposures of concern to O₃ concentrations at or above 60 ppb, particularly the occurrence of two or

more exposures. When she further considered that not all exposures of concern lead to adverse effects, and that the NAAQS are not meant to be zero-risk or background standards, the Administrator judged that alternative standard levels below 65 ppb are not needed to further reduce such exposures.

b. Comments on Level

A number of groups representing medical, public health, or environmental organizations; some state agencies; and many individuals submitted comments on the appropriate level of a revised primary O₃ standard.¹²⁸ Virtually all of these commenters supported setting the standard level within the range recommended by CASAC (*i.e.*, 60 to 70). Some expressed support for the overall CASAC range, without specifying a particular level within that range, while others expressed a preference for the lower part of the CASAC range, often emphasizing support for a level of 60 ppb. Some of these commenters stated that if the EPA does not set the level at 60 ppb, then the level should be set no higher than 65 ppb (*i.e.*, the lower bound of the proposed range of standard levels).

To support their views on the level of a revised standard, some commenters focused on overarching issues related to the statutory requirements for the NAAQS. For example, some commenters maintained that the primary NAAQS must be set at a level at which there is an absence of adverse effects in sensitive populations. While this argument has some support in the case law and in the legislative history to the 1970 CAA (see *Lead Industries Ass'n v. EPA*, 647 F. 2d 1147, 1153 (D.C. Cir. 1980)), it is well established that the NAAQS are not meant to be zero risk standards. See *Lead Industries v. EPA*, 647 F.2d at 1156 n.51; *Mississippi v. EPA*, 744 F. 3d at 1351. From the inception of the NAAQS standard-setting process, the EPA and the courts have acknowledged that scientific uncertainties in general, and the lack of clear thresholds in pollutant effects in particular, preclude any such definitive determinations. *Lead Industries*, 647 F. 2d at 1156 (setting standard at a level which would remove most but not all

¹²⁸ In general, commenters who expressed the view that the EPA should retain the current O₃ NAAQS (*i.e.*, commenters representing industry and business groups, and some states) did not provide comments on alternative standard levels. As a result, this section focuses primarily on comments from commenters who expressed support for the proposed decision to revise the current primary O₃ standard.

sub-clinical effects). Likewise, the House report to the 1977 amendments addresses this question (H. Rep. 95–294, 95th Cong. 1st sess. 127):¹²⁹

Some have suggested that since the standards are to protect against all known or anticipated effects and since no safe threshold can be established, the ambient standards should be set at zero or background levels. Obviously, this no-risk philosophy ignores all economic and social consequences and is impractical. This is particularly true in light of the legal requirement for mandatory attainment of the national primary standards within 3 years.

Thus, post-1970 jurisprudence makes clear the impossibility, and lack of legal necessity, for NAAQS removing all health risk. See *ATA III*, 283 F. 3d at 360 (“[t]he lack of a threshold concentration below which these pollutants are known to be harmless makes the task of setting primary NAAQS difficult, as EPA must select standard levels that reduce risks sufficiently to protect public health even while recognizing that a zero-risk standard is not possible”); *Mississippi*, 744 F. 3d at 1351 (same); see also *id.* at 1343 (“[d]etermining what is ‘requisite’ to protect the ‘public health’ with an ‘adequate’ margin of safety may indeed require a contextual assessment of acceptable risk. See *Whitman*, 531 U.S. at 494–95 (Breyer J. concurring)”).

In this review, EPA is setting a standard based on a careful weighing of available evidence, including a weighing of the strengths and limitations of the evidence and underlying scientific uncertainties therein. The Administrator's choice of standard level is rooted in her evaluation of the evidence, which reflects her legitimate uncertainty as to the O₃ concentrations at which the public would experience adverse health effects. This is a legitimate, and well recognized, exercise of “reasoned decision-making.” *ATA III*, 283 F. 3d at 370; see also *id.* at 370 (“EPA's inability to guarantee the accuracy or increase the precision of the . . . NAAQS in no way undermines the standards' validity. Rather, these limitations indicate only that significant scientific uncertainty remains about the health effects of fine particulate matter at low atmospheric concentration. . . .”); *Mississippi*, 744 F. 3d at 1352–53 (appropriate for EPA to balance scientific uncertainties in determining level of revised O₃ NAAQS).

¹²⁹ Similarly, Senator Muskie remarked during the floor debates on the 1977 Amendments that “there is no such thing as a threshold for health effects. Even at the national primary standard level, which is the health standard, there are health effects that are not protected against”. 123 Cong. Rec. S9423 (daily ed. June 10, 1977).

In an additional overarching comment, some commenters also fundamentally objected to the EPA's consideration of exposure estimates in reaching conclusions on the primary O₃ standard. These commenters' general assertion was that NAAQS must be established so as to be protective, with an adequate margin of safety, regardless of the activity patterns that feed into exposure estimates. They contended that "[a]ir quality standards cannot rely on avoidance behavior in order to protect the public health and sensitive groups" and that "[i]t would be unlawful for EPA to set the standard at a level that is contingent upon people spending most of their time indoors" (e.g., ALA *et al.*, p. 124). To support these comments, for example, ALA *et al.* analyzed ambient monitoring data from Core-Based Statistical Areas (CBSAs) with design values between 66–70 ppb (Table 17, pp. 145–151 in ALA *et al.*) and 62–65 ppb (Table 18, pp. 153–154 in ALA *et al.*) and pointed out that there are many more days with ambient concentrations above the benchmark levels than were estimated in the EPA's exposure analysis (i.e., at and above the benchmark level of 60, 70 and 80 ppb).

The EPA disagrees with these commenters' conclusions regarding the appropriateness of considering exposure estimates, and notes that NAAQS must be "requisite" (i.e., "sufficient, but not more than necessary" (*Whitman*, 531 U.S. at 473)) to protect the "public health" ("the health of the public" (*Whitman*, 531 U.S. at 465)). Estimating exposure patterns based on extensive available data¹³⁰ is a reasonable means of ascertaining that standards are neither under- nor over-protective, and that standards address issues of public health rather than health issues pertaining only to isolated individuals.¹³¹ Behavior patterns are critical in assessing whether ambient concentrations of O₃ may pose a public health risk.¹³² Exposures to ambient or near-ambient O₃ concentrations have only been shown to result in potentially

adverse effects if the ventilation rates of people in the exposed populations are raised to a sufficient degree (e.g., through physical exertion) (U.S. EPA, 2013, section 6.2.1.1).¹³³ Ignoring whether such elevated ventilation rates are actually occurring, as advocated by these commenters, would not provide an accurate assessment of whether the public health is at risk. Indeed, a standard established without regard to behavior of the public would likely lead to a standard which is more stringent than necessary to protect the public health.

While setting the primary O₃ standard based only on ambient concentrations, without consideration of activity patterns and ventilation rates, would likely result in a standard that is over-protective, the EPA also concludes that setting a standard based on the assumption that people will adjust their activities to avoid exposures on high-pollution days would likely result in a standard that is under-protective. The HREA's exposure assessment does not make this latter assumption.¹³⁴ The time-location-activity diaries that provided the basis for exposure estimates reflect actual variability in human activities. While some diary days may reflect individuals spending less time outdoors than would be typical for them, it is similarly likely that some days reflect individuals spending more time outdoors than would be typical. Considering the actual variability in time-location-activity patterns is at the least a permissible way of identifying standards that are neither over- nor under-protective.¹³⁵

Further, the EPA sees nothing in the CAA that prohibits consideration of the O₃ exposures that could result in effects of public health concern. While a number of judicial opinions have upheld the EPA's decisions in other NAAQS reviews to place little weight on particular risk or exposure analyses (i.e., because of scientific uncertainties

in those analyses), none of these opinions have suggested that such analyses are irrelevant because actual exposure patterns do not matter. See, e.g. *Mississippi*, 744 F. 3d at 1352–53; *ATA III*, 283 F. 3d at 373–74. Therefore, because behavior patterns are critical in assessing whether ambient concentrations of O₃ may pose a public health risk, the EPA disagrees with the views expressed by these commenters objecting to the consideration of O₃ exposures in reaching decisions on the primary O₃ standard.

In addition to these overarching comments, a number of commenters supported their views on standard level by highlighting specific aspects of the scientific evidence, exposure/risk information, and/or CASAC advice. Key themes expressed by these commenters included the following: (1) Controlled human exposure studies provide strong evidence of adverse lung function decrements and airway inflammation in healthy adults following exposures to O₃ concentrations as low as 60 ppb, and at-risk populations would be likely to experience more serious effects or effects at even lower concentrations; (2) epidemiologic studies provide strong evidence for associations with mortality and morbidity in locations with ambient O₃ concentrations below 70 ppb, and in many cases in locations with concentrations near and below 60 ppb; (3) quantitative analyses in the HREA are biased such that they understate O₃ exposures and risks, and the EPA's interpretation of lung function risk estimates is not appropriate and not consistent with other NAAQS; and (4) the EPA must give deference to CASAC advice, particularly CASAC's policy advice to set the standard level below 70 ppb. The next sections discuss comments related to each of these points, and provide the EPA's responses to those comments. More detailed discussion of individual comments, and the EPA's responses, is provided in the Response to Comments document.

i. Effects in Controlled Human Exposure Studies

Some commenters who advocated for a level of 60 ppb (or absent that, for 65 ppb) asserted that controlled human exposure studies have reported adverse respiratory effects in healthy adults following exposures to O₃ concentrations as low as 60 ppb. These commenters generally based their conclusions on the demonstration of FEV₁ decrements ≥ 10% and increased airway inflammation following exposures of healthy adults to 60 ppb O₃. They concluded that even more serious effects would occur in at-risk

¹³⁰ The CHAD database used in the HREA's exposure assessment contains over 53,000 individual daily diaries including time-location-activity patterns for individuals of both sexes across a wide range of ages (U.S. EPA, 2014a, Chapter 5).

¹³¹ CASAC generally agreed with the EPA's methodology for characterizing exposures of concern (Frey, 2014a, pp. 5–6).

¹³² See 79 FR 75269 ("The activity pattern of individuals is an important determinant of their exposure. Variation in O₃ concentrations among various microenvironments means that the amount of time spent in each location, as well as the level of activity, will influence an individual's exposure to ambient O₃. Activity patterns vary both among and within individuals, resulting in corresponding variations in exposure across a population and over time" (internal citations omitted)).

¹³³ For healthy young adults exposed at rest for 2 hours, 500 ppb is the lowest O₃ concentration reported to produce a statistically significant O₃-induced group mean FEV₁ decrement (U.S. EPA, 2013, section 6.2.1.1).

¹³⁴ The EPA was aware of the possibility of averting behavior during the development of the HREA, and that document includes sensitivity analyses to provide perspective on the potential role of averting behavior in modifying O₃ exposures. As discussed further above (II.B.2.c), these sensitivity analyses were limited and the results were discussed in the proposal within the context of uncertainties in the HREA assessment of exposures of concern.

¹³⁵ See *Mississippi*, 744 F. 3d at 1343 ("[d]etermining what is 'requisite' to protect the 'public health' with an 'adequate' margin of safety may indeed require a contextual assessment of acceptable risk. See *Whitman*, 531 U.S. at 494–95 (Breyer, J. concurring . . .)"))

populations exposed to 60 ppb O₃, and that such populations would experience adverse effects following exposures to O₃ concentrations below 60 ppb.

While the EPA agrees that information from controlled human exposure studies conducted at 60 ppb can help to inform the Administrator's decision on the standard level, the Agency does not agree that this information necessitates a level below 70 ppb. In fact, as discussed in the proposal, a revised O₃ standard with a level of 70 ppb can be expected to provide substantial protection against the effects shown to occur following various O₃ exposure concentrations, including those observed following exposures to 60 ppb. This is because the degree of protection provided by any NAAQS is due to the combination of all of the elements of the standard (*i.e.*, indicator, averaging time, form, level). In the case of the fourth-high form of the O₃ NAAQS, which the Administrator is retaining in the current review (II.C.3), the large majority of days in areas that meet the standard will have 8-hour O₃ concentrations below the level of the standard, with most days well below the level. Therefore, as discussed in the proposal, in considering the degree of protection provided by an O₃ standard with a particular level, it is important to consider the extent to which that standard would be expected to limit population exposures of concern to the broader range of O₃ exposure concentrations shown in controlled human exposure studies to result in health effects. The Administrator's consideration of such exposures of concern is discussed below (II.C.4.c).

Another important part of the Administrator's consideration of exposure estimates is the extent to which she judges that adverse effects could occur following specific O₃ exposures. While controlled human exposure studies provide a high degree of confidence regarding the extent to which specific health effects occur following exposures to O₃ concentrations from 60 to 80 ppb, the Administrator notes that there are no universally accepted criteria by which to judge the adversity of the observed effects. Therefore, in making judgments about the extent to which the effects observed in controlled human exposure studies have the potential to be adverse, the Administrator considers the recommendations of ATS and advice from CASAC (II.A.1.c, above).

As an initial matter, with regard to the effects shown in controlled human exposure studies following O₃ exposures, the Administrator notes the following:

1. The largest respiratory effects, and the broadest range of effects, have been studied and reported following exposures to 80 ppb O₃ or higher, with most exposure studies conducted at these higher concentrations.

Specifically, 6.6-hour exposures of healthy young adults to 80 ppb O₃, while engaged in quasi-continuous, moderate exertion, can decrease lung function, increase airway inflammation, increase respiratory symptoms, result in airway hyperresponsiveness, and decrease lung host defenses.

2. Exposures of healthy young adults for 6.6 hours to O₃ concentrations as low as 72 ppb, while engaged in quasi-continuous, moderate exertion, have been shown to both decrease lung function and result in respiratory symptoms.

3. Exposures of healthy young adults for 6.6 hours to O₃ concentrations as low as 60 ppb, while engaged in quasi-continuous, moderate exertion, have been shown to decrease lung function and to increase airway inflammation.

To inform her judgments on the potential adversity to public health of these effects reported in controlled human exposure studies, as in the proposal, the Administrator considers the ATS recommendation that "reversible loss of lung function in combination with the presence of symptoms should be considered adverse" (ATS, 2000a). She notes that this combination of effects has been shown to occur following 6.6-hour exposures to O₃ concentrations at or above 72 ppb. In considering these effects, CASAC observed that "the combination of decrements in FEV₁ together with the statistically significant alterations in symptoms in human subjects exposed to 72 ppb ozone meets the American Thoracic Society's definition of an adverse health effect" (Frey, 2014c, p. 5).

Regarding the potential for adverse effects following exposures to lower concentrations, the Administrator notes the CASAC judgment that the adverse combination of lung function decrements and respiratory symptoms "almost certainly occur in some people" following exposures to O₃ concentrations below 72 ppb (Frey, 2014c, p. 6). In particular, when commenting on the extent to which the study by Schelegle et al. (2009) suggests the potential for adverse effects following O₃ exposures below 72 ppb, CASAC judged that:

[I]f subjects had been exposed to ozone using the 8-hour averaging period used in the standard [rather than the 6.6-hour exposures evaluated in the study], adverse effects could have occurred at lower concentration.

Further, in our judgment, the level at which adverse effects might be observed would likely be lower for more sensitive subgroups, such as those with asthma (Frey, 2014c, p. 5).

Though CASAC did not provide advice as to how far below 72 ppb adverse effects would likely occur, the Administrator agrees that such effects could occur following exposures at least somewhat below 72 ppb.

The Administrator notes that while adverse effects could occur following exposures at least somewhat below 72 ppb, the combination of statistically significant increases in respiratory symptoms and decrements in lung function has not been reported following 6.6-hour exposures to average O₃ concentrations of 60 ppb or 63 ppb, though studies have evaluated the potential for such effects (Adams, 2006; Schelegle *et al.*, 2009; Kim *et al.*, 2011). In the absence of this combination, the Administrator looks to additional ATS recommendations and CASAC advice in order to inform her judgments regarding the potential adversity of the effects that have been observed following O₃ exposures as low as 60 ppb.

With regard to ATS, she first notes the recommendations that "a small, transient loss of lung function, by itself, should not automatically be designated as adverse" and that "[f]ew . . . biomarkers have been validated sufficiently that their responses can be used with confidence to define the point at which a response should be equated to an adverse effect warranting preventive measures" (ATS, 2000a).¹³⁶ Based on these recommendations, compared to effects following exposures at or above 72 ppb, the Administrator has less confidence in the adversity of the respiratory effects that have been observed following exposures to 60 or 63 ppb.

She further notes that some commenters who advocated for a level of 60 ppb also focused on ATS recommendations regarding population-level risks. These commenters specifically stated that lung function decrements "may be adverse in terms of 'population risk,' where exposure to air pollution increases the risk to the population even though it might not harm lung function to a degree that is, on its own, 'clinically important' to an individual" (*e.g.*, ALA *et al.*, p. 118). These commenters asserted that the EPA

¹³⁶ With regard to this latter recommendation, as discussed above (II.A.1.c), the ATS concluded that elevations of biomarkers such as cell numbers and types, cytokines, and reactive oxygen species may signal risk for ongoing injury and more serious effects or may simply represent transient responses, illustrating the lack of clear boundaries that separate adverse from nonadverse events.

has not appropriately considered the potential for such population-level risk. Contrary to the views expressed by these commenters, the Administrator carefully considers the potential for population risk, particularly within the context of the ATS recommendation that “a shift in the risk factor distribution, and hence the risk profile of the exposed population, should be considered adverse, even in the absence of the immediate occurrence of frank illness” (ATS, 2000a). Given that exposures to 60 ppb O₃ have been shown in controlled human exposure studies to cause transient and reversible decreases in group mean lung function, the Administrator notes the potential for such exposures to result in similarly transient and reversible shifts in the risk profile of an exposed population. However, in contrast to commenters who advocated for a level of 60 ppb, the Administrator also notes that the available evidence does not provide information on the extent to which a short-term, transient decrease in lung function in a population, as opposed to a longer-term or permanent decrease, could affect the risk of other, more serious respiratory effects (*i.e.*, change the risk profile of the population). This uncertainty, together with the additional ATS recommendations noted above, indicates to the Administrator that her judgment that there is uncertainty in the adversity of the effects shown to occur at 60 ppb is consistent with ATS recommendations.¹³⁷

With regard to CASAC advice, the Administrator notes that, while CASAC clearly advised the EPA to consider the health effects shown to occur following exposures to 60 ppb O₃, its advice regarding the adversity of those effects is less clear. In particular, she notes that CASAC was conditional about whether the lung function decrements observed in some people at 60 ppb (*i.e.*, FEV₁ decrements \geq 10%) are adverse. Specifically, CASAC stated that these decrements “*could be adverse in individuals with lung disease*” (Frey, 2014c, p. 7, *emphasis added*) and that they provide a “*surrogate for adverse health outcomes for people with asthma and lung disease*” (Frey, 2014c, p. 3, *emphasis added*). Further, CASAC did not recommend considering standard levels low enough to eliminate O₃-induced FEV₁ decrements \geq 10% (Frey,

2014c). With regard to the full range of effects shown to occur at 60 ppb (*i.e.*, FEV₁ decrements, airway inflammation), CASAC stated that exposures of concern for the 60 ppb benchmark are “*relevant for consideration*” with respect to people with asthma (Frey, 2014c, p. 6, *italics added*). In addition, “[t]he CASAC concurs with EPA staff regarding the finding based on scientific evidence that a level of 60 ppb corresponds to the lowest exposure concentration demonstrated to result in lung function decrements large enough to be judged an abnormal response by ATS and that *could be adverse in individuals with lung disease*” (Frey, 2014c, p. 7, *italics added*). The Administrator contrasts these statements with CASAC’s clear advice that “the combination of decrements in FEV₁ together with the statistically significant alterations in symptoms in human subjects exposed to 72 ppb ozone meets the American Thoracic Society’s definition of an adverse health effect” (Frey, 2014c, p. 5).

Based on her consideration of all of the above recommendations and advice noted above, the Administrator judges that, compared to exposure concentrations at and above 72 ppb, there is greater uncertainty with regard to the adversity of effects shown to occur following O₃ exposures as low as 60 ppb. However, based on the effects that have been shown to occur at 60 ppb (*i.e.*, lung function decrements, airway inflammation), and CASAC advice indicating the importance of considering these effects (though its advice regarding the adversity of effects at 60 ppb is less clear), she concludes that it is appropriate to give some consideration to the extent to which a revised standard could allow such effects.

In considering estimates of exposures of concern for the 60, 70, and 80 ppb benchmarks within the context of her judgments on adversity, the Administrator notes that, due to interindividual variability in responsiveness, not every occurrence of an exposure of concern will result in an adverse effect. As discussed above (II.B.2.b.i), this point was highlighted by some commenters who opposed revision of the current standard, based on their analysis of effects shown to occur following exposures to 72 ppb O₃. This point was also highlighted by some commenters who advocated for a level of 60 ppb, based on the discussion of O₃-induced inflammation in the proposal. In particular, this latter group of commenters highlighted discussion from the proposal indicating that “[i]nflammation induced by a single O₃

exposure can resolve entirely but, as noted in the ISA (U.S. EPA, 2013, p. 6–76), ‘continued acute inflammation can evolve into a chronic inflammatory state’” (*e.g.*, ALA *et al.*, p. 48). Consistent with these comments, and with her consideration of estimated exposures of concern in the proposal, the Administrator judges that the types of respiratory effects that can occur following exposures of concern, particularly if experienced repeatedly, provide a plausible mode of action by which O₃ may cause other more serious effects. Because of this, as in the proposal, the Administrator is most concerned about protecting against repeated occurrences of exposures of concern.

The Administrator’s consideration of estimated exposures of concern is discussed in more detail below (II.C.4.b.iv, II.C.4.c). In summary, contrary to the conclusions of commenters who advocated for a level of 60 ppb, the Administrator judges that a revised standard with a level of 70 ppb will effectively limit the occurrence of the O₃ exposures for which she is most confident in the adversity of the resulting effects (*i.e.*, based on estimates for the 70 and 80 ppb benchmarks). She further concludes that such a standard will provide substantial protection against the occurrence of O₃ exposures for which there is greater uncertainty in the adversity of effects (*i.e.*, based on estimates for the 60 ppb benchmark).

As noted above, commenters also pointed out that benchmark concentrations are based on studies conducted in healthy adults, whereas at-risk populations are likely to experience more serious effects and effects at lower O₃ exposure concentrations. In considering this issue, the EPA notes CASAC’s endorsement of 60 ppb as the lower end of the range of benchmarks for evaluation, and its advice that “the 60 ppb-8hr exposure benchmark is relevant for consideration with respect to adverse effects on asthmatics” (Frey, 2014c, p. 6). As discussed in detail below (II.C.4.c), the Administrator has carefully considered estimated exposures of concern for the 60 ppb benchmark. In addition, though the available information does not support the identification of specific benchmarks below 60 ppb that could be appropriate for consideration for at-risk populations, and though CASAC did not recommend consideration of any such benchmarks, the EPA expects that a revised standard with a level of 70 ppb will also reduce the occurrence of exposures to O₃ concentrations at least somewhat below 60 ppb (U.S. EPA,

¹³⁷ ATS provided additional recommendations to help inform judgments regarding the adversity of air pollution-related effects (*e.g.*, related to “quality of life”), though it is not clear whether, or how, such recommendations should be applied to the respiratory effects observed in controlled human exposure studies following 6.6-hour O₃ exposures (ATS, 2000a, p. 672).

2014a, Figures 4–9 and 4–10).¹³⁸ Thus, even if some members of at-risk populations may experience effects following exposures to O₃ concentrations somewhat below 60 ppb, a revised level of 70 ppb would be expected to reduce the occurrence of such exposures.¹³⁹ Therefore, the EPA has considered O₃ exposures that could be relevant for at-risk populations such as children and people with asthma, and does not agree that controlled human exposure studies reporting respiratory effects in healthy adults following exposures to 60 ppb O₃ necessitate a standard level below 70 ppb.

ii. Epidemiologic Studies

Commenters representing environmental and public health organizations also highlighted epidemiologic studies that, in their view, provide strong evidence for associations with mortality and morbidity in locations with ambient O₃ concentrations near and below 60 ppb. These commenters focused both on the epidemiologic studies evaluated in the PA's analyses of study location air quality (U.S. EPA, 2014c, Chapter 4) and on studies that were not explicitly analyzed in the PA, and in some cases on studies that were not included in the ISA.

The EPA agrees that epidemiologic studies can provide perspective on the degree to which O₃-associated health effects have been identified in areas with air quality likely to have met various standards. However, as discussed below, we do not agree with the specific conclusions drawn by these commenters regarding the implications of epidemiologic studies for the standard level. As an initial matter in considering epidemiologic studies, the EPA notes its decision, consistent with CASAC advice, to place the most emphasis on information from controlled human exposure studies (II.B.2 and II.B.3, above). This decision reflects the greater certainty in using information from controlled human exposure studies to link specific O₃ exposures with health effects, compared to using air quality information from epidemiologic studies of O₃ for this purpose.

¹³⁸ Air quality analyses in the HREA indicate that reducing the level of the primary standard from 75 ppb to 70 ppb will result in reductions in the O₃ concentrations in the upper portions of ambient distributions. This includes 8-hour ambient O₃ concentrations at, and somewhat below, 60 ppb (U.S. EPA, 2014a, Figures 4–9 and 4–10).

¹³⁹ The uncertainty associated with the potential adversity of any such effects would be even greater than that discussed above for the 60 ppb benchmark.

While being aware of the uncertainties discussed above (II.B.2.b.ii), in considering what epidemiologic studies can tell us, the EPA notes analyses in the PA (U.S. EPA, 2014c, section 4.4.1) indicating that a revised standard with a level at or below 70 ppb would be expected to maintain distributions of short-term ambient O₃ concentrations below those present in the locations of all of the single-city epidemiologic studies analyzed. As discussed in the PA (U.S. EPA, 2014c, section 4.4.1), this includes several single-city studies conducted in locations that would have violated the current standard, and the study by Mar and Koenig (2009) that reported positive and statistically significant associations with respiratory emergency department visits with children and adults in a location that would have met the current standard over the entire study period, but would have violated a standard with a level of 70 ppb.¹⁴⁰ While these analyses provide support for a level at least as low as 70 ppb, the Administrator judges that they do not provide a compelling basis for distinguishing between the appropriateness of 70 ppb and lower standard levels.

As in the proposal, the EPA acknowledges additional uncertainty in interpreting air quality in locations of multicity epidemiologic studies of short-term O₃ for the purpose of evaluating alternative standard levels (U.S. EPA, 2014c, sections 3.1.4.2, 4.4.1). In particular, the PA concludes that interpretation of such air quality information is complicated by uncertainties in the extent to which multicity effect estimates (*i.e.*, which are based on combining estimates from multiple study locations) can be attributed to ambient O₃ in the subset of study locations that would have met a particular standard, versus O₃ in the study locations that would have violated the standard. While giving only limited weight to air quality analyses in these study areas because of this uncertainty, the EPA also notes PA analyses indicating that a standard level at or below 70 ppb would require additional reductions, beyond those required by the current standard, in the ambient O₃ concentrations that provided the basis for statistically significant O₃ health effect associations in multicity epidemiologic studies. As

¹⁴⁰ As noted above (II.B.2.b.ii and II.B.3), the studies by Silverman and Ito (2010) and Strickland *et al.* (2010) provided support for the Administrator's decision to revise the current primary O₃ standard, but do not provide insight into the appropriateness of specific standard levels below 75 ppb.

was the case for the single-city studies, and contrary to the views expressed by the commenters noted above, the Administrator judges that these studies do not provide a compelling basis for distinguishing between the appropriateness of alternative standard levels at or below 70 ppb.

In some cases, commenters highlighted studies that were assessed in the 2008 review of the O₃ NAAQS, but were not included in the ISA in the current review. These commenters asserted that such studies support the occurrence of O₃ health effect associations in locations with air quality near or, in some cases, below 60 ppb. Specifically, commenters highlighted a number of studies included in the 2007 Staff Paper that were not included in the ISA, claiming that these studies support a standard level below 70 ppb, and as low as 60 ppb.

As an initial matter with regard to these studies, the EPA notes that the focus of the ISA is on assessing the most policy-relevant scientific evidence. In the current review, the ISA considered over 1,000 new studies that have been published since the last review. Thus, it is not surprising that, as the body of evidence has been strengthened since the last review, some of the studies considered in the last review are no longer among the most policy relevant. However, based on the information included in the 2007 Staff Paper, the EPA does not agree that the studies highlighted by commenters provide compelling support for a level below 70 ppb. In fact, as discussed in the Staff Paper in the last review (U.S. EPA, 2007, p. 6–9; Appendix 3B), the O₃ concentrations reported for these studies, and the concentrations highlighted by commenters, were based on averaging across multiple monitors in study areas. Given that the highest monitor in an area is used to determine whether that area meets or violates the NAAQS, the averaged concentrations reported in the Staff Paper are thus not appropriate for direct comparison to the level of the O₃ standard. When the Staff Paper considered the O₃ concentrations measured at individual monitors for the subset of these study areas with particularly low concentrations, they were almost universally found to be above, and in many cases well above, even the current standard level of 75 ppb.¹⁴¹ Based on the above

¹⁴¹ For one study conducted in Vancouver, where data from individual monitors did indicate ambient concentrations below the level of the current standard (Vedal *et al.*, 2003), the Staff Paper noted that the study authors questioned whether O₃, other gaseous pollutants, and PM in this study may be

Continued

considerations, and consistent with the Administrator's overall decision to place less emphasis on air quality in locations of epidemiologic studies to select a standard level, the EPA disagrees with commenters who asserted that epidemiologic studies included in the last review, but not cited in the ISA or PA in this review, necessitate a level below 70 ppb. In fact, the EPA notes that these studies are consistent with the majority of the U.S. studies evaluated in the PA in the current review, in that most were conducted in locations that would have violated the current O₃ NAAQS over at least part of the study periods.

iii. Exposure and Risk Assessments

Some commenters supporting levels below 70 ppb also asserted that quantitative analyses in the HREA are biased such that they understate O₃ exposures of concern and risks of O₃-induced FEV₁ decrements. Many of these comments are discussed above within the context of the adequacy of the current standard (II.B.2.b.i), including comments pointing out that exposure and risk estimates are based on information from healthy adults rather than at-risk populations; comments noting that the exposure assessment evaluates 8-hour O₃ exposures rather than the 6.6-hour exposures used in controlled human exposure studies; and comments asserting that the EPA's exposure and risk analyses rely on people staying indoors on high pollution days (*i.e.*, averting behavior).

As discussed in section II.B.2.b.i above, while the EPA agrees with certain aspects of these commenters' assertions, we do not agree with their overall conclusions. In particular, there are aspects of the HREA's quantitative analyses that, if viewed in isolation, would tend to either overstate or understate O₃ exposures and/or health risks. While commenters tended to focus on those aspects of the assessments that support their position, they tended to ignore aspects of the assessments that do not support their position (points that were often raised by commenters on the other side of the issue). Rather than viewing the potential implications of these aspects of the HREA assessments in isolation, the EPA considers them together, along with

acting as surrogate markers of pollutant mixes that contain more toxic compounds, "since the low measured concentrations were unlikely, in their opinion, to cause the observed effects" (U.S. EPA, 2007, p. 6–16). The Staff Paper further noted that another study conducted in Vancouver failed to find statistically significant associations with O₃ (Villeneuve *et al.*, 2003).

other issues and uncertainties related to the interpretation of exposure and risk estimates.

For example, some commenters who advocated for a level below 70 ppb asserted that the exposure assessment could underestimate O₃ exposures for highly active populations, including outdoor workers and children who spend a large portion of time outdoors during summer. In support of these assertions, commenters highlighted sensitivity analyses conducted in the HREA. However, as noted in the HREA (U.S. EPA, 2014a, Table 5–10), this aspect of the assessment is likely to have only a "low to moderate" impact on the magnitude of exposure estimates. To put this magnitude in perspective, HREA sensitivity analyses conducted in a single urban study area indicate that, regardless of whether exposure estimates for children are based on all available diaries or on a subset of diaries restricted to simulate highly exposed children, a revised standard with a level of 70 ppb is estimated to protect more than 99% of children from experiencing two or more exposures of concern at or above 70 ppb (U.S. EPA, 2014a, Chapter 5 Appendices, Figure 5G–9).¹⁴² ¹⁴³ In contrast to the focus of commenters who supported a level below 70 ppb, other aspects of quantitative assessments, some of which were highlighted by commenters who opposed revising the current standard (II.B.2), tend to result in overestimates of O₃ exposures. These aspects are characterized in the HREA as having either a "low," a "low-to-moderate," or a "moderate" impact on the magnitudes of exposure estimates.

In its reviews of the HREA and PA, CASAC recognized many of the uncertainties and issues highlighted by commenters. Even considering these uncertainties, CASAC endorsed the approaches adopted by the EPA to assess O₃ exposures and health risks, and CASAC used exposure and risk estimates as part of the basis for their recommendations on the primary O₃ NAAQS (Frey, 2014c). Thus, as discussed in section II.B.2.b.i above, the

¹⁴² More specifically, based on all children's diaries, just under 0.1% of children are estimated to experience two or more exposures of concern at or above 70 ppb. Based on simulated profiles of highly exposed children, this estimate increased to just over 0.1% (U.S. EPA, 2014a, Chapter 5 Appendices, Figure 5G–9).

¹⁴³ In addition, when diaries were selected to mimic exposures that could be experienced by outdoor workers, the percentages of modeled individuals estimated to experience exposures of concern were generally similar to the percentages estimated for children (*i.e.*, using the full database of diary profiles) in the worst-case cities and years (*i.e.*, cities and years with the highest exposure estimates) (U.S. EPA, 2014, section 5.4.3.2, Figure 5–14).

EPA disagrees with commenters who claim that the aspects of the quantitative assessments that they highlight lead to overall underestimates of exposures or health risks.¹⁴⁴

Some commenters further contended that the level of the primary O₃ standard should be set below 70 ppb in order to compensate for the use of a form that allows multiple days with concentrations higher than the standard level. These groups submitted air quality analyses to support their point that the current fourth-high form allows multiple days per year with ambient O₃ concentrations above the level of the standard. While the EPA does not dispute the air quality analyses submitted by these commenters, and agrees that fourth-high form allows multiple days per year with ambient O₃ concentrations above the level of the standard (3 days per year, on average over a 3-year period), the Agency disagrees with commenters' assertion that, because of this, the level of the primary O₃ standard should be set below 70 ppb. As discussed above (II.A.2), the quantitative assessments that informed the Administrator's proposed decision, presented in the HREA and considered in the PA and by CASAC, estimated O₃ exposures and health risks associated with air quality that "just meets" various standards with the current 8-hour averaging time and fourth-high, 3-year average form. Thus, in considering the degree of public health protection appropriate for the primary O₃ standard, the Administrator has considered quantitative exposure and risk estimates that are based a fourth-high form, and therefore on a standard that, as these commenters point out, allows multiple days per year with ambient O₃ concentrations above the level of the standard.

iv. CASAC Advice

Many commenters, including those representing major medical, public health, or environmental groups; some state agencies; and a large number of individual commenters, focused on CASAC advice in their rationale supporting levels below 70 ppb, and as low as 60 ppb. These commenters generally asserted that the EPA must

¹⁴⁴ As discussed in II.B.2.b above, in weighing the various uncertainties, which can bias exposure results in different directions but tend to have impacts that are similar in magnitude (U.S. EPA, 2014a, Table 5–10), and in light of CASAC's advice based on its review of the HREA and the PA, the EPA continues to conclude that the approach to considering estimated exposures of concern in the HREA, PA, and the proposal reflects an appropriate balance, and provides an appropriate basis for considering the public health protectiveness of the primary O₃ standard.

give deference to CASAC. In some cases, these commenters expressed strong objections to a level of 70 ppb, noting CASAC policy advice that such a level would provide little margin of safety.

The EPA agrees that CASAC advice is an important consideration in reaching a decision on the standard level (see *e.g.* CAA section 307 (d)(3)),¹⁴⁵ though not with commenters' conclusion that CASAC advice necessitates a standard level below 70 ppb. As discussed above (II.C.4.a), the Administrator carefully considered CASAC advice in the proposal, and she judged that her proposed decision to revise the level to within the range of 65 to 70 ppb was consistent with CASAC advice, based on the available science.

As in the proposal, in her final decision on level the Administrator notes CASAC's overall conclusion that "based on the scientific evidence from clinical studies, epidemiologic studies, animal toxicology studies, as summarized in the ISA, the findings from the exposure and risk assessments as summarized in the HREA, and the interpretation of the implications of all of these sources of information as given in the Second Draft PA . . . there is adequate scientific evidence to recommend a range of levels for a revised primary ozone standard from 70 ppb to 60 ppb" (Frey, 2014c, p. 8). Thus, CASAC used the health evidence and exposure/risk information to inform its range of recommended standard levels, a range that included an upper bound of 70 ppb based on the scientific evidence, and it did not use the evidence and information to recommend setting the primary O₃ standard at any specific level within the range of 70 to 60 ppb. In addition, CASAC further stated that "the choice of a level within the range recommended based on scientific evidence [*i.e.*, 70 to 60 ppb] is a policy judgment under the statutory mandate of the Clean Air Act" (Frey, 2014c, p. ii).

In addition to its advice based on the scientific evidence, CASAC offered the "policy advice" to set the level below 70 ppb, stating that a standard level of 70 ppb "may not meet the statutory requirement to protect public health with an adequate margin of safety" (Frey, 2014c, p. ii). In supporting its policy advice to set the level below 70 ppb, CASAC noted the respiratory effects that have been shown to occur in controlled human exposure studies following exposures from 60 to 80 ppb

O₃, and the extent to which various standard levels are estimated to allow the occurrence of population exposures that can result in such effects (Frey, 2014c, pp. 7–8).

The EPA agrees that an important consideration when reaching a decision on level is the extent to which a revised standard is estimated to allow the types of exposures shown in controlled human exposure studies to cause respiratory effects. In reaching her final decision that a level of 70 ppb is requisite to protect public health with an adequate margin of safety (II.C.4.c, below), the Administrator carefully considers the potential for such exposures and effects. In doing so, she emphasizes the importance of setting a standard that limits the occurrence of the exposures about which she is most concerned (*i.e.*, those for which she has the most confidence in the adversity of the resulting effects, which are repeated exposures of concern at or above 70 or 80 ppb, as discussed above in II.C.4.b.i). Based on her consideration of information from controlled human exposure studies in light of CASAC advice and ATS recommendations, the Administrator additionally judges that there is important uncertainty in the extent to which the effects shown to occur following exposures to 60 ppb O₃ are adverse to public health (discussed above, II.C.4.b.i and II.C.4.b.iii). However, based on the effects that have been shown to occur, CASAC advice indicating the importance of considering these effects, and ATS recommendations indicating the potential for adverse population-level effects (II.C.4.b.i, II.C.4.b.iii), she concludes that it is appropriate to give some consideration to the extent to which a revised standard could allow the respiratory effects that have been observed following exposures to 60 ppb O₃.

When considering the extent to which a revised standard could allow O₃ exposures that have been shown in controlled human exposures studies to result in respiratory effects, the Administrator is most concerned about protecting the public, including at-risk populations, against repeated occurrences of such exposures of concern (II.C.4.b.i, above). In considering the appropriate metric for evaluating repeated occurrences of exposures of concern, the Administrator acknowledges that it is not clear from the evidence, or from the ATS recommendations, CASAC advice, or public comments, how particular numbers of exposures of concern could impact the seriousness of the resulting effects, especially at lower exposure

concentrations. Therefore, the Administrator judges that focusing on HREA estimates of two or more exposures of concern provides a health-protective approach to considering the potential for repeated occurrences of exposures of concern that could result in adverse effects. She notes that other possible metrics for considering repeated occurrences of exposures of concern (*e.g.*, 3 or more, 4 or more, etc.) would result in smaller exposure estimates.

As discussed further below (II.C.4.c), the Administrator notes that a revised standard with a level of 70 ppb is estimated to eliminate the occurrence of two or more exposures of concern to O₃ concentrations at or above 80 ppb and to virtually eliminate the occurrence of two or more exposures of concern to O₃ concentrations at or above 70 ppb (Table 1, above). For the 70 ppb benchmark, this reflects about a 90% reduction in the number of children estimated to experience two or more exposures of concern, compared to the current standard.¹⁴⁶ Even considering the worst-case urban study area and worst-case year evaluated in the HREA, a standard with a level of 70 ppb is estimated to protect more than 99% of children from experiencing two or more exposures of concern to O₃ concentrations at or above 70 ppb (Table 1).

Though the Administrator judges that there is greater uncertainty with regard to the occurrence of adverse effects following exposures as low as 60 ppb, she notes that a revised standard with a level of 70 ppb is estimated to protect the vast majority of children in urban study areas (*i.e.*, about 96% to more than 99% in individual areas) from experiencing two or more exposures of concern at or above 60 ppb. Compared to the current standard, this represents a reduction of more than 60% in exposures of concern for the 60 ppb benchmark (Table 1). Given the Administrator's uncertainty regarding the adversity of the effects following exposures to 60 ppb O₃, and her health-protective approach to considering repeated occurrences of exposures of concern, the Administrator judges that this degree of protection is appropriate and that it reflects substantial protection against the occurrence of O₃-induced effects, including effects for which she judges the adversity to public health is uncertain.

¹⁴⁵ The EPA notes, of course, that the CAA places the responsibility for judging what standard is requisite with the Administrator and only requires that, if her decision differs in important ways from CASAC's advice, she explain her reasoning for differing.

¹⁴⁶ Percent reductions in this section refer to reductions in the number of children in HREA urban study areas (averaged over the years evaluated in the HREA) estimated to experience exposures of concern, based on the information in Table 1 above.

While being less concerned about single occurrences of exposures of concern, especially at lower exposure concentrations, the Administrator also notes that a standard with a level of 70 ppb is estimated to (1) virtually eliminate all occurrences of exposures of concern at or above 80 ppb; (2) protect \geq about 99% of children in urban study areas from experiencing any exposures of concern at or above 70 ppb; and (3) to achieve substantial reductions (*i.e.*, about 50%), compared to the current standard, in the occurrence of one or more exposures of concern at or above 60 ppb (Table 1).

Given the information and advice noted above (and in II.C.4.b.i, II.C.4.b.iii), the Administrator judges that a revised standard with a level of 70 ppb will effectively limit the occurrence of the O₃ exposures for which she has the most confidence in the adversity of the resulting effects (*i.e.*, based on estimates for the 70 and 80 ppb benchmarks). She further judges that such a standard will provide a large degree of protection against O₃ exposures for which there is greater uncertainty in the adversity of effects (*i.e.*, those observed following exposures to 60 ppb O₃), contributing to the margin of safety of the standard. See *Mississippi*, 744 F. 3d at 1353 (“By requiring an ‘adequate margin of safety’, Congress was directing EPA to build a buffer to protect against uncertain and unknown dangers to human health”). Given the considerable protection provided against repeated exposures of concern for all of the benchmarks evaluated, including the 60 ppb benchmark, the Administrator judges that a standard with a level of 70 ppb will provide an adequate margin of safety against the adverse O₃-induced effects shown to occur following exposures at or above 72 ppb, and judged by CASAC likely to occur following exposures somewhat below 72 ppb.¹⁴⁷

Contrary to the conclusions of commenters who advocated for a level below 70 ppb, the Administrator notes that her final decision is consistent with CASAC’s advice, based on the scientific evidence, and with CASAC’s focus on

setting a revised standard to further limit the occurrence of the respiratory effects observed in controlled human exposure studies, including effects observed following exposures to 60 ppb O₃. Given her judgments and conclusions discussed above, and given that the CAA reserves the choice of the standard that is requisite to protect public health with an adequate margin of safety for the judgment of the EPA Administrator, she disagrees with commenters who asserted that CASAC advice necessitates a level below 70 ppb, and as low as 60 ppb. The Administrator’s final conclusions on level are discussed in more detail below (II.C.4.c).

c. Administrator’s Final Decision Regarding Level

Having carefully considered the public comments on the appropriate level of the primary O₃ standard, as discussed above and in the Response to Comments document, the Administrator believes her scientific and policy judgments in the proposal remain valid. In conjunction with her decisions to retain the current indicator, averaging time, and form (II.C.1 to II.C.3, above), the Administrator is revising the level of the primary O₃ standard to 70 ppb. In doing so, she is selecting a primary O₃ standard that is requisite to protect public health with an adequate margin of safety, in light of her judgments based on an interpretation of the scientific evidence and exposure/risk information that neither overstates nor understates the strengths and limitations of that evidence and information and the appropriate inferences to be drawn therefrom.

The Administrator’s decision to revise the level of the primary O₃ standard to 70 ppb builds upon her conclusion that the overall body of scientific evidence and exposure/risk information calls into question the adequacy of public health protection afforded by the current standard, particularly for at-risk populations and lifestages (II.B.3).¹⁴⁸ Consistent with the proposal, her decision on level places the greatest emphasis on the results of controlled human exposure studies and on quantitative analyses based on information from these studies, particularly analyses of O₃ exposures of concern. As in the proposal, and as discussed further below, she views the results of the lung function risk assessment, analyses of O₃ air quality in

locations of epidemiologic studies, and epidemiology-based quantitative health risk assessments as providing information in support of her decision to revise the current standard, but a more limited basis for selecting a particular standard level among a range of options. See *Mississippi*, 744 F. 3d at 1351–52 (studies can legitimately support a decision to revise the standard, but not provide sufficient information to justify their use in setting the level of a revised standard).

Given her consideration of the evidence, exposure/risk information, advice from CASAC, and public comments, the Administrator judges that a standard with a level of 70 ppb is requisite to protect public health with an adequate margin of safety. She notes that the determination of what constitutes an adequate margin of safety is expressly left to the judgment of the EPA Administrator. See *Lead Industries Association v. EPA*, 647 F.2d at 1161–62; *Mississippi*, 744 F. 3d at 1353. She further notes that in evaluating how particular standards address the requirement to provide an adequate margin of safety, it is appropriate to consider such factors as the nature and severity of the health effects, the size of sensitive population(s) at risk, and the kind and degree of the uncertainties present (I.B, above). Consistent with past practice and long-standing judicial precedent, the Administrator takes the need for an adequate margin of safety into account as an integral part of her decision-making on the appropriate level, averaging time, form, and indicator of the standard.¹⁴⁹

In considering the need for an adequate margin of safety, the Administrator notes that a standard with a level of 70 ppb O₃ would be expected to provide substantial improvements in public health, including for at-risk groups such as children and people with asthma. The following paragraphs summarize the basis for the Administrator’s conclusion that a revised primary O₃ standard with a level of 70 ppb is requisite to protect the public health with an adequate margin of safety.

As an initial matter, consistent with her conclusions on the need for revision of the current standard (II.B.3), in reaching a decision on level the Administrator places the most weight on information from controlled human exposure studies. In doing so, she notes that controlled human exposure studies provide the most certain evidence indicating the occurrence of health

¹⁴⁷ As discussed above (II.C.4.b.i), when commenting on the extent to which the study by Schelegle *et al.* (2009) suggests the potential for adverse effects following O₃ exposures below 72 ppb, CASAC stated the following: “[I]f subjects had been exposed to ozone using the 8-hour averaging period used in the standard [rather than the 6.6-hour exposures evaluated in the study], adverse effects could have occurred at lower concentration. Further, in our judgment, the level at which adverse effects might be observed would likely be lower for more sensitive subgroups, such as those with asthma” (Frey, 2014c, p. 5).

¹⁴⁸ At-risk populations include people with asthma; children and older adults; people who are active outdoors, including outdoor workers; people with certain genetic variants; and people with reduced intake of certain nutrients.

¹⁴⁹ See, *e.g.* *NRDC v. EPA*, 902 F. 2d 962, 973–74 (D.C. Cir. 1990).

effects in humans following specific O₃ exposures. In particular, she notes that the effects reported in controlled human exposure studies are due solely to O₃ exposures, and interpretation of study results is not complicated by the presence of co-occurring pollutants or pollutant mixtures (as is the case in epidemiologic studies). The Administrator also observes that her emphasis on information from controlled human exposure studies is consistent with CASAC's advice and interpretation of the scientific evidence (Frey, 2014c).

With regard to the effects shown in controlled human exposure studies following specific O₃ exposures, as discussed in more detail above (II.B, II.C.4.b.i), the Administrator notes that (1) the largest respiratory effects, and the broadest range of effects, have been studied and reported following exposures to 80 ppb O₃ or higher (*i.e.*, decreased lung function, increased airway inflammation, increased respiratory symptoms, AHR, and decreased lung host defense); (2) exposures to O₃ concentrations as low as 72 ppb have been shown to both decrease lung function and result in respiratory symptoms; and (3) exposures to O₃ concentrations as low as 60 ppb have been shown to decrease lung function and to increase airway inflammation.

While such controlled human exposure studies provide a high degree of confidence regarding the occurrence of health effects following exposures to O₃ concentrations from 60 to 80 ppb, there are no universally accepted criteria by which to judge the adversity of the observed effects. To inform her judgments on the potential adversity to public health of effects reported in controlled human exposure studies, the Administrator considers ATS recommendations and CASAC advice, as described in detail above (II.B.2, II.C.4.b.i, II.C.4.b.iii, II.C.4.b.iv). Based on her consideration of such recommendations and advice, the Administrator is confident that the respiratory effects that have been observed following exposures to 72 ppb O₃ or above can be adverse. In addition, she judges that adverse effects are likely to occur following exposures somewhat below 72 ppb (II.C.4.b.i). However, as described above (II.C.4.b.i, II.C.4.b.iii, II.C.4.b.iv), the Administrator is notably less confident in the adversity to public health of the respiratory effects that have been observed following exposures to O₃ concentrations as low as 60 ppb, given her consideration of the following: (1) ATS recommendations indicating uncertainty in judging adversity based

on lung function decrements alone; (2) uncertainty in the extent to which a short-term, transient population-level decrease in FEV₁ would increase the risk of other, more serious respiratory effects in that population (*i.e.*, per ATS recommendations on population-level risk); and (3) compared to 72 ppb, CASAC advice is less clear regarding the potential adversity of effects at 60 ppb.

Taken together, the Administrator concludes that the evidence from controlled human exposure studies provides strong support for her conclusion that a revised standard with a level of 70 ppb is requisite to protect the public health with an adequate margin of safety. She bases this conclusion, in part, on the fact that such a standard level would be well below the O₃ exposure concentration shown to result in the widest range of respiratory effects (*i.e.*, 80 ppb), and below the lowest O₃ exposure concentration shown to result in the adverse combination of lung function decrements and respiratory symptoms (*i.e.*, 72 ppb). See *Lead Industries*, 647 F. 2d at 1160 (setting NAAQS at level well below the level where the clearest adverse effects occur, and at a level eliminating most "sub-clinical effects" provides an adequate margin of safety).

As discussed above (II.C.4.b.i), the Administrator also notes that a revised O₃ standard with a level of 70 ppb can provide substantial protection against the broader range of O₃ exposure concentrations that have been shown in controlled human exposure studies to result in respiratory effects, including exposure concentrations below 70 ppb. The degree of protection provided by any NAAQS is due to the combination of all of the elements of the standard (*i.e.*, indicator, averaging time, form, level) and, in the case of the fourth-high form of the revised primary O₃ standard (II.C.3), the large majority of days in areas that meet the revised standard will have 8-hour O₃ concentrations below 70 ppb, with most days having 8-hour O₃ concentrations well below this level. In addition, the degree of protection provided by the O₃ NAAQS is also dependent on the extent to which people experience health-relevant O₃ exposures in locations meeting the NAAQS. As discussed above, for a pollutant like O₃ where adverse responses are critically dependent on ventilation rates, the Administrator notes that it is important to consider activity patterns in the exposed population. Not considering activity patterns, and corresponding ventilation rates, can result in a standard that provides more protection than is requisite. Therefore, as discussed in the

proposal, in considering the degree of protection provided by a revised primary O₃ standard, the Administrator considers the extent to which that standard would be expected to limit population exposures of concern (*i.e.*, which take into account activity patterns and estimated ventilation rates) to the broader range of O₃ exposure concentrations shown to result in health effects.

Due to interindividual variability in responsiveness, the Administrator notes that not every occurrence of an exposure of concern will result in an adverse effect (II.C.4.b.i). Moreover, repeated occurrences of some of the effects demonstrated following exposures of concern could increase the likelihood of adversity (U.S. EPA, 2013, Section 6.2.3, p. 6–76). In particular, she notes that the types of respiratory effects that can occur following exposures of concern, particularly if experienced repeatedly, provide a plausible mode of action by which O₃ may cause other more serious effects. Therefore, as in the proposal, the Administrator is most concerned about protecting at-risk populations against repeated occurrences of exposures of concern. In considering the appropriate metric for evaluating repeated occurrences of exposures of concern, the Administrator acknowledges that it is not clear from the evidence, or from the ATS recommendations, CASAC advice, or public comments, how particular numbers of exposures of concern could impact the seriousness of the resulting effects, especially at lower exposure concentrations. Therefore, the Administrator judges that focusing on HREA estimates of two or more exposures of concern provides a health-protective approach to considering the potential for repeated occurrences of exposures of concern that could result in adverse effects.

Based on her consideration of adversity discussed above, the Administrator places the most emphasis on setting a standard that appropriately limits repeated occurrences of exposures of concern at or above the 70 and 80 ppb benchmarks. She notes that a revised standard with a level of 70 ppb is estimated to eliminate the occurrence of two or more exposures of concern to O₃ concentrations at or above 80 ppb and to virtually eliminate the occurrence of two or more exposures of concern to O₃ concentrations at or above 70 ppb for all children and children with asthma, even in the worst-case year and location evaluated.

While she is less confident that adverse effects will occur following exposures to O₃ concentrations as low as 60 ppb, as discussed above, the

Administrator judges that it is also appropriate to consider estimates of exposures of concern for the 60 ppb benchmark. Consistent with this judgment, although CASAC advice regarding the potential adversity of effects at 60 ppb was less definitive than for effects at 72 ppb, CASAC did clearly advise the EPA to consider the extent to which a revised standard is estimated to limit the effects observed following 60 ppb exposures (Frey, 2014c). Therefore, the Administrator considers estimated exposures of concern for the 60 ppb benchmark, particularly considering the extent to which the health protection provided by a revised standard includes a margin of safety against the occurrence of adverse O₃-induced effects. The Administrator notes that a revised standard with a level of 70 ppb is estimated to protect the vast majority of children in urban study areas (*i.e.*, about 96% to more than 99% of children in individual areas) from experiencing two or more exposures of concern at or above 60 ppb. Compared to the current standard, this represents a reduction of more than 60%.

Given the considerable protection provided against repeated exposures of concern for all of the benchmarks evaluated, including the 60 ppb benchmark, the Administrator judges that a standard with a level of 70 ppb will incorporate a margin of safety against the adverse O₃-induced effects shown to occur following exposures at or above 72 ppb, and judged likely to occur following exposures somewhat below 72 ppb.

While the Administrator is less concerned about single occurrences of O₃ exposures of concern, especially for the 60 ppb benchmark, she judges that estimates of one or more exposures of concern can provide further insight into the margin of safety provided by a revised standard. In this regard, she notes that a standard with a level of 70 ppb is estimated to (1) virtually eliminate all occurrences of exposures of concern at or above 80 ppb; (2) protect the vast majority of children in urban study areas from experiencing any exposures of concern at or above 70 ppb (*i.e.*, \geq about 99%, based on mean estimates; Table 1); and (3) to achieve substantial reductions, compared to the current standard, in the occurrence of one or more exposures of concern at or above 60 ppb (*i.e.*, about a 50% reduction; Table 1). The Administrator judges that these results provide further support for her conclusion that a standard with a level of 70 ppb will incorporate an adequate margin of safety against the occurrence of O₃ exposures

that can result in effects that are adverse to public health.

The Administrator additionally judges that a standard with a level of 70 ppb would be expected to result in important reductions, compared to the current standard, in the population-level risk of O₃-induced lung function decrements ($\geq 10\%$, $\geq 15\%$) in children, including children with asthma. Specifically, a revised standard with a level of 70 ppb is estimated to reduce the risk of two or more O₃-induced decrements by about 30% and 20% for decrements ≥ 15 and 10%, respectively (Table 2, above). However, as discussed above (II.C.4.b.i), the Administrator judges that there are important uncertainties in using lung function risk estimates as a basis for considering the occurrence of adverse effects in the population given (1) the ATS recommendation that “a small, transient loss of lung function, by itself, should not automatically be designated as adverse” (ATS, 2000a); (2) uncertainty in the extent to which a transient population-level decrease in FEV₁ would increase the risk of other, more serious respiratory effects in that population (*i.e.*, per ATS recommendations on population-level risk); and (3) that CASAC did not advise considering a standard that would be estimated to eliminate O₃-induced lung function decrements ≥ 10 or 15% (Frey, 2014c). Moreover, as at proposal, the Administrator notes that the variability in lung function risk estimates across urban study areas is often greater than the differences in risk estimates between various standard levels (Table 2, above).¹⁵⁰ Given this, and the resulting considerable overlap between the ranges of lung function risk estimates for different standard levels, the Administrator puts limited weight on the lung function risk estimates for distinguishing between the degrees of public health protection provided by alternative standard levels. Therefore, the Administrator judges that while a standard with a level of 70 ppb would be expected to result in important reductions, compared to the current standard, in the population-level risk of O₃-induced lung function decrements ($>10\%$, 15%) in children, including children with asthma, she also judges that estimated risks of O₃-induced lung function decrements provide a more limited basis than exposures of concern for distinguishing between the

¹⁵⁰ For example, the average percentage of children estimated to experience two or more decrements $\geq 10\%$ ranges from approximately 6 to 11% for a standard level of 70 ppb, up to about 9% for a level of 65 ppb, and up to about 6% for a level of 60 ppb (Table 2, above).

appropriateness of the health protection afforded by a standard level of 70 ppb versus lower levels.

The Administrator also considers the epidemiologic evidence and the quantitative risk estimates based on information from epidemiologic studies. As discussed in the proposal, and above in the EPA’s responses to significant comments, although the Administrator acknowledges the important uncertainties in using the O₃ epidemiologic studies as a basis for selecting a standard level, she notes that these studies can provide perspective on the degree to which O₃-associated health effects have been identified in areas with air quality likely to have met various standards. Specifically, the Administrator notes analyses in the PA (U.S. EPA, 2014c, section 4.4.1) indicating that a revised standard with a level of 70 ppb would be expected to require additional reductions, beyond those required by the current standard, in the short- and long-term ambient O₃ concentrations that provided the basis for statistically significant O₃ health effect associations in both the single-city and multicity epidemiologic studies evaluated. As discussed above in the response to comments, while the Administrator concludes that these analyses support a level at least as low as 70 ppb, based on a study reporting health effect associations in a location that met the current standard over the entire study period but that would have violated a revised standard with a level of 70 ppb,¹⁵¹ she further judges that they are of more limited utility for distinguishing between the appropriateness of the health protection estimated for a standard level of 70 ppb and the protection estimated for lower levels. Thus, the Administrator notes that a revised standard with a level of 70 ppb will provide additional public health protection, beyond that provided by the current standard, against the clearly adverse effects reported in

¹⁵¹ As discussed above (II.B.2.c.ii and II.B.3), the study by Mar and Koenig (2009) reported positive and statistically significant associations with respiratory emergency department visits in a location that would have met the current standard over the entire study period, but violated a standard with a level of 70 ppb. In addition, air quality analyses in the locations of two additional studies highlighted in sections II.B.2 and II.B.3 (Silverman and Ito, 2010; Strickland *et al.*, 2010) were used in the PA to inform staff conclusions on the adequacy of the current primary O₃ standard. However, they did not provide insight into the appropriateness of standard levels below 75 ppb and, therefore, these analyses were not used to inform conclusions on potential alternative standard levels lower than 75 ppb (U.S. EPA, 2014c, Chapters 3 and 4). See *Mississippi*, 744 F. 3d at 1352–53 (study appropriate for determining causation may not be probative for determining level of a revised standard).

epidemiologic studies. She judges that a standard with a level of 70 ppb strikes an appropriate balance between setting the level to require reductions in the ambient O₃ concentrations associated with statistically significant health effects in epidemiologic studies, while not being more protective than necessary in light of her considerable uncertainty in the extent to which studies clearly show O₃-attributable effects at lower ambient O₃ concentrations. This judgment is consistent with the Administrator's conclusions based on information from controlled human exposure studies, as discussed above.

With regard to epidemiology-based risk estimates, the Administrator takes note of the CASAC conclusion that "[a]lthough the estimates for short-term exposure impacts are subject to uncertainty, the data supports a conclusion that there are meaningful reductions in mean premature mortality associated with ozone levels lower than the current standard" (Frey, 2014a, p. 10). While she concludes that epidemiology-based risk analyses provide only limited support for any specific standard level, consistent with CASAC advice the Administrator judges that, compared to the current standard, a revised standard with a level of 70 ppb will result in meaningful reductions in the mortality and respiratory morbidity risk that is associated with short-or long-term ambient O₃ concentrations.

Given all of the evidence and information discussed above, the Administrator judges that a standard with a level of 70 ppb is requisite to protect public health with an adequate margin of safety, and that a level below 70 ppb would be more than "requisite" to protect the public health. In reaching this conclusion, she notes that a decision to set a lower level would place a large amount of emphasis on the potential public health importance of (1) further reducing the occurrence of O₃ exposures of concern, though the exposures about which she is most concerned are estimated to be almost eliminated with a level of 70 ppb, and lower levels would be expected to achieve virtually no additional reductions in these exposures (see Table 1, above); (2) further reducing the risk of O₃-induced lung function decrements >10 and 15%, despite having less confidence in judging the potential adversity of lung function decrements alone and the considerable overlap between risk estimates for various standard levels that make it difficult to distinguish between the risk reductions achieved; (3) further reducing ambient O₃ concentrations, relative to those in

locations of epidemiologic studies, though associations have not been reported for air quality that would have met a standard with a level of 70 ppb across all study locations and over entire study periods, and despite her consequent judgment that air quality analyses in epidemiologic study locations are not informative regarding the additional degree of public health protection that would be afforded by a standard set at a level below 70 ppb; and (4) further reducing epidemiology-based risk estimates, despite the important uncertainties in those estimates. As discussed in this section and in the responses to significant comments above, the Administrator does not agree that it is appropriate to place significant weight on these factors or to use them to support the appropriateness of standard levels below 70 ppb O₃. Compared to an O₃ standard level of 70 ppb, the Administrator concludes that the extent to which lower standard levels could result in further public health improvements becomes notably less certain.

Thus, having carefully considered the evidence, information, CASAC advice, and public comments relevant to her decision on the level of the primary O₃ standard, as discussed above and in the Response to Comments document, the Administrator is revising the level of the primary O₃ standard to 70 ppb. She is mindful that the selection of a primary O₃ standard that is requisite to protect public health with an adequate margin of safety requires judgments based on an interpretation of the scientific evidence and exposure/risk information that neither overstate nor understate the strengths and limitations of that evidence and information and the appropriate inferences to be drawn therefrom. Her decision places the greatest emphasis on the results of controlled human exposure studies and on quantitative analyses based on information from these studies, particularly analyses of O₃ exposures of concern. As in the proposal, and as discussed above, she views the results of the lung function risk assessment, analyses of O₃ air quality in locations of epidemiologic studies, and epidemiology-based quantitative health risk assessments as providing information in support of her decision to revise the current standard, but a more limited basis for selecting a particular standard level among a range of options.

In making her decision to revise the level of the primary O₃ standard to 70 ppb, the Administrator judges that a revised standard with a level of 70 ppb

strikes the appropriate balance between limiting the O₃ exposures about which she is most concerned and not going beyond what would be required to effectively limit such exposures. Specifically, the Administrator judges it appropriate to set a standard estimated to eliminate, or almost eliminate, repeated occurrences of exposures of concern for the 70 and 80 ppb benchmarks. She further judges that a lower standard level would not be appropriate given that lower levels would be expected to achieve virtually no additional reductions in repeated occurrences of exposures of concern for these benchmarks. For the 60 ppb benchmark, a level of 70 ppb is estimated to protect the vast majority of children (including children with asthma) in urban study areas from experiencing two or more exposures of concern, reflecting important reductions in such exposures compared to the current standard and indicating that the revised primary O₃ standard provides an adequate margin of safety. Given these results, including the considerable protection provided against repeated exposures of concern for the 60 ppb benchmark, the Administrator judges that a standard with a level of 70 ppb incorporates an adequate margin of safety against the occurrence of adverse O₃-induced effects.

For all of the above reasons, the Administrator concludes that a primary O₃ standard with an 8-hour averaging time; a 3-year average, fourth-high form; and a level of 70 ppb is requisite to protect public health, including the health of at-risk populations, with an adequate margin of safety. Therefore, in this final rule she is setting the level of the primary O₃ standard at 70 ppb.

D. Decision on the Primary Standard

For the reasons discussed above, and taking into account information and assessments presented in the ISA, HREA, and PA, the advice and recommendations of the CASAC Panel, and the public comments, the Administrator has decided to revise the existing 8-hour primary O₃ standard. Specifically, the Administrator is revising the level of the primary O₃ standard to 70 ppb. The revised 8-hour primary standard, with a level of 70 ppb, would be met at an ambient air monitoring site when the 3-year average of the annual fourth-highest daily maximum 8-hour average O₃ concentration is less than or equal to 70 ppb. Data handling conventions are specified in the new Appendix U that is adopted, as discussed in section V below.

At this time, EPA is also promulgating revisions to the Air Quality Index (AQI) for O₃ to be consistent with the revisions to the primary O₃ standard and the health information evaluated in this review of the standards. These revisions are discussed below in section III.

III. Communication of Public Health Information

Information on the public health implications of ambient concentrations of criteria pollutants is currently made available primarily through EPA's AQI program. The AQI has been in use since its inception in 1999 (64 FR 42530). It provides accurate, timely, and easily understandable information about daily levels of pollution. It is designed to tell individual members of the public how clean or unhealthy their air is, whether health effects might be a concern, and, if so, measures individuals can take to reduce their exposure to air pollution.¹⁵² See CAA section 127. The AQI focuses on health effects individuals may experience within a few hours or days after breathing unhealthy air. The AQI establishes a nationally uniform system of indexing pollution concentrations for O₃, CO, NO₂, PM and SO₂. The AQI converts pollutant concentrations in a community's air to a number on a scale from 0 to 500. Reported AQI values enable the public to know whether air pollution concentrations in a particular location are characterized as good (0–50), moderate (51–100), unhealthy for sensitive groups (101–150), unhealthy (151–200), very unhealthy (201–300), or

hazardous (301–500). The AQI index value of 100 typically corresponds to the level of the short-term NAAQS for each pollutant. For the 2008 O₃ NAAQS, an 8-hour average concentration of 75 ppb corresponds to an AQI value of 100. An AQI value greater than 100 means that a pollutant is in one of the unhealthy categories (*i.e.*, unhealthy for sensitive groups, unhealthy, very unhealthy, or hazardous) on a given day; an AQI value at or below 100 means that a pollutant concentration is in one of the satisfactory categories (*i.e.*, moderate or good). An additional consideration in selecting breakpoints is for each category to span at least a 15 ppb range to allow for more accurate air pollution forecasting. Decisions about the pollutant concentrations at which to set the various AQI breakpoints, that delineate the various AQI categories, draw directly from the underlying health information that supports the NAAQS review.

A. Proposed Revisions to the AQI

Recognizing the importance of revising the AQI in a timely manner to be consistent with any revisions to the NAAQS, EPA proposed conforming changes to the AQI, in connection with the Agency's proposed decision on revisions to the O₃ NAAQS. These conforming changes included setting the 100 level of the AQI at the same level as the revised primary O₃ NAAQS and also making adjustments based on health information from this NAAQS review to AQI breakpoints at the lower end of each range (*i.e.*, AQI values of 50, 150, 200 and 300). The EPA did not propose to change the level at the top of the index (*i.e.*, AQI value of 500) that typically is set equal to the Significant Harm Level (40 CFR 51.16), which would apply to state contingency plans.

The EPA proposed to revise the AQI for O₃ by setting an AQI value of 100 equal to the level of the revised O₃ standard (65–70 ppb). The EPA also proposed to revise the following breakpoints: an AQI value of 50 to within a range from 49–54 ppb; an AQI value of 150 to 85 ppb; an AQI value of 200 to 105 ppb, and an AQI value of 300 to 200 ppb. All these levels are averaged over 8 hours. The EPA proposed to set an AQI value of 50, the breakpoint between the good and moderate categories, at 15 ppb below the value of the proposed standard, *i.e.* to within a range from 49 to 54 ppb. The EPA took comment on what level within this range to select, recognizing that there is no health message for either at-risk or healthy populations in the good category. Thus, the level selected should be below the lowest concentration (*i.e.*,

60 ppb) that has been shown in controlled human exposure studies of young, healthy adults exposed to O₃ while engaged in quasi-continuous moderate exercise for 6.6 hours to cause moderate lung function decrements (*i.e.*, FEV₁ decrements ≥ 10%, which could be adverse to people with lung disease) and airway inflammation.¹⁵³ The EPA proposed to set an AQI value of 150, the breakpoint between the unhealthy for sensitive groups and unhealthy categories, at 85 ppb. At this level, controlled human exposure studies of young, healthy adults indicate that up to 25% of exposed people are likely to have moderate lung function decrements (*i.e.*, 25% have FEV₁ decrements ≥ 10%; 12% have FEV₁ decrements ≥ 15%) and up to 7% are likely to have large lung function decrements (*i.e.*, FEV₁ decrements ≥ 20%) (McDonnell *et al.*, 2012; Figure 7). Large lung function decrements would likely interfere with normal activity for many healthy people. For most people with lung disease, large lung function decrements would not only interfere with normal activity but would increase the likelihood that they would seek medical treatment (72 FR 37850, July 11, 2007). The EPA proposed to set an AQI value of 200, the breakpoint between the unhealthy and very unhealthy categories, at 105 ppb. At this level, controlled human exposure studies of young, healthy adults indicate that up to 38% of exposed people are likely to have moderate lung function decrements (*i.e.*, 38% have FEV₁ decrements ≥ 10%; 22% have FEV₁ decrements ≥ 15%) and up to 13% are likely to have large lung function decrements (*i.e.*, FEV₁ decrements ≥ 20%). The EPA proposed to set an AQI value of 300, the breakpoint between the very unhealthy and hazardous categories, at 200 ppb. At this level, controlled human exposure studies of healthy adults indicate that up to 25% of exposed individuals are likely to have large lung function decrements (*i.e.*, FEV₁ decrements ≥ 20%), which would interfere with daily activities for many of them and likely cause people with lung disease to seek medical attention.

EPA stated that the proposed breakpoints reflect an appropriate balance between reflecting the health evidence that is the basis for the proposed primary O₃ standard and providing category ranges that are large enough to be forecasted accurately, so

¹⁵² EPA issued the AQI in 1999, updating the previous Pollutant Standards Index (PSI) to send "a clear and consistent message to the public by providing nationally uniform information on air quality." The rule requires metropolitan areas of 350,000 and larger to report the AQI [and associated health effects] daily; all other AQI-related activities—including real-time ozone and particle pollution reporting, next-day air quality forecasting and action days—are voluntary and are carried out at the discretion of state, local and tribal air agencies. In the 1999 rule, we acknowledged these other programs, noting, for example, that while states primarily use the AQI "to provide general information to the public about air quality and its relationship to public health," some state, local or tribal agencies use the index to call "action days." Action days encourage additional steps, usually voluntary, that the public, business or industry could take to reduce emissions when higher levels of pollution are forecast to occur. As the 1999 rule notes, agencies may have several motivations for calling action days, including: providing health information to the public; attaining or maintaining NAAQS attainment status; meeting specific emission reduction targets; and managing or reducing traffic congestion. State, local and tribal agencies should consider whether non-voluntary emissions or activity curtailments are necessary (as opposed to a suite of voluntary measures) for days when the AQI is forecasted to be on the lower end of the moderate category.

¹⁵³ Exposures to 50 ppb have not been evaluated experimentally, but are estimated to potentially affect only a small proportion of healthy adults and with only a half to a third of the moderate to large lung function decrements observed at 60 ppb (McDonnell *et al.*, 2012; Figure 7).

that the new AQI for O₃ can be implemented more easily in the public forum for which the AQI ultimately exists. However, the EPA recognized alternative approaches to viewing the evidence and information and solicited comment on the proposed revisions to the AQI.

With respect to reporting requirements (40 CFR part 58, section 58.50), EPA proposed to revise 40 CFR part 58, section 58.50 (c) to determine the areas subject to AQI reporting requirements based on the latest available census figures, rather than the most recent decennial U.S. census.¹⁵⁴ This change is consistent with our current practice of using the latest population figures to make monitoring requirements more responsive to changes in population.

B. Comments on Proposed Revisions to the AQI

EPA received many comments on the proposed changes to the AQI. Three issues came up in the comments, including: (1) Whether the AQI should be revised at all, even if the primary standard is revised; (2) whether an AQI value of 100 should be set equal to the level of the primary standard and the other breakpoints adjusted accordingly; and, (3) whether the AQI reporting requirements should be based on the latest available census figures rather than the most recent decennial census.

With respect to the first issue, some industry commenters stated that the AQI should not be revised at all, even if the level of the primary O₃ standard is revised. In support of this position, these commenters stated that the proposed conforming changes to the AQI would lower O₃ levels in each category, and would mean that air quality that is actually improving would be reported as less healthy. According to commenters, the revised AQI would fail to capture these improvements and potentially mislead the public into thinking that air quality has degraded and that EPA and state regulators are not doing their jobs. These commenters noted that there is no requirement to revise the AQI, and that the CAA does not tie the AQI to the standards, stating that the purpose of section 319(a) of the CAA is to provide a consistent, uniform means of gauging air quality. These commenters further asserted that EPA's proposed changes run counter to that uniformity by changing the air quality significance of a given index value and category and that retention of the

¹⁵⁴ Under 40 CFR 58.50, any MSA with a population exceeding 350,000 is required to report AQI data.

current AQI breakpoints would allow continued uniform information on air quality. Commenters stated that it is important that the EPA clearly communicates that the immediate increases in moderate rated days are due to AQI breakpoint adjustment and not due to a sudden decline in air quality. One commenter estimated the increased proportion of days in the moderate category and above in 10 metropolitan areas for 2013 and also for 2025 for 4 cities from the original 10 that were estimated to attain a standard below 70 ppb, to compare with 2013. This commenter noted that the change in the proposed AQI breakpoint between "good" and "moderate" would result in a larger number of days that did not meet the "good" criteria. They went further to claim that the change in breakpoints would result in fewer "good" days in the year 2025 (using the new breakpoint) than occurred in 2013 (using the old breakpoints) despite substantial improvement in air quality over that time period.

On the other hand, state and local agencies and their organizations, environmental and medical groups, and members of the public overwhelmingly supported revising the AQI when the level of the standard is revised. Even state agencies that did not support revising the standard, expressed support for revising the AQI at the same time as the standard, if the standard is revised.

Recognizing the importance of the AQI as a communication tool that allows members of the public to take exposure reduction measures when air quality poses health risks, the EPA agrees with these comments about revising the AQI at the same time as the primary standard. The EPA agrees with state and local agency commenters that its historical approach of setting an AQI value of 100 equal to the level of the revised 8-hour primary O₃ standard is appropriate, both from a public health and a communication perspective.

EPA disagrees with commenters who stated that the AQI should not be linked to the primary standards. As noted in the August 4, 1999, rulemaking (64 FR 149, 42531) that established the current AQI, the EPA established the nationally uniform air quality index, called the Pollutant Standards Index (PSI), in 1976 to meet the needs of state and local agencies with the following advantages: It sends a clear and consistent message to the public by providing nationally uniform information on air quality; it is keyed as appropriate to the NAAQS and the Significant Harm Level which have a scientific basis relating air quality and public health; it is simple and easily understood by the public; it provides a

framework for reflecting changes to the NAAQS; and it can be forecasted to provide advance information on air quality. Both the PSI and AQI have historically been normalized across pollutants by defining an index value of 100 as the numerical level of the short-term (*i.e.*, averaging time of 24-hours or less) primary NAAQS for each pollutant. Moreover, this approach does not mislead the public. Since the establishment of the AQI, the EPA and state and local air agencies and organizations have developed experience in educating the public about changes in the standards and, concurrently, related changes to AQI breakpoints and advisories. When the standards change, EPA and state and local agencies have tried to help the public understand that air quality is not getting worse, it's that the health evidence underlying the standards and the AQI has changed. EPA's Air Quality System (AQS), the primary repository for air quality monitoring data, is also adjusted to reflect the revised breakpoints. Specifically, all historical AQI values in AQS are recomputed with the revised breakpoints, so that all data queries and reports downstream of AQS will show appropriate trends in AQI values over time.¹⁵⁵

In general, commenters who supported revising the AQI when the standard is revised, also supported setting an AQI value of 100 equal to the level of the 8-hour primary O₃ standard. The EPA agrees with these commenters. With respect to an AQI value of 100, the EPA is taking final action to set an AQI value of 100 equal to the level of the 8-hour primary standard at 70 ppb O₃.

With respect to proposed changes to other AQI breakpoints, some state and local agency commenters expressed general support for all the changes in O₃ breakpoints (in Table 2 of Appendix G). In addition, we received a few comments specifically about the breakpoint between the good and moderate categories. One state expressed the view that forecasting the AQI for O₃ is not an exact science, so it is important to provide a range large enough to reasonably predict O₃

¹⁵⁵ Although we do not contest the assertion that the new AQI breakpoints will lead to fewer green days in the near future, we do not agree that commenters' analysis sufficiently demonstrates that there would be fewer green days in 2025 than in 2013. In their analysis, they compared observed 2013 data with modeled 2025 data without doing any model performance evaluation for AQI categories or comparison of current year modeled and observed data. The current year observations are not directly comparable to the future-year modeling data without some such evaluation and, as such, we cannot support their quantitative conclusions.

concentrations for the following day (\geq 20 ppb). Although not supporting revision of the standard, this state recommended that if the primary standard was revised to 70 ppb, the lower end of moderate category should be set at 50 ppb to allow for a 20 ppb spread in that category. Several commenters recommending a breakpoint between the good and moderate categories of no higher than 50 ppb stated that this breakpoint should be set on health information, pointing to epidemiologic data and the World Health organization guidelines. The Agency agrees that AQI breakpoints should take into consideration health information when possible, and also that it is important for AQI categories to span ranges large enough to support accurate forecasting. The EPA is setting the breakpoint at the lower end of the moderate category at 55 ppb, which is 15 ppb below the level of the standard of 70 ppb. This is consistent with past practice of making a proportional adjustment to this AQI breakpoint, relative to an AQI value of 100 (*i.e.*, 70 ppb), and also retains the current practice of providing a 15 ppb range in the moderate category to allow for accurate forecasting. This level is below the lowest concentration (*i.e.*, 60 ppb) that has been shown in controlled human exposure studies of healthy adults to cause moderate lung function decrements (*i.e.*, FEV₁ decrements \geq 10%, which could be adverse to people with lung disease), large lung function decrements (*i.e.*, FEV₁ decrements \geq 20%) in a small proportion of people, and airway inflammation, notwithstanding the Administrator's judgment that there is uncertainty in the adversity of the effects shown to occur at 60 ppb.

We received fewer comments on proposed changes to the AQI values of 150, 200 and 300. Again, some state and local agency commenters expressed general support for proposed changes to the AQI. Some states specifically supported these breakpoints. However, a commenter suggested setting an AQI value at the lower end of the unhealthy category, at a level much lower than 85 ppb, since they state that it is a key threshold that is often used in air quality action day programs as a trigger to encourage specific behavior modifications or reduce emissions of O₃ precursors (*e.g.*, by taking public transportation to work). This commenter stated that setting the breakpoint at 85 ppb would, in the Agency's own rationale, not require the triggering of these pollution reduction measures until air quality threatened to impact

25% of people exposed. We disagree with this commenter because EPA does not have any requirements for voluntary programs. State and local air agencies have discretion to set the trigger for voluntary action programs at whatever level they choose, and they are currently set at different levels, not just at the unhealthy breakpoint specified in the comment. For example, Houston, Galveston and Brazoria TX metropolitan area calls ozone action days when air quality reaches the unhealthy for sensitive groups category. For more information about action days programs across the U.S. see the AirNow Web site (www.airnow.gov) and click on the link to AirNow Action Days. The unhealthy category represents air quality where there are general population-level effects. We believe that setting the breakpoint between the unhealthy for sensitive groups and unhealthy categories, at 85 ppb where, as discussed in section IIIA above, controlled human exposure studies of young, healthy adults exposed to O₃ while engaged in quasi-continuous moderate exercise for 6.6 hours indicate that up to 25% of exposed people are likely to have moderate lung function decrements and up to 7% are likely to have large lung function decrements (McDonnell *et al.*, 2012; Figure 7) is appropriate. A smaller proportion of inactive or less active individuals would be expected to experience lung function decrements at 85 ppb. Moreover, a breakpoint at 85 ppb allows for category ranges large enough for accurate forecasting. Accordingly, the EPA is adopting the proposed revisions to the AQI values of 150, 200 and 300.

As noted earlier, the EPA proposed to revise 40 CFR part 58, section 58.50(c) to determine the areas subject to AQI reporting requirements based on the latest available census figures, rather than the most recent decennial U.S. census.

A total of five state air monitoring agencies provided comments on this proposed change. Four agencies supported the proposal. One state commenter did not support the proposal, noting that the change would unnecessarily complicate AQI reporting and possibly increase reporting burdens in an unpredictable manner.

The EPA notes that the majority of monitoring network minimum requirements listed in Appendix D to Part 58 include a reference to "latest available census figures." Minimum network requirements for O₃, PM_{2.5}, SO₂, and NO₂ all include this language in the regulatory text and monitoring agencies have successfully adopted these processes into their planning

activities and the subsequent revision of their annual monitoring network plans which are posted for public review. Annual population estimates are easily obtainable from the U.S. Census Bureau and the EPA does not believe the burden in tracking these annual estimates is excessive or complicated.¹⁵⁶ Although the changes in year to year estimates are typically modest, there are MSAs that are approaching (or have recently exceeded) the 350,000 population AQI reporting limit and there is great value in having the AQI reported for these areas when the population threshold is exceeded versus waiting potentially up to 10 years for a revision to the decennial census. Accordingly, the EPA is finalizing the proposed revision to 40 CFR part 58, section 58.50(c) to require the AQI reporting requirements to be based on the latest available census figures.

One state requested additional guidance on the frequency of updating the AQI reporting threshold, and recommended linking the AQI reporting requirement evaluation with the annual air monitoring network plan requirements, and recommended requiring AQI reporting to begin no later than January 1 of the following year. The EPA notes that the census bureau estimates appear to be released around July 1 of each year which would not provide sufficient time for monitoring agencies to incorporate AQI reporting in their annual plans for that year, which are also due by July 1 each year. EPA believes that it should be unnecessary for monitoring agencies to wait until the implementation of the following year's annual plan (*i.e.*, approximately 18 months later) to begin AQI reporting. Accordingly, EPA is not at this time including a specific deadline for commencement of AQI reporting for newly-subject areas in 40 CFR part 58, but will work with agencies to implement additional AQI reporting as needed to ensure that information is being disseminated in a timely fashion.

C. Final Revisions to the AQI

For the reasons discussed above, the EPA is revising the AQI for O₃ by setting an AQI value of 100 equal to 70 ppb, 8-hour average, the level of the revised primary O₃ standard. The EPA is also revising the following breakpoints: An AQI value of 50 is set at 54 ppb; an AQI value of 150 is set at 85 ppb; an AQI value of 200 is set at 105 ppb; and an AQI value of 300 is set at 200 ppb. All of these levels are averaged over 8 hours. The revisions to all of the

¹⁵⁶ <http://www.census.gov/popest/data/metro/totals/2014/CBSA-EST2014-alldata.html>.

breakpoints are based on estimated health outcomes at relevant ambient concentrations and to allow for each category to span at least a 15–20 ppb category range to allow for more accurate air pollution forecasting. The EPA believes that the revised breakpoints provide a balance between adjustments to reflect the health information supporting the revised O₃ standard and providing category ranges that are large enough to be forecasted accurately, so that the AQI can be implemented more easily in the public forum for which the AQI ultimately exists. With respect to AQI reporting requirements (40 CFR part 58, section 58.50), the EPA is revising 40 CFR part 58, section 58.50(c) to make the AQI reporting requirements based on the latest available census figures, rather than the most recent decennial U.S. census. This change is consistent with our current practice of using the latest population figures to make monitoring requirements more responsive to changes in population.

IV. Rationale for Decision on the Secondary Standard

A. Introduction

This section (IV) presents the rationale for the Administrator's decisions regarding the need to revise the current secondary standard for O₃, and the appropriate revision. Based on her consideration of the full body of welfare effects evidence and related analyses, including the evidence of effects associated with cumulative seasonal exposures of the magnitudes allowed by the current standard, the Administrator has concluded that the current secondary standard for O₃ does not provide the requisite protection of public welfare from known or anticipated adverse effects. She has decided to revise the level of the current secondary standard to 0.070 ppm, in conjunction with retaining the current indicator, averaging time and form.

The Administrator has made this decision based on judgments regarding the currently available welfare effects evidence, the appropriate degree of public welfare protection for the revised standard, and currently available air quality information on seasonal cumulative exposures that may be allowed by such a standard. In so doing, she has focused on O₃ effects on tree seedling growth as a proxy for the full array of vegetation-related effects of O₃, ranging from effects on sensitive species to broader ecosystem-level effects. Using this proxy in judging effects to public welfare, the Administrator has concluded that the requisite protection

from adverse effects to public welfare will be provided by a standard that limits cumulative seasonal exposures to 17 ppm-hrs or lower, in terms of a 3-year W126 index, in nearly all instances, and she has also concluded that such control of cumulative seasonal exposures may be achieved by revising the level of the current standard to 70 ppb. Based on all of these considerations, the Administrator has decided that a secondary standard with a level of 0.070 ppm, and the current form and averaging time, will provide the requisite protection of public welfare from known or anticipated adverse effects.

As discussed more fully below, this decision is based on a thorough review, in the ISA, of the latest scientific information on O₃-induced environmental effects. This decision also takes into account (1) staff assessments in the PA of the most policy-relevant information in the ISA regarding evidence of adverse effects of O₃ to vegetation and ecosystems, information on biologically-relevant exposure metrics, WREA analyses of air quality, exposure, and ecological risks and associated ecosystem services, and staff analyses of relationships between levels of a W126-based metric and a metric based on the form and averaging time of the current standard summarized in the PA and in the proposal notice; (2) CASAC advice and recommendations; and (3) public comments received during the development of these documents, either in connection with CASAC meetings or separately, and on the proposal notice.

This decision draws on the ISA's integrative synthesis of the entire body of evidence, generally published through July 2011, on environmental effects associated with the presence of O₃ and related photochemical oxidants in the ambient air (U.S. EPA, 2013, ISA chapters 9–10), and includes more than four hundred new studies that build on the extensive evidence base from the last review. In addition to reviewing the most recent scientific information as required by the CAA, this rulemaking incorporates the EPA's response to the judicial remand of the 2008 secondary O₃ standard in *State of Mississippi v. EPA*, 744 F. 3d 1334 (D.C. Cir. 2013) and, in accordance with the court's decision in that case, fully explains the Administrator's conclusions as to the level of air quality that provides the requisite protection of public welfare from known or anticipated adverse effects. In drawing conclusions on the secondary standard, the decision described in this rulemaking is a public welfare policy judgment made by the

Administrator. The Administrator's decision draws upon the available scientific evidence for O₃-attributable welfare effects and on analyses of exposures and public welfare risks based on impacts to vegetation, ecosystems and their associated services, as well as judgments about the appropriate weight to place on the range of uncertainties inherent in the evidence and analyses. As described in sections IV.B.3 and IV.C.3 below, such judgments in the context of this review include judgments on the weight to place on the evidence of specific vegetation-related effects estimated to result across a range of cumulative seasonal concentration-weighted O₃ exposures; on the weight to give associated uncertainties, including those related to the variability in occurrence of such effects in areas of the U.S., especially areas of particular public welfare significance; and on the extent to which such effects in such areas may be considered adverse to public welfare.

Information related to vegetation and ecosystem effects, biologically relevant exposure indices, and vegetation exposure and risk assessments were summarized in sections IV.A through IV.C of the proposal (79 FR at 75314–75329), respectively, and key observations from the proposal are briefly outlined in sections IV.A.1 to IV.A.3 below. Subsequent sections of this preamble provide a more complete discussion of the Administrator's rationale, in light of key issues raised in public comments, for concluding that the current standard is not requisite to protect public welfare from known or anticipated adverse effects (section IV.B), and that it is appropriate to revise the current secondary standard to provide additional public welfare protection by revising the level while retaining the current indicator, form and averaging time (section IV.C). A summary of the final decisions on revisions to the secondary standard is presented in section IV.D.

1. Overview of Welfare Effects Evidence a. Nature of Effects

In the more than fifty years that have followed identification of O₃'s phytotoxic effects, extensive research has been conducted both in and outside of the U.S. to examine the impacts of O₃ on plants and their associated ecosystems (U.S. EPA, 1978, 1986, 1996a, 2006a, 2013). As was established in prior reviews, O₃ can interfere with carbon gain (photosynthesis) and allocation of carbon within the plant, making fewer carbohydrates available

for plant growth, reproduction, and/or yield. For seed-bearing plants, these reproductive effects will culminate in reduced seed production or yield (U.S. EPA, 1996a, pp. 5–28 and 5–29). Recent studies, assessed in the ISA, together with this longstanding and well-established literature on O₃-related vegetation effects, further contribute to the coherence and consistency of the vegetation effects evidence (U.S. EPA, 2013, chapter 9).

The strongest evidence for effects from O₃ exposure on vegetation is from controlled exposure studies, which “have clearly shown that exposure to O₃ is causally linked to visible foliar injury, decreased photosynthesis, changes in reproduction, and decreased growth” in many species of vegetation (U.S. EPA, 2013, p. 1–15). Such effects at the plant scale can also be linked to an array of effects at larger spatial scales, with the currently available evidence indicating that “ambient O₃ exposures can affect ecosystem productivity, crop yield, water cycling, and ecosystem community composition” (U.S. EPA, 2013, p. 1–15; Chapter 9, section 9.4). The current body of O₃ welfare effects evidence confirms and strengthens support for the conclusions reached in the last review on the nature of O₃-induced welfare effects and is summarized in the ISA as follows (U.S. EPA, 2013, p. 1–8).

The welfare effects of O₃ can be observed across spatial scales, starting at the subcellular and cellular level, then the whole plant and finally, ecosystem-level processes. Ozone effects at small spatial scales, such as the leaf of an individual plant, can result in effects along a continuum of larger spatial scales. These effects include altered rates of leaf gas exchange, growth, and reproduction at the individual plant level, and can result in broad changes in ecosystems, such as productivity, carbon storage, water cycling, nutrient cycling, and community composition.

Based on assessment of this extensive body of science, the EPA has determined that, with respect to vegetation and ecosystems, a causal relationship exists between exposure to O₃ in ambient air and visible foliar injury effects on vegetation, reduced vegetation growth, reduced productivity in terrestrial ecosystems, reduced yield and quality of agricultural crops and alteration of below-ground biogeochemical cycles (U.S. EPA, 2013, Table 1–2). In consideration of the evidence of O₃ exposure and alterations in stomatal performance, “which may affect plant and stand transpiration and therefore possibly affecting hydrological cycling,” the ISA concludes that “[a]lthough the direction of the response

differed among studies,” the evidence is sufficient to conclude a likely causal relationship between O₃ exposure and the alteration of ecosystem water cycling (U.S. EPA, 2013, section 2.6.3). The evidence is also sufficient to conclude a likely causal relationship between O₃ exposure and the alteration of community composition of some terrestrial ecosystems (U.S. EPA, 2013, section 2.6.5). Related to the effects on vegetation growth, productivity and, to some extent, below-ground biogeochemical cycles, the EPA has additionally determined that a likely causal relationship exists between exposures to O₃ in ambient air and reduced carbon sequestration (also termed carbon storage) in terrestrial ecosystems (U.S. EPA, 2013, p. 1–10 and section 2.6.2). Modeling studies available in this review consistently found negative impacts of O₃ on carbon sequestration, although the severity of impact was influenced by “multiple interactions of biological and environmental factors” (U.S. EPA, 2013, p. 2–39).

Ozone in the troposphere is also a major greenhouse gas and radiative forcing agent,¹⁵⁷ with the ISA formally concluding that “the evidence supports a causal relationship between changes in tropospheric O₃ concentrations and radiative forcing” (U.S. EPA, 2013, p. 1–13 and section 2.7.1). While tropospheric O₃ has been ranked third in importance after carbon dioxide and methane, there are “large uncertainties in the magnitude of the radiative forcing estimate attributed to tropospheric O₃, making the impact of tropospheric O₃ on climate more uncertain than the effect of the longer-lived greenhouse gases” (U.S. EPA, 2013, p. 2–47). The ISA notes that “[e]ven with these uncertainties, global climate models indicate that tropospheric O₃ has contributed to observed changes in global mean and regional surface temperatures” and concludes that “[a]s a result of such evidence presented in climate modeling studies, there is likely to be a causal relationship between changes in tropospheric O₃ concentrations and effects on climate” (U.S. EPA, 2013, p. 2–47).¹⁵⁸ The ISA additionally states that “[i]mportant

¹⁵⁷ As described in the ISA, “[r]adiative forcing by a greenhouse gas or aerosol is a metric used to quantify the change in balance between radiation coming into and going out of the atmosphere caused by the presence of that substance” (U.S. EPA, 2013, p. 1–13).

¹⁵⁸ Climate responses, including increased surface temperature, have downstream climate-related ecosystem effects (U.S. EPA, 2013, p. 10–7). As noted in section I.D above, such effects may include an increase in the area burned by wildfires, which, in turn, are sources of O₃ precursor emissions.

uncertainties remain regarding the effect of tropospheric O₃ on future climate change” (U.S. EPA, 2013, p. 10–31).

b. Vegetation Effects

Given the strong evidence base and the findings of causal or likely causal relationships with O₃ in ambient air, including the quantitative assessments of relationships between O₃ exposure and occurrence and magnitude of effects, this review has given primary consideration to three main kinds of vegetation effects, some of which contribute to effects at scales beyond the plant level, such as at the ecosystem level and on ecosystem services. The three kinds of effects are addressed below in the following order: 1) Visible foliar injury, 2) impacts on tree growth, productivity and carbon storage, and 3) crop yield loss.

Visible foliar injury resulting from exposure to O₃ has been well characterized and documented over several decades of research on many tree, shrub, herbaceous, and crop species (U.S. EPA, 2013, p. 1–10; U.S. EPA, 2006a, 1996a, 1986, 1978). Ozone-induced visible foliar injury symptoms on certain plant species, such as black cherry, yellow-poplar and common milkweed, are considered diagnostic of exposure to O₃ based on the consistent association established with experimental evidence (U.S. EPA, 2013, p. 1–10). The evidence has found that visible foliar injury occurs only when sensitive plants are exposed to elevated O₃ concentrations in a predisposing environment; a major modifying factor is the amount of available soil moisture during the year (U.S. EPA, 2013, section 9.4.2).

The significance of O₃ injury at the leaf and whole plant levels depends on an array of factors, and therefore, it is difficult to quantitatively relate visible foliar injury symptoms to vegetation effects such as individual tree growth, or effects at population or ecosystem levels (U.S. EPA, 2013, p. 9–39). The ISA notes that visible foliar injury “is not always a reliable indicator of other negative effects on vegetation” (U.S. EPA, 2013, p. 9–39). Factors that influence the significance to the leaf and whole plant include the amount of total leaf area affected, age of plant, size, developmental stage, and degree of functional redundancy among the existing leaf area (U.S. EPA, 2013, section 9.4.2). Although there remains a lack of robust exposure-response functions that would allow prediction of visible foliar injury severity and incidence under varying air quality and environmental conditions, “[e]xperimental evidence has clearly

established a consistent association of visible injury with O₃ exposure, with greater exposure often resulting in greater and more prevalent injury” (U.S. EPA, 2013, section 9.4.2, p. 9–41).

By far the most extensive field-based dataset of visible foliar injury incidence is that obtained by the U.S. Forest Service Forest Health Monitoring/Forest Inventory and Analysis (USFS FHM/FIA) biomonitoring network program (U.S. EPA, 2013, section 9.4.2.1; Smith, 2012; Coulston *et al.*, 2007). A recently published trend analysis of data from the sites located in 24 states of the northeast and north central U.S. for the 16-year period from 1994 through 2009 (Smith, 2012) describes evidence of visible foliar injury occurrence in the field as well as some insight into the influence of changes in air quality and soil moisture on visible foliar injury and the difficulty inherent in predicting foliar injury response under different air quality and soil moisture scenarios (Smith, 2012; U.S. EPA, 2013, section 9.4.2.1). Study results showed that incidence and severity of foliar injury were dependent on local site conditions for soil moisture availability and O₃ exposure (U.S. EPA, 2013, p. 9–41). Although the study indicated that moderate O₃ exposures continued to cause visible foliar injury at sites throughout the study area, there was an overall declining trend in the incidence of visible foliar injury as peak O₃ concentrations declined (U.S. EPA, 2013, p. 9–40).

Ozone has been shown to affect a number of important U.S. tree species with respect to growth, productivity, and carbon storage. Ambient O₃ concentrations have long been known to cause decreases in photosynthetic rates and plant growth. As discussed in the ISA, research published since the 2006 AQCD substantiates prior conclusions regarding O₃-related effects on forest tree growth, productivity and carbon storage, and further strengthens the support for those conclusions. A variety of factors in natural environments can either mitigate or exacerbate predicted O₃-plant interactions and are recognized sources of uncertainty and variability. Such factors include multiple genetically influenced determinants of O₃ sensitivity, changing sensitivity to O₃ across vegetative growth stages, co-occurring stressors and/or modifying environmental factors (U.S. EPA, 2013, section 9.4.8). In considering of the available evidence, the ISA states, “previous O₃ AQCDs concluded that there is strong evidence that exposure to O₃ decreases photosynthesis and growth in numerous plant species” and that “[s]tudies published since the 2008

review support those conclusions” (U.S. EPA, 2013, p. 9–42). The available studies come from a variety of different study types that cover an array of different species, effects endpoints, levels of biological organization and exposure methods and durations. The O₃-induced effects at the scale of the whole plant may translate to the ecosystem scale, with changes in productivity and carbon storage. As stated in the ISA, “[s]tudies conducted during the past four decades have demonstrated unequivocally that O₃ alters biomass allocation and plant reproduction” (U.S. EPA, 2013, p. 1–10).

The strong evidence of O₃ impacts on trees includes robust exposure-response (E–R) functions for reduced growth, termed relative biomass loss (RBL),¹⁵⁹ in seedlings of 11 species. These functions were developed under the National Health and Environmental Effects Research Laboratory–Western Ecology Division program, a series of experiments that used open top chambers (OTCs) to investigate seedling growth response for a single growing season under a variety of O₃ exposures (ranging from near background to well above current ambient concentrations) and growing conditions (U.S. EPA, 2013, section 9.6.2; Lee and Hogsett, 1996). The evidence from these studies shows that there is a wide range in sensitivity across the studied species in the seedling growth stage over the course of a single growing season, with some species being extremely sensitive and others being very insensitive over the range of cumulative O₃ exposures studied (U.S. EPA, 2014c, Figure 5–1). At the other end of the organizational spectrum, field-based studies of species growing in natural stands have compared observed plant responses across a number of different sites and/or years when exposed to varying ambient O₃ exposure conditions. For example, a study conducted in forest stands in the southern Appalachian Mountains during a period when O₃ concentrations exceeded the current standard found that the cumulative effects of O₃ decreased seasonal stem growth (measured as a change in circumference) by 30–50 percent for most of the examined tree species (*i.e.*, tulip poplar, black cherry, red maple, sugar maple) in a high-O₃ year in comparison to a low-O₃ year (U.S. EPA, 2013, section 9.4.3.1; McLaughlin *et al.*, 2007a). The study also reported that

¹⁵⁹These functions for RBL estimate reduction in a year’s growth as a percentage of that expected in the absence of O₃ (U.S. EPA, 2013, section 9.6.2; U.S. EPA, 2014b, section 6.2).

high ambient O₃ concentrations can increase whole-tree water use and in turn reduce late-season streamflow (McLaughlin *et al.*, 2007b; U.S. EPA, 2013, p. 9–43).

The magnitude of O₃ impact on ecosystem productivity and on forest composition can vary among plant communities based on several factors, including the type of stand or community in which the sensitive species occurs (*e.g.*, single species *versus* mixed canopy), the role or position of the species in the stand (*e.g.*, dominant, sub-dominant, canopy, understory), and the sensitivity of co-occurring species and environmental factors (*e.g.*, drought and other factors). For example, recent studies found O₃ to have little impact on white fir, but to greatly reduce growth of ponderosa pine in southern California locations, with associated reductions in ponderosa pine abundance in the community, and to cause decreased net primary production of most forest types in the mid-Atlantic region, with only small impacts on spruce-fir forest (U.S. EPA, 2013, section 9.4.3.4).

There is previously and newly available evidence of the potential for O₃ to alter biomass allocation and plant reproduction in seasons subsequent to exposure (U.S. EPA, 2013, section 9.4.3). For example, several studies published since the 2006 AQCD further demonstrate that O₃ can alter the timing of flowering and the number of flowers, fruits and seeds in herbaceous and woody plant species (U.S. EPA, 2013, section 9.4.3.3). Further, limited evidence in previous reviews reported that vegetation effects from a single year of exposure to elevated O₃ could be observed in the following year. For example, growth affected by a reduction in carbohydrate storage in one year may result in the limitation of growth in the following year. Such “carry-over” effects have been documented in the growth of some tree seedlings and in roots (U.S. EPA, 2013, section 9.4.8; Andersen *et al.*, 1997). In the current review, additional field-based evidence expands the EPA’s understanding of the consequences of single and multi-year O₃ exposures in subsequent years.

A number of studies were conducted at a planted forest at the Aspen free-air carbon-dioxide and ozone enrichment (FACE) experiment site in Wisconsin. These studies, which occurred in a field setting (more similar to natural forest stands than OTC studies), observed tree growth responses when grown in single or two species stands within 30-m diameter rings and exposed over a period of ten years to existing ambient conditions and elevated O₃

concentrations. Some studies indicate the potential for carry-over effects, such as those showing that the effects of O₃ on birch seeds (reduced weight, germination, and starch levels) could lead to a negative impact on species regeneration in subsequent years, and that the O₃-attributable effect of reduced aspen bud size might have been related to the observed delay in spring leaf development. These effects suggest that elevated O₃ exposures have the potential to alter carbon metabolism of overwintering buds, which may have subsequent effects in the following year (Darbah, *et al.*, 2008, 2007; Riikonen *et al.*, 2008; U.S. EPA, 2013, section 9.4.3). Other studies found that, in addition to affecting tree heights, diameters, and main stem volumes in the aspen community, elevated O₃ over a 7-year study period was reported to increase the rate of conversion from a mixed aspen-birch community to a community dominated by the more tolerant birch, leading the authors to conclude that elevated O₃ may alter intra- and inter-species competition within a forest stand (U.S. EPA, 2013, section 9.4.3; Kubiske *et al.*, 2006; Kubiske *et al.*, 2007). These studies confirm earlier FACE results of aspen growth reductions from exposure to elevated O₃ during the first seven years of stand growth and of cumulative biomass impacts associated with changes in annual production in studied tree communities (U.S. EPA, 2013, section 9.4.3; King *et al.*, 2005).

Robust and well-established E-R functions for RBL are available for 11 tree species: black cherry, Douglas fir, loblolly pine, ponderosa pine, quaking aspen, red alder, red maple, sugar maple, tulip poplar, Virginia pine, and white pine (U.S. EPA, 2013; U.S. EPA, 2014c). While these 11 species represent only a small fraction (0.8 percent) of the total number of native tree species in the contiguous U.S. (1,497), this small subset includes eastern and western species, deciduous and coniferous species, and species that grow in a variety of ecosystems and represent a range of tolerance to O₃ (U.S. EPA, 2013, section 9.6.2; U.S. EPA, 2014b, section 6.2, Figure 6-2, Table 6-1). Supporting the E-R functions for each of these species are studies in OTCs, with most species studied multiple times under a wide range of exposure and/or growing conditions, with separate E-R functions developed for each combination of species, exposure condition and growing condition scenario (U.S. EPA, 2013, section 9.6.1). Based on these separate E-R functions, species-specific composite E-R functions have been

developed and successfully used to predict the biomass loss response from tree seedling species over a range of cumulative exposure conditions (U.S. EPA, 2013, section 9.6.2). These 11 composite functions, as well as the E-R function for eastern cottonwood (derived from a field study in which O₃ and climate conditions were not controlled),¹⁶⁰ are described in the ISA and graphed in the WREA to illustrate the predicted responses of these species over a wide range of cumulative exposures (U.S. EPA, 2014b, section 6.2, Table 6-1 and Figure 6-2; U.S. EPA, 2013, section 9.6.2). For some of these species, the E-R function is based on a single study (*e.g.*, red maple), while for other species there were as many as 11 studies available (*e.g.*, ponderosa pine). In total, the E-R functions developed for these 12 species (the 11 with robust composite E-R functions plus eastern cottonwood) reflect 52 tree seedling studies. A stochastic analysis in the WREA, summarized in section IV.C of the proposal, indicates the potential for within-species variability in these relationships for each species. Consideration of biomass loss estimates in the PA and in discussions below, however, is based on conventional methods and focuses on estimates for the 11 species for which the robust datasets from OTC experiments are available, in consideration of CASAC advice.

The “detrimental effect of O₃ on crop production has been recognized since the 1960s” (U.S. EPA, 2013, p. 1-10, section 9.4.4). On the whole, the newly available evidence supports and strengthens previous conclusions that exposure to O₃ reduces growth and yield of crops. The ISA describes average crop yield loss reported across a number of recently published meta-analyses and identifies several new exposure studies that support prior findings for a variety of crops of decreased yield and biomass with increased O₃ exposure (U.S. EPA, 2013, section 9.4.4.1, Table 9-17). Studies have also “linked increasing O₃ concentration to decreased photosynthetic rates and accelerated aging in leaves, which are related to

¹⁶⁰The CASAC cautioned the EPA against placing too much emphasis on the eastern cottonwood data. In comments on the draft PA, the CASAC stated that the eastern cottonwood response data from a single study “receive too much emphasis,” explaining that these “results are from a gradient study that did not control for ozone and climatic conditions and show extreme sensitivity to ozone compared to other studies” and that “[a]lthough they are important results, they are not as strong as those from other experiments that developed E-R functions based on controlled ozone exposure” (Frey, 2014c, p. 10).

yield” and described effects of O₃ on crop quality, such as nutritive quality of grasses, macro- and micronutrient concentrations in fruits and vegetable crops and cotton fiber quality (U.S. EPA, 2013, p. 1-10, section 9.4.4). The findings of the newly available studies do not change the basic understanding of O₃-related crop yield loss since the last review and little additional information is available in this review on factors that influence associations between O₃ levels and crop yield loss (U.S. EPA, 2013, section 9.4.4). However, the evidence available in this review continues to support the conclusion that O₃ in ambient air can reduce the yield of major commodity crops in the U.S. Further, the recent evidence increases our confidence in the use of crop E-R functions based on OTC experiments to characterize the quantitative relationship between ambient O₃ concentrations and yield loss (U.S. EPA, 2013, section 9.4.4).

The new evidence has strengthened support for previously established E-R functions for 10 crops (barley, field corn, cotton, kidney bean, lettuce, peanut, potato, grain sorghum, soybean and winter wheat), reducing two important areas of uncertainty, especially for soybean, as summarized in more detail in section IV.A of the proposal. The established E-R functions for relative yield loss (RYL)¹⁶¹ were developed from OTC-type experiments from the National Crop Loss Assessment Network (NCLAN) (U.S. EPA, 2013, section 9.6.3; U.S. EPA, 2014b, section 6.2; U.S. EPA, 2014c, Figure 5-4 and section 6.3). With regard to the first area of uncertainty reduced, evaluations in the ISA found that yield loss in soybean from O₃ exposure at the SoyFACE (Soybean Free Air Concentration Enrichment) field experiment was reliably predicted by soybean E-R functions developed from NCLAN data (U.S. EPA, 2013, section 9.6.3.1),¹⁶² demonstrating a robustness of the NCLAN-based E-R functions for predicting relative yield loss from O₃ exposure. A second area of uncertainty that was reduced is that regarding the

¹⁶¹These functions for RYL estimate reduction in a year's growth as a percentage of that expected in the absence of O₃ (U.S. EPA, 2013, section 9.6.2; U.S. EPA, 2014b, section 6.2).

¹⁶²The NCLAN program, which was undertaken in the early to mid-1980s, assessed multiple U.S. crops, locations, and O₃ exposure levels, using consistent methods, to provide the largest, most uniform database on the effects of O₃ on agricultural crop yields (U.S. EPA 1996a; U.S. EPA, 2006a; U.S. EPA, 2013, sections 9.2, 9.4, and 9.6, Frey, 2014c, p. 9). The SoyFACE experiment was a chamberless (or free-air) field-based exposure study conducted in Illinois from 2001-2009 (U.S. EPA, 2013, section 9.2.4).

application of the NCLAN E-R functions to more recent cultivars currently growing in the field. Recent studies, especially those focused on soybean, provide little evidence that crops are becoming more tolerant of O₃ (U.S. EPA, 2006a; U.S. EPA, 2013, sections 9.6.3.1 and 9.6.3.4 and p. 9–59). The ISA comparisons of NCLAN and SoyFACE data referenced above also “confirm that the response of soybean yield to O₃ exposure has not changed in current cultivars” (U.S. EPA, 2013, p. 9–59; section 9.6.3.1). Additionally, a recent assessment of the relationship between soybean yield loss and O₃ in ambient air over the contiguous area of Illinois, Iowa, and Indiana found a relationship that correlates well with previous results from FACE- and OTC-type experiments (U.S. EPA, 2013, section 9.4.4.1).

c. Biologically Relevant Exposure Metric

In assessing biologically based indices of exposure pertinent to O₃ effects on vegetation, the ISA states the following (U.S. EPA, 2013, p. 2–44).

The main conclusions from the 1996 and 2006 O₃ AQCDs [Air Quality Criteria Documents] regarding indices based on ambient exposure remain valid. These key conclusions can be restated as follows: ozone effects in plants are cumulative; higher O₃ concentrations appear to be more important than lower concentrations in eliciting a response; plant sensitivity to O₃ varies with time of day and plant development stage; [and] quantifying exposure with indices that cumulate hourly O₃ concentrations and preferentially weight the higher concentrations improves the explanatory power of exposure/response models for growth and yield, over using indices based on mean and peak exposure values.

The long-standing body of available evidence upon which these conclusions are based includes a wealth of information on aspects of O₃ exposure that are important in influencing plant response (U.S. EPA, 1996a; U.S. EPA, 2006a; U.S. EPA, 2013). Specifically, a variety of “factors with known or suspected bearing on the exposure-response relationship, including concentration, time of day, respite time, frequency of peak occurrence, plant phenology, predisposition, etc.,” have been identified (U.S. EPA, 2013, section 9.5.2). In addition, the importance of the duration of the exposure and the relatively greater importance of higher concentrations over lower concentrations in determining plant response to O₃ have been consistently well documented (U.S. EPA, 2013, section 9.5.3). Based on improved understanding of the biological basis for plant response to O₃ exposure, a large number of “mathematical approaches

for summarizing ambient air quality information in biologically meaningful forms for O₃ vegetation effects assessment purposes” have been developed (U.S. EPA, 2013, section 9.5.3), including those that cumulate exposures over some specified period while weighting higher concentrations more than lower (U.S. EPA, 2013, section 9.5.2). As with any summary statistic, these exposure indices retain information on some, but not all, characteristics of the original observations.

Based on extensive review of the published literature on different types of exposure-response metrics, including comparisons between metrics, the EPA has focused on cumulative, concentration-weighted indices, recognizing them as the most appropriate biologically based metrics to consider in this context (U.S. EPA, 1996a; U.S. EPA, 1996b; U.S. EPA, 2006a; U.S. EPA, 2013). In the last two reviews of the O₃ NAAQS, the EPA concluded that the risk to vegetation comes primarily from cumulative exposures to O₃ over a season or seasons¹⁶³ and focused on metrics intended to characterize such exposures: SUM06¹⁶⁴ in the 1997 review (61 FR 65716, December 13, 1996) and W126 in the 2008 review (72 FR 37818, July 11, 2007). Although in both reviews the policy decision was made not to revise the form and averaging time of the secondary standard, the Administrator, in both cases, also concluded, consistent with CASAC advice, that a cumulative, seasonal index was the most biologically relevant way to relate exposure to plant growth response (62 FR 38856, July 18, 1997; 73 FR 16436, March 27, 2008). This approach for characterizing O₃ exposure concentrations that are biologically relevant with regard to potential vegetation effects received strong support from CASAC in the last review and again in this review, including strong support for use of such a metric as the form for the secondary standard (Henderson, 2006, 2008; Samet, 2010; Frey, 2014c).

Alternative methods for characterizing O₃ exposure to predict plant response have, in recent years,

¹⁶³ In describing the form as “seasonal,” the EPA is referring generally to the growing season of O₃-sensitive vegetation, not to the seasons of the year (*i.e.*, spring, summer, fall, winter).

¹⁶⁴ The SUM06 index is a threshold-based approach described as the sum of all hourly O₃ concentrations greater or equal to 0.06 ppm observed during a specified daily and seasonal time window (U.S. EPA, 2013, section 9.5.2). The W126 index is a non-threshold approach, described more fully below.

included flux models, which some researchers have claimed may “better predict vegetation responses to O₃ than exposure-based approaches” because they estimate the ambient O₃ concentration that actually enters the leaf (*i.e.*, flux or deposition). However, the ISA notes that “[f]lux calculations are data intensive and must be carefully implemented” (U.S. EPA, 2013, p. 9–114). Further, the ISA states, “[t]his uptake-based approach to quantify the vegetation impact of O₃ requires inclusion of those factors that control the diurnal and seasonal O₃ flux to vegetation (*e.g.*, climate patterns, species and/or vegetation-type factors and site-specific factors)” (U.S. EPA, 2013, p. 9–114). In addition to these data requirements, each species has different amounts of internal detoxification potential that may protect species to differing degrees. The lack of detailed species- and site-specific data required for flux modeling in the U.S. and the lack of understanding of detoxification processes have continued to make this technique less viable for use in vulnerability and risk assessments at the national scale in the U.S. (U.S. EPA, 2013, section 9.5.4).

Therefore, consistent with the ISA conclusions regarding the appropriateness of considering cumulative exposure indices that preferentially weight higher concentrations over lower for predicting O₃ effects of concern based on the well-established conclusions and supporting evidence described above, and in light of continued CASAC support, we continue to focus on cumulative concentration-weighted indices as the most biologically relevant metrics for consideration of O₃ exposures eliciting vegetation-related effects. Quantifying exposure in this way “improves the explanatory power of exposure/response models for growth and yield over using indices based on mean and peak exposure values” (U.S. EPA, 2013, section 2.6.6.1, p. 2–44). In this review, as in the last review, we use the W126-based cumulative, seasonal metric (U.S. EPA, 2013, sections 2.6.6.1 and 9.5.2) for consideration of the effects evidence and in the exposure and risk analyses in the WREA.

This metric, commonly called the W126 index, is a non-threshold approach described as the sigmoidally weighted sum of all hourly O₃ concentrations observed during a specified daily and seasonal time window, where each hourly O₃ concentration is given a weight that increases from zero to one with increasing concentration (U.S. EPA, 2014c, p. 5–6; U.S. EPA 2013, p. 9–101).

The first step in calculating the seasonal W126 index, as described and considered in this review, is to sum the weighted ambient O₃ concentrations

during daylight hours (defined as 8:00 a.m. to 8:00 p.m.) within each calendar month, resulting in monthly index values (U.S. EPA, 2014b, pp. 4–5 to

4–6). As more completely described in the WREA, the monthly W126 index values are calculated from hourly O₃ concentrations as follows:

$$\text{Monthly W126} = \sum_{d=1}^N \sum_{h=8}^{19} \frac{C_{dh}}{1+4403 \cdot \exp(-126 \cdot C_{dh})}$$

where *N* is the number of days in the month, *d* is the day of the month (*d* = 1, 2, . . . , *N*), *h* is the hour of the day (*h* = 0, 1, . . . , 23), and *C_{dh}* is the hourly O₃ concentration observed on day *d*, hour *h*, in parts per million. The seasonal W126 index value for a specific year is the maximum sum of the monthly index values for three consecutive months. Three-year W126 index values are calculated by taking the average of seasonal W126 index values for three consecutive years (U.S. EPA, 2014b, pp. 4–5 to 4–6; Wells, 2014a).

2. Overview of Welfare Exposure and Risk Assessment

This section outlines the information presented in section IV.C of the proposal regarding the WREA conducted for this review, which built upon similar analyses performed in the last review. The WREA focuses primarily on analyses related to two types of effects on vegetation: Reduced growth (biomass loss) in both trees and agricultural crops, and foliar injury. The assessments of O₃-associated reduced growth in native trees and crops (specifically, RBL and RYL, respectively) include analysis of associated changes in related ecosystem services, including pollution removal, carbon sequestration or storage, and hydrology, as well as economic impacts on the forestry and agriculture sectors of the economy. The foliar injury assessments include cumulative analyses of the proportion of USFS biosite index scores¹⁶⁵ above zero (or five, in a separate set of analyses) with increasing W126 exposure index estimates, with and without consideration of soil moisture conditions. The implications of visible foliar injury in national parks were considered in a screening level assessment and three case studies.¹⁶⁶

¹⁶⁵ Sampling sites in the FIA/FHM O₃ biomonitoring program, called “biosites”, are plots of land on which data are collected regarding the incidence and severity of visible foliar injury on a variety of O₃-sensitive plant species. Biosite index scores are derived from these data (U.S. EPA, 2014b, section 7.2.1).

¹⁶⁶ All of the analyses are described in detail in the WREA and summarized in the PA and in section IV.C of the proposal (U.S. EPA, 2014a; U.S.

Growth-related effects were assessed for W126-based exposure estimates in five scenarios of national-scale¹⁶⁷ air quality: Recent conditions (2006 to 2008), the existing secondary standard, and W126 index values of 15 ppm-hrs, 11 ppm-hrs, and 7 ppm-hrs, using 3-year averages (U.S. EPA, 2014b, chapter 4). For each of these scenarios, 3-year average W126 exposure index values were estimated for 12 kilometer (km) by 12 km grid cells in a national-scale spatial surface. The method for creating these grid cell estimates generally involved two steps (summarized in Table 5–4 of the PA).

The first step in creating the grid cell estimates for each scenario was calculation of the average W126 index value (across the three years) at each monitor location. For the recent conditions scenario, this value was based on unadjusted O₃ concentrations from monitoring data. For the other four scenarios, the W126 index value for each monitor location was calculated from model-adjusted hourly O₃ concentrations. The adjusted concentrations were based on model-predicted relationships between O₃ at each monitor location and reductions in NO_x. Adjustments were applied independently for each of the nine U.S. regions (see U.S. EPA, 2014b, section 4.3.4.1).¹⁶⁸ The existing standard scenario was created first, with the result being a national dataset for which the highest monitor location in each U.S. region had a design value equal to the level of the current standard.¹⁶⁹ The W126 scenarios were created from the hourly concentrations used to create the existing standard scenario, with model-

EPA, 2014b; 79 FR 75324–75329, December 17, 2014).

¹⁶⁷ Although the scenarios and the grid cell O₃ concentrations on which they are based were limited to the contiguous U.S., we have generally used the phrase “national-scale” in reference to the WREA scenarios and surfaces.

¹⁶⁸ The U.S. regions referenced here and in section IV.C below are NOAA climate regions, as shown in Figure 2B–1 of the PA.

¹⁶⁹ The adjustment results in broad regional reductions in O₃ and includes reductions in O₃ at some monitors that were already at or below the target level. These reductions do not represent an optimized control scenario, but rather characterize one potential distribution of air quality across a region that meets the scenario target (U.S. EPA, 2014b, sections 4.3.4.2 and 4.4).

based adjustments made at all monitor sites in those regions with a site not already at or below the target W126 value for that scenario (U.S. EPA, 2014b, section 4.3.4.1).¹⁷⁰

After completing step one for all the scenarios, the second step involved creating the national-scale spatial surfaces (composed of 3-year W126 index values at grid cell centroids). These were created by applying the Voronoi Neighbor Averaging (VNA) spatial interpolation technique to the monitor-location, 3-year W126 index values (described in step 1).¹⁷¹ This step of creating the gridded spatial surfaces resulted in further reduction of the highest values in each modeling region, as demonstrated by comparing the W126 index values from steps one and two for the existing standard scenario. After the step-one adjustment of the monitor location concentrations such that the highest location in each NOAA region just met the existing standard (using relationships mentioned above), the maximum 3-year average W126 values in the nine regions ranged from 18.9 ppm-hrs in the West region to 2.6 ppm-hrs in the Northeast region (U.S. EPA, 2014b, Table 4–3). After application of the VNA technique in the second step, however, the highest 3-year average W126 values across the national surface grid cells, which were in the Southwest region, were below 15 ppm-hrs (U.S. EPA, 2014b, Figure 4–7).¹⁷²

All of the assessments based on growth impacts relied on the W126 index estimates from the national-scale spatial surfaces (created from the 3-year average monitor location values as described above). Among the analyses related to visible foliar injury, a small component of the screening-level

¹⁷⁰ In regions where the air quality adjustment was applied, it was based on emissions reductions determined necessary for the highest monitor in that region to just equal the existing standard or the W126 target for the scenario. Concentrations at all other monitor locations in the region were also adjusted based on the same emissions reductions assumptions.

¹⁷¹ The VNA technique is described in the WREA (U.S. EPA, 2014b, Appendix 4A).

¹⁷² Thus, it can be seen that application of the VNA interpolation method to estimate W126 index values at the centroid of every 12 km x 12 km grid cell rather than only at each monitor location results in a lowering of the highest values in each region.

national park assessment and also the three national park case studies involved summarizing 3-year W126 index estimates from the four air quality scenarios. However, the visible foliar injury cumulative proportion analyses and a component of the national park screening-level assessment relied on national-scale spatial surfaces of single-year, unadjusted W126 index values created for each year from 2006 through 2010 using the VNA interpolation technique applied to the monitor location index values for these years (U.S. EPA, 2014b, section 4.3.2, Appendix 4A).

Because the W126 estimates generated for the different air quality scenarios assessed are inputs to the vegetation risk analyses for tree biomass and crop yield loss, and also used in some components of the visible foliar injury assessments, limitations and uncertainties in the air quality analyses, which are discussed in detail in the WREA and some of which are mentioned here, are propagated into those analyses (U.S. EPA, 2014b, chapters 4 and 8 and section 8.5, Table 4–5). An important uncertainty in the analyses is the application of regionally determined emissions reductions to meet the existing standard (U.S. EPA, 2014b, section 8.5.1). The model adjustments are based on emissions reductions in NO_x and characterize only one potential distribution of air quality across a region when all monitor locations meet the standard, as well as for the W126 scenarios (U.S. EPA, 2014b, section 4.3.4.2).¹⁷³

An additional uncertainty related to the W126 index estimates in the national surfaces for each air quality scenario, and to the estimates for the single-year surfaces used in the visible foliar injury cumulative analysis, comes with the creation of the national-scale spatial surfaces of grid cells from the monitor-location O₃ data.¹⁷⁴ In general, spatial interpolation techniques perform better in areas where the O₃ monitoring network is denser. Therefore, the W126 index values estimated using this

¹⁷³ The adjustment is applied to all monitor locations in each region. In this way, the adjustment results in broad regional reductions in O₃ and includes reductions in O₃ at some monitors that were already meeting or below the target level. Thus, the adjustments performed to develop a scenario meeting a target level at the highest monitor in each region did result in substantial reduction below the target level in some areas of the region. This result at the monitors already well below the target indicates an uncertainty with regard to air quality expected from specific control strategies that might be implemented to meet a particular target level.

¹⁷⁴ Some uncertainty is inherent in any approach to characterizing O₃ air quality over broad geographic areas based on concentrations at monitor locations.

technique in rural areas in the West, Northwest, Southwest, and West North Central regions where there are few or no monitors (U.S. EPA, 2014b, Figure 2–1) are more uncertain than those estimated for areas with denser monitoring. Further, as described above, this interpolation method generally underpredicts the highest W126 exposure index values. Due to the important influence of higher exposures in determining risks to plants, the potential for the VNA interpolation approach to dampen peak W126 index values could result in an underestimation of risks to vegetation in some areas.¹⁷⁵

The vegetation analyses performed in the WREA, along with key observations, insights, uncertainties and limitations were summarized in sections IV.C.2 through IV.C.3 of the proposal. Highlights for the three categories of biomass loss and foliar injury assessments are summarized here.

a. Tree Growth, Productivity and Carbon Storage

These assessments rely on the species-specific E–R functions described in section IV.A.1.b above. For the air quality scenarios described above, the WREA applied the species-specific E–R functions to develop estimates of O₃-associated RBL and associated effects on productivity, carbon storage and associated ecosystem services (U.S. EPA, 2014b, Chapter 6). More specifically, the WREA derived species-specific and weighted RBL estimates for grid cells across the continental U.S. and summarized the estimates by counties and national parks. Additional WREA case study analyses focused on selected urban areas. The WREA estimates indicate substantial heterogeneity in plant responses to O₃, both within species (*e.g.*, study-specific variation), between species, and across regions of the U.S. National variability in the estimates (*e.g.*, eastern vs western U.S.) is influenced by there being different sets of resident species (with different E–R functions) in different areas of the U.S., as well as differences in number of national parks and O₃ monitors. For example, the eastern U.S. has different resident species compared to the western U.S., and the eastern U.S. has far more such species. Additionally, there are more national parks in the western than the eastern U.S., yet fewer O₃ monitors (U.S. EPA, 2014b, chapter 8).

¹⁷⁵ In the visible foliar injury dataset used for the cumulative analysis, underestimation of W126 index values at sites with injury would contribute to overestimates of the cumulative proportion of sites with injury plotted for the lower W126 values.

Relative biomass loss nationally (across all of the air quality surface grid cells) was estimated for each of the 12 studied species from the composite E–R functions for each species described above and information on the distribution of those species across the U.S. (U.S. EPA, 2014b, section 6.2.1.3 and Appendix 6A). In consideration of CASAC advice (summarized in section IV.A.1.b above), the WREA derived RBL and weighted RBL (wRBL) estimates separately, both with and without the eastern cottonwood, and the PA and proposal gave primary focus to analyses that exclude cottonwood. These analyses provided estimates of per-species and cross-species RBL in the different air quality scenarios. Air quality scenario estimates were also developed in terms of proportion of basal area affected at different magnitudes of RBL. The wRBL analysis integrated the species-specific estimates, providing an indication of potential magnitude of ecological effect possible in some ecosystems. The county analyses also included analyses focused on the median species response. The WREA also used the E–R functions to estimate RBL across tree lifespans and the resulting changes in consumer and producer/farmer economic surplus in the forestry and agriculture sectors of the economy. Case studies in five urban areas provided comparisons across air quality scenarios of estimates for urban tree pollutant removal and carbon storage or sequestration.

The array of uncertainties associated with estimates from these tree RBL analyses are summarized in the proposal and described in detail in the WREA, including the potential for the air quality scenarios to underestimate the higher W126 index values and associated implications for the RBL-related estimates, as referenced above.

b. Crop Yield Loss

These assessments rely on the species-specific E–R functions described in section IV.A.1.b above. For the different air quality scenarios, the WREA applied the species-specific E–R functions to develop estimates of O₃ impacts related to crop yield, including annual yield losses estimated for 10 commodity crops grown in the U.S. and how these losses affect producer and consumer economic surpluses (U.S. EPA, 2014b, sections 6.2, 6.5). The WREA derived estimates of crop RYL nationally and in a county-specific analysis, relying on information regarding crop distribution (U.S. EPA, 2014b, section 6.5). As with the tree analyses described above, the county analysis included estimates based on

the median O₃ response across the studied crop species (U.S. EPA, 2014b, section 6.5.1, Appendix 6B).

Overall effects on agricultural yields and producer and consumer surplus depend on the ability of producers/farmers to substitute other crops that are less O₃ sensitive, and the responsiveness, or elasticity, of demand and supply (U.S. EPA, 2014b, section 6.5). The WREA discusses multiple areas of uncertainty associated with the crop yield loss estimates, including those associated with the model-based adjustment methodology as well as those associated with the projection of yield loss using the Forest and Agriculture Sector Optimization Model (with greenhouse gases) at the estimated O₃ concentrations (U.S. EPA, 2014b, Table 6–27, section 8.5). Because the W126 index estimates generated in the air quality scenarios are inputs to the vegetation risk analyses for crop yield loss, any uncertainties in the air quality scenario estimation of W126 index values are propagated into those analyses (U.S. EPA, 2014b, Table 6–27, section 8.5). Therefore, the air quality scenarios in the crop yield analyses have the same uncertainties and limitations as in the biomass loss analyses (summarized above), including those associated with the model-based adjustment methodology (U.S. EPA, 2014b, section 8.5).

c. Visible Foliar Injury

The WREA presents a number of analyses of O₃-related visible foliar injury and associated ecosystem services impacts (U.S. EPA, 2014b, Chapter 7). In the initial analysis, the WREA used the biomonitoring site data from the USFS FHM/FIA Network (USFS, 2011),¹⁷⁶ associated soil moisture data during the sample years, and national surfaces of ambient air O₃ concentrations based on spatial interpolation of monitoring data from 2006 to 2010 in a cumulative analysis of the proportion of biosite records with any visible foliar injury, as indicated by a nonzero biosite index score (U.S. EPA, 2014b, section 7.2). This analysis was done for all records together, and also for subsets based on soil moisture conditions (normal, wet or dry).

In each cumulative analysis, the biosite records were ordered by W126 index and then, moving from low to high W126 index, the records were cumulated into a progressively larger dataset. With the addition of each new

data point (composed of biosite index score and W126 index value for a biosite and year combination) to the cumulative dataset, the percentage of sites with a nonzero biosite index score was derived and plotted versus the W126 index estimate for the just added data point. The cumulative analysis for all sites indicates that (1) as the cumulative set of sites grows with addition of sites with progressively higher W126 index values, the proportion of the dataset for which no foliar injury was recorded changes (increases) noticeably prior to about 10 ppm-hrs (10.46 ppm-hrs), and (2) as the cumulative dataset grows still larger with the addition of records for higher W126 index estimates, the proportion of the cumulative dataset with no foliar injury remains relatively constant (U.S. EPA, 2014b, Figure 7–10). The data for normal moisture years are very similar to the dataset as a whole, with an overall proportion of about 18 percent for presence of any foliar injury. The data for relatively wet years have a much higher proportion of biosites showing injury, approximately 25% when all data are included, and a proportion of approximately 20% when data for W126 index estimates up to about 5–8 ppm-hrs are included (U.S. EPA, 2014b, Figure 7–10).¹⁷⁷ The overall proportion showing injury for the subset for relatively dry conditions is much lower, less than 15% for the subset (U.S. EPA, 2014b, section 7.2.3, Figures 7–10). While these analyses indicate the potential for foliar injury to occur under conditions that meet the current standard, the extent of foliar injury that might be expected under different exposure conditions is unclear from these analyses.

Criteria derived from the cumulative analyses were then used in two additional analyses. The national-scale screening-level assessment compared W126 index values estimated within 214 national parks using the VNA technique described above for the individual years from 2006 to 2010 with benchmark criteria developed from the biosite data analysis (U.S. EPA, 2014b, Appendix 7A and section 7.3). Separate case study analyses described visits, as well as visitor uses and expenditures for three national parks, and the 3-year

W126 index estimates in those parks for the four air quality scenarios (U.S. EPA, 2014b, section 7.4). Uncertainties associated with these analyses, included those associated with the W126 index estimates, are discussed in the WREA, sections 7.5 and 8.5.3, and in WREA Table 7–24, and also summarized in the PA (e.g., U.S. EPA, 2014c, section 6.3).

3. Potential Impacts on Public Welfare

As provided in the CAA, section 109(b)(2), the secondary standard is to “specify a level of air quality the attainment and maintenance of which in the judgment of the Administrator . . . is requisite to protect the public welfare from any known or anticipated adverse effects associated with the presence of such air pollutant in the ambient air.” Effects on welfare include, but are not limited to, “effects on soils, water, crops, vegetation, man-made materials, animals, wildlife, weather, visibility, and climate, damage to and deterioration of property, and hazards to transportation, as well as effects on economic values and on personal comfort and well-being” (CAA section 302(h)). The secondary standard is not meant to protect against all known or anticipated O₃-related effects, but rather those that are judged to be adverse to the public welfare, and a bright-line determination of adversity is not required in judging what is requisite (78 FR 8312, January 15, 2013; see also 73 FR 16496, March 27, 2008). Thus, the level of protection from known or anticipated adverse effects to public welfare that is requisite for the secondary standard is a public welfare policy judgment to be made by the Administrator. In the current review, the Administrator’s judgment is informed by conclusions drawn with regard to adversity of effects to public welfare in decisions on secondary O₃ standards in past reviews.

As indicated by the Administrator in the 2008 decision, the degree to which O₃ effects on vegetation should be considered to be adverse to the public welfare depends on the intended use of the vegetation and the significance of the vegetation to the public welfare (73 FR 16496, March 27, 2008). Such judgments regarding public welfare significance in the last O₃ NAAQS decision gave particular consideration to O₃ effects in areas with special federal protections, and lands set aside by states, tribes and public interest groups to provide similar benefits to the public welfare (73 FR 16496, March 27, 2008). For example, in reaching his conclusion regarding the need for revision of the secondary standard in the 2008 review, the Administrator took

¹⁷⁶ Data were not available for several western states (Montana, Idaho, Wyoming, Nevada, Utah, Colorado, Arizona, New Mexico, Oklahoma, and portions of Texas).

¹⁷⁷ As discussed in section IV.C.2 below, as the cumulative set increases, with increasing W126 values, the overall prevalence of visible foliar injury in the cumulative set is more and more influenced by data for the lower W126 values. Accordingly, the “leveling off” observed above ~10 ppm-hrs in the ‘all sites’ analysis likely reflects the counterbalancing of visible foliar injury occurrence at the relatively fewer higher O₃ sites by the larger representation within the subset of the lower W126 conditions associated with which there is lower occurrence or extent of foliar injury.

note of “a number of actions taken by Congress to establish public lands that are set aside for specific uses that are intended to provide benefits to the public welfare, including lands that are to be protected so as to conserve the scenic value and the natural vegetation and wildlife within such areas, and to leave them unimpaired for the enjoyment of future generations” (73 FR 16496, March 27, 2008). As further recognized in the 2008 notice, “[s]uch public lands that are protected areas of national interest include national parks and forests, wildlife refuges, and wilderness areas” (73 FR 16496, March 27, 2008).^{178 179} Such areas include Class I areas¹⁸⁰ which are federally mandated to preserve certain air quality related values. Additionally, as the Administrator recognized, “States, Tribes and public interest groups also set aside areas that are intended to provide similar benefits to the public welfare, for residents on State and Tribal lands, as well as for visitors to those areas” (73 FR 16496, March 27, 2008). The Administrator took note of the “clear public interest in and value of maintaining these areas in a condition that does not impair their intended use and the fact that many of these lands contain O₃-sensitive species” (73 FR 16496, March 27, 2008).

The concept described in the 2008 notice regarding the degree to which effects on vegetation in specially protected areas, such as those identified above, may be judged adverse also applies beyond the species level to the ecosystem level, such that judgments

¹⁷⁸ For example, the National Park Service Organic Act of 1916 established the National Park Service (NPS) and, in describing the role of the NPS with regard to “Federal areas known as national parks, monuments, and reservations”, stated that the “fundamental purpose” for these federal areas “is to conserve the scenery and the natural and historic objects and the wild life therein and to provide for the enjoyment of the same in such manner and by such means as will leave them unimpaired for the enjoyment of future generations.” 16 U.S.C. 1.

¹⁷⁹ As a second example, the Wilderness Act of 1964 defines designated “wilderness areas” in part as areas “protected and managed so as to preserve [their] natural conditions” and requires that these areas “shall be administered for the use and enjoyment of the American people in such manner as will leave them unimpaired for future use and enjoyment as wilderness, and so as to provide for the protection of these areas, [and] the preservation of their wilderness character . . .” 16 U.S.C. 1131 (a).

¹⁸⁰ Areas designated as Class I include all international parks, national wilderness areas which exceed 5,000 acres in size, national memorial parks which exceed 5,000 acres in size, and national parks which exceed six thousand acres in size, provided the park or wilderness area was in existence on August 7, 1977. Other areas may also be Class I if designated as Class I consistent with the CAA.

can depend on the intended use¹⁸¹ for, or service (and value) of, the affected vegetation, ecological receptors, ecosystems and resources and the significance of that use to the public welfare (73 FR 16496, March 27, 2008). Uses or services provided by areas that have been afforded special protection can flow in part or entirely from the vegetation that grows there. Aesthetic value and outdoor recreation depend, at least in part, on the perceived scenic beauty of the environment (U.S. EPA, 2014b, chapters 5 and 7). Further, analyses have reported that the American public values—in monetary as well as nonmonetary ways—the protection of forests from air pollution damage. In fact, studies that have assessed willingness-to-pay for spruce-fir forest protection in the southeastern U.S. from air pollution and insect damage have found that values held by the survey respondents for the more abstract services (existence, option and bequest)¹⁸² were greater than those for recreation or other services (U.S. EPA, 2014b, Table 5–6; Haefele *et al.*, 1991; Holmes and Kramer, 1995).

The spatial, temporal and social dimensions of public welfare impacts are also influenced by the type of service affected. For example, a national park can provide direct recreational services to the thousands of visitors that come each year, but also provide an indirect value to the millions who may not visit but receive satisfaction from knowing it exists and is preserved for the future (U.S. EPA, 2014b, chapter 5, section 5.5.1). Similarly, ecosystem services can be realized over a range of temporal scales. An evaluation of adversity to the public welfare might also consider the likelihood, type, and magnitude of the effect, as well as the potential for recovery and any uncertainties relating to these

¹⁸¹ Ecosystem services have been defined as “the benefits that people obtain from ecosystems” (U.S. EPA, 2013, Preamble, p. 1xxii; UNEP, 2003) and thus are an aspect of the use of a type of vegetation or ecosystem. Similarly, a definition used for the purposes of the EPA benefits assessments states that ecological goods and services are the “outputs of ecological functions or processes that directly or indirectly contribute to social welfare or have the potential to do so in the future” and that “[s]ome outputs may be bought and sold, but most are not marketed” (U.S. EPA, 2006b). Ecosystem services analyses were one of the tools used in the last review of the secondary standards for oxides of nitrogen and sulfur to inform the decisions made with regard to adequacy and as such, were used in conjunction with other considerations in the discussion of adversity to public welfare (77 FR 20241, April 3, 2012).

¹⁸² Public surveys have indicated that Americans rank as very important the existence of resources, the option or availability of the resource and the ability to bequest or pass it on to future generations (Cordell *et al.*, 2008).

conditions, as stated in the preamble of the 2012 final notice of rulemaking on the secondary standards for oxides of nitrogen and sulfur (77 FR 20232, April 3, 2012).

The three main categories of effects on vegetation discussed in section IV.A.1.b above differ with regard to aspects important to judging their public welfare significance. Judgments regarding crop yield loss, for example, depend on considerations related to the heavy management of agriculture in the U.S., while judgments regarding the other categories of effects generally relate to considerations regarding forested areas. For example, while both tree growth-related effects and visible foliar injury have the potential to be significant to the public welfare through impacts in Class I and other protected areas, they differ in how they might be significant and with regard to the clarity of the data that describe the relationship between the effect and the services potentially affected.

With regard to effects on tree growth, reduced growth is associated with effects on an array of ecosystem services including reduced productivity, altered forest and forest community (plant, insect and microbe) composition, reduced carbon storage and altered water cycling (U.S. EPA, 2013, Figure 9–1, sections 9.4.1.1 and 9.4.1.2; U.S. EPA, 2014b, section 6.1). For example, forest or forest community composition can be affected through O₃ effects on growth and reproductive success of sensitive species in the community, with the extent of compositional changes dependent on factors such as competitive interactions (U.S. EPA, 2013, sections 9.4.3 and 9.4.3.1). Depending on the type and location of the affected ecosystem, services benefitting the public in other ways can be affected as well. For example, other services valued by people that can be affected by reduced tree growth, productivity and carbon storage include aesthetic value, food, fiber, timber, other forest products, habitat, recreational opportunities, climate and water regulation, erosion control, air pollution removal, and desired fire regimes (U.S. EPA 2013, sections 9.4.1.1 and 9.4.1.2; U.S. EPA, 2014b, section 6.1, Figure 6–1, section 6.4, Table 6–13). Further, impacts on some of these services (e.g., forest or forest community composition) may be considered of greater public welfare significance when occurring in Class I or other protected areas.

Consideration of the magnitude of tree growth effects that might cause or contribute to adverse effects for trees, forests, forested ecosystems or the public welfare is complicated by aspects

of, or limitations in, the available information. For example, the evidence on tree seedling growth effects, deriving from the E-R functions for 11 species (described in section IV.A.1 above), provides no clear threshold or breakpoint in the response to O₃ exposure. Additionally, there are no established relationships between magnitude of tree seedling growth reduction and forest ecosystem impacts and, as noted in section IV.A.1.b above, other factors can influence the degree to which O₃-induced growth effects in a sensitive species affect forest and forest community composition and other ecosystem service flows from forested ecosystems. These include (1) the type of stand or community in which the sensitive species is found (*i.e.*, single species versus mixed canopy); (2) the role or position the species has in the stand (*i.e.*, dominant, sub-dominant, canopy, understory); (3) the O₃ sensitivity of the other co-occurring species (O₃ sensitive or tolerant); and (4) environmental factors, such as soil moisture and others. The lack of such established relationships complicates judgments as to the extent to which different estimates of impacts on tree seedling growth would indicate significance to the public welfare and thus be an important consideration in the level of protection for the secondary standard.

During the 1997 review of the secondary standard, views related to this issue were provided by a 1996 workshop of 16 leading scientists in the context of discussing their views for a secondary O₃ standard (Heck and Cowling, 1997). In their consideration of tree growth effects as an indicator for forest ecosystems and crop yield reduction as an indicator of agricultural systems, the workshop participants identified annual percentages, of RBL for forest tree seedlings and RYL for agricultural crops, considered important to their judgments on the standard. With regard to forest ecosystems and seedling growth effects as an indicator, the participants selected a range of 1–2% RBL per year “to avoid cumulative effects of yearly reductions of 2%.” With regard to crops, they indicated an interest in protecting against crop yield reductions of 5% RYL yet noted uncertainties surrounding such a percentage which led them to identifying 10% RYL for the crop yield endpoint (Heck and Cowling, 1997). The workshop report provides no explicit rationale for the percentages identified (1–2% RBL and 5% or 10% RYL); nor does it describe their connection to ecosystem impacts of a specific

magnitude or type, nor to judgments on significance of the identified effects for public welfare, *e.g.*, taking into consideration the intended use and significance of the affected vegetation (Heck and Cowling, 1997). In recognition of the complexity of assessing the adversity of tree growth effects and effects on crop yield in the broader context of public welfare, the EPA’s consideration of those effects in both the 1997 and 2008 reviews extended beyond the consideration of various benchmark responses for the studied species, and, with regard to crops, additionally took note of their extensive management (62 FR 38856, July 18, 1997; 73 FR 16436, March 27, 2008).

While, as noted above, public welfare benefits of forested lands can be particular to the type of area in which the forest occurs, some of the potential public welfare benefits associated with forest ecosystems are not location dependent. A potentially extremely valuable ecosystem service provided by forested lands is carbon storage, a regulating service that is “of paramount importance for human society” (U.S. EPA, 2013, section 2.6.2.1 and p. 9–37). As noted above, the EPA has concluded that this ecosystem service has a likely causal relationship with O₃ in ambient air. The service of carbon storage is potentially important to the public welfare no matter in what location the sensitive trees are growing or what their intended current or future use. In other words, the benefit exists as long as the tree is growing, regardless of what additional functions and services it provides. Another example of locations potentially vulnerable to O₃-related impacts but not necessarily identified for such protection might be forested lands, both public and private, where trees are grown for timber production. Forests in urbanized areas also provide a number of services that are important to the public in those areas, such as air pollution removal, cooling, and beautification. There are also many other tree species, such as species identified by the USFS and various ornamental and agricultural species (*e.g.*, Christmas trees, fruit and nut trees), that provide ecosystem services that may be judged important to the public welfare but whose vulnerability to O₃ impacts has not been quantitatively characterized (U.S. EPA, 2014b, Chapter 6).

As noted above, in addition to tree growth-related effects, O₃-induced visible foliar injury also has the potential to be significant to the public welfare through impacts in Class I and other similarly protected areas. Visible

foliar injury is a visible bioindicator of O₃ exposure in species sensitive to this effect, with the injury affecting the physical appearance of the plant. Accordingly visible foliar injury surveys are used by federal land managers as tools in assessing potential air quality impacts in Class I areas. These surveys may focus on plant species that have been identified as potentially sensitive air quality related values (AQRVs) due to their sensitivity to O₃-induced foliar injury (USFS, NPS, FWS, 2010). An AQRV is defined by the National Park Service as a “resource, as identified by the [federal land manager] for one or more Federal areas that may be adversely affected by a change in air quality,” and the resource “may include visibility or a specific scenic, cultural, physical, biological, ecological, or recreational resource identified by the [federal land manager] for a particular area” (USFS, NPS, USFWS, 2010).¹⁸³ No criteria have been established, however, regarding a level or prevalence of visible foliar injury considered to be adverse to the affected vegetation, and, as noted in section IV.A.1.b above, there is not a clear relationship between visible foliar injury and other effects, such as reduced growth and productivity.¹⁸⁴ Thus, key considerations with regard to public welfare significance of this endpoint

¹⁸³ The identification, monitoring and assessment of AQRVs with regard to an adverse effect is an approach used for assessing the potential for air pollution impacts in Class I areas from pending permit actions (USFS, NPS, USFWS, 2010). An adverse impact is recognized by the National Park Service as one that results in diminishment of the Class I area’s national significance or the impairment of the ecosystem structure or functioning, as well as impairment of the quality of the visitor experience (USFS, NPS, USFWS, 2010). Federal land managers make such adverse impact determinations on a case-by-case basis, using technical and other information that they provide for consideration by permitting authorities. The National Park Service has developed a document describing an overview of approaches related to assessing projects under the National Environmental Policy Act and other planning initiatives affecting the National Park System (http://www.nature.nps.gov/air/Pubs/pdf/AQGuidance_2011-01-14.pdf).

¹⁸⁴ The National Park Service identifies various ranges of W126 index values in providing approaches for assessing air quality-related impacts of various development projects which appear to be based on the 1996 workshop report (Heck and Cowling, 1997), and may, at the low end, relate to a benchmark derived for the highly sensitive species, black cherry, for growth effects (10% RBL), rather than visible foliar injury (Kohut, 2007; Lefohn *et al.*, 1997). As noted in section IV.A.1.b above, visible foliar injury is not always a reliable indicator of other negative effects on vegetation (U.S. EPA, 2013, p. 9–39). We also note that the USFS biomonitoring analyses of visible foliar injury biomonitoring data commonly make use of a set of biosite index categories for which risk assumptions have been assigned, providing a relative scale of possible impacts (Campbell *et al.*, 2007); however, little information is available on the studies, effects and judgments on which these categories are based.

have related to qualitative consideration of the plant's aesthetic value in protected forested areas. Depending on the extent and severity, O₃-induced visible foliar injury might be expected to have the potential to impact the public welfare in scenic and/or recreational areas during the growing season, particularly in areas with special protection, such as Class I areas.

The ecosystem services most likely to be affected by O₃-induced visible foliar injury (some of which are also recognized above for tree growth-related effects) are cultural services, including aesthetic value and outdoor recreation. In addition, several tribes have indicated that many of the species identified as O₃ sensitive (including bioindicator species) are culturally significant (U.S. EPA, 2014c, Table 5-1). The geographic extent of protected areas that may be vulnerable to such public welfare effects of O₃ is potentially appreciable. Sixty-six plant species that occur on U.S. National Park Service (NPS) and U.S. Fish and Wildlife Service lands¹⁸⁵ have been identified as sensitive to O₃-induced visible foliar injury, and some also have particular cultural importance to some tribes (U.S. EPA, 2014c, Table 5-1 and Appendix 5-A; U.S. EPA, 2014b, section 6.4.2). Not all species are equally sensitive to O₃, however, and quantitative E-R relationships for O₃ exposure and other important effects, such as seedling growth reduction, are only available for a subset of 12 of the 66, as summarized in section IV.A.1.b above. A diverse array of ecosystem services has been identified for these twelve species (U.S. EPA, 2014c, Table 5-1). Two species in this group that are slightly more sensitive than the median for the group with regard to effects on growth are the ponderosa pine and quaking aspen (U.S. EPA, 2014b, section 6.2), the ranges for which overlap with many lands that are protected or preserved for enjoyment of current and future generations (consistent with the discussion above on Class I and other protected areas), including such lands located in the west and southwest regions of the U.S. where ambient O₃ concentrations and associated cumulative seasonal exposures can be highest (U.S. EPA, 2014c, Appendix 2B).¹⁸⁶

With regard to agriculture-related effects, the EPA has recognized other complexities, stating that the degree to

which O₃ impacts on vegetation that could occur in areas and on species that are already heavily managed to obtain a particular output (such as commodity crops or commercial timber production) would impair the intended use at a level that might be judged adverse to the public welfare has been less clear (73 FR 16497, March 27, 2008). As noted in section IV.B.2 of the proposal, while having sufficient crop yields is of high public welfare value, important commodity crops are typically heavily managed to produce optimum yields. Moreover, based on the economic theory of supply and demand, increases in crop yields would be expected to result in lower prices for affected crops and their associated goods, which would primarily benefit consumers. These competing impacts on producers and consumers complicate consideration of these effects in terms of potential adversity to the public welfare (U.S. EPA, 2014c, sections 5.3.2 and 5.7). When agricultural impacts or vegetation effects in other areas are contrasted with the emphasis on forest ecosystem effects in Class I and similarly protected areas, it can be seen that the Administrator has in past reviews judged the significance to the public welfare of O₃-induced effects on sensitive vegetation growing within the U.S. to differ depending on the nature of the effect, the intended use of the sensitive plants or ecosystems, and the types of environments in which the sensitive vegetation and ecosystems are located, with greater significance ascribed to areas identified for specific uses and benefits to the public welfare, such as Class I areas, than to areas for which such uses have not been established (FR 73 16496-16497, March 27, 2008).

In summary, several considerations are recognized as important to judgments on the public welfare significance of the array of effects of different O₃ exposure conditions on vegetation. While there are complexities associated with the consideration of the magnitude of key vegetation effects that might be concluded to be adverse to ecosystems and associated services, there are numerous locations where O₃-sensitive tree species are present that may be vulnerable to impacts from O₃ on tree growth, productivity and carbon storage and their associated ecosystems and services. Cumulative exposures that may elicit effects and the significance of the effects in specific situations can vary due to differences in exposed species sensitivity, the importance of the observed or predicted O₃-induced effect, the role that the species plays in the ecosystem, the intended use of the

affected species and its associated ecosystem and services, the presence of other co-occurring predisposing or mitigating factors, and associated uncertainties and limitations. These factors contribute to the complexity of the Administrator's judgments regarding the adversity of known and anticipated effects to the public welfare.

B. Need for Revision of the Secondary Standard

The initial issue to be addressed in this review of the secondary standard for O₃ is whether, in view of the currently available scientific evidence, exposure and risk information and air quality analyses, as reflected in the record, the standard should be retained or revised. In drawing conclusions on adequacy of the current O₃ secondary standard, the Administrator has taken into account both evidence-based and quantitative exposure- and risk-based considerations, as well as advice from CASAC and public comment. Evidence-based considerations draw upon the EPA's assessment and integrated synthesis of the scientific evidence from experimental and field studies evaluating welfare effects related to O₃ exposure, with a focus on policy-relevant considerations, as discussed in the PA. Air quality analyses inform these considerations with regard to cumulative, seasonal exposures occurring in areas of the U.S. that meet the current standard. Exposure- and risk-based considerations draw upon the EPA assessments of risk of key welfare effects, including O₃ effects on forest growth, productivity, carbon storage, crop yield and visible foliar injury, expected to occur in model-based scenarios for the current standard, with appropriate consideration of associated uncertainties.

In evaluating whether it is appropriate to revise the current standard, the Administrator's considerations build on the general approach used in the last review, as summarized in section IV.A of the proposal, and reflect the body of evidence and information available during this review. The approach used is based on an integration of the information on vegetation effects associated with exposure to O₃ in ambient air, as well as policy judgments on the adversity of such effects to public welfare and on when the standard is requisite to protect public welfare from known or anticipated adverse effects. Such judgments are informed by air quality and related analyses, quantitative assessments, when available, and qualitative assessment of impacts that could not be quantified. The Administrator has taken into

¹⁸⁵ See <http://www2.nature.nps.gov/air/Pubs/pdf/flag/NPSozonesenspFLAG06.pdf>.

¹⁸⁶ Basal area for resident species in national forests and parks are available in files accessible at: <http://www.fs.fed.us/foresthealth/technology/nidrm2012.shtml>. Basal area is generally described as the area of ground covered by trees.

account both evidence of effects on vegetation and ecosystems and public uses of these entities that may be important to the public welfare. The decision on adequacy of the protection provided by the current standard has also considered the 2013 remand of the secondary standard by the D.C. Circuit such that this decision incorporates the EPA's response to this remand.

Section IV.B.1 below summarizes the basis for the proposed decision by the Administrator that the current secondary standard should be revised. Significant comments received from the public on the proposal are discussed in section IV.B.2 and the Administrator's final decision is described in section IV.B.3.

1. Basis for Proposed Decision

In evaluating whether it was appropriate to propose to retain or revise the current standard, as discussed in section IV.D of the proposal, the Administrator carefully considered the assessment of the current evidence in the ISA, findings of the WREA, including associated limitations and uncertainties, considerations and staff conclusions and associated rationales presented in the PA, views expressed by CASAC, and public comments that had been offered up to that point. In the paragraphs below, we summarize the proposal presentation of the PA considerations with regard to adequacy of the current secondary standard, advice from the CASAC, and the Administrator's proposed conclusions, drawing from section IV.D of the proposal, where a fuller discussion is presented.

a. Considerations and Conclusions in the PA

The PA evaluation is based on the longstanding evidence for O₃ effects and the associated conclusions in the current review of causal and likely causal relationships between O₃ in ambient air and an array of welfare effects at a range of biological and ecological scales of organization, as summarized in section IV.A.1 above (and described in detail in the ISA). Drawing from the ISA and CASAC advice, the PA emphasizes the strong support in the evidence for the conclusion that effects on vegetation are attributable to cumulative seasonal O₃ exposures, taking note of the improved "explanatory power" (for effects on vegetation) of the W126 index over other exposure metrics, as summarized in section IV.A.1.c above. The PA further recognizes the strong basis in the evidence for the conclusion that it is appropriate to use a cumulative

seasonal exposure metric, such as the W126 index, to judge impacts of O₃ on vegetation; related effects on ecosystems and services, such as carbon storage; and the level of public welfare protection achieved for such effects (U.S. EPA, 2014c, p. 5–78). As a result, based on the strong support in the evidence and advice from CASAC in the current and past reviews, the PA concludes that the most appropriate and biologically relevant way to relate O₃ exposure to plant growth, and to determine what would be adequate protection for public welfare effects attributable to the presence of O₃ in ambient air, is to characterize exposures in terms of a cumulative seasonal form, and in particular the W126 metric (U.S. EPA, 2014c, pp. 5–7 and 5–78). Accordingly, in considering the evidence with regard to level of protection provided by the current secondary standard, the PA considers air quality data and exposure-response relationships for vegetation effects, particularly those related to forest tree growth, productivity and carbon storage, in terms of the W126 index (U.S. EPA, 2014c, section 5.2; 79 FR 75330–75333, December 17, 2014).

In considering the extent to which such growth-related effects might be expected to occur under conditions that meet the current secondary standard, the PA focused particularly on tree seedling RBL estimates for the 11 species for which robust E–R functions have been developed, noting the CASAC concurrence with use of O₃-related tree biomass loss as a surrogate for related effects extending to the ecosystem scale (U.S. EPA, 2014c, p. 5–80, Frey, 2014c, p. 10). The PA evaluation relied on RBL estimates for these 11 species derived using the robust OTC-based E–R functions, noting that analyses newly performed in this review have reduced the uncertainty associated with using OTC E–R functions to predict tree growth effects in the field (U.S. EPA, 2014c, section 5.2.1; U.S. EPA, 2013, section 9.6.3.2).

In considering the RBL estimates for different O₃ conditions associated with the current standard, the PA focused primarily on the median of the species-specific (composite) E–R functions. In so doing, in the context of considering the adequacy of protection afforded by the current standard, the PA takes note of CASAC's view regarding a 6% median RBL (Frey, 2014c, p. 12). Based on the summary of RBL estimates in the PA, the PA notes that the median species RBL estimate, across the 11 estimates derived from the robust species-specific E–R functions, is at or above 6% for W126 index values of 19

ppm-hrs and higher (U.S. EPA, 2014c, Tables 6–1 and 5C–3).

In recognition of the potential significance to public welfare of vegetation effects in Class I areas, the proposal described in detail findings of the PA analysis of the occurrence of O₃ concentrations associated with the potential for RBL estimates above benchmarks of interest in Class I areas that meet the current standard, focusing on 22 Class I areas for which air quality data indicated the current standard was met and cumulative seasonal exposures, in terms of a 3-year average W126 index, were at or above 15 ppm-hrs (79 FR 75331–75332, Table 7, December 17, 2014; U.S. EPA, 2014c, Table 5–2). The PA noted that W126 index values (both annual and 3-year average values) in many such areas, distributed across multiple states and NOAA climatic regions, were above 19 ppm-hrs. The highest 3-year average value was over 22 ppm-hrs and the highest annual value was over 27 ppm-hrs, exposure values for which the corresponding median species RBL estimates markedly exceed 6%, which CASAC has termed "unacceptably high" (U.S. EPA, 2014c, section 5.2). The PA additionally considered the species-specific RBL estimates for two tree species (quaking aspen and ponderosa pine) that are found in many of these Class I areas and that have a sensitivity to O₃ exposure that places them slightly more sensitive than the median of the group for which robust E–R functions have been established (U.S. EPA, 2014c, sections 5.2 and 5.7). As further summarized in the proposal, the PA describes the results of this analysis, particularly in light of advice from CASAC regarding the significance of the 6% RBL benchmark, as evidence of the occurrence in Class I areas, during periods when the current standard is met, of cumulative seasonal O₃ exposures of a magnitude for which the tree growth impacts indicated by the associated RBL estimates might reasonably be concluded to be important to public welfare (79 FR 75332; U.S. EPA, 2014c, sections 5.2.1 and 5.7).

The proposal also noted that the PA additionally considered findings of the WREA analyses of O₃ effects on tree growth and an array of ecosystem services provided by forests, including timber production, carbon storage and air pollution removal (79 FR 75332–75333; U.S. EPA, 2014b, sections 6.2–6.8; U.S. EPA, 2014c, section 5.2). While recognizing that these analyses provide quantitative estimates of impacts on tree growth and associated services for several different air quality scenarios,

the PA takes note of the large uncertainties associated with these analyses (see U.S. EPA, 2014b, Table 6–27) and the potential for these findings to underestimate the response at the national scale. While noting the potential usefulness of considering predicted and anticipated impacts to these services in assessing the extent to which the current information supports or calls into question the adequacy of the protection afforded by the current standard, the PA also recognizes significant uncertainties associated with the absolute magnitude of the estimates for these ecosystem service endpoints which limited the weight placed on these results (U.S. EPA, 2014c, sections 5.2 and 5.7).

As described in the proposal, the PA also considered O₃ effects on crops, taking note of the extensive and long-standing evidence of the detrimental effect of O₃ on crop production, which continues to be confirmed by evidence newly available in this review (79 FR 75333; U.S. 2014c, sections 5.3 and 5.7). With regard to consideration of the quantitative impacts of O₃ exposures under exposure conditions associated with the current standard, the PA focused on RYL estimates that had strong support in the current evidence (as characterized in the ISA, section 9.6) in light of CASAC comments regarding RYL benchmarks (Frey, 2014c, pp. iii and 14). In considering such evidence-based analyses, as well as the exposure/risk-based information for crops, the PA notes the CASAC comments regarding the use of crop yields as a surrogate for consideration of public welfare impacts, which noted that “[c]rops provide food and fiber services to humans” and that “[e]valuation of market-based welfare effects of O₃ exposure in forestry and agricultural sectors is an appropriate approach to take into account damage that is adverse to public welfare” (Frey, 2014c, p. 10; U.S. EPA, 2014c, section 5.7). The PA additionally notes, however, as recognized in section IV.A.3 above that the determination of the point at which O₃-induced crop yield loss becomes adverse to the public welfare is still unclear, given that crops are heavily managed (*e.g.*, with fertilizer, irrigation) for optimum yields, have their own associated markets and that benefits can be unevenly distributed between producers and consumers (79 FR 75322; U.S. EPA, 2014c, sections 5.3 and 5.7).

With regard to visible foliar injury, as summarized in the proposal, the PA recognizes the long-standing evidence that has established that O₃ causes diagnostic visible foliar injury symptoms on studied bioindicator

species and also recognizes that such O₃-induced impacts have the potential to impact the public welfare in scenic and/or recreational areas, with visible foliar injury associated with important cultural and recreational ecosystem services to the public, such as scenic viewing, wildlife watching, hiking, and camping, that are of significance to the public welfare and enjoyed by millions of Americans every year, generating millions of dollars in economic value (U.S. EPA, 2014b, section 7.1). In addition, several tribes have indicated that many of the O₃-sensitive species (including bioindicator species) are culturally significant (U.S. EPA, 2014c, Table 5–1). Similarly, the PA notes CASAC comments that “visible foliar injury can impact public welfare by damaging or impairing the intended use or service of a resource,” including through “visible damage to ornamental or leafy crops that affects their economic value, yield, or usability; visible damage to plants with special cultural significance; and visible damage to species occurring in natural settings valued for scenic beauty or recreational appeal” (Frey, 2014c, p. 10). Given the above, and taking note of CASAC views, the PA recognizes visible foliar injury as an important O₃ effect which, depending on severity and spatial extent, may reasonably be concluded to be of public welfare significance, especially when occurring in nationally protected areas, such as national parks and other Class I areas.

As summarized in the proposal, the PA additionally takes note of the evidence described in the ISA regarding the role of soil moisture conditions that can decrease the incidence and severity of visible foliar injury under dry conditions (U.S. EPA, 2014c, sections 5.4 and 5.7). As recognized in the PA, this area of uncertainty complicates characterization of the potential for visible foliar injury and its severity or extent of occurrence for given air quality conditions and thus complicates identification of air quality conditions that might be expected to provide a specific level of protection from this effect (U.S. EPA, 2014c, sections 5.4 and 5.7). While noting the uncertainties associated with describing the potential for visible foliar injury and its severity or extent of occurrence for any given air quality conditions, the PA notes the occurrence of O₃-induced visible foliar injury in areas, including federally protected Class I areas that meet the current standard, and suggests it may be appropriate to consider revising the standard for greater protection. In so doing, however, the PA recognizes that

the degree to which O₃-induced visible foliar injury would be judged important and potentially adverse to public welfare is uncertain (U.S. EPA, 2014c, section 5.7).

As noted in the proposal, with regard to other welfare effects, for which the ISA determined a causal or likely causal relationships with O₃ in ambient air, such as alteration of ecosystem water cycling and changes in climate, the PA concludes there are limitations in the available information that affect our ability to consider potential impacts of air quality conditions associated with the current standard.

Based on the considerations described in the PA, summarized in the proposal and outlined here, the PA concludes that the currently available evidence and exposure/risk information call into question the adequacy of the public welfare protection provided by the current standard and provide support for considering potential alternative standards to provide increased public welfare protection, especially for sensitive vegetation and ecosystems in federally protected Class I and similarly protected areas. In this conclusion, staff gives particular weight to the evidence indicating the occurrence in Class I areas that meet the current standard of cumulative seasonal O₃ exposures associated with estimates of tree growth impacts of a magnitude that may reasonably be considered important to public welfare.

b. CASAC Advice

The proposal also summarized advice offered by the CASAC in the current review, based on the updated scientific and technical record since the 2008 rulemaking. The CASAC stated that it “[supports] the conclusion in the Second Draft PA that the current secondary standard is not adequate to protect against current and anticipated welfare effects of ozone on vegetation” (Frey, 2014c, p. iii) and that the PA “clearly demonstrates that ozone-induced injury may occur in areas that meet the current standard” (Frey, 2014c, p. 12). The CASAC further stated “[w]e support the EPA’s continued emphasis on Class I and other protected areas” (Frey, 2014c, p. 9). Additionally, the CASAC indicated support for the concept of ecosystem services “as part of the scope of characterizing damage that is adverse to public welfare” and “concur[red] that trees are important from a public welfare perspective because they provide valued services to humans, including aesthetic value, food, fiber, timber, other forest products, habitat, recreational opportunities, climate regulation, erosion control, air

pollution removal, and hydrologic and fire regime stabilization” (Frey, 2014c, p. 9). Similar to comments from CASAC in the last review, and comments on the proposed reconsideration, the current CASAC also endorsed the PA discussions and conclusions on biologically relevant exposure metrics and the focus on the W126 index accumulated over a 12-hour period (8 a.m.–8 p.m.) over the 3-month summation period of a year resulting in the maximum value (Frey, 2014c, p. iii).

In addition, CASAC stated that “relative biomass loss for tree species, crop yield loss, and visible foliar injury are appropriate surrogates for a wide range of damage that is adverse to public welfare,” listing an array of related ecosystem services (Frey, 2014c, p. 10). With respect to RBL for tree species, CASAC states that it is appropriate to identify in the PA “a range of levels of alternative W126-based standards that include levels that aim for not greater than 2% RBL for the median tree species” and that a median tree species RBL of 6% is “unacceptably high” (Frey, 2014c, pp. 13 and 14). With respect to crop yield loss, CASAC points to a benchmark of 5%, stating that a crop RYL for median species over 5% is “unacceptably high” and described crop yield as a surrogate for related services (Frey, 2014c, p. 13).

c. Administrator’s Proposed Conclusions

At the time of proposal, the Administrator took into account the information available in the current review with regard to the nature of O₃-related effects on vegetation and the adequacy of protection provided by the current secondary standard. The Administrator recognized the appropriateness and usefulness of the W126 metric in evaluating O₃ exposures of potential concern for vegetation effects, additionally noting support conveyed by CASAC for such a use for this metric. Further, the Administrator took particular note of (1) the PA analysis of the magnitude of tree seedling growth effects (biomass loss) estimated for different cumulative, seasonal, concentration-weighted exposures in terms of the W126 metric; (2) the monitoring analysis in the PA of cumulative exposures (in terms of W126 index) occurring in locations where the current standard is met, including those locations in or near Class I areas, and associated estimates of tree seedling growth effects; and (3) the analyses in the WREA illustrating the geographic distribution of tree species for which E–R functions are available and estimates of O₃-related growth impacts for

different air quality scenarios, taking into account the identified potential for the WREA’s existing standard scenario to underestimate the highest W126-based O₃ values that would be expected to occur.

With regard to considering the adequacy of public welfare protection provided by the current secondary standard at the time of proposal, the Administrator focused first on welfare effects related to reduced native plant growth and productivity in terrestrial systems, taking note of the following: (a) The ISA conclusion of a causal relationship between O₃ in the ambient air and these welfare effects, and supporting evidence related to O₃ effects on vegetation growth and productivity, including the evidence from OTC studies of tree seedling growth that support robust E–R functions for 11 species; (b) the evidence, described in section IV.D.1 of the proposal and summarized above, of the occurrence of cumulative seasonal O₃ exposures for which median species RBL estimates are of a magnitude that CASAC has termed “unacceptably high” in Class I areas during periods where the current standard is met; (c) actions taken by Congress to establish public lands that are set aside for specific uses intended to provide benefits to the public welfare, including lands that are to be protected so as to conserve the scenic value and the natural vegetation and wildlife within such areas for the enjoyment of future generations, such as national parks and forests, wildlife refuges, and wilderness areas (many of which have been designated Class I areas); and (d) PA conclusions that the current information calls into question the adequacy of the current standard, based particularly on impacts on tree growth (and the potential for associated ecosystem effects), estimated for Class I area conditions meeting the current standard, that are reasonably concluded to be important from a public welfare standpoint in terms of both the magnitude of the vegetation effects and the significance to public welfare of such effects in such areas.

At the time of proposal, the Administrator also recognized the causal relationships between O₃ in the ambient air and visible foliar injury, reduced yield and quality of agricultural crops, and alteration of below-ground biogeochemical cycles associated with effects on growth and productivity. As to visible foliar injury, she took note of the complexities and limitations in the evidence base regarding characterizing air quality conditions with respect to the magnitude and extent of risk for visible foliar injury, and she

additionally recognized the challenges of associated judgments with regard to adversity of such effects to public welfare. In taking note of the conclusions with regard to crops, she recognized the complexity of considering adverse O₃ impacts to public welfare due to the heavy management common for achieving optimum yields and market factors that influence associated services and additionally took note of the PA conclusions that placing emphasis on the protection afforded to trees inherently also recognizes a level of protection afforded for crops.

Based on her consideration of the conclusions in the PA, and with particular weight given to PA findings pertaining to tree growth-related effects, as well as with consideration of CASAC’s conclusion that the current standard is not adequate, the Administrator proposed to conclude that the current standard is not requisite to protect public welfare from known or anticipated adverse effects and that revision is needed to provide the requisite public welfare protection, especially for sensitive vegetation and ecosystems in federally protected Class I areas and in other areas providing similar public welfare benefits. The Administrator further concluded that the scientific evidence and quantitative analyses on tree growth-related effects provide strong support for consideration of alternative standards that would provide increased public welfare protection beyond that afforded by the current O₃ secondary standard. She further noted that a revised standard would provide increased protection for other growth-related effects, including for carbon storage and for areas for which it is more difficult to determine public welfare significance, as recognized in section IV.B.2 of the proposal, as well as other welfare effects of O₃, including visible foliar injury and crop yield loss.

2. Comments on the Need for Revision

In considering comments on the need for revision, we first note the advice and recommendations from CASAC with regard to the adequacy of the current standard. In its review of the second draft PA, CASAC stated that it “supports the scientific conclusion in the Second Draft PA that the current secondary standard is not adequate to protect against current and anticipated welfare effects of ozone on vegetation” (Frey, 2014c).

General comments received from the public on the proposal that are based on relevant factors and either supported or opposed the proposed decision to revise

the current O₃ secondary standard are addressed in this section. Comments on specific issues or information that relate to consideration of the appropriate elements of a revised secondary standard are addressed below in section IV.C. Other specific comments related to standard setting, as well as general comments based on implementation-related factors that are not a permissible basis for considering the need to revise the current standard, are addressed in the Response to Comments document.

Public comments on the proposal were divided with regard to support for the Administrator's proposed decision to revise the current secondary standard. Many state and local environmental agencies or government bodies, tribal agencies and organizations, and environmental organizations agreed with the EPA's proposed conclusion on the need to revise the current standard, stating that the available scientific information shows that O₃-induced vegetation and ecosystem effects are occurring under air quality conditions allowed by the current standard and, therefore, provides a strong basis and support for the conclusion that the current secondary standard is not adequate. In support of their view, these commenters relied on the entire body of evidence available for consideration in this review, including evidence assessed previously in the 2008 review. These commenters variously pointed to the information and analyses in the PA and the conclusions and recommendations of CASAC as providing a clear basis for concluding that the current standard does not provide adequate protection of public welfare from O₃-related effects. Many of these commenters generally noted their agreement with the rationale provided in the proposal with regard to the Administrator's proposed conclusion on adequacy of the current standard, and some gave additional emphasis to several aspects of that rationale, including the appropriateness of the EPA's attention to sensitive vegetation and ecosystems in Class I areas and other public lands that provide similar public welfare benefits and of the EPA's reliance on the strong evidence of impacts to tree growth and growth-related effects.

Comments from tribal organizations additionally noted that many Class I areas are of sacred value to tribes or provide treaty-protected benefits to tribes, including the exercise of gathering rights. Tribal organizations also noted the presence in Class I areas of large numbers of culturally important plant species, which they indicate to be impacted by air quality conditions

allowed by the current standard. The impacts described include visible foliar injury, loss in forest growth and crop yield loss, which these groups describe as especially concerning when occurring on lands set aside for the benefit of the public or that are of sacred value to tribes or provide treaty-protected benefits to tribes.

As described in section IV.B.3 below, the EPA generally agrees with the view of these commenters regarding the need for revision of the current secondary standard and with CASAC that the evidence provides support for the conclusions that the current secondary standard is not adequate to protect public welfare from known or anticipated adverse effects, particularly with respect to effects on vegetation.

A number of industries, industry associations, or industry consultants, as well as some state governors, attorneys general and environmental agencies, disagreed with the EPA's proposed conclusion on the adequacy of the current standard and recommended against revision. In support of their position, these commenters variously stated that the available evidence is little changed from that available at the time of the 2008 decision, and that the evidence is too uncertain, including with regard to growth-related effects and visible foliar injury, to support revision, and does not demonstrate adverse effects to public welfare for conditions associated with the current standard, with some commenters stating particularly that the EPA analysis of Class I areas did not document adverse effects to public welfare. They also cited the WREA modeling analyses as indicating that any welfare improvements associated with a revised standard would be marginal; in particular, compared to the benefits of achieving the current standard. Further, they state that, because of long-range transport of O₃ and precursors, it is not appropriate for the EPA to draw conclusions about the level of protection offered by the current standard based on current air quality conditions; in support of this view, these commenters point to different modeling analyses as demonstrating that under conditions where the current standard is met throughout the U.S., the associated W126 values would all be below the upper end of the range proposed as providing requisite public welfare protection and nearly all below the lower end of 13 ppm-hrs.

As an initial matter, we note that, as noted in sections I.C and IV.A above, the EPA's 2008 decision on the secondary standard was remanded back to the Agency because in setting the

2008 secondary standard, the EPA failed to specify what level of air quality was requisite to protect public welfare from known or anticipated adverse effects or explain why any such level would be requisite. So, in addressing the court remand, the EPA has more explicitly considered the extent to which protection is provided from known or anticipated effects that the Administrator may judge to be adverse to public welfare, and has described how the air quality associated with the revised standard would provide requisite public welfare protection, consistent with CAA section 109(b)(2) and the court's decision remanding the 2008 secondary standard. In undertaking this review, consistent with the direction of the CAA, the EPA has considered the current air quality criteria.

While we recognize, as stated in the proposal, that the evidence newly available in this review is largely consistent with the evidence available at the time of the last review (completed in 2008) with regard to the welfare effects of O₃, we disagree with the commenters' interpretations of the evidence and analyses available in this review and with their views on the associated uncertainties. As summarized in section IV.A above, the ISA has determined causal relationships to exist between several vegetation and ecosystem endpoints and O₃ in ambient air (U.S. 2013, section 9.7). The ISA characterized the newly available evidence as largely consistent with and supportive of prior conclusions, as summarized in section IV.A above. This is not to say, however, that there is no newly available evidence and information in this review or that it is identical to that available in the last review. In some respects, the newly available evidence has strengthened the evidence available in the last review and reduced important uncertainties. As summarized in section IV.A.1.b above, newly available field studies confirm the cumulative effects and effects on forest community composition over multiple seasons. Additionally, among the newly available evidence for this review are analyses documented in the ISA that evaluate the RBL and RYL E-R functions for aspen and soybean, respectively, with experimental datasets that were not used in the derivation of the functions (U.S. 2013, section 9.6.3). These evaluations confirm the pertinence of the tree seedling RBL estimates for aspen, a species with sensitivity roughly midway in the range of sensitivities for the studied species, across multiple years in older trees.

With regard to crops, the ISA evaluations demonstrate a robustness of the E-R functions to predict O₃-attributable RYL and confirm the relevance of the crop RYL estimates for more recent cultivars currently growing in the field. Together, the information newly available in this review confirms the basis for the E-R functions and strengthens our confidence in interpretations drawn from their use in other analyses newly available in this review that have been described in the WREA and PA.

With regard to comments on uncertainties associated with estimates of RBL, we first note that these established, robust E-R functions, which the EPA gave particular emphasis in this review, are available for seedling growth for 11 tree species native to the U.S., as summarized in section IV.A.1.b above and described in the proposal. These E-R functions are based on studies of multiple genotypes of 11 tree species grown for up to three years in multiple locations across the U.S. (U.S. EPA, 2013, section 9.6.1). We have recognized the uncertainty regarding the extent to which the studied species encompass the O₃ sensitive species in the U.S. and also the extent to which they represent U.S. vegetation as a whole (U.S. EPA, 2014b, section 6.9). However, the studied species include both deciduous and coniferous trees with a wide range of sensitivities and species native to every region across the U.S. and in most cases are resident across multiple states and NOAA climatic regions (U.S. EPA, 2014b, Appendix 6A). While the CASAC stated that there is “considerable uncertainty in extrapolating from the [studied] forest tree species to all forest tree species in the U.S.,” it additionally expressed the view that it should be anticipated that there are highly sensitive vegetation species for which we do not have E-R functions and others that are insensitive.¹⁸⁷ In so doing, the CASAC stated that it “should not be assumed that species of unknown sensitivity are tolerant to ozone” and “[i]t is more appropriate to assume that the sensitivity of species without E-R functions might be similar to the range of sensitivity for those species with E-R functions” (Frey, 2014c, p. 11). Accordingly, we disagree with commenters’ view that effects on these species are not appropriate

¹⁸⁷ Use of RBL estimates in the proposal, and in this final decision, focuses on the RBL for the studied species as a surrogate for a broad array of growth-related effects of potential public welfare significance, consistent with the CASAC advice.

considerations for evaluation of the adequacy of the current standard.

In support of their view that RBL estimates are too uncertain to inform a conclusion that the current standard is not adequately protective of public welfare, some commenters state that some of the 11 E-R functions are based on as few as one study. The EPA agrees that there are two species for which there is only one study supporting the E-R function (Virginia pine and red maple). We also note, however, that those two species are appreciably less sensitive than the median (Lee and Hogsett, 1996; U.S. EPA, 2014c, Table 5C-1). Thus, in the relevant analyses, they tend to influence the median toward a relatively less (rather than more) sensitive response. Further, there are four species for which the E-R functions are based on more than five studies,¹⁸⁸ contrary to the commenters’ claims of there being no functions supported by that many studies. That said, the EPA has noted the relatively greater uncertainty in the species for which fewer studies are available, and it is in consideration of such uncertainties that the EPA focused in the proposal on the median E-R function across the 11 species, rather than a function for a species much more (or less) sensitive than the median. The EPA additionally notes that it gave less emphasis to the E-R function available for one species, eastern cottonwood, based on CASAC advice that the study results supporting that E-R function were not as strong as the results of the other experiments that support the other, robust E-R functions and that the eastern cottonwood study results showed extreme sensitivity to O₃ compared to other studies (Frey, 2014c, p. 10). Accordingly, the EPA has appropriately considered the strength of the scientific evidence and the associated uncertainties in considering revision of the secondary standard.

Other commenters stated that the scientific evidence does not support revising the NAAQS, pointing to uncertainty related to interpretation of the RBL estimates (based on tree seedling studies) with regard to effects on older tree lifestages. Some of these commenters’ claim that mature canopy trees experience reduced O₃ effects. The EPA agrees that the quantitative information for O₃ growth effects on older tree lifestages is available for a more limited set of species than that available for tree seedlings. We note,

¹⁸⁸ These four species, aspen, Douglas fir, ponderosa pine and red alder, range broadly in sensitivities that fall above, below and at the median for the 11 species (Lee and Hogsett, 1996; U.S. EPA, 2014c, Table 5C-1).

however, that this is an area for which there is information newly available in this review. A detailed analysis of study data for seedlings and older lifestages of aspen shows close agreement between the O₃-attributable reduced growth observed in the older trees and reductions predicted from the seedling E-R function (U.S. EPA, 2013, section 9.6.3.2; discussed in the PA, section 5.2.1 as noted in the proposal, p. 75330). This finding, newly available in this review and documenting impacts on mature trees, improves our confidence in conclusions drawn with regard to the significance of RBL estimates for this species, which is prevalent across multiple regions of the U.S.¹⁸⁹ It is also noteworthy that this species is generally more sensitive to O₃ effects on growth than the median of the 11 species with robust E-R functions (as shown in U.S. EPA 2014c, Table 5C-1). Other newly available studies, summarized in section IV.A.1.b above and section IV.B.1.b of the proposal, provide additional evidence of O₃ impacts on mature trees, including a meta-analysis reporting older trees to be more affected by O₃ than younger trees (U.S. EPA, 2013, p. 9-42; Wittig et al., 2007). We additionally note that CASAC “concur[red] that biomass loss in trees is a relevant surrogate for damage to tree growth that affects ecosystem services such as habitat provision for wildlife, carbon storage, provision of food and fiber, and pollution removal” additionally stating that “[b]iomass loss may also have indirect process-related effects such as on nutrient and hydrologic cycles” leading them to conclude that “[t]herefore, biomass loss is a scientifically valid surrogate of a variety of adverse effects to public welfare” (Frey, 2014c, p. 10).

As noted in section IV.A above and discussed below, the Administrator’s final decision on the adequacy of the current standard draws upon, among other things, the available evidence and quantitative analyses as well as judgments about the appropriate weight to place on the range of uncertainties inherent in the evidence and analyses. The strengthening in this review, as compared with the last review, of the basis for the robust E-R functions for tree seedling RBL, as well as other newly available quantitative analyses,

¹⁸⁹ The WREA notes a few additional, limited analyses using modeling tools and data from previous publications that indicate there may be species-specific differences in the extent of similarities between seedling and adult growth response to O₃, with some species showing greater and some lesser response for seedlings as compared to mature tree, but a general comparability (U.S. EPA 2014b, section 6.2.1.1 and p. 6-67).

will, accordingly, contribute to judgments made by the Administrator with regard to these effects in reaching her final decisions in this review.

Amongst the newly available information in this review is a new analysis describing W126-based exposures occurring in counties containing Class I areas for which monitoring data indicated compliance with the current standard. The PA gave particular attention to this analysis in consideration of the adequacy of the current standard, and this analysis was also described in the proposal (U.S. EPA, 2014c, Appendix 5B and pp. 5–27 to 5–29; 79 FR 75331–75332, December 17, 2014). Some of the commenters who disagreed with the EPA’s conclusion on adequacy of the current standard variously stated that this analysis does not demonstrate growth effects are occurring in Class I areas and that the analysis is too uncertain for reliance on by the Administrator in her judgment on adequacy of the current standard. While the EPA agrees with commenters that data on the occurrence of growth effects in the areas and time periods identified are not part of this analysis, we note that this is because such data have not been collected and consequently cannot be included. As a result, the EPA has utilized measurements of O₃ in or near these areas in combination with the established E–R functions to estimate the potential for growth impacts in these areas under conditions where the current standard is met. The EPA additionally notes that species for which E–R functions have been developed have been documented to occur within these areas (see Table 3).

The EPA disagrees with commenters regarding the appropriateness of this analysis for the Administrator’s consideration. This analysis documents the occurrence of cumulative growing

season exposures in these ecosystems which the EPA and CASAC have interpreted, through the use of the established E–R functions for tree seedling growth effects summarized in section IV.A.1.b above (and described in the ISA, PA and proposal), as indicating the potential for growth effects of significance in these protected areas. To the extent that these comments imply that the Administrator may only consider welfare effects that are certain in judging the adequacy of the current standard, we note that section 109(b)(2) of the CAA plainly provides for consideration of both known and anticipated adverse effects in establishing or revising secondary NAAQS.

In support of some commenters’ view that this analysis is too uncertain to provide a basis for the Administrator’s proposed conclusion that the current standard is not adequate, one commenter observed that the O₃ monitors used for six of the 22 Class I areas in the analysis, although in the same county, were sited outside of the Class I areas. This was the case due to the analysis being focused on the highest monitor in the county that met the current standard. To clarify the presentation, however, we have refocused the presentation, restricting it to data for monitors sited in or within 15 kilometers of a Class I area,¹⁹⁰ and note that the results are little changed, continuing to call into question the adequacy of the current standard. As shown in Table 3, the dataset in the refocused presentation, which now spans 1998 up through 2013, includes 17 Class I areas for which monitors were identified in this manner. For context, we note that this represents nearly a quarter of the Class I areas for which there are O₃ monitors within 15 km.¹⁹¹

In recognition of the influence that other environmental factors can exert in the natural environment on the relationship between ambient O₃ exposures and RBL, potentially modifying the impact predicted by the E–R functions, the PA and proposal took particular note of the occurrence of 3-year average W126 index values at or above 19 ppm-hrs. In the re-focused analysis in Table 3, there are 11 areas, distributed across four states in two NOAA climatic regions, for which the 3-year W126 exposure index values ranged at or above 19 ppm-hrs, a value for which the corresponding median species RBL estimate for a growing season’s exposure is 6%, a magnitude termed “unacceptably high” by CASAC (Frey, 2014c, p. 13). The highest 3-year W126 index values in these 11 areas ranged from 19.0 up to 22.2 ppm-hrs, a cumulative seasonal exposure for which the median species RBL estimate is 9% for a single growing season. The annual W126 index values range above 19 ppm-hrs in 15 of the areas in the re-focused table provided here; these areas are distributed across six states (AZ, CA, CO, KY, SD, UT) and four regions (West, Southwest, West North Central and Central).¹⁹² The highest index values in the areas with annual index values above 19 ppm-hrs range from 19.1 to 26.9 ppm-hrs. As is to be expected from the focus on a smaller dataset, the number of states with 1-year W126 index values above 19 ppm-hrs is smaller in the refocused analysis (15 as compared to 20), although the number of regions affected is the same. More importantly, however, the number of areas with 3-year W126 index values at or above 19 ppm-hrs is the same, 11 Class I areas across two regions, supporting the prior conclusions.

TABLE 3—O₃ CONCENTRATIONS FOR CLASS I AREAS DURING PERIOD FROM 1998 TO 2013 THAT MET THE CURRENT STANDARD AND WHERE 3-YEAR AVERAGE W126 INDEX VALUE WAS AT OR ABOVE 15 ppm-hrs

Class I area (distance away, if monitor is not at/ within boundaries)	State/ County	Design value (ppb)*	3-Year average W126 (ppm-hrs)* (# ≥ 19 ppm-hrs, range)	Annual W126 (ppm-hrs)* (# ≥ 19 ppm-hrs, range)	Number of 3-year periods
Bridger Wilderness Area ^{QA, DF} (8.9 km).	WY/Sublette	70–72	16.2–17.0	13.9–18.8	4
Canyonlands National Park ^{QA, DF, PP} .	UT/San Juan	70–73	15.4–19.5 (2, 19.1–19.5)	9.6–23.6 (4, 19.2–23.6)	8
Chiricahua National Monument ^{DF, PP} (12 km).	AZ/Cochise	69–73	15.2–19.8 (1, 19.8)	11.7–21.9 (2, 19.8–21.9)	10
Grand Canyon National Park ^{QA, DF, PP} .	AZ/Coconino	68–74	15.3–22.2 (7, 19.1–22.2)	10.1–26.9 (6, 19.8–26.9)	12
Desolation Wilderness ^{PP} (3.9 km) ..	CA/El Dorado	75	19.8 (1, 19.8)	15.6–22.9 (2, 21.0–22.9)	1

¹⁹⁰ The 15 km distance was selected as a natural breakpoint in distance of O₃ monitoring sites from Class I areas and as still providing similar surroundings to those occurring in the Class I area. We note that given the strict restrictions on

structures and access within some of these areas, it is common for monitors intended to collect data pertaining to air quality in these types of areas to be sited outside their boundaries.

¹⁹¹ There is an O₃ monitor within fewer than 15% of all Class I areas, and fewer than half of all Class I areas have a monitor within 15 km.

¹⁹² This compares to 20 areas in eight states and four regions in the earlier analysis.

TABLE 3—O₃ CONCENTRATIONS FOR CLASS I AREAS DURING PERIOD FROM 1998 TO 2013 THAT MET THE CURRENT STANDARD AND WHERE 3-YEAR AVERAGE W126 INDEX VALUE WAS AT OR ABOVE 15 ppm-hrs—Continued

Class I area (distance away, if monitor is not at/ within boundaries)	State/ County	Design value (ppb)*	3-Year average W126 (ppm-hrs)* (# ≥ 19 ppm-hrs, range)	Annual W126 (ppm-hrs)* (# ≥ 19 ppm-hrs, range)	Number of 3-year periods
Lassen Volcanic National Park DF, PP	CA/Shasta	72–74	15.3–15.6	11.5–19.1 (1, 19.1)	2
Mammoth Cave National Park BC, C, LP, RM, SM, VP, YP (0.1 km).	KY/Edmonson	74	15.7	12.3–22.0 (1, 22.0)	1
Maroon Bells-Snowmass Wilderness Area QA, DF (0.8 km).	CO/Gunnison	68–73	15.6–20.2 (1, 20.2)	13.0–23.8 (3, 21.3–23.8)	8
Mazatzal Wilderness DF, PP (10.9 km).	AZ/Maricopa	74–75	17.8–19.9 (1, 19.9)	10.3–26.2 (3, 19.7–26.2)	2
Mesa Verde National Park DF	CO/Montezuma	67–73	15.4–20.7 (1, 20.7)	10.7–23.4 (4, 19.5–23.4)	11
Petrified Forest National Park C	AZ/Navajo	70	15.4–16.9	12.7–18.6	2
Rocky Mountain National Park QA, DF, PP (0.9 km).	CO/Larimer	73–74	15.3–18.4	8.3–26.2 (4, 19.4–26.2)	5
Saguaro National Park DF, PP (0.1 km)**.	AZ/Pima	69–74	15.4–19.0 (1, 19.0)	7.3–22.9 (3, 19.6–22.9)	6
Superstition Wilderness Area PP (6.3, 14.9 km and 7.2 km)**.	AZ/Gila	72–75	16.6–20.9 (2, 19.0–20.9)	13.8–25.5 (4, 19.0–25.5)	5
	AZ/Maricopa	70–75	15–20.2 (1, 20.2)	6.3–23.9 (4, 19.6–23.9)	4
	AZ/Pinal	72–75	15.3–21.1 (1, 21.1)	10.2–24.7 (4, 21.4–24.7)	7
Weminuche Wilderness Area QA, DF, PP (14.9 km).	CO/La Plata	70–74	15.1–19.1 (1, 19.1)	10.8–21.0 (2, 20.8–21.0)	6
Wind Cave National Park QA, PP	SD/Custer	70	15.4	12.3–20.5 (1, 20.5)	1
Zion National Park QA, DF, PP (3.6 km).	UT/Washington	70–73	17.0–20.1 (2, 19.4–20.1)	14.2–23.2 (3, 19.8–23.2)	6

* Based on hourly O₃ concentration data retrieved from AQS on June 25, 2014, and additional CASTNET data downloaded from http://java.epa.gov/castnet/epa_jsp/prepackageddata.jsp on June 25, 2014. Design values shown above are derived in accordance with Appendix P to 40 CFR Part 50. Annual W126 index values are derived as described in section IV.A.1 above; three consecutive year annual values are averaged for 3-year averages. Prior to presentation, both types of W126 index values are rounded to one decimal place. The full list of monitoring site identifiers and individual statistics is available in the docket for this rulemaking.

** No monitor was sited within these Areas and multiple monitors were sited within 15 km. Data for the closest monitor per county are presented.

Superscript letters refer to species present for which E–R functions have been developed. QA=Quaking Aspen, BC=Black Cherry, C=Cottonwood, DF=Douglas Fir, LP=Loblolly Pine, PP=Ponderosa Pine, RM=Red Maple, SM=Sugar Maple, VP=Virginia Pine, YP=Yellow (Tulip) Poplar. Sources include USDA–NRCS (2014, <http://plants.usda.gov>), USDA–FS (2014, <http://www.fs.fed.us/foresthealth/technology/nidrm2012.shtml>) UM–CFCWI (2014, <http://www.wilderness.net/printFactSheet.cfm?WID=583>), NPS (<http://www.nps.gov/pefo/planyourvisit/upload/Common-Plants-Site-Bulletin-sb-2013.pdf>) and Phillips and Comus (2000).

As support for their view that the Class I area analysis is too uncertain to provide a basis for the Administrator’s proposed conclusion that the current standard is not adequate, some commenters stated that forests in Class I areas were composed of mature trees and that the tree seedling E–R functions do not predict growth impacts in mature forests. The EPA disagrees with the commenters’ statement that Class I areas are only made up of mature trees. Seedlings exist throughout forests as part of the natural process of replacing aging trees and overstory trees affected by periodic disturbances.¹⁹³ Seedlings also tend to occur in areas affected by natural disturbances, such as fires, insect infestations and flooding, and such disturbances are common in many natural forests. As noted above, information newly available in this review strengthens our understanding regarding O₃ effects on mature trees for

aspen, an important and O₃-sensitive species (U.S. EPA, 2013, section 9.6.3.2).

One commenter additionally stated that the EPA has not shown reduced biomass to be adverse to public welfare, variously citing individual studies, most of which are not considering O₃, as support for their view that such an effect of O₃ may not occur in the environment and may be of no significance if it does. With regard to the occurrence of O₃-related reduced growth in the field, we note the strength of the evidence from field OTC studies on which the E–R functions are based, and evidence from comparative studies with open-air chamberless control treatments suggests that characteristics particular to the OTC did not significantly affect plant response (U.S. EPA, 2013, p. 9–5). Thus, we view the OTC systems as combining aspects of controlled exposure systems with field conditions to facilitate a study providing data that represent the role of the studied pollutant in a natural system.

Further, we disagree with the commenters on the significance of O₃-

attributable reduced growth in natural ecosystems. Even in the circumstances cited by the commenter (e.g., subsequent to large-scale disturbances, nutrient limited system, multigeneration exposure), O₃ can affect growth of seedlings and older trees, with the potential for effects on ecosystem productivity, handicapping the sensitive species and affecting community dynamics and associated community composition, as well as ecosystem hydrologic cycles (U.S. EPA, 2013, p. 1–8). For example, two recent studies report on the role of O₃ exposure in affecting water use in a mixed deciduous forest and indicated that O₃ increased water use in the forest and also reduced growth rate (U.S. EPA, 2013, p. 9–43, McLaughlin, 2007a, 2007b). Contrary to the lesser effects implied by the commenters, the authors of these two studies noted implications of their findings with regard to the potential for effects to be amplified under conditions of increased temperature and associated reduced water availability (McLaughlin, 2007a). We additionally note comments from

¹⁹³ Basic information on forest processes, including the role of seedlings is available at: http://www.na.fs.fed.us/stewardship/pubs/NE_forest_regeneration_handbook_revision_130829_desktop.pdf.

the CASAC, summarized above, in which it concurs with a focus on biomass loss and the use of RBL estimates, calling biomass loss in trees a “relevant surrogate for damage to tree growth” that affects an array of ecosystem services (Frey, 2014c, p. 10), and identifies 6% RBL as “unacceptably high” (Frey, 2014c, p. 13). The evidence we presented includes evidence related to RBL estimates above that benchmark. Thus, while we agree that some reductions in tree growth may not be concluded to be adverse to public welfare, we disagree with commenters that we have not presented the evidence, which includes RBL estimates well above the 6% magnitude identified by CASAC, that supports the Administrator’s judgments on adversity that may be indicated by such estimates and her conclusion that adequate protection is not provided by the current standard, as described in section IV.B.3 below.

Some commenters disagree with the EPA’s consideration of the Class I areas analysis, stating that it is not appropriate for the EPA to evaluate the level of protection offered by the current primary O₃ standard under current conditions due to the long-range transport of O₃ and O₃ precursors to Class I areas from upwind non-attainment areas. It is the view of these commenters that once the upwind areas make emissions reductions to attain the current standard, downwind areas will see improvements in air quality and decreasing W126 levels. In support of this view, commenters point to several modeling analyses. Some commenters point to air quality modeling conducted by an environmental consultant that projects all sites to have W126 index values below 13 ppm-hrs when emissions are adjusted such that all upwind monitors are modeled to meet the current standard. Detailed methodology, results and references for the commenter’s modeling analysis were not provided, precluding a thorough evaluation and comparison to the EPA’s modeling. While the EPA agrees that transport of O₃ and O₃ precursors can affect downwind monitors, we disagree with commenters regarding the conclusions that are appropriate to draw from modeling simulations for the reasons noted below.

As support for their view that the current standard provides adequate protection, some commenters pointed to estimates drawn from the EPA’s air quality modeling performed for the RIA, stating that this modeling for an alternative standard level of 70 ppb indicates “only a handful” of monitoring sites approaching as high as

13 ppm-hrs as a 3-year average (*e.g.*, UARG, p. 76). These commenters further point to the WREA modeling, noting that those estimates project that attainment of the current standard would result in only 5 sites above 15 ppm-hrs. Based on these statements, these commenters state that the current standard is likely to provide conditions with no site having a monitor over 17 ppm-hrs and a “minimal number” likely exceeding 13 ppm-hrs (*e.g.*, UARG, p. 77). We disagree with commenters’ interpretation of the modeling information from the two different assessments. As we summarized in section IV.C.1 of the proposal with regard to the WREA modeling, the modeling estimates are each based on a single set of precursor emissions reductions that are estimated to achieve the desired target conditions, which is also the case for the RIA modeling¹⁹⁴ (U.S. EPA, 2014c, pp. 5–40 to 5–41; see also section 1.2.2 of the 2014 RIA).

As noted in section IV.A.2 above, and in the proposal, the model-adjusted air quality in the WREA scenario for the current standard does not represent an optimized control scenario that just meets the current standard, but rather characterizes one potential distribution of air quality across a region when all monitor locations meet the standard (79 FR 75322; U.S. EPA, 2014b, section 4.3.4.2). Alternate precursor emissions reductions would be expected to produce different patterns of O₃ concentrations and associated differences in W126 index values. Specifically, the precursor emissions reductions scenarios examined in the WREA focuses on regional reductions over broad areas rather than localized cuts that may focus more narrowly on areas violating the current standard (U.S. EPA, 2014b, p. 4–35). The assumption of regionally determined across-the-board emissions reductions is a source of potential uncertainty with the potential to overestimate W126 scenario benefits (U.S. EPA, 2014b, Table 4–5 [row G]). The application of emissions reductions to all locations in each region to bring down the highest monitor in the region to meet the

¹⁹⁴ Although commenters cite to both analyses as if providing the same information, there are many differences in specific aspects of the RIA approach from that of the WREA, which derive, at least in part, from their very different purposes. The RIA is not developed for consideration in the NAAQS review. Rather, it is intended to provide insights and analysis of an illustrative control strategy that states might adopt to meet the revised standard. The EPA does not consider this analysis informative to consideration of the protection provided by the current standard, and the results of the RIA have not been considered in the EPA’s decisions on the O₃ standards.

current standard could potentially lead to W126 index underestimates at some locations, as noted in the WREA: “[w]hile the scenarios implemented in this analysis show that [] bringing down the highest monitor in a region would lead to reductions below the targeted level through the rest of the region, to the extent that the regional reductions from on-the-books controls are supplemented with more local controls the additional benefit may be overestimated” (U.S. EPA, 2014b, p. 4–36; U.S. EPA, 2014c, pp. 5–40 to 5–41). This point was emphasized by CASAC in their comments on the 2nd draft WREA. CASAC noted that, “[m]eeting a target level at the highest monitor requires substantial reductions below the targeted level through the rest of the region” and stated that “[t]his artificial simulation does not represent an actual control strategy and may conflate differences in control strategies required to meet different standards” (Frey, 2014b, p. 2).

Due to the uncertainty about what actual future emissions control strategies might be and their associated emissions reductions, and the impact such uncertainty might have on modeling estimates involving reductions from recent conditions, we believe it is important to place weight on ambient air monitoring data for recent conditions in drawing conclusions regarding W126 index values that would be expected in areas that meet the current standard. The analysis of air quality data for Class I areas described in the proposal, and updated in Table 3 above (1998–2013), indicates the occurrence of 3-year W126 exposure index values well above 19 ppm-hrs, a cumulative exposure value for which CASAC termed the associated median RBL estimate “unacceptably high,” in multiple Class I areas that meet the current standard (79 FR 75312, December 17, 2014, Table 7; updated in Table 3 above). Additionally, analysis of recent air quality data (2011–2013) for all locations across the U.S. indicates 10 monitor locations distributed across two NOAA climatic regions that meet the current standard and at which 3-year W126 index values are above 19 ppm-hrs, with the highest values extending up to 23 ppm-hrs (Wells, 2015b).

In support of their view that the EPA’s modeling supports the conclusion that W126 index values of interest are achieved under the current secondary standard, some commenters additionally state that the W126 values in the WREA are overestimated in unmonitored rural areas due to the much greater prevalence of urban monitors across the U.S. The EPA

disagrees with this conclusion. In order to estimate O₃ concentrations in grid cells across a national-scale spatial surface, the WREA applied the VNA spatial interpolation technique after applying the HDDM technique to adjust O₃ concentrations at monitoring sites based on the emissions reductions necessary to just meet the current standard. In estimating concentrations in unmonitored areas, the VNA method considers only the “neighboring” monitors, using an inverse distance squared weighting formula, which assigns the greatest influence to the nearest neighboring monitor (U.S. EPA, 2014b, p. 4A–6). By this approach, monitors in less-densely monitored areas contribute to the concentration estimates over much larger areas than do monitors in more-densely monitored areas. In an urban area, neighboring monitors may be quite close to one another, such that any one monitor may only be influencing concentration estimates for a handful of spatial grid cells in the immediate vicinity. By contrast, monitors in rural areas may influence hundreds of grid cells. A specific example of this is the monitor in Great Basin National Park in eastern Nevada. The VNA algorithm assigns very high weights to this monitor for all of the grid cells covering a 100 km radius around it, simply because there are no other monitors in that area and it is the closest. On the other hand, a monitor near downtown Las Vegas may only get a high weight for, and thus exert influence on the concentration estimate in, the one grid cell containing it. We agree with the commenter that urban monitors may influence the spatial surface for some distance away from the urban areas, although the influence wanes with increasing distance from that area and decreasing distance to the next closest monitor. As we lack data for the intervening locations, however, we have no reason to conclude that the VNA surface is overestimating the W126 index values. Further, as was summarized in section IV.A.2 above, and in the WREA, the PA and the proposal (U.S. EPA, 2014b, Table 6–27, section 8.5; U.S. EPA, 2014c, p. 5–49; 79 FR 75323, December 17, 2014), the VNA approach results in a lowering of the highest W126 index values at monitoring sites, which contributes to underestimates of the highest W126 index values in each region.

In support of their view that the current standard is adequate, some industry commenters additionally cite WREA analyses for the current standard scenario, including the W126 index

estimates in national parks, as showing that the current standard provides more than adequate protection, with alternative scenarios providing only marginal and increasingly uncertain benefits. As we noted in the proposal and section IV.A.2 above, there are an array of uncertainties associated with the W126 index estimates, in the current standard scenario and in the other scenarios, which, as they are inputs to the vegetation risk analyses, are propagated into those analyses (79 FR 75323; December 17, 2014). As a result, consistent with the approach in the proposal, the Administrator has not based her decision with regard to adequacy of the current standard in this review on these air quality scenario analyses.

In support of their view that the current standard provides adequate protection and should not be revised, some commenters described their concerns with any consideration of visible foliar injury in the decision regarding the secondary standard. These commenters variously stated that visible foliar injury cannot be reliably evaluated for adversity given lack of available information, is not an adverse effect on public welfare that must be addressed through a secondary standard, and is not directly relatable to growth suppression (and the EPA’s use of RBL captures that effect anyway). Additionally, some state that any associated ecosystem services effects are not quantifiable. In sum, the view of these commenters is that it is not appropriate for the Administrator to place any weight on this O₃ effect in determining the adequacy of the current standard. As an initial matter, the EPA agrees with the comment that the current evidence does not include an approach for relating visible foliar injury to growth suppression,¹⁹⁵ as recognized in section IV.A.1.b above. Further, we note that, similar to decisions in past O₃ reviews, the Administrator’s proposed decision in this review recognized the “complexities and limitations in the evidence base regarding characterizing air quality conditions with respect to

¹⁹⁵ The current evidence indicates that “[t]he significance of O₃ injury at the leaf and whole plant levels depends on how much of the total leaf area of the plant has been affected, as well as the plant’s age, size, developmental stage, and degree of functional redundancy among the existing leaf area” and “in some cases, visible foliar symptoms have been correlated with decreased vegetative growth . . . and with impaired reproductive function” (U.S. EPA, 2013, p. 9–39). The ISA concludes, however, “it is not presently possible to determine, with consistency across species and environments, what degree of injury at the leaf level has significance to the vigor of the whole plant” (U.S. EPA, 2013, p. 9–39).

the magnitude and extent of risk for visible foliar injury” and the “challenges of associated judgments with regard to adversity of such effects to public welfare” (79 FR 75336; December 17, 2014). Contrary to the implications of the commenters, although the Administrator took into consideration the potential for adverse effects on public welfare from visible foliar injury, she placed weight primarily on growth-related effects of O₃, both in her proposed decision on adequacy and with regard to proposed judgments on what revisions would be appropriate. Although visible foliar injury may impact the public welfare and accordingly has the potential to be adverse to the public welfare (as noted in section IV.B.2 of the proposal), the Administrator placed less weight on visible foliar injury considerations in identifying what revisions to the standard would be appropriate to propose. In considering these effects for this purpose, she recognized “significant challenges” in light of “the variability and the lack of clear quantitative relationship with other effects on vegetation, as well as the lack of established criteria or objectives that might inform consideration of potential public welfare impacts related to this vegetation effect” (79 FR 75349; December 17, 2014). As summarized in section IV.A.1.a above, the evidence demonstrates a causal relationship of O₃ with visible foliar injury. Accordingly, we note that the uncertainty associated with visible foliar injury is not with regard to whether O₃ causes visible foliar injury. Rather, the uncertainty is, as discussed in sections IV.A.1.b and IV.A.3 above, with the lack of established, quantitative exposure-response functions that document visible foliar injury severity and incidence under varying air quality and environmental conditions and information to support associated judgments on the significance of such responses with regard to associated public welfare impacts. As with the Administrator’s proposed decisions on the standard, such considerations also informed her final decisions, described in sections IV.B.3 and IV.C.3 below.

In support of their view that the current standard should be retained, some commenters note the WREA finding for the current standard scenario of no U.S. counties with RYL estimates at or above 5%, the RYL value emphasized by CASAC and state that policy reasons provide support for not focusing on crops in the decision; other commenters state that additional studies on crops and air quality are needed. As

described previously in this section, and in section IV.A.2 above, an aspect of uncertainties associated with the WREA air quality scenarios, including the current standard scenario, is underestimation of the highest W126 index values, contributing to underestimates in the effects associated with the current standard scenario. The EPA agrees with commenters that additional studies on crops and air quality will be useful to future reviews. Additionally, however, as noted above, the Administrator's proposed conclusion on adequacy of the current standard, as well as her final decision described in section IV.B.3 below, gives less weight to consideration of effects on agricultural crops in recognition of the complicating role of heavy management in that area.

Lastly, we note that many commenters cited the costs of compliance as supporting their view that the standard should not be revised, although as we have described in section I.B above, the EPA may not consider the costs of compliance in determining what standard is requisite to protect public welfare from known or anticipated adverse effects.

3. Administrator's Conclusions on the Need for Revision

Having carefully considered the advice from CASAC and public comments, as discussed above, the Administrator believes that the fundamental scientific conclusions on the welfare effects of O₃ in ambient air reached in the ISA and summarized in the PA and in section IV.B of the proposal remain valid. Additionally, the Administrator believes the judgments she reached in the proposal (section IV.D.3) with regard to consideration of the evidence and quantitative assessments and advice from CASAC remain appropriate. Thus, as described below, the Administrator concludes that the current secondary standard is not requisite to protect public welfare from known and anticipated adverse effects associated with the presence of O₃ in the ambient air and that revision is needed to provide additional protection.

In considering the adequacy of the current secondary O₃ standard, the Administrator has carefully considered the available evidence, analyses and conclusions contained in the ISA, including information newly available in this review; the information, quantitative assessments, considerations and conclusions presented in the PA; the advice and recommendations from CASAC; and public comments. The Administrator gives primary consideration to the evidence of growth

effects in well-studied tree species and information, presented in the PA and represented with a narrower focus in section IV.B.2 above, on cumulative exposures occurring in Class I areas when the current standard is met. This information indicates the occurrence of exposures associated with Class I areas during periods when the current standard is met for which associated estimates of growth effects, in terms of the tree seedling RBL in the median species for which E-R functions have been established, extend above a magnitude considered to be "unacceptably high" by CASAC. This analysis estimated such cumulative exposures occurring under the current standard for nearly a dozen areas, distributed across two NOAA climatic regions of the U.S. The Administrator gives particular weight to this analysis, given its focus in Class I areas. Such an emphasis on lands afforded special government protections, such as national parks and forests, wildlife refuges, and wilderness areas, some of which are designated Class I areas under the CAA, is consistent with such emphasis in the 2008 revision of the secondary standard (73 FR 16485, March 27, 2008). As noted in section IV.A above, Congress has set such lands aside for specific uses that are intended to provide benefits to the public welfare, including lands that are to be protected so as to conserve the scenic value and the natural vegetation and wildlife within such areas, and to leave them unimpaired for the enjoyment of future generations. The Administrator additionally recognizes that states, tribes and public interest groups also set aside areas that are intended to provide similar benefits to the public welfare for residents on those lands, as well as for visitors to those areas.

As noted in prior reviews, judgments regarding effects that are adverse to public welfare consider the intended use of the ecological receptors, resources and ecosystems affected. Thus, the Administrator recognizes that the median RBL estimate for the studied species is a quantitative tool within a larger framework of considerations pertaining to the public welfare significance of O₃ effects on the public welfare. Such considerations include effects that are associated with effects on growth and that the ISA has determined to be causally or likely causally related to O₃ in ambient air, yet for which there are greater uncertainties affecting our estimates of impacts on public welfare. These other effects include reduced productivity in terrestrial ecosystems, reduced carbon

sequestration in terrestrial ecosystems, alteration of terrestrial community composition, alteration of below-grown biogeochemical cycles, and alteration of terrestrial ecosystem water cycles, as summarized in section IV.A.1. Thus, in her attention to CASAC's characterization of a 6% estimate for tree seedling RBL in the median studied species as "unacceptably high", the Administrator, while mindful of uncertainties with regard to the magnitude of growth impact that might be expected in mature trees, is also mindful of related, broader, ecosystem-level effects for which our tools for quantitative estimates are more uncertain and those for which the policy foundation for consideration of public welfare impacts is less well established. She finds her consideration of tree growth effects consistent with CASAC advice regarding consideration of O₃-related biomass loss as a surrogate for the broader array of O₃ effects at the plant and ecosystem levels.

The Administrator also recognizes that O₃-related effects on sensitive vegetation can occur in other areas that have not been afforded special federal protections, including effects on vegetation growing in managed city parks and residential or commercial settings, such as ornamentals used in urban/suburban landscaping or vegetation grown in land use categories that are heavily managed for commercial production of commodities such as timber. In her consideration of the evidence and quantitative information of O₃ effects on crops, the Administrator recognizes the complexity of considering adverse O₃ impacts to public welfare due to the heavy management common for achieving optimum yields and market factors that influence associated services. In so doing, she notes that her judgments that place emphasis on the protection of forested ecosystems inherently also recognize a level of protection for crops. Additionally, for vegetation used for residential or commercial ornamental purposes, the Administrator believes that there is not adequate information specific to vegetation used for those purposes, but notes that a secondary standard revised to provide protection for sensitive natural vegetation and ecosystems would likely also provide some degree of protection for such vegetation.

The Administrator also takes note of the long-established evidence of consistent association of the presence of visible foliar injury with O₃ exposure and the currently available information that indicates the occurrence of visible foliar injury in sensitive species of

vegetation during recent air quality in public forests across the U.S. She additionally notes the PA conclusions regarding difficulties in quantitatively relating visible foliar injury symptoms to vegetation effects such as growth or related ecosystem effects. As at the time of the last review, the Administrator believes that the degree to which such effects should be considered to be adverse depends on the intended use of the vegetation and its significance. The Administrator also believes that the significance of O₃-induced visible foliar injury depends on the extent and severity of the injury and takes note of studies in the evidence base documenting increased severity and/or prevalence with higher O₃ exposures. However, the Administrator takes note of limitations in the available information with regard to judging the extent to which the extent and severity of visible foliar injury occurrence associated with conditions allowed by the current standard may be considered adverse to public welfare.

Based on these considerations, and taking into consideration the advice and recommendations of CASAC, the Administrator concludes that the protection afforded by the current secondary O₃ standard is not sufficient and that the standard needs to be revised to provide additional protection from known and anticipated adverse effects to public welfare, related to effects on sensitive vegetation and ecosystems, most particularly those occurring in Class I areas. The Administrator additionally recognizes that states, tribes and public interest groups also set aside areas that are intended to provide similar benefits to the public welfare for residents on those lands, as well as for visitors to those areas. Given the clear public interest in and value of maintaining these areas in a condition that does not impair their intended use, and the fact that many of these areas contain O₃-sensitive vegetation, the Administrator further concludes that it is appropriate to revise the secondary standard in part to provide increased protection against O₃-caused impairment to vegetation and ecosystems in such areas, which have been specially protected to provide public welfare benefits. She further notes that a revised standard would provide increased protection for other growth-related effects, including for crop yield loss, reduced carbon storage and for areas for which it is more difficult to determine public welfare significance, as recognized in section IV.A.3 above, as well other welfare

effects of O₃, such as visible foliar injury.

C. Conclusions on Revision of the Secondary Standard

The elements of the standard—indicator, averaging time, form, and level—serve to define the standard and are considered collectively in evaluating the welfare protection afforded by the secondary standard. Section IV.C.1 below summarizes the basis for the proposed revision. Significant comments received from the public on the proposal are discussed in section IV.C.2 and the Administrator's final decision on revisions to the secondary standard is described in section IV.C.3.

1. Basis for Proposed Revision

At the time of proposal, in considering what revisions to the secondary standard would be appropriate, the Administrator considered the ISA conclusions regarding the weight of the evidence for a range of welfare effects associated with O₃ in ambient air and associated areas of uncertainty; quantitative risk and exposure analyses in the WREA for different adjusted air quality scenarios and associated limitations and uncertainties; staff evaluations of the evidence, exposure/risk information and air quality information in the PA; additional air quality analyses of relationships between air quality metrics based on form and averaging time of the current standards and a cumulative seasonal exposure index; CASAC advice; and public comments received as of that date in the review. In the paragraphs below, we summarize the proposal presentation with regard to key aspects of the PA considerations, advice from the CASAC, air quality analyses of different air quality metrics and the Administrator's proposed conclusions, drawing from section IV.E of the proposal.

a. Considerations and Conclusions in the PA

As summarized in the proposal, in identifying alternative secondary standards appropriate to consider in this review, the PA focused on standards based on a cumulative, seasonal, concentration-weighted form consistent with the CASAC advice in the current and last review. Based on conclusions of the ISA, as also summarized in section IV.A above, the PA considered a cumulative, seasonal, concentration-weighted exposure index to provide the most scientifically defensible approach for characterizing vegetation response to ambient O₃ and comparing study findings, as well as for defining indices

for vegetation protection, as summarized in the proposal section IV.E.2.a. With regard to the appropriate index, the PA considered the evidence for a number of different such indices, as described in the proposal, and noted the ISA conclusion that the W126 index has some important advantages over other similarly weighted indices. The PA additionally considered the appropriate diurnal and seasonal exposure periods in a given year by which to define the seasonal W126 index and based on the evidence in the ISA and CASAC advice, as summarized in the proposal, decided on the 12-hour daylight window (8:00 a.m. to 8:00 p.m.) and the 3-consecutive-month period providing the maximum W126 index value.

Based on these considerations, the PA concluded it to be appropriate to retain the current indicator of O₃ and to consider a secondary standard form that is an average of the seasonal W126 index values (derived as described in section IV.A.1.c above) across three consecutive years (U.S. EPA, 2014c, section 6.6). In so doing, the PA recognized that there is limited information to discern differences in the level of protection afforded for cumulative growth-related effects by potential alternative W126-based standards of a single-year form as compared to a 3-year form (U.S. EPA, 2014c, pp. 6–30). The PA concluded a 3-year form to be appropriate for a standard intended to provide the desired level of protection from longer-term effects, including those associated with potential compounding, and that such a form might be concluded to contribute to greater stability in air quality management programs, and thus, greater effectiveness in achieving the desired level of public welfare protection than might result from a single-year form. (U.S. EPA, 2014c, section 6.6).

As summarized in the proposal, the PA noted that, due to the variability in the importance of the associated ecosystem services provided by different species at different exposures and in different locations, as well as differences in associated uncertainties and limitations, it is essential to consider the species present and their public welfare significance, together with the magnitude of the ambient concentrations in drawing conclusions regarding the significance or magnitude of public welfare impacts. Therefore, in development of the PA conclusions, staff took note of the complexity of judgments to be made by the Administrator regarding the adversity of known and anticipated effects to the

public welfare and recognized that the Administrator's ultimate judgments on the secondary standard will most appropriately reflect an interpretation of the available scientific evidence and exposure/risk information that neither overstates nor understates the strengths and limitations of that evidence and information. In considering an appropriate range of levels to consider for an alternative standard, the PA primarily considered tree growth, crop yield loss, and visible foliar injury, as well as impacts on the associated ecosystem services, while noting key uncertainties and limitations.

In specifically evaluating exposure levels, in terms of the W126 index, as to their appropriateness for consideration in this review with regard to providing the desired level of vegetation protection for a revised secondary standard, the PA focused particularly on RBL estimates for the median across the 11 tree species for which robust E-R functions are available. Table 4 below presents these estimates (U.S. EPA, 2014c, Appendix 5C, Table 5C-3; also summarized in Table 8 of the proposal). In so doing and recognizing the longstanding, strong evidence base supporting these relationships, the PA also noted

uncertainties regarding inter-study variability for some species, as well as with regard to the extent to which tree seedling E-R functions can be used to represent mature trees. As summarized in the proposal, the PA conclusions on a range of W126 levels appropriate to consider are based on specific advice from CASAC with regard to median tree seedling RBL estimates that might be considered unacceptably high (6%), as well as its judgment on a RBL benchmark (2%) for identification of the lower end of a W126 index value range for consideration that might give more emphasis to the more sensitive tree seedlings (Frey, 2014c, p. 14).¹⁹⁶

TABLE 4—TREE SEEDLING BIOMASS LOSS AND CROP YIELD LOSS ESTIMATED FOR O₃ EXPOSURE OVER A SEASON

W126 index value for exposure period	Tree seedling biomass loss ^A		Crop yield loss ^B	
	Median value	Individual species	Median value	Individual species
23 ppm-hrs	Median species w. 7.6% loss	≤ 2% loss: 3/11 species ≤ 5% loss: 4/11 species ≤10% loss: 8/11 species ... ≤15% loss: 10/11 species ... >40% loss: 1/11 species ...	Median species w. 8.8% loss	≤ 5% loss: 4/10 species >5,<10% loss: 1/10 species >10,<20% loss: 4/10 species >20: 1/10 species
22 ppm-hrs	Median species w. 7.2% loss	≤ 2% loss: 3/11 species ≤ 5% loss: 4/11 species ≤10% loss: 7/11 species ... ≤15% loss: 10/11 species ... >40% loss: 1/11 species ...	Median species w. 8.2% loss	≤ 5% loss: 4/10 species >5,<10% loss: 1/10 species >10,<20% loss: 4/10 species >20: 1/10 species
21 ppm-hrs	Median species w. 6.8% loss	≤ 2% loss: 3/11 species ≤ 5% loss: 4/11 species ≤10% loss: 7/11 species ... ≤15% loss: 10/11 species ... >40% loss: 1/11 species ...	Median species w. 7.7% loss	≤ 5% loss: 4/10 species >5,<10% loss: 3/10 species >10,<20% loss: 3/10 species
20 ppm-hrs	Median species w. 6.4% loss	≤ 2% loss: 3/11 species ≤ 5% loss: 5/11 species ≤10% loss: 7/11 species ... ≤15% loss: 10/11 species ... >40% loss: 1/11 species ...	Median species w. 7.1% loss	≤ 5% loss: 5/10 species >5,<10% loss: 3/10 species >10,<20% loss: 2/10 species
19 ppm-hrs	Median species w. 6.0% loss	≤ 2% loss: 3/11 species ≤5% loss: 5/11 species ≤10% loss: 7/11 species ... ≤15% loss: 10/11 species ... >30% loss: 1/11 species ...	Median species w. 6.4% loss	≤ 5% loss: 5/10 species >5, <10% loss: 3/10 species >10,<20% loss: 2/10 species
18 ppm-hrs	Median species w. 5.7% loss	≤ 2% loss: 5/11 species ≤ 5% loss: 5/11 species ≤10% loss: 7/11 species ... ≤15% loss: 10/11 species ... >30% loss: 1/11 species ...	Median species w. 5.7% loss	≤ 5% loss: 5/10 species >5,<10% loss: 3/10 species >10,<20% loss: 2/10 species
17 ppm-hrs	Median species w. 5.3% loss	≤ 2% loss: 5/11 species ≤5% loss: 5/11 species ≤10% loss: 9/11 species ... ≤15% loss: 10/11 species ... >30% loss: 1/11 species ...	Median species w. 5.1% loss	≤ 5% loss: 5/10 species >5, <10% loss: 3/10 species >10,<20% loss: 2/10 species
16 ppm-hrs	Median species w. 4.9% loss	≤ 2% loss: 5/11 species ≤ 5% loss: 6/11 species ≤10% loss: 10/11 species ... >30% loss: 1/11 species ...	Median species w. ≤5.0% loss	≤ 5% loss: 5/10 species >5,<10% loss: 4/10 species >10,<20% loss: 1/10 species
15 ppm-hrs	Median species w. 4.5% loss	≤ 2% loss: 5/11 species ≤5% loss: 6/11 species ≤10% loss: 10/11 species ... >30% loss: 1/11 species ...	Median species w. ≤5.0% loss	≤ 5% loss: 6/10 species >5, <10% loss: 4/10 species
14 ppm-hrs	Median species w. 4.2% loss	≤ 2% loss: 5/11 species ≤ 5% loss: 6/11 species ≤10% loss: 10/11 species ... >30% loss: 1/11 species ...	Median species w. ≤5.0% loss	≤ 5% loss: 6/10 species >5,<10% loss: 4/10 species
13 ppm-hrs	Median species w. 3.8% loss	≤ 2% loss: 5/11 species <5% loss: 7/11 species <10% loss: 10/11 species ... >20% loss: 1/11 species ...	Median species w. ≤5.0% loss	≤ 5% loss: 6/10 species >5, <10% loss: 4/10 species

¹⁹⁶ The CASAC provided several comments related to 2% RBL for tree seedlings both with

regard to its use in summarizing WREA results and with regard to consideration of the potential

significance of vegetation effects, as summarized in sections IV.D.2 and IV.E.3 of the proposal.

TABLE 4—TREE SEEDLING BIOMASS LOSS AND CROP YIELD LOSS ESTIMATED FOR O₃ EXPOSURE OVER A SEASON—Continued

W126 index value for exposure period	Tree seedling biomass loss ^A		Crop yield loss ^B	
	Median value	Individual species	Median value	Individual species
12 ppm-hrs	Median species w. 3.5% loss	≤ 2% loss: 5/11 species ≤ 5% loss: 8/11 species ≤10% loss: 10/11 species >20% loss: 1/11 species ...	Median species w. ≤5.0% loss	≤ 5% loss: 8/10 species >5, <10% loss: 2/10 species
11 ppm-hrs	Median species w. 3.1% loss	≤ 2% loss: 5/11 species ≤5% loss: 8/11 species ≤10% loss: 10/11 species >20% loss: 1/11 species ...	Median species w. ≤5.0% loss	≤ 5% loss: 9/10 species >5, <10% loss: 1/10 species
10 ppm-hrs	Median species w. 2.8% loss	≤ 2% loss: 5/11 species ≤ 5% loss: 9/11 species <10% loss: 10/11 species >20% loss: 1/11 species ...	Median species w. ≤5.0% loss	≤ 5% loss: 9/10 species >5, <10% loss: 1/10 species
9 ppm-hrs	Median species w. 2.4% loss	≤ 2% loss: 5/11 species ≤ 5% loss: 10/11 species .. >20% loss: 1/11 species ...	Median species w. ≤5.0% loss	≤ 5% loss: all species
8 ppm-hrs	Median species w. 2.0% loss	≤ 2% loss: 5/11 species ≤ 5% loss: 10/11 species .. >15% loss: 1/11 species ...	Median species w. ≤5.0% loss	≤ 5% loss: all species
7 ppm-hrs	Median species w. <2.0% loss	≤ 2% loss: 7/11 species ≤5% loss: 10/11 species ... >15% loss: 1/11 species ...	Median species w. ≤5.0% loss	≤ 5% loss: all species

^A Estimates here are based on the E-R functions for 11 species described in the WREA, section 6.2 and discussed in the PA, section 5.2.1. The cottonwood was excluded to address CASAC comments (Frey, 2014c; U.S. EPA, 2014b, U.S. EPA, 2014c, Appendix 6F). The median is the median of the 11 composite E-R functions (U.S. EPA, 2014c, Appendix 5C).

^B Estimates here are based on the 10 E-R functions for crops described in the WREA, section 6.2 and discussed in the PA, section 5.3.1. The median is the median of the 10 composite E-R functions (U.S. EPA, 2014b; U.S. EPA, 2014c, Appendix 5C).

With regard to secondary standard revisions appropriate to consider in this review, as summarized in the proposal, the PA concluded it to be appropriate to consider a W126-based secondary standard with index values within the range of 7 to 17 ppm-hrs and a form averaged over 3 years (U.S. EPA, 2014c, section 6.7). The PA additionally recognized the role of policy judgments required of the Administrator with regard to the public welfare significance of identified effects, the appropriate weight to assign the range of uncertainties inherent in the evidence and analyses, and ultimately, in identifying the requisite protection for the secondary O₃ standard.

The PA additionally recognized that to the extent the Administrator finds it useful to consider the public welfare protection that might be afforded by revising the level of the current standard, this is appropriately judged by evaluating the impact of associated O₃ exposures in terms of the cumulative seasonal W126-based index, an exposure metric considered appropriate for evaluating impacts on vegetation (U.S. EPA, 2014c, section 6.7). Accordingly, the PA included several air quality data analyses that might inform such consideration (U.S. EPA, 2014c, section 6.4). Additional air quality analyses were performed subsequent to the PA, described in the proposal and are summarized below.

b. CASAC Advice

Advice received from the CASAC during the current review, similar to that in the last review, recommended retaining O₃ as the indicator, while also recommending consideration of a secondary standard with a revised form and averaging time based on the W126 index (Frey, 2014c, p. iii). The CASAC concurred with the 12-hour period (8 a.m. to 8 p.m.) and 3-month summation period resulting in the maximum W126 index value, as described in the PA, while recommending a somewhat narrower range of levels from 7 ppm-hrs to 15 ppm-hrs. While the CASAC recommended a W126 index limited to a single year, in contrast with the PA's conclusion that it was appropriate to consider the W126 index averaged across three years, it also noted that the Administrator may prefer, as a policy matter, to base the secondary standard on a 3-year averaging period. In such a case, the CASAC recommended revising downward the level for such a metric to avoid a seasonal W126 index value above a level in their recommended range in any given year of the 3-year period, indicating an upper end of 13 ppm-hrs as an example for such a 3-year average W126 index range (Frey, 2014c, p. iii and iv).

c. Air Quality Analyses

The proposal additionally summarized several analyses of air quality that considered relationships

between metrics based on a 3-year W126 index and based on the form and averaging time of the current standard, the “fourth-high” metric (U.S. EPA, 2014c, Chapter 2, Appendix 2B and section 6.4; Wells, 2014a), as well as describing the uncertainties and limitations associated with these analyses. The proposal concluded that these analyses suggest that, depending on the level, a standard of the current averaging time and form can be expected to control cumulative seasonal O₃ exposures to such that they may meet specific 3-year average W126 index values. The fourth-high and W126 metrics, and changes in the two metrics over the past decade, were found to be highly correlated (U.S. EPA, 2014c, section 6.4 and Appendix 2B; Wells, 2014a). From these analyses, it was concluded that future control programs designed to help meet a standard based on the fourth-high metric are also expected to result in reductions in values of the W126 metric (Wells, 2014a). Further, the second analysis also found that the Southwest and West NOAA climatic regions, which showed the greatest potential for sites to measure elevated cumulative, seasonal O₃ exposures without the occurrence of elevated daily maximum 8-hour average O₃ concentrations, exhibited the greatest reduction in W126 metric value per unit reduction in fourth-high metric (Wells, 2014a, Figures 5b and 12 and Table 6).

Analyses of the most recent periods studied in the two analyses (2009–2011 and 2011–2013) had similar findings regarding the highest W126 metric values occurring at monitoring sites that meet alternative levels of the fourth-high metric (U.S. EPA, 2014c, section 6.4; Wells, 2014a). In both analyses, the highest W126 metric values were in the Southwest and West NOAA climatic regions. In both analyses, no monitoring sites for which the fourth-high metric was at or below 70 ppb had a W126 metric value above 17 ppm-hrs (U.S. EPA, 2014c, Figure 2B–3b; Wells, 2014a, Table 4). All U.S. regions were represented in these subsets. In the 2011–2013 subset of sites for which the fourth-high metric was at or below a potential alternative primary standard level of 65 ppb, no monitoring sites had W126 metric values above 11 ppm-hrs (Wells, 2014a, Table 4).

d. Administrator's Proposed Conclusions

At the time of proposal, the Administrator concluded it to be appropriate to continue to use O₃ as the indicator for a secondary standard that is intended to address effects associated with exposure to O₃ alone and in combination with related photochemical oxidants. While the complex atmospheric chemistry in which O₃ plays a key role has been highlighted in this review, no alternatives to O₃ have been advanced as being a more appropriate surrogate for ambient photochemical oxidants and their effects on vegetation. The CASAC agreed that O₃ should be retained as the indicator for the standard (Frey, 2014c, p. iii). In proposing to retain O₃ as the indicator, the Administrator recognized that measures leading to reductions in ecosystem exposures to O₃ would also be expected to reduce exposures to other photochemical oxidants.

The Administrator proposed to retain the current averaging time and form and to revise the level of the current secondary standard to a level within the range of 0.065 to 0.070 ppm. She based this proposal on her provisional conclusions regarding the level of cumulative seasonal O₃ exposures that would provide the requisite protection against known or anticipated adverse effects to the public welfare and on a policy option that would provide this level of protection. With regard to the former, the Administrator concluded that in judging the extent of public welfare protection that might be afforded by a revised standard and whether it meets the appropriate level of protection, it is appropriate to use a cumulative, seasonal concentration-

weighted exposure metric. For this purpose, the Administrator concluded it to be appropriate to use the W126 index value, averaged across three years, with each year's value identified as that for the 3-month period yielding the highest seasonal value and with daily O₃ exposures within a 3-month period cumulated for the 12-hour period from 8:00 a.m. to 8:00 p.m.

To identify the range of cumulative seasonal exposures, in terms of the W126 index, expected to be associated with the appropriate degree of public welfare protection, the Administrator gave primary consideration to growth-related impacts, using tree seedling RBL estimates for a range of W126 exposure index values and CASAC advice regarding such estimates. Additionally taking into account judgments on important uncertainties and limitations inherent in the current available scientific evidence and quantitative assessments, and judgments regarding the extent to which different RBL estimates might be considered indicative of effects adverse to public welfare, the Administrator proposed that ambient O₃ concentrations resulting in cumulative seasonal O₃ exposures of a level within the range from 13 ppm-hrs to 17 ppm-hrs, in terms of a W126 index averaged across three consecutive years, would provide the requisite protection against known or anticipated adverse effects to the public welfare. In identifying policy options for a revised secondary standard that would control exposures to such an extent, the Administrator considered the results of air quality analyses that examined the responsiveness of cumulative exposures (in terms of the W126 index) to O₃ reductions in response to the current and prior standard for which the form and averaging time are summarized as a fourth-high metric, and also examined the extent to which cumulative exposures (in terms of the W126 index) may be limited by alternative levels of a metric based on the current standard averaging time and form. Based on the results of these analyses, she proposed that revision of the level of the current secondary standard to within the range of 0.065 to 0.070 ppm would be expected to provide the requisite public welfare protection, depending on final judgments concerning such requisite protection.

2. Comments on Proposed Revision

Significant comments from the public regarding revisions to the secondary standard are addressed in the subsections below. We first discuss comments related to our consideration of growth-related effects and visible

foliar injury in identifying appropriate revisions to the standard (sections IV.C.2.a and IV.C.2.b). Next, we address comments related to the use of the W126 metric in evaluating vegetation effects and public welfare protection and comments related to the form and averaging time for the revised standard (sections IV.C.2.c and IV.C.2.d). Comments on revisions to the level of the standard are described in section IV.C.2.e, and those related to the way in which today's rulemaking addresses the 2013 court remand are addressed in section IV.C.2.f. Other significant comments related to consideration of a revised secondary standard, and that are based on relevant factors, are addressed in the Response to Comments document.

a. Consideration of Growth-Related Effects

In considering public comments received on the consideration of growth-related effects of O₃ in the context of the proposed decision on a revised secondary standard, we first note related advice and comments from the CASAC provided during development of the PA, stating, as summarized in section IV.B.1.b above, that "relative biomass loss for tree species, crop yield loss, and visible foliar injury are appropriate surrogates for a wide range of damage that is adverse to public welfare" (Frey, 2014c, p. 10). Additionally, in the context of different standard levels they considered appropriate for the EPA to consider, CASAC stated that it is appropriate to "include[] levels that aim for not greater than 2% RBL for the median tree species" and that a median tree species RBL of 6% is "unacceptably high" (Frey, 2014c, p. 14).¹⁹⁷ With respect to crop yield loss, CASAC points to a benchmark of 5%, stating that a crop RYL for median species over 5% is "unacceptably high" (Frey, 2014c, p. 13).

In addition, regarding consideration of RBL benchmarks for tree seedlings, the CASAC stated that "[a] 2% biomass loss is an appropriate scientifically based value to consider as a benchmark of adverse impact for long-lived perennial species such as trees, because effects are cumulative over multiple

¹⁹⁷ The CASAC made this comment while focusing on Table 6–1 in the second draft PA and the entry for 17 ppm-hrs (Frey, 2014c, p. 14). That table was revised for inclusion in the final PA in consideration of CASAC comments on the E–R function for eastern cottonwood, and after that revision, the median RBL estimate for 17 ppm-hrs in the final table (see Table 4 above) is below the value of 6% that CASAC described in this way.

years” (Frey, 2014c, p. 14).¹⁹⁸ With regard to this benchmark, the CASAC also commented that “it is appropriate to identify a range of levels of alternative W126-based standards that includes levels that aim for not greater than 2% RBL for the median tree species” in the PA (Frey, 2014c, p. 14). The CASAC noted that the “level of 7 ppm-hrs is the only level analyzed for which the relative biomass loss for the median tree species is less than or equal to 2 percent,” indicating that 7 ppm was appropriate as a lower bound for the recommended range (Frey, 2014c, p. 14).¹⁹⁹

With regard to consideration of effects on crops, in addition to their comments regarding a median species RYL over 5% yield loss, noted above (Frey, 2014c, p. 13), the CASAC further noted that “[c]rop loss appears to be less sensitive than these other indicators, largely because of the CASAC judgment that a 5% yield loss represents an adverse impact, and in part due to more opportunities to alter management of annual crops” (Frey, 2014c, p. 14).

Comments from the public with regard to how the EPA considered growth-related effects in the proposed decision on a revised secondary standard varied. Generally, those commenters who recommended against revision of the standard expressed the view that RBL estimates based on the established E-R functions for the 11 studied species, and their pertinence to mature trees, were too uncertain to serve as a basis for judgments regarding public welfare protection afforded by the secondary standard. The EPA generally disagrees with this view, as discussed in section IV.B.2 above, and addressed in more detail in the Response to Comments document.

Some commenters also took note of the unclear basis for CASAC’s 2% benchmark, stating that the CASAC advice on this point is “not wholly scientific,” given that it referenced the 1996 workshop, which provided little specificity as to scientific basis for such a benchmark; based on this, the

¹⁹⁸ The CASAC provided several comments related to 2% RBL for tree seedlings both with regard to its use in summarizing WREA results and with regard to consideration of the potential significance of vegetation effects, as summarized in sections IV.D.2 and IV.E.3 of the proposal.

¹⁹⁹ The CASAC made this comment while focusing on Table 6-1 in the second draft PA, which included odd-numbered W126 index values and in which the median RBL values were based on 12 species. That table was revised for inclusion in the final PA in consideration of CASAC comments on the E-R function for eastern cottonwood, such that the median RBL species estimate for both 7 ppm-hrs and 8 ppm-hrs are less than or equal to 2.0% in the final table (see Table 4 above and Table 5C-3 of the final PA).

commenters described this CASAC advice as a policy judgment and described the important role of the EPA’s judgment in such instances. As noted in section IV.E.3 of the proposal, we generally agree with these commenters regarding the unclear scientific basis for the 2% value. Consistent with this advice from CASAC, however, the range of levels for a revised secondary standard that the PA concluded was appropriate for the Administrator to consider did include a level for which the estimated median RBL across the 11 studied tree species would be 2%, as well as a level for which the median RBL would be below 2% (U.S. EPA, 2014c, section 6.7 and Tables 6-1 and 5C-3), and, as described in the proposal, the Administrator considered the conclusions of the PA in reaching her proposed decision that it was appropriate to consider a range for the revised secondary standard that did not focus on this benchmark. The Administrator has further considered and explained any differences from CASAC’s recommendations on this point in her final decision, as described in section IV.C.3 below.

Some of the state and local environmental agencies and organizations and environmental groups that supported the EPA’s proposed decision to revise the secondary standard additionally indicated their view that the EPA should give more weight to growth-related effects by setting the standard at a level for which the estimated RBL would be at or below 2% in the median studied species. In support of this recommendation, the commenters cited the CASAC advice and stated that the EPA’s rationale deviates from that advice with regard to consideration of RBL. In so doing, the commenters implied incorrectly that the EPA’s proposal did not put the most weight on the median RBL. In fact, in considering RBL as a metric for growth effects, the Administrator’s proposed conclusions focused solely on the median RBL estimates, indicating that appreciable weight was given to growth-related effects and on the median RBL. Additionally, the commenters implied that the EPA misconstrued the CASAC comment on 6% RBL to indicate that it was acceptable. Yet, the proposal notes CASAC’s view that a 6% RBL is “unacceptably high” nine times, and, in section IV.B.3 above, the Administrator takes note of this view in reaching the decision that the current standard should be revised. The EPA considers this statement from CASAC, provided in the context of considering effects related to different W126 index values, to be of

a different nature than CASAC advice discussed above that options for the EPA consideration “include” a level that aims for median RBL at or below 2%.

The comments that state that the standard should control cumulative exposures to levels for which the estimated median species RBL is at or below 2% provided little rationale beyond citing to CASAC advice. We note, however, that the CASAC did not specify that the revised secondary standard be set to limit cumulative exposures to that extent. Nor, in identifying a range of alternatives for the EPA to consider, did CASAC recommend that the EPA consider *only* W126 index levels associated with median RBL estimates at or below 2%. Rather, the CASAC stated that “it is appropriate to identify a range of levels of alternative W126-based standards that *includes* {emphasis added} levels that aim for not greater than 2% RBL for the median tree species” (Frey, 2014c, p. 14) and seven of the nine levels in the CASAC-recommended range of W126 index levels were associated with higher RBL estimates (as shown in Table 4 above).

In citing to CASAC advice, commenters quoted the CASAC characterization of a 2% RBL as “an appropriate scientifically based value to consider as a benchmark of adverse impact for long-lived perennial species such as trees, because effects are cumulative over multiple years” (Frey, 2014, p. 14). Presumably to indicate reasoning for this statement, the subsequent sentence in the same CASAC letter referenced findings for biomass loss in aspen exposed to elevated O₃ over seven years, citing Wittig et al., 2009. As noted in the proposal, however, the way in which these findings would provide a basis for CASAC’s view with regard to 2% is unclear, as the original publication that is the source for the 7-year biomass loss value (King, et al., 2005) and which is cited in Wittig et al. (2009) indicates yearly RBL values during this 7-year exposure that are each well above 2%, and, in fact, are all above 20% (King, et al., 2005). In the same paragraph, the CASAC letter additionally referenced the report of the 1996 workshop sponsored by the Southern Oxidants Study group (Heck and Cowling, 1997, noted in section IV.A.3 above). The workshop report identified 1–2% per year growth reduction (based on a stated interest in avoiding 2% cumulative effects) as an appropriate endpoint for consideration of growth effects in trees, although an explicit rationale for the identified percentages is not provided

(Frey, 2014c, p. 14).²⁰⁰ Like the 1996 workshop, the CASAC describes 2% RBL as providing the basis for consideration of 7 ppm-hrs, the lower end of their recommended W126 range (Frey, 2014c, p. 14). As a result, the specific scientific basis for judging a value of 2% RBL in the median studied species as an appropriate benchmark of adverse impact for trees and other long-lived perennials is not clear, which, as described in the proposal, contributed to the Administrator noting the greater uncertainty regarding the extent to which estimates of benefits in terms of ecosystem services and reduced effects on vegetation at O₃ exposures below her identified range of 13 to 17 ppm-hrs might be judged significant to the public welfare.

Some commenters recommended revision of the standard to 7 ppm-hrs as a W126 form stating that such a change is needed to protect against climate change. In so doing, one commenter expressed the view that the relatively lesser weight the EPA placed on the WREA estimates of carbon storage (in terms of CO₂) in consideration of a proposed revision to the secondary standard is inconsistent with the emphasis that the EPA placed on CO₂ emissions reductions estimated for the proposed Clean Power Plan (79 FR 34830, 34931–33). As support for this view of inconsistency, the commenter compared the WREA 30-year estimate of the amount of CO₂ removed from the air and stored in vegetation with estimated reductions in CO₂ emissions from power plants over a 4-year period. We note, however, some key distinctions between the two types of estimates which appropriately lead to different levels of emphasis by the EPA in the two actions. First, we note that the lengths of time pertaining to the two estimates that the commenter states to be “roughly equal” (e.g., ALA et al., p. 211) differ by more than a factor of seven (4 years compared to 30). Second, the CPP estimates are for reductions in CO₂ produced and emitted from power plants, while the WREA estimates are for amounts of CO₂ removed from the air and stored in vegetation as a result of plant photosynthesis occurring across the U.S. This leads to two important differences. The first is whether a ton of additional carbon uptake by plants is equal to a ton of reduced emissions from fossil fuels. This is still an active area of discussion due in part to the potentially transient

²⁰⁰ The report of the 1996 workshop provides no more explicit rationale for the percentages identified or specification with regard to number or proportion of species for which such percentages should be met (Heck and Cowling, 1997).

nature of the carbon storage in vegetation. The second is that there are much larger uncertainties involved in attempting to quantify the additional carbon uptake by plants which requires complex modeling of biological and ecological processes and their associated sources of uncertainty. Therefore, as summarized in section IV.C.3 below, the Administrator is judging, as at the time of proposal, that the quantitative uncertainties are too great to support identification of a revised standard based specifically on the WREA quantitative estimates of carbon storage benefits to climate. In so doing, she notes that a revised standard, established primarily based on other effects for which our quantitative estimates are less uncertain, can be expected to also provide increased protection in terms of carbon storage.

b. Consideration of Visible Foliar Injury

In considering public comments received on the EPA’s consideration of visible foliar injury in its decision on a revised secondary standard, the EPA first notes related advice and comments from the CASAC received during development of the PA. The CASAC stated that “[w]ith respect to the secondary standard, the CASAC concurs with the EPA’s identification of adverse welfare effects related to . . . damage to resource use from foliar injury” (Frey, 2014, p. iii). In its comments on levels of a W126-based standard, the CASAC, seemingly in reference to the WREA visible foliar injury analyses, additionally stated that “[a] level below 10 ppm-hrs is required to reduce foliar injury” (Frey, 2014, pp. iii and 15), with “W126 values below 10 ppm-hr required to reduce the number of sites showing visible foliar injury” (Frey, 2014, p. 14).

Public comments were generally split between two views, either that visible foliar injury was not appropriate to consider in decisions regarding the standard, based on variously identified reasons, or that it should be considered and it would lead the EPA to focus on a W126 value below approximately 10 ppm-hrs. Comments of the former type are discussed in section IV.B.2 above, with, in some cases, additional detail in the Response to Comments document. Commenters expressing the latter view variously cite CASAC advice and figures from the WREA cumulative analysis of USFS biosite data with WREA W126 index value estimates. The EPA disagrees that only a reduction in cumulative exposures to W126 index values below 10 ppm-hrs will affect the occurrence or extent of visible foliar injury. In so doing, we note that the

extensive evidence, which is summarized in the ISA (including studies of the USFS biomonitoring program), analyses in the 2007 Staff Paper and also observations based on the WREA dataset do not support this conclusion.

The evidence regarding visible foliar injury as an indicator of O₃ exposure is well established and generally documents a greater extent and severity of visible foliar injury with higher O₃ exposures and a modifying role of soil moisture conditions (U.S. EPA, 2013, section 9.4.2). As stated in the ISA, “[v]isible foliar injury resulting from exposure to O₃ has been well characterized and documented over several decades of research on many tree, shrub, herbaceous and crop species” and “[o]zone-induced visible foliar injury symptoms on certain bioindicator plant species are considered diagnostic as they have been verified experimentally” (U.S. EPA, 2013, p. 9–41). Further, a recent study highlighted in the ISA, which analyzed trends in the incidence and severity of foliar injury, reported a declining trend in the incidence of foliar injury as peak O₃ concentrations declined (U.S. EPA, 2013, p. 9–40; Smith, 2012). Another study available in this review that focused on O₃-induced visible foliar injury in forests of west coast states observed that both percentage of biosites with injury and average biosite index were higher for sites with average cumulative O₃ concentrations above 25 ppm-hrs in terms of SUM06 (may correspond to W126 of approximately 21 ppm-hrs [U.S. EPA, 2007, p. 8–26, Appendix 7B]) as compared to groups of sites with lower average cumulative exposure concentrations, with much less clear differences between the two lower exposure groups (Campbell et al., 2007, Figures 27 and 28 and p. 30). A similar finding was reported in the 2007 Staff Paper which reported on an analysis that showed a smaller percentage of injured sites among the group of sites with O₃ exposures below a SUM06 metric of 15 ppm-hrs or a fourth-high metric of 74 ppb as compared to larger groups that also included sites with SUM06 values up to 25 ppm-hrs or fourth-high metric up to 84 ppb, respectively (U.S. EPA 2007, pp. 7–63 to 7–64).

With regard to the comments referencing the WREA cumulative analysis of USFS FHM/FIA biosite data or related CASAC comments, we note some clarification of this analysis. This analysis does not show, as implied by the comments, that at W126 index values above 10 ppm-hrs, there is little change with increasing W126 index in

the proportion of records with any visible foliar injury (biosite index above 0). As the analysis is a cumulative analysis, each point graphed in the analysis includes the records for the same and lower W126 index values, so the analysis does not compare results for groups of records with differing, non-overlapping W126 index values. Rather, the points represent groups with records (and W126 index values) in common and the number of records in the groups is greater for higher W126 index values (U.S. EPA, 2014b, section 7.2). Additionally, we note that the pattern observed in the cumulative analysis is substantially influenced by the large number of records for which the W126 index estimates are at or below 11 ppm-hrs, more than two thirds of the dataset (Smith and Murphy, 2015, Table 1).

To more fully address the comments related to this WREA analysis, we have drawn several additional observations from the WREA dataset, re-presenting the same data in a different format in a technical memorandum to the docket (Smith and Murphy, 2015). Contrary to the implication of the statements from the commenters and CASAC that no reduction in the occurrence of visible foliar injury can be achieved with exposures above 10 ppm-hrs, both the proportion of records with injury and the average biosite index are lower for groups of records with W126 index estimates at or below 17 ppm-hrs compared to the group for the highest W126 index range. This is true when considered regardless of soil moisture conditions (all records), as well as for dry, normal and wet records, separately (Smith and Murphy, 2015, Table 2). The pattern of the two measures across record groups with lower W126 index values differs with moisture level, with the wetter than normal records generally showing decreasing proportions of injured sites and decreasing average biosite index with lower W126 index values, while little difference in these measures is seen among the middle W126 values although they are lower than the highest W126 index group and higher than the lowest W126 index group (Smith and Murphy, 2015, Table 2). In summary, the EPA disagrees with commenters, noting that the available information, including additional observations from the WREA dataset, indicate declines in the occurrence of visible foliar injury across decreasing W126 index values that are higher than 10 ppm-hrs.

c. Use of W126 Metric in Evaluating Vegetation Effects and Public Welfare Protection

In considering public comments received on the EPA's use of the W126 exposure index in its decision on a revised secondary standard, the EPA first notes related advice and comments from the CASAC received during development of the PA. Although we recognize that CASAC's comments on the W126 index were provided in the context of its recommendation for a secondary standard of that form, we find them to also relate to our use of the W126 metric in evaluating the magnitude and extent of vegetation effects that might be expected and conversely the level of protection that might be provided under different air quality conditions. In comments on the first draft PA, the CASAC stated that "discussions and conclusions on biologically relevant exposure metrics are clear and compelling and the focus on the W126 form is appropriate" (Frey and Samet, 2012a). With regard to specific aspects of the W126 index, the CASAC concurred with the second draft PA focus on "the biologically-relevant W126 index accumulated over a 12-hour period (8 a.m.–8 p.m.) over the 3-month summation period of a single year resulting in the maximum value of W126" (Frey, 2014c, p. iii).

The CASAC advice on levels of the W126 index on which to focus for public welfare protection recommended a level within the range of 7 ppm-hrs to 15 ppm-hrs (Frey, 2014c, p. iii). We note, however, as summarized in section IV.E.3 of the proposal, that this advice was provided in the context of the CASAC review of the second draft PA, which concluded that a range from 7 to 17 ppm-hrs was appropriate to consider. In considering the upper end of this range, the CASAC consulted Table 6–1 of the second draft PA which indicated for a W126 index value of 17 ppm-hrs an RBL estimate of 6%, a magnitude that CASAC described as "unacceptably high" and that contributed to a lack CASAC support for W126 exposures values higher than 15 ppm-hrs (Frey, 2014c, p. 14; U.S. EPA 2014d, Table 6–1). As noted in section IV.E.3 of the proposal, revisions to the RBL estimate table in the final PA, which were made in consideration of other CASAC comments, have resulted in changes to the median species RBL estimate associated with each W126 index value, such that the median species RBL estimate for a W126 index value of 17 ppm-hrs in this table in the final PA was 5.3%, rather than the "unacceptably high" value of 6% (U.S.

EPA, 2014c, Table 6–1; U.S. EPA, 2014d, Table 6–1; Frey, 2014c, p. 14).²⁰¹ Additionally, the CASAC recognized that the Administrator may, as a policy matter, prefer to use a 3-year average, and stated that in that case, the range of levels should be revised downward (Frey, 2014c, p. iii–iv).

The majority of comments on the W126 index concurred with its use for assessing O₃ exposures, while some commenters additionally expressed the view that this index should be used as the form of the secondary standard (as discussed in section IV.C.2.d below). Most submissions from state and local environmental agencies or governments, as well as organizations of state agencies, that provided comments on the magnitude of cumulative exposure, in terms of the W126 index, appropriate to consider for a revised secondary standard, recommended that the EPA focus on an index value within the EPA's proposed range of 13 to 17 ppm-hrs, as did the industry commenters. These commenters variously noted their agreement with the rationale provided by the EPA in the proposal or cited to CASAC comments, including for a downward adjustment of its recommended values if a 3-year average W126 was used rather than a single year index. Some other commenters, including two groups of environmental organizations, submitted comments recommending a focus on a W126 index level as low as 7 ppm-hrs based on reasons generally focused on consideration of visible foliar injury.

Some aspects of these comments have been addressed in sections IV.C.2.a and IV.C.2.b above. In the Response to Comments document, we have additionally addressed other comments that recommend a focus on W126 index values for specific reasons other than generally citing the CASAC recommended range. Further, in her consideration of a target level of protection for the revised secondary standard in section IV.C.3 below, the Administrator has considered comments from the CASAC regarding the basis for their recommended range.

An additional comment from an organization of western state air quality managers indicated a concern with the use of W126 for vegetation in arid and high altitude regions, such as those in the western states, which the

²⁰¹ We additionally note that the median species RBL estimate for 17 ppm-hrs in the final PA is nearly identical to the estimate for 15 ppm-hrs (the value corresponding to the upper end of the CASAC-identified range) that was in the second draft PA (5.2%) which was the subject of the CASAC review (U.S. EPA, 2014c, Table 6–1; U.S. EPA, 2014d, Table 6–1).

commenter hypothesized may have reduced sensitivity. The commenters did not provide evidence of this hypothesis, calling for further research in order to characterize the sensitivity of vegetation in such areas. The EPA agrees that additional research would be useful in more completely characterizing the response of species in such areas, as well as other less well studied areas, but does not find support in the currently available evidence for the commenter's suggestion that species in arid and high altitude regions may be less sensitive than those in other areas.²⁰²

Among the small number of commenters recommending against using the W126 metric to assess O₃ exposure, a few expressed the view that some other, not-yet-identified cumulative exposure metric should be used. These commenters cited a variety of concerns that they state are not addressed by the W126 index: that plant exposure to and uptake of O₃ are not always equivalent because of variations in stomatal conductance and plant defenses and their respective diel patterns, which will also influence plant response; that the duration between harmful O₃ exposures affects the plant's ability to repair damage; and, that nighttime exposures may be important. These commenters do not identify an alternative to the W126 index that they conclude to better represent exposures relevant to considering O₃ effects on vegetation and particularly for growth effects. The EPA has considered the items raised by these commenters, recognizing some as areas of uncertainty (U.S. EPA, 2013, pp. 9–109 to 9–113), yet has concluded that based on the information available at this time, exposure indices that cumulate and differentially weight the higher hourly average concentrations while also including the “mid-level” values offer the most appropriate approach for use in developing response functions and comparing studies of O₃ effects on vegetation (U.S. EPA, 2013, p. 9–117). When considering the response of vegetation to O₃ exposures represented by the threshold (*e.g.*, SUM06) and non-threshold (*e.g.*, W126) indices, the ISA notes that “the W126 metric does not have a cut-off in the weighting scheme as does SUM06 and thus it includes consideration of potentially damaging exposures below 60 ppb” and that “[t]he

W126 metric also adds increasing weight to hourly concentrations from about 40 ppb to about 100 ppb” (U.S. EPA, 2013, p. 9–104). This aspect of W126 is one way it differs from cut-off metrics such as the SUM06 where all concentrations above 60 ppb are treated equally and is identified by the ISA as “an important feature of the W126 since as hourly concentrations become higher, they become increasingly likely to overwhelm plant defenses and are known to be more detrimental to vegetation” (U.S. EPA, 2013, p. 9–104). Further, we note the concurrence by CASAC with the EPA's focus on the W126 exposure index, as noted above.

Some commenters also raised concerns regarding the sensitivity of vegetation in desert areas where plants take in ambient air during nighttime rather than daylight hours, such that little exposure occurs from 8 a.m. to 8 p.m., stating that the W126 index as defined by the EPA to cumulate hourly O₃ from 8 a.m. to 8 p.m. may result in an overly stringent exposure level in areas with such vegetation. The EPA recognizes that plants, such as cacti, that commonly occur in desert systems exhibit a particular type of metabolism (referred to as CAM photosynthesis) such that they only open their stomata at night (U.S. EPA, 2013, p. 9–109). We note, however, that few if any O₃ exposure studies of these species are available²⁰³ to further inform our characterization of these species' responses to O₃, and we have no basis on which to conclude that an exposure level based on the studied species and a daylight exposure metric would be overly or underly stringent in areas where only species utilizing CAM photosynthesis occur. As summarized above, the CASAC advice concurred with the use of an 8am to 8pm diurnal period for the W126 exposure index. Thus, we conclude that for our purposes in this review the focus on daylight hours is appropriate. Our use of the W126 index in this review has been for purposes of characterizing the potential harm and conversely the potential protection that might be afforded from the well-characterized effects of O₃ on vegetation, while recognizing associated uncertainties and limitations. We note that different ecosystems across the U.S. will be expected to be of varying sensitivities with regard to the effects of O₃. For example, large water bodies without vegetation extending above the water's surface would be expected to be less sensitive than forests of sensitive

species. The EPA notes, however, that the NAAQS are set with applicability to all ambient air in the U.S., such that the secondary O₃ standard provides protection in areas across the U.S. regardless of site-specific aspects of vegetation sensitivity to O₃. In considering the evidence on O₃ and associated welfare effects, we recognize variability in sensitivity that may relate to a number of factors, as discussed in the ISA (U.S. EPA, 2013, section 9.4.8). This variability is among the Administrator's considerations in setting the secondary standard for O₃ that is requisite to protect public welfare against anticipated or known adverse effects.

Further, some commenters who agreed with a focus on the W126 exposure index also stated that the EPA's definition of the index for the daylight hours of 8 a.m. to 8 p.m. and a 3-month period was not appropriate, stating that derivation of the W126 metric should involve summing concentrations for all 24 hours in each day and all months in each year to avoid underestimating O₃ exposure that the commenters viewed as pertinent. Support for the EPA's definition of the W126 index, with which CASAC concurred (Frey, 2014c, p. iii), is based on the assessment of the evidence in the ISA (U.S. EPA, 2013, section 9.5.3.2) and the context for use of the W126 index in relating O₃ exposure to magnitude and/or extent of O₃ response. This context has a particular focus on growth effects for the purposes of judging the potential for public welfare impacts, as well as the level of protection, associated with different exposure circumstances. We note that the ISA stated there is a lack of information that would allow consideration of the extent to which nocturnal exposures that may be of interest occur (U.S. EPA, 2013, p. 9–109). Additionally, in our use of the W126 index, we are relying on E–R functions based on studies that were generally of 3-month duration and involved controlled exposures during the daylight period. Accordingly we have relied on the E–R function derived for 12-hour and 3-month W126 indices, as described in section IV.A.1 above. To apply these E–R functions to the W126 estimates derived using 24 hours-per-day index values would inaccurately represent the response observed in the study (producing an overestimate). Similarly, with regard to the 3-month duration, “[d]espite the possibility that plants may be exposed to ambient O₃ longer than 3 months in some locations, there is generally a lack of exposure experiments conducted for longer than

²⁰² For example, we note that among the 11 species for which robust E–R functions have been established for O₃ effects on tree seedling growth, the sensitivity of ponderosa pine, a species occurring in arid and high altitude regions of the western U.S., is similar to the median (U.S. EPA, 2014c, Table 5C–1).

²⁰³ No O₃ exposure studies on cacti or other species that utilize CAM photosynthesis are reported in the ISA (U.S. EPA, 2013).

3 months” (U.S. EPA, 2014c, p. 9–112). Thus, in consideration of the lack of support in the current evidence for characterizing exposure for purposes of estimating RBL based on cumulative exposures derived from a combination of daytime and nighttime exposures and consideration of year-round O₃ concentrations across the U.S., we disagree with the commenters’ view of the appropriateness of using an exposure index based on 24-hour, year-round O₃ concentrations.

The commenters supporting the use of the W126 exposure index were divided with regard to whether the EPA should focus on an annual index or one averaged over three years. Some of the commenters indicating support for the EPA’s proposed focus on a 3-year average W126 index stated that this was appropriate in light of the wide variations in W126 index values that can occur on a year-to-year basis as a result of the natural variation of climatic conditions that have a direct impact on O₃ formation; in their view, these factors are mitigated by use of a 3-yr average, which thus provides “stability” in the assessment dampening out the natural variation of climatic conditions that have a direct impact on O₃ formation. Others noted that use of a 3-year average may be supported as matter of policy. We generally concur with the relevance of these points, among others, to a focus on the 3-year average W126. Other commenters expressed the view that the EPA should focus on an annual W126 index, generally making these comments in the context of expressing their support for a secondary standard with a W126 form. These commenters variously cited CASAC advice and its rationale for preferring a single year W126 form, stated that vegetation damage occurs on an annual basis, and/or questioned the EPA’s statements of greater confidence in conclusions as to O₃ impacts based on a 3-year average exposure metric.

The EPA agrees with commenters that, as discussed in the PA and the proposal, depending on the exposure conditions, O₃ can contribute to measurable effects on vegetation in a single year. We additionally recognize that, as described in the PA and proposal, there is generally a greater significance for effects associated with multiple-year exposures. The proposal described a number of considerations raised in the PA as influencing the Administrator’s decision to focus on a 3-year average W126 index (79 FR 75347, December 17, 2014). These included, among others, the observation of a greater significance for effects associated with multiple-year exposures, and the

uncertainties associated with consideration of annual effects relative to multiple-year effects.

Further, we note that among the judgments contributing to the Administrator’s decision on the level of protection appropriate for the secondary standard are judgments regarding the weight to place on the evidence of specific vegetation-related effects estimated to result across a range of cumulative seasonal concentration-weighted O₃ exposures and judgments on the extent to which such effects in such areas may be considered adverse to public welfare (79 FR 75312, December 17, 2014). Thus, conclusions regarding the extent to which the size and/or prevalence of effects on vegetation in a single year and any ramifications for future years represent an adverse effect to the public welfare, conclusions that are also inherently linked to overall magnitudes of exposures, are dependent on the Administrator’s judgment. Accordingly, the decision regarding the need to focus on a 1-year or 3-year W126 index value is also a judgment of the Administrator, informed by the evidence, staff evaluations and advice from CASAC, as described in section IV.C.3 below.

d. Form and Averaging Time

In considering comments received on the proposed form for the revised standard, the EPA first notes the advice and comments from the CASAC, received in its review of the second draft PA. Similar to its advice in the last review, the CASAC recommended “establishing a revised form of the secondary standard to be the biologically relevant W126 index” (Frey, 2014c, p. iii). With regard to its reasons for this view, the CASAC cites the PA in stating that it “concur[s] with the justification in [section 5.7] that the form of the standard should be changed from the current 8-hr form to the cumulative W126 index” (Frey, 2014c, p. 12). In addressing specific aspects of this index, the CASAC concurred with the EPA’s focus on the 3-month period with the highest index value and further states that “[a]ccumulation over the 08:00 a.m.–08:00 p.m. daytime 12-hour period is a scientifically acceptable and recommended means of generalizing across latitudes and seasons” (Frey, 2014c, p. 13). As section 5.7 of the PA discusses the W126 index in the context of the support in the evidence for use of the W126 exposure index for assessing impacts of O₃ on vegetation and the extent of protection from such impacts, we interpret CASAC’s statement on this point to indicate that the basis for CASAC’s view with regard to the form

for the secondary standard relates to the appropriateness of the W126 exposure index for those assessment purposes.²⁰⁴ ²⁰⁵

The public comments on the form for a revised secondary standard were divided. Most of the state and local environmental agencies or governments, and all of the tribal agencies and organizations that provided comments on the form for the secondary standard concurred with the EPA’s proposed decision, as did the industry commenters. These commenters generally indicated agreement with the rationale provided in the proposal that drew from the EPA analyses of recent air quality data examining relationships at sites across the U.S. between values of the fourth-high metric (the current design value) and values of a 3-year average W126-based metric, stating that this analysis showed that a standard in the form of the fourth-high metric, as proposed, can provide air quality consistent with or below the range of 3-year W126 exposure index values identified in the proposal. Some commenters additionally stated that the choice of form was a policy decision for the EPA and that little or no additional protection of public welfare would be gained by adopting a W126-based form. Some of these commenters provided analyses of data for their state or region that further supported this view. As

²⁰⁴ Section 5.7 of the PA states that “the evidence continues to provide a strong basis for concluding that it is appropriate to judge impacts of O₃ on vegetation, related effects and services, and the level of public welfare protection achieved, using a cumulative, seasonal exposure metric, such as the W126-based metric,” references the support of CASAC for a W126-based secondary standard, and then concludes that “based on the consistent and well-established evidence described above, . . . the most appropriate and biologically relevant way to relate O₃ exposure to plant growth, and to determine what would be adequate protection for public welfare effects attributable to the presence of O₃ in the ambient air, is to characterize exposures in terms of a cumulative seasonal form, and in particular the W126 metric” (U.S. EPA, 2014c, p. 5–78).

²⁰⁵ The CASAC also mentioned its support for revising the secondary standard to a W126 index-based form in its review of Chapter 6 of the second draft PA (Frey, 2014c, p. 13). Similar to section 5.7, in that chapter of the PA staff concluded that “specific features associated with the W126 index still make it the most appropriate and biologically relevant cumulative concentration-weighted form for use in the context of the secondary O₃ NAAQS review” (U.S. EPA, 2014c, p. 6–5) and also concluded that “it is appropriate to consider a revised secondary standard in terms of the cumulative, seasonal, concentration-weighted form, the W126 index” (U.S. EPA, 2014c, p. 6–57).

²⁰⁶ The term design value is commonly used to refer to the metric for the standard. Consistent with the summary in section I.D above, a design value is the statistic that describes the air quality of a given location in terms of the indicator, form and averaging time of the standard such that it can then be compared to the level of the standard.

described in section IV.C.3 below, the EPA generally agrees with these commenters.

Some commenters, including a regional organization of state agencies and two groups of environmental organizations, submitted comments recommending revision of the standard to a cumulative, seasonal form based on the W126 index. In support of their position, these commenters generally cited CASAC advice, variously additionally indicating their view that the standard form should be a metric described as biologically relevant, and that the existing form, with a level in the proposed range, would not provide adequate ecosystem protection. Some commenters additionally suggested that the EPA cannot lawfully retain the form and averaging time that were initially established for purposes of the primary standard when the EPA has identified the W126 index as a metric appropriate for judging vegetation-related effects on public welfare. With regard to the EPA air quality analyses, summarized in the proposal, of the W126 index values at sites where O₃ concentrations met different levels of fourth-high metric, some of these commenters stated that the analyses showed widespread variation in W126 values for each fourth-high metric examined. Further, some commenters disagreed with the EPA that the analyses indicated that a revised standard level within the proposed range would be expected to limit W126 exposures in the future to the extent suggested by the analyses of data from the past.

We agree with public commenters and CASAC regarding the appropriateness of the W126 index (the sum of hourly concentrations over a specified period) as a biologically relevant metric for assessing exposures of concern for vegetation-related public welfare effects, as discussed in the proposal, PA and ISA. Accordingly, we agree that this metric is appropriate for use in considering the protection that might be expected to be afforded by potential alternative secondary standards, as discussed in section IV.C.2.c above. We disagree with commenters, however, that use of the W126 metric for this purpose dictates that we must establish a secondary standard with a W126 index form.

In support of this position, we note the common use, in assessments conducted for NAAQS reviews, of exposure metrics that differ in a variety of ways from the ambient air concentration metrics of those

standards.²⁰⁶ Across reviews for the various NAAQS pollutants, we have used a variety of exposure metrics to evaluate the protection afforded by the standards. These exposure metrics are based on the health or welfare effects evidence for the specific pollutant and commonly, in assessments for primary standards, on established exposure-response relationships or health-based benchmarks (doses or exposures of concern) for effects associated with specific exposure circumstances. Some examples of exposure metrics used to evaluate health impacts in primary standard reviews include the concentration of lead in blood of young children and a 5-minute exposure concentration for sulfur dioxide. In contrast, the health-based standards for these two pollutants are the 3-month concentration of lead in total suspended particles and the average across three years of the 99th percentile of 1-hour daily maximum concentration of sulfur dioxide in ambient air, respectively (73 FR 66964, November 12, 2008; 75 FR 35520, June 22, 2010). In somewhat similar manner, in the 2012 PM review, the EPA assessed the extent to which the existing 24-hour secondary standard for PM_{2.5}, expressed as a 24-hour concentration (of PM_{2.5} mass per cubic meter of air) not to be exceeded more than once per year on average over three years, could provide the desired protection from effects on visibility in terms of the 90th percentile, 24-hour average PM_{2.5} light extinction, averaged over three years, based on speciated PM_{2.5} mass concentrations and relative humidity data (79 FR 3086, January 15, 2013). Additionally, in the case of the screening-level risk analyses in the 2008 review of the secondary standard for lead, concentrations of lead in soil, surface water and sediment were evaluated to assess the potential for welfare effects related to lead deposition from air, while the standard is expressed in terms of the concentration of lead in particles suspended in air (73 FR 67009, November 12, 2008).

Further, depending on the evidence base, some NAAQS reviews may consider multiple exposure metrics in assessing risks associated with a particular pollutant in ambient air in order to judge the adequacy of an existing standard in providing the required level of protection. And a standard with an averaging time of one

²⁰⁶The term design value is commonly used to refer to the metric for the standard. Consistent with the summary in section I.D above, a design value is the statistic that describes the air quality of a given location in terms of the indicator, form and averaging time of the standard such that it can then be compared to the level of the standard.

duration may provide protection against effects elicited by exposures of appreciably shorter or longer durations. For example, in the current review of the primary O₃ standard, as described in section II above, we have considered the potential for effects associated with both short- and long-term exposures and concluded, based on a combination of air quality and risk analyses and the health effects evidence, that the existing standard with its short (8-hour) averaging time provides control of both the long and short term exposures (*e.g.*, from one hour to months or years) that may be of concern to public health. Similarly, during the 1996 review of the NO₂ primary standard, while health effects were recognized to result from both long-term and short-term exposures to NO₂, the primary standard, which was a long-term (annual) standard, was concluded to provide the requisite protection against both long- and short-term exposures (61 FR 52852, Oct 8 1996). In the subsequent review of the NO₂ primary standard in which the available air quality information indicated that the annual standard was not providing the needed control of the shorter term exposures, an additional short-term standard was established (75 FR 6474, February 9, 2010).

Thus, we note that different metrics may logically, reasonably, and for technically sound reasons, be used in assessing exposures of concern or characterizing risk as compared to the metric of the standard which is used to control air quality to provide the desired degree of protection. That is, exposure metrics are used to assess the likely occurrence and/or frequency and extent of effects under different air quality conditions, while the air quality standards are intended to control air quality to the extent requisite to protect from the occurrence of public health or welfare effects judged to be adverse. In this review of the secondary standard for O₃, the EPA agrees that, for the reasons summarized in section IV.A.1 above and described in the ISA, the W126 index—and not an 8-hour daily maximum concentration that has relevance in human health risk characterization, as described in section II above—is the appropriate metric for assessing exposures of concern for vegetation, characterizing risk to public welfare, and evaluating what air quality conditions might provide the desired degree of public welfare protection. We disagree, however, that the secondary standard must be established using that same metric.

Moreover, we note that the CAA does not require that the secondary O₃ standard be established in a specific

form. Section 109(b)(2) provides only that any secondary NAAQS “shall specify a level of air quality the attainment and maintenance of which in the judgment of the Administrator, based on [the air quality] criteria, is requisite to protect the public welfare from any known or anticipated adverse effects associated with the presence of such air pollutant in the ambient air. . . . [S]econdary standards may be revised in the same manner as promulgated.” The EPA interprets this provision to leave it considerable discretion to determine whether a particular form is appropriate, in combination with the other aspects of the standard (averaging time, level and indicator), for specifying the air quality that provides the requisite protection, and to determine whether, once a standard has been established in a particular form, that form must be revised. Moreover, nothing in the Act or the relevant case law precludes the EPA from establishing a secondary standard equivalent to the primary standard in some or all respects, as long as the Agency has engaged in reasoned decision-making.²⁰⁷

With regard to the commenter’s emphasis on advice from CASAC on the form of the secondary standard, the EPA agrees with the importance of giving such advice careful consideration. The EPA further notes, however, that the Administrator is not legally precluded from departing from CASAC’s recommendations, when she has provided an explanation of the reasons for such differences.²⁰⁸ Accordingly, in reaching conclusions on the revised secondary standard in this review, the Administrator has given careful consideration to the CASAC advice in this review and, when she has differed from CASAC recommendations, she has fully explained the reasons and judgments that led her to a different conclusion, as described in section IV.C.3 below.

In disagreeing with the EPA’s conclusions drawn from analyses of recent air quality data on the extent to which cumulative seasonal exposures might be limited to within or below the identified 3-year average W126 index values by controlling air quality using different values for the fourth-high

metric, one group of environmental organizations emphasized the range of W126 index values that occur at monitors with concentrations at or below specific values for the fourth-high metric. For monitor observations for which the fourth-high metric was at or below 70 ppb, this commenter group stated that some sites have 3-year average W126 index values above 17 ppm-hrs and noted a maximum 3-year W126 index value of 19.1 ppm-hrs, while additionally noting occurrences of other W126 values above the CASAC range of 7 to 15 ppm-hrs. This commenter additionally stated that the air quality data “do not support a claim of congruence” between the fourth-high and W126 metrics (e.g., ALA et al., p. 196), that there is no basis for concluding that there is some fundamental underlying relationship that assures meeting the fourth-high metric will mean meeting any of the W126 options, and that the relationship between the metrics is non-linear with significant spread in the data (citing visual inspection of a graph).

The EPA does not agree with the commenter’s statements regarding the relationship between the two metrics.²⁰⁹ We have not, as stated by the commenter, claimed there to be “congruence” between the two metrics (e.g., ALA et al., p. 196), or that the two metrics coincide exactly. Rather, at any location, values of both metrics are a reflection of the temporal distribution of hourly O₃ concentrations across the year and both vary in response to changes in that distribution. While the EPA’s air quality analysis shows that the specific relationship differs among individual sites, it documents an overall strong, positive, non-linear relationship between the two metrics (Wells, 2014a, p. 6, Figures 5a and 5b; Wells, 2015b). Further, this analysis finds the amount of year-to-year variability in the two metrics tended to decrease over time with decreasing O₃ concentrations, especially for the W126 metric, as described in section IV.E.4 of the proposal (Wells, 2014a; Wells, 2015b).

With regard to the highest 3-year average W126 exposure index values that might reasonably be expected in the future in areas where a revised standard with a fourth-high form is met, we disagree with the commenters as to the

significance of the W126 index value of 19.1 ppm-hrs in the 13-year dataset. This value, for a site during the period 2006–2008, is the only occurrence at or above 19 ppm-hrs in the nearly 4000 3-year W126 index values—across the 11 3-year periods extending back in time from 2013—for which the fourth-high metric for the same monitor location is at or below 70 ppb. This is clearly an isolated occurrence.

In considering this comment, we have expanded the technical memorandum that was available at the time of proposal (Wells, 2014a). The expanded memorandum describes the same air quality analyses for 3-year periods from 2001 through 2013 as the 2014 memorandum, and includes additional summary tables for all 3-year periods from 2001 through 2013 as well as tables for the most recent period, 2011–2013 (Wells, 2015b). After the 3-year W126 index value of 19 ppm-hrs, the next three highest 3-year average W126 index values, which are the only other such values above 17 ppm-hrs in the 13-year dataset, and which also occur during periods in the past, round to 18 ppm-hrs (Wells, 2015b). Additionally, we note that reductions in the fourth-high metric over the 13-year period analyzed are strongly associated with reductions in the cumulative W126 index (Wells, 2014a, Figure 11, Table 6; Wells, 2015b). Specifically, the regression analysis of changes in W126 index between the 2001–2003 period and the 2011–2013 period with changes in the fourth-high metric across the same periods indicates a fairly linear and positive relationship between reductions of the two types of metrics, with, on average, a change of approximately 0.7 ppm-hr in the W126 index per ppb change in the fourth-high metric value. From this information we conclude that W126 exposures above 17 ppm-hrs at sites for which the fourth-high metric is at or below 70 ppb would be expected to continue to be rare in the future, particularly as steps are taken to meet a 70 ppb standard.

With regard to the comment that the relationship between the two metrics varies across locations, the EPA agrees that there is variation in cumulative seasonal O₃ exposure (in terms of a 3-year average W126 index) among locations that are at or below the same fourth-high metric. As noted in the proposal, the analysis illustrates this variation, with the locations in the West and Southwest NOAA climatic regions tending to have the highest cumulative seasonal exposures for the same fourth-high metric value. In considering expectations for the future in light of this observation, however, we note that

²⁰⁷ In fact, the D.C. Circuit has upheld secondary NAAQS that were identical to the corresponding primary standard for the pollutant (e.g., *ATA III*, 283 F.3d at 375, 380 [D.C. Cir. 2002, upholding secondary standards for PM_{2.5} and O₃ that were identical to primary standards]).

²⁰⁸ See CAA sections 307(d)(3) and 307(d)(6)(A); see also *Mississippi v. EPA*, 744 F.3d 1334, 1354 (D.C. Cir. 2013) (“Although EPA is not bound by CASAC’s recommendations, it must fully explain its reasons for any departure from them”).

²⁰⁹ The EPA additionally notes that commenters contradict their own assertion when, after stating their view that no relationship exists between the 4th high and W126 metrics, the commenter then states that there is a nonlinear relationship and yet then relies on a predicted *linear* relationship to estimate W126 values occurring when air quality meets different values for the 4th high metric at 11 national parks.

the regional regressions of reductions in W126 metric with reductions in the fourth-high metric indicate that the Southwest and West regions, which had the greatest potential for sites having 3-year W126 index values greater than the various W126 values of interest when fourth-high values are less than or equal to the various fourth-high metric values of interest, also exhibited the greatest reduction in the W126 index values per unit reduction in the fourth-high values (Wells, 2015b). Thus, in considering the potential for occurrences of values above 17 ppm-hrs in the future in areas that meet a fourth-high of 70 ppb, the EPA notes that the analysis indicates that those areas that exhibited the greatest likelihood of occurrence of a 3-year W126 index above a level of interest (e.g., the commenters' example in the Southwest region of a value of 19.1 ppm-hrs [2006–2008] in comparison to the W126 level of 17 ppm-hrs) also exhibit the greatest improvement in W126 per unit decrease in fourth-high metric.²¹⁰ It is expected that future control programs designed to meet a standard with a fourth-high form would provide similar improvements in terms of the W126 metric.

As part of their rationale in support of revising the current form and averaging time, one commenter pointed to the regional variation in the highest W126 index values expected at sites that just meet a fourth-high metric of 70 ppb, based on the EPA's analysis of recent air quality data available at the time of the proposal (Wells, 2014a). This commenter observed that, while in some U.S. regions, locations that meet a potential alternative standard with the current form and a level of 70 ppb also have 3-year average W126 index values no higher than 17 ppm-hrs, the highest W126 index values in other parts of the country are lower. As a result, the commenter concluded that such a standard would result in regionally differing levels of welfare protection. The commenter additionally states that, for extreme values, a W126 form for the secondary standard would also offer different levels of protection, although with the primary standard setting the upper boundary for such values.

The EPA recognizes that a standard with the current form might be expected to result in regionally differing

distributions of W126 exposure index values (including different maximum values) depending on precursor sources, local meteorology, and patterns of O₃ formation. Variation in exposures is to be expected with any standard (secondary or primary) of any form. In fact, variation in exposures and any associated variation in welfare or health risk is generally an inherent aspect of the Administrator's judgment on a specific standard, and any associated variation in welfare or health protection may play a role in the Administrator's judgment with regard to public welfare or public health protection objectives for a national standard. In considering the comment, however, we have focused only on the extent to which the commenter's conclusion that a secondary standard of the current form and averaging time would provide regionally varying welfare protection might indicate that the specified air quality is more (or less) than necessary to achieve the purposes of the standard. In so doing, we additionally respond to a separate comment that the EPA needs to address how the revised secondary standard is neither more or less than necessary to protect the public welfare.

The CAA requirement in establishing a standard is that it be set at a level of air quality that is requisite, meaning "sufficient, but not more than necessary" (*Whitman v. American Trucking Ass'n*, 531 U.S. 457, 473 [2001]). We note that the air quality that is specified by the revised primary standard has been concluded to be "necessary" and it may be reasonable and appropriate to consider the stringency of the secondary standard in light of what is identified as "necessary" for the primary standard. The EPA considered the stringency of the O₃ secondary standard in this way in the 1979 decision (44 FR 8211, February 8, 1979), which was upheld in subsequent litigation (*API v. Costle*, 665 F.2d 1176 [D.C. Cir. 1991]). We note that, in similar manner, the commenter considered public welfare protection that might be afforded by the primary standard in noting that the primary standard would be expected to provide welfare protection from extreme values.²¹¹

²¹¹ As described earlier in this section, the EPA has also considered the air quality specified by one secondary standard in a decision on the need for a second secondary standard. In the decision not to adopt a second PM_{2.5} secondary standard specific to visibility-related welfare effects, the Administrator, after describing the public welfare protection objective related to visibility effects, considered analyses that related air quality associated with the existing secondary standard to that expected for the proposed visibility-focused secondary standard. From these analyses, she

In addressing the remand of the 2008 secondary standard in this rulemaking, as discussed in section IV.C.2.e below, the EPA recognizes that it must explain the basis for concluding that the standard selected by the Administrator specifies air quality that will provide the degree of public welfare protection needed from the secondary standard (*Mississippi v. EPA*, 744 F.3d 1334, 1360–61 [D.C. Cir. 2013]). In this review, the Administrator describes the degree or level of public welfare protection needed from the secondary standard and fully explains the basis for concluding that the standard selected specifies air quality that will provide that degree of protection. If the Administrator concludes that the level of air quality specified by the primary standard would provide sufficient protection against known or anticipated adverse public welfare effects, the EPA believes that a secondary standard with that indicator, level, form and averaging time could be considered to be requisite. If the level of air quality that areas will need to achieve or maintain for purposes of the primary standard also provides a level of air quality that is adequate to provide the level of protection identified for the secondary standard, there would be little purpose in requiring the EPA to establish a less stringent secondary standard. For these reasons, the expectation of regionally differing cumulative exposures under a secondary standard of the current form and averaging time does not lead us to conclude that the air quality specified by such a standard would be more (or less) than necessary (and thus not requisite) for the desired level of public welfare protection.

e. Revisions to the Standard Level

Some comments specifically addressed the level for a revised secondary standard of the current form and averaging time. Of the comments that addressed this, some from states or industry groups generally supported a level within the proposed range, frequently specifying the upper end of the range (70 ppb), while comments

concluded sufficient protection against visibility effects would be provided by the existing standard, and to the extent that the existing standard would provide more protection than had been her objective for such effects, adoption of a second secondary standard focused on visibility would not change that result (78 FR 3227–3228, January 15, 2013). This decision responded to a court remand of the prior EPA decision that visibility protection would be afforded by a secondary standard set equal to the primary standard based on the court's conclusion that the EPA had not adequately described the Administrator's objectives for visibility-related public welfare protection under the standard (*American Farm Bureau*, 559 F.3d at 530–531).

from tribes and tribal organizations, and a few others, recommended a level no higher than 65 ppb. The Administrator has considered such comments in reaching her decision on the appropriate revisions to the standard, described in section IV.C.3. Detailed aspects of these comments are discussed in the Response to Comments document.

f. 2013 Court Remand and Levels of Protection

Both industry groups and a group of environmental advocacy organizations submitted comments on the extent to which the proposal addressed the July 2013 remand of the secondary standard by the U.S. Court of Appeals for the D.C. Circuit. The former generally concluded that the proposal had adequately addressed the remand, while the latter expressed the view that the EPA had failed to comply with the court's remand because it had failed to identify the target levels of vegetation protection for which the proposed range of standards would provide the requisite protection, claiming that the identified W126 index range of 13–17 ppm-hrs was not based on a proposed level of protection against biomass loss, carbon storage loss, or foliar injury that the EPA had identified as requisite for public welfare.

We agree with the comments that state that we have addressed the court's remand. More specifically, with this rulemaking, including today's decision and the Administrator's conclusions described in section IV.C.3 below, the EPA has fully addressed the remand of the 2008 secondary O₃ standard. In *Mississippi v. EPA*, the D.C. Circuit remanded the 2008 secondary O₃ standard to the EPA for reconsideration because it had not adequately explained why that standard provided the requisite public welfare protection. 744 F.3d 1334, 1360–61 (D.C. Cir. 2013). In doing so, the court relied on the language of CAA section 109(b)(2), and the court's prior decision, *American Farm Bureau Federation v. EPA*, 559 F.3d 512, 528–32 (D.C. Cir. 2009), which came to the same conclusion for the 2006 secondary PM_{2.5} standard. Both decisions recognize that the plain language of section 109(b)(2) requires the EPA to “specify a level of air quality the maintenance of which . . . is requisite to protect the public welfare from any known or anticipated adverse effects” (*Mississippi*, 744 F.3d at 1360 [citing *American Farm Bureau*, 559 F.3d at 530]). Further, explaining that it was insufficient for the EPA “merely to compare the level of protection afforded by the primary standard to possible secondary standards and to find the two

roughly equivalent” (*Mississippi*, 744 F.3d at 1360), the court rejected the EPA's justification for setting the secondary standard equivalent to the primary standard because that justification was based on comparing the protection from the primary standard to that expected from one possible standard with a cumulative, seasonal form (21 ppm-hrs) without stating that such a cumulative seasonal standard would be requisite to protect welfare or explaining why that would be so. Because the EPA had “failed to determine what level of protection was ‘requisite to protect the public welfare’” (*Mississippi*, 744 F.3d at 1362), the court found that the EPA's rationale failed to satisfy the requirements of the Act.

Today's rulemaking both satisfies the requirements of section 109(b)(2) of the Act and addresses the issues raised in the court's remand. In this rulemaking, the Administrator has established a revised secondary standard that replaces the remanded 2008 secondary standard. In so doing, based on her consideration of the currently available evidence and quantitative exposure and air quality information, as well as advice from CASAC and input from public comments, the Administrator has described the requisite public welfare protection for the secondary standard and explained how the standard selected specifies air quality that will provide that protection. As explained in detail in IV.C.3 below, in this review the Administrator is describing the public welfare protection she finds requisite in terms of seedling RBL in the median species, which serves as a surrogate for a broader array of O₃ effects at the plant and ecosystem levels. This description of the desired protection sufficiently articulates the standard that the Administrator is using to evaluate welfare protection. Further, the Administrator has considered air quality analyses in determining how to achieve the air quality conditions associated with the desired protection. Based on these analyses, the Administrator is determining that revising the level of the secondary standard to 70 ppb, while retaining the current form, averaging time, and indicator, specifies a level of air quality that will provide the requisite public welfare protection.

To the extent the comments suggest that the EPA is required in establishing a standard to identify a precise and quantified level of public welfare protection that is requisite with respect to every potentially adverse public welfare impact (e.g., visible foliar injury, crop yield loss) that is considered in establishing the standard, we disagree. While the D.C. Circuit has required the

EPA to “qualitatively describe the standard governing its selection of particular NAAQS,” it has expressly “rejected the notion that the Agency must establish a measure of the risk to safety it considers adequate to protect public health every time it establishes a NAAQS” (*ATA III*, 283 F.3d at 369 [internal marks and citations omitted]). That is, the EPA must “engage in reasoned decision-making,” but is not required to “definitively identify pollutant levels below which risks to public health are negligible” (*ATA III*, 283 F.3d at 370). This principle recognizes that the Act requires the EPA to establish NAAQS even when the risks or effects of a pollutant cannot be quantified or precisely identified because of scientific uncertainty concerning such effects at atmospheric concentrations (*ATA III*, 283 F.3d at 370). Though these decisions specifically address setting a primary standard under CAA section 109(b)(1), we believe the same principles apply to the parallel provision in section 109(b)(2) governing secondary standards. Accordingly, while the EPA recognizes that it must explain the basis for concluding that the standard selected by the Administrator specifies air quality that will provide the protection against adverse effects on public welfare needed from the secondary standard (*Mississippi v. EPA*, 744 F.3d 1334, 1360–61 [D.C. Cir. 2013]), the CAA does not require the EPA to precisely quantify the measure of protection that is necessary to protect the public welfare in establishing a secondary standard. In light of the Administrator's description of the desired public welfare protection in IV.C.3 below, which has both qualitative and quantitative components, the EPA is not required to further reduce this description to a precise, quantitative target level of vegetation protection. Moreover, nothing in the CAA or in case law requires the EPA to identify a target level of protection for any particular public welfare effect, such as vegetation effects, but rather leaves the Administrator discretion in judging how to describe the public welfare protection that she concludes is requisite. In IV.C.3 below, the Administrator explains her reasoning for giving primary focus to growth-related effects in describing the requisite welfare protection, rather than to other welfare effects such as foliar injury, for which there are more uncertainties and less predictability with respect to the severity of the effects that would be expected from varying O₃ exposures in the natural environment

and the significance of the associated impacts to public welfare.

3. Administrator's Conclusions on Revision

In reaching her decision on the appropriate revisions to the secondary standard, the Administrator has drawn on (1) the ISA conclusions regarding the weight of the evidence for a range of welfare effects associated with O₃ in ambient air, quantitative findings regarding air quality and ecosystem exposures associated with such effects, and associated limitations and uncertainties; (2) staff evaluations in the PA of the evidence summarized in the ISA, the exposure/risk information developed in the WREA and analyses of air quality monitoring information; (3) additional air quality analyses of relationships between air quality metrics based on form and averaging time of the current standard and the W126 cumulative seasonal exposure index; (4) CASAC advice; and (5) consideration of public comments. After giving careful consideration to all of this information, the Administrator believes that the conclusions and policy judgments supporting her proposed decision remain valid.

The Administrator concludes it is appropriate to continue to use O₃ as the indicator for a secondary standard intended to address adverse effects to public welfare associated with exposure to O₃ alone and in combination with related photochemical oxidants. In this review, no alternatives to O₃ have been advanced as being a more appropriate surrogate for ambient photochemical oxidants. Advice from CASAC concurs with the appropriateness of retaining the current indicator. Thus, as is the case for the primary standard (discussed above in section II.C.1), the Administrator has decided to retain O₃ as the indicator for the secondary standard. In so doing, she recognizes that measures leading to reductions in ecosystem exposures to O₃ would also be expected to reduce exposures to other photochemical oxidants.

In her decision on the other elements of the standard, the Administrator has considered the body of evidence and information in a systematic fashion, giving appropriate consideration to the important findings of the ISA as to the effects of O₃ in ambient air that may present risks to the public welfare, measures of exposure best formulated for assessment of these effects, associated evidence regarding ecosystem exposures and air quality associated with such effects; judgments regarding the weight to place on strengths, limitations and uncertainties

of this full body of information; and public welfare policy judgments on the appropriate degree of protection and the form and level of a revised standard that will provide such protection. In reaching her decision, the Administrator recognizes that the Act does not require that NAAQS be set at zero-risk or background levels, but rather at levels that reduce risk sufficiently to protect public welfare from known or anticipated adverse effects. In addition, we note that the elements of the standard (indicator, level, form, and averaging time) are considered together in assessing the protection provided by a new or revised standard, and the EPA's approach for considering the elements of a new or revised standard is part of the exercise of the judgment of the Administrator.

As an initial matter, the Administrator recognizes the robustness of the longstanding evidence, described in the ISA, of O₃ effects on vegetation and associated terrestrial ecosystems. The newly available studies and analyses have strengthened the evidence for the current review that provides the foundation for the Administrator's consideration of O₃ effects, associated public welfare protection objectives, and the revisions to the current standard needed to achieve those objectives. In light of the extensive evidence base in this regard, the Administrator focuses on protection against adverse public welfare effects of O₃ related effects on vegetation. In so doing, she takes note of effects that compromise plant function and productivity, with associated effects on ecosystems. She is particularly concerned about such effects in natural ecosystems, such as those in areas with protection designated by Congress for current and future generations, as well as areas similarly set aside by states, tribes and public interest groups with the intention of providing similar benefits to the public welfare. She additionally recognizes that providing protection for this purpose will also provide a level of protection for other vegetation that is used by the public and potentially affected by O₃ including timber, produce grown for consumption and horticultural plants used for landscaping.

A central issue in this review of the secondary standard, as in the last review (completed in 2008), has been consideration of the role for a cumulative seasonal exposure index. In the last review, the Administrator proposed such an index as one of two options for the form of a revised standard. The Administrator's decision in that review was to retain the existing

form and averaging time, while revising the standard level to provide the desired level of protection. As described in section IV.A above, this decision was remanded to the EPA in 2013 by the DC Circuit. In the current review, the ISA evaluates the evidence and concludes that, among the approaches investigated, quantifying exposure with a cumulative seasonal index best captures the aspects of exposure that relate to effects on vegetation, particularly those related to growth and yield. The PA considered this finding both in the context of assessing potential impacts, and, conversely, the protection from such impacts that might be realized, as well as in the context of using a cumulative seasonal exposure index as a form for the secondary standard. In the proposal, the Administrator focused on the former context, as an exposure index, while additionally soliciting comment on use of the index as the form for the revised standard. Advice from CASAC, all of which was received prior to the proposal, has largely emphasized the latter context, and that was also the focus of some comments.

In considering revisions to the secondary standard that will specify a level of air quality to provide the necessary public welfare protection, the Administrator focuses on use of a cumulative seasonal exposure index, including specifically the W126 index as defined in the proposal, for assessing exposure, both for making judgments with regard to the potential harm to public welfare posed by conditions allowed by various levels of air quality and for making the associated judgments regarding the appropriate degree of protection against such potential harm. In so doing, the Administrator takes note of the conclusions in the ISA and PA, with which the CASAC concurred, that, based on the currently available evidence, a cumulative seasonal concentration-weighted index best captures the aspects of ecosystem exposure to O₃ in ambient air that impact vegetation. In considering the public comments in this area, she notes the broad support for use of such a metric as an exposure index, with many additionally supporting its use as the form for a revised standard, in light of CASAC advice on that point. Thus, based on the substantial support in the evidence and CASAC advice, and in consideration of public comments, the Administrator concludes that it is appropriate to use such a cumulative seasonal concentration-weighted index for purposes of assessing the potential

public welfare risks, and similarly, for assessing the potential protection achieved against such risks on a national scale.

The Administrator has considered conclusions of the ISA and PA, as well as advice from CASAC and public comments, regarding different cumulative, concentration-weighted metrics, and different temporal definitions of aspects of these metrics. The Administrator takes note of the PA conclusions in support of the W126 exposure index, recognized by the ISA for its strength in weighting potentially damaging O₃ concentrations that contributes to the advantages it offers over other weighted cumulative indices. With regard to the relevant definitions for the temporal aspects of this index, conclusions in the ISA and PA, and such considerations in the last review, have led to a focus on a maximum 3-month, 12-hour index, defined by the 3-consecutive-month period within the O₃ season with the maximum sum of W126-weighted hourly O₃ concentrations during the period from 8:00 a.m. to 8:00 p.m. each day (as explained in section IV.A.1.c above). The Administrator takes note of the support in the ISA and PA, as well as CASAC recommendations for consideration of the W126 index defined in this way. While recognizing that no one definition of an exposure metric used for the assessment of protection for multiple effects at a national scale will be exactly tailored to every species or each vegetation type, ecosystem and region of the country, as discussed in section IV.C.2 above, the Administrator judges that on balance, a W126 index derived in this way, and averaged over three years, as discussed below, will be appropriate for such purposes.

In considering the appropriate exposure index to facilitate assessment of the level of protection afforded to the public welfare by alternative secondary standards in the proposal, the Administrator concluded that a 3-year average W126 index was appropriate for these purposes. A number of considerations raised in the PA influenced the Administrator's conclusion at the time of proposal, in combination with public welfare judgments regarding the weight to place on the evidence of specific vegetation-related effects estimated to result across a range of cumulative seasonal concentration-weighted O₃ exposures and judgments on the extent to which such effects in such areas may be considered adverse to public welfare (79 FR 76347, 75312, December 17, 2014.). Some comments were received from the

public on this aspect of the proposed decision, as discussed in section IV.C.2 above, and have been considered in the conclusions reached here.

The Administrator continues to place weight on key aspects raised in the PA and summarized in the proposal on the appropriateness of considering a 3-year average index. The Administrator notes the PA consideration of the potential for multiple consecutive years of critical O₃ exposures to result in larger impacts on forested areas than intermittent occurrences of such exposures due to the potential for compounding effects on tree growth. The Administrator additionally notes the evidence, as considered in the PA and summarized in the proposal, for some perennial species of some effects associated with a single year's exposure of a critical magnitude that may have the potential for some "carry over" of effects on plant growth or reproduction in the subsequent season. Further, the Administrator notes the occurrence of visible foliar injury and growth or yield loss in annual plants or crops associated with exposures of a critical magnitude. While the Administrator appreciates that the scientific evidence documents the effects on vegetation resulting from individual growing season exposures of specific magnitude, including those that can affect the vegetation in subsequent years, she is also mindful, both of the strengths and limitations of the evidence, and of the information on which to base her judgments with regard to adversity of effects on the public welfare. The Administrator also recognizes uncertainties associated with interpretation of the public welfare significance of effects resulting from a single-year exposure, and that the public welfare significance of effects associated with multiple years of critical exposures are potentially greater than those associated with a single year of such exposure.

As she did for the proposal, the Administrator has considered advice from CASAC in this area, including the CASAC comments that it favors a W126-based secondary standard with a single year form, that its recommended range of levels relates to such a form, and that a lower range (e.g., with 13 ppm-hrs at the upper end) would pertain to a 3-year form. The Administrator also notes CASAC's recognition that her decision on use of a 3-year average over a single-year W126 index may be a matter of policy. While recognizing the potential for effects on vegetation associated with a single-year exposure, the Administrator concludes that use of a 3-year average metric can address the potential for adverse effects to public

welfare that may relate to shorter exposure periods, including a single year.

While the Administrator recognizes the scientific information and interpretations, as well as CASAC advice, with regard to a single-year exposure index, she also takes note of uncertainties associated with judging the degree of vegetation impacts for annual effects that would be adverse to public welfare. Even in the case of annual crops, the assessment of public welfare significance is unclear for the reasons discussed below related to agricultural practices. The Administrator is also mindful of the variability in ambient air O₃ concentrations from year to year, as well as year-to-year variability in environmental factors, including rainfall and other meteorological factors, that influence the occurrence and magnitude of O₃-related effects in any year, and contribute uncertainties to interpretation of the potential for harm to public welfare over the longer term. As noted above, the Administrator also recognizes that the public welfare significance of effects associated with multiple years of critical exposures are potentially greater than those associated with a single year of such exposure. Based on all of these considerations, the Administrator recognizes greater confidence in judgments related to public welfare impacts based on a 3-year average metric. Accordingly, the considerations identified here lead the Administrator to conclude it is appropriate to use an index averaged across three years for judging public welfare protection afforded by a revised secondary standard.

In reaching a conclusion on the amount of public welfare protection from the presence of O₃ in ambient air that is appropriate to be afforded by a revised secondary standard, the Administrator has given particular consideration to the following: (1) The nature and degree of effects of O₃ on vegetation, including her judgments as to what constitutes an adverse effect to the public welfare; (2) the strengths and limitations of the available and relevant information; (3) comments from the public on the Administrator's proposed decision, including comments related to identification of a target level of protection; and (4) CASAC's views regarding the strength of the evidence and its adequacy to inform judgments on public welfare protection. The Administrator recognizes that such judgments include judgments about the interpretation of the evidence and other information, such as the quantitative analyses of air quality monitoring,

exposure and risk. She also recognizes that such judgments should neither overstate nor understate the strengths and limitations of the evidence and information nor the appropriate inferences to be drawn as to risks to public welfare. The CAA does not require that a secondary standard be protective of all effects associated with a pollutant in the ambient air but rather those known or anticipated effects judged adverse to the public welfare (as described in section IV.A.3 above). The Administrator additionally recognizes that the choice of the appropriate level of protection is a public welfare policy judgment entrusted to the Administrator under the CAA taking into account both the available evidence and the uncertainties.

The Administrator finds the coherence and strength of the weight of evidence concerning effects on vegetation from the large body of available literature compelling. The currently available evidence addresses a broad array of O₃-induced effects on a variety of tree species across a range of growth stages (*i.e.*, seedlings, saplings and mature trees) using diverse field-based (*e.g.*, free air, gradient and ambient) and OTC exposure methods. The Administrator gives particular attention to the effects related to native tree growth and productivity, recognizing their relationship to a range of ecosystem services, including forest and forest community composition. She is also mindful of the significance of community composition changes, particularly in protected areas, such as Class I areas. At the same time, she recognizes, while the evidence strongly supports conclusions regarding O₃ impacts on growth and the evidence showing effects on tree seedlings, as well as on older trees, there are limitations in our ability to predict impacts in the environment or to estimate air quality or exposures that will avoid such impacts. Such limitations relate to the variability of environmental factors or characteristics that can influence the extent of O₃ effects.

In recognition of the CASAC advice and the potential for adverse public welfare effects, the Administrator has considered the nature and degree of effects of O₃ on the public welfare. In so doing, the Administrator recognizes that the significance to the public welfare of O₃-induced effects on sensitive vegetation growing within the U.S. can vary, depending on the nature of the effect, the intended use of the sensitive plants or ecosystems, and the types of environments in which the sensitive vegetation and ecosystems are located.

Any given O₃-related effect on vegetation and ecosystems (*e.g.*, biomass loss, visible foliar injury), therefore, may be judged to have a different degree of impact on the public depending, for example, on whether that effect occurs in a Class I area, a residential or commercial setting, or elsewhere. The Administrator notes that such a distinction is supported by CASAC advice in this review. In her judgment, like those of the Administrator in the last review, it is appropriate that this variation in the significance of O₃-related vegetation effects should be taken into consideration in making judgments with regard to the level of ambient O₃ concentrations that is requisite to protect the public welfare from any known or anticipated adverse effects. As a result, the Administrator concludes that of those known and anticipated O₃-related vegetation and ecosystem effects identified and discussed in this notice, particular significance should be ascribed to those that may occur on sensitive species that are known to or are likely to occur in federally protected areas such as Class I areas or on lands set aside by states, tribes and public interest groups to provide similar benefits to the public welfare, for residents on those lands, as well as visitors to those areas.

Likewise, the Administrator also notes that less protection related to growth effects may be called for in the case of other types of vegetation or vegetation associated with other uses or services. For example, the maintenance of adequate agricultural crop yields is extremely important to the public welfare and currently involves the application of intensive management practices. With respect to commercial production of commodities, the Administrator notes that judgments about the extent to which O₃-related effects on commercially managed vegetation are adverse from a public welfare perspective are particularly difficult to reach, given that the extensive management of such vegetation (which, as CASAC noted, may reduce yield variability) may also to some degree mitigate potential O₃-related effects. The management practices used on these lands are highly variable and are designed to achieve optimal yields, taking into consideration various environmental conditions. In addition, changes in yield of commercial crops and commercial commodities, such as timber, may affect producers and consumers differently, further complicating the question of assessing overall public welfare impacts. Thus, the Administrator

concludes, while research on agricultural crop species remains useful in illuminating mechanisms of action and physiological processes, information from this sector on O₃-induced effects is considered less useful in informing judgments on what specific standard would provide the appropriate public welfare protection. In so doing, the Administrator notes that a standard revised to increase protection for forested ecosystems would also be expected to provide some increased protection for agricultural crops and other commercial commodities, such as timber.

The Administrator also recognizes that O₃-related effects on sensitive vegetation can occur in other areas that have not been afforded special federal or other protections, including effects on vegetation growing in managed city parks and residential or commercial settings, such as ornamentals used in urban/suburban landscaping or vegetation grown in land use categories involving commercial production of commodities, such as timber. For vegetation used for residential or commercial ornamental purposes, the Administrator believes that there is not adequate information at this time to establish a secondary standard based specifically on impairment of these categories of vegetation, but notes that a secondary standard revised to provide protection for sensitive natural vegetation and ecosystems would likely also provide some degree of protection for such vegetation.

Based on the above considerations, in identifying the appropriate level of protection for the secondary standard, the Administrator finds it appropriate to focus on sensitive trees and other native species known or anticipated to occur in protected areas such as Class I areas or on other lands set aside by the Congress, states, tribes and public interest groups to provide similar benefits to the public welfare, for residents on those lands, as well as visitors to those areas. In light of their public welfare significance, the Administrator gives particular weight to protecting such vegetation and ecosystems. Given the reasons for the special protection afforded such areas (identified in section I.A.3 above), she recognizes the importance of protecting these natural forests from O₃-induced impacts, including those related to O₃ effects on growth, and including those extending in scale from individual plants to the ecosystem. The Administrator also recognizes that the impacts identified for O₃ range from those for which the public welfare significance may be more easily judged, but for which quantitative relationships

with O₃ in ambient air are less well established, such as impacts on forest community composition in protected wilderness areas, carbon storage and other important ecosystem services, to specific plant-level effects, such as growth impacts (in terms of RBL) in tree seedlings, for which our quantitative estimates are more robust.

For considering the appropriate public welfare protection objective for a revised standard, the Administrator finds appropriate and useful the estimates of tree seedling growth impacts (in terms of RBL) associated with a range of W126-based index values developed from the robust E-R functions for 11 tree species, that were described in the PA and proposal and are summarized in Table 4 above. In making judgments based on those observations, however, the Administrator has considered the broader evidence base and public welfare implications, including associated strengths, limitations and uncertainties. Thus, in drawing on estimates from this table, she is not making judgments simply about a specific magnitude of growth effect in seedlings that would be acceptable or unacceptable in the natural environment. Rather, the Administrator is using the estimates in the table, as suggested by CASAC and emphasized by some commenters, as a surrogate or proxy for consideration of the broader array of vegetation-related effects of potential public welfare significance, that include effects on growth of individual sensitive species and extend to ecosystem-level effects, such as community composition in natural forests, particularly in protected public lands, as well as forest productivity. In so doing, she notes that CASAC similarly viewed biomass loss as “a scientifically valid surrogate of a variety of adverse effects to public welfare” (Frey, 2014c, p. 10). Thus, in considering the appropriate level of public welfare protection for the revised standard, the Administrator gives primary attention to the relationship between W126 exposures and estimates of RBL in tree seedlings in Table 4, finding this to be a useful quantitative tool to inform her judgments in this matter.

In considering the RBL estimates in Table 4 above (drawn from the final PA), the Administrator takes note of comments from CASAC that also give weight to these relationships in formulating its advice and notes the CASAC comments on specific RBL values (Frey, 2014c). In so doing, she considers and contrasts comments and

their context on RBL estimates of 2% and 6% for the median studied species.

With regard to the CASAC advice regarding 2% RBL for the median studied tree species, the Administrator notes, as an initial matter, the unclear basis for such a focus, as described in section IV.C.2 above and in the proposal. Further, she notes that the CASAC advice related to this RBL value was that it would be appropriate for the range of levels identified in the PA for the Administrator’s consideration to “include[] levels that aim for not greater than 2% RBL for the median tree species” (Frey, 2014c, p. 14). As described in the proposal, the range identified in the PA, which the Administrator considered, extended down to W126 index levels for which the estimated RBL in the median tree species is less than or equal to 2%, consistent with the CASAC advice. In addition, the Administrator notes that only the lowest portion of this range (7–8 ppm-hrs) corresponds to an estimated RBL for the median tree species of less than or equal to 2%, with the remainder of CASAC’s range (up to 15 ppm-hrs) associated with higher median RBL estimates. Thus, the Administrator understands CASAC to have identified 2% RBL for the median tree species as a benchmark falling within, and at one end of, the range of levels of protection that the CASAC considers appropriate for the revised standard to provide. However, the fact that the CASAC range included levels for which the RBL estimates were appreciably greater than 2% indicates that CASAC did not judge it necessary that the revised standard be based on the 2% RBL benchmark. Accordingly, the Administrator proposed revisions to the secondary standard based on options related to higher RBL estimates and associated exposures. After also considering public comments, the Administrator continues to consider the uncertainty regarding the extent to which associated effects on vegetation at lower O₃ exposures would be adverse to public welfare to be too great to provide a foundation for public welfare protection objectives for a revised secondary standard.

With regard to the CASAC comments on a 6% RBL estimate, the Administrator takes particular note of their characterization of this level of effect in the median studied species as “unacceptably high” (Frey, 2014c, pp. iii, 13, 14). These comments were provided in the context of CASAC’s considering the significance of effects associated with a range of alternatives for the secondary standard. Moreover, the range recommended by CASAC excluded W126 index values for which

the median species was estimated to have a 6% RBL,²¹² based on the information before CASAC at the time (Frey, 2014c, p. 12–13). Accordingly, the EPA interprets these comments regarding 6% RBL to be of a different nature than the CASAC advice regarding a 2% median RBL, both because these two comments are framed to address different questions and because CASAC treated them differently in its recommended range.

In the Administrator’s consideration of the RBL estimates to inform judgments on O₃ exposures of concern to public welfare and the appropriate protection that the secondary standard should provide from such exposures, she has given particular consideration to the current evidence for the relationship of reduced growth of sensitive tree species with ecosystem effects (as described in the ISA), CASAC’s view of 6% RBL for the median studied species as unacceptably high, and the role of the Administrator’s judgments regarding public welfare impacts of effects in specially protected natural systems, such as Class I areas. With regard to a point of focus among the median RBL estimates extending below 6% for purposes of judging the appropriate public welfare protection objectives for a revised secondary standard, the Administrator is mindful of the CASAC advice to consider lower levels if using a 3-year average, rather than annual, W126 index value.

In considering the CASAC advice, the Administrator notes that her judgments on a 3-year average index focus on the level of confidence in conclusions that might be drawn with regard to single as compared to multiple year impacts, as described above. For example, the Administrator, while recognizing the strength of the evidence with regard to quantitative characterization of O₃ effects on growth of tree seedlings and crops, and in addition to noting the additional difficulties for assessing the welfare impacts of O₃ on crops, takes note of the uncertainty associated with

²¹² As summarized in IV.C.2 above (and noted in section IV.E.3 of the proposal), revisions to this table in the final PA, made in consideration of other CASAC comments, have resulted in changes to the median species RBL estimates such that the median species RBL estimate for a W126 index value of 17 ppm-hrs in this table in the final PA (5.3%) is nearly identical to the median species estimate for 15 ppm-hrs (the value corresponding to the upper end of the CASAC-identified range) in the second draft PA (5.2%), the review of which was the context for CASAC’s advice on this point (Frey, 2014c). The median RBL estimate ranges from 5.3% to 3.8% across the range of W126 exposures (17 ppm-hrs to 13 ppm-hrs) that the Administrator proposed to conclude would provide the appropriate public welfare protection for a revised secondary standard.

drawing conclusions with regard to the extent to which small percent reductions in annual growth contribute to adverse effects on public welfare and the role of annual variability in environmental factors that affect plant responses to O₃. Moreover, as explained above, the Administrator concludes that concerns related to the possibility of a single unusually damaging year, inclusive of those described by the CASAC, can be addressed through use of a 3-year average metric. Thus, similar to the CASAC's view that a lower level would be appropriate with a 3-year form, the Administrator considers it appropriate to focus on a standard that would generally limit cumulative exposures to those for which the median RBL estimate would be somewhat lower than 6%.

In focusing on cumulative exposures associated with a median RBL estimate somewhat below 6%, the Administrator considers the relationships in Table 4, noting that the median RBL estimate is 6% for a cumulative seasonal W126 exposure index of 19 ppm-hrs. Considering somewhat lower values, the median RBL estimate is 5.7% (which rounds to 6%) for a cumulative seasonal W126 exposure index of 18 ppm-hrs and the median RBL estimate is 5.3% (which rounds to 5%) for 17 ppm-hrs. In light of her decision that it is appropriate to use a 3-year cumulative exposure index for assessing vegetation effects (described above), the potential for single-season effects of concern, and CASAC comments on the appropriateness of a lower value for a 3-year average W126 index, the Administrator concludes it is appropriate to identify a standard that would restrict cumulative seasonal exposures to 17 ppm-hrs or lower, in terms of a 3-year W126 index, in nearly all instances. In reaching this conclusion, based on the current information to inform consideration of vegetation effects and their potential adversity to public welfare, she additionally judges that the RBL estimates associated with marginally higher exposures in isolated, rare instances are not indicative of effects that would be adverse to the public welfare, particularly in light of variability in the array of environmental factors that can influence O₃ effects in different systems and uncertainties associated with estimates of effects associated with this magnitude of cumulative exposure in the natural environment.

While giving primary consideration to growth effects using the surrogate of RBL estimates based on tree seedling effects, the Administrator also

recognizes the longstanding and robust evidence of O₃ effects on crop yield. She takes note of CASAC concurrence with the PA description of such effects as of public welfare significance and agrees. As recognized in the proposal, the maintenance of adequate agricultural crop yields is extremely important to the public welfare. Accordingly, research on agricultural crop species remains important for further illumination of mechanisms of action and physiological processes. Given that the extensive management of such vegetation, which as CASAC noted may reduce yield variability, may also to some degree mitigate potential O₃-related effects, however, judgments about the extent to which O₃-related effects on crop yields are adverse from a public welfare perspective are particularly difficult to reach. Further, management practices for agricultural crops are highly variable and generally designed to achieve optimal yields, taking into consideration various environmental conditions. As a result of this extensive role of management in optimizing crop yield, the Administrator notes the potential for greater uncertainty with regard to estimating the impacts of O₃ exposure on agricultural crop production than that associated with O₃ impacts on vegetation in natural forests. For all of these reasons, the Administrator is not giving the same weight to CASAC's statement regarding crop yield loss as a surrogate for adverse effects on public welfare, or the magnitude that would represent an adverse impact to public welfare, as to the CASAC's comments on RBL as a surrogate for an array of growth-related effects. Similarly, given the considerations summarized above and in the proposal, the Administrator concludes that agricultural crops do not have the same need for additional protection from the NAAQS as forested ecosystems and finds protection of public welfare from crop yield impacts to be a less important consideration in this review for the reasons identified, including the extensive management of crop yields and the dynamics of agricultural markets. Thus, the Administrator is not giving a primary focus to crop yield loss in selecting a revised secondary standard. She notes, however, that a standard revised to increase protection for forested ecosystems would also be expected to provide some increased protection for agricultural crops.

The Administrator has additionally considered the evidence and analyses of visible foliar injury. In so doing, the Administrator notes the ISA conclusion

that "[e]xperimental evidence has clearly established a consistent association of visible injury with O₃ exposure, with greater exposure often resulting in greater and more prevalent injury" (U.S. EPA, 2013, section 9.4.2, p. 9–41). The Administrator also recognizes the potential for this effect to affect the public welfare in the context of affecting values pertaining to natural forests, particularly those afforded special government protection, as discussed in section IV.A.3 above. However, she recognizes significant challenges in judging the specific extent and severity at which such effects should be considered adverse to public welfare, in light of the variability in the occurrence of visible foliar injury and the lack of clear quantitative relationships with other effects on vegetation, as well as the lack of established criteria or objectives that might inform consideration of potential public welfare impacts related to this vegetation effect.

Further, the Administrator takes note of the range of evidence on visible foliar injury and the various related analyses, including additional observations drawn from the WREA biosite dataset in response to comments, as summarized in section IV.C.2 above. In so doing, she does not agree with CASAC's comment that a level of W126 exposure below 10 ppm-hrs is required to reduce foliar injury, noting some lack of clarity in the WREA and PA presentations of the WREA cumulative proportion analysis findings and their meaning (described in section IV.C.2.b above). She notes that the additional observations summarized in section IV.C.2 above indicate declines in proportions of sites with any visible foliar injury and biosite index scores with reductions in cumulative W126 exposure across a range of values extending at the high end well above 20 ppm-hrs, down past and including 17 ppm-hrs. In considering this information, however, the Administrator takes note of the current lack of robust exposure-response functions that would allow prediction of visible foliar injury severity and incidence under varying air quality and environmental conditions, as recognized in section IV.A.1.b above. Thus, while the Administrator notes that the evidence is not conducive to use for identification of a specific quantitative public welfare protection objective, due to uncertainties and complexities described in sections IV.A.1.b and IV.A.3 above, she concludes that her judgments above, reached with a focus on RBL estimates, would also be expected to provide an additional

desirable degree of protection against visible foliar injury in sensitive vegetation. Accordingly, she considers a conclusion on the appropriateness of selecting a standard that will generally limit cumulative exposures above 17 ppm-hrs to be additionally supported by evidence for visible foliar injury, while not based on specific consideration of this effect.

With the public welfare protection objectives identified above in mind, the Administrator turns to her consideration of form and level for the revised secondary standard. In considering whether the current form should be retained or revised in order to provide the appropriate degree of public welfare protection, the Administrator has considered the analyses of air quality data from the last 13 years that describe the cumulative exposures, in terms of a 3-year W126 index, occurring at monitoring sites across the U.S. when the air quality metric at that location, in terms of the current standard's form and averaging time, is at or below different alternative levels. The Administrator notes both the conclusions drawn from analyses of the strong, positive relationship between these metrics and the findings that indicate the amount of control provided by the fourth-high metric.

The Administrator has also considered advice from CASAC and public commenters that support revision of the form to the W126 exposure index. The Administrator concurs with the underlying premise that O₃ effects on vegetation are most directly assessed using a cumulative seasonal exposure index, specifically the W126 exposure index. The Administrator additionally recognizes, based on analyses of the last 13 years of monitoring data, and consideration of modeling analyses with associated limitations and uncertainties, that cumulative seasonal exposures appear to have a strong relationship with design values based on the current form and averaging time. She additionally notes the correlation of reductions in W126 index values with reductions in precursor emissions over the past decade that were targeted at meeting the current O₃ standards (with fourth-high form), which indicate the control of cumulative seasonal exposures that can be achieved with a standard of the current form and averaging time.

With regard to recommendations from the CASAC that the form for the revised secondary standard should be the biologically relevant exposure metric, and related comments from the public indicating that the secondary standard must have such a form, the

Administrator disagrees. In so doing, she notes that CAA section 109 does not impose such a requirement on the form or averaging time for the NAAQS, as explained in IV.C.2 above. She further notes that the averaging time and form of primary standards are often not the same as the exposure metrics used in reviews of primary standards, in which specific information on quantitative relationships between different exposure metrics and health risk is more often available than it is in reviews of secondary NAAQS. As discussed in section IV.C.2 above, with examples, a primary standard with a particular averaging time and form may provide the requisite public health protection from health effects that are most appropriately assessed using an exposure metric of a different averaging time and form and indicator, and the same principle can apply when establishing or revising secondary standards. The Administrator recognizes that the exposure metric and the standard metric can be quite similar, as in the case of consideration of short-term health effects with the primary O₃ standard. She also notes, however, as illustrated by the examples described in section IV.C.2 above, that it is not uncommon for the EPA to retain or adopt elements of an existing standard that the Administrator judges in combination across all elements, including in some cases a revised level, to provide the requisite protection under the Act, even if those elements do not neatly correspond to the exposure metric. Accordingly, she concludes that the Act does not require that the secondary O₃ standard be revised to match the exposure metric identified as biologically relevant in this review, as long as the revised standard provides the degree of protection required under CAA section 109(b)(2).

Based on the considerations described here, including the use of an exposure metric that CASAC has agreed to be biologically relevant and appropriate, related considerations summarized in the proposal with regard to air quality analyses and common uses of exposure metrics in other NAAQS reviews, the Administrator finds that, in combination with a revised level, the current form and averaging time for a revised secondary standard can be expected to provide the desired level of public welfare protection. Accordingly, she next turns to the important consideration of a level that, in combination with the form and averaging time, will yield a standard that specifies the requisite air quality for protection of public welfare. In so

doing, she has recognized the recommendation by CASAC for revision of the form and averaging time and provided the basis for her alternative view, as described above. Further, in the context of the Administrator's decision on objectives for public welfare protection of a revised secondary standard, and with consideration of the advice from CASAC on levels for a W126-based standard, the Administrator has also reached the conclusion, as described above, that in order to provide the appropriate degree of public welfare protection, the revised secondary standard should restrict cumulative seasonal exposures to 17 ppm-hrs or lower, in terms of a 3-year average W126 index, in nearly all instances. Thus, the Administrator finds it appropriate to revise the standard level to one that, in combination with the form and averaging time, will exert this desired degree of control for cumulative seasonal exposures.

In considering a revised standard level, the Administrator has, in light of public comments, revisited the information she considered in reaching her proposed decision on a level within the range of 65 to 70 ppb, and additional information or insights conveyed with public comments. The primary focus of the Administrator's considerations in reaching her proposed decision was the multi-faceted analysis of air quality data from 2001 through 2013 documented in the technical memo in the docket (Wells, 2014a), as well as the earlier analyses and related information described in the PA (as summarized in section IV.E.4 of the proposal). This analysis describes the occurrences of 3-year W126 index values of a magnitude from 17 ppm-hrs through 7 ppm-hrs at monitor locations where O₃ concentrations met different alternative standards with the current form and averaging time, and has been expanded in consideration of public comments to present in summary form the more extensive historical dataset accompanying this analysis (Wells, 2015b). Focusing first on the air quality analyses for the most recent period for which data are available (2011–2013) and with the protection objectives identified above in mind, the Administrator observes that across the sites meeting the current standard of 75 ppb, the analysis finds 25 sites distributed across different NOAA climatic regions with 3-year average W126 index values above 17 ppm-hrs, with the values at nearly half of the sites extending above 19 ppm-hrs, with some well above. In comparison, she observes that across sites meeting an alternative

standard of 70 ppb, the analysis for the period from 2011–2013 finds no occurrences of W126 metric values above 17 ppm-hrs and less than a handful of occurrences that equal 17 ppm-hrs. The more than 500 monitors that would meet an alternative standard of 70 ppb during the 2011–2013 period are distributed across all nine NOAA climatic regions and 46 of the 50 states (Wells, 2015b and associated dataset in the docket).

The Administrator notes that some public commenters, who disagreed with her proposed decision on form and averaging time, emphasized past occurrences of cumulative W126 exposure values above the range identified in the proposal (of 13 to 17 ppm-hrs). For example, these commenters emphasize data from farther back across the full time period of the dataset analyzed in the technical memorandum (2001–2013), identifying a value of 19.1 ppm-hrs at a monitor for which the fourth-high metric is 70 ppb for the 3-year period of 2006–2008. The Administrator notes, as discussed in section IV.C.2 above, that this was one of fewer than a handful of isolated occurrences of sites for which the fourth-high was at or below 70 ppb and the W126 index value was above 17 ppm-hrs, all but one of which were below 19 ppm-hrs. The Administrator additionally recognizes her underlying objective of a revised secondary standard that would limit cumulative exposures in nearly all instances to those for which the median RBL estimate would be somewhat lower than 6%. She observes that the single occurrence of 19 ppm-hrs identified by the commenter among the nearly 4000 3-year W126 index values from across the most recently available 11 3-year periods of data at monitors for which the fourth-high metric is at or below 70 ppb is reasonably regarded as an extremely rare and isolated occurrence (Wells, 2015b). As such, it is unclear whether it would recur, particularly as areas take further steps to reduce O₃ to meet revised primary and secondary standards. Further, based on the currently available information, the Administrator does not judge RBL estimates associated with marginally higher exposures in isolated, rare instances to be indicative of adverse effects to the public welfare. Thus, the Administrator concludes that a standard with a level of 70 ppb and the current form and averaging time may be expected to limit cumulative exposures, in terms of a 3-year average W126 exposure index, to values at or below 17 ppm-hrs, in nearly all instances, and

accordingly, to eliminate or virtually eliminate cumulative exposures associated with a median RBL of 6% or greater.

The Administrator recognizes that any standard intended to exert a very high degree of control on cumulative seasonal exposures, with the objective of limiting exposures above 17 ppm-hrs across the U.S., in nearly all instances, will, due to regional variation in meteorology and sources of O₃ precursors, result in cumulative seasonal exposures well below 17 ppm-hrs in many areas. Even implementation of a standard set in terms of the cumulative seasonal exposure metric, while limiting the highest exposures, would, due to regional variation in meteorology and sources of O₃ precursors, result in many areas with much lower exposures. Such variation in exposures occurring under a specific standard is not unexpected and the overall distribution of exposures estimated to occur with air quality conditions associated with different alternative standards is a routine part of the consideration of public health protection in reviews of primary standards, and can also play a role in the review of secondary standards. For these reasons, and in light of the discussion in section IV.C.2.d above on consideration of “necessary” protection, the Administrator notes that an expectation of differing exposures is not, in itself, a basis for concluding that the air quality would be more (or less) than necessary (and thus not requisite) for the desired level of public welfare protection.

The Administrator has also considered the protection afforded by a revised standard against other effects studied in this review, such as visible foliar injury and reduced yield for agricultural crops, and also including those associated with climate change. While noting the evidence supporting a relationship of O₃ in ambient air with climate forcing effects, as concluded in the ISA, the Administrator judges the quantitative uncertainties to be too great to support identification of a standard specific to such effects such that she concludes it is more important to focus, as she has done above, on setting a standard based on providing protection against vegetation-related effects which would be expected to also have positive implications for climate change protection through the protection of ecosystem carbon storage.

The Administrator additionally considers the extent of control for cumulative seasonal exposures exerted by a revised standard level of 65 ppb, the lower end of the proposed range. In

focusing on the air quality analyses for the most recent 3-year period for which data are available, the Administrator observes that across the sites meeting a fourth-high metric of 65 ppb, the analysis finds no occurrences of W126 metric values above 11 ppm-hrs and 35 occurrences of a value between 7 ppm-hrs and 11 ppm-hrs, scattered across NOAA climatic regions. The Administrator finds these magnitudes of cumulative seasonal exposures to extend appreciably below the objectives she identified above for affording public welfare protection. In considering this alternative level, she additionally notes that data for only 276 monitors (less than 25 percent of the total with valid fourth-high and W126 metric values) were at or below a fourth-high value of 65 ppb during the period from 2011–2013. In so noting, she recognizes the appreciably smaller and less geographically extensive dataset available and the associated uncertainty for conclusions based on such an analysis.

Thus, based on the support provided by currently available information on air quality, the evidence base of O₃ effects on vegetation and her public welfare policy judgments, and after carefully taking the above comments and considerations into account, fully considering the scientific views of the CASAC, and also taking note of CASAC’s policy views, the Administrator has decided to retain the current indicator, form and averaging time and to revise the secondary standard level to 70 ppb. In the Administrator’s judgment, based on the currently available evidence and quantitative exposure and air quality information, a standard set at this level, in combination with the currently specified form, averaging time and indicator would be requisite to protect the public welfare from known or anticipated adverse effects. A standard set at this level provides an appreciable increase in protection compared to the current standard. The Administrator judges that such a standard would protect natural forests in Class I and other similarly protected areas against an array of adverse vegetation effects, most notably including those related to effects on growth and productivity in sensitive tree species. The Administrator believes that a standard set at 70 ppb would be sufficient to protect public welfare from known or anticipated adverse effects and believes that a lower standard would be more than what is necessary to provide such protection. This judgment by the Administrator appropriately recognizes

that the CAA does not require that standards be set at a zero-risk level, but rather at a level that reduces risk sufficiently so as to protect the public welfare from known or anticipated adverse effects. Accordingly, the Administrator concludes that it is appropriate to revise the level for the secondary standard to 70 ppb (0.070 ppm), in combination with retaining the current form, indicator, and averaging time, in order to specify the level of air quality that provides the requisite protection to the public welfare from any known or anticipated adverse effects associated with the presence of O₃ in the ambient air.

D. Decision on the Secondary Standard

For the reasons discussed above, and taking into account information and assessments presented in the ISA and PA, the advice and recommendations of CASAC, and the public comments, as well as public welfare judgments, the Administrator is revising the level of the current secondary standard. Specifically, the Administrator has decided to revise the level of the secondary standard to a level of 0.070 ppm, in conjunction with retaining the current indicator, averaging time and form. Accordingly the revised secondary standard is 0.070 ppm O₃, as the annual fourth-highest daily maximum 8-hour average concentration, averaged over three years.

V. Appendix U: Interpretation of the Primary and Secondary NAAQS for O₃

A. Background

The EPA is finalizing the proposed Appendix U to 40 CFR part 50: Interpretation of the Primary and Secondary National Ambient Air Quality Standards for Ozone. The proposed Appendix U addressed the selection of ambient O₃ monitoring data to be used in making comparisons with the NAAQS, data reporting and data handling conventions for comparing ambient O₃ monitoring data with the level of the NAAQS, and data completeness requirements. The EPA solicited public comment on four elements where the proposed Appendix U differed from Appendix P to 40 CFR part 50, which addressed data handling conventions for the previous O₃ NAAQS. These included the following: (1) the addition of a procedure to combine data collected from two or more O₃ monitors operating simultaneously at the same physical location, (2) the addition of a provision allowing the Regional Administrator to approve "site combinations", or the combination of data from two nearby

monitoring sites for the purpose of calculating a valid design value, (3) a change from the use of one-half of the method detection limit (½ MDL) to zero (0.000 ppm) as the substitution value in 8-hour average data substitution tests, and 4) a new procedure for calculating daily maximum 8-hour average O₃ concentrations for the revised NAAQS.

The EPA is also finalizing, as proposed, exceptional events scheduling provisions in 40 CFR 50.14 that will apply to the submission of information supporting claimed exceptional events affecting pollutant data that are intended to be used in the initial area designations for any new or revised NAAQS. The new scheduling provisions will apply to initial area designations for the 2015 O₃ NAAQS.

B. Data Selection Requirements

The EPA proposed this section in Appendix U to clarify which data are to be used in comparisons with the revised O₃ NAAQS. The EPA is finalizing this section in Appendix U as proposed.

First, the EPA proposed to combine data at monitoring sites with two or more O₃ monitoring instruments operating simultaneously into a single site-level data record for determining compliance with the NAAQS, and proposed an analytical approach to perform this combination (79 FR 75351–75352, December 17, 2014). Several commenters supported the EPA's proposed approach, including the State of Iowa, where 15 of the 20 monitoring sites currently operating two O₃ monitors simultaneously are located. Commenters supporting the proposal noted that a similar approach is already being used for lead and particulate monitoring, and that the proposed approach will help states meet data completeness requirements.

A few commenters supported the EPA's proposed approach with the additional restrictions that the monitoring instruments must use identical methods and be operated by the same monitoring agency. The EPA notes that at the time of this rulemaking, all monitors reporting O₃ concentration data to the EPA for regulatory use were FEMs. All current O₃ FEMs use an ultraviolet photometry sampling methodology and have been found to meet the performance criteria in 40 CFR part 53. Therefore, the EPA has no reason to believe that O₃ concentration data should not be combined across monitoring methods at the site level. Regarding the commenters' suggestion that data should not be combined when two or more monitors at the same site are operated by different monitoring agencies, the EPA is aware of only one

instance where this presently occurs. In this instance, the monitors have been assigned distinct site ID numbers in the AQS database, so that data will not be combined across these monitors. Should future instances arise where two or more monitoring agencies decide to operate O₃ monitors at the same site, the EPA encourages these agencies to work together to establish a plan for how the data collected from these monitors should be used in regulatory decision making.

One state objected to combining data across monitors because the secondary monitors at their sites were used only for quality assurance purposes and data from these monitors should not be combined with data reported from the primary monitors. The EPA notes that concentration data collected to meet quality assurance requirements (*i.e.* precision and bias data) are reported and stored in a separate location within the AQS database and are not used for determining compliance with the NAAQS. The required quality assurance data are derived from O₃ standards and not from a separate O₃ monitor. However, if a separate O₃ monitor is used strictly for quality assurance purposes and does not meet the applicable monitoring requirements, it can be distinguished in AQS in such a manner that data from the secondary monitor would not be combined with data from the primary monitor.

Another commenter objected to the proposal because it would reduce the total number of comparisons made with the NAAQS. While this is true, the number of physical locations being compared with the NAAQS will not decrease under the proposed approach, and in fact may increase due to additional sites meeting the data completeness requirements.

Finally, two commenters submitted similar comments citing the EPA's evaluation of collocated O₃ monitoring data and precision data in the ISA (U.S. EPA, 2013, section 3.5.2), and stated that although the median differences in concentrations reported by the pairs of monitoring instruments were near zero, the extreme values were close to +/– 3.5%. The commenter argued that since the O₃ NAAQS are based on the fourth-highest annual value, data should not be combined across monitors because of the imprecision in the extreme values. The EPA disagrees, noting that the data presented in the ISA are based on hourly concentrations, while design values for the O₃ NAAQS are based on a 3-year average of 8-hour average concentrations. Thus, the random variability in the hourly O₃ concentration data due to monitoring

imprecision will be reduced when concentrations are averaged for comparison with the NAAQS. Additionally, the precision data are typically collected at concentrations at or above the level of the NAAQS, thus the EPA expects that the level of precision documented in the ISA analysis is consistent with the level of precision in the fourth-highest daily maximum concentrations used for determining compliance with the NAAQS.

The EPA is finalizing this addition in Appendix U as proposed. In addition, the AQS database will be updated to require state agencies to designate a primary monitor at O₃ monitoring sites that report data under more than one Pollutant Occurrence Code (POC), a numeric indicator in AQS used to identify individual monitoring instruments. O₃ design value calculations in AQS will be updated so that the data will automatically be combined across POCs at a site, and a single design value will be reported for each site. The EPA notes that the substitution approach described above will only be applied to design value calculations for the revised O₃ standards, and that design values for previous O₃ standards will continue to be calculated at the monitor level, in accordance with the applicable appendices of 40 CFR part 50.

Second, the EPA proposed to add a provision in Appendix U that would allow the Regional Administrator to approve “site combinations”, or to combine data across two nearby monitors for the purpose of calculating a valid design value. Although data handling appendices for previous O₃ standards do not explicitly mention site combinations, the EPA has approved over 100 site combinations since the promulgation of the first 8-hour O₃ NAAQS in 1997. Thus, the EPA’s intention in proposing this addition was merely to codify an existing convention, and to improve transparency by implementing site combinations in AQS design value calculations.

Public commenters unanimously supported this proposed addition. Two commenters suggested that the EPA should require monitoring agencies to provide technical documentation supporting the similarities between sites approved for combining data, including a requirement for simultaneous monitoring whenever possible. One state requested that the EPA provide more detailed acceptability criteria for approving site combinations, while another state urged the EPA not to create a regulatory burden by

prescribing detailed requirements codified in regulations.

The EPA is finalizing this addition as proposed in Appendix U. The EPA believes that approval of site combinations should be handled on a case-by-case basis, and that any requests for supporting documentation should be left to the discretion of the Regional Administrator. The EPA may issue future guidance providing general criteria for determining an acceptable level of similarity in air quality concentrations between monitored locations, but is not prescribing detailed criteria for approval of site combinations in this rulemaking.

Additionally, the AQS database will be updated with new fields for monitoring agencies to request site combinations, and an additional field indicating Regional Administrator approval. All pre-existing site combinations will be initially entered into the database as having already been approved by the Regional Administrator. Since this provision has already been used in practice under previous O₃ standards, site combinations will be applied to AQS design value calculations for both the revised O₃ standards and previous O₃ standards.

C. Data Reporting and Data Handling Requirements

First, the EPA proposed a change in Appendix U to the pre-existing 8-hour average data substitution test (40 CFR part 50, Appendix P, section 2.1) which is used to determine if a site would have had a valid 8-hour average greater than the NAAQS when fewer than 6 hourly O₃ concentration values are available for a given 8-hour period. The EPA proposed to change the value substituted for the missing hourly concentrations from one-half of the method detection limit of the O₃ monitoring instrument (½ MDL) to zero (0.000 ppm).

Several commenters supported the proposed change, stating that the use of a constant substitution value instead of ½ MDL, which can vary across O₃ monitoring methods, would simplify design value calculations. One commenter noted that with a substitution value of zero, the data substitution test for an 8-hour average value greater than the NAAQS is equivalent to a sum of hourly O₃ concentrations greater than 0.567 ppm (*i.e.*, if the sum is 0.568 ppm or higher, the resulting 8-hour average must be at least 0.071 ppm, which is greater than the revised O₃ NAAQS of 0.070 ppm). Finally, one commenter opposed the proposed change in favor of some type

of mathematical or statistical interpolation approach, but did not provide a specific recommendation.

The EPA is finalizing the proposed change in Appendix U, with the addition of a short clause making note of the equivalent summation approach described above. The purpose of the data substitution test is to identify 8-hour periods that do not meet the requirements for a valid 8-hour average, yet the reported hourly concentration values are so high that the NAAQS would have been exceeded regardless of the magnitude of the missing concentration values. The EPA believes that zero, being the lowest measured O₃ concentration physically possible, is the most appropriate value to substitute in this situation. Additionally, the EPA does not support the use of interpolation or other means of filling in missing monitoring data for O₃ NAAQS comparisons. Such an approach would be contrary to the EPA’s long-standing policy of using only quality-assured and certified ambient air quality measurement data to determine compliance with the O₃ NAAQS.

Second, the EPA proposed a new procedure in Appendix U for determining daily maximum 8-hour O₃ concentrations for the revised NAAQS.²¹³ The EPA proposed to determine the daily maximum 8-hour O₃ concentration based on 17 consecutive moving 8-hour periods in each day, beginning with the 8-hour period from 7:00 a.m. to 3:00 p.m., and ending with the 8-hour period from 11:00 p.m. to 7:00 a.m. In addition, the EPA proposed that a daily maximum value would be considered valid if 8-hour averages were available for at least 13 of the 17 consecutive moving 8-hour periods, or if the daily maximum value was greater than the level of the NAAQS. This procedure is designed to eliminate “double counting” exceedances of the NAAQS based on overlapping 8-hour periods from two consecutive days with up to 7 hours in common, which was allowed under previous 8-hour O₃ NAAQS. A dozen public commenters expressed support for the proposed procedure, including several states.

One regional air quality management organization and three of its member states submitted similar comments stating that they agreed with the principle of eliminating “double counting” exceedances of the NAAQS

²¹³ This procedure will be adopted only for the revised O₃ NAAQS. Design values for the 1997 8-hour O₃ NAAQS and the 2008 8-hour O₃ NAAQS will continue to be calculated according to Appendix I and Appendix P of 40 CFR part 50, respectively.

based on overlapping 8-hour periods, but suggested an alternative calculation procedure that would accomplish the same objective. The alternative procedure iteratively finds the highest 8-hour period in a given year, then removes this 8-hour period and all other 8-hour periods associated with that day, including any overlapping 8-hour periods on adjacent days, from the data until a daily maximum value is determined for each day of the year with sufficient monitoring data. The EPA examined a similar iterative procedure in a previous data analysis supporting the proposal (Wells, 2014b, Method 1). The EPA compared this procedure to the procedure proposed by the commenters using the data from the original analysis and found the resulting daily maximum 8-hour values to be nearly identical (Wells, 2015a). Additionally, the commenters' procedure suffers from the same limitations the EPA identified previously in the original analysis: added complexity in design value calculations, longer computational time, and challenges to real-time O₃ data reporting systems, which would have to re-calculate daily maximum 8-hour values for the entire year each time the system was updated with new data.

Three states submitted comments stating that they agreed with the proposed calculation procedure, but disagreed with the proposed requirements for determining a valid daily maximum 8-hour O₃ concentration. These states were primarily concerned that the proposed requirements would only allow a monitoring site to have four missing 8-hour averages during a day before the entire day would be invalidated, compared with six missing 8-hour averages allowed previously. Two of these states also stated concerns that the proposed requirements would be more difficult to meet while maintaining compliance with existing monitoring requirements such as biweekly quality assurance checks. The EPA compared annual data completeness rates calculated using the Appendix U requirements to annual data completeness rates calculated using the requirements under the previous O₃ standards across all U.S. monitoring sites based on data from 2004–2013 (Wells, 2015a). The national mean annual data completeness rate was 0.1% higher under the proposed Appendix U requirements than under the previous O₃ standards, and the national median annual data completeness rates were identical. In addition, the EPA notes that the Appendix U requirements allow

for biweekly quality assurance checks and other routine maintenance to be performed between 5:00 a.m. and 9:00 a.m. local time without affecting data completeness. Thus, the EPA does not believe that the proposed daily data completeness requirements in Appendix U will be more difficult for monitoring agencies to meet.

Finally, two public commenters opposed the proposed procedures for determining daily maximum 8-hour concentrations. These commenters expressed similar concerns, primarily that not considering 8-hour periods starting midnight to 6:00 a.m. is less protective of public health than the procedure used to determine daily maximum 8-hour concentrations for the previous O₃ standards. The EPA believes that this approach provides the appropriate degree of protection for public health, noting that the hourly concentrations from midnight to 7:00 a.m. are covered under the 8-hour period from 11:00 p.m. to 7:00 a.m., which is included in the design value calculations proposed in Appendix U. At the same time, the proposed approach ensures that individual hourly concentrations may not contribute to multiple exceedances of the NAAQS, which the EPA believes is inappropriate given that people are only exposed once.

The EPA is finalizing as proposed in Appendix U the procedure for determining daily maximum 8-hour concentrations. The EPA does not believe that daily maximum 8-hour concentrations for two consecutive days should be based on overlapping 8-hour periods, since the exposures experienced by individuals only occur once. The EPA believes that the new procedure will avoid this outcome while continuing to make use of all hourly concentrations in determining attainment of the standards, without introducing unnecessary complexity into design value calculations, and without creating additional difficulties for monitoring agencies to meet the data completeness requirements.

D. Exceptional Events Information Submission Schedule

The “Treatment of Data Influenced by Exceptional Events; Final Rule” (72 FR 13560, March 22, 2007), known as the Exceptional Events Rule and codified at 40 CFR 50.14, contains generic deadlines for an air agency to submit to the EPA specified information about exceptional events and associated air pollutant concentration data. As discussed in this section and in more detail in the O₃ NAAQS proposal, without revisions to 40 CFR 50.14, an

air agency may not be able to flag and submit documentation for some relevant data either because the generic deadlines may have already passed by the time a new or revised NAAQS is promulgated or because the generic deadlines require submission of documentation at least 12 months prior to the date by which the EPA must make a regulatory decision, which may be before air agencies have collected some of the potentially affected data. Specific to the revised O₃ NAAQS, revisions to 40 CFR 50.14 are needed because it is not possible for air agencies to flag and submit documentation for any exceptional events that occur in October through December of 2016 by 1 year before the designations are made in October 2017, as is required by the existing generic schedule.

The EPA is finalizing exceptional events scheduling provisions in 40 CFR 50.14, as proposed and as supported by multiple commenters, that will apply to the submission of information supporting claimed exceptional events affecting pollutant data that are intended to be used in the initial area designations for any new or revised NAAQS. The new scheduling provisions will apply to initial area designations for the revised O₃ NAAQS. The provisions that we are promulgating use a “delta schedule” that calculates the timelines associated with flagging data potentially influenced by exceptional events, submitting initial event descriptions and submitting exceptional events demonstrations based on the promulgation date of a new or revised NAAQS. The general data flagging deadlines in the Exceptional Events Rule at 40 CFR 50.14(c)(2)(iii) and the general schedule for submission of demonstrations at 40 CFR 50.14(c)(3)(i) continue to apply to data used in regulatory decisions other than those related to the initial area designations process under a new or revised NAAQS.²¹⁴

The EPA acknowledges the concern raised by several commenters that a strengthened O₃ NAAQS may result in numerous demonstrations for exceptional events occurring between 2014 and 2016, the data years that the EPA will presumably use for initial area designation decisions made in October 2017.²¹⁵ Commenters noted that the proposed schedule is particularly burdensome for agencies needing to submit exceptional events packages for

²¹⁴ The EPA intends to consider changes to these retained scheduling requirements as part of the planned notice and comment rulemaking revisions to the 2007 Exceptional Events Rule.

²¹⁵ Governors may also use 2013 data to formulate their recommendations regarding designations.

the third year to be used in a 3-year design value (*i.e.*, 2016 data). Several commenters recommended that the EPA either establish no defined schedule for data flagging and exceptional events demonstration submittal or allow a minimum of 2 years from the setting of any new or revised NAAQS for air agencies to provide a complete exceptional events demonstration. Given the CAA requirement that the EPA follow a 2-year designations schedule, the EPA cannot remove submittal schedules entirely for data influenced by exceptional events or provide a minimum 2-year period from the setting of a new or revised NAAQS for documentation submittal. Neither of these options would ensure that the EPA has time to consider event-influenced data in initial area designation decisions. Rather, the EPA is promulgating in this action an exceptional events schedule that provides air agencies with the maximum amount of time available to prepare exceptional events demonstrations and will still allow the EPA sufficient time to consider such exceptional events demonstrations in the designations process in advance of the date by which the EPA must send 120-day notification letters to states.²¹⁶ The EPA recognizes that the schedule promulgated in this action is compressed, particularly for the third year of data to be used in a 3-year design value, and we will work cooperatively with air agencies to accommodate this scenario.

Under the schedule promulgated in this action and assuming initial area designation decisions in October 2017 for the revised O₃ NAAQS, affected air agencies would need to flag data, submit initial event descriptions and submit demonstrations for exceptional events occurring in 2016 by May 31, 2017. This schedule provides approximately 5 months between the EPA's receipt of the demonstration package and the expected date of designation decisions and approximately 1 month between the EPA's receipt of a package and the date by which the EPA must notify states and tribes of intended modifications to the Governors' recommendations for designations (*i.e.*, 120-day letters).

While, for the third year of data anticipated to be used in a 3-year design value for the revised O₃ NAAQS, the promulgated schedule provides for demonstration submission 5 months after the end of the calendar year, the EPA expects that most submitting

agencies will have additional time to prepare documentation as we expect the majority of potential O₃-related exceptional events to occur during the warmer months (*e.g.*, March through October). Additionally, the EPA will soon propose rule revisions to the 2007 Exceptional Events Rule and will release through a **Federal Register** Notice of Availability a draft guidance document to address Exceptional Events Rule criteria for wildfires that could affect O₃ concentrations. We expect to promulgate Exceptional Events Rule revisions and finalize the new guidance document before the October 2016 date by which states, and any tribes that wish to do so, are required to submit their initial designation recommendations for the revised O₃ NAAQS. Considered together, the EPA believes the exceptional events scheduling dates promulgated in this action, the upcoming Exceptional Events Rule revisions, the forthcoming guidance, and the existing guidance and examples of submitted demonstrations currently on the EPA's exceptional events Web site at <http://www2.epa.gov/air-quality-analysis/treatment-data-influenced-exceptional-events>, will help air agencies submit information in a timely manner.

Applying the "delta schedule" promulgated in this action for air quality data collected in 2013 through 2014 that could be influenced by exceptional events and be considered during the initial area designations process for the revised O₃ NAAQS, results in extending to July 1, 2016, the otherwise applicable generic deadlines of July 1, 2014, and July 1, 2015, respectively, for flagging data and providing an initial description of an event (40 CFR 50.14(c)(2)(iii)). The schedule promulgated in this action also results in a July 1, 2016, date for flagging data and providing an initial description of an event for air quality data collected in 2015. The July 1, 2016, date for data collected in 2015 is the same as that which would apply under the existing generic deadline in the 2007 Exceptional Events Rule. Under the schedule promulgated in this action, October 1, 2016 is the deadline for submitting exceptional events demonstrations for data years 2013 through 2015. As noted previously, under the schedule promulgated in this action, affected air agencies would need to flag, submit initial event descriptions and submit demonstrations for exceptional events occurring in 2016 by May 31, 2017. The EPA believes these revisions will provide adequate time for air agencies to review potential O₃

exceptional events influencing compliance with the revised O₃ NAAQS, to notify the EPA by flagging the relevant data and providing an initial event description in AQS, and to submit documentation to support exceptional events demonstrations. The schedule revisions promulgated in this action will also allow the EPA to consider and act on the submitted information during the initial area designation process.

While the EPA will make every effort to designate areas for any new or revised NAAQS on a 2-year schedule, the EPA recognizes that under some circumstances we may need up to an additional year for the designations process to ensure that air agencies and the EPA base designations decisions on complete and sufficient information. The promulgated schedule accounts for the possibility that the EPA might announce after promulgating a new or revised NAAQS that we are extending the designations schedule beyond 2 years using authority provided in CAA section 107(d)(B)(i). If the EPA determines that we will follow a 3-year designation schedule, the deadline is 2 years and 7 months after promulgation of a new or revised NAAQS for states to flag data influenced by exceptional events, submit initial event descriptions and submit exceptional events demonstrations for the last year of data that will be used in the designations (*e.g.*, if the EPA were to designate areas in October 2018, the exceptional events submittal deadline for 2017 data would be May 31, 2018). If the EPA notifies states and tribes of a designations schedule between 2 and 3 years, the deadline for states to flag data affected by exceptional events, submit initial event descriptions, and submit exceptional events demonstrations associated with data from the last year to be considered would be 5 months prior to the date specified for designation decisions.

Therefore, using the authority provided in CAA section 319(b)(2) and in the 2007 Exceptional Events Rule at 40 CFR 50.14(c)(2)(vi), the EPA is modifying the schedule for flagging data and submitting exceptional events demonstrations considered for initial area designations by replacing the deadlines and information in Table 1 in 40 CFR 50.14 with the deadlines and information presented in Table 5. As we did in the O₃ NAAQS proposal, we are also providing Table 6 to illustrate how the promulgated schedule might apply to the designations process for the revised O₃ NAAQS and to designations

²¹⁶ See Section VIII.B for additional detail on the initial area designations process for the revised O₃ NAAQS.

processes for other future new or revised NAAQS.²¹⁷ schedules for initial area designations for new or revised NAAQS, the EPA, as proposed, is removing obsolete regulatory language in 40 CFR 50.14(c)(2)(iv) and (v) and 40 CFR 50.14(c)(3)(ii) and (iii) associated with exceptional events schedules for all historical standards.

Additionally, in conjunction with promulgating exceptional events

TABLE 5—SCHEDULE FOR FLAGGING AND DOCUMENTATION SUBMISSION FOR DATA INFLUENCED BY EXCEPTIONAL EVENTS FOR USE IN INITIAL AREA DESIGNATIONS

Exceptional events/Regulatory action	Exceptional events deadline schedule ^d
Flagging and initial event description deadline for data years 1, 2 and 3 ^a .	If state and tribal initial designation recommendations for a new/revised NAAQS are due August through January, then the flagging and initial event description deadline will be the July 1 prior to the recommendation deadline. If state and tribal recommendations for a new/revised NAAQS are due February through July, then the flagging and initial event description deadline will be the January 1 prior to the recommendation deadline.
Exceptional events demonstration submittal deadline for data years 1, 2 and 3 ^a .	No later than the date that state and tribal recommendations are due to the EPA.
Flagging, initial event description and exceptional events demonstration submittal deadline for data year 4 ^b and, where applicable, data year 5 ^c .	By the last day of the month that is 1 year and 7 months after promulgation of a new/revised NAAQS, unless either option a or b applies. a. If the EPA follows a 3-year designation schedule, the deadline is 2 years and 7 months after promulgation of a new/revised NAAQS. b. If the EPA notifies the state/tribe that it intends to complete the initial area designations process according to a schedule between 2 and 3 years, the deadline is 5 months prior to the date specified for final designations decisions in such EPA notification.

^a Where data years 1, 2, and 3 are those years expected to be considered in state and tribal recommendations.
^b Where data year 4 is the additional year of data that the EPA may consider when it makes final area designations for a new/revised NAAQS under the standard designations schedule.
^c Where data year 5 is the additional year of data that the EPA may consider when it makes final area designations for a new/revised NAAQS under an extended designations schedule.
^d The date by which air agencies must certify their ambient air quality monitoring data in AQS is annually on May 1 of the year following the year of data collection as specified in 40 CFR 58.15(a)(2). In some cases, however, air agencies may choose to certify a prior year's data in advance of May 1 of the following year, particularly if the EPA has indicated its intent to promulgate final designations in the first 8 months of the calendar year. Data flagging, initial event description and exceptional events demonstration deadlines for "early certified" data will follow the deadlines for "year 4" and "year 5" data.

²¹⁷ The range of dates identified in Table 6 is illustrative of the dates for the revised O₃ NAAQS. Users could increment these dates by any constant number (for example by 6 years for a hypothetical NAAQS promulgated in 2021) to develop a table with dates relevant to NAAQS promulgated in the future.

Table 6. Examples by Month of Applying the Promulgated Revised Schedule for Flagging and Documentation Submission for Data Influenced by Exceptional Events for Use in Initial Area Designations

Exceptional Events / Regulatory Action	Month of NAAQS Promulgation, State and Tribal Recommendation, and Final Designations												
	Oct 2015	Nov 2015	Dec 2015	Jan 2016	Feb 2016	Mar 2016	Apr 2016	May ^d 2016	Jun ^d 2016	Jul ^d 2016	Aug ^d 2016	Sep 2016	Oct 2016
Exceptional Events Deadline Schedule ^e	July 1, 2016 (data years 2013, 2014, 2015)	July 1, 2016 (data years 2013, 2014, 2015)	July 1, 2016 (data years 2013, 2014, 2015)	July 1, 2016 (data years 2013, 2014, 2015)	Jan 1, 2017 (data years 2013, 2014, 2015)	Jan 1, 2017 (data years 2013, 2014, 2015)	Jan 1, 2017 (data years 2013, 2014, 2015)	Jan 1, 2017 (data years 2013, 2014, 2015)	Jan 1, 2017 (data years 2013, 2014, 2015)	Jan 1, 2017 (data years 2013, 2014, 2015)	Jan 1, 2017 (data years 2013, 2014, 2015)	Jan 1, 2017 (data years 2013, 2014, 2015)	Jan 1, 2017 (data years 2013, 2014, 2015)
Flagging and initial event description for data years 1, 2, and 3. ^a	July 1, 2016 (data years 2013, 2014, 2015)	July 1, 2016 (data years 2013, 2014, 2015)	July 1, 2016 (data years 2013, 2014, 2015)	July 1, 2016 (data years 2013, 2014, 2015)	Jan 1, 2017 (data years 2013, 2014, 2015)	Jan 1, 2017 (data years 2013, 2014, 2015)	Jan 1, 2017 (data years 2013, 2014, 2015)	Jan 1, 2017 (data years 2013, 2014, 2015)	Jan 1, 2017 (data years 2013, 2014, 2015)	Jan 1, 2017 (data years 2013, 2014, 2015)	Jan 1, 2017 (data years 2013, 2014, 2015)	Jan 1, 2017 (data years 2013, 2014, 2015)	Jan 1, 2017 (data years 2013, 2014, 2015)
Exceptional events demonstration submittal deadline for data years 1, 2, and 3. ^a	by Oct 2016 (data years 2013, 2014, 2015)	by Nov 2016 (data years 2013, 2014, 2015)	by Dec 2016 (data years 2013, 2014, 2015)	by Jan 2017 (data years 2013, 2014, 2015)	by Feb 2017 (data years 2013, 2014, 2015)	by Mar 2017 (data years 2013, 2014, 2015)	by Apr 2017 (data years 2013, 2014, 2015)	by May 2017 (data years 2013, 2014, 2015)	by June 2017 (data years 2013, 2014, 2015)	by July 2017 (data years 2013, 2014, 2015)	by Aug 2017 (data years 2013, 2014, 2015)	by Sep 2017 (data years 2013, 2014, 2015)	by Oct 2017 (data years 2013, 2014, 2015)
AQS quality assurance and data certification	May 1	May 1	May 1	May 1	May 1	May 1	May 1	May 1	May 1	May 1	May 1	May 1	May 1
Flagging, initial event description and exceptional events demonstration submittal deadline for data year 4 ^b and, where applicable, data year 5. ^c	by May 31, 2017 (data year 2016)	by June 30, 2017 (data year 2016)	by July 31, 2017 (data year 2016)	by Aug 31, 2017 (data year 2016)	by Sep 30, 2017 (data year 2016)	by Oct 31, 2017 (data year 2016)	by Nov 30, 2017 (data year 2016)	by Dec 31, 2017 (data year 2016)	by Jan 31, 2018 (data year 2017)	by Feb 28/29, 2018 (data year 2017)	by Mar 31, 2018 (data year 2017)	by Apr 30, 2018 (data year 2017)	by May 31, 2018 (data year 2017)
State & Tribal Recommendations to EPA	Oct 2016	Nov 2016	Dec 2016	Jan 2017	Feb 2017	Mar 2017	Apr 2017	May 2017	June 2017	July 2017	Aug 2017	Sep 2017	Oct 2017
EPA notifies States/Tribes of intended modifications to recommendations (EPA sends 120-day letters)	June 2017	July 2017	Aug 2017	Sept 2017	Oct 2017	Nov 2017	Dec 2017	Jan 2018	Feb 2018	Mar 2018	Apr 2018	May 2018	June 2018
Administrator Promulgates Final Designations	Oct 2017	Nov 2017	Dec 2017	Jan 2018	Feb 2018	Mar 2018	Apr 2018	May 2018	June 2018	July 2018	Aug 2018	Sep 2018	Oct 2018

^a Where data years 1, 2, and 3 are those years expected to be considered in state and tribal recommendations.
^b Where data year 4 is the additional year of data that the EPA may consider when it makes final area designations for a new/revised NAAQS under the standard designations schedule.
^c Where data year 5 is the additional year of data that the EPA may consider when it makes final area designations for a new/revised NAAQS under an extended designations schedule.
^d The date by which air agencies must certify their ambient air quality monitoring data in AQS is annually on May 1 of the year following the year of data collection as specified in 40 CFR 58.15(a)(2). In some cases, however, air agencies may choose to certify a prior year's data in advance of May 1 of the following year, particularly if the EPA has indicated its intent to promulgate final designations in the first 8 months of the calendar year. Data flagging, initial event description and exceptional events demonstration deadlines for "year 4" and "year 5" data.

VI. Ambient Monitoring Related to O₃ Standards

A. Background

The EPA proposed to revise the state-by-state O₃ monitoring seasons; the PAMS monitoring requirements; the FRM for measuring O₃; and the FEM performance requirement specifications for automated O₃ analyzers. The EPA also proposed to make additional minor changes to the FEM analyzer performance testing requirements for NO₂ and particulate matter in part 53.

The EPA is finalizing changes to the length of the required O₃ monitoring season for 32 states and the District of Columbia. Section VI.B of this preamble provides an overview of the proposed changes to the length of the required O₃ monitoring seasons, a summary of significant public comments and our responses, and a summary of the final decisions made to the O₃ monitoring seasons for each state.

The EPA is finalizing changes to the PAMS monitoring requirements in 40 CFR part 58, Appendix D Section 5. Section VI.C of this preamble provides background on the PAMS program and current monitoring requirements, a summary of the proposed changes to the PAMS requirements, a summary of significant public comments and our responses, and a summary of the changes to the PAMS requirements in this final rule.

The EPA is finalizing changes to the FRM for O₃ in Section VI.D of this preamble and to the associated FEM performance requirement specifications for automated O₃ analyzers in Section VI.E. A summary of significant public comments and our responses are provided and a summary of the final changes to the FRM and FEM requirements in this final rule. The EPA is also finalizing minor additional changes to Part 53 including conforming changes to the FEM performance testing requirements in Table B-1 and Figure B-5 for NO₂; extending the period of time for the Administrator to take action on a request for modification of a FRM or FEM from 30 days to 90 days in part 53.14; and removing an obsolete provision for manufacturers to submit Product Manufacturing Checklists for fine and coarse particulate matter monitors in part 53.9.

B. Revisions to the Length of the Required O₃ Monitoring Seasons

Unlike the ambient monitoring requirements in 40 CFR part 58 for other criteria pollutants that mandate year-round monitoring at State and Local Air Monitoring Stations (SLAMS), O₃ monitoring is only required during the

seasons of the year that are conducive to O₃ formation. These seasons vary in length from place-to-place as the conditions conducive to the formation of O₃ (*i.e.*, seasonally-dependent factors such as ambient temperature, strength of solar insolation, and length of day) differ by location. In some locations, conditions conducive to O₃ formation are limited to the summer months of the year. In other states with warmer climates (*e.g.*, California, Nevada, and Arizona), the currently required O₃ season is year-round. Elevated levels of winter-time O₃ have also been measured in some western states where precursor emissions can interact with sunlight off the snow cover under very shallow, stable boundary layer conditions (U.S. EPA 2013).

The EPA has determined that the proposed lengthening of the O₃ monitoring seasons in 32 states and the District of Columbia is appropriate. Ambient O₃ concentrations in these areas could approach or exceed the level of the NAAQS, more frequently and during more months of the year compared with the current season lengths. It is important to monitor for O₃ during the periods when ambient concentrations could approach the level of the NAAQS to ensure that the public is informed when exposure to O₃ could reach or has reached a level of concern.

The EPA completed an analysis to address whether extensions of currently required monitoring seasons are appropriate (Rice, 2014). In this analysis, we used all available data in AQS, including data from monitors that collected O₃ data year-round during 2010–2013. More than half of O₃ monitors are voluntarily operated on a year-round basis by monitoring agencies. We determined the number of days where one or more monitors had a daily maximum 8-hour O₃ average equal to or above 0.060 ppm in the months outside each state’s current O₃ monitoring season and the pattern of those days in the out-of-season months. We believe that a threshold of 0.060 ppm, taking into consideration reasonable uncertainty, serves as an appropriate indicator of ambient conditions that may be conducive to the formation of O₃ concentrations that approach or exceed the NAAQS. We also considered regional consistency, particularly for those states with little available data. We note that seasonal O₃ patterns vary year-to-year due primarily to highly variable meteorological conditions conducive to the formation of elevated O₃ concentrations early or late in the season in some years and not others. The EPA believes it is important that O₃ monitors operate during all

periods when there is a reasonable possibility of ambient levels approaching the level of the NAAQS.

Basing O₃ monitoring season requirements on the goal of ensuring monitoring when ambient O₃ levels approach or exceed the level of the NAAQS supports established monitoring network objectives described in Appendix D of Part 58, including the requirement to provide air pollution data to the general public in a timely manner²¹⁸ and to support comparisons of an area’s air pollution levels to the NAAQS. The operation of O₃ monitors during periods of time when ambient levels approach or exceed the level of the NAAQS ensures that unusually sensitive people and sensitive groups are alerted to O₃ levels of potential health concern allowing them to take precautionary measures. The majority of O₃ monitors in the U.S. report to AIRNOW,²¹⁹ as well as to state-operated Web sites and automated phone reporting systems. These programs support many objectives including real-time air quality reporting to the public, O₃ forecasting, and the verification of real-time air quality forecast models.

1. Proposed Changes to the Length of the Required O₃ Monitoring Seasons

The EPA proposed to extend the length of the required O₃ monitoring season in 32 states and the District of Columbia. The proposed changes were an increase of one month for 22 states (Connecticut, Delaware, Idaho, Illinois, Iowa, Kansas, Maryland, Massachusetts, Minnesota, Missouri, Nebraska, New Hampshire, New Jersey, New York, North Carolina, Ohio, Pennsylvania, Rhode Island, South Carolina, Texas (northern portion only), Virginia, and West Virginia) and the District of Columbia, an increase of one and one half months for Wisconsin, an increase of two months for four states (Indiana, Michigan, Montana, and North Dakota), an increase of four months for Florida and South Dakota, an increase of five months for Colorado, and an increase of seven months for Utah. For Wyoming, we proposed to add three months at the beginning of the season and remove one month at the end of the season, resulting in a net increase of two months. Ozone season requirements are currently split by Air Quality Control Region (AQCR) in Louisiana and Texas. We proposed lengthening the required season in the northern part of Texas (AQCR 022, 210,

²¹⁸ Public reporting requirements are detailed in 40 CFR part 58 Appendix G, Uniform Air Quality Index (AQI) and Daily Reporting.

²¹⁹ See <http://airnow.gov/>.

211, 212, 215, 217, and 218) by one month and leaving the year-round O₃ season in the southern part of Texas (AQCRs 106, 153, 213, 214, and 216) unchanged. No changes were proposed for the AQCRs in Louisiana. As noted earlier, in a few states with limited available data and few exceedance days outside the currently-required season (Iowa, Missouri, and West Virginia), the proposed changes were made by considering supporting information from the surrounding states. These changes involved the proposed addition of one month (March) to the currently-required O₃ seasons for these states.

The EPA also proposed that O₃ monitors at all National Core Multipollutant Monitoring Stations (NCore) be operated year-round, January through December, regardless of the length of the required O₃ season for the remainder of the SLAMS within each state.

We noted that the EPA Regional Administrators have previously approved deviations from the required O₃ monitoring seasons as allowed by paragraph 4.1(i) of 40 CFR part 58, Appendix D. We proposed to retain the rule language permitting such deviations from the required O₃ monitoring seasons, but note that finalized changes to O₃ monitoring season requirements would revoke all existing Regional Administrator-granted waiver approvals. As appropriate, monitoring agencies could seek new approvals for seasonal deviations. Any seasonal deviations based on the Regional Administrator's waiver of requirements must be described in the state's annual monitoring network plan and updated in the AQS.

Given the timing of the final rulemaking and any associated burden on state/local monitoring agencies to implement the extended O₃ seasons, we proposed that implementation of the revised O₃ seasons would become effective at SLAMS (including NCore sites) on January 1, 2017. We solicited comment on whether the revised seasons could be implemented beginning January 1, 2016, for all monitors or for a subset of monitors, such as those currently operating year-round or on a schedule that corresponds to the proposed O₃ season.

2. Comments on the Length of the Required O₃ Monitoring Seasons

We received several comments on the proposed revisions to O₃ monitoring seasons. Several commenters supported the proposed O₃ season length changes and agreed that O₃ monitoring seasons should reflect the times of year when O₃ may approach or exceed the level of the

NAAQS. A few commenters noted the complexities that would arise in the implementation of multi-state planning agreements if states that shared an MSA had different required O₃ monitoring seasons. Two state agencies that supported season length changes also recommended changes to neighboring states' O₃ seasons. New York recommended that Connecticut's proposed O₃ season be further extended (adding the month of October) to match the proposed season in New York (March–October) because they share a major MSA and nonattainment area, and the highest design value monitor in the nonattainment area is often in Connecticut. The results from the EPA's analysis did not support the addition of October for Connecticut. The EPA recognizes that there may be value in having a consistent O₃ season across multi-state planning areas. We recommend that monitoring agency representatives from New York and Connecticut contact their respective EPA Regional Office to jointly develop a monitoring plan to provide coverage of the MSA for a longer period of time. Consistent with the results from the EPA's analysis and consistent with our proposal, the EPA is finalizing the March–October season in New York and the March–September season in Connecticut.

Although no changes were proposed for Arkansas, the Arkansas Department of Environmental Quality recommended that the O₃ season in the nonattainment area that includes Crittenden County, Arkansas (March–November) be consistent with the O₃ seasons in Tennessee (March–October) and Mississippi (March–October) by either shortening the O₃ season in Arkansas or lengthening the O₃ season by one month in Tennessee and Mississippi. Based on the results from the EPA's analysis and consistent with our proposal, the EPA is not finalizing any changes to the current O₃ seasons in Arkansas, Tennessee, or Mississippi. There is currently one monitor operating in Crittenden County. We recommend that Arkansas work with their EPA Regional Administrator to consider a waiver for the monitor(s) in Crittenden County to allow a deviation (shortened season) from the required O₃ season if the agency demonstrates that such a deviation is appropriate for consistency in the nonattainment area.

Two commenters noted the need to extend seasons to capture wintertime O₃ events. One commenter urged the EPA to extend monitoring to year-round in the intermountain west (specifically Wyoming) to adequately capture summer and winter O₃ problem days

and noted especially two monitors in the Pinedale area of Wyoming that should be operated year-round. The EPA's analysis showed that there were no days that were ≥ 0.060 ppm in Wyoming for the months of October–December and that the Wyoming Department of Environmental Quality is currently operating about 70% of their O₃ monitors year-round including all O₃ monitors in Sublette County, which includes the Pinedale area. Another commenter supported lengthening the seasons for states in the western U.S. where wintertime O₃ could be an issue in light of the unique and growing O₃ pollution problems caused by oil and gas development activities. They also recommended that the EPA expand the O₃ monitoring season to year-round for North Dakota, South Dakota, and Montana beyond what was proposed. The number of observed days that were ≥ 0.060 ppm in the months outside the season proposed for these states (one day for North Dakota and no days observed for South Dakota and Montana) do not support a further extension to the length of the O₃ monitoring season beyond what was proposed. These states are already operating a large percentage of their monitors year-round (89% in North Dakota, 100% in South Dakota, and 78% in Montana). The EPA is finalizing the seasons as proposed in Wyoming (January–September), North Dakota (March–September), South Dakota (March–October), and Montana (April–September). The EPA encourages these states to continue year-round operation of their monitors to determine what areas are affected by elevated levels of winter-time O₃.

The commenters who opposed lengthening the O₃ monitoring seasons noted concerns with the threshold (0.060 ppm) used as the basis for the changes and the length of time (2010–2013) for which ambient data were retrieved and analyzed. Many of those with concerns recommended that levels in the proposed range (e.g., 0.065 ppm or 0.070 ppm) or the current NAAQS level of 0.075 ppm be used as the appropriate threshold for determining the O₃ season. With regard to the 0.060 ppm threshold used, this value is consistent with the 85 percent threshold used to require additional O₃ monitoring based on Appendix D requirements, which include the MSA population and design value.²²⁰ As noted previously, year-to-year variability occurs in seasonal O₃ patterns based on highly variable and unpredictable meteorological

²²⁰ See 40 CFR part 58, appendix D, Table D–2.

conditions, which can support the formation of early or late season elevated O₃ concentrations in some years and not in other years. This threshold serves as an appropriate indicator of ambient conditions that may be conducive to the formation of O₃ concentrations that approach or exceed the level of the NAAQS.

Certain logistical complexities were noted if longer seasons were required, including site access during winter and the challenge of getting the monitoring equipment ready in time. Four states noted concerns with operator safety and anticipated their inability to access sites due to early spring snowfall. The EPA agrees that site access could be an issue depending on weather conditions and notes that specific site monitoring season deviations may be appropriate. We suggest that this be addressed through the monitoring season waiver process with the EPA Regional Administrator. Any deviations based on the Regional Administrator's waiver of requirements must be described in the state's annual monitoring network plan and updated in AQS.

Several commenters had concerns about the additional cost and resources needed to expand the O₃ monitoring seasons. There was some disagreement with the EPA's total annual average cost estimate of \$230,000 which took into account the number of O₃ monitors already operating year-round across the country. Commenters noted specifically that the proposed extension of required monitoring seasons would increase operational costs and potentially impact the resources available for other monitoring efforts. The added cost of operating O₃ monitors over a longer period was noted by some commenters, referencing both the cost of staff to operate the monitors, as well as the additional wear and tear those O₃ monitors would experience over a longer operational period. They noted that extending their required monitoring season by adding the month of March would increase staffing requirements for monitor operation and quality assurance. They also noted that the life expectancy of equipment would be reduced due to increased wear and tear. The EPA acknowledges that operational costs for O₃ monitoring networks will incrementally increase in states where required seasons have been lengthened. We encourage monitoring agencies to review available technology and operational procedures to institute practices that could potentially reduce such costs, such as the automation of quality control and calibration checks and remote access to evaluate monitor operations. As noted earlier, all states

operated at least a portion of their O₃ monitoring network outside of the required O₃ season during the 2010–2013 data period and reported the data to AQS. In addition, many states are operating more than the minimum number of monitors required to support the basic monitoring objectives described in 40 CFR part 58, Appendix D. Some states have a large percentage of their total O₃ monitors operating outside the currently-required O₃ season and some states have a small percentage. In situations where states are already operating a large number of their O₃ monitors outside their current O₃ season, the actual cost increase will be less. In cases where states have a small number of monitors operating outside their current O₃ season, in addition to automation and remote access, those states could investigate with their Regional Administrator the process in 40 CFR part 58.14 for reducing the total number of operating monitors that are above the number required by 40 CFR, part 58, appendix D to offset the cost of extending the O₃ monitoring season in their state.

Two commenters had concerns about the 4-year period of time evaluated in the EPA's analysis and noted that the 4-year period of time evaluated does not take into account meteorological anomalies and other weather induced situations and is not consistent with the 3 years used to calculate design values. One state agency's comments referenced their own analysis showing concentrations going back 20 years. They noted that 2010 was an unusual year and inclusion of such an unusual year in the 4-year period (2010–2013) of the EPA's analysis provides too much weight on those data. As noted earlier, year-to-year variability occurs in seasonal O₃ patterns based on variable meteorological conditions and given the impracticality of forecasting such conditions that affect O₃ photochemistry, the EPA believes it is important that O₃ monitors operate when there is a reasonable possibility of ambient levels approaching the level of the NAAQS. Another state agency commented that 4 years appeared to be an unusual number of years given that design values are based on 3 years. To support the proposed rule in 2014, the EPA's analysis of O₃ seasons began in 2013. At that time the EPA's analysis considered the most recent 3 years of certified data (2010–2012) and updated the analysis to add a fourth year (2013) when the data were quality-assured, certified, and available in AQS. We used 4 years of data, including the most recent year (2013) to include an

additional year of potentially-variable meteorological conditions to propose changes to the seasons. The EPA treated all years equally and did not put any more weight on the 2010 data than any of the other years used in the analysis. The EPA believes that using recently-available data across multiple years to capture varying meteorological conditions was appropriate to support the decisions on extending the O₃ seasons. One commenter disagreed with the EPA's definition of year-round (at least 20 daily observations in all 12 months of at least 1 year of the 4-year period). The definition of year-round was used to estimate the number of monitors being operated outside a state's required O₃ season and also used for the EPA's Information Collection Request (ICR). All available data in AQS were used for the O₃ season analysis, including data from year-round monitors.

Two commenters noted that "regional consistency" is not a scientific reason and is not needed for making changes to the O₃ seasons. One commenter noted that significant geographical, meteorological and demographic differences exist between neighboring states that may not warrant identical monitoring seasons. The EPA notes that regional consistency was considered, but only important for a few states where little data were available and the neighboring states had more available data and a sufficient number of days that were ≥ 0.060 ppm to support the proposed O₃ season changes. Regional consistency was not important for other states.

Some commenters expressed support for the proposed requirement that NCore O₃ sites operate year-round. They questioned whether data from NCore stations outside the O₃ season will be used for designations and requested that the EPA exclude those data from the designations process. Consistent with the designations process for all criteria pollutants, the states, tribes, and the EPA use all data available in AQS that meet the quality assurance requirements in 40 CFR part 58, Appendix A for the designations process. Given that O₃ data from NCore stations will meet these requirements, there is no rational basis for excluding these data from comparison to the NAAQS. Accordingly, such data from NCore stations cannot be excluded and will be treated in a manner equivalent to all other O₃ data in AQS. The EPA expects that the highest O₃ values will occur during the required O₃ season; therefore, we don't anticipate that NCore data from the out-of-season months will contribute to the design value used in

the designations process. The EPA is finalizing the requirement for year-round O₃ monitoring at NCore stations.

The EPA Regional Administrators have previously approved deviations from the required O₃ monitoring seasons through rulemakings (64 FR 3028, January 20, 1999; 67 FR 57332, September 10, 2002; and 69 FR 52836, August 30, 2004). The current ambient monitoring rule, in paragraph 4.1(i) of 40 CFR part 58, Appendix D (71 FR 61319, October 17, 2006), allows the EPA Regional Administrators to approve changes to the O₃ monitoring season without rulemaking. The EPA is retaining the rule language allowing such deviations from the required O₃ monitoring seasons without rulemaking. In the finalized revision to paragraph 4.1(i) of 40 CFR part 58, Appendix D, the EPA is clarifying the minimum considerations that should be taken into account when reviewing requests, and clarifying that changes to the O₃ seasons finalized in this rule revoke all previously approved seasonal deviations. The EPA clarifies that all O₃ season waivers will be revoked when this final rule becomes effective. We encourage monitoring agencies with existing waivers to engage their EPA Regions as soon as possible to evaluate whether new or continued waivers are appropriate given the level of the revised O₃ NAAQS.

We received three comments for and three comments against early implementation of the revised O₃ seasons by the start of the applicable O₃ season in each state by January 1, 2016. Those commenters in favor of early implementation of the revised O₃ seasons are already operating a large percentage of O₃ monitors year-round or outside the current O₃ monitoring season in their state. Those commenters against early implementation cited concerns with the need for additional time to implement the revised O₃ seasons, especially in areas where access in order to service and support the monitoring equipment may be problematic during winter weather conditions, and the undue burden on already constrained state resources. One commenter noted that given the date for the final rule (October 1, 2015) that there is insufficient time for public review of their annual monitoring network plan due July 1, 2015, for early implementation in 2016. The EPA encourages those agencies who are able to implement the O₃ season changes early to do so by the start of the applicable O₃ season in their state in 2016. However, taking into consideration the timing and potential burden on monitoring agencies, the EPA

is finalizing the requirement for implementing the revised O₃ seasons no later than the start of the applicable O₃ monitoring season in 2017, as proposed.

3. Final Decisions on the Length of the Required O₃ Monitoring Seasons

Final changes to the required O₃ monitoring seasons are summarized in this section as well as in revised Table D-3 in 40 CFR part 58, Appendix D.

Detailed state-by-state technical information has been placed in the docket to document the basis for the EPA's decision on each state. This information includes state-by-state maps and number of days that were ≥ 0.060 ppm; distribution charts of the number of days that were ≥ 0.060 ppm by month and state; and detailed information regarding AQS site IDs, dates and concentrations of all occurrences of the 8-hour daily maximum of at least 0.060 ppm between 2010 and 2013. Summaries have also been prepared for each state including the former and proposed O₃ monitoring seasons.

No changes to the required O₃ monitoring season were proposed or finalized for these states: Alabama, Alaska, Arizona, Arkansas, California, Georgia, Hawaii, Kentucky, Northern Louisiana (AQCR ²²¹ 019, 022), Southern Louisiana (AQCR 106), Maine, Mississippi, Nevada, New Mexico, Oklahoma, Oregon, Tennessee, Southern Texas (AQCR 106, 153, 213, 214, 216), Vermont, Washington, Puerto Rico, Virgin Islands, Guam, and American Samoa. All existing O₃ season deviations or waivers are revoked.

Changes to the required O₃ monitoring seasons are finalized as follows for these states and the District of Columbia and all existing O₃ season deviations or waivers are revoked.

Colorado: Proposed addition of January, February, October, November, and December is finalized. The required season is revised to January–December.

Connecticut: Proposed addition of March is finalized, revising season to March–September.

Delaware: Proposed addition of March is finalized, revising season to March–October.

District of Columbia: Proposed addition of March is finalized, revising season to March–October.

Florida: Proposed addition of January, February, November, and December is finalized. The required season is revised to January–December.

Idaho: Proposed addition of April is finalized, revising season to April–September.

Illinois: Proposed addition of March is finalized, revising season to March–October.

Indiana: Proposed addition of March and October, revising season to March–October.

Iowa: Proposed addition of March is finalized, revising season to March–October.

Kansas: Proposed addition of March is finalized, revising season to March–October.

Maryland: Proposed addition of March is finalized, revising season to March–October.

Massachusetts: Proposed addition of March is finalized, revising season to March–September.

Michigan: Proposed addition of March and October is finalized, revising season to March–October.

Minnesota: Proposed addition of March is finalized, revising season to March–October.

Missouri: Proposed addition of March is finalized, revising season to March–October.

Montana: Proposed addition of April and May is finalized, revising season to April–September.

Nebraska: Proposed addition of March is finalized, revising season to March–October.

New Hampshire: Proposed addition of March is finalized, revising season to March–September.

New Jersey: Proposed addition of March is finalized, revising season to March–October.

New York: Proposed addition of March is finalized, revising season to March–October.

North Carolina: Proposed addition of March is finalized, revising season to March–October.

North Dakota: Proposed addition of March and April is finalized, revising season to March–September.

Ohio: Proposed addition of March is finalized, revising season to March–October.

Pennsylvania: Proposed addition of March is finalized, revising season to March–October.

Rhode Island: Proposed addition of March is finalized, revising season to March–September.

South Carolina: Proposed addition of March is finalized, revising season to March–October.

South Dakota: Proposed addition of March, April, May, and October is finalized, revising season to March–October.

Texas (Northern AQCR 022, 210, 211, 212, 215, 217, 218): Proposed addition of November is finalized, revising season to March–November.

Utah: Proposed addition of January, February, March, April, October,

²²¹ Air Quality Control Region.

November, and December is finalized. The required season is revised to January–December.

Virginia: Proposed addition of March is finalized, revising season to March–October.

West Virginia: Proposed addition of March is finalized, revising season to March–October.

Wisconsin: Proposed addition of March and April 1–15 is finalized, revising season to March–October 15.

Wyoming: Proposed addition of January, February, March, and removal of October is finalized, revising season to January–September.

Finally, we are finalizing the required O₃ monitoring season for all NCore stations to be year-round (January–December) regardless of the required monitoring season for the individual state in which the NCore station is located.

C. Revisions to the PAMS Network Requirements

Section 182 (c)(1) of the CAA required the EPA to promulgate rules for enhanced monitoring of O₃, NO_x, and VOCs for nonattainment areas classified as serious (or above) to obtain more comprehensive and representative data on O₃ air pollution. In addition, Section 185B of the CAA required the EPA to work with the National Academy of Sciences (NAS) to conduct a study on the role of O₃ precursors in tropospheric O₃ formation and control. As a result of this study, the NAS issued the report entitled, “Rethinking the Ozone Problem in Urban and Regional Air Pollution”, (NAS, 1991).

In response to the CAA requirements and the recommendations of the NAS report, on February 12, 1993 (58 FR 8452), the EPA revised the ambient air quality surveillance regulations to require PAMS in each O₃ nonattainment area classified as serious, severe, or extreme (“PAMS areas”). As noted in the EPA’s Technical Assistance Document (TAD) for Sampling and Analysis of Ozone Precursors (U.S. EPA, 1998), the current objectives of the PAMS program are to: (1) Provide a speciated ambient air database that is both representative and useful in evaluating control strategies and understanding the mechanisms of pollutant transport by ascertaining ambient profiles and distinguishing among various individual volatile organic compounds (VOCs); (2) provide local, current meteorological and ambient data to serve as initial and boundary condition information for photochemical grid models; (3) provide a representative, speciated ambient air database that is characteristic of source

emission impacts to be used in analyzing emissions inventory issues and corroborating progress toward attainment; (4) provide ambient data measurements that would allow later preparation of unadjusted and adjusted pollutant trends reports; (5) provide additional measurements of selected criteria pollutants for attainment/nonattainment decisions and to construct NAAQS maintenance plans; and (6) provide additional measurements of selected criteria and non-criteria pollutants to be used for evaluating population exposure to air toxics as well as criteria pollutants.

The original requirements called for two to five fixed sites per PAMS area depending on the area’s population. Four types of PAMS sites were identified including upwind (Type 1), maximum precursor emission rate (Type 2), maximum O₃ concentration (Type 3), and extreme downwind (Type 4) sites. Each PAMS site was required to measure O₃, nitrogen oxide (NO), NO₂, speciated VOCs, selected carbonyl compounds, and selected meteorological parameters. In addition, upper air meteorological monitoring was required at one site in each PAMS area.

In the October 17, 2006 monitoring rule (71 FR 61236), the EPA revised the PAMS requirements to only require two sites per PAMS area. The intent of the revision was to “allow PAMS monitoring to be more customized to local data needs rather than meeting so many specific requirements common to all subject O₃ nonattainment areas; the changes also gave states the flexibility to reduce the overall size of their PAMS programs—within limits—and to use the associated resources for other types of monitoring they consider more useful.” In addition to reducing the number of required sites per PAMS area, the 2006 revisions also limited the requirement for carbonyl measurements (specifically formaldehyde, acetaldehyde, and acetone) to areas classified as serious or above for the 8-hour O₃ standards. This change was made in recognition of carbonyl sampling issues which were believed to cause significant uncertainty in the measured concentrations.

Twenty-two areas were classified as serious or above O₃ nonattainment at the time the PAMS requirements were promulgated in 1993. On July 18, 1997 (62 FR 38856), the EPA revised the averaging time of the O₃ NAAQS from a 1-hour averaging period to an 8-hour averaging period. On June 15, 2005 (70 FR 44470), the EPA revoked the 1-hour; however, PAMS requirements were identified as requirements that had to be

retained in the anti-backsliding provisions included in that action. Therefore, PAMS requirements continue to be applicable to areas that were classified as serious or above nonattainment for the 1-hour O₃ standards as of June 15, 2004. Currently, 25 areas are subject to the PAMS requirements with a total of 75 sites. As will be discussed in detail later, the current PAMS sites are concentrated in the Northeast U.S. and California with relatively limited coverage in the rest of the country (Cavender, 2014).

The first PAMS sites began operation in 1994, and have been in operation for over 20 years. Since the start of the program, there have been many changes to the nature and scope of the O₃ problem in the U.S. as well as to our understanding of it. The O₃ standards has been revised multiple times since the PAMS program was first implemented. On July 18, 1997, the EPA revised the O₃ NAAQS to a level of 0.08 parts per million (ppm), with a form based on the 3-year average of the annual fourth-highest daily maximum 8-hour average O₃ concentration. On March 28, 2008 (73 FR 16436), the EPA revised the O₃ standards to a level of 0.075 ppm, with a form based on the 3-year average of the annual fourth-highest daily maximum 8-hour average O₃ concentration. These changes in the level and form of the O₃ NAAQS, along with notable decreases in O₃ levels in most parts of the U.S., have changed the landscape of O₃ NAAQS violations in the U.S. At the time of the first round of designations for the 8-hour standards (June 15, 2005), only 5 areas were classified as serious or above for the 8-hour standards as compared to 22 areas that were classified as serious or above for the 1-hour standards. While the number of serious and above areas decreased, the number of nonattainment areas remained nearly the same. In addition to the change in the landscape of O₃ nonattainment issues, much of the equipment used at PAMS sites is outdated and in need of replacement. New technologies have been developed since the inception of the PAMS program that should be considered for use in the network to simplify procedures and improve data quality. For these reasons, the EPA determined that it would be appropriate to re-evaluate the PAMS program as explained below.

In 2011, the EPA initiated an effort to re-evaluate the PAMS requirements in light of changes in the needs of PAMS data users and the improvements in monitoring technology. The EPA consulted with the Clean Air Science Advisory Committee (CASAC), Air

Monitoring and Methods Subcommittee (AMMS) to seek advice on potential revisions to the technical and regulatory aspects of the PAMS program; including changes to required measurements and associated network design requirements. The EPA also requested advice on appropriate technology, sampling frequency, and overall program objectives in the context of the most recently revised O₃ NAAQS and changes to atmospheric chemistry that have occurred over the past 10–15 years in the significantly impacted areas. The CASAC AMMS met on May 16 and May 17, 2011, and provided a report with their advice on the PAMS program on September 28, 2011 (U.S. EPA, 2011f). In addition, the EPA met multiple times with the National Association of Clean Air Agencies (NACAA) Monitoring Steering Committee (MSC) to seek advice on the PAMS program. The MSC includes monitoring experts from various State and local agencies actively engaged in ambient air monitoring and many members of the MSC have direct experience with running PAMS sites. Specific advice obtained from the CASAC AMMS and the MSC that was considered in making the proposed changes to the PAMS requirements is discussed in the appropriate sections below.

Based on the findings of the PAMS evaluation and the consultations with the CASAC AMMS and NACAA MSC, the EPA proposed to revise several aspects of the PAMS monitoring requirements including changes in (1) network design, (2) VOC sampling, (3) carbonyl sampling, (4) nitrogen oxides sampling, and (5) meteorology measurements. The following paragraphs summarize the proposed changes, the comments received, and the final changes and supporting rationale.

1. Network Design

As discussed above, the current PAMS network design calls for two sites (a Type 2, and a Type 1 or Type 3) per PAMS area. In their report (U.S. EPA, 2011f), the CASAC AMMS found “that the existing uniform national network design model for PAMS is outdated and too resource intensive,” and recommended “that greater flexibility for network design and implementation of the PAMS program be transferred to state and local monitoring agencies to allow monitoring, research, and data analysis to be better tailored to the specific needs of each O₃ problem area.” While stating that the current PAMS objectives were appropriate, the AMMS report also stated that “objectives may need to be revised to include both a

national and regional focus because national objectives may be different from regional objectives.” The NACAA MSC also advised the EPA that the existing PAMS requirements were too prescriptive and may hinder state efforts to collect other types of data that were more useful in understanding their local O₃ problems.

The EPA agrees with CASAC that the PAMS objectives include both local and national objectives, and believes that the current PAMS network design is no longer suited for meeting either sets of objectives. As part of the PAMS evaluation, it was determined that at the national level the primary use of the PAMS data has been to evaluate photochemical model performance. Due to the locations of the current PAMS areas and the current network design, existing PAMS sites are clustered along the northeast and west coasts leading to significant redundancy in these areas and very limited coverage throughout the remainder of the country (Cavender, 2014). The resulting uneven spatial coverage greatly limits the value of the PAMS data for evaluation of model performance. CASAC (U.S. EPA, 2011f) noted the spatial coverage issue and advised that the EPA should consider requiring PAMS measurements in areas in addition to “areas classified as serious and above for the O₃ NAAQS to improve spatial coverage.” The EPA also agrees with CASAC and NACAA that the PAMS requirements should be revised to provide monitoring agencies greater flexibility in meeting local objectives.

The EPA proposed changes to the network design requirements to better serve both national and local objectives. The EPA proposed a two part network design. The first part of the design included a network of fixed sites (“required PAMS sites”) intended to support O₃ model development and the tracking of trends of important O₃ precursor concentrations. The second part of the network design required states with O₃ non-attainment areas to develop and implement Enhanced Monitoring Plans (EMPs) which were intended to allow monitoring agencies the needed flexibility to implement additional monitoring capabilities to suit the needs of their area.

To implement the fixed site portion of the network design, the EPA proposed to require PAMS measurements at any existing NCore site in an O₃ nonattainment area in lieu of the current PAMS network design requirements.²²²

²²² The EPA noted that the proposed change would expand the PAMS applicability beyond that required in 182(c)(1) of the CAA. Thus, in this final

The NCore network is a multi-pollutant monitoring network consisting of 80 sites (63 urban, 17 rural) sited in typical neighborhood scale locations and supports multiple air quality objectives including some of the objectives of the PAMS program including the development and evaluation of photochemical models (including both PM_{2.5} and O₃ models), development and evaluation of control strategies, and the tracking of regional precursor trends.

The EPA recognized that in limited situations existing NCore sites may not be the most appropriate locations for making PAMS measurements. For example, an existing PAMS site in an O₃ nonattainment area may be sited at a different location than the existing NCore site. In this case, it may be appropriate to continue monitoring at the existing PAMS site to support ongoing research and to maintain trends information. To account for these situations, the EPA also proposed to provide the EPA Regional Administrator the authority to approve an alternative location for a required PAMS site where appropriate. The EPA also solicited comments on alternative frameworks using other benchmarks such as attainment status or population to ensure an appropriately sized fixed PAMS monitoring network. The EPA received several comments on the proposed changes to the network design, primarily from state and local monitoring agencies. The following paragraphs summarize the major comments made on the proposed network design, our response, and final network design requirements.

Most commenters agreed with the need to revise the existing network design. One commenter agreed that “requiring PAMS monitoring at already existing NCore locations will benefit national and local objectives to understand ozone formation and would also provide significant cost efficiencies.” Another commenter stated that they supported the proposed changes, “especially the flexibility provided by EMPs designed to meet local objectives and achieve a better understanding of photochemical precursors.” Another commenter supporting the changes stated that the “proposed network revision will provide states the flexibility to use their resources effectively.” One commenter stated that the proposed changes “reflect a more efficient use of state and local monitoring resources by availing

rule, the EPA is relying on the authority provided in Sections 103(c), 110(a)(2)(B), 114(a) and 301(a)(1) of the CAA to expand the PAMS applicability to areas other than those that are serious or above O₃ nonattainment.

monitoring agencies of existing NCore infrastructure to fulfill PAMS requirements.”

A number of concerns were also raised with the proposed network design. Several commenters stated that the proposal “would drastically reduce the PAMS network in the Northeast.” One commenter stated that “this is not acceptable for the Northeast and Mid-atlantic Corridor, which requires monitoring of the complex transport from multiple large metropolitan areas in the region.” One commenter recognized that the EPA had intended to allow states to use EMPs to address upwind and downwind data needs, but raised concerns that states with historically important upwind and downwind sites in the Ozone Transport Region ²²³ (OTR) may not be required to develop an EMP since those sites would be in states that are attaining the O₃ NAAQS. One commenter suggested that “the EPA consider the entire OTR when designing a PAMS network rather than pockets of nonattainment areas in the region.” The EPA agrees that the reduction of sites in the OTR is a potential issue and that many important existing PAMS sites would not be part of the required PAMS sites based on the proposed network design. As noted by several commenters, the EPA intended the state directed EMPs to give states flexibility in determining data needed to understand local O₃ formation, including transport in the Northeast. However, the EPA also agrees that as proposed many states in the OTR would not be required to develop EMPs and, therefore, may not be provided PAMS resources. To address these concerns and ensure adequate network coverage in the OTR, the EPA is adding a requirement that all states in the OTR develop and implement an EMP regardless of O₃ attainment status. This change will help ensure that an EMP appropriate for the entire OTR can be implemented.

Concerns were raised by some states that existing NCore sites may not be the most appropriate location for making PAMS measurements. One commenter noted that their NCore site was inland but that their “most significant ozone problems occur along the shoreline due to transport along the lake”, and that “the NCore site cannot provide insight into these important lakeshore ozone processes.” Another commenter stated that “while it was laudable to leverage

²²³ Section 184(c) of the CAA establishes the OTR as comprised of the states of Connecticut, Delaware, Maine, Maryland, Massachusetts, New Hampshire, New Jersey, New York, Pennsylvania, Rhode Island, Vermont, and Consolidated Metropolitan Statistical Area that includes the District of Columbia.

sites where data is already being collected, it is unclear whether NCore sites adequately meet the objectives of the PAMS program”, and that “the current NCore network may not be adequate to depict boundary conditions or areas of maximum emissions.” One commenter stated that “in some nonattainment areas an NCore site may be an appropriate location for a PAMS monitor, but in other areas it would be preferable to install the PAMS monitoring in a location downwind of a source region where higher ozone exposures occur” and that “State and local boundaries should not be part of the network design criteria.” One commenter noted that while the EPA had proposed to allow waivers, it was unclear if waivers would be allowed where the alternative site was in a different CBSA or state than the required PAMS site. As stated in our proposal, the EPA recognizes that in some cases existing PAMS sites (or other sites) may be better suited to meet local and national data needs. For this reason, we had proposed to allow waivers in these situations. We do agree that it is appropriate in some cases to allow these waivers to cross CBSA and state boundaries. Therefore, we have added specific language to the final waiver provisions to clarify that waivers can be allowed to cross CBSA and state boundaries. Where a monitoring agency receives a waiver from siting a monitor in reliance on a monitor operated by a different monitoring agency (*e.g.*, across state lines), the waiver will be conditioned on the monitor being properly included in the other agency’s network plan, and operated in accordance with the requirements of Part 58, including the relevant appendices.

In addition to the concerns raised about closing important existing PAMS sites discussed above, some commenters raised concerns that many of the newly required PAMS sites would be in locations that were expected to attain the revised O₃ NAAQS soon after the new sites would be installed. One commenter noted that “requiring marginal nonattainment areas to install PAMS sites would result in a large undertaking in an area that would most likely be back in attainment at or around the time the PAMS site started collecting data.” One commenter stated that by tying the network requirement to NAAQS attainment “threatens to underserve areas that are very close to exceeding the revised ozone NAAQS and results in significant gaps in the spatial coverage of the PAMS network” and “has the potential to introduce

undesirable uncertainty on the size and spatial extent of the PAMS network over the long term.” Another commenter was concerned that the proposed network would be unstable, and would experience frequent changes as areas came into attainment or went out of attainment thus reducing the value of the data collected, and resulting in inefficient use of resources. One commenter noted that “a more stable monitoring network design will allow for the examination of trends from spatially robust, long running sites and will allow states to firmly establish the infrastructure costs.”

The EPA noted in the proposal that the size and locations of the proposed required PAMS network is sensitive to the level of the revised O₃ NAAQS and future O₃ concentrations. We recognize and agree that if current downward trends in O₃ concentrations continue, many initially required sites may no longer be required to make PAMS measurements soon after the sites were installed. Non-required sites could be closed, soon after being installed, at the state’s discretion. We agree this would result in an inefficient use of resources. We also note that if these sites were closed following a potential reclassification to attainment, the loss of those sites could lead to a network with poor spatial coverage. Therefore, the EPA is making changes to the proposed revisions to the network design to improve the stability of the fixed site network. As explained below, the final requirements are based on options for which we requested comments in the proposal and the comments we have received.

We requested comments on additional options to define the fixed PAMS network component of the new network design. These options were further discussed in a memorandum to the docket (Cavender, 2014). One option discussed was to require PAMS measurements at all NCore sites irrespective of the O₃ attainment status of the area. One commenter noted that “requiring PAMS monitoring at all NCore sites, regardless of ozone attainment status, provides the most spatially robust and stable monitoring network.” We noted that this requirement would result in a network of approximately 80 sites, which would be larger than the current network. In the supporting memorandum, we noted that a fixed network of 80 sites would strain existing resources and would not allow adequate resources to implement the state directed EMPs.

Another option discussed in the proposal included requiring PAMS measurements at NCore sites in O₃

nonattainment areas with a population greater than 1,000,000. We noted that this option would result in a network of between 31 and 37 sites depending on the level of the revised O₃ NAAQS. We also noted that focusing the applicability of PAMS to those NCore sites in larger CBSAs would still provide the desired improvement in geographic distribution while reducing the number of required sites down to a level that would provide sufficient resources to implement the state-directed EMP portion of the network. One commenter stated that they “supported a 1,000,000 population threshold because it would help prioritize resources to areas based on the greatest human health impacts.” In addition, a number of commenters, while not commenting on the need for a population limit, did raise concerns about their ability to acquire and retain staff with the necessary expertise to collect PAMS measurements in less urbanized areas. As with the proposed network design, we recognize that the total number of sites and the ultimate spatial coverage under this option is also sensitive to changes in O₃ concentrations. If current downward trends in O₃ concentrations continue, many initially required sites would not be required soon after they were installed. As with the proposed option, this option could result in an unstable network resulting in an inefficient use of resources and inadequate spatial coverage to meet the network goals discussed above.

Upon further consideration and in response to the comments received, we are finalizing a network design that includes a requirement for states to make PAMS measurements at all NCore sites in CBSAs with a population of 1,000,000 people or more, irrespective of O₃ attainment status. We believe this requirement will result in an appropriately sized network (roughly 40 sites) that will provide adequate spatial coverage to meet national model evaluation needs (Cavender, 2015). Redundancy is greatly reduced while important network coverage is added in the midwest, southeast, and mountain west. The improved spatial coverage will also strengthen the EPA’s ability to track trends in precursor concentrations regionally.

Because the network requirement is not tied to attainment status, this final requirement will ensure network stability and allows for more efficient use of available resources. This final requirement also removes uncertainty as to applicability and aids planning and logistics involved with implementing the new requirements. Monitoring

agencies can determine the applicability of the fixed site requirements to their areas today, and begin to make plans for investments in equipment, shelter improvements, and staffing and training needs necessary to implement the fixed site requirements without having to wait for the designations process to be completed. In addition, this final requirement should alleviate concerns raised by monitoring agencies in more rural locations over the ability to attract and retain staff with the skills necessary to make PAMS measurements.

By adding the PAMS measurements to existing NCore sites, significant efficiencies can be obtained which should further reduce the costs of the fixed site network as NCore sites currently make many of the PAMS measurements. Furthermore, adding the additional PAMS measurements (*e.g.*, speciated VOCs, carbonyls, and mixing height) to existing NCore sites will improve our ability to assess other pollutants (*e.g.*, air toxics and PM_{2.5}).

Although, as discussed in comment and summarized above, we believe there are good reasons for not tying the requirement for fixed PAMS sites to O₃ attainment status, we continue to believe that requiring PAMS measurements in areas that historically have had low O₃ concentrations is unlikely to provide data of significant value to warrant the expense and effort of making such measurements. Therefore, we have included a provision that would allow a monitoring agency to obtain a waiver, based on Regional Administrator approval, in instances where CBSA-wide O₃ design values are equal to or less than 85% of the 8-hour O₃ NAAQS and where the site is not considered an important upwind or downwind site for other nonattainment areas. The EPA selected 85% as the threshold for this waiver provision as it has been used historically to identify locations needing additional monitoring for both the O₃ and PM_{2.5} NAAQS. The EPA will work with the monitoring agencies and the Regions to help ensure consistent implementation of this waiver provision.

The second part of the proposed PAMS network design included monitoring agency directed enhanced O₃ monitoring activities intended to provide data needed to understand an area’s specific O₃ issues. To implement this part of the PAMS network design, the EPA proposed to add a requirement for states with O₃ nonattainment areas to develop an EMP. The purpose of the EMP was to improve monitoring for ambient concentrations of O₃, NO_x, total

reactive nitrogen (NO_y)²²⁴, VOC, and meteorology. The EPA suggested that types of activities that might be included in the state’s EMP could include additional PAMS sites (*e.g.*, upwind or downwind sites), additional O₃ and NO_x monitoring, ozonesondes or other aloft measurements, rural measurements, mobile PAMS sites, additional meteorological measurements, and episodic or intensive studies. The intent of the EMPs is to allow monitoring agencies flexibility in determining and collecting the information they need to understand their specific O₃ problems.

We received comments on the proposed requirement for an EMP in states with O₃ nonattainment areas. Most comments supported the requirement, but other comments raised a number of concerns. A number of commenters questioned the need for EMPs in Marginal and Moderate O₃ nonattainment areas. They noted that in most cases, Marginal O₃ nonattainment areas were expected to come into compliance without state-specific controls. One commenter stated that “nonattainment areas projected to attain the standard without additional state-level actions may not need the PAMS resources and additional monitoring to develop a better understanding of their ozone issues.” One commenter noted that “marginal ozone nonattainment areas are given only a few requirements because it is assumed that the areas will reach attainment within three years.” Another commenter stated “requiring enhanced monitoring for any marginal or moderate area should only be implemented where such analyses show the need for this data.” The EPA agrees that based on current trends in O₃ concentrations and the EPA’s own projections, states in Marginal nonattainment areas likely will comply with the revised NAAQS without additional state-directed controls, and as such, an EMP is not necessary in Marginal O₃ attainment areas. Accordingly, the EPA is finalizing a requirement for EMPs in areas classified as Moderate or above O₃ nonattainment and, thereby, removing the applicability of the requirement for Marginal areas. We believe this final requirement will provide the desired flexibility to allow states to identify enhanced monitoring needs while focusing resources for EMPs in areas of greater need of enhanced monitoring data.

Commenters expressed concerns over the lack of detail on what an approvable EMP would entail. As proposed, the

²²⁴ NO_y includes NO, NO₂, and other oxidized nitrogen compounds (NO_x).

EMPs would be reviewed and approved by the EPA Regional Administrator as part of the annual monitoring plan review process. One commenter recommended that the “EPA detail the requirements of the EMPs for ozone nonattainment areas in future implementation guidance.” One commenter stated that the “EPA should provide some coordination between regional offices and technical guidance to state agencies that would be of assistance in developing and executing the EMPs.” The requirements for the EMPs were intentionally left quite general in order to maximize the flexibility for states in identifying their specific data needs. Regional approval of the plans is required to ensure the enhanced monitoring planned will be commensurate with grant funds provided for EMPs. Nonetheless, the EPA understands the need for guidance on developing EMPs and commits to working with monitoring agencies and the regions to develop appropriate guidance on developing and reviewing EMPs.

2. Speciated VOC Measurements

Measurement of speciated VOCs important to O₃ formation is a key aspect of the PAMS program. The existing PAMS requirements allow for a number of options in measuring speciated VOCs at PAMS sites which include (1) hourly measurements using an automatic gas chromatograph (“autoGC”), (2) eight 3-hour samples daily using canisters, or (3) one morning and one afternoon sample with a 3-hour or less averaging time daily using canisters plus continuous Total Non-methane Hydrocarbon (TNMHC) measurements.

The EPA believes that the current options provided for VOC measurement limit the comparative value of the data being collected, and proposed that required PAMS sites must measure and report hourly speciated VOCs, which effectively would require them to use an autoGC to measure VOCs in lieu of canisters. More complete and consistent speciated VOC data nationally would better help meet certain objectives of the PAMS program described above (e.g., a speciated ambient air database useful in evaluating control strategies, analyzing emissions inventory issues, corroborating progress toward attainment, and evaluating population exposure to air toxics). Furthermore, as noted by the CASAC AMMS, hourly VOC data are “particularly useful in evaluating air quality models and performing diagnostic emission attribution studies. These data can be provided on a near real-time basis and

presented along with other precursor species (e.g., oxides of nitrogen and carbon monoxide) collected over similar averaging times.” Longer time-averaged data are of significantly lower value for model evaluation. In addition, creating consistent monitoring requirements across the network would provide better data for analyzing regional trends and spatial patterns.

At the time the original PAMS requirements were promulgated, the canister options were included because the EPA recognized that the technologies necessary to measure hourly average speciated VOC concentrations were relatively new and may not have been suitable for broad network use. At that time, GCs designed for laboratory use were equipped with auto-samplers designed to “trap” the VOC compounds from a gas sample, and then “purge” the compounds onto the GC column. The EPA did not believe that autoGCs were universally appropriate due to the technical skill and effort necessary at that time to properly operate an autoGC.

While the basic principles of autoGC technology have not changed, the hardware and software of modern autoGCs are greatly improved over that available at the time of the original PAMS requirements. Based on advice from the CASAC AMMS, the EPA initiated an evaluation of current autoGCs potentially suitable for use in the PAMS network. Based on the preliminary results, the EPA believes that typical site operators, with appropriate training, will have the skill necessary to operate a modern autoGC successfully. Considering the advances in autoGC technology, the added value obtained from hourly data, and the proposed move of PAMS measurements to NCore sites in O₃ nonattainment areas, the EPA proposed to require hourly speciated VOC sampling at all PAMS sites. The EPA noted that this proposed requirement would effectively prevent the use of canisters to collect speciated VOCs at the required PAMS sites but that canister sampling may continue to be an appropriate method for collecting speciated VOCs at other locations as part of discretionary monitoring designed within the EMPs.

While the EPA believes that the proposed transition to hourly speciated VOC sampling is the appropriate strategy to take advantage of improved technology and to broaden the utility of collected data, we are also mindful of the additional rigidity that the proposed mandatory use of autoGCs may have for monitoring agencies, especially those that have experience with and have established effective and reliable

canister sampling programs. Therefore, the EPA requested comment on the proposed requirement for hourly VOC sampling as well as the range of alternatives that might be appropriate in lieu of a strict requirement.

The EPA received a number of comments on the requirement to measure hourly VOCs at required PAMS sites. Many commenters agreed with requiring hourly VOC data. One commenter agreed that “hourly VOC data collection is the most appropriate and useful for PAMS monitors” and that “it is only appropriate to approve an alternative data collection interval if it is believed that the high ozone in an area is due to other pollutants, such as NO_x or methane.” One commenter stated they “supported the movement towards hourly PAMS VOC speciated measurements with flexibility to use canisters if programmatic or logistical needs indicate.”

However, some commenters raised concerns with the hourly VOC requirement. Some commenters questioned if autoGCs would be capable of measuring important VOC species in their environment. One commenter noted that in their location (high desert) “the largest VOC present in our inventory is creosote, a compound not commonly measured with this instrumentation.” One commenter stated that the “Southeastern United States is dominated by biogenic VOC emissions” and questioned “the benefits of an autoGC in understanding ozone formation in any potential nonattainment area in our State.”²²⁵ Some questioned the detection capabilities of autoGCs as compared to canister sampling. One commenter found that the method detection limit (MDL) for their canister sampling was “consistently equal to or less than the autoGC instrumentation” based on the EPA’s autoGC evaluation laboratory report (RTI, 2014). Another commenter noted that the MDLs for many of the compounds and systems reported in the laboratory report were too high to be useful at PAMS sites. Another commenter stated that they found that “retention-time shifts made it difficult for instant identification of chemical peaks” and that “states should be allowed the flexibility to continue using canisters instead of autoGC.”

As noted in the preamble, and the comments received, the EPA is currently completing an evaluation of

²²⁵ The EPA notes that isoprene (the dominant biogenic compound in the Southeast) is well measured using autoGCs. The EPA is also evaluating the potential of modern autoGC’s to measure alpha and beta pinene; however that work is not complete.

commercially available autoGCs. A copy of the report for the laboratory phase of the study is available in the docket (RTI, 2014). As noted in the laboratory report, the MDL estimates made for the laboratory study were not conducted according to normal MDL testing procedures and as such the results should only be used to compare the various instruments being tested against each other.²²⁶ As part of the evaluation, the EPA identified the manufacturer's specifications for MDL. Most of the systems that are being evaluated have a manufacturer's estimated MDL in the range of 0.1 ppb to 0.5 ppb. Based on the evaluation of MDL capabilities and typical ambient concentrations of O₃ precursors, the EPA believes that autoGCs are an appropriate method for gathering VOC data at most urban locations. However, canister sampling may be more appropriate in locations with low VOC concentrations.

For the reasons discussed above and in the proposed rule, the EPA is finalizing a requirement for hourly speciated VOC measurements at required PAMS sites. The EPA believes that hourly VOC measurements will provide a more complete and consistent speciated VOC database to help meet the PAMS program objectives described above. Hourly VOC data are particularly useful in evaluating air quality models and performing diagnostic emission attribution studies. Longer time-averaged data are of lower value for model evaluation. Consistent monitoring requirements across the network will provide better data for analyzing regional trends and spatial patterns.

However, the EPA agrees that there may be locations where an autoGC may not be the most appropriate method for VOC measurement and that it is appropriate to allow for canister sampling in limited situations. Accordingly, the EPA is adding a waiver option (to be approved by the EPA Regional Administrator) to allow three 8-hour average samples every 3rd day as an alternative in cases where VOCs are not well measured by autoGC due to low concentrations of target compounds

²²⁶ Several factors combined to result in the high relative MDL estimates reported in laboratory report. The MDL testing in the laboratory was conducted during concurrent tests for interferences from humidity and temperature. In addition, the MDL testing was conducted at relatively high concentrations compared to the concentrations testing would be conducted at for conventional MDL testing. Finally, as noted in the laboratory report, a number of instruments were having technical difficulties during the testing which greatly impacted their MDL results. The EPA is continuing the autoGC evaluation and has conducted a field study during the summer of 2015. A final report is expected in early 2016.

or where the predominant VOC compounds cannot be measured using autoGC technology (e.g., creosote in high desert environments). This alternative sampling frequency was selected to be consistent with the sampling frequency selected for carbonyls, which is discussed later in this preamble.

3. Carbonyl Measurements

Carbonyls include a number of compounds important to O₃ formation that cannot currently be measured using the autoGCs or canisters used at PAMS sites to measure speciated VOCs. The current method for measuring carbonyls in the PAMS program is Compendium Method TO-11A (U.S. EPA, 1999). In this method, carbonyl compounds are adsorbed and converted into stable hydrazones using dinitrophenylhydrazine (DNPH) cartridges. These cartridges are then analyzed for the individual carbonyl compounds using liquid chromatography (LC) techniques. Three carbonyls are currently required to be measured in the PAMS program—formaldehyde, acetaldehyde, and acetone.

In 2006, the EPA revised the PAMS requirements such that carbonyl sampling was only required in areas classified as serious or above nonattainment for O₃ under the 8-hour O₃ standard which effectively reduced the applicability of carbonyl sampling to a few areas in California. This change was made in recognition that there were a number of issues with Method TO-11A that raised concerns with the uncertainty in the carbonyl data being collected. These issues include interferences (humidity and O₃) and breakthrough (i.e., overloading of the DNPH cartridge) at high concentrations. While solutions for these issues have been investigated, these improvements have not been incorporated into Method TO-11A.

A recent evaluation of the importance of VOCs and carbonyls to O₃ formation determined that carbonyls, especially formaldehyde, are very important to O₃ formation (Cavender, 2013). CASAC AMMS (U.S. EPA, 2011f) also noted the importance of carbonyls stating that "There are many compelling scientific reasons to measure carbonyls. They are a very important part of O₃ chemistry almost everywhere." Although the EPA recognizes the issues that have been raised about the current method of measuring carbonyls, due to the importance of carbonyls to understanding O₃ chemistry, the EPA proposed to require all required PAMS sites to measure carbonyls.

Several commenters agreed with the need for carbonyl data at PAMS sites. However, a number of commenters questioned the proposed frequency of eight 3-hour samples every day during the PAMS sampling season (June through August). Several commenters indicated that the frequency was too high. One commenter noted that the requirement would require 800 samples per season at each PAMS site and pointed out that this requirement, which was required at the inception of the PAMS program in the 1990s was "found to be prohibitively expensive, technically unsustainable, and qualitatively compromised." Another commenter stated that "this level of sampling would require a substantial amount of agency resources and seems unduly burdensome." A number of commenters also questioned the commercial availability of an 8-channel carbonyl sampler that would be needed to take eight 3-hour samples daily. In light of the comments and upon further review, the EPA agrees that the proposed frequency is unduly burdensome and is finalizing a requirement with a lower frequency.

A number of alternative frequencies were suggested in the comments. Several commenters suggested a frequency of three 8-hour samples on either a 1-in-6 day or 1-in-3 day basis. Another commenter suggested a frequency of eight 3-hour samples on a 1 in 6 day basis. The EPA notes that sampling on a 1-in-6 day frequency would lead to as little as 15 sampling days per PAMS sampling season. The EPA believes that 15 sampling days is too few to provide a meaningful representation of carbonyl concentrations over the PAMS sampling period. A sampling frequency of 1-in-3 days would lead to 30 sampling days per season with each day of the week being represented at least 4 times per sampling season. With regards to samples per day, a 3-hour sampling duration provides a better diurnal representation of carbonyl sampling compared with an 8-hour sampling duration; however 8-hour sampling can provide information useful for evaluating diurnal differences in carbonyl concentrations. Upon further consideration and in light of the comments received, the EPA is finalizing a carbonyl sampling requirement with a frequency of three 8-hour samples on a 1-in-3 day basis. This final requirement will result in approximately 90 samples per PAMS sampling season which the EPA believes is not unduly burdensome and

will provide a reasonable representation of carbonyl concentrations.

A number of commenters noted the ongoing development of continuous formaldehyde instruments, and recommended that EPA allow for continuous formaldehyde measurements as an alternative to the manual cartridge based TO-11A method. The EPA agrees that continuous formaldehyde, with the ability to obtain hourly averaged measurements, would be a significantly more valuable than the longer averaged measurements. As a result, the EPA has added an option to allow for continuous formaldehyde as an alternative to the carbonyl measurements using TO-11A.

4. Nitrogen Oxides Measurements

It is well known that NO and NO₂ play important roles in O₃ formation (U.S. EPA, 2013, Section 3.2.2). Under the current network design, Type 2 PAMS sites are required to measure NO_x (which by definition is the sum of NO and NO₂), and Types 1, 3, and 4 sites are required to measure NO_y. NCore sites are currently required to measure NO_y but are not required to measure NO₂ separately.

In conventional NO_x analyzers, NO₂ is determined as the difference between the measured NO and NO_x concentrations. However, due to the non-selective reduction of oxidized nitrogen compounds by the molybdenum converter used in conventional NO_x monitors, the NO₂ measurement made by conventional NO_x monitors can be biased high due to the varying presence of NO_z compounds that may be reported as NO₂. The unknown bias from the NO_z compounds is undesirable when attempting to understand O₃ chemistry.

Improvements in reactive nitrogen measurements have been made since the original PAMS requirements were promulgated that allow for improved NO₂ measurements. Selective photolytic converters have been developed that are not significantly biased by NO_z compounds (Ryerson et al., 2000). Monitors using photolytic converters are commercially available and have been approved as FEMs for the measurement of NO₂. In addition, methods that directly read NO₂ have been developed that allow for very accurate readings of NO₂ without some of the issues inherent to the "difference method" used in converter-based NO_x analyzers. However, these direct reading NO₂ analyzers generally do not provide an NO estimate, and would need to be paired with a converter-based NO_x monitor or NO_y monitor in order to also measure NO.

As discussed above, the EPA is finalizing a PAMS network design such that PAMS measurements will be required at existing NCore sites in CBSAs with a population of 1,000,000 people or more. NCore sites currently are required to measure NO and NO_y. NCore sites are not currently required to measure NO₂. Due to the importance of accurate NO₂ data to the understanding of O₃ formation, the EPA proposed to require NO₂ measurements at required PAMS sites. Since existing NCore sites currently measure NO_y, either a direct reading NO₂ analyzer or a photolytic-converter NO_x analyzer could be used to meet the proposed requirement. The EPA believes conventional NO_x analyzers would not be appropriate for making PAMS measurements due to the uncertainty caused by interferences from NO_z compounds.

A number of commenters questioned the need for both NO_y and NO₂ measurements at PAMS sites. One commenter stated that "in dense urban areas an NO/NO₂/NO_x instrument may be adequate but in a more rural area an NO/NO_y instrument may be preferable." Another commenter stated that due to the size of the grid cells used in grid models that "the impact of NO_z interferences would be very small compared to other modeling uncertainties such as emission inventories and mixing heights." Another commenter suggested that "EPA should provide clear and specific guidance on how agencies can request that the NO_y monitoring be eliminated from the NCore suite based on comparative data between the NO₂ and NO_y monitors."

The comments suggest that the model's ability to simulate the partitioning of reactive nitrogen is unimportant because there may be other errors in the model. The EPA believes that measurements should be routinely collected so that it can be demonstrated that the chemistry, meteorology, and emissions in the model are all of sufficient reliability for use in informing air quality management decisions. Monitoring sites rarely fall into simple categories of urban or rural, and the speciation of NO_y varies considerably as a function of meteorology and time of day at a given site. The state-of-the-science in regulatory air quality modeling is such that accurate measurements of key O₃ precursors must be available to demonstrate the credibility of the model predictions. The increased availability of special field study observations is leading to increased scrutiny of the chemical mechanisms used in regulatory modeling. Comprehensive and accurate

measurement sites are needed to demonstrate the adequacy of the models and to respond to these challenges.

Measurements of NO, NO₂, and NO_y concentrations are critical to understanding atmospheric aging and photochemistry. These measurements will provide essential information about whether NO_y compounds are fresh or aged which is important for understanding both local photochemistry (*i.e.* through indicator ratios to distinguish NO_x vs VOC limited conditions) as well as for characterizing transport from upwind regions. These evaluations may be conducted using observations, box modeling or through complex photochemical grid based modeling. Accurate speciated and total NO_y measurements are necessary for all three types of analysis. For these reasons, the EPA is finalizing the requirement for required PAMS sites to measure true NO₂ in addition to NO and NO_y.

5. Meteorology Measurements

The current PAMS requirements require monitoring agencies to collect surface meteorology at all required PAMS sites. As noted in the EPA's Technical Assistance Document (U.S. EPA, 1998) for the PAMS program, the PAMS requirements do not provide specific surface meteorological parameters to be monitored. As part of the implementation efforts for the original PAMS program, a list of recommended parameters was developed and incorporated into the TAD which includes wind direction, wind speed, temperature, humidity, atmospheric pressure, precipitation, solar radiation, and ultraviolet (UV) radiation. Currently, NCore sites are required to measure the above parameters with the exceptions of atmospheric pressure, precipitation, solar radiation, and UV radiation. In recognition of the importance of these additional measurements for understanding O₃ formation, the EPA proposed to specify that required PAMS sites are required to collect wind direction, wind speed, temperature, humidity, atmospheric pressure, precipitation, solar radiation, and UV radiation. Since NCore sites are currently required to measure several of these surface meteorological parameters, the net impact of the proposal was to add the requirement for the monitoring of atmospheric pressure, precipitation, solar radiation, and UV radiation at affected NCore sites. The EPA received no significant comments on this portion of the proposal, and therefore is finalizing the requirement as proposed.

The existing PAMS requirements also require the collection of upper air meteorological measurements at one site in each PAMS area. The term upper air meteorological is not well defined in the existing PAMS requirements. As part of the implementation efforts for the original PAMS program, mixing height was added to the PAMS TAD as a recommended meteorological parameter to be monitored. Most monitoring agencies installed radar profilers to meet the requirement to collect upper air meteorology. Radar profilers provide data on wind direction and speed at multiple heights in the atmosphere. Radio acoustic sounding system (RASS) profilers are often included with radar profilers to obtain atmospheric temperature at multiple heights in the atmosphere and to estimate mixing height. The EPA recognizes that the upper air data on wind speed and wind direction from radar profilers can be very useful in O₃ modeling. However, many of the current PAMS radar profilers are old and in need of replacement or expensive maintenance. In addition, the cost to install and operate radar profilers at all required PAMS sites would be prohibitive. Therefore, the EPA did not propose to add upper air wind speed and direction as required meteorological parameters to be monitored at required PAMS sites. Where monitoring agencies find the radar profiler data valuable, continued operation of existing radar profilers or the installation of new radar profilers would be appropriate to consider as part of the state's EMP.

As discussed above, mixing height is one upper air meteorological measurement that has historically been measured at PAMS sites. A number of methods can be used to measure mixing height in addition to radar profiler technology discussed above. Recent developments in ceilometer technology allow for the measurement of mixing height by changes in particulate concentrations at the top of the boundary layer (Eresmaa et al., 2006). Ceilometers provide the potential for continuous mixing height data at a fraction of the cost of radar profilers. Due to the importance of mixing height measurements for O₃ modeling, the EPA proposed to add the requirement for monitoring agencies to measure mixing height at required PAMS sites.

A number of commenters questioned the need for mixing height measurements at PAMS sites. One commenter stated, "the photochemical modeling community has a long history of relying upon National Weather Service measurements for mixing height." Another commenter stated that

"in some areas of the country the models used to predict mixing height are adequate, but in other mountainous or marine areas model-predicted mixing height data is inadequate." Accurate estimates of mixing height are important for appropriately characterizing concentrations of O₃ and O₃ precursors. Mixing height is also important for characterizing how modeled O₃ may change as a result of changing NO_x and VOC concentrations. For instance, if the modeled mixing height is too low causing unrealistically high concentration of NO_x, then O₃ destruction could be predicted when O₃ production may be happening in the atmosphere. When this or the opposite situation exists in modeling it may lead O₃ response to emissions changes that are less reliable for air quality planning purposes. While models are believed to do a reasonable job of predicting mixing height during the day, there is considerably more uncertainty in predicting this parameter during morning and evening transition periods and at night. Model O₃ predictions are particularly sensitive to mixing height during the time periods for which uncertainty in this parameter is greatest.

Several commenters noted that nearby National Oceanic and Atmospheric Administration (NOAA) Automated Surface Observing System (ASOS) sites may be a better alternative for collection of mixing height data. As indicated in the proposal, the EPA is aware of the network of ceilometers operated by NOAA as part of ASOS. The EPA has been in discussions with NOAA regarding the potential for these systems to provide the needed mixing height data. However, the ASOS ceilometers are not currently equipped to provide mixing height data and NOAA has no current plans to measure continuous mixing height in the future. Nonetheless, the EPA will continue to work with NOAA to determine if the ASOS ceilometers can be upgraded to meet the need for mixing height data, and included proposed regulatory language that will allow states a waiver to use nearby mixing height data from ASOS (or other sources) to meet the requirement to collect mixing height data at required PAMS sites when such data are suitable and available.

The EPA is finalizing the requirement for the measurement of mixing height at required PAMS sites due to the importance of mixing height in O₃ modeling. A waiver option, to be approved by the Regional Administrator, is also being included to allow mixing height measurements to be obtained from other nearby sites (e.g., NOAA ASOS sites).

6. PAMS Season

Currently, PAMS measurements are required to be taken during the months of June, July, and August. This 3-month period is referred to as the "PAMS Season." As part of the PAMS re-evaluation, the EPA considered changes to the PAMS season. The 3-month PAMS season was originally selected to represent the most active period for O₃ formation. However, the EPA notes that in many areas the highest O₃ concentrations are observed outside of the PAMS season. As an example, the highest O₃ concentrations in the mountain-west often occur during the winter months. Data collected during the current PAMS season would have limited value in understanding winter O₃ episodes.

The CASAC AMMS (U.S. EPA, 2011f) noted in their report to the EPA that "it would be desirable to extend the PAMS monitoring season beyond the current June, July, August sampling period." But that "the monitoring season should not be mandated and rigid; it should be flexible and adopted and coordinated on a regional airshed basis." The EPA agrees with CASAC on the need for flexibility in determining when PAMS measurements should be taken to meet local monitoring needs but also agrees with CASAC that the flexibility "should not conflict with national goals for the PAMS program." A significant benefit of the standard PAMS season is that it ensures data availability from all PAMS sites for national- or regional-scale modeling efforts.

While the EPA agrees with the potential benefit of extending the availability of PAMS measurements outside of the current season, we also considered the burden of requiring monitoring agencies to operate additional PAMS measurements (e.g., hourly speciated VOC) for periods that in some cases, might be much longer than the current 3-month season, for example, if the PAMS season was extended to match each state's required O₃ monitoring season. Being mindful of the potential burden associated with a lengthening of the PAMS season as well as the potential benefits of the additional data, the EPA proposed to maintain the current 3-month PAMS monitoring season for required PAMS sites rather than extending the PAMS season to other periods where elevated O₃ may be expected. No significant comments were received on the proposed PAMS season, and as such, for the reasons stated here and in the proposal, the EPA is not changing the 3-month PAMS season of June, July, and August.

The EPA believes that the 3-month PAMS season will provide a consistent data set of O₃ and O₃ precursor measurements for addressing the national PAMS objectives. Monitoring agencies are strongly encouraged to consider collecting PAMS measurements in additional periods beyond the required PAMS season as part of their EMP. The monitoring agencies should consider factors such as the periods of expected peak O₃ concentrations and regional consistency when determining potential expansion of their specific monitoring periods beyond the required PAMS season.

7. Timing and Other Implementation Issues

The EPA recognizes that the changes to the PAMS requirements will require resources and a reasonable timeline in order to be successfully implemented. The PAMS program is funded, in part, as part of the EPA's section 105 grants. The EPA believes that the current national funding level of the PAMS program is sufficient to support these final changes, but changes in the distribution of PAMS funds will need to be made. The network design changes will require some monitoring agencies to start collection of new PAMS measurements, while other monitoring agencies will see reductions in PAMS measurement requirements. The EPA will work with the NAACA, AAPCA, and other monitoring agencies to develop an appropriate PAMS grant distribution strategy.

In addition to resources, the affected monitoring agencies will need time to implement the revised PAMS requirements. For the required PAMS sites, monitoring agencies can determine now which NCore sites will be required to make PAMS measurements based on readily available census data. However, monitoring agencies will still need time to evaluate and seek approval for alternative sites or alternative VOC methods. In addition, monitoring agencies will need time to make capital investments (primarily for the installation of autoGCs, NO₂ monitors, and ceilometers), prepare appropriate QA documents, and develop the expertise needed to successfully collect PAMS measurements via training or otherwise. In order to ensure monitoring agencies have adequate time to plan and successfully implement the revised PAMS requirements, the EPA is requiring that monitoring agencies identify their plans to implement the PAMS measurements at NCore sites in their Annual Network Plan due July 1, 2018, and to begin making PAMS

measurements at NCore sites by June 1, 2019. The EPA believes some monitoring agencies may be able to begin making PAMS measurements sooner than June 2019 and encourages early deployment where possible.

Monitoring agencies will need to wait until O₃ designations are made to officially determine the applicability of the EMP requirement. The EPA proposed to allow two years after designations to develop EMPs, and that the EMPs would be submitted as part of their Annual Network Plan. Several commenters stated that due to the level of planning and coordination required for the EMPs, that the plans should instead be included as part of the 5-year network assessment. While the EPA agrees that the EMPs will require a substantial amount of planning and coordination, the next 5 year network assessment will not be due until July 1, 2020—nearly 5 years from the date of this final rulemaking. The EPA believes that it would be inappropriate to wait 5-years from the date of this rulemaking to develop plans for enhanced O₃ monitoring. In addition, the EPA believes that the first round of EMP development should receive additional focus and review that may not be afforded as part of the larger network assessment. Finally, most monitoring agencies will be aware of their likely O₃ attainment status well in advance of the official designations. In order to ensure timely development of the initial EMPs, the EPA is requiring affected monitoring agencies to submit their initial EMPs no later than two years following designations. States in the OTR do not need to wait until designations to determine EMP applicability and may not be classified as Moderate or above. As such, the final rule includes a requirement for states in the OTR to submit their initial EMPs by October 1, 2019 (which is consistent with the expected timeline for the remaining EMPs). However, subsequent review and revisions to the EMPs are to be made as part of the 5-year network assessments beginning with the assessments due in 2025.

D. Addition of a New FRM for O₃

The use of FRM analyzers for the collection of air monitoring data provides uniform, reproducible measurements of concentrations of criteria pollutants in ambient air. FRMs for various pollutants are described in several appendixes to 40 CFR part 50. For most gaseous criteria pollutants (including O₃ in Appendix D of part 50), the FRM is described as a particular measurement principle and calibration procedure to be implemented, with

further reference to specific analyzer performance requirements specified in 40 CFR part 53.

The EPA allows new or alternative monitoring technologies—identified as FEMs—to be used in lieu of FRMs, provided that such alternative methods produce measurements closely comparable to corresponding FRM measurements. Part 53 sets forth the specific performance requirements as well as the performance test procedures required by the EPA for determining and designating both FRM and FEM analyzers by brand and model.

To be used in a determination of compliance with the O₃ NAAQS, ambient O₃ monitoring data must be obtained using either a FRM or a FEM, as defined in parts 50 and 53. For O₃, nearly all the monitoring methods currently used by state and local monitoring agencies are FEM (not FRM) continuous analyzers that utilize an alternative measurement principle based on quantitative measurement of the absorption of UV light by O₃. This type of O₃ analyzer was introduced into monitoring networks in the 1980s and has since become the predominant type of method used because of its all-optoelectronic design and its ease of installation and operation.

The existing O₃ FRM specifies a measurement principle based on quantitative measurement of chemiluminescence from the reaction of ambient O₃ with ethylene (ET-CL). Ozone analyzers based on this FRM principle were once widely deployed in monitoring networks, but now they are no longer used for routine O₃ field monitoring because readily available UV-type FEMs are substantially less difficult to install and operate. In fact, the extent of the utilization of UV-type FEMs over FRMs for O₃ monitoring is such that FRM analyzers have now become commercially unavailable. The last new commercial FRM analyzer was designated by the EPA in 1979. The current list of all approved FRMs and FEMs capable of providing ambient O₃ data for use in NAAQS attainment decisions may be found on the EPA's Web site and in the docket for this action (U.S. EPA, 2014e). However, that list does not indicate whether or not each listed method is still commercially available.

1. Proposed Changes to the FRM for O₃

Although the existing O₃ FRM is still a technically sound methodology, the lack of commercially available FRM O₃ analyzers severely impeded the use of FRM analyzers, which are needed for quality control purposes and as the standard to which candidate FEMs are

required to be compared. Therefore, the EPA proposed to establish a new FRM measurement technique for O₃ based on NO-chemiluminescence (NO-CL) methodology. This new chemiluminescence technique is very similar to the existing ET-CL methodology with respect to operating principle, so the EPA proposed to incorporate it into the existing O₃ FRM as a variation of the existing ET-CL methodology, coupled with the same existing FRM calibration procedure.

A revised Appendix D to 40 CFR part 50 was proposed to include both the original ET-CL methodology as well as the new NO-CL methodology, such that use of either measurement technique would be acceptable for implementation in commercial FRM analyzers. Currently, two O₃ analyzer models (from the same manufacturer) employing the NO-CL methodology have been designated by the EPA as FEMs and would qualify for re-designation as FRMs under the revised O₃ FRM. The rationale for selecting the new NO-CL FRM methodology, including what other methodologies were also considered, and additional information to support its selection are discussed in the preamble to the proposal for this action (79 FR 75366-75368). No substantive change was proposed to the existing O₃ FRM calibration procedure, which would be applicable to both chemiluminescence FRM methodologies.

The proposed FRM in part 50, Appendix D also included numerous editorial changes to provide clarification of some provisions, some revised wording, additional details, and a more refined numbering system and format consistent with that of two other recently revised FRMs (for SO₂ and CO).

As noted in the proposal, there is substantial similarity between the new and previously existing FRM measurement techniques, and comparative field data show excellent agreement between ambient O₃ measurements made with the two techniques (U.S. EPA 2014f). Therefore, the EPA believes that there will be no significant impact on the comparability between existing ambient O₃ monitoring data based on the original ET-CL methodology and new monitoring data that may be based on the NO-CL methodology.

The proposed FRM retains the original ET-CL methodology, so all existing FEMs, which were designated under part 53 based on demonstrated comparability to that ET-CL methodology, will retain their FEM designations. Thus, there will be no negative consequences or disruption to

monitoring agencies, which will not be required to make any changes to their O₃ monitors due to the revised O₃ FRM. New FEMs would be designated under part 53, based on demonstrated acceptable comparability to either FRM methodology.

2. Comments on the FRM for O₃

Comments that were received from the public on the proposed new O₃ FRM technique are addressed in this section. Most commenters expressed general support for the proposed changes, although a few commenters expressed some concerns. The most significant issue discussed in comments was the relatively small but nevertheless potentially significant interference of water vapor observed in the ET-CL technique. As some comments pointed out, this interference is positive and could possibly affect NAAQS attainment decisions. The available NO-CL FEM analyzers include a sample dryer, which minimizes this interference. As noted previously, very few, if any, ET-CL FRM analyzers are still in operation. The ET-CL (with and without a sample dryer), the proposed NO-CL FRM, and all designated FEM analyzers have demonstrated compliance with the substantially reduced water vapor interference equivalent limit specified in 40 CFR part 53.

The proposed FRM mentioned the need for a sample air dryer for both ET-CL and NO-CL FRM analyzers. In response to these comments, the wording of the ET-CL FRM has been augmented to clarify the requirement for a dryer in all newly designated FRMs (the only change being made by the EPA to the existing ET-CL FRM as proposed). Also, the interference equivalent limit for water vapor in part 53 was proposed to be substantially reduced from the current 0.02 ppm to 0.002 ppm. The interference equivalent test for water vapor applicable to the new NO-CL candidate FRM analyzers (specified in Table B-3 of part 53) was proposed to be more stringent than the corresponding existing test for ET-CL FRM analyzers by requiring that water vapor be mixed with O₃. This mixing requirement was not part of the existing test for ET-CL candidate analyzers (denoted by footnote 3 in Table B-3). However, in further response to these commenters' concerns, the EPA has modified Table B-3 to extend this water vapor mixing requirement to newly designated ET-CL analyzers, as well. These measures should insure that potential water vapor interference is minimized in all newly designated FRM analyzers.

Several comments indicated concern that currently-designated FEM analyzers retain their designation without retesting if the new FRM were promulgated. The current ET-CL FRM is being retained; therefore, it is not necessary to make these new requirements retroactive to existing designated FEM analyzers. The existing FEM analyzers will not be required to be retested, and their FEM designation will be retained so that there will be no disruption to current monitoring networks.

Although beyond the scope of this rulemaking, other comments concerned potential hazards of the NO compressed gas supply required for NO-CL analyzer operation, and the current non-availability of a photolytic converter to provide an alternative source of NO from a less hazardous nitrous oxide gas supply. With regard to the photolytic converter, the EPA would approve such a converter as a source of NO if requested by an FRM analyzer manufacturer, upon demonstration of adequate functionality.

A few commenters liked the "scrubberless UV absorption" (SL-UV) measurement technique. The EPA has identified the SL-UV method as a potentially advantageous candidate for the O₃ FRM, but could not propose adopting it until additional test and performance information becomes available. A related comment requested clarification that promulgation of the proposed revised FRM would not preclude future consideration of other O₃ measurement techniques such as SL-UV. In response, the EPA can always consider new technologies for FRMs under 40 CFR 53.16 (Supersession of reference methods). However, a revised or amended FRM that included the SL-UV technique, as set forth in Appendix D of 40 CFR part 50, would have to be promulgated as part of a future rulemaking, before a SL-UV analyzer could be approved as an FRM under 40 CFR part 53.

One comment suggested that the value for the absorption cross section of O₃ at 254 nm used by the FRM's calibration procedure should be changed. The comment indicated that the nearly 2% difference effectively lowers the O₃ NAAQS by that amount. Using the corrected value would resolve much of the difference observed between O₃ measurements calibrated against the UV standard reference photometer versus those calibrated using NO gas phase titration and it would allow the EPA to adopt the less complex and more economical Gas Phase Titration (GPT) technique as the primary calibration standard for the

FRM. The EPA will await the results of further studies determining the value of the O₃ cross section at 254 nm before making a change to the calibration procedures and will not finalize changes to the calibration procedures in this final rule.

E. Revisions to the Analyzer Performance Requirements

1. Proposed Changes to the Analyzer Performance Requirements

In close association with the proposed O₃ FRM, the EPA also proposed changes to the associated analyzer performance requirements for designation of FRMs and FEMs for O₃, as set forth in 40 CFR part 53. These changes were largely confined to Table B-1, which specifies performance requirements for FRM and FEM analyzers for SO₂, CO, O₃, and NO₂, and to Table B-3, which specifies test concentrations for the various interfering agent (interferent) tests. Minor changes were also proposed for Figure B-5 and the general provisions in subpart A of part 53. All of these proposed changes are described and discussed more fully in the preamble to the proposal for this action (79 FR 75368-75369).

Modest changes proposed for Table B-3 would add new interferent test concentrations specifically for NO-CL O₃ analyzers, which include a test for NO₂ interference.

Several changes to Table B-1 were proposed. Updated performance requirements for “standard range” analyzers were proposed to be more consistent with current O₃ analyzer performance capabilities, including reduced limits for noise allowance, lower detectable limit (LDL), interference equivalent, zero drift, span drift, and lag, rise, and fall times. The previous limit on the total of all interferents was proposed to be withdrawn as unnecessary and to be consistent with that same change made previously for SO₂ and CO analyzers. Also, the span drift limit at 20% of the upper range limit (URL) was proposed to be withdrawn because it has similarly been shown to be unnecessary and to maintain consistency with that same change made previously for SO₂ and CO analyzers.

The form of the precision limits at both 20% and 80% of the URL was proposed to be changed from ppm to percent. The proposed new limits (in percent) were set to be equivalent to the previously existing limits (in ppm) and thus remain effectively unchanged. This change in form of the precision limits in Table B-1 has been previously made for SO₂ and CO analyzers, and was

proposed to extend also to analyzers for NO₂, (again with equivalent limits) for consistency and to simplify Table B-1 across all types of analyzers to which the table applies. A new footnote proposed for Table B-1 clarifies the new form for precision limits as “standard deviation expressed as percent of the URL.” Also proposed was a revision to Figure B-5 (Calculation of Zero Drift, Span Drift, and Precision) to reflect the changes proposed in the form of the precision limits and the withdrawal of the limits for total interference equivalent.

Concurrent with the proposed changes to the performance requirements for candidate O₃ analyzers, the EPA conducted a review of all designated FRM and FEM O₃ analyzers currently in production or being used, and verified that all meet the proposed new performance requirements. Therefore, none would require withdrawal or cancellation of their current FRM or FEM respective designations.

Finally, the EPA proposed new, optional, “lower range” performance limits for O₃ analyzers operating on measurement ranges lower (*i.e.*, more sensitive) than the standard range specified in Table B-1. The new performance requirements are listed in a new “lower range” column in Table B-1 and will provide for more stringent performance in applications where more sensitive O₃ measurements are needed.

Two minor changes were proposed to the general, administrative provisions in Subpart A of part 53. These include an increase in the time allowed for the EPA to process requests for approval of modifications to previously designated FRMs and FEMs in 53.14 and the withdrawal of a requirement for annual submission of Product Manufacturing Checklists associated with FRMs and FEMs for PM_{2.5} and PM_{10-2.5} in 53.9. No comments were received on these proposed changes and the EPA will be finalizing these revisions in this rulemaking.

2. Comments on the Analyzer Performance Requirements

Several comments were received related to the proposed changes to the analyzer performance requirements of part 53, and most were supportive. Comments from a few monitoring agencies suggested that the more stringent performance requirements proposed might be difficult to achieve or would increase monitor maintenance and cost. The EPA is also clarifying that these requirements apply only to the performance qualification requirements for designations of new FRM and FEM

analyzers and will have no impact on a monitoring agency’s operation of existing O₃ analyzers.

More specific comments from an analyzer manufacturer pointed out that the proposed lower limits for noise and LDL may be too stringent, the former because low-cost portable analyzers may have shorter absorption cells, and the latter because of limitations of current calibration technology. After further consideration of available analyzer performance data in light of these comments, the EPA agrees and is changing the noise limits from the proposed values of 1 ppb and 0.5 ppb (for the standard and lower ranges, respectively) to 2.5 ppb and 1 ppb (respectively). The EPA is also changing the LDL limit from the proposed values of 3 ppb and 1 ppb (respectively) to 5 ppb and 2 ppb (respectively). These new limits are still considerably more stringent than the previous limits (for the standard range) and are also consistent with those recommended by the commenter and the current performance capabilities of existing analyzer/calibration technology.

This commenter also pointed out that the proposed lower limit for 12-hour zero drift, together with the way the prescribed test is carried out, resulted in the test being dominated by analyzer noise rather than drift. The EPA agrees with this comment in general but believes that further study is needed before any specific changes can be proposed for the 12-hour zero drift test, particularly since any such changes would affect analyzers for other gaseous pollutants, as well.

Other comments suggested that there was no need for the proposed new, low-range performance requirements, because of cost and that available calibrators would be inadequate for calibration of such low ranges. The EPA disagrees with these comments and believes, as noted in the proposal preamble, that there is a definite need for low-level O₃ measurements in some applications and that suitable calibration for such low-level measurement ranges can be adequately carried out. As stated previously, the new “low range” specifications for O₃ analyzers are optional.

Several comments pointed out some typographical errors related to footnotes in Table B-3, as proposed; these errors have been corrected in the version of Table B-3 being finalized today.

EPA is finalizing the proposed amendments to both the O₃ FRM in Appendix D of part 50 and provisions in part 53, modified as described above, in response to the comments received.

VII. Grandfathering Provision for Certain PSD Permits

This section addresses the grandfathering provision for certain Prevention of Significant Deterioration (PSD) permit applications that is being finalized in this rule. Section VIII.C of this preamble contains a description of the PSD and Nonattainment New Source Review (NNSR) permitting programs and additional discussion of the implementation of those programs for the O₃ NAAQS.

A. Summary of the Proposed Grandfathering Provision

The EPA proposed to amend the PSD regulations to add a transition plan that would address the extent to which the revised O₃ NAAQS will apply to pending PSD permit applications. This transition plan is reflected in a grandfathering provision that applies to permit applications that meet certain milestones in the review process prior to either the signature date or effective date of the revised O₃ NAAQS. Absent such a grandfathering provision in the EPA's regulations, the EPA interprets section 165(a)(3)(B) of the CAA and the implementing PSD regulations at 40 CFR 52.21(k)(1) and 51.166(k)(1) to require that PSD permit applications include a demonstration that emissions from the proposed facility will not cause or contribute to a violation of any NAAQS that is in effect as of the date the PSD permit is issued. The proposal included a grandfathering provision that would enable eligible PSD applications to make the demonstration that the proposed project would not cause or contribute to a violation of any NAAQS with respect to the O₃ NAAQS in effect at the time the relevant permitting benchmark for grandfathering was reached, rather than the revised O₃ NAAQS. We proposed that the grandfathering provision would apply specifically to either of two categories of pending PSD permit applications: (1) Applications for which the reviewing authority has formally determined that the application is complete on or before the signature date of the final rule revising the O₃ NAAQS; and (2) applications for which the reviewing authority has first published a public notice of the draft permit or preliminary determination before the effective date of the revised NAAQS.

In the proposal, we also noted that for sources subject to the federal PSD program under 40 CFR 52.21, the EPA and air agencies that have been delegated authority to implement the federal PSD program for the EPA would apply the grandfathering provision to

any PSD application that satisfies either of the two criteria that make an application eligible for grandfathering. Accordingly, if a particular application does not qualify under the first criterion based on a complete application determination, it may qualify under the second criterion based on a public notice announcing the draft permit or preliminary determination. Conversely, a source may qualify for grandfathering under the first criterion, even if it does not satisfy the second.

The EPA also proposed revisions to the PSD regulations at 40 CFR 51.166 that would afford air agencies that issue PSD permits under a SIP-approved PSD permit program the discretion to adopt provisions into the SIP that allow for grandfathering of pending PSD permits under the same circumstances as set forth in the federal PSD regulations. With regard to implementing the grandfathering provision, we also explained that air agencies with EPA-approved PSD programs in their SIPs would have additional flexibility for implementing the proposed grandfathering provision to the extent that any alternative approach is at least as stringent as the federal provision. In addition, the proposal recognized that some air agencies do not make formal completeness determinations; thus, only the latter criterion based on the issuance of a public notice would be relevant in such cases and the state could elect to adopt only that criterion into its SIP. Accordingly, the EPA proposed to add a grandfathering provision to 40 CFR 51.166 containing the same two criteria as proposed for 40 CFR 52.21.

B. Comments and Responses

Many of the comments supported the concept of grandfathering. Some of these comments, mostly by state and local air agencies, supported the grandfathering provision as proposed. Many others recommended alternative approaches to grandfathering based on several different dates. Several comments recommended that air agencies be allowed to grandfather certain PSD permit applications and issue a PSD permit based on the 2008 O₃ NAAQS after the area is designated nonattainment for the revised O₃ NAAQS. An opposing set of comments, representing a coalition of eight environmental groups and one health advocacy group, strongly objected to the proposal for grandfathering, claiming that the EPA did not have any authority under the CAA to exempt or grandfather permit applicants from the statutory PSD permitting requirements. We are addressing some of these comments below and others in the Response to

Comment Document that is included in the docket for this rule.

Comments that recommended broadening the scope of the proposed grandfathering provision suggested a variety of approaches. Some air agency and industry comments recommended that the EPA adopt a grandfathering provision applicable only to those PSD applications for which the reviewing authority has determined the application to be complete on or before the signature date of the revised NAAQS. Other air agency and industry comments recommended that grandfathered status be determined only on the basis of whether the relevant permitting milestone has been achieved by the effective date of the revised NAAQS.

The EPA disagrees with these comments; the final rule uses separate dates for the two grandfathering milestones, as proposed. If the effective date of the revised NAAQS were used as the date for the complete application milestone, this could lead to pressure on state permitting authorities to prematurely issue completeness determinations in order to qualify for the grandfathering provision in the time period between signature of this final rule and the effective date. Using the signature date of the revised O₃ NAAQS as the date for the grandfathering milestone based on the completeness determination is thus intended to help preserve the integrity of the completeness determination process. Permit applications that have not yet been determined complete can be supplemented or revised to address the revised O₃ standards before the completeness determination is issued. Conversely, the amount and type of work required for a preliminary determination or a draft permit reduces the risk that such a document would be released prematurely merely to qualify for grandfathering. Similarly, because these documents are released for the purpose of providing an adequate opportunity for public participation in the permitting process, it would not behoove a reviewing authority to precipitately release such documents merely to satisfy the grandfathering milestone. Accordingly, the EPA does not have the same concerns about using the effective date of this final rule for the preliminary determination or draft permit milestone and further finds it reasonable to provide additional time for satisfying this milestone. Moreover, using the proposed milestones and corresponding dates is consistent with the milestones and corresponding dates that were used in the grandfathering provisions for the 2012 PM_{2.5} NAAQS.

Several other comments recommended that the grandfathering provision apply to all PSD applications for which a final PSD permit will be issued prior to the effective date of the area designations for the revised NAAQS. Some of these comments explained that without some transition provisions in the final rule, it may be impossible for a source to demonstrate attainment if the current ambient air monitoring data indicates a revised, lowered standard is not being met. The comments also suggested that the extended period for grandfathering a source from the revised NAAQS would provide states with additional time to establish offset banks or similar systems for new nonattainment areas.

Other comments recommended that air agencies be allowed to grandfather either all or certain PSD permit applications received before the effective date of the final nonattainment designations for the revised O₃ NAAQS. These comments supported allowing air agencies to issue PSD permits to grandfathered sources even after the area in which the source proposes to locate is designated nonattainment for the revised O₃ NAAQS. One comment saw this as being necessary because the development of the regulatory framework that will support the revised NAAQS, such as development of a credit market or even a transition into NNSR permitting, does not instantaneously accompany the revised standard. Hence, the comment added that “[d]uring the Interim Period (the time between the revision of the NAAQS rule and development of the regulatory framework) the project may be unable to secure offsets and no offsets would be available for purchase.” Another comment explained that the extended period for grandfathering sources from the revised O₃ NAAQS was needed to “minimize disruption to complex projects that may have been under development since before the EPA published the proposed NAAQS revision.” This comment noted the “PSD projects commonly undergo years of engineering and other development resources before an air permit application can be prepared.”

The EPA does not agree with the comments recommending that the EPA use a date after the effective date of the revised O₃ NAAQS as the date by which the permit application must reach the relevant milestone to qualify for grandfathering. The EPA does not believe it is appropriate to unreasonably or unnecessarily delay implementation of these revised standards under the PSD program. As explained in more detail below, the purpose of the

grandfathering provision is to provide a reasonable transition mechanism for certain PSD applications and the EPA believes that the milestones proposed and finalized here strike the appropriate balance in providing for such a reasonable transition. Moreover, in some cases, some of these recommended approaches could enable a situation where a PSD permit would be issued to a source during a future period when the area is designated nonattainment for the revised O₃ NAAQS. As explained below, the EPA does not believe that this specific outcome is permissible under the CAA.

The EPA does not agree with the comments suggesting that the grandfathering provision should be expanded to apply to any PSD application received before the effective date of the final nonattainment designations for the revised O₃ NAAQS. Because the process for reviewing PSD permit applications and issuing a final PSD permit is time consuming, such an approach could allow issuance of PSD permits to grandfathered sources even after the area in which the source proposes to locate is designated nonattainment for the revised O₃ NAAQS. The EPA does not agree that grandfathering should be extended in a way that would allow a source located in an area designated as nonattainment for a pollutant at the time of permit issuance to obtain a PSD permit for that pollutant rather than a NNSR permit. The EPA does not interpret the CAA or its implementing regulations to allow such an outcome. The PSD requirements under CAA section 165 only apply in areas designated attainment or unclassifiable for the pollutant. *Alabama Power v. Costle*, 636 F.2d 323, 365–66, 368 (D.C. Cir. 1980). Accordingly, the PSD implementing regulations at 40 CFR 52.21(i)(2) contain an exemption that provides that the substantive PSD requirements shall not apply to a pollutant if the owner or operator demonstrates that the facility is located in an area designated nonattainment for that pollutant under CAA section 107 of the Act. *See also* 40 CFR 51.166(i)(2) (allowing for the same exemption in SIP-approved PSD permitting programs). In addition, under CAA section 172(c)(5) implementation plans must require that permits issued to new or modified stationary sources “anywhere in the nonattainment area” meet the requirements of CAA section 173, which contains the NNSR permit requirements. *See* 40 CFR part 51, Appendix S, IV.A (providing that, if a major new source or major modification that would locate in an area designated

as nonattainment for a pollutant for which the source or modification would be major, approval to construct may be granted only if the specific conditions for NNSR are met, including obtaining emission offsets and an emission limitation that specifies the lowest achievable emissions rate). Moreover, given the adverse air quality conditions that already exist in a nonattainment area and the congressional directive to reach attainment as expeditiously as practicable, construction of a major stationary source that significantly increases emissions in such an area should be expected to address all of the NNSR requirements, which are designed to ensure that a new or modified major stationary source will not interfere with reasonable progress toward attainment, even if this could cause delay to the permit applicant.

With respect to the comments that suggested the effective date of the NAAQS should be used as the date for both milestones, the EPA does not agree that such a change is necessary. The purpose of the grandfathering provision is to provide a reasonable transition mechanism in the following circumstances: first, the PSD application is one for which both the applicant and the reviewing authority have committed substantial resources; and, second, this situation is one where the need to satisfy the demonstration requirement under CAA section 165(a)(3) could impact the reviewing authority’s ability to meet the statutory deadline for issuing a permit within one year of the completeness determination. In situations where the reviewing authority has not yet issued a completeness determination as of the signature date of the revised O₃ NAAQS, both the permit applicant and the reviewing authority have sufficient notice of the revised standard so that it can be addressed before the completeness determination is issued and the one-year clock begins to run. The grandfathering provision issued in this rulemaking is crafted to draw a reasonable balance that accommodates the requirements under both CAA sections 165(a)(3) and 165(c). Any modification of the dates further than is necessary to accommodate these concerns could upset this balance.

With respect to the comments that suggested adopting a grandfathering provision applicable only to those PSD applications for which the reviewing authority has determined the application to be complete on or before the signature date of the revised NAAQS, the EPA is not making this change because we understand that not all reviewing authorities issue formal completeness determinations. Including

a grandfathering provision based on the publication of a public notice of the draft permit or preliminary determination provides a reasonable transition mechanism for PSD applications in situations where the reviewing authority does not issue formal completeness determinations, but the applicant and the reviewing authority have both committed substantial resources to the pending permit application at the time the revisions to the O₃ NAAQS are finalized.

An opposing set of comments—submitted by a consortium of eight environmental groups and one health advocacy group—challenged the proposed grandfathering provision on the basis that the EPA did not have the legal authority to grandfather sources from PSD requirements. These commenters argued that the plain language of CAA section 165 forecloses the EPA's proposed approach and raised several other legal considerations. The EPA disagrees with these comments, including the interpretations of the CAA that they offer. As summarized in the rationale for the final action below in section VII.C of this preamble, the EPA believes that the CAA provides it authority and discretion to establish a PSD grandfathering provision such as the one being adopted today through a rulemaking process. The EPA is providing a further, detailed analysis fully responding to this set of comments, as well as other comments related to the grandfathering provision, in the Response to Comment Document in the docket for this rule.

C. Final Action and Rationale

After consideration and evaluation of all the public comments received on the grandfathering provision, the EPA is finalizing this provision as proposed, with minor revisions that enhance the clarity of the grandfathering provision, without changing its substantive effect. While these revisions lead to slight differences in wording for the grandfathering provision for the 2012 PM_{2.5} NAAQS and the grandfathering provision finalized in this rulemaking, those differences are not intended to create a different meaning; rather, the grandfathering provision finalized in this rulemaking is intended to have the same substantive effect and meaning for the revised O₃ standards as the grandfathering provision for the 2012 PM_{2.5} NAAQS had for the revised PM standards. Other than those clarifying revisions, this final rule includes the same rule language for the grandfathering provision as previously proposed for the PSD regulations at 40

CFR 52.21(i)(12) and 51.166(i)(11), respectively. The provision in the final rule reflects the same two milestones and corresponding dates as the proposed grandfathering provision. Thus, under the grandfathering provision as finalized, either of the following two categories of pending PSD permit applications would be eligible for grandfathering: (1) Applications for which the reviewing authority has formally determined that the application is complete on or before the signature date of the revised O₃ NAAQS, or (2) applications for which the reviewing authority has first published a notice of a draft permit or preliminary determination before the effective date of the revised O₃ NAAQS. The EPA believes that it continues to be appropriate to include the two proposed milestones for pending permit applications to be eligible for grandfathering. While a completeness determination is often the first event, some air agencies do not determine applications complete as part of their permit process.

Under 40 CFR 52.21, a permit application may qualify for grandfathering under either of the two sets of milestones and dates contained in the provision. Where the EPA is the reviewing authority, the EPA intends to apply the grandfathering provision to PSD applicants pursuant to PSD regulations at 40 CFR 52.21 primarily through the use of the completeness determination milestone because the EPA Regional Offices make a formal completeness determination for any PSD application that they receive and review. The EPA is including the second criterion in 40 CFR 52.21 so that pending applications can still qualify for grandfathering under the second criterion if any air agency that incorporates 40 CFR 52.21 into a SIP-approved program does not make formal completeness determinations as part of its permit review process.

The EPA is also amending the PSD regulations at 40 CFR 51.166 to enable states and other air agencies that issue PSD permits under SIP-approved PSD programs to adopt a comparable grandfathering provision. Nevertheless, such air agencies have discretion to not grandfather PSD applications or to apply grandfathering under their approved PSD programs in another manner as long as that program is at least as stringent as the provision being added to 40 CFR 51.166. Accordingly, an air agency may elect to rely on both sets of milestones and dates or it may grandfather on the sole basis of only one set. However, the EPA anticipates that once a decision is made concerning the

use of either set of milestones and dates, the air agency will apply grandfathering consistently to all pending PSD permit applications.

As explained in more detail in the proposal, absent a regulatory grandfathering provision, the EPA interprets section 165(a)(3)(B) of the CAA and the implementing PSD regulations at 40 CFR 52.21(k)(1) and 51.166(k)(1) to require that PSD permit applications include a demonstration that emissions from the proposed facility will not cause or contribute to a violation of any NAAQS that is in effect as of the date the PSD permit is issued. However, reading CAA section 165(a)(3)(B) in context with other provisions of the Act and the legislative history, the EPA interprets the Act to provide the EPA with authority to establish grandfathering provisions through regulation. The EPA has explained its interpretation of its authority to promulgate grandfathering provisions in previous rulemaking actions, most recently in the rule establishing the grandfathering provision for the 2012 PM_{2.5} NAAQS (78 FR 3086, 3254–56, January 15, 2013), as well as in the proposal for this final action. The EPA is providing additional discussion of this authority in the Response to Comment Document contained in the docket for this final action.

To summarize briefly, the addition of this grandfathering provision is permissible under the discretion provided by the CAA for the EPA to craft a reasonable implementation regulation that balances competing objectives of the statutory PSD program found in CAA section 165. Specifically, section 165(a)(3) requires a permit applicant to demonstrate that its proposed project will not cause or contribute to a violation of any NAAQS, while section 165(c) requires that a PSD permit be granted or denied within one year after the permitting authority determines the application for such permit to be complete. Section 109(d)(1) of the CAA requires the EPA to review existing NAAQS and make appropriate revisions every five years. When these provisions are considered together, a statutory ambiguity arises concerning how the requirements under CAA section 165(a)(3)(B) should be applied to a limited set of pending PSD permit applications when the O₃ NAAQS is revised. The Act does not clearly address how the requirements of CAA section 165(a)(3)(B) should be met for PSD permit applications that are pending when the NAAQS are revised, particularly when the EPA also determines that complying with the

demonstration requirement for the revised NAAQS could hinder compliance with the requirement under section 165(c) to issue a permit within one year of the completeness determination for a certain subset of pending permits. The CAA also does not address how the requirements of CAA sections 165(a)(3) and 165(c) should be balanced in light of the statutory requirement to review the NAAQS every five years. As Congress has not spoken precisely to this issue, the EPA has the discretion to apply a permissible interpretation of the Act that balances the statutory requirements to make a decision on a permit application within one year and to ensure the new and modified sources will only be authorized to construct after showing they can meet the substantive permitting criteria. *See Chevron, U.S.A., Inc. v. Natural Res. Def. Council, Inc.*, 467 U.S. 837, 843–44 (1984).

In addressing these gaps in the CAA and the tension that may arise in section 165 in these circumstances, the EPA also applies CAA section 301, where the Administrator is authorized “to prescribe such regulations as are necessary to carry out his functions under this chapter.” Sections 165(a)(3) and 165(c) of the CAA make clear that the interests behind CAA section 165 include both protection of air quality and timely decision-making on pending permit applications. The legislative history illustrates congressional intent to avoid delays in permit processing. S. Rep. No. 94–717, at 26 (1976) (“nothing could be more detrimental to the intent of this section and the integrity of this Act than to have the process encumbered by bureaucratic delay”). Thus, when read in combination, these provisions of the CAA provide the EPA with the discretion to issue regulations to grandfather pending permit applications from having to address a revised NAAQS where necessary to achieve both CAA objectives—to protect the NAAQS and to avoid delays in processing PSD permit applications. Accordingly, the EPA is seeking in this action to balance the requirements in the CAA to make a decision on a permit application within one year and to ensure that new and modified sources will only be authorized to construct after showing they can meet the substantive permitting criteria that apply to them. The EPA is achieving this balance by determining through rulemaking which O₃ NAAQS apply to certain permit applications that are pending when the EPA finalizes the revisions to the O₃ NAAQS in this final rule. We are clarifying, for the limited

purpose of satisfying the requirements under section 165(a)(3)(B) for those permits, which O₃ NAAQS are applicable to those permit applications and must be addressed in the source’s demonstration that its emissions do not cause or contribute to a violation of the NAAQS.

This approach is consistent with a recent opinion by the U.S. Court of Appeals for the Ninth Circuit, which recognized the EPA’s traditional exercise of grandfathering authority through rulemaking. The court observed that this approach was consistent with the statutory requirement to “enforce whatever regulations are in effect at the time the agency makes a final decision” because it involved identifying “an operative date, incident to setting the new substantive standard, and the grandfathering of pending permit applications was explicitly built into the new regulations.” *Sierra Club v. EPA*, 762 F.3d 971, 983 (9th Cir. 2014). As discussed in more detail in the EPA’s Response to Comment Document contained in the docket for this rule, this case supports the EPA’s action in this rulemaking. The court favorably discussed prior adoption of regulatory grandfathering provisions that are similar to the action in this rulemaking, such as the grandfathering provision that the EPA promulgated when revising the PM_{2.5} NAAQS that became effective in 2013. *See id.* at 982–83.²²⁷

This adoption of a grandfathering provision in this action is also consistent with previous actions in which the EPA has recognized that the CAA provides discretion for the EPA to establish grandfathering provisions for PSD permit applications through regulations. Some examples of previous

²²⁷ This case specifically involved an action by the EPA to issue an individual PSD permit, which grandfathered a specific permit applicant from certain requirements without any revision to the regulations that were in effect. The court’s reasoning in this case distinguishes that type of permit-specific grandfathering from establishing grandfathering provisions through a rulemaking process. While the court was not persuaded that there was a conflict between the requirements of sections 165(a)(3) and 165(c) of the CAA that supported the permit-specific grandfathering at issue in that case, it did not extend that uncertainty to its discussion of the EPA’s rulemaking authority. In fact, in its favorable discussion of the EPA’s authority to grandfather pending permit applications through regulation, the court noted that the power of an administrative agency “to administer a congressionally created and funded program necessarily requires the formulation of policy and the making of rules to fill any gap left, implicitly or explicitly, by Congress” though “such decision cannot be made on an ad hoc basis.” *Sierra Club v. EPA*, 762 F.3d 971, 983 (9th Cir. 2014) (internal quotations and marks omitted). This indicates that the court believed there is a gap in the CAA that supports including grandfathering provisions in regulations.

references to the EPA’s authority to grandfather certain applications through rulemaking include 45 FR 52683, August 7, 1980; 52 FR 24672, July 1, 1987; and most recently 78 FR 3086, January 15, 2013.

This grandfathering provision does not apply to any applicable PSD requirements related to O₃ other than the requirement to demonstrate that the proposed source does not cause or contribute to a violation of the revised O₃ NAAQS. Sources with projects qualifying under the grandfathering provision will be required to meet all the other applicable PSD requirements, including applying BACT to all applicable pollutants, demonstrating that emissions from the proposed facility will not cause or contribute to a violation of the O₃ NAAQS in effect at the time of the relevant grandfathering milestone, and addressing any Class I area and additional O₃-related impacts in accordance with the applicable PSD requirements. In addition, this grandfathering provision would not apply to any permit application for a new or modified major stationary source of O₃ located in an area designated nonattainment for O₃ on the date the permit is issued.

VIII. Implementation of the Revised O₃ Standards

This section provides background information for understanding the implications of the revised O₃ NAAQS and describes the EPA’s plans for providing revised rules or additional guidance on some subjects in a timely manner to assist states with their implementation efforts under the requirements of the CAA. This section also describes existing EPA rules, interpretations of CAA requirements, and other EPA guidance relevant to implementation of the revised O₃ NAAQS. Relevant CAA provisions that provide potential flexibility with regard to meeting implementation timelines are highlighted and discussed. This section also contains a discussion of how existing requirements to reduce the impact on O₃ concentrations from the stationary source construction in permit programs under the CAA are affected by the revisions to the O₃ NAAQS. These are the PSD and Nonattainment New Source Review (NNSR) programs. As discussed in section VII of this preamble, to facilitate a smooth transition to the PSD requirements for the revised O₃ NAAQS, the EPA is finalizing as part of this rulemaking a grandfathering provision that applies to certain PSD permit applications that are pending and have met certain milestones in the permitting process

when the revised O₃ NAAQS is signed or before the effective date of the revised O₃ NAAQS, depending on the milestone.

In the preamble for the O₃ NAAQS proposal, the EPA solicited comments on several issues related to implementing the revised O₃ NAAQS that the agency anticipated addressing in future guidance or regulatory actions, but for which the EPA was not at that time proposing any action. The EPA received numerous comments on those and other implementation issues. Consistent with what the EPA indicated in the O₃ NAAQS proposal (79 FR 75370), the agency is not responding to the implementation comments that are not related to a specific proposal. However, the EPA intends to take these comments under advisement as the agency develops rules and guidance to assist with implementation of the revised NAAQS. Because the EPA did specifically propose and is finalizing provisions in the regulations addressing grandfathering for certain PSD permit applications and requirements, as discussed in section VII of this preamble, the EPA is responding to comments on the proposed PSD grandfathering provisions.

A. NAAQS Implementation Plans

1. Cooperative Federalism

As directed by the CAA, reducing pollution to meet national air quality standards always has been a shared task, one involving the federal government, states, tribes and local air quality management agencies. The EPA develops regulations and strategies to reduce pollution on a broad scale, while states and tribes are responsible for implementation planning and any additional emission reduction measures necessary to bring specific areas into attainment. The agency supports implementation planning with technical resources, guidance, and program rules where necessary, while air quality management agencies use their knowledge of local needs and opportunities in designing emission reduction strategies that will work best for their industries and communities.

This partnership has proved effective since the EPA first issued O₃ standards more than three decades ago. For example, 101 areas were designated as nonattainment for the 1-hour O₃ standards issued in 1979. As of the end of 2014, air quality in all but one of those areas meets the 1-hour standards. The EPA strengthened the O₃ standards in 1997, shifting to an 8-hour standard to improve public health protection, particularly for children, the elderly,

and other sensitive individuals. The 1997 standards drew significant public attention when they were proposed, with numerous parties voicing concerns about states' ability to comply. However, after close collaboration between the EPA, states, tribes and local governments to reduce O₃-forming pollutants, significant progress has been made. Air quality in 108 of the original 115 areas designated as nonattainment for the 1997 O₃ NAAQS now meets those standards. Air quality in 18 of the original 46 areas designated as nonattainment for the 2008 O₃ NAAQS now meets those standards.

The revisions to the primary and secondary O₃ NAAQS discussed in sections II.D and IV.D of this preamble trigger a process under which states²²⁸ make recommendations to the Administrator regarding area designations. Then, the EPA promulgates the final area designations. States also are required to review capacity and authorities in their existing SIPs to ensure the CAA requirements associated with the new standards can be carried out, and modify or supplement their existing SIPs as needed. The O₃ NAAQS revisions also apply to the transportation conformity and general conformity determinations, and affect which preconstruction permitting requirements apply to sources of O₃ precursor emissions, and the nature of those requirements.

The EPA has regulations in place addressing the general requirements for SIPs, and there are also provisions in these existing rules that cover O₃ SIPs (40 CFR part 51). States likewise have provisions in their existing SIPs to address air quality for O₃ and to implement the existing O₃ NAAQS. In the course of the past 45 years of regulating criteria pollutants, including O₃, the EPA has also provided general guidance on the development of SIPs and administration of construction permitting programs, as well as specific guidance on implementing the O₃ NAAQS in some contexts under the CAA and the EPA regulations.

The EPA has considered the extent to which existing EPA regulations and guidance are sufficient to implement the revised standards. The CAA does not require that the EPA promulgate new implementing regulations or issue new guidance for states every time that a NAAQS is revised. Likewise, the CAA does not require the issuance of additional implementing regulations or

²²⁸This and all subsequent references to "state" are meant to include state, local, and tribal agencies responsible for the implementation of an O₃ control program.

guidance by the EPA before a revised NAAQS becomes effective. It is important to note that the existing EPA regulations in 40 CFR part 51 applicable to SIPs generally and to particular pollutants, including O₃ and O₃ precursors, continue to apply unless and until they are updated. Accordingly, the discussion below provides the EPA's current thoughts about the extent to which revisions to existing regulations and additional guidance are appropriate to aid in the implementation of the revised O₃ NAAQS.

2. Additional New Rules and Guidance

The EPA has received comments from a variety of states and organizations asking for rules and guidance associated with a revised NAAQS to be issued in a timely manner. As explained above, and consistent with the proposal, the EPA is not responding to these comments at this time because they are not related to any changes to existing regulations that EPA proposed in this rule. Moreover, although issuance of such rules and guidance is not a part of the NAAQS review process, *National Ass'n of Manufacturers v. EPA*, 750 F.3d 921, 926–27 (D.C. Cir. 2014), toward that end, the EPA intends to develop appropriate revisions to necessary implementation rules and provide additional guidance in time frames that are useful to states when developing implementation plans that meet CAA requirements.

Certain requirements under the PSD preconstruction permit review program apply immediately to a revised NAAQS upon the effective date of that NAAQS, unless the EPA has established a grandfathering provision through rulemaking. To ensure a smooth transition to a revised O₃ NAAQS, the EPA is finalizing a grandfathering provision similar to the provision finalized in the 2012 PM_{2.5} NAAQS Rule. See section VII.C of this preamble for more details on the PSD program and the final grandfathering provision.

Promulgation or revision of the NAAQS starts a clock for the EPA to designate areas as either attainment or nonattainment. State recommendations for area designations are due to the EPA within 12 months of promulgating or revising the NAAQS. In an effort to allow states to make more informed recommendations for these particular standards, the EPA intends to issue additional guidance concerning the designations process for these standards within four months of promulgation of the NAAQS, or approximately eight months before state recommendations are due. The EPA generally completes

area designations two years after promulgation of a NAAQS. See section VIII.B of this preamble for additional information on the initial area designation process.

Under CAA section 110, a NAAQS revision triggers the review and, as necessary, revision of SIPs to be submitted within three years of promulgation of a revised NAAQS. These SIPs are referred to as “infrastructure SIPs.” The EPA issued general guidance on submitting infrastructure SIPs on September 13, 2013.²²⁹ It should be noted that this guidance did not address certain state planning and emissions control requirements related to interstate pollution transport. This guidance remains relevant for the revised O₃ NAAQS. See section VIII.A.4 of this preamble for additional information on infrastructure SIPs.

While much of the existing rules and guidance for prior ozone standards remains applicable to the new standards, the EPA intends to propose to adopt revised rules on some subjects to facilitate air agencies’ efforts to implement the revised O₃ NAAQS within one year after the revised NAAQS is established. The rules would address nonattainment area classification methodologies and attainment dates, attainment plan and NNSR SIP submission due dates, and any other necessary revisions to existing regulations for other required implementation programs. The EPA anticipates finalizing these rules by the time areas are designated nonattainment. Finalizing rules and guidance on these subjects by this time would assist air quality management agencies with development of any CAA-required SIPs associated with nonattainment areas. See section VIII.A.5 of this preamble for additional information on nonattainment SIPs and section VIII.C.3 for additional information on nonattainment New Source Review requirements applicable to new major sources and major modifications of existing sources.

3. Background O₃

The EPA and state, local and tribal air agencies, strive to determine how to most effectively and efficiently use the CAA’s various provisions to provide required public health and welfare

²²⁹ See memorandum from Stephen D. Page to Regional Air Directors, “Guidance on Infrastructure State Implementation Plan (SIP) Elements under Clean Air Act Sections 110(a)(1) and 110(a)(2)” September 13, 2013, which is available at http://www3.epa.gov/airquality/urbanair/sipstatus/docs/Guidance_on_Infrastructure_SIP_Elements_Multipollutant_FINAL_Sept_2013.pdf.

protection from the harmful effects of O₃. In most cases, reducing man-made emissions of NO_x and VOCs within the U.S. will reduce O₃ formation and provide additional health and welfare protection. The EPA recognizes, however, that there can be infrequent events where daily maximum 8-hour O₃ concentrations approach or exceed 70 ppb largely due to the influence of wildfires or stratospheric intrusions, which contribute to U.S. background (USB) levels but may also qualify for consideration under the Exceptional Events Rule. See section I.D; but see section II.A.2.a above (percentage of anthropogenic O₃ tends to increase on high O₃ days relative to percentage of background, including in intermountain west).

The term “background” O₃ is often used to refer to O₃ that originates from natural sources of O₃ (e.g., wildfires and stratospheric O₃ intrusions) and O₃ precursors, as well as from man-made international emissions of O₃ precursors. Using the term generically, however, can lead to confusion as to what sources of O₃ are being considered. Relevant to the O₃ implementation provisions of the CAA, we define background O₃ the same way the EPA defines USB: O₃ that would exist in the absence of any man-made emissions inside the U.S.

While the great majority of modeled O₃ exceedances have local and regional emissions as their primary cause, there can be events where O₃ levels approach or exceed the concentration level of the revised O₃ standards in large part due to background sources. These cases of high USB levels on high O₃ days typically result from stratospheric intrusions of O₃ or wildfire O₃ plumes. These events are infrequent and the CAA contains provisions that can be used to help deal, in particular, with stratospheric intrusion and wildfire events with O₃ contributions of this magnitude, including providing varying degrees of regulatory relief for air agencies and potential regulated entities. The EPA intends to work closely with states to identify affected locations and ensure that the appropriate regulatory mechanisms are employed.

Statutory and regulatory relief associated with U.S. background O₃ may include:²³⁰

²³⁰ Note that the relief mechanisms discussed here do not include the CAA’s interstate transport provisions found in sections 110(a)(2)(D) and 126. The interstate transport provisions are intended to address the cross-state transport of O₃ and O₃ precursor emissions from man-made sources within the continental U.S. rather than background O₃ as it is defined in this section. As noted in section II.A.2.a above, many of the instances where

- Relief from designation as a nonattainment area through exclusion of data affected by exceptional events;
- Relief from the more stringent requirements of higher nonattainment area classifications through treatment as a rural transport area, through exclusion of data affected by exceptional events, or through international transport provisions;
- Relief from having to demonstrate attainment and having to adopt more than reasonable controls on local sources through international transport provisions.

Further discussion of these mechanisms is provided in sections VIII.B.2 (exceptional events), VIII.B.1 (rural transport areas), and VIII.E.2 (international transport).

Although these relief mechanisms require some level of assessment or demonstration by a state and/or the EPA to invoke, they have been used successfully in the past under appropriate circumstances. For example, the EPA has historically acted on every exceptional events demonstration that has affected a regulatory decision regarding initial area designations. See e.g., *Idaho: West Silver Valley Nonattainment Area—Area Designations for the 2012 primary annual PM_{2.5} NAAQS Technical Support Document*, pp. 10–14, December 2014. For the revised O₃ standards, the areas that would most likely need to use the mechanisms discussed in this section as part of attaining the revised O₃ standards are locations in the western U.S. where we have estimated the largest seasonal average values of background O₃ occur. We expect some of these areas to use the provisions in the Exceptional Events Rule during the designations process for the revised O₃ standards. The EPA will then give priority to exceptional events demonstrations submitted by air agencies with areas whose designation decision could be influenced by the exclusion of data under the Exceptional Events Rule. In addition, as discussed in more detail in sections V.D and VIII.B.2 of this action, to streamline the exceptional events process, the EPA will soon propose revisions to the 2007 Exceptional Events Rule and will release through a **Federal Register Notice of Availability** a draft guidance document to address Exceptional Events Rule criteria for wildfires that could affect O₃ concentrations. We expect to

commenters pointed to remote monitored locations having O₃ exceedances due to background O₃ in fact reflected sizeable contributions from domestic sources, including interstate contributions (including from the Los Angeles Basin and other California locations).

promulgate Exceptional Events Rule revisions and finalize the new guidance document before the October 2016 date by which states, and any tribes that wish to do so, are required to submit their initial designation recommendations for the revised O₃ NAAQS.

4. Section 110 State Implementation Plans

The CAA section 110 specifies the general requirements for SIPs. Within three years after the promulgation of revised NAAQS (or such shorter period as the Administrator may prescribe²³¹) each state must adopt and submit “infrastructure” SIPs to the EPA to address the requirements of section 110(a)(1) and (2), as applicable. These “infrastructure SIP” submissions establish the basic state programs to implement, maintain, and enforce revised NAAQS and provide assurances of state resources and authorities. States are required to develop and maintain an air quality management infrastructure that includes enforceable emission limitations, a permitting program, an ambient monitoring program, an enforcement program, air quality modeling capabilities, and adequate personnel, resources, and legal authority. Because the revised primary NAAQS and secondary NAAQS are identical, the EPA does not at present discern any need for there to be any significant substantive difference in the infrastructure SIP elements for the two standards and thus believes it would be more efficient for states and the EPA if each affected state submits a single section 110 infrastructure SIP that addresses both standards at the same time (*i.e.*, within three years of promulgation of the O₃ NAAQS). Accordingly the EPA is not extending the SIP deadline for purposes of a revised secondary standard.

It is the responsibility of each state to review its air quality management program’s compliance with the infrastructure SIP provisions in light of each new or revised NAAQS. Most states have revised and updated their infrastructure SIPs in recent years to address requirements associated with the 2008 O₃ NAAQS. We expect that the result of these prior updates is that, in most cases, states will already have adequate state regulations previously adopted and approved into the SIP to address a particular requirement with respect to the revised O₃ NAAQS. For

²³¹ While the CAA allows the EPA to set a shorter time for submission of these SIPs, the EPA does not currently intend to do so for this revision to the O₃ NAAQS.

such portions of the state’s infrastructure SIP submission, the state may provide a “certification” specifying that certain existing provisions in the SIP are adequate to meet applicable requirements. Although the term “certification” does not appear in the CAA as a type of infrastructure SIP submittal, the EPA sometimes uses the term in the context of infrastructure SIPs, by policy and convention, to refer to a state’s SIP submission. If a state determines that its existing EPA-approved SIP provisions are adequate in light of the revised O₃ NAAQS with respect to a given infrastructure SIP element (or sub-element), then the state may make a “certification” that the existing SIP contains provisions that address those requirements of the specific CAA section 110(a)(2) infrastructure elements. In the case of a certification, the submittal does not have to include another copy of the relevant provision (*e.g.*, rule or statute) itself. Rather, the submission may provide citations to the already SIP-approved state statutes, regulations, or non-regulatory measures, as appropriate, which meet the relevant CAA requirement. Like any other SIP submission, such certification can be made only after the state has provided reasonable notice and opportunity for public hearing. This “reasonable notice and opportunity for public hearing” requirement for infrastructure SIP submittals appears at section 110(a), and it comports with the more general SIP requirement at section 110(l) of the CAA. Under the EPA’s regulations at 40 CFR part 51, if a public hearing is held, an infrastructure SIP submission must include documentation by the state that the public hearing was held in accordance with the EPA’s procedural requirements for public hearings. See 40 CFR part 51, Appendix V, paragraph 2.1(g), and 40 CFR 51.102. In the event that a state’s existing SIP does not already meet applicable requirements, then the infrastructure SIP submission must include the modifications or additions to the state’s SIP in order to update it to meet the relevant elements of section 110(a)(2).

5. Nonattainment Area Requirements

Part D of the CAA describes the various program requirements that apply to states with nonattainment areas for different NAAQS. Clean Air Act Section 182 (found in subpart 2 of part D) includes the specific SIP requirements that govern the O₃ program, and supplements the more general nonattainment area requirements in CAA sections 172 and 173. Under CAA section 182, states

generally are required to submit attainment demonstration SIPs within three or four years after the effective date of area designations promulgated by the EPA, depending on the classification of the area.²³² These SIP submissions need to show how the nonattainment area will attain the primary O₃ standard “as expeditiously as practicable,” but no later than within the relevant time frame from the effective date of designations associated with the classification of the area.

The EPA believes that the overall framework and policy approach of the implementation rules associated with the 2008 O₃ NAAQS provide an effective and appropriate template for the general approach states would follow in planning for attainment of the revised O₃ standard.²³³ However, to assist with the implementation of the revised O₃ standards, the EPA intends to develop and propose an additional O₃ NAAQS Implementation Rule that will address certain subjects specific to the new O₃ NAAQS finalized here. This will include establishing air quality thresholds associated with each nonattainment area classification (*i.e.*, Marginal, Moderate, etc.), associated attainment deadlines, and deadlines for submitting attainment planning SIP elements (*e.g.*, RACT for major sources, RACT VOC control techniques guidelines, etc.). The rulemaking will also address whether to revoke the 2008 O₃ NAAQS, and to impose appropriate anti-backsliding requirements to ensure that the protections afforded by that standard are preserved. The EPA intends to propose this implementation rule within one year after the revised O₃ NAAQS is promulgated, and finalize this implementation rule by no later than the time the area designations process is finalized (approximately two years after promulgation of the revised O₃ NAAQS).

We know that developing the implementation plans that outline the steps a nonattainment area will take to

²³² Section 181(a)(1) of the CAA establishes classification categories for areas designated nonattainment for the primary O₃ NAAQS. These categories range from “Marginal,” the lowest O₃ classification with the fewest requirements associated with it, to “Extreme,” the highest classification with the most required programs. Areas with worse O₃ problems are given more time to attain the NAAQS and more associated emission control requirements.

²³³ Implementation of the 2008 National Ambient Air Quality Standards for Ozone: State Implementation Plan Requirements; Final Rule (80 FR 12264; March 6, 2015) and Implementation of the 2008 National Ambient Air Quality Standards for Ozone: Nonattainment Area Classifications Approach, Attainment Deadlines and Revocation of the 1997 Ozone Standards for Transportation Conformity Purposes (77 FR 30160; May 21, 2012).

meet an air quality standard requires a significant amount of work on the part of state, tribal or local air agencies. The EPA routinely looks for ways to reduce this workload, including assisting with air quality modeling by providing inputs such as emissions, meteorological and boundary conditions; and sharing national-scale model results that states can leverage in their development of attainment demonstrations.

B. O₃ Air Quality Designations

1. Area Designation Process

After the EPA establishes or revises a NAAQS, the CAA directs the EPA and the states to take steps to ensure that the new or revised NAAQS is met. One of the first steps, known as the initial area designations, involves identifying areas of the country that either meet or do not meet the new or revised NAAQS, along with any nearby areas that contribute to areas that do not meet the new or revised NAAQS.

Section 107(d)(1) of the CAA provides that, “By such date as the Administrator may reasonably require, but not later than 1 year after promulgation of a new or revised national ambient air quality standard for any pollutant under section 109, the Governor of each state shall . . . submit to the Administrator a list of all areas (or portions thereof) in the state” that designates those areas as nonattainment, attainment, or unclassifiable. The EPA must then promulgate the area designations according to a specified process, including procedures to be followed if the EPA intends to modify a state’s initial recommendation.

Clean Air Act Section 107(d)(1)(B)(i) further provides, “Upon promulgation or revision of a national ambient air quality standard, the Administrator shall promulgate the designations of all areas (or portions thereof) . . . as expeditiously as practicable, but in no case later than 2 years from the date of promulgation of the new or revised national ambient air quality standard. Such period may be extended for up to one year in the event the Administrator has insufficient information to promulgate the designations.” By no later than 120 days prior to promulgating area designations, the EPA is required to notify states of any intended modifications to their recommendations that the EPA may deem necessary. States then have an opportunity to demonstrate why any proposed modification is inappropriate. Whether or not a state provides a recommendation, the EPA must timely

promulgate the designation that the agency deems appropriate.

While section 107 of the CAA specifically addresses states, the EPA intends to follow the same process for tribes to the extent practicable, pursuant to CAA section 301(d) regarding tribal authority and the Tribal Authority Rule (63 FR 7254, February 12, 1998). To provide clarity and consistency in doing so, the EPA issued a 2011 guidance memorandum on working with tribes during the designation process.²³⁴

As discussed in sections II and IV of this preamble, the EPA is revising both the primary and secondary O₃ NAAQS. Accordingly, the EPA intends to complete designations for both NAAQS following the standard 2-year process discussed above. In accordance with section 107(d)(1) of the CAA, state Governors (and tribes, if they choose) should submit their initial designation recommendations for a revised primary and secondary NAAQS by 1 year after October 1, 2015. If the EPA intends to modify any state recommendation, the EPA would notify the appropriate state Governor (or tribal leader) no later than 120 days prior to making final designation decisions. A state or tribe that believes the modification is inappropriate would then have the opportunity to demonstrate to the EPA why it believes its original recommendation (or a revised recommendation) is more appropriate. The EPA would take any additional input into account in making the final designation decisions.

The CAA defines an area as nonattainment if it is violating the NAAQS or if it is contributing to a violation in a nearby area. Consistent with previous area designations processes, the EPA intends to use area-specific analysis of multiple factors to support area boundary decisions. The EPA intends to evaluate information related to the following factors for designations: air quality data, emissions and emissions-related data, meteorology, geography/topography, and jurisdictional boundaries. Additional guidance on the designation process and how these factors may be evaluated and inform the process will be issued by the EPA early in 2016 to assist states in developing their recommendations.

²³⁴ Page, S. (2011). Guidance to Regions for Working with Tribes during the National Ambient Air Quality Standards (NAAQS) Designations Process, Memorandum from Stephen D. Page, Director, EPA Office of Air Quality Planning and Standards to Regional Air Directors, Regions I–X, December 20, 2011. Available: http://www.epa.gov/ttn/oarp/t1/memoranda/20120117naaqs_guidance.pdf.

Areas that are designated as nonattainment are also classified at the time of designation by operation of law according to the severity of their O₃ problem. The classification categories are Marginal, Moderate, Serious, Severe, and Extreme. Ozone nonattainment areas are subject to specific mandatory measures depending on their classification. As indicated previously, the thresholds for the classification categories will be established in a future O₃ implementation rule.

Clean Air Act section 182(h) authorizes the EPA Administrator to determine that an area designated nonattainment can be treated as a rural transport area. Regardless of its classification, a rural transport area is deemed to have fulfilled all O₃-related planning and control requirements if it meets the CAA’s requirements for areas classified Marginal, which is the lowest classification specified in the CAA. In accordance with the statute, a nonattainment area may qualify for this determination if it meets the following criteria:

- The area does not contain emissions sources that make a significant contribution to monitored O₃ concentrations in the area, or in other areas; and
- The area does not include and is not adjacent to a Metropolitan Statistical Area.

Historically, the EPA has listed four nonattainment areas as rural transport areas under this statutory provision.²³⁵ The EPA has not issued separate written guidance to further elaborate on the interpretation of these CAA qualification criteria. However, the EPA developed draft guidance in 2005 that explains the kinds of technical analyses that states could use to establish that transport of O₃ and/or O₃ precursors into the area is so overwhelming that the contribution of local emissions to an observed 8-hour O₃ concentration above the level of the NAAQS is relatively minor and determine that emissions within the area do not make a significant contribution to the O₃ concentrations measured in the area or in other areas.²³⁶ While this guidance

²³⁵ For the 1979 1-hour O₃ standard, Door County Area, Wisconsin; Edmonson County Area, Kentucky; Essex County Area (Whiteface Mountain), New York; and Smyth County Area (White Top Mountain), Virginia were recognized by the EPA as rural transport areas. No rural transport areas were recognized for the 1997 or 2008 8-hour O₃ standards.

²³⁶ U.S. Environmental Protection Agency (2005). Criteria For Assessing Whether an Ozone Nonattainment Area is Affected by Overwhelming Transport [Draft EPA Guidance]. U.S. Environmental Protection Agency, Research Triangle Park, NC. June 2005. Available at <http://>

was not prepared specifically for rural transport areas, it could be useful to states for developing technical information to support a request that the EPA treat a specific O₃ nonattainment area as a rural transport area. The EPA will work with states to ensure nonattainment areas eligible for treatment as rural transport areas are identified.

2. Exceptional Events

During the initial area designations process, the EPA intends to evaluate multiple factors, including air quality data, when identifying and determining boundaries for areas of the country that meet or do not meet the revised O₃ NAAQS. In some cases, these data may be influenced by exceptional events. Under the Exceptional Events Rule, an air agency can request and the EPA can agree to exclude data associated with event-influenced exceedances or violations of a NAAQS, including the revised O₃ NAAQS, provided the event meets the statutory requirements in section 319(b) of the CAA, which requires that:

- the event “affects air quality;”
- the event “is not reasonably controllable or preventable;”
- the event is “caused by human activity that is unlikely to recur at a particular location or [is] a natural event,”²³⁷ and
- that “a clear causal relationship must exist between the measured exceedances of a [NAAQS] and the exceptional event. . . .”

The EPA’s implementing regulations, the Exceptional Events Rule, further specify certain requirements for air agencies making exceptional events demonstrations.²³⁸

The ISA contains discussions of natural events that may contribute to O₃ or O₃ precursors. These include stratospheric O₃ intrusion and wildfire events.²³⁹ As indicated above, to satisfy the exceptional events requirements and to qualify for data exclusion under the Exceptional Events Rule, an air agency must develop and submit a

www.epa.gov/scram001/guidance/guide/owt_guidance_07-13-05.pdf.

²³⁷ A natural event is further described in 40 CFR 50.1(k) as “an event in which human activity plays little or no direct causal role.”

²³⁸ 72 FR 13,560 (March 22, 2007), “Treatment of Data Influenced by Exceptional Events,” Final Rule; see also 40 CFR parts 50 and 51.

²³⁹ The preamble to the Exceptional Events Rule (72 FR 13560) identifies both stratospheric O₃ intrusions and wildfires as natural events that could also qualify as exceptional events under the CAA and Exceptional Event Rule criteria. Note that O₃ resulting from routine natural emissions from vegetation, microbes, animals and lightning are not exceptional events authorized for exclusion under the section 319 of the CAA.

demonstration, including evidence, addressing each of the identified criteria. The extent to which a stratospheric O₃ intrusion event or a wildfire event contributes to O₃ levels can be uncertain, and in most cases requires detailed analyses to determine.

Strong stratospheric O₃ intrusion events, most prevalent at high elevation sites during winter or spring, can be identified based on measurements of low relative humidity, evidence of deep atmospheric mixing, and a low ratio of CO to O₃ based on ambient measurements. Accurately determining the extent of weaker intrusion events remains challenging (U.S. EPA 2013, p. 3–34). Although states have submitted only a few exceptional events demonstrations for stratospheric O₃ intrusion, the EPA recently approved a demonstration from Wyoming for a June 2012 stratospheric O₃ event.²⁴⁰

While stratospheric O₃ intrusions can increase monitored ground-level ambient O₃ concentrations, wildfire plumes can either suppress or enhance O₃ depending upon a variety of factors including fuel type, combustion stage, plume chemistry, aerosol effects, meteorological conditions and distance from the fire (Jaffe and Wigder, 2012). As a result, determining the impact of wildfire emissions on specific O₃ observations is challenging. The EPA recently approved an exceptional events demonstration for wildfires affecting 1-hour O₃ levels in Sacramento, California in 2008 that successfully used a variety of analytical tools (e.g., regression modeling, back trajectories, satellite imagery, etc.) to support the exclusion of O₃ data affected by large fires.²⁴¹

In response to previously expressed stakeholder feedback regarding implementation of the Exceptional Events Rule and specific stakeholder concerns regarding the burden of exceptional events demonstrations, the EPA is currently engaged in a rulemaking process to amend the Exceptional Events Rule. As part of an upcoming notice and comment rulemaking effort (and related activities, including the issuance of relevant guidance documents), the EPA sees opportunities to standardize best

²⁴⁰ U.S. EPA (2014) Treatment of Data Influenced by Exceptional Events: Examples of Reviewed Exceptional Event Submissions. U.S. Environmental Protection Agency, Research Triangle Park, NC, available at <http://www.epa.gov/ttn/analysis/exevents.htm>.

²⁴¹ U.S. EPA (2014) Treatment of Data Influenced by Exceptional Events: Examples of Reviewed Exceptional Event Submissions. U.S. Environmental Protection Agency, Research Triangle Park, NC. Examples of O₃-related exceptional event submissions, available at <http://www.epa.gov/ttn/analysis/exevents.htm>.

practices for collaboration between the EPA and air agencies, clarify and simplify demonstrations, and improve tools and consistency.

Additionally, the EPA intends to develop guidance to address implementing the Exceptional Events Rule criteria for wildfires that could affect ambient O₃ concentrations. Wildfire emissions are a component of background O₃ (Jaffe and Wigder, 2012) and in some locations can significantly contribute to periodic high O₃ levels (Emery, 2012). The threat from wildfires can be mitigated through management of wildland vegetation. Planned and managed fires are one tool that land managers can use to reduce fuel load, unnatural understory and tree density, thus helping to reduce the risk of catastrophic wildfires. Allowing some wildfires to continue and the thoughtful use of prescribed fire can influence the occurrence of catastrophic wildfires, which may reduce the probability of fire-induced smoke impacts and subsequent health effects. Thus, appropriate use of prescribed fire may help manage the contribution of wildfires to both background and periodic peak O₃ air pollution. Several commenters expressed concern that the revised O₃ NAAQS could limit the future use of prescribed fire. Under the current Exceptional Events Rule, prescribed fires meeting the rule criteria may also qualify as exceptional events. The EPA intends to further clarify the Exceptional Events Rule criteria for prescribed fire on wildland in its upcoming rulemaking.

The EPA is committed to working with federal land managers, other federal agencies, tribes and states to effectively manage prescribed fire use to reduce the impact of wildfire-related emissions on O₃ through policies and regulations implementing these standards.

C. How do the New Source Review (NSR) requirements apply to the revised O₃ NAAQS?

1. NSR Requirements for Major Stationary Sources for the Revised O₃ NAAQS

The CAA, at parts C and D of title I, contains preconstruction review and permitting programs applicable to new major stationary sources and major modifications of existing major sources. The preconstruction review of each new major stationary source and major modification applies on a pollutant-specific basis, and the requirements that apply for each pollutant depend on whether the area in which the source is situated is designated as attainment (or

unclassifiable) or nonattainment for that pollutant. In areas designated attainment or unclassifiable for a pollutant, the PSD requirements under part C apply to construction at major sources. In areas designated nonattainment for a pollutant, the NNSR requirements under part D apply to major source construction. Collectively, those two sets of permit requirements are commonly referred to as the “major New Source Review” or “major NSR” programs.

Until an area is formally designated with respect to the revised O₃ NAAQS, the NSR provisions applicable under that area’s current designation for the 2008 O₃ NAAQS (including any applicable anti-backsliding requirements) will continue to apply. That is, for areas designated as attainment/unclassifiable for the 2008 O₃ NAAQS, PSD will apply for new major stationary sources and major modifications that trigger major source permitting requirements for O₃; areas designated nonattainment for the 2008 O₃ NAAQS must comply with the NNSR requirements for new major stationary sources and major modifications that trigger major source permitting requirements for O₃. When the new designations for the revised O₃ NAAQS become effective, under the current rules, those designations will generally serve to determine whether PSD or NNSR applies to O₃ and its precursors. The PSD regulations at 40 CFR 51.166(i)(2) and 52.21(i)(2) provide that the substantive PSD requirements do not apply for a particular pollutant if the owner or operator of the new major stationary source or major modification demonstrates that the area in which the source is located is designated nonattainment for that pollutant under CAA section 107. Thus, new major sources and modifications will generally be subject to the PSD program requirements for O₃ if they are locating in an area that does not have a current nonattainment designation under CAA section 107 for O₃. These rules further provide that nonattainment designations for a revoked NAAQS, as contained in 40 CFR part 81, are not viewed as current designations under CAA section 107 for purposes of determining the applicability of such PSD requirements.²⁴²

The EPA’s major NSR regulations define the term “regulated NSR pollutant” to include any pollutant for which a NAAQS has been promulgated

²⁴² This description of paragraph (i)(2) of the PSD regulations at 40 CFR 51.166 and 52.21 reflects revisions made in the final 2008 O₃ NAAQS SIP Requirements Rule. See 80 FR 12264 at 12287 (March 6, 2015).

and any pollutant identified in EPA regulations as a constituent or precursor to such pollutant.²⁴³ Both the PSD and NNSR regulations identify VOC and NO_x as precursors to O₃. Accordingly, the major NSR programs for O₃ are applied to emissions of VOC and NO_x as precursors of O₃.²⁴⁴

2. Prevention of Significant Deterioration (PSD) Program

The statutory requirements for a PSD permit program set forth under part C of title I of the CAA (sections 160 through 169) are addressed by the EPA’s PSD regulations found at 40 CFR 51.166 (minimum requirements for an approvable PSD SIP) and 40 CFR 52.21 (PSD permitting program for permits issued under the EPA’s federal permitting authority). Both sets of regulations already apply for O₃ when the area is designated attainment or unclassifiable for O₃ and when the new source or modification triggers PSD requirements for O₃.

For PSD, a “major stationary source” is one that emits or has the potential to emit 250 tons per year (tpy) or more of any regulated NSR pollutant, unless the new or modified source is classified under a list of 28 source categories contained in the statutory definition of “major emitting facility” in section 169(1) of the CAA. For those 28 source categories, a “major stationary source” is one that emits or has the potential to emit 100 tpy or more of any regulated NSR pollutant. A “major modification” is a physical change or a change in the method of operation of an existing major stationary source that results first, in a significant emissions increase of a regulated NSR pollutant for the project, and second, in a significant net emissions increase of that pollutant at the source. See 40 CFR 51.166(b)(2)(i), 40 CFR 52.21(b)(2)(i).

Among other things, for each regulated NSR pollutant emitted or increased in significant amounts, the PSD program requires a new major stationary source or a major modification to apply Best Available Control Technology and to conduct an air quality impact analysis to demonstrate that the proposed source or project will not cause or contribute to a violation of any NAAQS or PSD increment (see CAA section 165(a)(3)–

²⁴³ The definition of “regulated NSR pollutant” is found in the PSD regulations at 40 CFR 51.166(b)(49) and 52.21(b)(50), and in the NNSR regulations at 40 CFR 51.165(a)(1)(xxxvii).

²⁴⁴ VOC and NO_x are defined as precursors of ozone in the PSD regulations at 40 CFR 51.166(b)(49)(i)(b)(1) and 52.21(b)(50)(i)(b)(1), and in the NNSR regulations at 40 CFR 51.165(a)(1)(xxxvii)(B) and (C)(1) and part 51, Appendix S, II.A.31(ii)(b)(1).

(4), 40 CFR 51.166(j)–(k), 40 CFR 52.21(j)–(k)). The PSD requirements may also include, in appropriate cases, an analysis of potential adverse impacts on Class I areas (see CAA sections 162 and 165).²⁴⁵ The EPA has generally interpreted the requirement for an air quality impact analysis under CAA section 165(a)(3) and the implementing regulations to include a requirement to demonstrate that emissions from the proposed facility will not cause or contribute to a violation of any NAAQS that is in effect as of the date a PSD permit is issued.²⁴⁶ See, e.g., 73 FR 28321, 28324, 28340 (May 16, 2008); 78 FR 3253 (Jan. 15, 2013); Memorandum from Stephen D. Page, Director, Office of Air Quality Planning & Standards, “Applicability of the Federal Prevention of Significant Deterioration Permit Requirements to New and Revised National Ambient Air Quality Standards” (April 1, 2010). Consistent with this interpretation, the demonstration required under CAA section 165(a)(3) and 40 CFR 51.166(k) and 52.21(k) will apply to any revised O₃ NAAQS when such NAAQS become effective, except to the extent that a pending permit application is subject to a grandfathering provision that the EPA establishes through rulemaking. In addition, the other existing requirements of the PSD program will remain applicable to O₃ after the revised O₃ NAAQS takes effect.

Because the complex chemistry of O₃ formation in the atmosphere poses significant challenges for the assessing the impacts of individual stationary sources on O₃ formation, the EPA’s judgment historically has been that it is not technically sound to designate a

²⁴⁵ Congress established certain Class I areas in section 162(a) of the CAA, including international parks, national wilderness areas, and national parks that meet certain criteria. Such Class I areas, known as mandatory federal Class I areas, are afforded special protection under the CAA. In addition, states and tribal governments may establish Class I areas within their own political jurisdictions to provide similar special air quality protection.

²⁴⁶ An exception occurs in cases where the EPA has included a grandfathering provision in its PSD regulations for a particular pollutant. The EPA historically has exercised its discretion to transition the implementation of certain new requirements through grandfathering, under appropriate circumstances, either by rulemaking or through a case-by-case determination for a specific permit application. In 2014, the United States Court of Appeals for the Ninth Circuit vacated a decision by the EPA to issue an individual PSD permit grandfathering a permit applicant from certain requirements. See *Sierra Club v. EPA*, 762 F.3d 971 (9th Cir. 2014). In light of that decision, the EPA is no longer asserting authority to grandfather permit applications on a case-by-case basis. This decision is addressed in more detail in the discussion of the grandfathering provisions that the EPA is issuing through this rulemaking in section VII of this preamble.

specific air quality model that must be used in the PSD permitting process to make this demonstration for O₃. To address ambient impacts of emissions from proposed individual stationary sources on O₃, the EPA proposed amendments to Appendix W to 40 CFR part 51 in July 2015 that would, among other things, revise the Appendix W provisions relating to the analytical techniques for demonstrating that an individual PSD source or modification does not cause or contribute to a violation of the O₃ NAAQS (80 FR 45340, July 29, 2015). Until any revisions are finalized and in effect, PSD permit applicants should continue to follow the current provisions in the applicable regulations and Appendix W in order to demonstrate that a proposed source or modification does not cause or contribute to a violation of the O₃ NAAQS.

a. What transition plan is the EPA providing for implementing the PSD requirements for the revised O₃ NAAQS?

In this rulemaking, the EPA is amending the PSD regulations at 40 CFR 51.166 and 40 CFR 52.21 to include a grandfathering provision that will allow reviewing authorities to continue to review certain pending PSD permit applications in accordance with the O₃ NAAQS that was in effect when a specific permitting milestone was reached, rather than the revised O₃ NAAQS. The EPA is finalizing the grandfathering provision as proposed with two trigger dates—the signature date of the revised O₃ NAAQS rule for complete applications and the effective date of the revised O₃ NAAQS for a draft permit or preliminary determination. A more detailed discussion of the final provision, comments received and our responses to those comments is provided in section VII of this preamble, which addresses this change to the PSD regulations, as well as the Response to Comment Document contained in the docket for this rulemaking.

b. What screening and compliance demonstration tools are used to implement the PSD program?

The EPA has historically allowed the use of screening and compliance demonstration tools to help facilitate the implementation of the NSR program by reducing the source's burden and streamlining the permitting process for circumstances where the emissions or ambient impacts of a particular pollutant could be considered *de minimis*. For example, the EPA has established significant emission rates, or SERs, that are used as screening tools to

determine when a pollutant would be considered to be emitted in a significant amount and, accordingly, when the NSR requirements should be applied to that pollutant. See 40 CFR 51.166(b)(23) and 52.21(b)(23). For O₃, the EPA established a SER of 40 tpy for emissions of each O₃ precursor—VOC and NO_x. For PSD, the O₃ SER applies independently to emissions of VOC and NO_x (emissions of precursors are not added together) to determine when the proposed major stationary source or major modification must undergo PSD review for that precursor and whether individual PSD requirements, such as BACT, apply to that precursor.²⁴⁷

In the context of the PSD air quality impact analysis, the EPA has also used a value called a significant impact level (SIL) as a compliance demonstration tool. The SIL, expressed as an ambient concentration of a pollutant, may be used first to determine the geographical scope of the ambient impact analysis that must be completed for the applicable pollutant to satisfy the air quality demonstration requirement under CAA section 165(a)(3). A second use is to guide the determination of whether the impact of the source is considered to cause or contribute to a violation of any NAAQS. The EPA has not established a SIL for O₃. The EPA is currently considering development of a SIL for O₃ through either guidance or a rulemaking process. Such a SIL would complement proposed revisions to Appendix W mentioned above (80 FR 45340, July 29, 2015) and would assist in the implementation of the PSD air quality analysis requirement for protection of the O₃ NAAQS. However, the EPA is not making revisions in this rulemaking to address the PSD air quality analysis for O₃. Until any rulemaking to amend existing PSD regulations for O₃ is completed, permitting decisions should continue to be based on the existing provisions in the applicable regulations.

Several commenters addressed statements that the EPA made concerning screening tools for O₃ in the preamble to the O₃ NAAQS proposal. These statements were not linked to any proposed amendments to EPA regulations. Aside from adopting the grandfathering provision addressed in section VII of this preamble, the EPA is not revising the PSD requirements for O₃ in this final rule. Therefore, the EPA

²⁴⁷ See *In re Footprint Power Salem Harbor Development, LP*, 16 E.A.D. ___, PSD Appeal No. 14-02, at 20-25 (EAB, Sept. 2, 2014) (including description of EPA's position on application of BACT to ozone precursors) available at [http://yosemite.epa.gov/oa/EAB_Web_Docket.nsf/PSD+Permit+Appeals+\(CAA\)?OpenView](http://yosemite.epa.gov/oa/EAB_Web_Docket.nsf/PSD+Permit+Appeals+(CAA)?OpenView).

is not responding to those comments at this time, consistent with the EPA's general approach to comments on implementation topics described above.

c. Other PSD Transition Issues

The EPA anticipates that the existing O₃ air quality in some areas currently designated attainment or unclassifiable for O₃ will not meet the revised O₃ NAAQS upon its effective date and that some of these areas will ultimately be designated “nonattainment” for the revised O₃ NAAQS through the formal area designation process set forth under the CAA (see section VIII.B above). However, until the EPA issues such nonattainment designations, proposed new major sources and major modifications situated in any area designated attainment or unclassifiable for the 2008 O₃ NAAQS will continue to be required to address O₃ in a PSD permit.²⁴⁸ As mentioned above, the PSD permitting program requires that proposed new major stationary sources and major modifications must demonstrate that the emissions from the proposed source or modification will not cause or contribute to a violation of any NAAQS. In the notice of proposed rulemaking, the EPA provided information concerning its views on the possibility that some PSD permit applications could satisfy the air quality analysis requirements for O₃ by obtaining air quality offsets (called PSD offsets).²⁴⁹ Several commenters expressed concern that without some transition provisions in the final rule exempting PSD permit applications for sources located in such areas from meeting the air quality analysis requirements for the revised O₃ NAAQS, such applications might not be able to satisfy the demonstration requirement, as the current ambient air monitoring data indicate the revised lower standards are not being met. The O₃ NAAQS proposal included no proposed revisions to PSD regulations on this

²⁴⁸ Any proposed major stationary source or major modification subject to PSD for O₃ that does not receive its PSD permit by the effective date of a new O₃ nonattainment designation for the area where the source would locate would then be required to satisfy all of the applicable NNSR preconstruction permit requirements for O₃, even if such source had been grandfathered under the PSD regulations from the demonstration requirement under CAA section 165(a)(3) for O₃.

²⁴⁹ The EPA has historically recognized in regulations and through other actions that sources applying for PSD permits may have the option of utilizing offsets as part of the required PSD demonstration under CAA section 165(a)(3)(B). See, e.g., *In re Interpower of New York, Inc.*, 5 E.A.D. 130, 141 (EAB 1994) (describing an EPA Region 2 PSD permit that relied in part on offsets to demonstrate the source would not cause or contribute to a violation of the NAAQS). 52 FR 24698 (July 1, 1987); 78 FR 3261-62 (Jan. 15, 2013).

topic and the EPA is not making any revisions to the PSD requirements for O₃ in this action to address this issue. Therefore, the EPA is not responding to those comments at this time, consistent with its general approach to comments on implementation topics described above. However, to help address this concern raised by commenters, the EPA is considering issuing additional guidance on how PSD offsets can be implemented.

3. Nonattainment NSR

Part D of title I of the CAA includes preconstruction review and permitting requirements for new major stationary sources and major modifications when they locate in areas designated nonattainment for a particular pollutant. The relevant part D requirements are typically referred to as the nonattainment NSR (NNSR) program. The EPA regulations for the NNSR program are contained at 40 CFR 51.165, 52.24 and part 51 Appendix S. The EPA's minimum requirements for a NNSR program to be approvable into a SIP are contained in 40 CFR 51.165. Appendix S to 40 CFR part 51 contains an interim NNSR program. This interim program enables implementation of NNSR permitting in nonattainment areas that lack a SIP-approved NNSR permitting program for the particular nonattainment pollutant, and the interim program can be applied during the time between the date of the relevant nonattainment designation and the date on which the EPA approves into the SIP a NNSR program or additional components of an NNSR program for a particular pollutant.²⁵⁰ This interim program is commonly known as the Emissions Offset Interpretative Rule, and is applicable to all criteria pollutants, including O₃.²⁵¹

The EPA is not modifying any existing NNSR requirements in this rulemaking. Under the CAA, area designations for new or revised NAAQS are addressed subsequent to the effective date of the new or revised NAAQS. If the EPA determines that any revisions to the existing NNSR requirements, including those in Appendix S, are appropriate, the EPA expects, at a later date contemporaneous with the designation process for the revised O₃ NAAQS, to propose those revisions. If any changes are proposed to Appendix S requirements, the EPA

anticipates that it would intend for those changes to become effective no later than the effective date of the area designations. This timing would allow air agencies that lack an approved NNSR program for O₃ to use the relevant Appendix S provisions to issue NNSR permits addressing O₃ on and after the effective date of designations of new nonattainment areas for O₃ until such time as a NNSR program for O₃ is approved into the SIP.²⁵²

For NNSR, new major stationary sources and major modifications for O₃ must comply with the Lowest Achievable Emission Rate (LAER) requirements as defined in the CAA and NNSR rules, and must perform other analyses and satisfy other requirements under section 173 of the CAA. For example, under CAA section 173(c) emissions reductions, known as emissions offsets, must be secured to offset the increased emissions of the air pollutant (including the relevant precursors) from the new or modified source by an equal or greater reduction, as applicable, of such pollutant. The appropriate emissions offset needed for a particular source will depend upon the classification for the O₃ nonattainment area in which the source or modification will locate, such that areas with more severe nonattainment classifications have more stringent offset requirements. This ranges from 1.1:1 for areas classified as Marginal to 1.5:1 for areas classified as Extreme. *See, e.g.,* CAA section 182, 40 CFR 51.165(a)(9) and 40 CFR part 51 Appendix S section IV.G.2.

To facilitate continued economic development in nonattainment areas, many states have established offset banks or registries.²⁵³ Such banks or registries can help new or modified major stationary source owners meet offset requirements by streamlining identification and access to available emissions reductions. Some states have established offset banks to help ensure a consistent method for generating, validating and transferring NO_x and VOC offsets. Offsets in these areas are generated by emissions reductions that meet specific creditability criteria set forth by the SIP consistent with the EPA regulations. *See* 40 CFR 51.165(a)(3)(ii)(A)-(J) and part 51 Appendix S section IV.C. The EPA

²⁵² States with SIP-approved NNSR programs for O₃ should evaluate that program to determine whether they can continue to issue permits under their approved program or whether revisions to their program are necessary to address the revised O₃ NAAQS.

²⁵³ *See, for example,* emission reduction credit banking programs in Ohio (OAC Chapter 3745-1111) and California (H&SC Section 40709).

received comments expressing concern about the limited availability of offsets in nonattainment areas. Since the EPA did not propose, and is not finalizing, any amendments related to the NNSR offset provisions, the EPA is not responding to those comments at this time, consistent with the EPA's general approach to comment on implementation topics as described above.

D. Transportation and General Conformity

1. What are transportation and general conformity?

Conformity is required under CAA section 176(c) to ensure that federal actions are consistent with ("conform to") the purpose of the SIP. Conformity to the purpose of the SIP means that federal activities will not cause new air quality violations, worsen existing violations, or delay timely attainment of the relevant NAAQS or interim reductions and milestones. Conformity applies to areas that are designated nonattainment, and those nonattainment areas redesignated to attainment with a CAA section 175A maintenance plan after 1990 ("maintenance areas").

The EPA's Transportation Conformity Rule (40 CFR 51.390 and part 93, subpart A) establishes the criteria and procedures for determining whether transportation activities conform to the SIP. These activities include adopting, funding or approving transportation plans, transportation improvement programs (TIPs) and federally supported highway and transit projects. For further information on conformity rulemakings, policy guidance and outreach materials, *see* the EPA's Web site at <http://www.epa.gov/otaq/stateresources/transconf/index.htm>. The EPA may issue future transportation conformity guidance as needed to implement a revised O₃ NAAQS.

With regard to general conformity, the EPA first promulgated general conformity regulations in November 1993. (40 CFR part 51, subpart W, 40 CFR part 93, subpart B) Subsequently the EPA finalized revisions to the general conformity regulations on April 5, 2010. (75 FR 17254-17279). Besides ensuring that federal actions not covered by the transportation conformity rule will not interfere with the SIP, the general conformity program also fosters communications between federal agencies and state/local air quality agencies, provides for public notification of and access to federal agency conformity determinations, and allows for air quality review of

²⁵⁰ *See* Appendix S, Part I; 40 CFR 52.24(k).

²⁵¹ As appropriate, certain NNSR requirements under 40 CFR 51.165 or Appendix S can also apply to sources and modifications located in areas that are designated attainment or unclassifiable in the Ozone Transport Region. *See, e.g.,* CAA 184(b)(2), 40 CFR 52.24(k).

individual federal actions. More information on the general conformity program is available at <http://www.epa.gov/air/genconform/>.

2. When would transportation and general conformity apply to areas designated nonattainment for the revised O₃ NAAQS?

Transportation and general conformity apply one year after the effective date of nonattainment designations for the revised O₃ NAAQS. This is because CAA section 176(c)(6) provides a 1-year grace period from the effective date of initial designations for any revised NAAQS before transportation and general conformity apply in areas newly designated nonattainment for a specific pollutant and NAAQS.

3. Impact of a Revised O₃ NAAQS on a State's Existing Transportation and/or General Conformity SIP

In this final rule, the EPA is revising the O₃ NAAQS, but is not making specific changes to its transportation or general conformity regulations. Therefore, states should not need to revise their transportation and/or general conformity SIPs. While we are not making any revisions to the general conformity regulations at this time, we recommend, when areas develop SIPs for a revised O₃ NAAQS, that state and local air quality agencies work with federal agencies with large emitting activities that are subject to the general conformity regulations to establish an emissions budget for those facilities and activities in order to facilitate future conformity determinations under the conformity regulations. Finally, states with existing conformity SIPs and new nonattainment areas may also need to revise their conformity SIPs in order to ensure the state regulations apply in any newly designated areas.

Because significant tracts of land under federal management may be included in nonattainment area boundaries, the EPA encourages state and local air quality agencies to work with federal agencies to assess and develop emissions budgets that consider emissions from projects subject to general conformity, including emissions from fire on wildland, in any baseline, modeling and SIP attainment inventory. Where appropriate, states, land managers, and landowners may also consider developing plans to ensure that fuel accumulations are addressed. Information is available from DOI and USDA Forest Service on the ecological role of fire and on smoke management

programs and basic smoke management practices.²⁵⁴

If this is the first time that transportation conformity will apply in a state, such a state is required by the statute and EPA regulations to submit a SIP revision that addresses three specific transportation conformity requirements that address consultation procedures and written commitments to control or mitigation measures associated with conformity determinations for transportation plans, TIPs or projects. (40 CFR 51.390) Additional information and guidance can be found in the EPA's "Guidance for Developing Transportation Conformity State Implementation Plans" (<http://www.epa.gov/otaq/stateresources/transconf/policy/420b09001.pdf>).

E. Regional and International Pollution Transport

1. Interstate Transport

The CAA contains provisions that specifically address and require regulation of the interstate transport of air pollution that does not otherwise qualify for data exclusion under the Act's exceptional events provisions. As previously noted, emissions from events, such as wildfires, may qualify as exceptional events and may be transported across jurisdictional boundaries. The EPA intends to address the transport of event-related emissions in our upcoming proposed revisions to the Exceptional Events Rule and draft guidance document addressing the Exceptional Events Rule criteria for wildfires that could affect O₃ concentrations. The EPA encourages affected air agencies to coordinate with their EPA regional office to identify approaches to evaluate the potential impacts of transported event-related emissions and determine the most appropriate information and analytical methods for each area's unique situation.

CAA section 110(a)(2)(D)(i)(I), *Interstate Transport*—CAA section 110(a)(2)(D)(i)(I) requires states to develop and implement a SIP to address the interstate transport of emissions. Specifically, this provision requires the SIP to prohibit "any source or other type of emissions activity within the state" that would "significantly contribute to nonattainment" of any NAAQS in another state, or that would "interfere with maintenance" of any NAAQS in another state. When EPA promulgates or

²⁵⁴ USDA Forest Service and Natural Resources Conservation Service, Basic Smoke Management Practices Tech Note, October 2011, http://www.nrcs.usda.gov/Internet/FSE_DOCUMENTS/stelprd_b1046311.pdf.

revises a NAAQS, each state is required to submit a SIP addressing this interstate transport provision within 3 years.

CAA section 126, *Interstate Transport*—CAA section 126(b) provides states and political subdivisions with a mechanism to petition the Administrator for a finding that "any major source or group of stationary sources emits or would emit any air pollution in violation of the prohibition of [CAA section 110(a)(2)(D)(i)(I)]." ²⁵⁵ Where the EPA makes such finding, the source is allowed to operate beyond a 3-month period after such finding only if the EPA establishes emissions limitations and a compliance schedule designated to bring the source into compliance as expeditiously as practicable, but no later than three years after such finding. This mechanism is available to downwind states and political subdivisions, regardless of designation status, that would be affected by emissions from upwind states.

2. International Transport

The agency is active in work to reduce the international transport of O₃ and other pollutants that can contribute to "background" O₃ levels in the U.S. Under the Convention on Long-Range Transboundary Air Pollution (LRTAP) of the United Nations Economic Commission for Europe, the U.S. has been a party to the Protocol to Abate Acidification, Eutrophication, and Ground-level Ozone (known as the Gothenburg Protocol) since 2005. The U.S. is also active in the LRTAP Task Force for Hemispheric Transport of Air Pollution. The U.S. has worked bilaterally with Canada under the US-Canada Air Quality Agreement to adopt an Ozone Annex to address transboundary O₃ impacts and continues to work with China on air quality management activities. This work includes supporting China's efforts to rapidly deploy power plant pollution controls that can achieve NO_x reductions of at least 80 to 90%. The U.S. also continues to work bilaterally with Mexico on the Border 2020 program to support efforts to improve environmental conditions in the border region. One of the main goals of the program is to reduce air pollution, including emissions that can cause transboundary O₃ impacts.

²⁵⁵ The text of section 126 codified in the United States Code cross references section 110(a)(2)(D)(ii) instead of section 110(a)(2)(D)(i). The courts have confirmed that this is a scrivener's error and the correct cross reference is to section 110(a)(2)(D)(i). See *Appalachian Power Co. v. EPA*, 249 F.3d 1032, 1040-44 (D.C. Cir. 2001).

Clean Air Act section 179B recognizes the possibility that certain nonattainment areas may be impacted by O₃ or O₃ precursor emissions from international sources beyond the regulatory jurisdiction of the state. The EPA's science review suggests that the influence of international sources on U.S. O₃ levels will be largest in locations that are in the immediate vicinity of an international border with Canada or Mexico. The science review also cites two recent studies which indicate that intercontinental transport of pollution, along with other natural sources and local pollutant sources, can affect O₃ air quality in the western U.S. under specific conditions. (U.S. EPA 2013, p. 3–140). Section 179B allows states to consider in their attainment plans and demonstrations whether an area might meet the O₃ NAAQS by the attainment date “but for” emissions contributing to the area originating outside the U.S. If a state is unable to demonstrate attainment of the NAAQS in such an area impacted by international transport after adopting all reasonably available control measures (e.g., RACM, including RACT, as required by CAA section 182(b)), the EPA can nonetheless approve the CAA-required state attainment plan and demonstration using the authority in section 179B.

When the EPA approves this type of attainment plan and demonstration, and there would be no adverse consequence for a finding that the area failed to attain the NAAQS by the relevant attainment date. States can also avoid potential sanctions and FIPs that would otherwise apply for failure to submit a required SIP submission or failure to submit an approvable SIP submission. For example, section 179B explicitly provides that the area shall not be reclassified to the next highest classification or required to implement a section 185 penalty fee program if a state meets the applicable criteria.

Section 179B authority does not allow an area to avoid a nonattainment designation or for the area to be classified with a lower classification than is indicated by actual ambient air quality. Section 179B also does not provide for any relaxation of mandatory emissions control measures (including contingency measures) or the prescribed emissions reductions necessary to achieve periodic emissions reduction progress requirements. In this way, section 179B insures that states will take actions to mitigate the public health impacts of exposure to ambient levels of pollution that violate the NAAQS by imposing reasonable control measures on the sources that are within the

jurisdiction of the state while also authorizing EPA to approve such attainment plans and demonstrations even though they do not fully address the public health impacts of international transport. Also, generally, monitoring data influenced by international transport may not be excluded from regulatory determinations. However, depending on the nature and scope of international emissions events affecting air quality in the U.S., the event-influenced data may qualify for exclusion under the Exceptional Events Rule. The EPA encourages affected air agencies to coordinate with their EPA regional office to identify approaches to evaluate the potential impacts of international transport and to determine the most appropriate information and analytical methods for each area's unique situation. The EPA will also work with states that are developing attainment plans for which section 179B is relevant, and ensure the states have the benefit of the EPA's understanding of international transport of ozone and ozone precursors.

The EPA has used section 179B authority previously to approve attainment plans for Mexican border areas in El Paso, TX (O₃, PM₁₀, and CO plans); and Nogales, AZ (PM₁₀ plan). The 24-hour PM₁₀ attainment plan for Nogales, AZ, was approved by EPA as sufficient to demonstrate attainment of the NAAQS by the Moderate classification deadline, but for international emissions sources in the Nogales Municipality, Mexico area (77 FR 38400, June 27, 2012).

States are encouraged to consult with their EPA Regional Office to establish appropriate technical requirements for these analyses.

IX. Statutory and Executive Order Reviews

Additional information about these statutes and Executive Orders can be found at <http://www2.epa.gov/laws-regulations/laws-and-executive-orders>.

A. Executive Order 12866: Regulatory Planning and Review and Executive Order 13563: Improving Regulation and Regulatory Review

This action is an economically significant regulatory action that was submitted to the Office of Management and Budget (OMB) for review. Any changes made in response to OMB recommendations have been documented in the docket. The EPA prepared an analysis of the potential costs and benefits associated with this action. This analysis is contained in the document, *Regulatory Impact Analysis*

of the Final National Ambient Air Quality Standards for Ground-Level Ozone, October 2015. A copy of the analysis is available in the RIA docket (EPA-HQ-OAR-2013-0169) and the analysis is briefly summarized here. The RIA estimates the costs and monetized human health and welfare benefits of attaining three alternative O₃ NAAQS nationwide. Specifically, the RIA examines the alternatives of 65 ppb and 70 ppb. The RIA contains illustrative analyses that consider a limited number of emissions control scenarios that states and Regional Planning Organizations might implement to achieve these alternative O₃ NAAQS. However, the CAA and judicial decisions make clear that the economic and technical feasibility of attaining ambient standards are not to be considered in setting or revising NAAQS, although such factors may be considered in the development of state plans to implement the standards. Accordingly, although an RIA has been prepared, the results of the RIA have not been considered in issuing this final rule.

B. Paperwork Reduction Act

The information collection requirements in this final rule have been submitted for approval to the Office of Management and Budget (OMB) under the Paperwork Reduction Act (PRA). The information collection requirements are not enforceable until OMB approves them. The Information Collection Request (ICR) document prepared by the EPA for these revisions has been assigned EPA ICR #2313.04.

The information collected and reported under 40 CFR part 58 is needed to determine compliance with the NAAQS, to characterize air quality and associated health and ecosystems impacts, to develop emission control strategies, and to measure progress for the air pollution program. We are extending the length of the required O₃ monitoring season in 32 states and the District of Columbia and the revised O₃ monitoring seasons will become effective on January 1, 2017. We are also revising the PAMS monitoring requirements to reduce the number of required PAMS sites while improving spatial coverage, and requiring states in moderate or above O₃ non-attainment areas and the O₃ transport region to develop an enhanced monitoring plan as part of the PAMS requirements. Monitoring agencies will need to comply with the PAMS requirements by June 1, 2019. In addition, we are revising the O₃ FRM to establish a new, additional technique for measuring O₃ in the ambient air. It will be

incorporated into the existing O₃ FRM, using the same calibration procedure in Appendix D of 40 CFR part 50. We are also making changes to the procedures for testing performance characteristics and determining comparability between candidate FEMs and reference methods.

For the purposes of ICR number 2313.04, the burden figures represent the burden estimate based on the requirements contained in this rule. The burden estimates are for the 3-year period from 2016 through 2018. The implementation of the PAMS changes will occur beyond the time frame of this ICR with implementation occurring in 2019. The cost estimates for the PAMS network (including revisions) will be captured in future routine updates to the Ambient Air Quality Surveillance ICR that are required every 3 years by OMB. The addition of a new FRM in 40 CFR part 50 and revisions to the O₃ FEM procedures for testing performance characteristics in 40 CFR part 53 does not add any additional information collection requirements.

The ICR burden estimates are associated with the changes to the O₃ seasons in this final rule. This information collection is estimated to involve 158 respondents for a total cost of approximately \$24,597,485 (total capital, labor, and operation and maintenance) plus a total burden of 339,930 hours for the support of all operational aspects of the entire O₃ monitoring network. The labor costs associated with these hours are \$20,209,966. Also included in the total are other costs of operations and maintenance of \$2,254,334 and equipment and contract costs of \$2,133,185. The actual labor cost increase to expand the O₃ monitoring seasons is \$2,064,707. In addition to the costs at the state, local, and tribal air quality management agencies, there is a burden to EPA of 41,418 hours and \$2,670,360. Burden is defined at 5 CFR 1320.3(b). State, local, and tribal entities are eligible for state assistance grants provided by the federal government under the CAA which can be used for related activities. An agency may not conduct or sponsor, and a person is not required to respond to, a collection of information unless it displays a currently valid OMB control number. The OMB control numbers for EPA's regulations in 40 CFR are listed in 40 CFR part 9.

C. Regulatory Flexibility Act (RFA)

I certify that this action will not have a significant economic impact on a substantial number of small entities under the RFA. This action will not impose any requirements on small

entities. Rather, this rule establishes national standards for allowable concentrations of O₃ in ambient air as required by section 109 of the CAA. See also *American Trucking Associations v. EPA*, 175 F. 3d at 1044–45 (NAAQS do not have significant impacts upon small entities because NAAQS themselves impose no regulations upon small entities). Similarly, the revisions to 40 CFR part 58 address the requirements for states to collect information and report compliance with the NAAQS and will not impose any requirements on small entities. Similarly, the addition of a new FRM in 40 CFR part 50 and revisions to the FEM procedures for testing in 40 CFR part 53 will not impose any requirements on small entities.

D. Unfunded Mandates Reform Act (UMRA)

This action does not contain an unfunded federal mandate of \$100 million or more as described in UMRA, 2 U.S.C. 1531–1538, and does not significantly or uniquely affect small governments. The revisions to the O₃ NAAQS impose no enforceable duty on any state, local, or tribal governments or the private sector beyond those duties already established in the CAA. The expected costs associated with the monitoring requirements are described in the EPA's ICR document, and these costs are not expected to exceed \$100 million in the aggregate for any year.

Furthermore, as indicated previously, in setting NAAQS the EPA cannot consider the economic or technological feasibility of attaining ambient air quality standards, although such factors may be considered to a degree in the development of state plans to implement the standards (see *American Trucking Associations v. EPA*, 175 F. 3d at 1043 [noting that because the EPA is precluded from considering costs of implementation in establishing NAAQS, preparation of a RIA pursuant to the UMRA would not furnish any information which the court could consider in reviewing the NAAQS]). With regard to the sections of the rule preamble discussing implementation of the revisions to the O₃ NAAQS, the CAA imposes the obligation for states to submit SIPs to implement the NAAQS for O₃. To the extent the EPA's discussion of implementation topics in this final rule may reflect some interpretations of those requirements, those interpretations do not impose obligations beyond the duties already established in the CAA and thus do not constitute a federal mandate for purposes of UMRA. The EPA is also adopting a grandfathering provision for

certain PSD permits in this action, as described above. However, that provision does not impose any mandate on any state, local, or tribal government or the private sector, but rather provides relief from requirements that would otherwise result from the new standards. In addition, the EPA is not requiring states to revise their SIPs to include such a provision.

E. Executive Order 13132: Federalism

This action does not have federalism implications. It will not have substantial direct effects on the states, on the relationship between the national government and the states, or on the distribution of power and responsibilities among the various levels of government.

F. Executive Order 13175: Consultation and Coordination With Indian Tribal Governments

This action does not have tribal implications as specified in Executive Order 13175. It does not have a substantial direct effect on one or more Indian tribes. This rule provides increased protection from adverse effects of ozone for the entire country, including for sensitive populations, and tribes are not obligated to adopt or implement any NAAQS. In addition, tribes are not obligated to conduct ambient monitoring for O₃ or to adopt the ambient monitoring requirements of 40 CFR part 58. Even if this action were determined to have tribal implications within the meaning of Executive Order 13175, it will neither impose substantial direct compliance costs on tribal governments, nor preempt tribal law. Thus, consultation under Executive Order 13175 was not required.

Nonetheless, consistent with the "EPA Policy on Consultation and Coordination with Indian Tribes", the EPA offered government-to-government consultation on the proposed rule. No tribe requested government-to-government consultation with the EPA on this rule. In addition, the EPA conducted outreach to tribal environmental professionals, which included participation in the Tribal Air call sponsored by the National Tribal Air Association, and two other calls available to tribal environmental professionals. During the public comment period we received comments on the proposed rule from seven tribes and three tribal organizations.

G. Executive Order 13045: Protection of Children From Environmental Health & Safety Risks

This action is subject to Executive Order 13045 because it is an

economically significant regulatory action as defined by Executive Order 12866, and the EPA believes that the environmental health risk addressed by this action may have a disproportionate effect on children. The rule will establish uniform NAAQS for O₃; these standards are designed to protect public health with an adequate margin of safety, as required by CAA section 109. However, the protection offered by these standards may be especially important for children because children, especially children with asthma, along with other at-risk populations²⁵⁶ such as all people with lung disease and people active outdoors, are at increased risk for health effects associated with exposure to O₃ in ambient air. Because children are considered an at-risk lifestage, we have carefully evaluated the environmental health effects of exposure to O₃ pollution among children. Discussions of the results of the evaluation of the scientific evidence, policy considerations, and the exposure and risk assessments pertaining to children are contained in sections II.B and II.C of this preamble.

H. Executive Order 13211: Actions That Significantly Affect Energy Supply, Distribution, or Use

This action is not a “significant energy action” because it is not likely to have a significant adverse effect on the supply, distribution, or use of energy. The purpose of this rule is to establish revised NAAQS for O₃, establish an additional FRM, revise FEM procedures for testing, and revises air quality surveillance requirements. The rule does not prescribe specific pollution control strategies by which these ambient standards and monitoring revisions will be met. Such strategies will be developed by states on a case-by-case basis, and the EPA cannot predict whether the control options selected by states will include regulations on energy suppliers, distributors, or users. Thus, the EPA concludes that this rule is not likely to have any adverse energy effects and does not constitute a significant energy action as defined in Executive Order 13211.

I. National Technology Transfer and Advancement Act

This rulemaking involves environmental monitoring and measurement. Consistent with the Agency’s Performance Based

Measurement System (PBMS), the EPA is not requiring the use of specific, prescribed analytical methods. Rather, the Agency is allowing the use of any method that meets the prescribed performance criteria. Ambient air concentrations of O₃ are currently measured by the FRM in 40 CFR part 50, Appendix D (Measurement Principle and Calibration Procedure for the Measurement of Ozone in the Atmosphere) or by FEM that meet the requirements of 40 CFR part 53. Procedures are available in part 53 that allow for the approval of an FEM for O₃ that is similar to the FRM. Any method that meets the performance criteria for a candidate equivalent method may be approved for use as an FEM. This approach is consistent with EPA’s PBMS. The PBMS approach is intended to be more flexible and cost-effective for the regulated community; it is also intended to encourage innovation in analytical technology and improved data quality. The EPA is not precluding the use of any method, whether it constitutes a voluntary consensus standard or not, as long as it meets the specified performance criteria.

J. Executive Order 12898: Federal Actions To Address Environmental Justice in Minority Populations and Low-Income Populations

The EPA believes that this action will not have disproportionately high and adverse human health or environmental effects on minority populations, low-income populations or indigenous peoples. The action described in this notice is to strengthen the NAAQS for O₃.

The primary NAAQS are established at a level that is requisite to protect public health, including the health of sensitive or at-risk groups, with an adequate margin of safety. The NAAQS decisions are based on an explicit and comprehensive assessment of the current scientific evidence and associated exposure/risk analyses. More specifically, EPA expressly considers the available information regarding health effects among at-risk populations, including that available for low-income populations and minority populations, in decisions on NAAQS. Where low-income populations or minority populations are among the at-risk populations, the decision on the standard is based on providing protection for these and other at-risk populations and lifestages. Where such populations are not identified as at-risk populations, a NAAQS that is established to provide protection to the at-risk populations would also be expected to provide protection to all

other populations, including low-income populations and minority populations.

The ISA, HREA, and PA for this review, which include identification of populations at risk from O₃ health effects, are available in the docket, EPA–HQ–OAR–2008–0699. The information on at-risk populations for this NAAQS review is summarized and considered earlier in this preamble (see section II.A). This final rule increases the level of environmental protection for all affected populations without having any disproportionately high and adverse human health or environmental effects on any population, including any minority populations, low-income populations or indigenous peoples. This rule establishes uniform national standards for O₃ in ambient air that, in the Administrator’s judgment, protect public health, including the health of sensitive groups, with an adequate margin of safety.

Although it is part of a separate docket (EPA–HQ–OAR–2013–0169) and is not part of the rulemaking record for this action, EPA has prepared a RIA of this decision. As part of the RIA, a demographic analysis was conducted. While, as noted in the RIA, the demographic analysis is not a full quantitative, site-specific exposure and risk assessment, that analysis examined demographic characteristics of persons living in areas with poor air quality relative to the proposed standard. Specifically, Chapter 9, section 9.10 (page 9–7) and Appendix 9A of the RIA describe this proximity and socio-demographic analysis. This analysis found that in areas with poor air quality relative to the revised standard,²⁵⁷ the representation of minority populations was slightly greater than in the U.S. as a whole. Because the air quality in these areas does not currently meet the revised standard, populations in these areas would be expected to benefit from implementation of the strengthened standard, and, thus, would be more affected by strategies to attain the revised standard. This analysis, which evaluates the potential implications for minority populations and low-income populations of future air pollution control actions that state and local agencies may consider in implementing the revised O₃ NAAQS described in this decision notice are discussed in Appendix 9A of the RIA. The RIA is available on the Web, through the EPA’s Technology Transfer Network Web site at http://www.epa.gov/ttn/naaqs/standards/ozone/s_o3_index.html and

²⁵⁷ This refers to monitored areas with O₃ design values above the revised and alternative standards.

²⁵⁶ As used here and similarly throughout this document, the term population refers to people having a quality or characteristic in common, including a specific pre-existing illness or a specific age or lifestage.

in the RIA docket (EPA–HQ–OAR–2013–0169). As noted above, although an RIA has been prepared, the results of the RIA have not been considered in issuing this final rule.

K. Congressional Review Act (CRA)

This action is subject to the CRA, and the EPA will submit a rule report to each House of the Congress and to the Comptroller General of the United States. This action is a “major rule” as defined by 5 U.S.C. 804(2).

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List of Subjects

40 CFR Part 50

Environmental protection, Air pollution control, Carbon monoxide, Lead, Nitrogen dioxide, Ozone, Particulate matter, Sulfur oxides.

40 CFR Part 51

Environmental protection, Administrative practices and

procedures, Air pollution control, Intergovernmental relations.

40 CFR Part 52

Environmental Protection, Administrative practices and procedures, Air pollution control, Incorporation by reference, Intergovernmental relations.

40 CFR Part 53

Environmental protection, Administrative practice and procedure, Air pollution control, Reporting and recordkeeping requirements.

40 CFR Part 58

Environmental protection, Administrative practice and procedure, Air pollution control, Intergovernmental relations, Reporting and recordkeeping requirements.

Dated: October 1, 2015.

Gina McCarthy,
 Administrator.

For the reasons set forth in the preamble, chapter I of title 40 of the Code of Federal Regulations is amended as follows:

PART 50—NATIONAL PRIMARY AND SECONDARY AMBIENT AIR QUALITY STANDARDS

■ 1. The authority citation for part 50 continues to read as follows:

Authority: 42 U.S.C. 7401 *et seq.*

■ 2. Amend § 50.14 by:

■ a. Revising paragraphs (c)(2)(iii) and (vi) and (c)(3)(i); and

■ b. Removing and reserving paragraphs (c)(2)(iv) and (v) and (c)(3)(ii) and (iii).

The revisions read as follows:

§ 50.14 Treatment of air quality monitoring data influenced by exceptional events.

* * * * *

(c) * * *

(2) * * *

(iii) Flags placed on data as being due to an exceptional event together with an initial description of the event shall be submitted to EPA not later than July 1st of the calendar year following the year in which the flagged measurement occurred, except as allowed under paragraph (c)(2)(vi) of this section.

* * * * *

(vi) Table 1 identifies the data submission process for a new or revised NAAQS. This process shall apply to those data that will or may influence the initial designation of areas for any new or revised NAAQS.

TABLE 1—SCHEDULE FOR FLAGGING AND DOCUMENTATION SUBMISSION FOR DATA INFLUENCED BY EXCEPTIONAL EVENTS FOR USE IN INITIAL AREA DESIGNATIONS

Exceptional events/regulatory action	Exceptional events deadline schedule ^d
Flagging and initial event description deadline for data years 1, 2 and 3. ^a	If state and tribal initial designation recommendations for a new/revised NAAQS are due August through January, then the flagging and initial event description deadline will be the July 1 prior to the recommendation deadline. If state and tribal recommendations for a new/revised NAAQS are due February through July, then the flagging and initial event description deadline will be the January 1 prior to the recommendation deadline.
Exceptional events demonstration submittal deadline for data years 1, 2 and 3. ^a	No later than the date that state and tribal recommendations are due to EPA.
Flagging, initial event description and exceptional events demonstration submittal deadline for data year 4 ^b and, where applicable, data year 5. ^c	By the last day of the month that is 1 year and 7 months after promulgation of a new/revised NAAQS, unless either option a or b applies. a. If the EPA follows a 3-year designation schedule, the deadline is 2 years and 7 months after promulgation of a new/revised NAAQS. b. If the EPA notifies the state/tribe that it intends to complete the initial area designations process according to a schedule between 2 and 3 years, the deadline is 5 months prior to the date specified for final designations decisions in such EPA notification.

^a Where data years 1, 2, and 3 are those years expected to be considered in state and tribal recommendations.
^b Where data year 4 is the additional year of data that the EPA may consider when it makes final area designations for a new/revised NAAQS under the standard designations schedule.
^c Where data year 5 is the additional year of data that the EPA may consider when it makes final area designations for a new/revised NAAQS under an extended designations schedule.
^d The date by which air agencies must certify their ambient air quality monitoring data in AQS is annually on May 1 of the year following the year of data collection as specified in 40 CFR 58.15(a)(2). In some cases, however, air agencies may choose to certify a prior year's data in advance of May 1 of the following year, particularly if the EPA has indicated its intent to promulgate final designations in the first 8 months of the calendar year. Data flagging, initial event description and exceptional events demonstration deadlines for "early certified" data will follow the deadlines for "year 4" and "year 5" data.

(3) *Submission of demonstrations.* (i) Except as allowed under paragraph (c)(2)(vi) of this section, a State that has flagged data as being due to an exceptional event and is requesting exclusion of the affected measurement data shall, after notice and opportunity for public comment, submit a demonstration to justify data exclusion to EPA not later than the lesser of 3 years following the end of the calendar quarter in which the flagged concentration was recorded or 12 months prior to the date that a regulatory decision must be made by

EPA. A State must submit the public comments it received along with its demonstration to EPA.
 * * * * *

■ 3. Section 50.19 is added to read as follows:

§ 50.19 National primary and secondary ambient air quality standards for ozone.

(a) The level of the national 8-hour primary ambient air quality standard for ozone (O₃) is 0.070 parts per million (ppm), daily maximum 8-hour average, measured by a reference method based on appendix D to this part and

designated in accordance with part 53 of this chapter or an equivalent method designated in accordance with part 53 of this chapter.

(b) The 8-hour primary O₃ ambient air quality standard is met at an ambient air quality monitoring site when the 3-year average of the annual fourth-highest daily maximum 8-hour average O₃ concentration is less than or equal to 0.070 ppm, as determined in accordance with appendix U to this part.

(c) The level of the national secondary ambient air quality standard for O₃ is 0.070 ppm, daily maximum 8-hour

average, measured by a reference method based on appendix D to this part and designated in accordance with part 53 of this chapter or an equivalent method designated in accordance with part 53 of this chapter.

(d) The 8-hour secondary O₃ ambient air quality standard is met at an ambient air quality monitoring site when the 3-year average of the annual fourth-highest daily maximum 8-hour average O₃ concentration is less than or equal to 0.070 ppm, as determined in accordance with appendix U to this part.

■ 4. Revise appendix D to part 50 to read as follows:

Appendix D to Part 50—Reference Measurement Principle and Calibration Procedure for the Measurement of Ozone in the Atmosphere (Chemiluminescence Method)

1.0 Applicability.

1.1 This chemiluminescence method provides reference measurements of the concentration of ozone (O₃) in ambient air for determining compliance with the national primary and secondary ambient air quality standards for O₃ as specified in 40 CFR part 50. This automated method is applicable to the measurement of ambient O₃ concentrations using continuous (real-time) sampling and analysis. Additional quality assurance procedures and guidance are provided in 40 CFR part 58, appendix A, and in Reference 14.

2.0 Measurement Principle.

2.1 This reference method is based on continuous automated measurement of the intensity of the characteristic chemiluminescence released by the gas phase reaction of O₃ in sampled air with either ethylene (C₂H₄) or nitric oxide (NO) gas. An ambient air sample stream and a specific flowing concentration of either C₂H₄ (ET-CL method) or NO (NO-CL method) are mixed in a measurement cell, where the resulting chemiluminescence is quantitatively

measured by a sensitive photo-detector. References 8–11 describe the chemiluminescence measurement principle.

2.2 The measurement system is calibrated by referencing the instrumental chemiluminescence measurements to certified O₃ standard concentrations generated in a dynamic flow system and assayed by photometry to be traceable to a National Institute of Standards and Technology (NIST) standard reference photometer for O₃ (see Section 4, Calibration Procedure, below).

2.3 An analyzer implementing this measurement principle is shown schematically in Figure 1. Designs implementing this measurement principle must include: an appropriately designed mixing and measurement cell; a suitable quantitative photometric measurement system with adequate sensitivity and wavelength specificity for O₃; a pump, flow control, and sample conditioning system for sampling the ambient air and moving it into and through the measurement cell; a sample air dryer as necessary to meet the water vapor interference limit requirement specified in subpart B of part 53 of this chapter; a means to supply, meter, and mix a constant, flowing stream of either C₂H₄ or NO gas of fixed concentration with the sample air flow in the measurement cell; suitable electronic control and measurement processing capability; and other associated apparatus as may be necessary. The analyzer must be designed and constructed to provide accurate, repeatable, and continuous measurements of O₃ concentrations in ambient air, with measurement performance that meets the requirements specified in subpart B of part 53 of this chapter.

2.4 An analyzer implementing this measurement principle and calibration procedure will be considered a federal reference method (FRM) only if it has been designated as a reference method in accordance with part 53 of this chapter.

2.5 *Sampling considerations.* The use of a particle filter on the sample inlet line of a chemiluminescence O₃ FRM analyzer is required to prevent buildup of particulate

matter in the measurement cell and inlet components. This filter must be changed weekly (or at least often as specified in the manufacturer's operation/instruction manual), and the sample inlet system used with the analyzer must be kept clean, to avoid loss of O₃ in the O₃ sample air prior to the concentration measurement.

3.0 Interferences.

3.1 Except as described in 3.2 below, the chemiluminescence measurement system is inherently free of significant interferences from other pollutant substances that may be present in ambient air.

3.2 A small sensitivity to variations in the humidity of the sample air is minimized by a sample air dryer. Potential loss of O₃ in the inlet air filter and in the air sample handling components of the analyzer and associated exterior air sampling components due to buildup of airborne particulate matter is minimized by filter replacement and cleaning of the other inlet components.

4.0 Calibration Procedure.

4.1 *Principle.* The calibration procedure is based on the photometric assay of O₃ concentrations in a dynamic flow system. The concentration of O₃ in an absorption cell is determined from a measurement of the amount of 254 nm light absorbed by the sample. This determination requires knowledge of (1) the absorption coefficient (α) of O₃ at 254 nm, (2) the optical path length (l) through the sample, (3) the transmittance of the sample at a nominal wavelength of 254 nm, and (4) the temperature (T) and pressure (P) of the sample. The transmittance is defined as the ratio I/I₀, where I is the intensity of light which passes through the cell and is sensed by the detector when the cell contains an O₃ sample, and I₀ is the intensity of light which passes through the cell and is sensed by the detector when the cell contains zero air. It is assumed that all conditions of the system, except for the contents of the absorption cell, are identical during measurement of I and I₀. The quantities defined above are related by the Beer-Lambert absorption law,

$$\text{Transmittance} = \frac{I}{I_0} = e^{-\alpha cl} \tag{1}$$

Where:

α = absorption coefficient of O₃ at 254 nm = 308 ± 4 atm⁻¹ cm⁻¹ at 0 °C and 760 torr,^{1, 2, 3, 4, 5, 6, 7}

c = O₃ concentration in atmospheres, and
 l = optical path length in cm.

A stable O₃ generator is used to produce O₃ concentrations over the required calibration

concentration range. Each O₃ concentration is determined from the measurement of the transmittance (I/I₀) of the sample at 254 nm with a photometer of path length l and calculated from the equation,

$$c(\text{atm}) = -\frac{1}{\alpha l} \left(\ln \frac{I}{I_0} \right) \tag{2a}$$

or

$$c(\text{ppm}) = -\frac{10^6}{\alpha l} \left(\ln \frac{I}{I_0} \right). \tag{2b}$$

The calculated O₃ concentrations must be corrected for O₃ losses, which may occur in the photometer, and for the temperature and pressure of the sample.

4.2 *Applicability.* This procedure is applicable to the calibration of ambient air O₃ analyzers, either directly or by means of a transfer standard certified by this procedure. Transfer standards must meet the requirements and specifications set forth in Reference 12.

4.3 *Apparatus.* A complete UV calibration system consists of an O₃ generator, an output port or manifold, a photometer, an appropriate source of zero air, and other components as necessary. The configuration must provide a stable O₃ concentration at the system output and allow the photometer to accurately assay the output concentration to the precision specified for the photometer (4.3.1). Figure 2 shows a commonly used configuration and serves to illustrate the calibration procedure, which follows. Other configurations may require appropriate variations in the procedural steps. All connections between components in the calibration system downstream of the O₃ generator must be of glass, Teflon, or other relatively inert materials. Additional information regarding the assembly of a UV photometric calibration apparatus is given in Reference 13. For certification of transfer standards which provide their own source of O₃, the transfer standard may replace the O₃ generator and possibly other components shown in Figure 2; see Reference 12 for guidance.

4.3.1 *UV photometer.* The photometer consists of a low-pressure mercury discharge lamp, (optional) collimation optics, an absorption cell, a detector, and signal-processing electronics, as illustrated in Figure 2. It must be capable of measuring the transmittance, I/I₀, at a wavelength of 254 nm with sufficient precision such that the standard deviation of the concentration measurements does not exceed the greater of 0.005 ppm or 3% of the concentration. Because the low-pressure mercury lamp radiates at several wavelengths, the photometer must incorporate suitable means to assure that no O₃ is generated in the cell by the lamp, and that at least 99.5% of the radiation sensed by the detector is 254 nm

radiation. (This can be readily achieved by prudent selection of optical filter and detector response characteristics.) The length of the light path through the absorption cell must be known with an accuracy of at least 99.5%. In addition, the cell and associated plumbing must be designed to minimize loss of O₃ from contact with cell walls and gas handling components. See Reference 13 for additional information.

4.3.2 *Air flow controllers.* Air flow controllers are devices capable of regulating air flows as necessary to meet the output stability and photometer precision requirements.

4.3.3 *Ozone generator.* The ozone generator used must be capable of generating stable levels of O₃ over the required concentration range.

4.3.4 *Output manifold.* The output manifold must be constructed of glass, Teflon, or other relatively inert material, and should be of sufficient diameter to insure a negligible pressure drop at the photometer connection and other output ports. The system must have a vent designed to insure atmospheric pressure in the manifold and to prevent ambient air from entering the manifold.

4.3.5 *Two-way valve.* A manual or automatic two-way valve, or other means is used to switch the photometer flow between zero air and the O₃ concentration.

4.3.6 *Temperature indicator.* A device to indicate temperature must be used that is accurate to ±1 °C.

4.3.7 *Barometer or pressure indicator.* A device to indicate barometric pressure must be used that is accurate to ±2 torr.

4.4 *Reagents.*

4.4.1 *Zero air.* The zero air must be free of contaminants which would cause a detectable response from the O₃ analyzer, and it must be free of NO, C₂H₄, and other species which react with O₃. A procedure for generating suitable zero air is given in Reference 13. As shown in Figure 2, the zero air supplied to the photometer cell for the I₀ reference measurement must be derived from the same source as the zero air used for generation of the O₃ concentration to be assayed (I measurement). When using the photometer to certify a transfer standard

having its own source of O₃, see Reference 12 for guidance on meeting this requirement.

4.5 *Procedure.*

4.5.1 *General operation.* The calibration photometer must be dedicated exclusively to use as a calibration standard. It must always be used with clean, filtered calibration gases, and never used for ambient air sampling. A number of advantages are realized by locating the calibration photometer in a clean laboratory where it can be stationary, protected from the physical shock of transportation, operated by a responsible analyst, and used as a common standard for all field calibrations via transfer standards.

4.5.2 *Preparation.* Proper operation of the photometer is of critical importance to the accuracy of this procedure. Upon initial operation of the photometer, the following steps must be carried out with all quantitative results or indications recorded in a chronological record, either in tabular form or plotted on a graphical chart. As the performance and stability record of the photometer is established, the frequency of these steps may be reduced to be consistent with the documented stability of the photometer and the guidance provided in Reference 12.

4.5.2.1 *Instruction manual.* Carry out all set up and adjustment procedures or checks as described in the operation or instruction manual associated with the photometer.

4.5.2.2 *System check.* Check the photometer system for integrity, leaks, cleanliness, proper flow rates, etc. Service or replace filters and zero air scrubbers or other consumable materials, as necessary.

4.5.2.3 *Linearity.* Verify that the photometer manufacturer has adequately established that the linearity error of the photometer is less than 3%, or test the linearity by dilution as follows: Generate and assay an O₃ concentration near the upper range limit of the system or appropriate calibration scale for the instrument, then accurately dilute that concentration with zero air and re-assay it. Repeat at several different dilution ratios. Compare the assay of the original concentration with the assay of the diluted concentration divided by the dilution ratio, as follows

$$E = \frac{A_1 - A_2/R}{A_1} \times 100\% \quad (3)$$

Where:

E = linearity error, percent

A₁ = assay of the original concentration

A₂ = assay of the diluted concentration

R = dilution ratio = flow of original concentration divided by the total flow

The linearity error must be less than 5%. Since the accuracy of the measured flow-rates will affect the linearity error as measured this way, the test is not necessarily conclusive. Additional information on verifying linearity is contained in Reference 13.

4.5.2.4 *Inter-comparison.* The photometer must be inter-compared annually, either directly or via transfer standards, with a

NIST standard reference photometer (SRP) or calibration photometers used by other agencies or laboratories.

4.5.2.5 *Ozone losses.* Some portion of the O₃ may be lost upon contact with the photometer cell walls and gas handling components. The magnitude of this loss must be determined and used to correct the calculated O₃ concentration. This loss must not exceed 5%. Some guidelines for quantitatively determining this loss are discussed in Reference 13.

4.5.3 *Assay of O₃ concentrations.* The operator must carry out the following steps to properly assay O₃ concentrations.

4.5.3.1 Allow the photometer system to warm up and stabilize.

4.5.3.2 Verify that the flow rate through the photometer absorption cell, F, allows the cell to be flushed in a reasonably short period of time (2 liter/min is a typical flow). The precision of the measurements is inversely related to the time required for flushing, since the photometer drift error increases with time.

4.5.3.3 Ensure that the flow rate into the output manifold is at least 1 liter/min greater than the total flow rate required by the photometer and any other flow demand connected to the manifold.

4.5.3.4 Ensure that the flow rate of zero air, Fz, is at least 1 liter/min greater than the flow rate required by the photometer.

4.5.3.5 With zero air flowing in the output manifold, actuate the two-way valve to allow the photometer to sample first the manifold zero air, then Fz. The two photometer readings must be equal ($I = I_0$).

Note: In some commercially available photometers, the operation of the two-way valve and various other operations in section

4.5.3 may be carried out automatically by the photometer.

4.5.3.6 Adjust the O₃ generator to produce an O₃ concentration as needed.

4.5.3.7 Actuate the two-way valve to allow the photometer to sample zero air until the absorption cell is thoroughly flushed and record the stable measured value of I₀.

4.5.3.8 Actuate the two-way valve to allow the photometer to sample the O₃ concentration until the absorption cell is

thoroughly flushed and record the stable measured value of I.

4.5.3.9 Record the temperature and pressure of the sample in the photometer absorption cell. (See Reference 13 for guidance.)

4.5.3.10 Calculate the O₃ concentration from equation 4. An average of several determinations will provide better precision.

$$[O_3]_{OUT} = \left(\frac{-1}{\alpha l}\right) \ln \left(\frac{I}{I_0}\right) \left(\frac{T}{273}\right) \left(\frac{760}{P}\right) \times \frac{10^6}{L} \quad (4)$$

Where:

[O₃]_{OUT} = O₃ concentration, ppm

α = absorption coefficient of O₃ at 254 nm = 308 atm – 1 cm – 1 at 0° C and 760 torr

l = optical path length, cm

T = sample temperature, K

P = sample pressure, torr

L = correction factor for O₃ losses from

4.5.2.5 = (1 – fraction of O₃ lost).

Note: Some commercial photometers may automatically evaluate all or part of equation 4. It is the operator's responsibility to verify that all of the information required for equation 4 is obtained, either automatically by the photometer or manually. For "automatic" photometers which evaluate the first term of equation 4 based on a linear approximation, a manual correction may be required, particularly at higher O₃ levels. See the photometer instruction manual and Reference 13 for guidance.

4.5.3.11 Obtain additional O₃ concentration standards as necessary by repeating steps 4.5.3.6 to 4.5.3.10 or by Option 1.

4.5.4 *Certification of transfer standards.* A transfer standard is certified by relating the output of the transfer standard to one or more O₃ calibration standards as determined according to section 4.5.3. The exact procedure varies depending on the nature

and design of the transfer standard. Consult Reference 12 for guidance.

4.5.5 *Calibration of ozone analyzers.* Ozone analyzers must be calibrated as follows, using O₃ standards obtained directly according to section 4.5.3 or by means of a certified transfer standard.

4.5.5.1 Allow sufficient time for the O₃ analyzer and the photometer or transfer standard to warm-up and stabilize.

4.5.5.2 Allow the O₃ analyzer to sample zero air until a stable response is obtained and then adjust the O₃ analyzer's zero control. Offsetting the analyzer's zero adjustment to +5% of scale is recommended to facilitate observing negative zero drift (if any). Record the stable zero air response as "Z".

4.5.5.3 Generate an O₃ concentration standard of approximately 80% of the desired upper range limit (URL) of the O₃ analyzer. Allow the O₃ analyzer to sample this O₃ concentration standard until a stable response is obtained.

4.5.5.4 Adjust the O₃ analyzer's span control to obtain the desired response equivalent to the calculated standard concentration. Record the O₃ concentration and the corresponding analyzer response. If substantial adjustment of the span control is necessary, recheck the zero and span adjustments by repeating steps 4.5.5.2 to 4.5.5.4.

4.5.5.5 Generate additional O₃ concentration standards (a minimum of 5 are recommended) over the calibration scale of the O₃ analyzer by adjusting the O₃ source or by Option 1. For each O₃ concentration standard, record the O₃ concentration and the corresponding analyzer response.

4.5.5.6 Plot the O₃ analyzer responses (vertical or Y-axis) versus the corresponding O₃ standard concentrations (horizontal or X-axis). Compute the linear regression slope and intercept and plot the regression line to verify that no point deviates from this line by more than 2 percent of the maximum concentration tested.

4.5.5.7 *Option 1:* The various O₃ concentrations required in steps 4.5.3.11 and 4.5.5.5 may be obtained by dilution of the O₃ concentration generated in steps 4.5.3.6 and 4.5.5.3. With this option, accurate flow measurements are required. The dynamic calibration system may be modified as shown in Figure 3 to allow for dilution air to be metered in downstream of the O₃ generator. A mixing chamber between the O₃ generator and the output manifold is also required. The flow rate through the O₃ generator (F_O) and the dilution air flow rate (F_D) are measured with a flow or volume standard that is traceable to a NIST flow or volume calibration standard. Each O₃ concentration generated by dilution is calculated from:

$$[O_3]'_{OUT} = [O_3]_{OUT} \left(\frac{F_O}{F_O + F_D}\right) \quad (5)$$

Where:

[O₃]'_{OUT} = diluted O₃ concentration, ppm
 FO = flow rate through the O₃ generator, liter/min

FD = diluent air flow rate, liter/min

Note: Additional information on calibration and pollutant standards is provided in Section 12 of Reference 14.

5.0 Frequency of Calibration.

5.1 The frequency of calibration, as well as the number of points necessary to establish the calibration curve, and the frequency of other performance checking will vary by analyzer; however, the minimum frequency, acceptance criteria, and subsequent actions are specified in Appendix D of Reference 14: Measurement Quality Objectives and Validation Templates. The user's quality control program shall provide guidelines for

initial establishment of these variables and for subsequent alteration as operational experience is accumulated. Manufacturers of analyzers should include in their instruction/operation manuals information and guidance as to these variables and on other matters of operation, calibration, routine maintenance, and quality control.

6.0 References.

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 14. QA Handbook for Air Pollution Measurement Systems—Volume II. Ambient Air Quality Monitoring Program. EPA–454/B–13–003, May 2013. [Available at <http://www.epa.gov/ttnamti1/files/ambient/pm25/qa/QA-Handbook-Vol-II.pdf>].

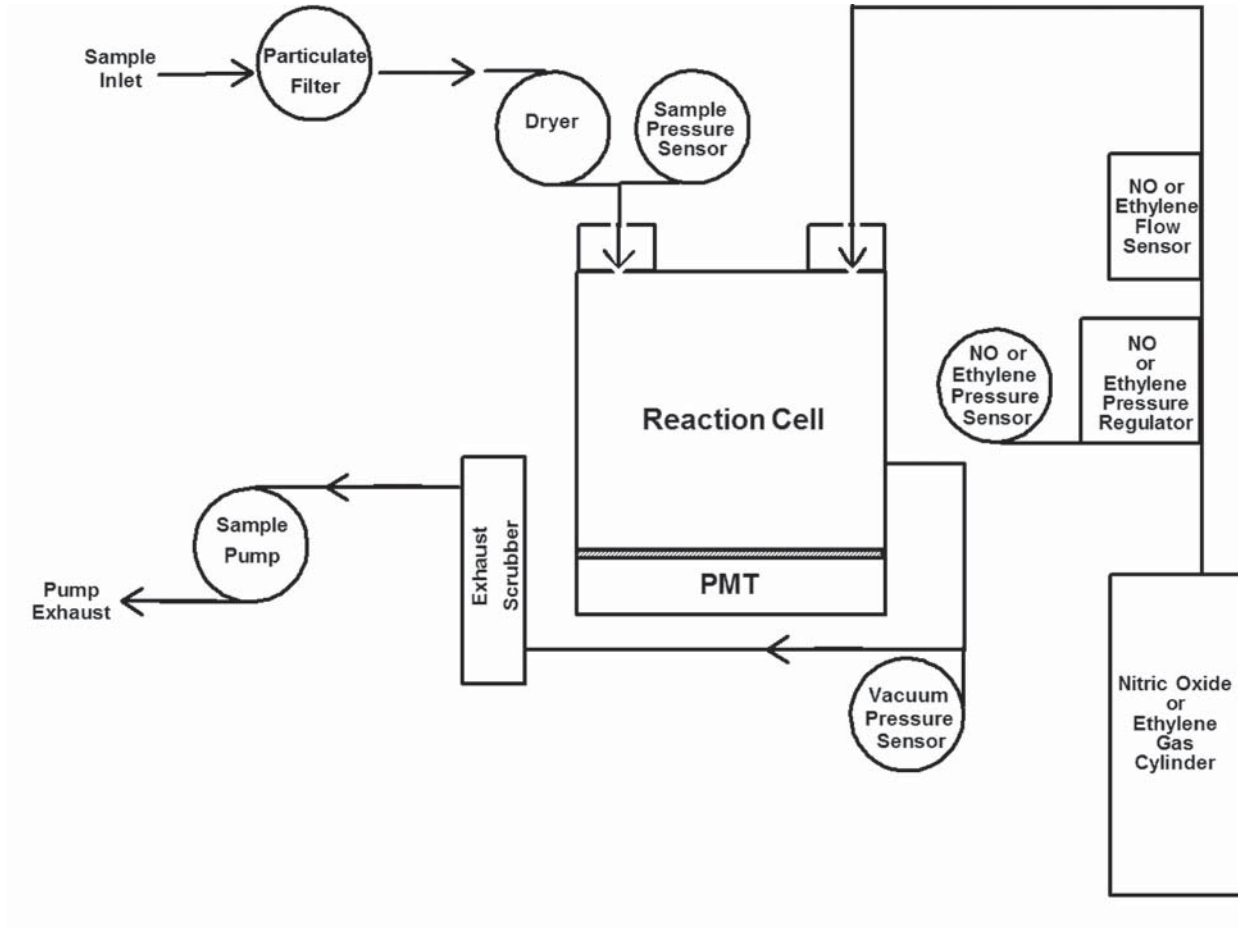


Figure 1. Gas-phase chemiluminescence analyzer schematic diagram, where PMT means photomultiplier tube.

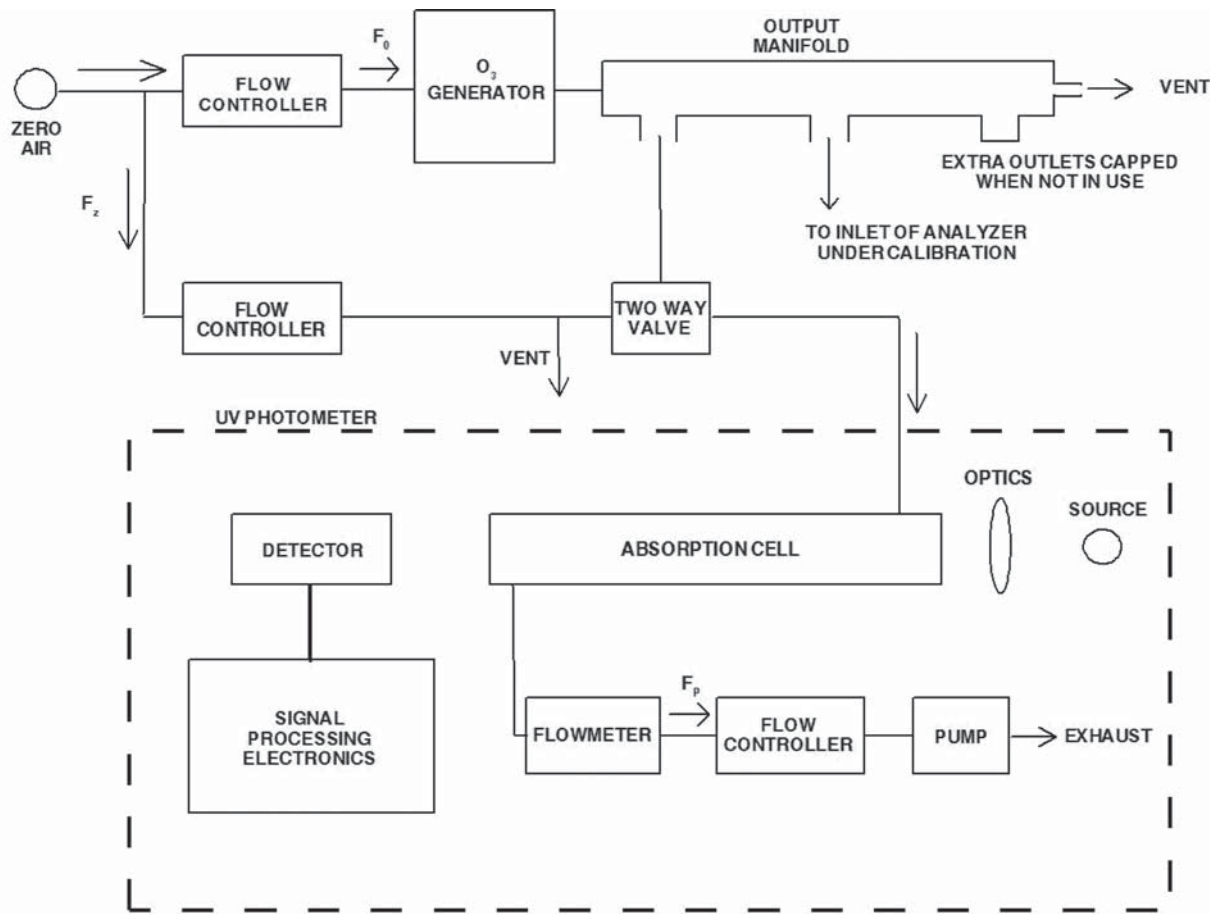


Figure 2. Schematic diagram of a typical UV photometric calibration system.

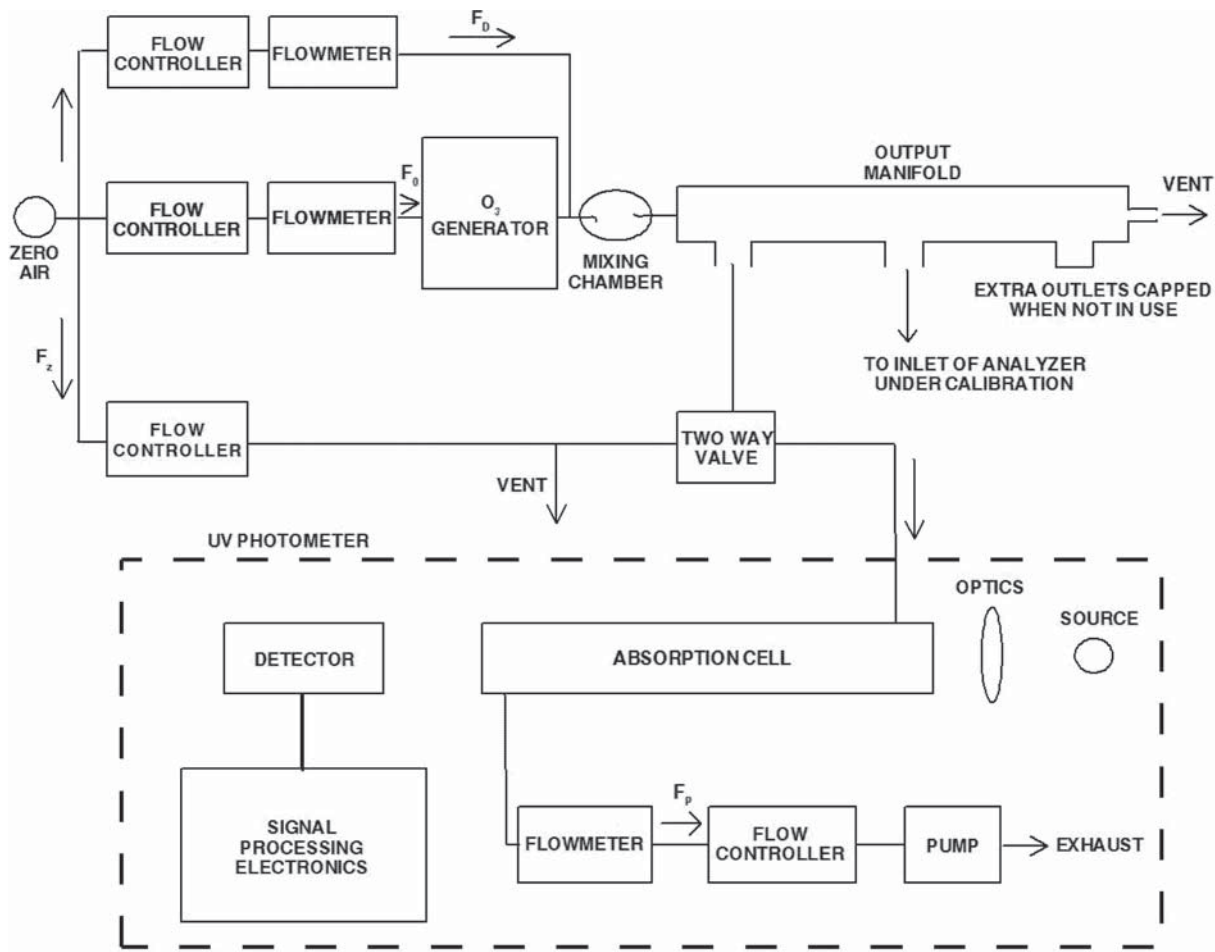


Figure 3. Schematic diagram of a typical UV photometric calibration system (Option 1).

■ 5. Add appendix U to Part 50 to read as follows:

Appendix U to Part 50—Interpretation of the Primary and Secondary National Ambient Air Quality Standards for Ozone

1. General

(a) This appendix explains the data handling conventions and computations necessary for determining whether the primary and secondary national ambient air quality standards (NAAQS) for ozone (O₃) specified in § 50.19 are met at an ambient O₃ air quality monitoring site. Data reporting, data handling, and computation procedures to be used in making comparisons between reported O₃ concentrations and the levels of the O₃ NAAQS are specified in the following sections.

(b) Whether to exclude or retain the data affected by exceptional events is determined by the requirements under §§ 50.1, 50.14 and 51.930.

(c) The terms used in this appendix are defined as follows:

8-hour average refers to the moving average of eight consecutive hourly O₃ concentrations

measured at a site, as explained in section 3 of this appendix.

Annual fourth-highest daily maximum refers to the fourth highest value measured at a site during a year.

Collocated monitors refers to the instance of two or more O₃ monitors operating at the same physical location.

Daily maximum 8-hour average O₃ concentration refers to the maximum calculated 8-hour average value measured at a site on a particular day, as explained in section 3 of this appendix.

Design value refers to the metric (*i.e.*, statistic) that is used to compare ambient O₃ concentration data measured at a site to the NAAQS in order to determine compliance, as explained in section 4 of this appendix.

Minimum data completeness requirements refer to the amount of data that a site is required to collect in order to make a valid determination that the site is meeting the NAAQS.

Monitor refers to a physical instrument used to measure ambient O₃ concentrations.

O₃ monitoring season refers to the span of time within a year when individual states are required to measure ambient O₃ concentrations, as listed in Appendix D to part 58 of this chapter.

Site refers to an ambient O₃ air quality monitoring site.

Site data record refers to the set of hourly O₃ concentration data collected at a site for use in comparisons with the NAAQS.

Year refers to calendar year.

2. Selection of Data for use in Comparisons With the Primary and Secondary Ozone NAAQS

(a) All valid hourly O₃ concentration data collected using a federal reference method specified in Appendix D to this part, or an equivalent method designated in accordance with part 53 of this chapter, meeting all applicable requirements in part 58 of this chapter, and submitted to EPA's Air Quality System (AQS) database or otherwise available to EPA, shall be used in design value calculations.

(b) All design value calculations shall be implemented on a site-level basis. If data are reported to EPA from collocated monitors, those data shall be combined into a single site data record as follows:

(i) The monitoring agency shall designate one monitor as the primary monitor for the site.

(ii) Hourly O₃ concentration data from a secondary monitor shall be substituted into

the site data record whenever a valid hourly O₃ concentration is not obtained from the primary monitor. In the event that hourly O₃ concentration data are available for more than one secondary monitor, the hourly concentration values from the secondary monitors shall be averaged and substituted into the site data record.

(c) In certain circumstances, including but not limited to site closures or relocations, data from two nearby sites may be combined into a single site data record for the purpose of calculating a valid design value. The appropriate Regional Administrator may approve such combinations after taking into consideration factors such as distance between sites, spatial and temporal patterns in air quality, local emissions and meteorology, jurisdictional boundaries, and terrain features.

3. Data Reporting and Data Handling Conventions

(a) Hourly average O₃ concentrations shall be reported in parts per million (ppm) to the third decimal place, with additional digits to the right of the third decimal place truncated. Each hour shall be identified using local standard time (LST).

(b) Moving 8-hour averages shall be computed from the hourly O₃ concentration data for each hour of the year and shall be stored in the first, or start, hour of the 8-hour period. An 8-hour average shall be considered valid if at least 6 of the hourly concentrations for the 8-hour period are available. In the event that only 6 or 7 hourly concentrations are available, the 8-hour average shall be computed on the basis of the hours available, using 6 or 7, respectively, as the divisor. In addition, in the event that 5 or fewer hourly concentrations are available, the 8-hour average shall be considered valid if, after substituting zero for the missing hourly concentrations, the resulting 8-hour average is greater than the level of the

NAAQS, or equivalently, if the sum of the available hourly concentrations is greater than 0.567 ppm. The 8-hour averages shall be reported to three decimal places, with additional digits to the right of the third decimal place truncated. Hourly O₃ concentrations that have been approved under § 50.14 as having been affected by exceptional events shall be counted as missing or unavailable in the calculation of 8-hour averages.

(c) The daily maximum 8-hour average O₃ concentration for a given day is the highest of the 17 consecutive 8-hour averages beginning with the 8-hour period from 7:00 a.m. to 3:00 p.m. and ending with the 8-hour period from 11:00 p.m. to 7:00 a.m. the following day (*i.e.*, the 8-hour averages for 7:00 a.m. to 11:00 p.m.). Daily maximum 8-hour average O₃ concentrations shall be determined for each day with ambient O₃ monitoring data, including days outside the O₃ monitoring season if those data are available.

(d) A daily maximum 8-hour average O₃ concentration shall be considered valid if valid 8-hour averages are available for at least 13 of the 17 consecutive 8-hour periods starting from 7:00 a.m. to 11:00 p.m. In addition, in the event that fewer than 13 valid 8-hour averages are available, a daily maximum 8-hour average O₃ concentration shall also be considered valid if it is greater than the level of the NAAQS. Hourly O₃ concentrations that have been approved under § 50.14 as having been affected by exceptional events shall be included when determining whether these criteria have been met.

(e) The primary and secondary O₃ design value statistic is the annual fourth-highest daily maximum 8-hour O₃ concentration, averaged over three years, expressed in ppm. The fourth-highest daily maximum 8-hour O₃ concentration for each year shall be determined based only on days meeting the

validity criteria in 3(d). The 3-year average shall be computed using the three most recent, consecutive years of ambient O₃ monitoring data. Design values shall be reported in ppm to three decimal places, with additional digits to the right of the third decimal place truncated.

4. Comparisons With the Primary and Secondary Ozone NAAQS

(a) The primary and secondary national ambient air quality standards for O₃ are met at an ambient air quality monitoring site when the 3-year average of the annual fourth-highest daily maximum 8-hour average O₃ concentration (*i.e.*, the design value) is less than or equal to 0.070 ppm.

(b) A design value greater than the level of the NAAQS is always considered to be valid. A design value less than or equal to the level of the NAAQS must meet minimum data completeness requirements in order to be considered valid. These requirements are met for a 3-year period at a site if valid daily maximum 8-hour average O₃ concentrations are available for at least 90% of the days within the O₃ monitoring season, on average, for the 3-year period, with a minimum of at least 75% of the days within the O₃ monitoring season in any one year.

(c) When computing whether the minimum data completeness requirements have been met, meteorological or ambient data may be sufficient to demonstrate that meteorological conditions on missing days were not conducive to concentrations above the level of the NAAQS. Missing days assumed less than the level of the NAAQS are counted for the purpose of meeting the minimum data completeness requirements, subject to the approval of the appropriate Regional Administrator.

(d) Comparisons with the primary and secondary O₃ NAAQS are demonstrated by examples 1 and 2 as follows:

EXAMPLE 1—SITE MEETING THE PRIMARY AND SECONDARY O₃ NAAQS

Year	Percent valid days within O ₃ monitoring season (Data completeness)	1st highest daily max 8-hour O ₃ (ppm)	2nd highest daily max 8-hour O ₃ (ppm)	3rd highest daily max 8-hour O ₃ (ppm)	4th highest daily max 8-hour O ₃ (ppm)	5th highest daily max 8-hour O ₃ (ppm)
2014	100	0.082	0.080	0.075	0.069	0.068
2015	96	0.074	0.073	0.065	0.062	0.060
2016	98	0.070	0.069	0.067	0.066	0.060
Average	98				0.065	

As shown in Example 1, this site meets the primary and secondary O₃ NAAQS because the 3-year average of the annual fourth-highest daily maximum 8-hour average O₃ concentrations (*i.e.*, 0.065666 ppm, truncated

to 0.065 ppm) is less than or equal to 0.070 ppm. The minimum data completeness requirements are also met (*i.e.*, design value is considered valid) because the average percent of days within the O₃ monitoring

season with valid ambient monitoring data is greater than 90%, and no single year has less than 75% data completeness.

EXAMPLE 2—SITE FAILING TO MEET THE PRIMARY AND SECONDARY O₃ NAAQS

Year	Percent valid days within O ₃ monitoring season (Data completeness)	1st highest daily max 8-hour O ₃ (ppm)	2nd highest daily max 8-hour O ₃ (ppm)	3rd highest daily max 8-hour O ₃ (ppm)	4th highest daily max 8-hour O ₃ (ppm)	5th highest daily max 8-hour O ₃ (ppm)
2014	96	0.085	0.080	0.079	0.074	0.072

EXAMPLE 2—SITE FAILING TO MEET THE PRIMARY AND SECONDARY O₃ NAAQS—Continued

Year	Percent valid days within O ₃ monitoring season (Data completeness)	1st highest daily max 8-hour O ₃ (ppm)	2nd highest daily max 8-hour O ₃ (ppm)	3rd highest daily max 8-hour O ₃ (ppm)	4th highest daily max 8-hour O ₃ (ppm)	5th highest daily max 8-hour O ₃ (ppm)
2015	74	0.084	0.083	0.072	0.071	0.068
2016	98	0.083	0.081	0.081	0.075	0.074
Average	89	0.073

As shown in Example 2, this site fails to meet the primary and secondary O₃ NAAQS because the 3-year average of the annual fourth-highest daily maximum 8-hour average O₃ concentrations (*i.e.*, 0.073333 ppm, truncated to 0.073 ppm) is greater than 0.070 ppm, even though the annual data completeness is less than 75% in one year and the 3-year average data completeness is less than 90% (*i.e.*, design value would not otherwise be considered valid).

PART 51—REQUIREMENTS FOR PREPARATION, ADOPTION, AND SUBMITTAL OF IMPLEMENTATION PLANS

■ 6. The authority citation for part 51 continues to read as follows:

Authority: 23 U.S.C. 101; 42 U.S.C. 7401–7671q.

Subpart I—Review of New Sources and Modifications

■ 8. Amend § 51.166 by adding paragraph (i)(11) to read as follows:

§ 51.166 Prevention of significant deterioration of air quality.

(i) * * *
 (11) The plan may provide that the requirements of paragraph (k)(1) of this section shall not apply to a permit application for a stationary source or modification with respect to the revised national ambient air quality standards for ozone published on October 26, 2015 if:

(i) The reviewing authority has determined the permit application subject to this section to be complete on or before October 1, 2015. Instead, the requirements in paragraph (k)(1) of this section shall apply with respect to the national ambient air quality standards for ozone in effect at the time the reviewing authority determined the permit application to be complete; or

(ii) The reviewing authority has first published before December 28, 2015 a public notice of a preliminary determination or draft permit for the permit application subject to this section. Instead, the requirements in

paragraph (k)(1) of this section shall apply with respect to the national ambient air quality standards for ozone in effect at the time of first publication of a public notice of the preliminary determination or draft permit.

* * * * *

PART 52—APPROVAL AND PROMULGATION OF IMPLEMENTATION PLANS

■ 8. The authority citation for part 52 continues to read as follows:

Authority: 42 U.S.C. 7401 *et seq.*

■ 9. Amend § 52.21 by adding paragraph (i)(12) to read as follows:

§ 52.21 Prevention of significant deterioration of air quality.

* * * * *

(i) * * *

(12) The requirements of paragraph (k)(1) of this section shall not apply to a permit application for a stationary source or modification with respect to the revised national ambient air quality standards for ozone published on October 26, 2015 if:

(i) The Administrator has determined the permit application subject to this section to be complete on or before October 1, 2015. Instead, the requirements in paragraph (k)(1) of this section shall apply with respect to the national ambient air quality standards for ozone in effect at the time the Administrator determined the permit application to be complete; or

(ii) The Administrator has first published before December 28, 2015 a public notice of a preliminary determination or draft permit for the permit application subject to this section. Instead, the requirements in paragraph (k)(1) of this section shall apply with respect to the national ambient air quality standards for ozone in effect on the date the Administrator first published a public notice of a preliminary determination or draft permit.

* * * * *

PART 53—AMBIENT AIR MONITORING REFERENCE AND EQUIVALENT METHODS

■ 10. The authority citation for part 53 continues to read as follows:

Authority: Sec. 301(a) of the Clean Air Act (42 U.S.C. 1857g(a)), as amended by sec. 15(c)(2) of Pub. L. 91–604, 84 Stat. 1713, unless otherwise noted.

Subpart A—General Provisions

§ 53.9 [Amended]

■ 11. Amend § 53.9 by removing paragraph (i).

■ 12. Amend § 53.14 by revising paragraph (c) introductory text to read as follows:

§ 53.14 Modification of a reference or equivalent method.

* * * * *

(c) Within 90 calendar days after receiving a report under paragraph (a) of this section, the Administrator will take one or more of the following actions:

* * * * *

Subpart B—Procedures for Testing Performance Characteristics of Automated Methods for SO₂, CO, O₃, and NO₂

■ 13. Amend § 53.23 by revising paragraph (e)(1)(vi) to read as follows:

§ 53.23 Test procedures.

* * * * *

(e) * * *

(1) * * *

(vi) *Precision:* Variation about the mean of repeated measurements of the same pollutant concentration, denoted as the standard deviation expressed as a percentage of the upper range limits.²⁵⁸

* * * * *

■ 14. Revise Table B–1 to Subpart B of Part 53 to read as follows:

²⁵⁸NO₂ precision in Table B–1 is also changed to percent to agree with the calculation specified in 53.23(e)(10)(vi).

TABLE B-1 TO SUBPART B OF PART 53—PERFORMANCE LIMIT SPECIFICATIONS FOR AUTOMATED METHODS

Performance parameter	Units ¹	SO ₂		O ₃		CO		NO ₂ (Std. range)	Definitions and test procedures
		Std. range ³	Lower range ^{2,3}	Std. range ³	Lower range ^{2,3}	Std. range ³	Lower range ^{2,3}		
1. Range	ppm	0-0.5	<0.5	0-0.5	<0.5	0-50	<50	0-0.5	Sec. 53.23(a)
2. Noise	ppm	0.001	0.0005	0.0025	0.001	0.2	0.1	0.005	Sec. 53.23(b)
3. Lower detectable limit	ppm	0.002	0.001	0.005	0.002	0.4	0.2	0.010	Sec. 53.23(c)
4. Interference equivalent Each interferent	ppm	±0.005	4 ±0.005	±0.005	±0.005	±1.0	±0.5	±0.02	Sec. 53.23(d)
Total, all interferents	ppm	—	—	—	—	—	—	0.04	Sec. 53.23(d)
5. Zero drift, 12 and 24 hour.	ppm	±0.004	±0.002	±0.004	±0.002	±0.5	±0.3	±0.02	Sec. 53.23(e)
6. Span drift, 24 hour 20% of upper range limit.	Percent	—	—	—	—	—	—	±20.0	Sec. 53.23(e)
80% of upper range limit.	Percent	±3.0	±3.0	±3.0	±3.0	±2.0	±2.0	±5.0	Sec. 53.23(e)
7. Lag time	Minutes	2	2	2	2	2.0	2.0	20	Sec. 53.23(e)
8. Rise time	Minutes	2	2	2	2	2.0	2.0	15	Sec. 53.23(e)
9. Fall time	Minutes	2	2	2	2	2.0	2.0	15	Sec. 53.23(e)
10. Precision 20% of upper range limit.	Percent ⁵	—	—	—	—	—	—	—	Sec. 53.23(e)
80% of upper range limit.	Percent ⁵	2	2	2	2	1.0	1.0	4	Sec. 53.23(e)
		—	—	—	—	—	—	—	Sec. 53.23(e)
		2	2	2	2	1.0	1.0	6	Sec. 53.23(e)

¹To convert from parts per million (ppm) to µg/m³ at 25 °C and 760 mm Hg, multiply by M/0.02447, where M is the molecular weight of the gas. Percent means percent of the upper measurement range limit.

²Tests for interference equivalent and lag time do not need to be repeated for any lower range provided the test for the standard range shows that the lower range specification (if applicable) is met for each of these test parameters.

³For candidate analyzers having automatic or adaptive time constants or smoothing filters, describe their functional nature, and describe and conduct suitable tests to demonstrate their function aspects and verify that performances for calibration, noise, lag, rise, fall times, and precision are within specifications under all applicable conditions. For candidate analyzers with operator-selectable time constants or smoothing filters, conduct calibration, noise, lag, rise, fall times, and precision tests at the highest and lowest settings that are to be included in the FRM or FEM designation.

⁴For nitric oxide interference for the SO₂ UVF method, interference equivalent is ±0.0003 ppm for the lower range.

⁵Standard deviation expressed as percent of the URL.

Table B-3 to Subpart B of Part 53—Interferent Test Concentration, 1 Parts per Million

Pollutant	Analyzer type	Hydrochloric acid	Ammonia	Hydrogen sulfide	Sulfur dioxide	Nitrogen dioxide	Nitric oxide	Carbon dioxide	Ethylene	Ozone	m-Xylene	Water vapor	Carbon monoxide	Methane	Ethane	Naphthalene
SO ₂	Ultraviolet fluorescence		⁵ 0.1	⁴ 0.1	⁴ 0.14	0.5	0.5			0.5	0.2	20,000				0.05
SO ₂	Flame photometric		0.01	⁴ 0.14	⁴ 0.14			750				³ 20,000	50			
SO ₂	Gas chromatography		0.1	⁴ 0.14	⁴ 0.14			750				³ 20,000	50			
SO ₂	Spectrophotometric -wet chemical (parosanaline)	0.2	0.1	⁴ 0.14	⁴ 0.14	0.5		750		0.5						
SO ₂	Electrochemical	0.2	0.1	⁴ 0.14	⁴ 0.14	0.5			0.2	0.5		³ 20,000				
SO ₂	Conductivity	0.2	0.1	⁴ 0.14	⁴ 0.14	0.5		750								
SO ₂	Spectrophotometric -gas phase, including DOAS			⁴ 0.14	⁴ 0.14	0.5				0.5	0.2					
O ₃	Ethylene chemiluminescence		0.1					750		⁴ 0.08		20,000				
O ₃	NO-chemiluminescence		0.1			0.5		750		⁴ 0.08		20,000				
O ₃	Electrochemical		³ 0.1	0.5		0.5				⁴ 0.08						
O ₃	Spectrophotometric -wet chemical (potassium iodide)		³ 0.1	0.5		0.5				⁴ 0.08						

1. Concentrations of interferences listed must be prepared and controlled to ± 10 percent of the stated value.
2. Analyzer types not listed will be considered by the Administrator as special cases.
3. Do not mix with the pollutant.
4. Concentration of pollutant used for test. These pollutant concentrations must be prepared to ± 10 percent of the stated value.
5. If candidate method utilizes an elevated-temperature scrubber for removal of aromatic hydrocarbons, perform this interference test.
6. If naphthalene test concentration cannot be accurately quantified, remove the scrubber, use a test concentration that causes a full scale response, reattach the scrubber, and evaluate response for interference.

CALCULATION OF ZERO DRIFT, SPAN DRIFT, AND PRECISION

Applicant _____ Date _____
 Analyzer _____ Pollutant _____

TEST PARAMETERS	CALCULATIONS	TEST DAY (n)														
		1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
ZERO DRIFT	12 HOUR $12ZD = C_{max} - C_{min}$															
	$Z = (L_1 + L_2)/2$															
	24 HOUR $24ZD = Z_n - Z_{n-1}$															
	$24ZD = Z'_n - Z'_{n-1}$															
SPAN DRIFT	$S_n = \frac{1}{6} \sum_{i=7}^{12} P_i$															
	24 HOUR $SD_n = \frac{S_n - S_{n-1}}{S_{n-1}} \times 100\%$															
	$SD_n = \frac{S'_n - S'_{n-1}}{S'_{n-1}} \times 100\%$															
PRECISION	20% URL (P_{20}) $P_{20} = \% \text{ STANDARD DEVIATION OF } (P_1...P_6)$															
	80% URL (P_{80}) $P_{80} = \% \text{ STANDARD DEVIATION OF } (P_7...P_{12})$															

Figure B-5. Form for calculating zero drift, span drift, and precision (§ 53.23(e)).

* * * * *

Subpart C—Procedures for Determining Comparability between Candidate Methods and Reference Methods

- 17. Amend § 53.32 by revising paragraph (g)(1)(iii) to read as follows:

§ 53.32 Test procedures for methods for SO₂, CO, O₃, and NO₂.

* * * * *

- (g) * * *
- (1) * * *

(iii) The measurements shall be made in the sequence specified in table C-2 of this subpart.

* * * * *

Figure E-2 to Subpart E of Part 53 [Removed]

- 18. Amend subpart E by removing figure E-2 to subpart E of part 53.

PART 58—AMBIENT AIR QUALITY SURVEILLANCE

- 19. The authority citation for part 58 continues to read as follows:

Authority: 42 U.S.C. 7403, 7405, 7410, 7414, 7601, 7611, 7614, and 7619.

Subpart B—Monitoring Network

- 20. Amend § 58.10 by adding paragraphs (a)(9) through (11) to read as follows:

§ 58.10 Annual monitoring network plan and periodic network assessment.

- (a) * * *

(9) The annual monitoring network plan shall provide for the required O₃ sites to be operating on the first day of the applicable required O₃ monitoring season in effect on January 1, 2017 as listed in Table D-3 of appendix D of this part.

(10) A plan for making Photochemical Assessment Monitoring Stations (PAMS) measurements, if applicable, in accordance with the requirements of appendix D paragraph 5(a) of this part shall be submitted to the EPA Regional Administrator no later than July 1, 2018. The plan shall provide for the required

PAMS measurements to begin by June 1, 2019.

(11) An Enhanced Monitoring Plan for O₃, if applicable, in accordance with the requirements of appendix D paragraph 5(h) of this part shall be submitted to the EPA Regional Administrator no later than October 1, 2019 or two years following the effective date of a designation to a classification of Moderate or above O₃ nonattainment, whichever is later.

* * * * *

- 21. Section § 58.11 is amended by revising paragraph (c) to read as follows:

§ 58.11 Network technical requirements.

* * * * *

(c) State and local governments must follow the network design criteria contained in appendix D to this part in designing and maintaining the SLAMS stations. The final network design and all changes in design are subject to approval of the Regional Administrator. NCore and STN network design and changes are also subject to approval of the Administrator. Changes in SPM stations do not require approvals, but a change in the designation of a monitoring site from SLAMS to SPM requires approval of the Regional Administrator.

* * * * *

- 22. Amend § 58.13 by adding paragraphs (g) and (h) to read as follows:

§ 58.13 Monitoring network completion.

* * * * *

(g) The O₃ monitors required under appendix D, section 4.1 of this part must operate on the first day of the applicable required O₃ monitoring season in effect January 1, 2017.

(h) The Photochemical Assessment Monitoring sites required under 40 CFR part 58 Appendix D, section 5(a) must be physically established and operating under all of the requirements of this part, including the requirements of appendix A, C, D, and E of this part, no later than June 1, 2019.

Subpart F—Air Quality Index Reporting

- 23. Amend § 58.50 by revising paragraph (c) to read as follows:

§ 58.50 Index reporting.

* * * * *

(c) The population of a metropolitan statistical area for purposes of index reporting is the latest available U.S. census population.

Subpart G—Federal Monitoring

- 24. Amend appendix D to part 58, under section 4, by revising section 4.1(i) and table D-3 to appendix D of part 58, and by revising section 5 to read as follows:

Appendix D to part 58—Network Design Criteria for Ambient Air Quality Monitoring

* * * * *

4. Pollutant-Specific Design Criteria for SLAMS Sites

* * * * *

*4.1 * * **

(i) Ozone monitoring is required at SLAMS monitoring sites only during the seasons of the year that are conducive to O₃ formation (*i.e.*, “ozone season”) as described below in Table D-3 of this appendix. These O₃ seasons are also identified in the AQS files on a state-by-state basis. Deviations from the O₃ monitoring season must be approved by the EPA Regional Administrator. These requests will be reviewed by Regional Administrators taking into consideration, at a minimum, the frequency of out-of-season O₃ NAAQS exceedances, as well as occurrences of the Moderate air quality index level, regional consistency, and logistical issues such as site access. Any deviations based on the Regional Administrator’s waiver of requirements must be described in the annual monitoring network plan and updated in AQS. Changes to the O₃ monitoring season requirements in Table D-3 revoke all previously approved Regional Administrator waivers. Requests for monitoring season deviations must be accompanied by relevant supporting information. Information on how to analyze O₃ data to support a change to the O₃ season in support of the 8-hour standard for the entire network in a specific state can be found in reference 8 to this appendix. Ozone monitors at NCore stations are required to be operated year-round (January to December).

TABLE D-3¹ TO APPENDIX D OF PART 58. OZONE MONITORING SEASON BY STATE

State	Begin Month	End Month
Alabama	March	October.
Alaska	April	October.
Arizona	January	December.
Arkansas	March	November.
California	January	December.
Colorado	January	December.
Connecticut	March	September.
Delaware	March	October.
District of Columbia	March	October.

TABLE D-3¹ TO APPENDIX D OF PART 58. OZONE MONITORING SEASON BY STATE—Continued

State	Begin Month	End Month
Florida	January	December.
Georgia	March	October.
Hawaii	January	December.
Idaho	April	September.
Illinois	March	October.
Indiana	March	October.
Iowa	March	October.
Kansas	March	October.
Kentucky	March	October.
Louisiana (Northern) AQCR 019, 022	March	October.
Louisiana (Southern) AQCR 106	January	December.
Maine	April	September.
Maryland	March	October.
Massachusetts	March	September.
Michigan	March	October.
Minnesota	March	October.
Mississippi	March	October.
Missouri	March	October.
Montana	April	September.
Nebraska	March	October.
Nevada	January	December.
New Hampshire	March	September.
New Jersey	March	October.
New Mexico	January	December.
New York	March	October.
North Carolina	March	October.
North Dakota	March	September.
Ohio	March	October.
Oklahoma	March	November.
Oregon	May	September.
Pennsylvania	March	October.
Puerto Rico	January	December.
Rhode Island	March	September.
South Carolina	March	October.
South Dakota	March	October.
Tennessee	March	October.
Texas (Northern) AQCR 022, 210, 211, 212, 215, 217, 218	March	November.
Texas (Southern) AQCR 106, 153, 213, 214, 216	January	December.
Utah	January	December.
Vermont	April	September.
Virginia	March	October.
Washington	May	September.
West Virginia	March	October.
Wisconsin	March	October 15.
Wyoming	January	September.
American Samoa	January	December.
Guam	January	December.
Virgin Islands	January	December.

¹ The required O₃ monitoring season for NCore stations is January through December.

* * * * *

5. Network Design for Photochemical Assessment Monitoring Stations (PAMS) and Enhanced Ozone Monitoring

(a) State and local monitoring agencies are required to collect and report PAMS measurements at each NCore site required under paragraph 3(a) of this appendix located in a CBSA with a population of 1,000,000 or more, based on the latest available census figures.

(b) PAMS measurements include:

- (1) Hourly averaged speciated volatile organic compounds (VOCs);
- (2) Three 8-hour averaged carbonyl samples per day on a 1 in 3 day schedule, or hourly averaged formaldehyde;
- (3) Hourly averaged O₃;

(4) Hourly averaged nitrogen oxide (NO), true nitrogen dioxide (NO₂), and total reactive nitrogen (NO_x);

- (5) Hourly averaged ambient temperature;
- (6) Hourly vector-averaged wind direction;
- (7) Hourly vector-averaged wind speed;
- (8) Hourly average atmospheric pressure;
- (9) Hourly averaged relative humidity;
- (10) Hourly precipitation;
- (11) Hourly averaged mixing-height;
- (12) Hourly averaged solar radiation; and
- (13) Hourly averaged ultraviolet radiation.

(c) The EPA Regional Administrator may grant a waiver to allow the collection of required PAMS measurements at an alternative location where the monitoring agency can demonstrate that the alternative location will provide representative data useful for regional or national scale modeling and the tracking of trends in O₃ precursors.

The alternative location can be outside of the CBSA or outside of the monitoring agencies jurisdiction. In cases where the alternative location crosses jurisdictions the waiver will be contingent on the monitoring agency responsible for the alternative location including the required PAMS measurements in their annual monitoring plan required under § 58.10 and continued successful collection of PAMS measurements at the alternative location. This waiver can be revoked in cases where the Regional Administrator determines the PAMS measurements are not being collected at the alternate location in compliance with paragraph (b) of this section.

(d) The EPA Regional Administrator may grant a waiver to allow speciated VOC measurements to be made as three 8-hour averages on every third day during the PAMS

season as an alternative to 1-hour average speciated VOC measurements in cases where the primary VOC compounds are not well measured using continuous technology due to low detectability of the primary VOC compounds or for logistical and other programmatic constraints.

(e) The EPA Regional Administrator may grant a waiver to allow representative meteorological data from nearby monitoring stations to be used to meet the meteorological requirements in paragraph 5(b) where the monitoring agency can demonstrate the data is collected in a manner consistent with EPA quality assurance requirements for these measurements.

(f) The EPA Regional Administrator may grant a waiver from the requirement to collect PAMS measurements in locations where CBSA-wide O₃ design values are equal to or less than 85% of the 8-hour O₃ NAAQS and where the location is not considered by the Regional Administrator to be an important upwind or downwind location for other O₃ nonattainment areas.

(g) At a minimum, the monitoring agency shall collect the required PAMS measurements during the months of June, July, and August.

(h) States with Moderate and above 8-hour O₃ nonattainment areas and states in the Ozone Transport Region as defined in 40 CFR 51.900 shall develop and implement an Enhanced Monitoring Plan (EMP) detailing enhanced O₃ and O₃ precursor monitoring activities to be performed. The EMP shall be submitted to the EPA Regional Administrator no later than October 1, 2019 or two years following the effective date of a designation to a classification of Moderate or above O₃ nonattainment, whichever is later. At a minimum, the EMP shall be reassessed and approved as part of the 5-year network assessments required under 40 CFR 58.10(d). The EMP will include monitoring activities deemed important to understanding the O₃ problems in the state. Such activities may include, but are not limited to, the following:

(1) Additional O₃ monitors beyond the minimally required under paragraph 4.1 of this appendix,

(2) Additional NO_x or NO_y monitors beyond those required under 4.3 of this appendix,

(3) Additional speciated VOC measurements including data gathered during different periods other than required under paragraph 5(g) of this appendix, or locations other than those required under paragraph 5(a) of this appendix, and

(4) Enhanced upper air measurements of meteorology or pollution concentrations.

* * * * *

■ 25. Appendix G of Part 58 is amended by revising table 2 to read as follows:

Appendix G to Part 58—Uniform Air Quality Index (AQI) and Daily Reporting

* * * * *

TABLE 2—BREAKPOINTS FOR THE AQI

These breakpoints							Equal these AQI's	
O ₃ (ppm) 8-hour	O ₃ (ppm) 1-hour ¹	PM _{2.5} (µg/m ³) 24-hour	PM ₁₀ (µg/m ³) 24-hour	CO (ppm) 8-hour	SO ₂ (ppb) 1-hour	NO ₂ (ppb) 1-hour	AQI	Category
0.000–0.054	—	0.0–12.0	0–54	0.0–4.4	0–35	0–53	0–50	Good.
0.055–0.070	—	12.1–35.4	55–154	4.5–9.4	36–75	54–100	51–100	Moderate.
0.071–0.085	0.125–0.164	35.5–55.4	155–254	9.5–12.4	76–185	101–360	101–150	Unhealthy for Sensitive Groups.
0.086–0.105	0.165–0.204	³ 55.5–150.4	255–354	12.5–15.4	⁴ 186–304	361–649	151–200	Unhealthy.
0.106–0.200	0.205–0.404	³ 150.5–250.4	355–424	15.5–30.4	⁴ 305–604	650–1249	201–300	Very Unhealthy.
0.201– ⁽²⁾	0.405–0.504	³ 250.5–350.4	425–504	30.5–40.4	⁴ 605–804	1250–1649	301–400	Hazardous.
⁽²⁾	0.505–0.604	³ 350.5–500.4	505–604	40.5–50.4	⁴ 805–1004	1650–2049	401–500	

¹ Areas are generally required to report the AQI based on 8-hour ozone values. However, there are a small number of areas where an AQI based on 1-hour ozone values would be more precautionary. In these cases, in addition to calculating the 8-hour ozone index value, the 1-hour ozone index value may be calculated, and the maximum of the two values reported.

² 8-hour O₃ values do not define higher AQI values (>301). AQI values > 301 are calculated with 1-hour O₃ concentrations.

³ If a different SHL for PM_{2.5} is promulgated, these numbers will change accordingly.

⁴ 1-hr SO₂ values do not define higher AQI values (≥200). AQI values of 200 or greater are calculated with 24-hour SO₂ concentration.

Exhibit 12



Regulatory Impact Analysis of the Final Revisions to the National Ambient Air Quality Standards for Ground-Level Ozone

EPA-452/R-15-007
September 2015

**Regulatory Impact Analysis of the Final Revisions
to the National Ambient Air Quality Standards for Ground-Level Ozone**

U.S. Environmental Protection Agency
Office of Air and Radiation
Office of Air Quality Planning and Standards
Research Triangle Park, NC 27711

EXECUTIVE SUMMARY

Overview

In setting primary and secondary national ambient air quality standards (NAAQS), the EPA's responsibility under the law is to establish standards that protect public health and welfare. The Clean Air Act (the Act) requires the EPA, for each criteria pollutant, to set a standard that protects public health with "an adequate margin of safety" and public welfare from "any known or anticipated adverse effects." As interpreted by the Agency and the courts, the Act requires the EPA to base the decision for the primary standard on health considerations only; economic factors cannot be considered. The prohibition against considering cost in the setting of the primary air quality standards does not mean that costs, benefits or other economic considerations are unimportant. The Agency believes that consideration of costs and benefits is an essential decision-making tool for the efficient implementation of these standards. The impacts of costs, benefits, and efficiency are considered by the States when they make decisions regarding what timelines, strategies, and policies are appropriate for their circumstances.

The Administrator concluded that the current primary standard for ozone does not provide requisite protection to public health with an adequate margin of safety, and that it should be revised to provide increased public health protection. Specifically, the EPA is retaining the indicator (ozone), averaging time (8-hour) and form (annual fourth-highest daily maximum, averaged over 3 years) of the existing primary standard and is revising the level of that standard to 70 ppb. The EPA has also concluded that the current secondary standard for ozone, set at a level of 75 ppb, is not requisite to protect public welfare from known or anticipated adverse effects, and is revising the standard to provide increased protection against vegetation-related effects on public welfare. Specifically, the EPA is retaining the indicator (ozone), averaging time (8-hour) and form (annual fourth-highest daily maximum, averaged over 3 years) of the existing secondary standard and is revising the level of that standard to 70 ppb.¹

¹ The EPA has concluded that this revision will effectively curtail cumulative seasonal ozone exposures above 17 ppm-hrs in terms of a three-year average seasonal W126 index value, based on the three consecutive month period within the growing season with the maximum index value, with daily exposures cumulated for the 12-hour period from 8:00 am to 8:00 pm.

The EPA performed an illustrative analysis of the potential costs, human health benefits, and welfare benefits of nationally attaining a revised primary ozone standard of 70 ppb and a primary alternative ozone standard level of 65 ppb. Because there are not additional costs and benefits of attaining the secondary standard, the EPA did not need to estimate any incremental costs and benefits associated with attaining a revised secondary standard. Per Executive Orders 12866 and 13563 and the guidelines of OMB Circular A-4, this Regulatory Impact Analysis (RIA) presents the analyses of the revised standard level of 70 ppb and an alternative standard level of 65 ppb. The cost and benefit estimates below are calculated incremental to a 2025 baseline that incorporates air quality improvements achieved through the projected implementation of existing regulations and full attainment of the existing ozone NAAQS (75 ppb). The 2025 baseline reflects, among other existing regulations, the 2017 and Later Model Year Light-Duty Vehicle Greenhouse Gas Emissions and Corporate Average Fuel Economy Standards, Greenhouse Gas Emissions Standards and Fuel Efficiency Standards for Medium- and Heavy-Duty Engines and Vehicles, the Tier 3 Motor Vehicle Emission and Fuel Standards, the Clean Power Plan, the Mercury and Air Toxics Standards,² and the Cross-State Air Pollution Rule, all of which will help many areas move toward attainment of the existing ozone standard (see Appendix 2, Section 2A.1.3 for additional information).

In this RIA we present the primary costs and benefits estimates for 2025. We assume that potential nonattainment areas everywhere in the U.S., excluding California, will be designated such that they are required to reach attainment by 2025, and we developed our projected baselines for emissions, air quality, populations, and premature mortality baseline rates for 2025. We recognize that there are areas that are not required to meet the existing ozone standard by 2025 -- the Clean Air Act allows areas with more significant air quality problems to take additional time to reach the existing standard. Several areas in California are not required to meet the existing standard by 2025 and may not be required to meet a revised standard until sometime between 2032 and 2037. Because of data and resource constraints, we were not able to project emissions and air quality beyond 2025 for California; however, we adjusted baseline air

² On June 29, 2015, the United States Supreme Court reversed the D.C. Circuit opinion affirming the Mercury and Air Toxics Standards (MATS). The EPA is reviewing the decision and will determine any appropriate next steps once the review is complete, however, MATS is still currently in effect. The first compliance date was April 2015, and many facilities have installed controls for compliance with MATS. MATS is included in the baseline for this analysis, and the EPA does not believe including MATS substantially alters the results of this analysis.

quality to reflect mobile source emissions reductions for California that would occur between 2025 and 2030.³ These emissions reductions were the result of mobile source regulations expected to be fully implemented by 2030.

The EPA will likely finalize designations for a revised ozone NAAQS in late 2017. Depending on the precise timing of the effective date of those designations, nonattainment areas classified as Marginal will likely have to attain in either late 2020 or early 2021. Nonattainment areas classified as Moderate will likely have to attain in either late 2023 or early 2024. If a Moderate nonattainment area qualifies for two 1-year extensions, the area may have as late as early 2026 to attain. Further, Serious nonattainment areas will likely have to attain in late 2026 or early 2027. As such, we selected 2025 as the primary year of analysis because it provided a good representation of the remaining air quality concerns that Moderate nonattainment areas would face and because most areas of the U.S. will likely be required to meet a revised ozone standard by 2025. States with areas classified as Moderate and higher are required to develop attainment demonstration plans for those nonattainment areas.

While there is uncertainty about the precise timing of emissions reductions and related costs for California, we assume costs associated with the installation of controls occur through the end of 2037 and beginning of 2038. In addition, we estimate benefits for California using projected population demographics and baseline mortality rates for 2038. Because of the different timing for incurring costs and accruing benefits and for ease of discussion throughout the analyses, we refer to the different time periods for potential attainment as 2025 and post-2025 to reflect that: (1) we did not project emissions and air quality for any year other than 2025; (2) for California, emissions controls and associated costs are assumed to occur through the end of 2037 and beginning of 2038; and (3) for California benefits are estimated using population demographics and baseline mortality rates for 2038. It is not straightforward to discount the post-2025 results for California to compare with or add to the 2025 results for the rest of the U.S. While we estimate benefits using 2038 information, we do not have good information on precisely when the costs of controls will be incurred. Because of these differences in timing

³ At the time of this analysis, there were no future year emissions for California beyond 2030, and projecting emissions beyond 2030 could introduce additional uncertainty.

related to California attaining a revised standard, the separate costs and benefits estimates for post-2025 should not be added to the primary estimates for 2025.

ES.1 Overview of Analytical Approach

This RIA consists of multiple analyses, including estimates of current and future emissions of relevant precursors (i.e., NO_x and VOC) that contribute to the air quality problem and estimates of current and future ozone concentrations (Chapter 2 – Emissions, Air Quality Modeling and Analytic Methodologies); development of illustrative control strategies to attain the revised standard of 70 ppb and an alternative primary standard level of 65 ppb (Chapter 3 – Control Strategies and Emissions Reductions); estimates of the incremental costs of attaining the revised and alternative standard levels (Chapter 4 – Engineering Cost Analysis and Economic Impacts); a discussion of the theoretical framework used to analyze regulation-induced employment impacts, as well as information on employment related to installation of NO_x controls on coal and gas-fired electric generating units, industrial boilers, and cement kilns (Chapter 5 – Qualitative Discussion of Employment Impacts of Air Quality); estimates of the incremental benefits of attaining the revised and alternative standard levels (Chapter 6 – Human Health Benefits Analysis Approach and Results); a qualitative discussion of the welfare benefits of attaining the revised standards (Chapter 7 – Impacts on Public Welfare of Attainment Strategies to Meeting Primary and Secondary Ozone NAAQS); a comparison and discussion of the benefits and costs (Chapter 8 – Comparison of Costs and Benefits); and an analysis of the impacts in the context of the relevant statutory and executive order requirements (Chapter 9 – Statutory and Executive Order Impact Analyses).

Because States are ultimately responsible for implementing strategies to meet revised standards, this RIA provides insights and analysis of a limited number of illustrative control strategies that states might adopt to meet a revised standard. The goal of this RIA is to provide estimates of the potential costs and benefits of the illustrative attainment strategies to meet the revised and alternative standard levels. The flowchart below (Figure ES-1) outlines the analytical steps taken to illustrate attainment with the revised and alternative standard levels, and the following discussion describes each of the major steps in the process.

ES.2 Results of Benefit-Cost Analysis

Below in Table ES-5, we present the primary costs and benefits estimates for 2025 for all areas except California. We anticipate that benefits and costs will likely begin occurring earlier than 2025, as states begin implementing control measures to show progress towards attainment. In these tables, ranges within the total benefits rows reflect multiple studies upon which the estimates associated with premature mortality were derived. PM_{2.5} co-benefits account for approximately 60 to 70 percent of the estimated benefits, depending on the standard analyzed and on the choice of ozone and PM mortality functions used. Assuming a 7 percent discount rate, for a standard of 70 ppb the total health benefits are comprised of between 29 and 34 percent ozone benefits and between 66 and 71 percent PM_{2.5} co-benefits. Assuming a 7 percent discount rate, for a standard of 65 ppb the total health benefits are comprised of between 29 and 35 percent ozone benefits and between 62 and 70 percent PM_{2.5} co-benefits. In addition for 2025, Table ES-6 presents the numbers of premature deaths avoided for the revised and alternative standard levels analyzed, as well as the other health effects avoided. Table ES-7 provides information on the costs by geographic region for the U.S., except California in 2025, and Table ES-8 provides a regional breakdown of benefits for 2025. See the tables in Chapter 6 for additional characterizations of the monetized benefits.

In the RIA we provide estimates of the costs of emissions reductions to attain the revised and alternative standard levels in three regions -- California, the rest of the western U.S., and the eastern U.S. In addition, we provide estimates of the benefits that accrue to each of these three regions resulting from both control strategies applied within the region and reductions in transport of ozone associated with emissions reductions in other regions.

The net benefits of emissions reductions strategies in a specific region reflect the benefits of the emissions reductions occurring both within and outside of the region minus the costs of the emissions reductions. Because the air quality modeling was conducted at the national level, we do not estimate separately the nationwide benefits associated with the emissions reductions occurring in any specific region.⁷ As a result, we are only able to provide net benefits estimates at the national level. The difference between the costs for a specific region and the benefits

⁷ For California, we provide separate estimates of the costs and nationwide estimates of benefits, so it is appropriate to calculate net benefits. As such, we provide net benefits for the post-2025 analysis for California.

accruing to that region is not an estimate of net benefits of the emissions reductions in that region.

**Table ES-5. Total Annual Costs and Benefits^{a,b} for U.S., except California in 2025
(billions of 2011\$, 7% Discount Rate)^c**

	Revised and Alternative Standard Levels	
	70 ppb	65 ppb
Total Costs^d	\$1.4	\$16
Total Health Benefits	\$2.9 to \$5.9 ^{e, f}	\$15 to \$30 ^{e, f}
Net Benefits	\$1.5 to \$4.5	-\$1.0 to \$14

^a All values are rounded to two significant figures.

^b Benefits are nationwide benefits of attainment everywhere except California.

^c The tables in Chapter 6 provide additional characterizations of the monetized benefits, including benefits estimated at a 3 percent discount rate. Estimating multiple years of costs and benefits is not possible for this RIA due to data and resource limitations. As a result, we provide a snapshot of costs and benefits in 2025, using the best available information to approximate social costs and social benefits recognizing uncertainties and limitations in those estimates.

^d The engineering costs in this table are annualized at a 7 percent discount rate to the extent possible. See Chapter 4 for more discussions.

^e Assuming a 7 percent discount rate, for a standard of 70 ppb the total health benefits are comprised of between 29 and 34 percent ozone benefits and between 66 and 71 percent PM_{2.5} co-benefits. Assuming a 7 percent discount rate, for a standard of 65 ppb the total health benefits are comprised of between 29 and 35 percent ozone benefits and between 62 and 70 percent PM_{2.5} co-benefits.

^f Excludes additional health and welfare benefits that could not be quantified (see Chapter 6, Section 6.6.3.8).

The guidelines of OMB Circular A-4 require providing comparisons of social costs and social benefits at discount rates of 3 and 7 percent. Ideally, streams of social costs and social benefits over time would be estimated and the net present values of each would be compared to determine net benefits of the illustrative attainment strategies. The three different uses of discounting in the RIA – (i) construction of annualized engineering costs, (ii) adjusting the value of mortality risk for lags in mortality risk decreases, and (iii) adjusting the cost of illness for non-fatal heart attacks to adjust for lags in follow up costs -- are all appropriate. Our estimates of net benefits are the approximations of the net value (in 2025) of benefits attributable to emissions reductions needed to attain just for the year 2025.

Table ES-6. Summary of Total Number of Annual Ozone and PM-Related Premature Mortalities and Premature Morbidity: 2025 National Benefits ^a

	Revised and Alternative Standard Levels	
	70 ppb	65 ppb
Ozone-related premature deaths avoided (all ages)	96 to 160	490 to 820
PM_{2.5}-related premature deaths avoided (age 30+)	220 to 500	1,100 to 2,500
Other health effects avoided		
Non-fatal heart attacks (age 18-99) (5 studies) ^{PM}	28 to 260	140 to 1,300
Respiratory hospital admissions (age 0-99) ^{O3, PM}	250	1,200
Cardiovascular hospital admissions (age 18-99) ^{PM}	80	400
Asthma emergency department visits (age 0-99) ^{O3, PM}	630	3,300
Acute bronchitis (age 8-12) ^{PM}	340	1,700
Asthma exacerbation (age 6-18) ^{O3, PM}	230,000	1,100,000
Lost work days (age 18-65) ^{PM}	28,000	140,000
Minor restricted activity days (age 18-65) ^{O3, PM}	620,000	3,100,000
Upper & lower respiratory symptoms (children 7-14) ^{PM}	11,000	53,000
School loss days (age 5-17) ^{O3}	160,000	790,000

^a Nationwide benefits of attainment everywhere except California. All values are rounded to two significant figures. Additional information on confidence intervals are available in the tables in Chapter 6.

Table ES-7. Summary of Total Control Costs (Identified + Unidentified Control Strategies) by Revised and Alternative Standard Levels for 2025 - U.S., except California (billions of 2011\$, 7% Discount Rate)^a

Revised and Alternative Standards Levels	Geographic Area	Total Control Costs (Identified and Unidentified)
70 ppb	East	1.4
	West	<0.05
	Total	\$1.4
65 ppb	East	15
	West	<0.75
	Total	\$16

^a All values are rounded to two significant figures. Costs are annualized at a 7 percent discount rate to the extent possible. Costs associated with unidentified controls are based on an average cost-per-ton methodology (see Chapter 4, Section 4.3 for more discussion on the average-cost methodology).

Table ES-8. Regional Breakdown of Monetized Ozone-Specific Benefits Results for 2025 (Nationwide Benefits of Attaining the Revised and Alternative Standard Levels Everywhere in the U.S., except California) ^a

Region	Revised and Alternative Standard Levels	
	70 ppb	65 ppb
East ^b	98%	96%
California	0%	0%
Rest of West	2%	4%

^a Because we use benefit-per-ton estimates to calculate the PM_{2.5} co-benefits, a regional breakdown for the co-benefits is not available. Therefore, this table only reflects the ozone benefits.

^b Includes Texas and states to the north and east.

To understand possible additional costs and benefits of fully attaining in California in a post-2025 timeframe, we provide separate results for California in Table ES-9. Because of the differences in the timing of achieving needed emissions reductions, incurring costs, and accruing benefits for California, the separate costs and benefits estimates for post-2025 should not be added to the primary estimates for 2025. For the post-2025 timeframe, Table ES-10 presents the numbers of premature deaths avoided for the revised and alternative standard levels analyzed, as well as the other health effects avoided. Table ES-11 provides information on the costs for California for post-2025, and Table ES-12 provides a regional breakdown of benefits for post-2025.

The EPA presents separate costs and benefits results for California because assuming attainment in an earlier year than would be required under the Clean Air Act would likely lead to an overstatement of costs and benefits because California might benefit from some existing federal or state programs that would be implemented between 2025 and the ultimate attainment years; because additional new technologies may become available between 2025 and the attainment years; and because the cost of existing technologies might fall over time. As such, we use the best available data to estimate costs and benefits for California in a post-2025 timeframe, but because of data limitations and additional uncertainty associated with not projecting emissions and air quality beyond 2025, we recognize that the estimates of costs and benefits for California in a post-2025 timeframe are likely to be relatively more uncertain than the national attainment estimates for 2025.

Table ES-9. Total Annual Costs and Benefits^a of the Identified + Unidentified Control Strategies Applied in California, Post-2025 (billions of 2011\$, 7% Discount Rate)^b

	Revised and Alternative Standard Levels	
	70 ppb	65 ppb
Total Costs^c	\$0.80	\$1.5
Total Health Benefits	\$1.2 to \$2.1 ^d	\$2.3 to \$4.2 ^d
Net Benefits	\$0.4 to \$1.3	\$0.8 to \$2.7

^a Benefits are nationwide benefits of attainment in California.

^b The guidelines of OMB Circular A-4 require providing comparisons of social costs and social benefits at discount rates of 3 and 7 percent. The tables in Chapter 6 provide additional characterizations of the monetized benefits, including benefits estimated at a 3 percent discount rate. Estimating multiple years of costs and benefits is not possible for this RIA due to data and resource limitations. As a result, we provide a snapshot of costs and benefits in 2025, using the best available information to approximate social costs and social benefits recognizing uncertainties and limitations in those estimates.

^c The engineering costs in this table are annualized at a 7 percent discount rate to the extent possible. See Chapter 4 for more discussions.

^d Excludes additional health and welfare benefits that could not be quantified (see Chapter 6, Section 6.6.3.8).

Table ES-10. Summary of Total Number of Annual Ozone and PM-Related Premature Mortalities and Premature Morbidity: Post-2025^a

	Revised and Alternative Standard Levels	
	70 ppb	65 ppb
Ozone-related premature deaths avoided (all ages)	72 to 120	150 to 240
PM_{2.5}-related premature deaths avoided (age 30+)	43 to 98	84 to 190
Other health effects avoided		
Non-fatal heart attacks (age 18-99) (5 studies) ^{PM}	6 to 51	11 to 100
Respiratory hospital admissions (age 0-99) ^{O3, PM}	150	300
Cardiovascular hospital admissions (age 18-99) ^{PM}	16	31
Asthma emergency department visits (age 0-99) ^{O3, PM}	380	760
Acute bronchitis (age 8-12) ^{PM}	64	130
Asthma exacerbation (age 6-18) ^{O3, PM}	160,000	330,000
Lost work days (age 18-65) ^{PM}	5,300	10,000
Minor restricted activity days (age 18-65) ^{O3, PM}	360,000	720,000
Upper & lower respiratory symptoms (children 7-14) ^{PM}	2,000	3,900
School loss days (age 5-17) ^{O3}	120,000	240,000

^a Nationwide benefits of attainment in California. All values are rounded to two significant figures. Additional information on confidence intervals are available in the tables in Chapter 6.

Table ES-11. Summary of Total Control Costs (Identified + Unidentified Control Strategies) by Revised and Alternative Standards for Post-2025 - California (billions of 2011\$, 7% Discount Rate)^a

Revised and Alternative Standard Level	Geographic Area	Total Control Costs (Identified and Unidentified)
70 ppb	California	\$0.80
65 ppb	California	\$1.5

^a All values are rounded to two significant figures. Costs are annualized at a 7 percent discount rate to the extent possible. Costs associated with unidentified controls are based on an average cost-per-ton methodology.

Table ES-12. Regional Breakdown of Monetized Ozone-Specific Benefits Results for Post-2025 (Nationwide Benefits of Attaining Revised and Alternative Standards just in California)^a

Region	Revised and Alternative Standard Levels	
	70 ppb	65 ppb
East ^b	3%	2%
California	90%	91%
Rest of West	7%	7%

^a Because we use benefit-per-ton estimates to calculate the PM_{2.5} co-benefits, a regional breakdown for the co-benefits is not available. Therefore, this table only reflects the ozone benefits.

^b Includes Texas and states to the north and east.

ES.3 Improvements between the Proposal and Final RIAs

In the regulatory impact analyses for both the proposed and final ozone NAAQS, there were two geographic areas outside of California where the majority of emissions reductions were needed to meet the revised standard level of 70 ppb – Texas and the Northeast. In analyzing 70 ppb in the final RIA, there were approximately 50 percent fewer emissions reductions needed in these two geographic areas. For an alternative standard of 65 ppb in the final RIA, emissions reductions needed nationwide were approximately 20 percent lower than at proposal. The primary reason for the difference in emissions reductions estimated for attainment is that in the final RIA we conducted more geographically-refined air quality sensitivity modeling to develop improved ozone response factors (see Chapter 2, Section 2.2.2 for a more detailed discussion of the air quality modeling) and focused the emissions reduction strategies on geographic areas closer to the monitors with the highest design values (see Chapter 3, Section 3.1.1 for a more detailed discussion of the emissions reduction strategies). The improvements in air quality modeling and emissions reduction strategies account for about 80 percent of the difference in needed emissions reductions between the proposal and final RIAs.

CHAPTER 6: HUMAN HEALTH BENEFITS ANALYSIS APPROACH AND RESULTS

6.1 Summary

This chapter of the Regulatory Impact Analysis (RIA) presents the estimated human health benefits for the revised National Ambient Air Quality Standards (NAAQS) for ozone. In this chapter, we quantify the health-related benefits of the ozone air quality improvements resulting from the illustrative emissions control strategies that reduce emissions of the ozone precursor pollutants (i.e., nitrogen oxides (NO_x) and volatile organic compounds (VOCs)) to reach the revised and alternative ozone NAAQS standard levels. We also estimate the health co-benefits of the fine particulate matter (PM_{2.5})-related air quality improvements that would occur as a result of reducing NO_x emissions.¹²³

We selected 2025 as the primary year of analysis because the Clean Air Act requires most areas of the U.S. to meet a revised ozone standard by 2025. The benefits of each standard alternative are estimated as being incremental to attaining the existing standard of 75 ppb.¹²⁴ These estimated benefits are incremental to the benefits estimated for several recent rules (e.g., U.S. EPA, 2011c and U.S. EPA, 2014a). We estimated the benefits of California attaining a revised standard in 2038 to account for the fact that many locations in this state must attain a revised standard later than the rest of the U.S. In this chapter, we refer to estimates of nationwide benefits of attaining an alternative standard everywhere in the U.S. except California as the *2025 scenario*. The *post-2025 scenario* refers to estimates of nationwide benefits of attaining an alternative standard just in California. Because we estimate incremental costs and benefits for these two distinct scenarios reflecting attainment in different years it is not appropriate to either sum, or directly compare, the estimates.

Table 6-1 summarizes the estimated monetized benefits (total and ozone only) of attaining the revised and alternative ozone standards of 70 ppb and 65 ppb, respectively, in 2025. Table 6-2 presents the same types of benefit estimates for the scenario. These estimates reflect the sum of the economic value of estimated morbidity and mortality effects related to changes in exposure to ozone and PM_{2.5}. Although these tables present ozone and PM_{2.5}-related benefits

¹²³ VOC reductions associated with simulated attainment of the revised and alternative ozone standards also have the potential to impact PM_{2.5} concentrations, but we were not able to estimate those effects.

¹²⁴ The current standard is the 4th highest daily maximum 8-hour ozone concentration of 75 ppb.

separately, it is not appropriate to compare the ozone-only benefits to total costs. Reduced levels of NO_x emissions needed to attain a more stringent ozone standard will affect levels of both ozone and PM_{2.5}. Following the standard practice for assessing the benefits of air quality rules (OMB, 2003; U.S. EPA, 2010e), this RIA quantifies the benefits of reducing both pollutants. For this reason, the costs of attaining a tighter standard should be compared against the sum of the ozone and PM_{2.5} benefits.

Compared with benefit estimates generated in the proposal RIA, the total benefit estimates generated for the 2025 scenario are ~55% lower for the revised standard (70 ppb) and ~22% lower for the alternative standard (65 ppb). Benefit estimates for the post-2025 scenarios are slightly higher than those generated at proposal (~6% for the revised standard and ~2% for the alternative standard). The proposal and final RIA estimates differ principally because as discussed in Chapter 2, Section 2.4.2 and Chapter 4, Section 4.6, the additional emissions sensitivity simulations and more refined ozone response factors allowed us to more accurately represent the increased effectiveness of emissions reductions closer to some monitor locations. The more refined air quality modeling resulted in approximately 50 percent fewer emissions reductions needed to reach a revised standard of 70 ppb and approximately 20 percent fewer emissions reductions needed to reach an alternative standard of 65 ppb. We have also slightly modified our approach to estimating morbidity benefits, which had a negligible (~1%) influence on the total monetized benefits in this RIA (see sections 6.3 and 6.6.3).

Table 6-1. Estimated Monetized Benefits of Attainment of the Revised and Alternative Ozone Standards for 2025 (nationwide benefits of attaining the standards everywhere in the U.S. except California) (billions of 2011\$)^a

	Discount Rate	70 ppb	65 ppb
Ozone-only Benefits ^c	^b	\$1.0 to \$1.7	\$5.3 to \$8.7
PM_{2.5} Co-benefits of NO_x Reductions ^d	3%	\$2.1 to \$4.7	\$10 to \$23
	7%	\$1.9 to \$4.2	\$9.3 to \$21
Total Benefits	3%	\$3.1 to \$6.4 ^e	\$16 to \$32 ^e
	7%	\$2.9 to \$5.9 ^e	\$15 to \$30 ^e

^a Rounded to two significant figures. It was not possible to quantify all benefits in this analysis due to data limitations. These estimates reflect the economic value of avoided morbidities and premature deaths using risk coefficients from the studies noted.

^b Ozone-only benefits reflect short-term exposure impacts and as such are assumed to occur in the same year as ambient ozone reductions. Consequently, social discounting is not applied to the benefits for this category.

^c Range reflects application of effect estimates from Smith et al. (2009) and Zanobetti and Schwartz (2008).

^d Range reflects application of effect estimates from Krewski et al. (2009) and Lepeule et al. (2012).

^e Excludes additional health and welfare benefits which could not be quantified (see section 6.6.3.8).

Table 6-2. Estimated Monetized Benefits of Attainment of the Revised and Alternative Ozone Standards for *post-2025* (nationwide benefits of attaining the standards just in California) (billions of 2011\$)^a

	Discount Rate	70 ppb	65 ppb
Ozone-only Benefits ^c	^b	\$0.79 to \$1.3	\$1.6 to \$2.6
PM _{2.5} Co-benefits of NO _x reductions ^d	3%	\$0.40 to \$0.91	\$0.79 to \$1.8
	7%	\$0.37 to \$0.82	\$0.71 to \$1.6
Total Benefits	3%	\$1.2 to \$2.2 ^e	\$2.4 to \$4.4 ^e
	7%	\$1.2 to \$2.1 ^e	\$2.3 to \$4.2 ^e

^a Rounded to two significant figures. It was not possible to quantify all benefits in this analysis due to data limitations. These estimates reflect the economic value of avoided morbidities and premature mortalities using risk coefficients from the studies noted.

^b Ozone-only benefits reflect short-term exposure impacts and as such are assumed to occur in the same year as ambient ozone reductions. Consequently, social discounting is not applied to the benefits for this category.

^c Range reflects application of effect estimates from Smith et al. (2009) and Zanobetti and Schwartz (2008).

^d Range reflects application of effect estimates from Krewski et al. (2009) and Lepeule et al. (2012).

^e Excludes additional health and welfare benefits which could not be quantified (see section 6.6.3.8).

The control measures (identified and unidentified) applied to reach the revised and alternative ozone standards would reduce other ambient pollutants, including VOCs and NO₂. However, because the method used in this analysis to simulate attainment does not account for changes in ambient concentrations of other pollutants, we were unable to quantify the co-benefits of reduced exposure to these pollutants. Due to limited data and methods, we were unable to estimate some anticipated health benefits associated with exposure to ozone and PM_{2.5}.

6.2 Overview

This chapter presents estimated health benefits for the revised and alternative ozone standards (70 ppb and 65 ppb, respectively) that the EPA could quantify, given the available resources, data and methods. This chapter characterizes the benefits of the application of the identified and unidentified control strategies identified in Chapter 3 for the revised and alternative ozone standards by answering three key questions:

1. What health effects are estimated to be avoided by reducing ambient ozone levels to attain the revised and alternative ozone standards?
2. What is the estimated economic value of these effects?

concentrations and the additional welfare benefits associated with reduced emissions of NO_x and VOC.

Table 7-1. Welfare Effects of NO_x and VOC Emissions

Pollutant	Atmospheric Effects		Atmospheric and Deposition Effects		Deposition Effects		
	Vegetation Injury (Ozone)	Visibility Impairment	Materials Damage	Climate	Ecosystem Effects—(Organics)	Acidification (freshwater)	Nitrogen Enrichment
NO _x	✓	✓	✓	✓		✓	✓
VOCs	✓	✓	✓		✓		

7.2 Welfare Benefits of Reducing Ozone

Ozone can affect ecological systems, leading to changes in the ecological community and influencing the diversity, health, and vigor of individual species (U.S. EPA, 2013). Ozone causes discernible injury to a wide array of vegetation (U.S. EPA, 2013). In terms of forest productivity and ecosystem diversity, ozone may be the pollutant with the greatest potential for region-scale forest impacts (U.S. EPA, 2013). Studies have demonstrated repeatedly that ozone concentrations observed in polluted areas can have substantial impacts on plant function (De Steiguer et al., 1990; Pye, 1988).

When ozone is present in ambient air, it can enter the leaves of plants, where it can cause significant cellular damage. Like carbon dioxide and other gaseous substances, ozone enters plant tissues primarily through the stomata in leaves in a process called “uptake” (Winner and Atkinson, 1986). Once sufficient levels of ozone (a highly reactive substance), or its reaction products, reaches the interior of plant cells, it can inhibit or damage essential cellular components and functions, including enzyme activities, lipids, and cellular membranes, disrupting the plant’s osmotic (i.e., water) balance and energy utilization patterns (U.S. EPA, 2013; Tingey and Taylor, 1982). With fewer resources available, the plant reallocates existing resources away from root growth and storage, above ground growth or yield, and reproductive processes, toward leaf repair and maintenance, leading to reduced growth and/or reproduction. Studies have shown that plants stressed in these ways may exhibit a general loss of vigor, which

can lead to secondary impacts that modify plants' responses to other environmental factors. Specifically, plants may become more sensitive to other air pollutants, or more susceptible to disease, pest infestation, harsh weather (e.g., drought, frost) and other environmental stresses, which can all produce a loss in plant vigor in ozone-sensitive species that over time may lead to premature plant death. Furthermore, there is evidence that ozone can interfere with the formation of mycorrhizae, an essential symbiotic fungus associated with the roots of most terrestrial plants, by reducing the amount of carbon available for transfer from the host to the symbiont (U.S. EPA, 2013).

This ozone damage may or may not be accompanied by visible injury on leaves, and likewise, visible foliar injury may or may not be a symptom of the other types of plant damage described above. Foliar injury is usually the first visible sign of injury to plants from ozone exposure and indicates impaired physiological processes in the leaves (Grulke, 2003). When visible injury is present, it is commonly manifested as chlorotic or necrotic spots, and/or increased leaf senescence (accelerated leaf aging). Visible foliar injury reduces the aesthetic value of ornamental vegetation and trees in urban landscapes and negatively affects scenic vistas in protected natural areas.

Ozone can produce both acute and chronic injury in sensitive species depending on the concentration level and the duration of the exposure. Ozone effects also tend to accumulate over the growing season of the plant, so that even lower concentrations experienced for a longer duration have the potential to create chronic stress on sensitive vegetation. Not all plants, however, are equally sensitive to ozone. Much of the variation in sensitivity between individual plants or whole species is related to the plant's ability to regulate the extent of gas exchange via leaf stomata (e.g., avoidance of ozone uptake through closure of stomata) and the relative ability of species to detoxify ozone-generated reactive oxygen free radicals (U.S. EPA, 2013; Winner, 1994). After injuries have occurred, plants may be capable of repairing the damage to a limited extent (U.S. EPA, 2013). Because of the differing sensitivities among plants to ozone, ozone pollution can also exert a selective pressure that leads to changes in plant community composition. Given the range of plant sensitivities and the fact that numerous other environmental factors modify plant uptake and response to ozone, it is not possible to identify threshold values above which ozone is consistently toxic for all plants.

Because plants are at the base of the food web in many ecosystems, changes to the plant community can affect associated organisms and ecosystems (including the suitability of habitats that support threatened or endangered species and below ground organisms living in the root zone). Ozone impacts at the community and ecosystem level vary widely depending upon numerous factors, including concentration and temporal variation of tropospheric ozone, species composition, soil properties and climatic factors (U.S. EPA, 2013). In most instances, responses to chronic or recurrent exposure in forested ecosystems are subtle and not observable for many years. These injuries can cause stand-level forest decline in sensitive ecosystems (U.S. EPA, 2013, McBride et al., 1985; Miller et al., 1982). It is not yet possible to predict ecosystem responses to ozone with certainty; however, considerable knowledge of potential ecosystem responses is available through long-term observations in highly damaged forests in the U.S. (U.S. EPA, 2013). Biomass loss due to ozone exposure affects climate regulation by reducing carbon sequestration. More carbon stays in the atmosphere because carbon uptake by forests is reduced. The studies cited in the Ozone ISA demonstrate a consistent pattern of reduced carbon uptake because of ozone damage, with some of the largest reductions projected over North America (U.S. EPA, 2013).

Ozone also directly contributes to climate change because tropospheric ozone traps heat, leading to increased surface temperatures. Projections of radiative forcing due to changing ozone concentrations over the 21st century show wide variation, due in large part to the uncertainty of future emissions of source gases (U.S. EPA 2014a). However, reduction of tropospheric ozone concentrations could provide an important means to slow climate change in addition to the added benefit of improving surface air quality (U.S. EPA, 2014a).

While it is clear that increases in tropospheric ozone lead to warming, the precursors of ozone also have competing effects on methane, complicating emissions reduction strategies. A decrease in carbon monoxide or VOC emissions would shorten the lifetime of methane, leading to an overall cooling effect. A decrease in NO_x emissions could lengthen the methane lifetime in certain regions, leading to warming (U.S. EPA, 2014a). Additionally, some strategies to reduce ozone precursor emissions could also lead to the reduced formation of aerosols (e.g., nitrates and sulfates) that currently have a cooling effect.

In the proposal RIA (U.S. EPA, 2014b), we were able to quantify only a small portion of the welfare impacts associated with reductions in ozone concentrations to meet alternative ozone standards. Using a model of commercial agriculture and forest markets, we analyzed the effects on consumers and producers of forest and agricultural products of changes in the W126 index resulting from meeting alternative standards within the proposed range of 70 to 65 ppb, as well as a lower standard level of 60 ppb. We also assessed the effects of those changes in commercial agricultural and forest yields on carbon sequestration and storage. This analysis provided limited quantitative information on the welfare benefits of meeting these alternative standards, focused only on one subset of ecosystem services. Commercial and non-commercial forests provide a number of additional services, including medicinal uses, non-commercial food and fiber production, arts and crafts uses, habitat, recreational uses, and cultural uses for Native American tribes. A more complete discussion of these additional ecosystem services is provided in the final *Welfare Risk and Exposure Assessment for Ozone* (WREA) (U.S. EPA, 2014a). Because we did not update this analysis for this RIA, we refer the reader to the results in the proposal RIA for an indication of the potential magnitude of these welfare benefits.

7.3 Additional Welfare Benefits of Strategies to Meet the Ozone NAAQS

Reductions in emissions of NO_x and VOC are associated with additional welfare benefits, including reductions in nutrient enrichment and acidification impacts on sensitive aquatic and terrestrial ecosystems and improvements in visibility in state and national parks, wilderness areas, and in the areas where people live and work.

Excess nitrogen deposition can lead to eutrophication of estuarine waters, which is associated with a range of adverse ecological effects. These include low dissolved oxygen (DO), harmful algal blooms (HABs), loss of submerged aquatic vegetation (SAV), and low water clarity. Low DO disrupts aquatic habitats, causing stress to fish and shellfish, which, in the short-term, can lead to episodic fish kills and, in the long-term, can damage overall growth in fish and shellfish populations. HAB are often toxic to fish and shellfish, lead to fish kills and aesthetic impairments of estuaries, and can in some instances be harmful to human health. SAV provides critical habitat for many aquatic species in estuaries and, in some instances, can also protect shorelines by reducing wave strength. Low water clarity is in part the result of

Exhibit 13



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exposure to O₃, are associated with morphologic changes such as mucous cell metaplasia⁵ of nasal epithelium, bronchiolar metaplasia of alveolar ducts and fibrotic changes in small airways (see Section 7.2.3 of the ISA, U.S. EPA, 2013). Mechanisms responsible for these responses are not well-understood. However, a recent study in mice demonstrated a key role for a signaling pathway in the deposition of collagen in the airway wall following chronic intermittent exposure to O₃. Chronic intermittent exposure to O₃ has also been shown to result in effects on the developing lung and immune system.

Systemic inflammation and vascular oxidative/nitrosative stress are also key events in the toxicity pathway of O₃. Extrapulmonary effects of O₃ occur in numerous organ systems, including the cardiovascular, central nervous, reproductive, and hepatic systems (see Sections 6.3 to 6.5 and Sections 7.3 to 7.5 of the ISA, U.S. EPA, 2013). It has been proposed that lipid oxidation products resulting from reaction of O₃ with lipids and/or cellular membranes in the ELF are responsible for systemic responses; however, it is not known whether they gain access to the circulation. Alternatively, release of diffusible mediators from the lung into the circulation may initiate or propagate inflammatory responses in the circulation or other organ systems.

Responses to O₃ exposure are variable within the population. Although studies have shown a large range of pulmonary function (i.e., spirometric) responses to O₃ among healthy young adults, responses within an individual are relatively consistent over time. Other responses to O₃ have also been characterized by a large degree of inter-individual variability. For example, a 3- to 20-fold difference among subjects in their studies in airways inflammation (i.e., neutrophilia influx) following O₃ exposure has been reported (Schelegle et al., 1991 and Devlin et al., 1991, respectively). Reproducibility of an individual's inflammatory response to O₃ exposure in humans, measured as sputum neutrophilia, was demonstrated by Holz et al. (1999). Since individual inflammatory responses were relatively consistent across time, it was thought that inflammatory responsiveness reflected an intrinsic characteristic of the subject (Mudway and Kelly, 2000). While the basis for the observed inter-individual variability in responsiveness to O₃ is not clear, section 5.4.2 of the ISA (U.S. EPA, 2013) discusses mechanisms that may underlie the variability in responses seen among individuals. Certain functional genetic polymorphisms, pre-existing conditions or diseases, nutritional status, lifestages, and co-exposures contribute to altered risk of O₃-induced effects.

⁵ Metaplasia is the reversible replacement of one differentiated cell type with another mature differentiated cell type. The change from one type of cell to another may generally be a part of normal maturation process or caused by some sort of abnormal stimulus. In simplistic terms, it is as if the original cells are not robust enough to withstand the new environment, and so they change into another type more suited to the new environment. If the stimulus that caused metaplasia is removed or ceases, tissues return to their normal pattern of differentiation.

Experimental evidence for such O₃-induced changes contributes to our understanding of the biological plausibility of adverse O₃-related health effects, including a range of respiratory effects as well as effects outside the respiratory system (e.g., cardiovascular effects) (U.S. EPA, 2013, Chapters 6 and 7).

3.1.2 Nature of Effects

- **To what extent does the currently available scientific evidence alter or strengthen our conclusions from the last review regarding health effects attributable to O₃ exposure in ambient air? Are previously identified uncertainties reduced or do important uncertainties remain?**

The health effects of ozone are described in detail in the assessment of the evidence available in this review which is largely consistent with conclusions of past Air Quality Criteria Documents (AQCD). In some categories of health effects, there is newly available evidence regarding some aspects of the effects described in the last review or that strengthens our conclusions regarding aspects of O₃ toxicity on a particular physiological system (U.S. EPA, 2013, Table 1-1). A sizeable number of studies on O₃ health effects are newly available in this review and are critically assessed in the ISA as part of the full body of evidence. Based on this assessment, the ISA determined that a causal relationship⁶ exists between short-term exposure to O₃ in ambient air⁷ and effects on the respiratory system and that a likely to be causal relationship⁸ exists between long-term exposure to O₃ in ambient air and respiratory effects (U.S. EPA, 2013, pp. 1-6 to 1-7). As stated in the ISA, “[c]ollectively, a very large amount of evidence spanning several decades supports a relationship between exposure to O₃ and a broad range of respiratory effects” (ISA, p. 1-6). Additionally, the ISA determined that the relationships between short-term exposures to O₃ in ambient air and both total mortality and cardiovascular effects are likely to be causal, based on expanded evidence bases in the current review (U.S. EPA, 2013, pp. 1-7 to 1-8). In the ISA, EPA additionally determined that the currently available evidence for additional endpoints is suggestive of causal relationships between short-term (central nervous system effects) and long-term exposure (cardiovascular effects, central nervous system effects and total mortality) to ambient O₃. Consistent with emphasis in past reviews on O₃ health effects for which

⁶ Since the last O₃ NAAQS review, the ISAs which have replaced CDs in documenting each review of the scientific evidence (or air quality criteria) employ a systematic framework for weighing the evidence and describing associated conclusions with regard to causality, using established descriptors, as summarized in section 1.3.1 above (U.S. EPA, 2013, Preamble).

⁷ In determining that a causal relationship exists for O₃ with specific health effects, EPA has concluded that “[e]vidence is sufficient to conclude that there is a causal relationship with relevant pollutant exposures” (ISA, p. lxiv).

⁸ In determining a likely to be a causal relationship exists for O₃ with specific health effects, EPA has concluded that “[e]vidence is sufficient to conclude that a causal relationship is likely to exist with relevant pollutant exposures, but important uncertainties remain” (ISA, p. lxiv).

the evidence is strongest, we place the greatest emphasis on studies of health effects that have been judged in the ISA to be caused by, or likely to be caused by, O₃ exposures (U.S. EPA, 2013, section 2.5.2).

This section presents our consideration of the evidence for health effects attributable to O₃ exposures, including respiratory morbidity and mortality effects attributable to short- and long-term exposures, and cardiovascular system effects (including mortality) and total mortality attributable to short-term exposures. We focus particularly on considering the extent to which the scientific evidence available in the current review has been strengthened since the last review, and the extent to which important uncertainties and limitations in the evidence from the last review have been addressed. In section 3.1.2.2, we then consider the extent to which the available evidence indicates health effects may be attributable to ambient O₃ concentrations likely to be allowed by the current O₃ NAAQS. In this section, we address the following specific question for each category of health effects considering the evidence available in the 2008 review of the standard as well as evidence that has become available since then. The ISA summarizes the longstanding body of evidence for O₃ respiratory effects as follows (U.S. EPA, 2013, p. 1-6).

The clearest evidence for health effects associated with exposure to O₃ is provided by studies of respiratory effects. Collectively, a very large amount of evidence spanning several decades supports a relationship between exposure to O₃ and a broad range of respiratory effects (see Section 6.2.9 and Section 7.2.8). The majority of this evidence is derived from studies investigating short-term exposures (i.e., hours to weeks) to O₃, although animal toxicological studies and recent epidemiologic evidence demonstrate that long-term exposure (i.e., months to years) may also harm the respiratory system.

The extensive body of evidence supporting a causal relationship between short-term O₃ exposures and respiratory effects is discussed in detail in Chapter 6 of the ISA (U.S. EPA, 2013), while evidence for respiratory effects associated with long-term or repeated O₃ exposures are discussed in chapter 7 of that document (U.S., EPA, 2013).

3.1.2.1 Respiratory Effects – Short-term Exposures

- **To what extent does the currently available scientific evidence, including related uncertainties, strengthen or alter our understanding from the last review of respiratory effects attributable to short-term O₃ exposures?**

The 2006 O₃ AQCD concluded that there was clear, consistent evidence of a causal relationship between short-term O₃ exposure and respiratory effects (U.S. EPA, 2006). This conclusion was substantiated by evidence from controlled human exposure and toxicological studies indicating a range of respiratory effects in response to short-term O₃ exposures, including pulmonary function decrements and increases in respiratory symptoms, lung inflammation, lung

permeability, and airway hyperresponsiveness. Toxicological studies provided additional evidence for O₃-induced impairment of host defenses. Combined, these findings from experimental studies provided support for epidemiologic evidence, in which short-term increases in ambient O₃ concentration were consistently associated with decreases in lung function in populations with increased outdoor exposures, especially children with asthma and healthy children; increases in respiratory symptoms and asthma medication use in children with asthma; and increases in respiratory-related hospital admissions and asthma-related ED visits (U.S. EPA, 2013, pp. 6-1 to 6-2).

As discussed in detail in the ISA (U.S. EPA, 2013, section 6.2.9), studies evaluated since the completion of the 2006 O₃ AQCD support and expand upon the strong body of evidence that, in the last review, indicated a causal relationship between short-term O₃ exposures and respiratory health effects. Recent controlled human exposure studies conducted in young, healthy adults with moderate exertion have reported FEV₁ decrements and pulmonary inflammation following prolonged exposures to O₃ concentrations as low as 60 ppb, and respiratory symptoms following exposures to concentrations as low as 70 ppb.⁹ Epidemiologic studies provide evidence that increases in ambient O₃ exposures can result in lung function decrements, increases in respiratory symptoms, and pulmonary inflammation in children with asthma; increases in respiratory-related hospital admissions and emergency department visits; and increases in respiratory mortality. Some of these studies report such associations even for O₃ concentrations at the low end of the distribution of daily concentrations. Recent epidemiologic studies report that associations with respiratory morbidity and mortality are stronger during the warm/summer months and remain robust after adjustment for copollutants. Recent toxicological studies reporting O₃-induced inflammation, airway hyperresponsiveness, and impaired lung host defense continue to support the biological plausibility and modes of action for the O₃-induced respiratory effects observed in the controlled human exposure and epidemiologic studies. Further support is provided by recent studies that found O₃-associated increases in indicators of airway inflammation and oxidative stress in children with asthma (U.S. EPA, 2013, section 6.2.9). Together, epidemiologic and experimental studies support a continuum of respiratory effects associated with O₃ exposure that can result in respiratory-related emergency department visits, hospital admissions, and/or mortality (U.S. EPA, 2013, section 6.2.9).

Across respiratory endpoints, evidence indicates antioxidant capacity may modify the risk of respiratory morbidity associated with O₃ exposure (U.S. EPA, 2013, section 6.2.9, p. 6-161) (section 3.1.1, above). The potentially elevated risk of populations with diminished

⁹ Schelegle et al. (2009) reported a statistically significant increase in respiratory symptoms in healthy adults at a target O₃ exposure concentration of 70 ppb. For this 70 ppb target, Schelegle et al. (2009) reported an actual exposure concentration, averaged over the study period, of 72 ppb.

antioxidant capacity and the reduced risk of populations with sufficient antioxidant capacity is supported by epidemiologic and controlled human exposure studies. Additional evidence characterizes O₃-induced decreases in antioxidant levels as a key event in the mode of action for downstream effects.

We describe key aspects of this evidence below with regard to lung function decrements; pulmonary inflammation, injury, and oxidative stress; airway hyperresponsiveness; respiratory symptoms and medication use; lung host defense; allergic and asthma-related responses; hospital admissions and emergency department visits; and respiratory mortality.

Lung Function Decrements

In the 2008 review, a large number of controlled human exposure studies reported O₃-induced lung function decrements in young, healthy adults engaged in intermittent, moderate exertion following 6.6 hour exposures to O₃ concentrations at or above 80 ppb. Although two studies also reported effects following exposures to lower concentrations, an important uncertainty in the last review was the extent to which exposures to O₃ concentrations below 80 ppb result in lung function decrements. In addition, in the last review epidemiologic panel studies had reported O₃-associated lung function decrements in a variety of different populations (e.g., children, outdoor workers) likely to experience increased exposures. In the current review, additional controlled human exposure studies are available that have evaluated exposures to O₃ concentrations of 60 or 70 ppb. The available evidence from controlled human exposure and panel studies is assessed in detail in the ISA (U.S. EPA, 2013, section 6.2.1) and is summarized below.

Controlled exposures to O₃ concentrations that can be found in the ambient air can result in a number of lung function effects, including decreased inspiratory capacity; mild bronchoconstriction; and rapid, shallow breathing patterns during exercise. Reflex inhibition of inspiration results in a decrease in forced vital capacity (FVC) and total lung capacity (TLC) and, in combination with mild bronchoconstriction, contributes to a decrease in the forced expiratory volume in 1 second (FEV₁) (U.S. EPA, 2013, section 6.2.1.1).¹⁰ Accumulating evidence indicates that such effects are mediated by activation of sensory nerves, resulting in the involuntary truncation of inspiration and a mild increase in airway obstruction due to bronchoconstriction (U.S. EPA, 2013, section 5.3.10).

¹⁰ The controlled human exposure studies emphasized in this PA utilize only healthy adult subjects. In the near absence of controlled human exposure data for children, HREA estimates of lung function decrements are based on the assumption that children exhibit the same lung function responses following O₃ exposures as healthy 18 year olds (U.S. EPA, 2014, sections 6.2.4 and 6.5). This assumption is justified in part by the findings of McDonnell et al. (1985), who reported that children 8-11 year old experienced FEV₁ responses similar to those observed in adults 18-35 years old. Thus, the conclusions about the occurrence of lung function decrements that follow generally apply to children as well as to adults.

Data from controlled human exposure studies indicate that increasing the duration of O₃ exposures and increasing ventilation rates decreases the O₃ exposure concentrations required to impair lung function. Ozone exposure concentrations well above those typically found in ambient air are required to impair lung function in healthy resting adults, while exposure to O₃ concentrations at or below those in the ambient air have been reported to impair lung function in healthy adults exposed for longer durations while undergoing intermittent, moderate exertion (U.S. EPA, 2013, section 6.2.1.1). With repeated O₃ exposures over several days, FEV₁ responses become attenuated in both healthy adults and adults with mild asthma, though this attenuation of response is lost after about a week without exposure (U.S. EPA, 2013, section 6.2.1.1; page 6-27).

When considering controlled human exposures studies of O₃-induced lung function decrements we evaluate both group mean changes in lung function and the interindividual variability in the magnitude of responses. An advantage of O₃ controlled human exposure studies (i.e., compared to the epidemiologic panel studies discussed below) is that reported effects necessarily result from exposures to O₃ itself.¹¹ To the extent studies report statistically significant decrements in mean lung function following O₃ exposures after controlling for other factors, we have more confidence that measured decrements are due to the O₃ exposure itself, rather than to chance alone. As discussed below, group mean changes in lung function are often small, especially following exposures to relatively low O₃ concentrations (e.g., 60 ppb). However, even when group mean decrements in lung function are small, some individuals could experience decrements that are “clinically meaningful” (Pellegrino et al., 2005; ATS, 1991) with respect to criteria for spirometric testing, and/or that could be considered “adverse” with respect to public health policy decisions (section 3.1.3 below).

At the time of the last review, a number of controlled human exposure studies had reported lung function decrements in young, healthy adults following prolonged (6.6-hour) exposures while at moderate exertion to O₃ concentrations at and above 80 ppb. In addition, there were two controlled human exposure studies by Adams (2002, 2006) that examined lung function effects following exposures to O₃ concentrations of 60 ppb. The EPA’s analysis of the data from the Adams (2006) study reported a small but statistically significant O₃-induced decrement in group mean FEV₁ following exposures of young, healthy adults, while at moderate exertion, to 60 ppb O₃, when compared with filtered air controls (Brown, 2008).¹² Further

¹¹ The ISA notes that the use of filtered air responses as a control for the assessment of responses following O₃ exposure in controlled human exposure studies serves to eliminate alternative explanations other than O₃ itself in causing the measured responses (U.S. EPA, 2013, section 6.2.1.1).

¹² Adams (2006) did not find effects on FEV₁ at 60 ppb to be statistically significant. In an analysis of the Adams (2006) data, even after removal of potential outliers, Brown et al. (2008) found the average effect on FEV₁ at 60 ppb to be small, but highly statistically significant ($p < 0.002$) using several common statistical tests.

examination of the post-exposure FEV₁ data, and mean data for other time points and other concentrations, indicated that the temporal pattern of the response to 60 ppb O₃ was generally consistent with the temporal patterns of responses to higher O₃ concentrations in this and other studies. (75 FR 2950, January 19, 2010). This suggested a pattern of response following exposures to 60 ppb O₃ that was consistent with a dose-response relationship, rather than random variability. See also *State of Mississippi v. EPA*, 744 F. 3d at 1347 (upholding EPA's interpretation of the Adams studies).

Figure 6-1 in the ISA summarizes the currently available evidence from multiple controlled human exposure studies evaluating group mean changes in FEV₁ following prolonged O₃ exposures (i.e., 6.6 hours) in young, healthy adults engaged in moderate levels of physical activity (U.S. EPA, 2013, section 6.2.1.1). With regard to the group mean changes reported in these studies, the ISA specifically notes the following (U.S. EPA, 2013, section 6.2.1.1, Figure 6-1):

1. Prolonged exposure to 40 ppb O₃ results in a small decrease in group mean FEV₁ that is not statistically different from responses following exposure to filtered air (Adams, 2002; Adams, 2006).
2. Prolonged exposure to an average O₃ concentration of 60 ppb results in group mean FEV₁ decrements ranging from 1.8% to 3.6% (Adams 2002; Adams, 2006;¹³ Schelegle et al., 2009;¹⁴ Kim et al., 2011). Based on data from multiple studies, the weighted average group mean decrement was 2.7%. In some analyses, these group mean decrements in lung function were statistically significant (Brown et al., 2008; Kim et al., 2011), while in other analyses they were not (Adams, 2006; Schelegle et al., 2009).¹⁵
3. Prolonged exposure to an average O₃ concentration of 70 ppb results in a statistically significant group mean decrement in FEV₁ of about 6% (Schelegle et al., 2009).¹⁶
4. Prolonged square-wave exposure to average O₃ concentrations of 80 ppb, 100 ppb, or 120 ppb O₃ results in statistically significant group mean decrements in FEV₁ ranging from 6 to 8%, 8 to 14%, and 13 to 16%, respectively (Folinsbee et al., 1988; Horstman et al., 1990; McDonnell et al., 1991; Adams, 2002; Adams, 2003; Adams, 2006).

¹³ Adams (2006; 2002) both provide data for an additional group of 30 healthy subjects that were exposed via facemask to 60 ppb (square-wave) O₃ for 6.6 hours with moderate exercise ($\dot{V}_E = 23$ L/min per m² BSA). These subjects are described on page 133 of Adams (2006) and pages 747 and 761 of Adams (2002). The FEV₁ decrement may be somewhat increased due to a target \dot{V}_E of 23 L/min per m² BSA relative to other studies having the target \dot{V}_E of 20 L/min per m² BSA. The facemask exposure is not expected to affect the FEV₁ responses relative to a chamber exposure.

¹⁴ Schelegle et al. (2009) reported an actual mean exposure concentration of 63 ppb for the target of 60 ppb.

¹⁵ Adams (2006) did not find effects on FEV₁ at 60 ppb to be statistically significant. In an analysis of the Adams (2006) data, Brown et al. (2008) addressed the more fundamental question of whether there were statistically significant differences in responses before and after the 6.6 hour exposure period and found the average effect on FEV₁ at 60 ppb to be small, but highly statistically significant using several common statistical tests, even after removal of potential outliers.

¹⁶ Schelegle et al. (2009) reported an actual mean exposure concentration of 72 ppb for the target of 70 ppb.

As illustrated in Figure 6-1 of the ISA, there is a smooth dose-response curve without evidence of a threshold for exposures between 40 and 120 ppb O₃ (U.S. EPA, 2013, Figure 6-1). When these data are taken together, the ISA concludes that “mean FEV₁ is clearly decreased by 6.6-h exposures to 60 ppb O₃ and higher concentrations in [healthy, young adult] subjects performing moderate exercise” (U.S. EPA, 2013, p. 6-9).

With respect to interindividual variability in lung function, in an individual with relatively “normal” lung function, with recognition of the technical and biological variability in measurements, within-day changes in FEV₁ of $\geq 5\%$ are clinically meaningful (Pellegrino et al., 2005; ATS, 1991). The ISA (U.S. EPA, 2013, section 6.1.) focuses on individuals with $>10\%$ decrements in FEV₁ for two reasons. A 10% FEV₁ decrement is accepted by the American Thoracic Society (ATS) as an abnormal response and a reasonable criterion for assessing exercise-induced bronchoconstriction (Dryden et al., 2010; ATS, 2000). (U.S. EPA, 2013, section 6.2.1.1). Also, some individuals in the Schelegle et al. (2009) study experienced 5-10% FEV₁ decrements following exposure to filtered air.

In previous NAAQS reviews, the EPA has made judgments regarding the potential implications for individuals experiencing FEV₁ decrements of varying degrees of severity.¹⁷ For people with lung disease, the EPA judged that moderate functional decrements (e.g., FEV₁ decrements ≥ 10 percent but < 20 percent, lasting up to 24 hours) would likely interfere with normal activity for many individuals, and would likely result in more frequent use of medication (75 FR 2973, January 19, 2010). In previous reviews CASAC has endorsed these conclusions. In the context of standard setting, in the last review O₃ review CASAC indicated that it is appropriate to focus on the lower end of the range of moderate functional responses (e.g., FEV₁ decrements ≥ 10 percent) when estimating potentially adverse lung function decrements in people with lung disease, especially children with asthma (Henderson, 2006). More specifically, CASAC stated that “[a] 10% decrement in FEV₁ can lead to respiratory symptoms, especially in individuals with pre-existing pulmonary or cardiac disease. For example, people with chronic obstructive pulmonary disease have decreased ventilatory reserve (i.e., decreased baseline FEV₁) such that a $\geq 10\%$ decrement could lead to moderate to severe respiratory symptoms” (Samet, 2011). In this review, CASAC reiterated its support for this conclusion, stating that “[a]n FEV₁ decrement of $\geq 10\%$ is a scientifically relevant surrogate for adverse health outcomes for people with asthma and lung disease” (Frey, 2014 p. 3). Therefore, in considering interindividual variability in

¹⁷ Such judgments have been made for decrements in FEV₁ as well as for increased airway responsiveness and symptomatic responses (e.g., cough, chest pain, wheeze). Ranges of pulmonary responses and their associated potential impacts are presented in Tables 3-2 and 3-3 of the Staff Paper (U.S. EPA, 2007).

O₃-induced lung function decrements in the current review, we also focus on the extent to which individuals were reported to experience FEV₁ decrements of 10% or greater.

New studies (Schelegle et al., 2009; Kim et al., 2011) add to the previously available evidence for interindividual variability in the responses of healthy adults following exposures to O₃. Following prolonged exposures to 80 ppb O₃ while at moderate exertion, the proportion of healthy adults experiencing FEV₁ decrements greater than 10% was 17% by Adams (2006), 26% by McDonnell (1996), and 29% by Schelegle et al. (2009). Following exposures to 60 ppb O₃, that proportion was 20% by Adams (2002), 3% by Adams (2006), 16% by Schelegle et al. (2009), and 5% by Kim et al. (2011). Based on these studies, the weighted average proportion of young, healthy adults with >10% FEV₁ decrements is 25% following exposure to 80 ppb O₃ and 10% following exposure to 60 ppb O₃ (U.S. EPA, 2013, page 6-19).¹⁸ The ISA notes that responses within an individual tend to be reproducible over a period of several months, indicating that interindividual differences reflect differences in intrinsic responsiveness. Given this, the ISA concludes that “a considerable fraction” of healthy individuals experience clinically meaningful decrements in lung function when exposed for 6.6 hours to 60 ppb O₃ during quasi continuous, moderate exertion (U.S. EPA, 2013, section 6.2.1.1, p. 6-20).

As discussed above (Figure 3-1) and in the ISA (U.S EPA, 2013, Section 5.3.2), secondary oxidation products formed following O₃ exposures can activate neural reflexes leading to decreased lung function. Two new quantitative models, discussed in section 6.2.1.1 of the ISA (U.S. EPA, 2013, p. 6-15), included mathematical approaches to simulate the protective effect of antioxidants in the ELF at lower ambient O₃ concentrations, and include a threshold below which changes in lung function do not occur (McDonnell et al., 2012; Schelegle et al., 2012).

McDonnell et al. (2012) and Schelegle et al. (2012) developed models using data on O₃ exposure concentrations, ventilation rates, duration of exposures, and lung function responses from a number of controlled human exposure studies. The McDonnell et al. (2012) and Schelegle et al. (2012) studies analyzed large datasets to fit compartmental models that included the concept of a dose of onset in lung function response or a response threshold based upon the inhaled O₃ dose. The first compartment in the McDonnell et al. (2012) model considers the level of oxidant stress in response to O₃ exposure to increase over time as a function of dose rate ($C \times \dot{V}_E$) and decrease by clearance or metabolism over time. In the second compartment of the McDonnell model, once oxidant stress reaches a threshold level the decrement in FEV₁ increases

¹⁸ The ISA also notes that by considering responses uncorrected for filtered air exposures, during which lung function typically improves (which would increase the size of the change, pre-and post-exposure), 10% is an underestimate of the proportion of healthy individuals that are likely to experience clinically meaningful changes in lung function following exposure for 6.6 hours to 60 ppb O₃ during intermittent moderate exertion (U.S. EPA, 2013, section 6.2.1.1).

as a sigmoid-shaped function. In the Schelegle et al. (2012) model, a first compartment acts as a reservoir in which oxidant stress builds up until the dose of onset, at which time it spills over into a second compartment. The second compartment is identical to the first compartment in McDonnell et al. (2012) model. The oxidant levels in the second compartment were multiplied by a responsiveness coefficient to predict FEV₁ responses for the Schelegle et al. (2012) model.

The McDonnell et al. (2012) model was fit to a large dataset consisting of the FEV₁ responses of 741 young, healthy adults (18-35 years of age) from 23 individual controlled exposure studies. Concentrations across individual studies ranged from 40 ppb to 400 ppb, activity level ranged from rest to heavy exercise, duration of exposure was from 2 to 7.6 hours. The extension of the McDonnell et al. (2012) model to children and older adults is discussed in section 6.2.1 of the ISA (U.S. EPA, 2013). Schelegle et al. (2012) also analyzed a large dataset with substantial overlap to that used by McDonnell et al. (2012). The Schelegle et al. (2012) model was fit to the FEV₁ responses of 220 young healthy adults (taken from a dataset of 704 individuals) from 21 individual controlled exposure studies. The resulting empirical models can estimate the frequency distribution of individual lung function responses for any exposure scenario as well as summary measures of the distribution such as the mean or median response and the proportions of individuals with FEV₁ decrements > 10%, 15%, and 20%.

The predictions of the McDonnell and Schelegle models are consistent with the observed results from the individual studies of O₃-induced FEV₁ decrements. Specifically, the model developed by McDonnell et al. (2012) predicts that 9% of healthy exercising adults would experience FEV₁ decrements greater than 10% following 6.6 hour exposure to 60 ppb O₃, and that 22% would experience such decrements following exposure to 80 ppb O₃ (U.S. EPA, 2013, p. 6-18 and Figure 6-3). The model developed by Schelegle et al. (2012) predicts that, for a prolonged (6.6 hours) O₃ exposure with moderate, quasi continuous exercise, the average dose of onset for FEV₁ decrement would be reached following 4 to 5 hours of exposure to 60 ppb, and following 3 to 4 hours of exposure to 80 ppb. However, 14% of the individuals had a dose of onset that was less than 40% of the average. Those individuals would reach their dose of onset following 1 to 2 hours of exposure to 50 to 80 ppb O₃ (U.S. EPA, 2013, p. 6-16), which is consistent with the threshold FEV₁ responses reported by McDonnell et al. (2012).

Epidemiologic studies¹⁹ have consistently linked short-term increases in ambient O₃ concentrations with lung function decrements in diverse populations and lifestages, including children attending summer camps, adults exercising or working outdoors, and groups with pre-existing respiratory diseases such as asthmatic children (U.S. EPA, 2013, section 6.2.1.2). Some

¹⁹ Unless otherwise specified, the epidemiologic studies discussed in this PA evaluate only adults.

of these studies reported ozone-associated lung function decrements accompanied by respiratory symptoms²⁰ in asthmatic children (Just et al., 2002; Mortimer et al., 2002; Ross et al., 2002; Gielen et al., 1997; Romieu et al., 1997; Thurston et al., 1997; Romieu et al., 1996). In contrast, studies of children in the general population have reported similar O₃-associated lung function decrements but without accompanying respiratory symptoms (Ward et al., 2002; Gold et al., 1999; Linn et al., 1996) (U.S. EPA, 2013, section 6.2.1.2).

Several epidemiologic panel studies reported that associations with lung function decrements persisted at relatively low ambient O₃ concentrations. For outdoor recreation or exercise, associations were reported in analyses restricted to 1-hour average O₃ concentrations less than 80 ppb (Spektor et al., 1988a; Spektor et al., 1988b), 60 ppb (Brunekreef et al., 1994; Spektor et al., 1988a), and 50 ppb (Brunekreef et al., 1994). Among outdoor workers, Brauer et al. (1996) found a robust association using daily 1-hour max O₃ concentrations less than 40 ppb. Ulmer et al. (1997) found a robust association in schoolchildren using 30-minute maximum O₃ concentrations less than 60 ppb. For 8-hour average O₃ concentrations, associations with lung function decrements in children with asthma were found to persist at concentrations less than 80 ppb in a U.S. multicity study (Mortimer et al., 2002) and less than 51 ppb in a study conducted in the Netherlands (Gielen et al., 1997).

Epidemiologic panel studies investigating the effects of short-term exposure to O₃ provided information on potential confounding by copollutants such as PM_{2.5}, PM₁₀, NO₂, or SO₂. These studies varied in how they evaluated confounding. Some studies of subjects exercising outdoors indicated that ambient concentrations of copollutants such as NO₂, SO₂, or acid aerosol were low, and thus not likely to confound associations observed for O₃ (Hoppe et al., 2003; Brunekreef et al., 1994; Hoek et al., 1993). In other studies of children with increased outdoor exposures, O₃ was consistently associated with decreases in lung function, whereas other pollutants such as PM_{2.5}, sulfate, and acid aerosol individually showed variable associations across studies (Thurston et al., 1997; Castillejos et al., 1995; Berry et al., 1991; Avol et al., 1990; Spektor et al., 1988a). Studies that conducted copollutant modeling generally found O₃-associated lung function decrements to be robust (i.e., most copollutant-adjusted effect estimates fell within the 95% CI of the single-pollutant effect estimates) (U.S. EPA, 2013, Figure 6-10 and Table 6-14). Most O₃ effect estimates for lung function were robust to adjustment for temperature, humidity, and copollutants such as PM_{2.5}, PM₁₀, NO₂, or SO₂. Although examined

²⁰ Reversible loss of lung function in combination with the presence of symptoms meets the ATS definition of adversity (ATS, 2000).

in only a few epidemiologic studies, O₃ also remained associated with decreases in lung function with adjustment for pollen or acid aerosols (U.S. EPA, 2013, section 6.2.1.2).

Several epidemiologic studies demonstrated the protective effects of vitamin E and vitamin C supplementation, and increased dietary antioxidant intake, on O₃-induced lung function decrements (Romieu et al., 2002) (U.S. EPA, 2013, Figure 6-7 and Table 6-8).²¹ These results provide support for the new, quantitative models (McDonnell et al., 2012; Schelegle et al., 2012), discussed above, which make use of the concept of oxidant stress to estimate the occurrence of lung function decrements following exposures to relatively low O₃ concentrations.

In conclusion, new information from controlled human exposure studies considerably strengthens the evidence and reduces the uncertainties, relative to the evidence that was available at the time of the 2008 review, regarding the presence and magnitude of lung function decrements in healthy adults following prolonged exposures to O₃ concentrations below 80 ppb. As discussed in Section 6.2.1.1 in the ISA (U.S. EPA, 2013, p. 6-12), there is information available from four separate studies that evaluated exposures to 60 ppb O₃ (Kim et al., 2011; Schelegle et al., 2009; Adams 2002; 2006). Although not consistently statistically significant, group mean FEV₁ decrements following exposures to 60 ppb O₃ are consistent among these studies. Moreover, as is illustrated in Figure 6-1 of the ISA (U.S. EPA, 2013), the group mean FEV₁ responses at 60 ppb fall on a smooth intake dose-response curve for exposures between 40 and 120 ppb O₃. These studies also indicate that, on average, 10% of young, healthy adults experience clinically meaningful decrements in lung function when exposed for 6.6 hours to 60 ppb O₃ during intermittent, moderate exertion. One recent study has also reported statistically significant decrements following exposures to 70 ppb O₃ (Schelegle et al., 2009). Predictions from newly developed quantitative models, based on the concept that O₃-induced oxidation results in lung function decrements, are consistent with these experimental results. Additionally, as discussed in more detail in section 3.1.4 below, epidemiologic studies continue to provide evidence of lung function decrements in people who are active outdoors, including people engaged in outdoor recreation or exercise, children, and outdoor workers, at low ambient O₃ concentrations. While few new epidemiologic studies of O₃-associated lung function decrements are available in this review, previously available studies have reported associations with decrements, including at relatively low ambient O₃ concentrations.

Pulmonary Inflammation, Injury, and Oxidative Stress

Ozone exposures result in increased respiratory tract inflammation and epithelial permeability. Inflammation is a host response to injury, and the induction of inflammation is

²¹ Evidence from controlled human exposure studies is mixed, suggesting that supplementation may be ineffective in the absence of antioxidant deficiency (U.S. EPA, 2013, p. 5-63).

evidence that injury has occurred. Oxidative stress has been shown to play a key role in initiating and sustaining O₃-induced inflammation. Secondary oxidation products formed as a result of reactions between O₃ and components of the ELF can increase the expression of molecules (i.e., cytokines, chemokines, and adhesion molecules) that can enhance airway epithelium permeability (U.S. EPA, 2013, Sections 5.3.3 and 5.3.4). As discussed in detail in the ISA (U.S. EPA, 2013, section 6.2.3), O₃ exposures can initiate an acute inflammatory response throughout the respiratory tract that has been reported to persist for at least 18-24 hours after exposure.

Inflammation induced by exposure of humans to O₃ can have several potential outcomes: (1) inflammation induced by a single exposure (or several exposures over the course of a summer) can resolve entirely; (2) continued acute inflammation can evolve into a chronic inflammatory state; (3) continued inflammation can alter the structure and function of other pulmonary tissue, leading to diseases such as asthma; (4) inflammation can alter the body's host defense response to inhaled microorganisms, particularly in potentially at-risk populations or lifestages such as the very young and old; and (5) inflammation can alter the lung's response to other agents such as allergens or toxins (U.S. EPA, 2013, Section 6.2.3). Thus, lung injury and the resulting inflammation provide a mechanism by which O₃ may cause other more serious morbidity effects (e.g., asthma exacerbations).

In the last review, controlled human exposure studies reported O₃-induced airway inflammation following exposures at or above 80 ppb. In the current review, the link between O₃ exposures and airway inflammation and injury has been evaluated in additional controlled human exposure studies, as well as in recent epidemiologic studies. Controlled human exposure studies have generally been conducted in young, healthy adults or in adults with asthma using lavage (proximal airway and bronchoalveolar), bronchial biopsy, and more recently, induced sputum. These studies have evaluated one or more indicators of inflammation, including neutrophil²² (PMN) influx, markers of eosinophilic inflammation, increased permeability of the respiratory epithelium, and/or prevalence of proinflammatory molecules (U.S. EPA, 2013, section 6.2.3.1). Epidemiologic studies have generally evaluated associations between ambient O₃ and markers of inflammation and/or oxidative stress, which plays a key role in initiating and sustaining inflammation (U.S. EPA, 2013, section 6.2.3.2).

There is an extensive body of evidence from controlled human exposure studies indicating that short-term exposures to O₃ can cause pulmonary inflammation. Previously

²² Referred to as either neutrophils or polymorphonuclear neutrophils (or PMNs), these are the most abundant type of white blood cells in mammals. PMNs are recruited to the site of injury following trauma and are the hallmark of acute inflammation. The presence of PMNs in the lung has long been accepted as a hallmark of inflammation and is an important indicator that O₃ causes inflammation in the lungs. Neutrophilic inflammation of tissues indicates activation of the innate immune system and requires a complex series of events, that then are normally followed by processes that clear the evidence of acute inflammation.

available evidence indicated that O₃ causes an inflammatory response in the lungs (U.S. EPA, 1996). A single acute exposure (1-4 hours) of humans to moderate concentrations of O₃ (200-600 ppb) while exercising at moderate to heavy intensities resulted in a number of cellular and biochemical changes in the lung, including inflammation characterized by increased numbers of PMNs, increased permeability of the epithelial lining of the respiratory tract, cell damage, and production of proinflammatory molecules (i.e., cytokines and prostaglandins, U.S. EPA, 2006). A meta-analysis of 21 controlled human exposure studies (Mudway and Kelly, 2004) using varied experimental protocols (80-600 ppb O₃ exposures; 1-6.6 hours exposure duration; light to heavy exercise; bronchoscopy at 0-24 hours post-O₃ exposure) reported that PMN influx in healthy subjects is linearly associated with total O₃ dose. Animal toxicological studies also provided evidence for increases in inflammation and permeability in rabbits at levels as low as 100 ppb O₃ (Section 2.5.3.1, ISA, U.S. EPA, 2013).

Several studies, including one published since the last review (Alexis et al., 2010), have reported O₃-induced increases in PMN influx and permeability following exposures at or above 80 ppb (Alexis et al., 2010; Peden et al., 1997; Devlin et al., 1991), and eosinophilic inflammation following exposures at or above 160 ppb (Scannell et al., 1996; Peden et al., 1997; Hiltermann et al., 1999; Vagaggini et al., 2002). In addition, one recent controlled human exposure study has reported O₃-induced PMN influx following exposures of healthy adults to O₃ concentrations of 60 ppb (Kim et al., 2011), the lowest concentration at which inflammatory responses have been evaluated in human studies.

As with FEV₁ responses to O₃, inflammatory responses to O₃ are generally reproducible within individuals, with some individuals experiencing more severe O₃-induced airway inflammation than indicated by group averages (Holz et al., 2005; Holz et al., 1999). Unlike O₃-induced decrements in lung function, which are attenuated following repeated exposures over several days (U.S. EPA, 2013, section 6.2.1.1), some markers of O₃-induced inflammation and tissue damage remain elevated during repeated exposures, indicating ongoing damage to the respiratory system (U.S. EPA, 2013, section 6.2.3.1, p. 6-81).

Most controlled human exposure studies have reported that asthmatics experience larger O₃-induced inflammatory responses than non-asthmatics. Specifically, asthmatics exposed to 200 ppb O₃ for 4-6 hours with exercise show significantly more neutrophils in bronchoalveolar lavage fluid (BALF) than similarly exposed healthy individuals (Scannell et al., 1996; Basha et al., 1994). Bosson et al. (2003) reported significantly greater expression of a variety of pro-inflammatory cytokines in asthmatics, compared to healthy subjects, following exposure to 200 ppb O₃ for 2 hours. In addition, research available in the last review, combined with a recent study newly available in this review, indicates that pretreatment of asthmatics with corticosteroids can prevent the O₃-induced inflammatory response in induced sputum, though

pretreatment did not prevent FEV₁ decrements (Vagaggini et al., 2001; 2007). In contrast, Stenfors et al. (2002) did not detect a difference in the O₃-induced increases in neutrophil numbers between 15 subjects with mild asthma and 15 healthy subjects by bronchial wash at the 6 hours postexposure time point, although the neutrophil increase in the asthmatic group was on top of an elevated baseline.

In people with allergic airway disease, including people with rhinitis and asthma, evidence available in the last review indicated that proinflammatory mediators also cause accumulation of eosinophils in the airways (Jorres et al., 1996; Peden et al., 1995 and 1997; Frampton et al., 1997; Hiltermann et al., 1999; Holz et al., 2002; Vagaggini et al., 2002). The eosinophil, which increases inflammation and allergic responses, is the cell most frequently associated with exacerbations of asthma (75 CFR 2969, January 19, 2010).

Studies reporting inflammatory responses and markers of lung injury have clearly demonstrated that there is important variation in the responses of exposed subjects (75 FR 2953, January 19, 2010). Some individuals also appear to be intrinsically more susceptible to increased inflammatory responses from O₃ exposure (Holz et al., 2005). In healthy adults exposed to each 80 and 100 ppb O₃, Devlin et al. (1991) observed group average increases in neutrophilic inflammation of 2.1- and 3.8-fold, respectively. However, there was a 20-fold range in inflammatory responses between individuals at both concentrations. Relative to an earlier, similar study conducted at 400 ppb (Koren et al., 1989), Devlin et al. (1991) noted that although some of the study population showed little or no increase in inflammatory and cellular injury indicators analyzed after exposures to lower levels of O₃ (i.e., 80 and 100 ppb), others had changes that were as large as those seen when subjects were exposed to 400 ppb O₃. The data suggest that as a whole the healthy population, on average, may have small inflammatory responses to near-ambient levels of O₃, though there may be a substantial subpopulation that is very sensitive to low levels of O₃. Devlin et al. (1991) expressed the view that “susceptible subpopulations such as the very young, elderly, and people with pulmonary impairment or disease may be even more affected.”

A number of studies report that O₃ exposures increase epithelial permeability. Increased BALF protein, suggesting O₃-induced changes in epithelial permeability, has been reported at 1 hour and 18 hours postexposure (Devlin et al., 1997; Balmes et al., 1996). A meta-analysis of results from 21 publications (Mudway and Kelly, 2004) for varied experimental protocols (80-600 ppb O₃; 1-6.6 hours duration; light to heavy exercise; bronchoscopy at 0-24 hours post-O₃ exposure; healthy subjects), showed that increased BALF protein is associated with total inhaled O₃ dose (i.e., the product of O₃ concentration, exposure duration, and \dot{V}_E). As noted in the 2009 PM ISA (U.S. EPA, 2009), it has been postulated that changes in permeability associated with acute inflammation may provide increased access of inhaled antigens, particles, and other

inhaled substances deposited on lung surfaces to the smooth muscle, interstitial cells, immune cells underlying the epithelium, and the blood (U.S. EPA, 2013, sections 5.3.4, 5.3.5). Animal toxicology studies have provided some support for this hypothesis (Adamson and Prieditis, 1995; Chen et al., 2006), though these studies did not specifically evaluate O₃ exposures (U.S. EPA, 2009). Because of this potentially increased access, it has been postulated that increases in epithelial permeability following O₃ exposure might lead to increases in airway responsiveness to specific and nonspecific agents. In a recent study, Que et al. (2011) investigated this hypothesis in healthy young adults (83M, 55 F) exposed to 220 ppb O₃ for 2.25 hours (alternating 15 min periods of rest and brisk treadmill walking). As has been observed for FEV₁ responses, within-individual changes in permeability were correlated between sequential O₃ exposures, indicating intrinsic differences among individuals in susceptibility to epithelial damage following O₃ exposures. However, increases in epithelial permeability at 1 day post-O₃ exposure were not correlated with changes in airway responsiveness assessed 1 day post-O₃ exposure. The authors concluded that changes in epithelial permeability is relatively constant over time in young healthy adults, although changes in permeability and AHR appear to be mediated by different physiologic pathways.

The limited epidemiologic evidence reviewed in the 2006 O₃ AQCD (U.S. EPA, 2006) demonstrated an association between short-term increases in ambient O₃ concentrations and airways inflammation in children (1-hour max O₃ of approximately 100 ppb). In the 2006 O₃ AQCD (U.S. EPA, 2006), there was limited evidence for increases in nasal lavage levels of inflammatory cell counts and molecules released by inflammatory cells (i.e., eosinophilic cationic protein, and myeloperoxidases). Since 2006, as a result of the development of less invasive methods, there has been a large increase in the number of studies assessing ambient O₃-associated changes in airway inflammation and oxidative stress, the types of biological samples collected, and the types of indicators. Most of these recent studies have evaluated biomarkers of inflammation or oxidative stress in exhaled breath, nasal lavage fluid, or induced sputum (U.S. EPA, 2013, section 6.2.3.2). These recent studies form a larger database to establish coherence with findings from controlled human exposure and animal studies that have measured the same or related biological markers. Additionally, results from these studies provide further biological plausibility for the associations observed between ambient O₃ concentrations and respiratory symptoms and asthma exacerbations.

A number of epidemiologic studies provide evidence that short-term increases in ambient O₃ exposure increase pulmonary inflammation and oxidative stress in children, including those with asthma (Sienra-Monge et al., 2004; Barraza-Villarreal et al., 2008; Romieu et al., 2008;

Berhane et al., 2011). Multiple studies examined and found increases in exhaled NO (eNO)²³ (Berhane et al., 2011; Khatri et al., 2009; Barraza-Villarreal et al., 2008). In some studies of subjects with asthma, increases in ambient O₃ concentration at the same lag were associated with both increases in pulmonary inflammation and respiratory symptoms (Khatri et al., 2009; Barraza-Villarreal et al., 2008). Although more limited in number, epidemiologic studies also found associations with cytokines such as IL-6 or IL-8 (Barraza-Villarreal et al., 2008; Sienna-Monge et al., 2004), eosinophils (Khatri et al., 2009), antioxidants (Sienna-Monge et al., 2004), and indicators of oxidative stress (Romieu et al., 2008) (ISA, Section 6.2.3.2, U.S. EPA, 2013). Because associations with inflammation were attenuated with higher antioxidant intake the study by Sienna-Monge et al. (2004) provides additional evidence that inhaled O₃ is likely to be an important source of reactive oxygen species in airways and/or may increase pulmonary inflammation via oxidative stress-mediated mechanisms among all age groups. Limitations in some recent studies have contributed to inconsistent results in adults (U.S. EPA, 2013, section 6.2.3.2).

Exposure to ambient O₃ on multiple days can result in larger increases in pulmonary inflammation and oxidative stress, as discussed in section 6.2.3.2 of the ISA (U.S. EPA, 2013). In studies that examined multiple O₃ lags, multiday averages of 8-hour maximum or 8-hour average concentrations were associated with larger increases in pulmonary inflammation and oxidative stress (Berhane et al., 2011; Delfino et al., 2010a; Sienna-Monge et al., 2004), consistent with controlled human exposure (U.S. EPA, 2013, section 6.2.3.1) and animal studies (U.S. EPA, 2013, section 6.2.3.3) reporting that some markers of pulmonary inflammation remain elevated with O₃ exposures repeated over multiple days. Evidence from animal toxicological studies also clearly indicates that O₃ exposures result in damage and inflammation in the lung (ISA, Section 5.3, U.S. EPA, 2013). In the few studies that evaluated the potential for confounding, O₃ effect estimates were not confounded by temperature or humidity, and were robust to adjustment for PM_{2.5} or PM₁₀ (Barraza-Villarreal et al., 2008; Romieu et al., 2008; Sienna-Monge et al., 2004).

In conclusion, a relatively small number of controlled human exposure studies evaluating O₃-induced airway inflammation have become available since the last review. For purposes of reviewing the current O₃ NAAQS, the most important of these recent studies reported a statistically significant increase in airway inflammation in healthy adults at moderate exertion following exposures to 60 ppb O₃, the lowest concentration that has been evaluated for inflammation. In addition, a number of recent epidemiologic studies report O₃-associated

²³ Exhaled NO has been shown to be a useful biomarker for airway inflammation in large population-based studies (ISA, U.S. EPA, 2013, Section 7.2.4).

increases in markers of pulmonary inflammation, particularly in children. Thus, recent studies continue to support the evidence for airway inflammation and injury that was available in previous reviews, with new evidence for such effects following exposures to lower concentrations than had been evaluated previously.

Airway Hyperresponsiveness

Airway hyperresponsiveness (AHR) refers to a condition in which the conducting airways undergo enhanced bronchoconstriction in response to a variety of stimuli. Airway hyperresponsiveness is an important consequence of exposure to ambient O₃ because its presence reflects a change in airway smooth muscle reactivity, and indicates that the airways are predisposed to narrowing upon inhalation of a variety of ambient stimuli including specific triggers (i.e., allergens) and nonspecific triggers (e.g., SO₂, and cold air). People with asthma are generally more sensitive to bronchoconstricting agents than those without asthma, and the use of an airway challenge to inhaled bronchoconstricting agents is a diagnostic test in asthma. Standards for airway responsiveness testing have been developed for the clinical laboratory (ATS, 2000), although variation in the methodology for administering the bronchoconstricting agent may affect the results (Cockcroft et al., 2005). There is a wide range of airway responsiveness in people without asthma, and responsiveness is influenced by a number of factors, including cigarette smoke, pollutant exposures, respiratory infections, occupational exposures, and respiratory irritants. Dietary antioxidants have been reported to attenuate O₃-induced bronchial hyperresponsiveness in people with asthma (Trenga et al., 2001).

Evidence for airway hyperresponsiveness following O₃ exposures is derived primarily from controlled human exposure and toxicological studies (U.S. EPA, 2013, section 6.2.2). Airway responsiveness is often quantified by measuring changes in pulmonary function following the inhalation of an aerosolized allergen or a nonspecific bronchoconstricting agent (e.g., methacholine), or following exposure to a bronchoconstricting stimulus such as cold air. In the last review, controlled human exposure studies of mostly adults (≥ 18 years of age) had shown that exposures to O₃ concentrations at or above 80 ppb increase airway responsiveness, as indicated by a reduction in the concentration of specific (e.g., ragweed) and non-specific (e.g., methacholine) agents required to produce a given reduction in lung function (e.g., as measured by FEV₁ or specific airway resistance) (U.S. EPA, 2013, section 6.2.2.1). This O₃-induced AHR has been reported to be dose-dependent (Horstman et al., 1990). Animal toxicology studies have reported O₃-induced airway hyperresponsiveness in a number of species, with some rat strains exhibiting hyperresponsiveness following 4-hour exposures to O₃ concentrations as low as 50 ppb (Depuydt et al., 1999). Since the last review, there have been relatively few new controlled human exposure and animal toxicology studies of O₃ and airway hyperresponsiveness, and no

new studies have evaluated exposures to O₃ concentrations at or below 80 ppb (U.S. EPA, 2013, section 6.2.2.1)

Airway hyperresponsiveness is linked with the accumulation and/or activation of eosinophils in the airways of asthmatics, which is followed by production of mucus and a late-phase asthmatic response (75 FR 2970, January 19, 2010). In a study of 16 intermittent asthmatics, Hiltermann et al. (1999) found that there was a significant inverse correlation between the O₃-induced change in the percentage of eosinophils in induced sputum and the concentration of methacholine causing a 20% decrease in FEV₁. Hiltermann et al. (1999) concluded that the results point to the role of eosinophils in O₃-induced airway hyperresponsiveness. Increases in O₃-induced nonspecific airway responsiveness incidence and duration could have important clinical implications for children and adults with asthma, such as exacerbations of their disease.

Airway hyperresponsiveness after O₃ exposure appears to resolve more slowly than changes in FEV₁ or respiratory symptoms (Folinsbee and Hazucha, 2000). Studies suggest that O₃-induced AHR usually resolves 18 to 24 hours after exposure, but may persist in some individuals for longer periods (Folinsbee and Hazucha, 1989). Furthermore, in studies of repeated exposure to O₃, changes in AHR tend to be somewhat less susceptible to attenuation with consecutive exposures than changes in FEV₁ (Gong et al., 1997; Folinsbee et al., 1994; Kulle et al., 1982; Dimeo et al., 1981) (U.S. EPA, 2013, section 6.2.2). In animal studies a 3-day continuous exposure resulted in attenuation of O₃-induced airway hyperresponsiveness (Johnston et al., 2005) while repeated exposures for 2 hours per day over 10 days did not (Chhabra et al., 2010), suggesting that attenuation could be lost when repeated exposures are interspersed with periods of rest (U.S. EPA, 2013, section 6.2.2.2).

Increases in airway responsiveness do not appear to be strongly associated with decrements in lung function or increases in symptoms (Aris et al., 1995). Recently, Que et al. (2011) assessed methacholine responsiveness in healthy young adults (83M, 55 F) one day after exposure to 220 ppb O₃ and filtered air for 2.25 hours (alternating 15 minute periods of rest and brisk treadmill walking). Increases in airways responsiveness at 1 day post-O₃ exposure were not correlated with FEV₁ responses immediately following the O₃ exposure or with changes in epithelial permeability assessed 1-day post-O₃ exposure. This indicates that airway hyperresponsiveness also appears to be mediated by a differing physiologic pathway.

As mentioned above, in addition to human subjects a number of species, including nonhuman primates, dogs, cats, rabbits, and rodents, have been used to examine the effect of O₃ exposure on airway hyperresponsiveness (see Table 6-14, (U.S. EPA, 1996) of the 1996 O₃ AQCD and Annex Table AX5-12 on page AX5-36 (U.S. EPA, 2006) of the 2006 O₃ AQCD). A body of animal toxicology studies, including some recent studies conducted since the last review,

provides support for the O₃-induced AHR reported in humans (U.S. EPA, 2013, section 6.2.2.2). Although most of these studies evaluated O₃ concentrations above those typically found in ambient air in cities in the United States (i.e., most studies evaluated O₃ concentrations of 100 ppb or greater), one study reported that a very low exposure concentration (50 ppb for 4 hours) induced AHR in some rat strains (Depuydt et al., 1999). Additional recent rodent studies reported O₃-induced AHR following exposures to O₃ concentrations from 100 to 500 ppb (Johnston et al., 2005; Chhabra et al., 2010; Larsen et al., 2010). In characterizing the relevance of these exposure concentrations, the ISA noted that a study using radiolabeled O₃ suggests that even very high O₃ exposure concentrations in rodents could be equivalent to much lower exposure concentrations in humans. Specifically, a 2000 ppb (2 ppm) O₃ exposure concentration in resting rats was reported to be roughly equivalent to a 400 ppb exposure concentration in exercising humans (Hatch et al., 1994). Given this relationship, the ISA noted that animal data obtained in resting conditions could underestimate the risk of effects for humans (U.S. EPA, 2013, section 2.4, p. 2-14).

The 2006 AQCD (U.S. EPA, 2006, p. 6-34) concluded that spirometric responses to O₃ are independent of inflammatory responses and markers of epithelial injury or integrity (Balmes et al., 1996; Blomberg et al., 1999; Torres et al., 1997). Significant inflammatory responses to O₃ exposures that did not elicit significant spirometric responses have been reported (Holz et al., 2005). A recent study (Que et al., 2011) indicates that airway hyper-responsiveness also appears to be mediated by a differing physiologic pathway. These results from controlled human exposure studies indicate that sub-populations of healthy study subjects consistently experience larger than average lung function decrements, greater than average inflammatory responses and pulmonary injury as expressed by increased epithelial permeability, and greater than average airway responsiveness, and that these effects are mediated by apparently different physiologic pathways. Except for lung function decrements, we do not have the concentration- or exposure-response function information about the other, potentially more sensitive,²⁴ clinical endpoints (i.e., inflammation, increased epithelial permeability, airway hyperresponsiveness) that would allow us to quantitatively estimate the size of the population affected and the magnitude of their responses. Moreover, some uncertainties about the exact physiological pathways underlying these endpoints prevents us from knowing whether the exaggerated responses are distributed in sub-populations evenly across the population, or may be clustered with more than one type of exaggerated response in particular sub-populations, or both.

²⁴ CASAC noted that "...[W]hile measures of FEV₁ are quantitative and readily obtainable in humans, they are not the only measures — and perhaps not the most sensitive measures — of the adverse health effects induced by ozone exposure." (Henderson, 2006).

In summary, a strong body of controlled human exposure and animal toxicological studies, most of which were available in the last review of the O₃ NAAQS, report O₃-induced airway hyperresponsiveness after either acute or repeated exposures (U.S. EPA, 2013, section 6.2.2.2). People with asthma often exhibit increased airway responsiveness at baseline relative to healthy controls, and they can experience further increases in responsiveness following exposures to O₃. Studies reporting increased airway responsiveness after O₃ exposure contribute to a plausible link between ambient O₃ exposures and increased respiratory symptoms in asthmatics, and increased hospital admissions and emergency department visits for asthma (U.S. EPA, 2013, section 6.2.2.2).

Respiratory Symptoms and Medication Use

Because respiratory symptoms are associated with adverse outcomes such as limitations in activity, and are the primary reason for people with asthma to use quick relief medication and seek medical care, studies evaluating the link between O₃ exposures and such symptoms allow a more direct characterization of the clinical and public health significance of ambient O₃ exposure than measures of lung function decrements and pulmonary inflammation. Controlled human exposure and toxicological studies have described modes of action through which short-term O₃ exposures may increase respiratory symptoms by demonstrating O₃-induced airway hyperresponsiveness (U.S. EPA, 2013, section 6.2.2) and pulmonary inflammation (U.S. EPA, 2013, section 6.2.3).

The link between subjective respiratory symptoms and O₃ exposures has been evaluated in both controlled human exposure and epidemiologic studies, and the link with medication use has been evaluated in epidemiologic studies. In the last review, several controlled human exposure studies reported respiratory symptoms following exposures to O₃ concentrations at or above 80 ppb. In addition, one study reported such symptoms following exposures to 60 ppb O₃, though the increase was not statistically different from filtered air controls. Epidemiologic studies reported associations between ambient O₃ and respiratory symptoms and medication use in a variety of locations and populations, including asthmatic children living in U.S. cities. In the current review, additional controlled human exposure studies have evaluated respiratory symptoms following exposures to O₃ concentrations below 80 ppb and recent epidemiologic studies have evaluated associations with respiratory symptoms and medication use (U.S. EPA, 2013, sections 6.2.1, 6.2.4).

In controlled human exposure studies available in the last review as well as newly available studies, statistically significant increases in respiratory symptoms have been consistently reported in healthy adult volunteers engaged in intermittent, moderate exertion following 6.6 hour exposures to average O₃ concentrations at or above 80 ppb (Adams, 2003; Adams, 2006; Schelegle et al., 2009). Such symptoms have been reported to increase with

increasing O₃ exposure concentrations, duration of exposure, and activity level (McDonnell et al., 1999). For example, in a study available during the last review, Adams (2006) reported an increase in respiratory symptoms in healthy adults during a 6.6 hour exposure protocol with an average O₃ exposure concentration of 60 ppb. This increase was significantly different from initial respiratory symptoms, but not from filtered air controls. Two recent controlled human exposure studies that have become available since the last review did not report statistically significant increases in respiratory symptoms following exposures of healthy adults to 60 ppb O₃ (Schelegle et al., 2009; Kim et al., 2011). A recent study by Schelegle et al. (2009) did report a statistically significant increase in respiratory symptoms in healthy adults following 6.6 hour exposures to an average O₃ concentration of 70 ppb. The findings for O₃-induced respiratory symptoms in controlled human exposure studies, and the evidence integrated across disciplines describing underlying modes of action, provide biological plausibility for epidemiologic associations observed between short-term increases in ambient O₃ concentration and increases in respiratory symptoms (U.S. EPA, 2013, section 6.2.4).

In epidemiologic studies of respiratory symptoms, data typically are collected by having subjects (or their parents) record symptoms and medication use in a diary without direct supervision by study staff. Several limitations of symptom reports are well recognized, as described in the ISA (U.S. EPA, 2013, section 6.2.4). Nonetheless, symptom diaries remain a convenient tool to collect individual-level data from a large number of subjects and allow modeling of associations between daily changes in O₃ concentration and daily changes in respiratory morbidity over multiple weeks or months. Importantly, many of the limitations in these studies are sources of random measurement error that can bias effect estimates to the null or increase the uncertainty around effect estimates (U.S. EPA, 2013, Section 6.2.4). Because respiratory symptoms are associated with limitations in activity and daily function and are the primary reason for using medication and seeking medical care, the evidence is directly coherent with the associations consistently observed between increases in ambient O₃ concentration and increases in asthma emergency department visits, discussed below (U.S. EPA, 2013, Section 6.2.4).

Most epidemiologic studies of O₃ and respiratory symptoms and medication use have been conducted in children and/or adults with asthma, with fewer studies, and less consistent results, in non-asthmatic populations (U.S. EPA, 2013, section 6.2.4). The 2006 AQCD (U.S. EPA, 2006, U.S. EPA, 2013, section 6.2.4) concluded that the collective body of epidemiologic evidence indicated that short-term increases in ambient O₃ concentrations are associated with increases in respiratory symptoms in children with asthma. A large body of single-city and single-region studies of asthmatic children provides consistent evidence for associations between

short-term increases in ambient O₃ concentrations and increased respiratory symptoms and asthma medication use in children with asthma (U.S. EPA, 2013, Figure 6-12, Table 6-20).

Methodological differences among studies make comparisons across recent multicity studies of respiratory symptoms difficult. Because of fewer person-days of data (Schildcrout et al., 2006) or examination of 19-day averages of ambient O₃ concentrations (O'Connor et al., 2008), the ISA did not give greater weight to results from recent multicity studies than results from single-city studies (U.S. EPA, 2013, section 6.2.4.5). While evidence from the few available U.S. multicity studies is less consistent (O'Connor et al., 2008; Schildcrout et al., 2006; Mortimer et al., 2002), the overall body of epidemiologic evidence with respect to the association between exposure to O₃ and respiratory symptoms in asthmatic children remains compelling (U.S. EPA, 2013, section 6.2.4.1). Findings from a small body of studies indicate that O₃ is also associated with increased respiratory symptoms in adults with asthma (Khatri et al., 2009; Feo Brito et al., 2007; Ross et al., 2002) (U.S. EPA, 2013, section 6.2.4.2).

Available evidence indicates that O₃-associated increases in respiratory symptoms are not confounded by temperature, pollen, or copollutants (primarily PM) (U.S. EPA, 2013, section 6.2.4.5; Table 6-25; Romieu et al., 1996; Romieu et al., 1997; Thurston et al., 1997; Gent et al., 2003). However, identifying the independent effects of O₃ in some studies was complicated due to the high correlations observed between O₃ and PM or different lags and averaging times examined for copollutants. Nonetheless, the ISA noted that the robustness of associations in some studies of individuals with asthma, combined with findings from controlled human exposure studies for the direct effects of O₃ exposure, provide substantial evidence supporting the independent effects of short-term ambient O₃ exposure on respiratory symptoms (U.S. EPA, 2013, section 6.2.4.5).

Epidemiologic studies of medication use have reported associations with 1-hour maximum O₃ concentrations and with multiday average O₃ concentrations (Romieu et al., 2006; Just et al., 2002). Some studies reported O₃ associations for both respiratory symptoms and asthma medication use (Escamilla-Nuñez et al., 2008; Romieu et al., 2006; Schildcrout et al., 2006; Jalaludin et al., 2004; Romieu et al., 1997; Thurston et al., 1997) while others reported associations for either respiratory symptoms or medication use (Romieu et al., 1996; Rabinovitch et al., 2004; Just et al., 2002; Ostro et al., 2001).

In summary, both controlled human exposure and epidemiologic studies have reported respiratory symptoms attributable to short-term O₃ exposures. In the last review, the majority of the evidence from controlled human exposure studies in young, healthy adults was for symptoms following exposures to O₃ concentrations at or above 80 ppb. Although studies that have become available since the last review have not reported respiratory symptoms in young, healthy adults following exposures with moderate exertion to 60 ppb, one recent study has reported increased

symptoms in young, healthy adults while at moderate exertion following exposures to O₃ concentrations as low as 70 ppb.²⁵ As was concluded in the 2006 O₃ AQCD (U.S. EPA, 2006, 1996), the collective body of epidemiologic evidence indicates that short-term increases in ambient O₃ concentration are associated with increases in respiratory symptoms in children with asthma (U.S. EPA, 2013, section 6.2.4). Recent studies of respiratory symptoms and medication use, primarily in asthmatic children, add to this evidence. In a smaller body of studies, increases in ambient O₃ concentration were associated with increases in respiratory symptoms in adults with asthma.

Lung Host Defense

The mammalian respiratory tract has a number of closely integrated defense mechanisms that, when functioning normally, provide protection from the potential health effects of exposures to a wide variety of inhaled particles and microbes. These defense mechanisms include mucociliary clearance, alveolobronchiolar transport mechanism, alveolar macrophages²⁶, and adaptive immunity²⁷ (U.S. EPA, 2013, section 6.2.5). The previous O₃ AQCD (U.S. EPA, 2006) concluded that animal toxicological studies provided evidence that acute exposure to O₃ concentrations as low as 100 to 500 ppb can increase susceptibility to infectious diseases due to modulation of these lung host defenses. This conclusion was based in large part on animal studies of alveolar macrophage functioning and mucociliary clearance (U.S. EPA, 2013, section 6.2.5).

With regard to alveolar macrophage functioning, the previous O₃ AQCD (U.S. EPA, 2006) concluded that short periods of O₃ exposure can cause a reduction in the number of free alveolar macrophages available for pulmonary defense, and that these alveolar macrophages are more fragile, less able to engulf particles (i.e., phagocytic), and exhibit decreased lysosomal²⁸ enzyme activities (U.S. EPA, 2013, section 6.2.5). These conclusions were based largely on studies conducted in animals exposed for several hours up to several weeks to O₃ concentrations from 100 to 250 ppb (Hurst et al., 1970; Driscoll et al., 1987; Cohen et al., 2002). Consistent with the animal evidence, a controlled human exposure study available in the last review had reported decrements in the ability of alveolar macrophages to phagocytize yeast following exposures of healthy volunteers to O₃ concentrations of 80 and 100 ppb for 6.6-hour during

²⁵As noted above, for the 70 ppb exposure concentration Schelegle et al. (2009) reported that the actual mean exposure concentration was 72 ppb.

²⁶ Phagocytic white blood cells within the alveoli of the lungs that ingest inhaled particles.

²⁷ The adaptive immune system, is also known as the acquired immune system. Acquired immunity creates immunological memory after an initial response to a specific pathogen, leading to an enhanced response to subsequent encounters with that same pathogen.

²⁸ Lysosomes are cellular organelles that contain acid hydrolase enzymes that break down waste materials and cellular debris.

moderate exercise (Devlin et al., 1991). Integrating the animal study results with human exposure evidence available in the 1996 Criteria Document, the 2006 Criteria Document concluded that available evidence indicates that short-term O₃ exposures have the potential to impair host defenses in humans, primarily by interfering with alveolar macrophage function. Any impairment in alveolar macrophage function may lead to decreased clearance of microorganisms or nonviable particles. Compromised alveolar macrophage functions in asthmatics may increase their susceptibility to other O₃ effects, the effects of particles, and respiratory infections (EPA, 2006, p. 8–26).

With regard to mucociliary clearance, in the last review a number of studies conducted in different animal species had reported morphological damage to the cells of the tracheobronchial tree from acute and sub-chronic exposure to O₃ concentrations at or above 200 ppb. The cilia were either completely absent or had become noticeably shorter or blunt. In general, functional studies of mucociliary transport had observed a delay in particle clearance soon after acute exposure, with decreased clearance more evident at higher doses (1 ppm) (U.S. EPA, 2013, section 6.2.5.1).

Alveolobronchiolar transport mechanisms refers to the transport of particles deposited in the deep lung (alveoli) which may be removed either up through the respiratory tract (bronchi) by alveolobronchiolar transport or through the lymphatic system. The pivotal mechanism of alveolobronchiolar transport involves the movement of alveolar macrophages with ingested particles to the bottom of the conducting airways. These airways are lined with ciliated epithelial cells and cells that produce mucous, which surrounds the macrophages. The ciliated epithelial cells move the mucous packets up the respiratory tract, hence the term “mucociliary escalator.” Although some studies show reduced tracheobronchial clearance after O₃ exposure, alveolar clearance of deposited material is accelerated, presumably due to macrophage influx, which in itself can be damaging.

With regard to adaptive immunity, a limited number of epidemiologic studies have examined associations between O₃ exposure and hospital admissions or ED visits for respiratory infection, pneumonia, or influenza. Results have been mixed, and in some cases conflicting (U.S. EPA, 2013, Sections 6.2.7.2 and 6.2.7.3). With the exception of influenza, it is difficult to ascertain whether cases of respiratory infection or pneumonia are of viral or bacterial etiology. A recent study that examined the association between O₃ exposure and respiratory hospital admissions in response to an increase in influenza intensity did observe an increase in respiratory hospital admissions (Wong et al., 2009), but information from toxicological studies of O₃ and viral infections is ambiguous.

In summary, relatively few studies conducted since the last review have evaluated the effects of O₃ exposures on lung host defense. When the available evidence is taken as a whole,

the ISA concludes that acute O₃ exposures impair the host defense capability of animals, primarily by depressing alveolar macrophage function and perhaps also by decreasing mucociliary clearance of inhaled particles and microorganisms. Coupled with limited evidence from controlled human exposure studies, this suggests that humans exposed to O₃ could be predisposed to bacterial infections in the lower respiratory tract (EPA, 2013, section 6.2.5.5). The seriousness of such infections may depend on how quickly bacteria develop virulence factors and how rapidly PMNs are mobilized to compensate for the deficit in alveolar macrophage function.

Allergic and Asthma-Related Responses

Effects resulting from combined exposures to O₃ and allergens have been studied in a variety of animal species, generally as models of experimental asthma. Pulmonary function and AHR in animal models of asthma are discussed in detail in Section 6.2.1.3 and Section 6.2.2.2, respectively, in the ISA (U.S. EPA, 2013). Studies of allergic and asthma-related responses are discussed in detail in sections 5.3.6 and 6.2.6 of the ISA (U.S. EPA, 2013).

Evidence available in the last review indicates that O₃ exposure skews immune responses toward an allergic phenotype. For example, Gershwin et al. (1981) reported that O₃ (800 and 500 ppb for 4 days) exposure caused a 34-fold increase in the number of IgE (allergic antibody)-containing cells in the lungs of mice. In general, the number of IgE-containing cells correlated positively with levels of anaphylactic sensitivity. In humans, allergic rhinoconjunctivitis symptoms are associated with increases in ambient O₃ concentrations (Riediker et al., 2001). Controlled human exposure studies have observed O₃-induced changes indicating allergic skewing. Airway eosinophils, which are white blood cells that participate in allergic disease and inflammation, were observed to increase in volunteers with atopy²⁹ and mild asthma (Peden et al., 1997). In a more recent study, expression of IL-5, a cytokine involved in eosinophil recruitment and activation, was increased in subjects with atopy but not in healthy subjects (Hernandez et al., 2010). Epidemiologic studies describe associations between eosinophils in both short- (U.S. EPA, 2013, Section 6.2.3.2) and long-term (U.S. EPA, 2013, Section 7.2.5) O₃ exposure, as do chronic exposure studies in non-human primates. Collectively, findings from these studies suggest that O₃ can induce or enhance certain components of allergic inflammation in individuals with allergy or allergic asthma.

Evidence available in the last review indicates that ozone may also increase AHR to specific allergen triggers (75 FR 2970, January 19, 2010). Two studies (Jörres et al., 1996; Holz et al., 2002) observed increased airway responsiveness to O₃ exposure with bronchial allergen

²⁹ Atopy is a predisposition toward developing certain allergic hypersensitivity reactions. A person with atopy typically presents with one or more of the following: eczema (atopic dermatitis), allergic rhinitis (hay fever), allergic conjunctivitis, or allergic asthma.

challenge in subjects with preexisting allergic airway disease. Ozone-induced exacerbation of airway responsiveness persists longer and attenuates more slowly than O₃-induced lung function decrements and respiratory symptom responses and can have important clinical implications for asthmatics.

Animal toxicology studies indicate that O₃ enhances inflammatory and allergic responses to allergen challenge in sensitized animals. In addition to exacerbating existing allergic responses, toxicology studies indicate that O₃ can also act as an adjuvant to produce sensitization in the respiratory tract. Along with its pro-allergic effects (inducing or enhancing certain components of allergic inflammation in individuals with allergy or allergic asthma), O₃ could also make airborne allergens more allergenic. When combined with NO₂, O₃ has been shown to enhance nitration of common protein allergens, which may increase their allergenicity Franze et al. (2005).

Hospital Admissions and Emergency Department Visits

The 2006 O₃ AQCD evaluated numerous studies of respiratory-related emergency department visits and hospital admissions. These were primarily time-series studies conducted in the U.S., Canada, Europe, South America, Australia, and Asia. Based on such studies, the 2006 O₃ AQCD concluded that “the overall evidence supports a causal relationship between acute ambient O₃ exposures and increased respiratory morbidity resulting in increased ED visits and [hospital admissions] during the warm season³⁰” (U.S. EPA, 2006). This conclusion was “strongly supported by the human clinical, animal toxicologic[al], and epidemiologic evidence for [O₃-induced] lung function decrements, increased respiratory symptoms, airway inflammation, and airway hyperreactivity” (U.S. EPA, 2006).

The results of recent studies largely support the conclusions of the 2006 O₃ AQCD (U.S. EPA, 2013, section 6.2.7). Since the completion of the 2006 O₃ AQCD, relatively fewer studies conducted in the U.S., Canada, and Europe have evaluated associations between short-term O₃ concentrations and respiratory hospital admissions and emergency department visits, with a growing number of studies conducted in Asia. This epidemiologic evidence is summarized in Appendix 3A and discussed in detail in the ISA (U.S. EPA, 2013, section 6.2.7).

In considering this body of evidence, the ISA focused primarily on multicity studies because they examine associations with respiratory-related hospital admissions and emergency department visits over large geographic areas using consistent statistical methodologies (U.S. EPA, 2013, section 6.2.7.1). The ISA also focused on single-city studies that encompassed a large number of daily hospital admissions or emergency department visits, included long study-

³⁰Epidemiologic associations for O₃ are more robust during the warm season than during cooler months (e.g., smaller measurement error, less potential confounding by copollutants). Rationale for focusing on warm season epidemiologic studies for O₃ can be found at 72 FR 37838-37840.

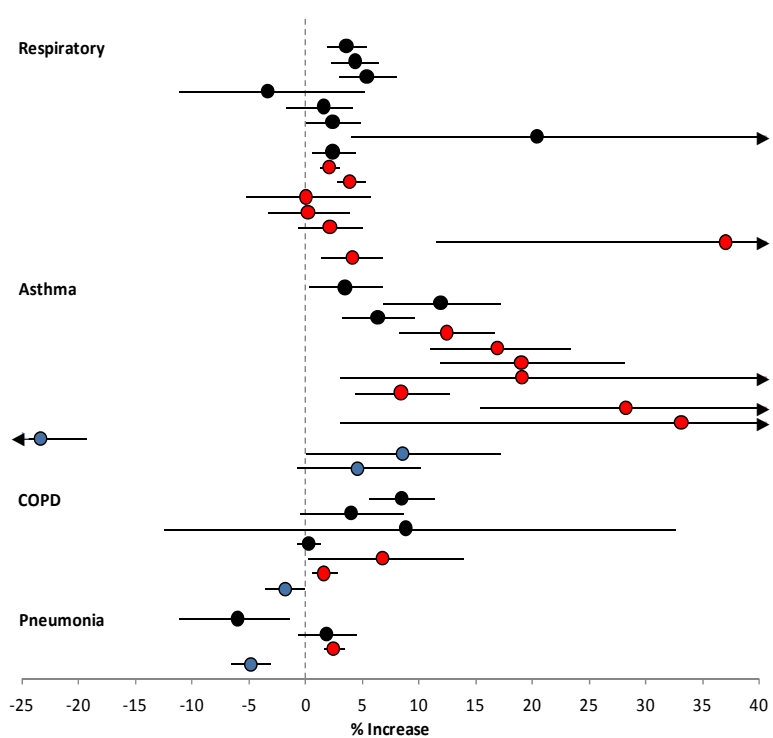
durations, were conducted in locations not represented by the larger studies, or examined population-specific characteristics that may increase the risk of O₃-related health effects but were not evaluated in the larger studies (U.S. EPA, 2013, section 6.2.7.1). When examining the association between short-term O₃ exposure and respiratory health effects that require medical attention, the ISA distinguishes between hospital admissions and emergency department visits because it is likely that a small percentage of respiratory emergency department visits will be admitted to the hospital; therefore, respiratory emergency department visits may represent potentially less serious, but more common outcomes (U.S. EPA, 2013, section 6.2.7.1).

Several recent multicity studies (e.g., Cakmak et al., 2006; Dales et al., 2006) and a multi-continent study (Katsouyanni et al., 2009) report associations between short-term O₃ concentrations and increased respiratory-related hospital admissions and emergency department visits. These multicity studies are supported by single-city studies also reporting consistent positive associations using different exposure assignment approaches (i.e., average of multiple monitors, single monitor, population-weighted average) and averaging times (i.e., 1-hour max and 8-hour max) (U.S. EPA, 2013, sections 6.2.7.1 to 6.2.7.5). When examining cause-specific respiratory outcomes, recent studies report positive associations with hospital admissions and emergency department visits for asthma (Strickland et al., 2010; Stieb et al., 2009) and COPD (Stieb et al., 2009; Medina-Ramon et al., 2006), with more limited evidence for pneumonia (Medina-Ramon et al., 2006; Zanobetti and Schwartz, 2006). In seasonal analyses (Figure 3-2 below; U.S. EPA, 2013, Figure 6-19, Table 6-28), stronger associations were reported in the warm season or summer months (red circles), when O₃ concentrations are higher, compared to the cold season (blue circles), particularly for asthma (Strickland et al., 2010; Ito et al., 2007) and COPD (Medina-Ramon et al., 2006).³¹ The available evidence indicates that children are at greatest risk for O₃-induced respiratory effects (Silverman and Ito, 2010; Strickland et al., 2010; Mar and Koenig, 2009; Villeneuve et al., 2007; Dales et al., 2006).

Although the collective evidence across studies indicates a mostly consistent positive association between O₃ exposure and respiratory-related hospital admissions and ED visits, the magnitude of these associations may be underestimated due to behavioral modification in response to air quality forecasts (U.S. EPA, 2013, Section 4.6.6).

³¹ The study by Strickland et al. (2010) is discussed in more detail in section 3.1.4.2, below.

Study	Location	Visit Type	Age	Lag
Wong et al. (2009)	Hong Kong	HA	All	0-1
Cakmak et al. (2006)	10 Canadian cities	HA	All	1,2
Dales et al. (2006)	11 Canadian cities	HA	0-27 days	2
Orazio et al. (2009) ^a	6 Italian cities	ED	0-2	0-6
Katsouyanni et al. (2009)	APHENA-Europe	HA	65+	0-1
	APHENA-U.S.	HA	65+	0-1
	APHENA-Canada	HA	65+	DL (0-2)
	APHENA-Canada	HA	65+	DL (0-2) ^b
Darrow et al. (2009)	Atlanta	ED	All	1
Tolbert et al. (2007)	Atlanta	ED	All	0-2
Biggeri et al. (2005) ^c	8 Italian cities	HA	All	0-3
Katsouyanni et al. (2009)	APHENA-Europe	HA	65+	0-1
	APHENA-U.S.	HA	65+	0-1
	APHENA-Canada	HA	65+	DL (0-2)
	APHENA-Canada	HA	65+	DL (0-2) ^b
Stieb et al. (2009)	7 Canadian Cities	ED	All	2
Villeneuve et al. (2007)	Alberta, CAN	ED	> 2	0-2
Strickland et al. (2010)	Atlanta	ED	Children	0-2
Silverman and Ito (2010) ^d	New York	HA	All	0-1
Ito et al. (2007)	New York	ED	All	0-1
Villeneuve et al. (2007)	Alberta, CAN	ED	> 2	0-2
Mar and Koenig (2009)	Seattle, WA	ED	18+	2
Strickland et al. (2010)	Atlanta	ED	Children	0-2
Silverman and Ito (2010) ^d	New York	HA	6-18	0-1
Mar and Koenig (2009)	Seattle, WA	ED	<18	0
Ito et al. (2007)	New York	ED	All	0-1
Villeneuve et al. (2007)	Alberta, CAN	ED	> 2	0-2
Strickland et al. (2010)	Atlanta	ED	Children	0-2
Wong et al. (2009)	Hong Kong	HA	All	0-1
Stieb et al. (2009)	7 Canadian Cities	ED	All	2
Yang et al. (2006)	Vancouver	HA	65+	0-3
Medina-Ramon et al. (2006)	36 U.S. cities	HA	65+	DL (0-1)
Stieb et al. (2009) ^e	7 Canadian Cities	ED	All	NR
Medina-Ramon et al. (2006)	36 U.S. cities	HA	65+	DL (0-1)
	36 U.S. cities	HA	65+	DL (0-1)
Zanobetti and Schwartz (2006)	Boston	HA	65+	0-1
Medina-Ramon et al. (2006)	36 U.S. cities	HA	65+	DL (0-1)
	36 U.S. cities	HA	65+	DL (0-1)
	36 U.S. cities	HA	65+	DL (0-1)



Note: Effect estimates are for a 20 ppb increase in 24-hour; 30 ppb increase in 8-hour max; and 40 ppb increase in 1-hour max O₃ concentrations. HA=hospital admission; ED=emergency department. Black=All-year analysis; Red=Summer only analysis; Blue=Winter only analysis.

^a Wheeze used as indicator of lower respiratory disease.

^b APHENA-Canada results standardized to approximate IQR of 5.1 ppb for 1-h max O₃ concentrations.

^c Study included 8 cities; but of those 8, only 4 had O₃ data.

^d non-ICU effect estimates.

^e The study did not specify the lag day of the summer season estimate.

Figure 3-2. Percent increase in respiratory-related hospital admission and emergency department visits in studies that presented all-year and/or seasonal results.

Studies examining the potential confounding effects of copollutants have reported that O₃ effect estimates remained relatively robust upon the inclusion of PM and gaseous pollutants in two-pollutant models (U.S. 2013, Figure 6-20, Table 6-29). Additional studies that conducted copollutant analyses, but did not present quantitative results, also support these conclusions (Strickland et al., 2010; Tolbert et al., 2007; Medina-Ramon et al., 2006) (U.S. 2013, section 6.2.7.5).

In the last review, studies had not evaluated the concentration-response relationship between short-term O₃ exposure and respiratory-related hospital admissions and emergency department visits. A preliminary examination of this relationship in studies that have become available since the last review found no evidence of a deviation from linearity when examining the association between short-term O₃ exposure and asthma hospital admissions (U.S. EPA, 2013, page 6-157, and Silverman and Ito, 2010). In addition, an examination of the concentration-response relationship for O₃ exposure and pediatric asthma emergency department visits found no evidence of a threshold at O₃ concentrations as low as 30 ppb (for daily maximum 8-hour concentrations) (Strickland et al., 2010). However, in both studies there is uncertainty in the shape of the concentration-response curve at the lower end of the distribution of O₃ concentrations due to the low density of data in this range (U.S. 2013, page 6-157).

Respiratory Mortality

The controlled human exposure, epidemiologic, and toxicological studies discussed in section 6.2 of the ISA (U.S. EPA, 2013, section 6.2) provide strong evidence for respiratory morbidity effects, including ED visits and hospital admissions, in response to short-term O₃ exposures. Moreover, evidence from experimental studies indicates multiple potential pathways of respiratory effects from short-term O₃ exposures, which support the continuum of respiratory effects that could potentially result in respiratory-related mortality in adults (U.S. EPA, 2013, section 6.2.8). The 2006 O₃ AQCD found inconsistent evidence for associations between short-term O₃ concentrations and respiratory mortality (U.S. EPA, 2006). Although some studies reported a strong positive association between O₃ and respiratory mortality, additional studies reported small associations or no associations. New epidemiologic evidence for respiratory mortality is discussed in detail in section 6.2.8 of the ISA (U.S. EPA, 2013). The majority of recent multicity studies have reported positive associations between short-term O₃ exposures and respiratory mortality, particularly during the summer months (U.S. EPA, 2013, Figure 6-36).

Specifically, recent multicity studies from the U.S. (Zanobetti and Schwartz, 2008b), Europe (Samoli et al., 2009), Italy (Stafoggia et al., 2010), and Asia (Wong et al., 2010), as well as a multi-continent study (Katsouyanni et al., 2009), reported associations between short-term O₃ concentrations and respiratory mortality (U.S. EPA, 2013, Figure 6-37, page 6-259). With

respect to respiratory mortality, summer-only analyses were consistently positive and most were statistically significant. In all-year analyses associations were positive, but smaller in magnitude.

Of the studies evaluated, only the studies by Katsouyanni et al. (2009) and by Stafoggia et al. (2010) analyzed the potential for copollutant confounding of the O₃-respiratory mortality relationship. Based on the results of these analyses, the ISA concluded that O₃ respiratory mortality risk estimates appear to be moderately to substantially sensitive (e.g., increased or attenuated) to inclusion of PM₁₀. However, in the APHENA study (Katsouyanni et al., 2009), the mostly every-6th-day sampling schedule for PM₁₀ in the Canadian and U.S. datasets greatly reduced their sample size and limits the interpretation of these results (U.S. EPA, 2013, section 6.2.8).

In summary, recent epidemiologic studies support and reinforce the epidemiologic evidence for O₃-associated respiratory hospital admissions and emergency department visits from the last review. In addition, the evidence for associations with respiratory mortality has been strengthened considerably since the last review, with the addition of several large multicity studies. The biological plausibility of the associations reported in these studies is supported by the experimental evidence for respiratory effects.

3.1.2.2 Respiratory Effects – Long-term Exposures

- **To what extent does the currently available scientific evidence, including related uncertainties, strengthen or alter our understanding from the last review of respiratory effects attributable to long-term O₃ exposures?**

As recognized in section 3.1.2.1, “the clearest evidence for health effects associated with exposure to O₃ is provided by studies of respiratory effects” (U.S. EPA, 2013, section 1, p. 1-6). Collectively, there is a vast amount of evidence spanning several decades that supports a causal association between exposure to O₃ and a continuum of respiratory effects (U.S. EPA, 2013, section 2.5). While the majority of this evidence is derived from studies investigating short-term exposures, evidence from animal toxicological studies and recent epidemiologic evidence indicate that long-term exposures (i.e., months to years) may also be detrimental to the respiratory system. Across this evidence, particularly the epidemiologic evidence, the exposures of focus vary, often involving repeated short concentrations extending over a long period, rather than a continuous long-term exposure period.

In the 2006 O₃ AQCD, evidence was examined for relationships between long-term O₃ exposure and effects on respiratory health outcomes including declines in lung function, increases in inflammation, and development of asthma in children and adults. Animal toxicology data provided a clearer picture indicating that long-term O₃ exposure may have lasting effects.

Thus, while the study by Jerrett et al. (2009) contributes to our understanding of health effects associated with ambient O₃ (as summarized in section 3.1.2 above), it is less informative regarding the extent to which those health effects may be occurring under air quality conditions allowed by the current standard.

3.1.5 Public Health Implications

In this section, we address the public health implications of O₃ exposures with respect to the factors that put populations at increased risk from exposures (section 3.1.5.1), the size of at-risk populations (section 3.1.5.2), and the potential effects of averting behavior on reducing O₃ exposures and associated health effects (section 3.1.5.3). Providing appropriate public health protection requires consideration of the factors that put populations at greater risk from O₃ exposure. In order to estimate potential public health impacts, it is important to consider not only the adversity of the health effects, but also the populations at greater risk and potential behaviors that may reduce exposure.

3.1.5.1 At-Risk Populations

In this section we address the following question:

- **To what extent does the currently available scientific evidence expand our understanding of at-risk populations?**

The currently available evidence expands our understanding of populations that were identified to be at greater risk of O₃-related health effects at the time of the last review (i.e., people who are active outdoors, people with lung disease, children and older adults and people with increased responsiveness to O₃) and supports the identification of additional factors that may lead to increased risk (U.S. EPA, 2006, section 3.6.2; U.S. EPA, 2013, chapter 8). Populations and lifestages may be at greater risk for O₃-related health effects due to factors that contribute to their susceptibility and/or vulnerability to ozone. The definitions of susceptibility and vulnerability have been found to vary across studies, but in most instances “susceptibility” refers to biological or intrinsic factors (e.g., lifestage, sex, preexisting disease/conditions) while “vulnerability” refers to non-biological or extrinsic factors (e.g., socioeconomic status [SES]) (U.S. EPA, 2013, p. 8-1). In some cases, the terms “at-risk” and “sensitive” have been used to encompass these concepts more generally. In the ISA and this PA, “at-risk” is the all-encompassing term used to define groups with specific factors that increase their risk of O₃-related health effects. Further discussion of at-risk populations can be found below.

There are multiple avenues by which groups may experience increased risk for O₃-related health effects. A population or lifestage⁶² may exhibit greater effects than other populations or lifestages exposed to the same concentration or dose, or they may be at greater risk due to increased exposure to an air pollutant (e.g., time spent outdoors). A group with intrinsically increased risk would have some factor(s) that increases risk through a biological mechanism and, in general, would have a steeper concentration-risk relationship, compared to those not in the group. Factors that are often considered intrinsic include pre-existing asthma, genetic background, and lifestage. A group of people could also have extrinsically increased risk, which would be through an external, non-biological factor, such as socioeconomic status (SES) and diet. Some groups are at risk of increased internal dose at a given exposure concentration, for example, because of breathing patterns. This category would include people who work or exercise outdoors. Finally, there are those who might be placed at increased risk for experiencing greater exposures by being exposed to higher O₃ concentrations. This would include, for example, groups of people with greater exposure to ambient O₃ due to less availability or use of home air conditioners such that they are more likely to be in locations with open windows on high ozone days. Some groups may be at increased risk of O₃-related health effects through a combination of factors. For example, children tend to spend more time outdoors when O₃ levels are high, and at higher levels of activity than adults, which leads to increased exposure and dose, and they also have biological, or intrinsic, risk factors (e.g., their lungs are still developing) (U.S. EPA, 2013, Chapter 8). An at-risk population or lifestage is more likely to experience adverse health effects related to O₃ exposures and/or, develop more severe effects from exposure than the general population.

People with Specific Genetic Variants

Overall, for variants in multiple genes there is adequate evidence for involvement in populations being more at-risk than others to the effects of O₃ exposure on health (U.S. EPA, 2013, section 8.1). Controlled human exposure and epidemiologic studies have reported evidence of O₃-related increases in respiratory symptoms or decreases in lung function with variants including GSTM1, GSTP1, HMOX1, and NQO1. NQO1 deficient mice were found to be resistant to O₃-induced AHR and inflammation, providing biological plausibility for results of studies in humans. Additionally, studies of rodents have identified a number of other genes that may affect O₃-related health outcomes, including genes related to innate immune signaling and pro- and anti-inflammatory genes, which have not been investigated in human studies.

People with Asthma

⁶² Lifestages, which in this case includes childhood and older adulthood, are experienced by most people over the course of a lifetime, unlike other factors associated with at-risk populations.

Previous O₃ AQCDs identified individuals with asthma as a population at increased risk of O₃-related health effects. Multiple new epidemiologic studies included in the ISA have evaluated the potential for increased risk of O₃-related health effects in people with asthma, including: lung function; symptoms; medication use; airway hyperresponsiveness (AHR); and airway inflammation (also measured as exhaled nitric oxide fraction, or FeNO). A study of lifeguards in Texas reported decreased lung function with short-term O₃ exposure among both individuals with and without asthma, however, the decrease was greater among those with asthma (Thaller et al., 2008). A Mexican study of children ages 6-14 detected an association between short-term O₃ exposure and wheeze, cough, and bronchodilator use among asthmatics but not non-asthmatics, although this may have been the result of a small non-asthmatic population (Escamilla-Nuñez et al., 2008). A study of modification by AHR (an obligate condition among asthmatics) reported greater short-term O₃-associated decreases in lung function in elderly individuals with AHR, especially among those who were obese (Alexeeff et al., 2007). With respect to airway inflammation, in one study, a positive association was reported for airway inflammation among asthmatic children following short-term O₃ exposure, but the observed association was similar in magnitude to that of non-asthmatics (Barraza-Villarreal et al., 2008). Similarly, another study of children in California reported an association between O₃ concentration and FeNO that persisted both among children with and without asthma as well as those with and without respiratory allergy (Berhane et al., 2011). Finally, Khatri et al. (2009) found no association between short-term O₃ exposure and altered lung function for either asthmatic or non-asthmatic adults, but did note a decrease in lung function among individuals with allergies.

New evidence for difference in effects among asthmatics has been observed in studies that examined the association between O₃ exposure and altered lung function by asthma medication use. A study of children with asthma living in Detroit reported a greater association between short-term O₃ and lung function for corticosteroid users compared with noncorticosteroid users (Lewis et al., 2005). Conversely, another study found decreased lung function among noncorticosteroid users compared to users, although in this study, a large proportion of non-users were considered to be persistent asthmatics (Hernández-Cadena et al., 2009). Lung function was not related to short-term O₃ exposure among corticosteroid users and non-users in a study taking place during the winter months in Canada (Liu et al., 2009). Additionally, a study of airway inflammation reported a counterintuitive inverse association with O₃ of similar magnitude for all groups of corticosteroid users and non-users (Qian et al., 2009).

Controlled human exposure studies that have examined the effects of O₃ on adults with asthma and healthy controls are limited. Based on studies reviewed in the 1996 and 2006 O₃ AQCDs, subjects with asthma appeared to be more sensitive to acute effects of O₃ in terms of

FEV₁ and inflammatory responses than healthy non-asthmatic subjects. For instance, Horstman et al. (1995) observed that mild-to-moderate asthmatics, on average, experienced double the O₃-induced FEV₁ decrement of healthy subjects (19% versus 10%, respectively, $p = 0.04$). Moreover, a statistically significant positive correlation between FEV₁ responses to O₃ exposure and baseline lung function was observed in individuals with asthma, i.e., responses increased with severity of disease. Minimal evidence exists suggesting that individuals with asthma have smaller O₃-induced FEV₁ decrements than healthy subjects (3% versus 8%, respectively) (Mudway et al., 2001). However, the asthmatics in that study also tended to be older than the healthy subjects, which could partially explain their lesser response since FEV₁ responses to O₃ exposure diminish with age. Individuals with asthma also had significantly more neutrophils in the BALF (18 hours postexposure) than similarly exposed healthy individuals (Peden et al., 1997; Scannell et al., 1996; Basha et al., 1994). Furthermore, a study examining the effects of O₃ on individuals with atopic asthma and healthy controls reported that greater numbers of neutrophils, higher levels of cytokines and hyaluronan, and greater expression of macrophage cell-surface markers were observed in induced sputum of atopic asthmatics compared with healthy controls (Hernandez et al., 2010). Differences in O₃-induced epithelial cytokine expression were noted in bronchial biopsy samples from asthmatics and healthy controls (Bosson et al., 2003). Cell-surface marker and cytokine expression results, and the presence of hyaluronan, are consistent with O₃ having greater effects on innate and adaptive immunity in these asthmatic individuals. In addition, studies have demonstrated that O₃ exposure leads to increased bronchial reactivity to inhaled allergens in mild allergic asthmatics (Kehrl et al., 1999; Jorres et al., 1996) and to the influx of eosinophils in individuals with pre-existing allergic disease (Vagaggini et al., 2002; Peden et al., 1995). Taken together, these results point to several mechanistic pathways which could account for the enhanced sensitivity to O₃ in subjects with asthma (see Section 5.4.2.2 in the ISA).

Toxicological studies provide additional evidence of the biological basis for the greater effects of O₃ among those with asthma or AHR (U.S. EPA, 2013, section 8.2.2). In animal toxicological studies, an asthmatic phenotype is modeled by allergic sensitization of the respiratory tract. Many of the studies that provide evidence that O₃ exposure is an inducer of AHR and remodeling utilize these types of animal models. For example, a series of experiments in infant rhesus monkeys have shown these effects, but only in monkeys sensitized to house dust mite allergen. Similarly, adverse changes in pulmonary function were demonstrated in mice exposed to O₃; enhanced inflammatory responses were in rats exposed to O₃, but only in animals sensitized to allergen. In general, it is the combined effects of O₃ and allergic sensitization which result in measurable effects on pulmonary function. In a pulmonary fibrosis model, exposure O₃ for 5 days increased pulmonary inflammation and fibrosis, along with the frequency of

bronchopneumonia in rats. Thus, short-term exposure to O₃ may enhance damage in a previously injured lung (U.S. EPA, 2013, section 8.2.2).

In the 2006 O₃ AQCD, the potential for individuals with asthma to have greater risk of O₃-related health effects was supported by a number of controlled human exposure studies, evidence from toxicological studies, and a limited number of epidemiologic studies. In section 8.2.2, the ISA reports that in the recent epidemiologic literature some, but not all, studies report greater risk of health effects among individuals with asthma. Studies examining effect measure modification of the relationship between short-term O₃ exposure and altered lung function by corticosteroid use provided limited evidence of O₃-related health effects. However, recent studies of behavioral responses have found that studies do not take into account individual behavioral adaptations to forecasted air pollution levels (such as avoidance and reduced time outdoors), which may underestimate the observed associations in studies that examined the effect of O₃ exposure on respiratory health (Neidell and Kinney, 2010). This could explain some inconsistency observed among recent epidemiologic studies. The evidence from controlled human exposure studies provides support for increased detriments in FEV₁ and greater inflammatory responses to O₃ in individuals with asthma than in healthy individuals without a history of asthma. The collective evidence for increased risk of O₃-related health effects among individuals with asthma from controlled human exposure studies is supported by recent toxicological studies which provide biological plausibility for heightened risk of asthmatics to respiratory effects due to O₃ exposure. Overall, the ISA finds there is adequate evidence for asthmatics to be an at-risk population.

Children

Children are considered to be at greater risk from O₃ exposure because their respiratory systems undergo lung growth until about 18-20 years of age and are therefore thought to be intrinsically more at risk for O₃-induced damage (U.S. EPA, 2006). It is generally recognized that children spend more time outdoors than adults, and therefore would be expected to have higher exposure to O₃ than adults. The ventilation rates also vary between children and adults, particularly during moderate/heavy activity. Children aged 11 years and older and adults have higher absolute ventilation rates than children aged 1-11 years. However, children have higher ventilation rates relative to their lung volumes, which tends to increase dose normalized to lung surface area. Exercise intensity has a substantial effect on ventilation rate, with high intensity activities resulting in nearly double the ventilation rate during moderate activity among children and those adults less than 31 years of age. For more information on time spent outdoors and ventilation rate differences by age group, see Section 4.4.1 in the ISA (U.S. EPA, 2013).

The 1996 O₃ AQCD reported clinical evidence that children, adolescents, and young adults (<18 years of age) appear, on average, to have nearly equivalent spirometric responses to

O₃ exposure, but have greater responses than middle-aged and older adults (U.S. EPA, 1996). Symptomatic responses (e.g., cough, shortness of breath, pain on deep inspiration) to O₃ exposure, however, appear to increase with age until early adulthood and then gradually decrease with increasing age (U.S. EPA, 1996). Complete lung growth and development is not achieved until 18-20 years of age in women and the early 20s for men; pulmonary function is at its maximum during this time as well.

Recent epidemiologic studies have examined different age groups and their risk to O₃-related respiratory hospital admissions and emergency department (ED) visits. Evidence for greater risk in children was reported in several studies. A study in Cyprus of short-term O₃ concentrations and respiratory hospital admissions (HA) detected possible effect measure modification by age with a larger association among individuals < 15 years of age compared with those > 15 years of age; the effect was apparent only with a 2-day lag (Middleton et al., 2008). Similarly, a Canadian study of asthma-ED visits reported the strongest O₃-related associations among 5- to 14-year olds compared to the other age groups (ages examined 0-75+) (Villeneuve et al., 2007). Greater O₃-associated risk in asthma-related ED visits were also reported among children (<15 years) as compared to adults (15 to 64 years) in a study from Finland (Halonen et al., 2009). A study of New York City hospital admissions demonstrated an increase in the association between O₃ exposure and asthma-related hospital admissions for 6- to 18-year olds compared to those < 6 years old and those > 18 years old (Silverman and Ito, 2010). When examining long-term O₃ exposure and asthma HA among children, associations were determined to be larger among children 1 to 2 years old compared to children 2 to 6 years old (Lin et al., 2008b). A few studies reported positive associations among both children and adults and no modification of the effect by age.

The evidence reported in epidemiologic studies is supported by recent toxicological studies which observed O₃-induced health effects in immature animals. Early life exposures of multiple species of laboratory animals, including infant monkeys, resulted in changes in conducting airways at the cellular, functional, ultra-structural, and morphological levels. The studies conducted on infant monkeys are most relevant for assessing effects in children. Carey et al. (2007) conducted a study of O₃ exposure in infant rhesus macaques, whose respiratory tract closely resemble that of humans. Monkeys were exposed either acutely or in episodes designed to mimic human exposure. All monkeys acutely exposed to O₃ had moderate to marked necrotizing rhinitis, with focal regions of epithelial exfoliation, numerous infiltrating neutrophils, and some eosinophils. The distribution, character, and severity of lesions in episodically exposed infant monkeys were similar to that of acutely exposed animals. Neither exposure protocol for the infant monkeys produced mucous cell metaplasia proximal to the lesions, an adaptation observed in adult monkeys exposed in another study (Harkema et al., 1987). Functional and

cellular changes in conducting airways were common manifestations of exposure to O₃ among both the adult and infant monkeys (Plopper et al., 2007). In addition, the lung structure of the conducting airways in the infant monkeys was significantly stunted by O₃ and this aberrant development was persistent 6 months postexposure (Fanucchi et al., 2006).

Age may also affect the inflammatory response to O₃ exposure. Toxicological studies reported that the difference in effects among younger lifestage test animals may be due to age-related changes in antioxidants levels and sensitivity to oxidative stress. Further discussion of these studies may be found in section 8.3.1.1 of the ISA (U.S. EPA, 2013, p. 8-18).

The previous and recent human clinical and toxicological studies reported evidence of increased risk from O₃ exposure for younger ages, which provides coherence and biological plausibility for the findings from epidemiologic studies. Although there was some inconsistency, generally, the epidemiologic studies reported positive associations among both children and adults or just among children. The interpretation of these studies is limited by the lack of consistency in comparison age groups and outcomes examined. However, overall, the epidemiologic, controlled human exposure, and toxicological studies provide adequate evidence that children are potentially at increased risk of O₃-related health effects.

Older Adults

The ISA notes that older adults are at greater risk of health effects associated with O₃ exposure through a variety of intrinsic pathways (U.S. EPA, 2013, section 8.3.1.2). In addition, older adults may differ in their exposure and internal dose. Older adults were outdoors for a slightly longer proportion of the day than adults aged 18-64 years. Older adults also have somewhat lower ventilation rates than adults aged 31 - less than 61 years. For more information on time spent outdoors and ventilation rate differences by age group, see Section 4.4 in the ISA (U.S. EPA, 2013). The gradual decline in physiological processes that occur with aging may lead to increased risk of O₃-related health effects (U.S. EPA, 2006). Respiratory symptom responses to O₃ exposure appears to increase with age until early adulthood and then gradually decrease with increasing age (U.S. EPA, 1996); lung function responses to O₃ exposure also decline from early adulthood (U.S. EPA, 1996). The reductions of these responses with age may put older adults at increased risk for continued O₃ exposure. In addition, older adults, in general, have a higher prevalence of preexisting diseases compared to younger age groups and this may also lead to increased risk of O₃-related health effects (U.S. EPA, 2013, section 8.3.1.2). With the number of older Americans increasing in upcoming years (estimated to increase from 12.4% of the U.S. population to 19.7% between 2000 to 2030, which is approximately 35 million and 71.5 million individuals, respectively) this group represents a large population potentially at risk of O₃-related health effects (SSDAN CensusScope, 2010; DeNavas-Walt et al., 2011).

The majority of recent studies reported greater effects of short-term O₃ exposure and mortality among older adults, which is consistent with the findings of the 2006 O₃ AQCD. A study (Medina-Ramón and Schwartz, 2008) conducted in 48 cities across the U.S. reported larger effects among adults ≥ 65 years old compared to those < 65 years; further investigation of this study population revealed a trend of O₃-related mortality risk that gets larger with increasing age starting at age 51 (Zanobetti and Schwartz, 2008a). Another study conducted in 7 urban centers in Chile reported similar results, with greater effects in adults ≥ 65 years old (Cakmak et al., 2007). More recently, a study conducted in the same area reported similar associations between O₃ exposure and mortality in adults aged < 64 years old and 65 to 74 years old, but the risk was increased among older age groups (Cakmak et al., 2011). A study performed in China reported greater effects in populations ≥ 45 years old (compared to 5 to 44 year olds), with statistically significant effects present only among those ≥ 65 years old (Kan et al., 2008). An Italian study reported higher risk of all-cause mortality associated with increased O₃ concentrations among individuals ≥ 85 year old as compared to those 35 to 84 years old (Stafoggia et al., 2010). The Air Pollution and Health: A European and North American Approach (APHENA) project examined the association between O₃ exposure and mortality for those < 75 and ≥ 75 years of age. In Canada, the associations for all-cause and cardiovascular mortality were greater among those ≥ 75 years old. In the U.S., the association for all-cause mortality was slightly greater for those < 75 years of age compared to those ≥ 75 years old in summer-only analyses. No consistent pattern was observed for CVD mortality. In Europe, slightly larger associations for all-cause mortality were observed in those < 75 years old in all-year and summer-only analyses. Larger associations were reported among those < 75 years for CVD mortality in all-year analyses, but the reverse was true for summer-only analyses (Katsouyanni et al., 2009).

With respect to epidemiologic studies of O₃ exposure and hospital admissions, a positive association was reported between short-term O₃ exposure and respiratory hospital admissions for adults ≥ 65 years old but not for those adults aged 15 to 64 years (Halonen et al., 2009). In the same study, no association was observed between O₃ concentration and respiratory mortality among those ≥ 65 years old or those 15 to 64 years old. No modification by age (40 to 64 year olds versus > 64 year olds) was observed in a study from Brazil examining O₃ levels and COPD ED visits.

Although some outcomes reported mixed findings regarding an increase in risk for older adults, recent epidemiologic studies report consistent positive associations between short-term O₃ exposure and mortality in older adults. The evidence from mortality studies is consistent with the results reported in the 2006 O₃ AQCD and is supported by toxicological studies providing biological plausibility for increased risk of effects in older adults. Also, older adults may be experiencing increased exposure compared to younger adults. Overall, the ISA (U.S. EPA, 2013)

concludes adequate evidence is available indicating that older adults are at increased risk of O₃-related health effects.

People with Diets Lower in Vitamins C and E

Diet was not examined as a factor potentially affecting risk in previous O₃ AQCDs, but recent studies have examined modification of the association between O₃ and health effects by dietary factors. Because O₃ mediates some of its toxic effects through oxidative stress, the antioxidant status of an individual is an important factor that may contribute to increased risk of O₃-related health effects. Supplementation with vitamins C and E has been investigated in a number of studies as a means of inhibiting O₃-mediated damage.

Two epidemiologic studies have examined effect measure modification by diet and found evidence that certain dietary components are related to the effect O₃ has on respiratory outcomes. In one recent study the effects of fruit/vegetable intake and Mediterranean diet were examined. Increases in these food patterns, which have been noted for their high vitamins C and E and omega-3 fatty acid content, were positively related to lung function in asthmatic children living in Mexico City, and modified by O₃ exposure (Romieu et al., 2009). Another study examined supplementation of the diets of asthmatic children in Mexico with vitamins C and E (Sienra-Monge et al., 2004). Associations were detected between short-term O₃ exposure and nasal airway inflammation among children in the placebo group but not in those receiving the supplementation.

The epidemiologic evidence is supported by controlled human exposure studies, discussed in section 8.4.1 of the ISA (U.S. EPA, 2013), that have shown that the first line of defense against oxidative stress is antioxidants-rich extracellular lining fluid (ELF) which scavenge free radicals and limit lipid peroxidation. Exposure to O₃ depletes antioxidant levels in nasal ELF probably due to scrubbing of O₃; however, the concentration and the activity of antioxidant enzymes either in ELF or plasma do not appear to be related to O₃ responsiveness. Controlled studies of dietary antioxidant supplementation have demonstrated some protective effects of α -tocopherol (a form of vitamin E) and ascorbate (vitamin C) on spirometric measures of lung function after O₃ exposure but not on the intensity of subjective symptoms and inflammatory responses. Dietary antioxidants have also afforded partial protection to asthmatics by attenuating postexposure bronchial hyperresponsiveness. Toxicological studies discussed in section 8.4.1 of the ISA (U.S. EPA, 2013) provide evidence of biological plausibility to the epidemiologic and controlled human exposure studies.

There is adequate evidence that individuals with diets lower in vitamins C and E are at risk for O₃-related health effects. The evidence from epidemiologic studies is supported by controlled human exposure and toxicological studies.

Outdoor Workers

Studies included in the 2006 O₃ AQCD reported that individuals who participate in outdoor activities or work outside to be a population at increased risk based on consistently reported associations between O₃ exposure and respiratory health outcomes in these groups (U.S. EPA, 2006). Outdoor workers are exposed to ambient O₃ concentrations for a greater period of time than individuals who spend their days indoors. As discussed in Section 4.7 of the ISA (U.S. EPA, 2013) outdoor workers sampled during the work shift had a higher ratio of personal exposure to fixed-site monitor concentrations than health clinic workers who spent most of their time indoors. Additionally, an increase in dose to the lower airways is possible during outdoor exercise due to both increases in the amount of air breathed (i.e., minute ventilation) and a shift from nasal to oronasal breathing. The association between FEV₁ responses to O₃ exposure and minute ventilation is discussed more fully in Section 6.2.3.1 of the 2006 O₃ AQCD.

Previous studies have shown that increased exposure to O₃ due to outdoor work leads to increased risk of O₃-related health effects, specifically decrements in lung function (U.S. EPA, 2006). The strong evidence from the 2006 O₃ AQCD which demonstrated increased exposure, dose, and ultimately risk of O₃-related health effects in this population supports the conclusion that there is adequate evidence to indicate that increased exposure to O₃ through outdoor work increases the risk of O₃-related health effects.

In some cases, it is difficult to determine a factor that results in increased risk of effects. For example, previous assessments have included controlled human exposure studies in which some healthy individuals demonstrate greater O₃-related health effects compared to other healthy individuals. Intersubject variability has been observed for lung function decrements, symptomatic responses, pulmonary inflammation, AHR, and altered epithelial permeability in healthy adults exposed to O₃ and these results tend to be reproducible within a given individual over a period of several months indicating differences in the intrinsic responsiveness. In many cases the reasons for the variability is not clear. This may be because one or some of the factors described above have not been evaluated in studies, or it may be that additional, unidentified factors influence individual responses to O₃ (U.S. EPA, 2013, section 8.5).

As discussed in chapter 8 of the ISA the challenges and limitations in evaluating the factors that can increase risk for experiencing O₃-related health effects may contribute to a lack of information about the factors that may increase risk from O₃ exposures. This lack of information may contribute to conclusions that evidence for some factors, such as sex, SES, and obesity provided “suggestive” evidence of increased risk, or that for a number of factors the evidence was inadequate to draw conclusions about potential increase in risk of effects. Overall, the factors for which the ISA concludes there is adequate evidence of increased risk for

experiencing O₃-related effects were related to asthma, lifestage (children and older adults), genetic variability, dietary factors, and working outdoors.

3.1.5.2 Size of At-Risk Populations and Lifestyles in the United States

One consideration in the assessment of potential public health impacts is the size of various population groups for which there is adequate evidence of increased risk for health effects associated with O₃-related air pollution exposure. The factors for which the ISA judged the evidence to be “adequate” with respect to contributing to increased risk of O₃-related effects among various populations and lifestyles included: asthma; childhood and older adulthood; diets lower in vitamins C and E; certain genetic variants and, working outdoors (U.S. EPA, 2013, section 8.5).

With regard to asthma, Table 3-7 below summarizes information on the prevalence of current asthma by age in the U.S. adult population in 2010 (Schiller et al., 2012; children - Bloom et al., 2011). Individuals with current asthma constitute a fairly large proportion of the population, including more than 25 million people. Asthma prevalence tends to be higher in children than adults.

Within the U.S., approximately 8.2% of adults have reported currently having asthma (Schiller et al., 2012) and 9.5% of children have reported currently having asthma (Bloom et al., 2011). Table 3-12 below provides more detailed information on prevalence of asthma by age in the U.S.

Table 3-7. Prevalence of asthma by age in the U.S.

Age (years)	N (in thousands)	Percent
0-4	1,285	6.0
5-11	3,020	10.5
12-17	2,672	10.9
18-44	8,902	8.1
45-64	6,704	8.4
65-74	1,849	8.7
75+	1,279	7.4

Asthma prevalence is reported for “still has asthma”

Source: Statistics for adults: Schiller et al. (2012); Statistics for children: Bloom et al. (2011)

With regard to lifestyles, based on U.S. census data from 2010 (Howden and Meyer, 2011), about 74 million people, or 24% of the U.S. population, are under 18 years of age and more than 40 million people, or about 13% of the U.S. population, are 65 years of age or older. Hence, a large proportion of the U.S. population, more than 33%, is included in age groups that are considered likely to be at increased risk for health effects from ambient O₃ exposure.

With regard to dietary factors, no statistics are available to estimate the size of an at-risk population based on nutritional status.

With regard to outdoor workers, in 2010 approximately 11.7% of the total number of people (143 million people) employed, or about 16.8 million people, worked outdoors one or more day per week (based on worker surveys).⁶³ Of these approximately 7.4% of the workforce, or about 7.8 million people, worked outdoors three or more days per week.

The health statistics data illustrate what is known as the “pyramid” of effects. At the top of the pyramid, there are approximately 2.5 million deaths from all causes per year in the U.S. population, with about 250 thousand respiratory-related deaths (CDC-WONDER⁶⁴). For respiratory health diseases, there are nearly 3.3 million hospital discharges per year (HCUP⁶⁵),

⁶³ The O*NET program is the nation's primary source of occupational information. Central to the project is the O*NET database, containing information on hundreds of standardized and occupation-specific descriptors. The database, which is available to the public at no cost, is continually updated by surveying a broad range of workers from each occupation. <http://www.onetcenter.org/overview.html>

http://www.onetonline.org/find/descriptor/browse/Work_Context/4.C.2/

⁶⁴ <http://wonder.cdc.gov/>

⁶⁵ <http://www.hcup-us.ahrq.gov/>

8.7 million respiratory ED visits (HCUP, 2007), 112 million ambulatory care visits (Woodwell and Cherry, 2004), and an estimated 700 million restricted activity days per year due to respiratory conditions (Adams et al., 1999). Combining small risk estimates with relatively large baseline levels of health outcomes can result in quite large public health impacts. Thus, even a small percentage reduction in O₃ health impacts on cardiopulmonary diseases would reflect a large number of avoided cases.

3.1.5.3 Averting Behavior

The activity pattern of individuals is an important determinant of their exposure (ISA, U.S. EPA, 2013, section 4.4.1). Variation in O₃ concentrations among various microenvironments means that the amount of time spent in each location, as well as the level of activity, will influence an individual's exposure to ambient O₃. Activity patterns vary both among and within individuals, resulting in corresponding variations in exposure across a population and over time. Individuals can reduce their exposure to O₃ by altering their behaviors, such as by staying indoors, being active outdoors when air quality is better, and by reducing their activity levels or reducing the time being active outdoors on high-O₃ days (U.S. EPA, 2013, section 4.4.2). The evidence in this topic area, while not addressed in the 2006 AQCD, is evaluated in the ISA for this review.

The widely reported Air Quality Index (AQI) conveys advice to the public, and particularly at-risk populations, on reducing exposure on days when ambient levels of common air pollutants are elevated (www.airnow.gov). The AQI describes the potential for health effects from O₃ (and other individual pollutants) in six color-coded categories of air-quality, ranging from Good (green), Moderate (yellow), Unhealthy for Sensitive Groups (orange), Unhealthy (red), and Very Unhealthy (purple), and Hazardous (maroon). Levels in the unhealthy ranges (i.e., Unhealthy for Sensitive Groups and above) come with recommendations about reducing exposure. Forecasted and actual AQI values for O₃ are reported to the public during the O₃ season. The AQI advisories explicitly state that children, older adults, people with lung disease, and people who are active outdoors, may be at greater risk from exposure to O₃. People are advised to reduce exposure depending on the predicted O₃ levels and the likelihood of risk. This advice includes being active outdoors when air quality is better, and reducing activity levels or reducing the time being active outdoors on high-O₃ days. Staying indoors to reduce exposure is not recommended until air quality reaches the Very Unhealthy or Hazardous categories.

Evidence of individual averting behaviors in response to AQI advisories has been found in several studies, including activity pattern and epidemiologic studies, especially for the at-risk populations, such as children, older adults, and people with asthma, who are targeted by the advisories. Such effects are less pronounced in the general population, possibly due to the opportunity cost of behavior modification. Epidemiologic evidence from a study (Neidell and

5 ADEQUACY OF THE CURRENT SECONDARY STANDARD

This chapter presents staff's considerations and conclusions regarding the adequacy of the current secondary O₃ NAAQS. In doing so, we pose the following overarching question:

- **Does the currently available scientific evidence- and exposure/risk-based information, as reflected in the ISA and WREA, support or call into question the adequacy and appropriateness of the protection afforded by the current secondary O₃ standard?**

In addressing this overarching question, we pose a series of more specific questions, as discussed in sections 5.1 through 5.5 below. We consider the nature of O₃-induced effects, including the nature of the exposures that drive the biological and ecological response and related biologically relevant exposure metrics (section 5.1); the scientific evidence and exposure/risk information, including that for associated ecosystem services, regarding (a) tree growth, productivity and carbon storage (section 5.2), (b) crop yield loss (section 5.3), (c) visible foliar injury (section 5.4), and (d) other welfare effects (section 5.5). Section 5.6 describes advice and recommendations received from CASAC. In section 5.7, we revisit the overarching question of this chapter and present staff conclusions on the adequacy and appropriateness of the current secondary standard.

5.1 NATURE OF EFFECTS AND BIOLOGICALLY RELEVANT EXPOSURE METRIC

- **Does the current evidence alter our conclusions from the previous review regarding the nature of O₃-induced welfare effects?**

As discussed further below, the current body of O₃ welfare effects evidence confirms and strengthens the conclusions reached in the last review on the nature of O₃-induced welfare effects. Ozone's phytotoxic effects were first identified on grape leaves in a study published in 1958 (Richards et al., 1958). In the more than fifty years that have followed, extensive research has been conducted both in and outside of the U.S. to examine the impacts of O₃ on plants and their associated ecosystems, since "of the phytotoxic compounds commonly found in the ambient air, O₃ is the most prevalent, impairing crop production and injuring native vegetation and ecosystems more than any other air pollutant" (U.S. EPA, 1989, 1996). Recent studies, assessed in the ISA, together with this longstanding and well established vegetation effects literature, further contribute to the coherence and consistency of the vegetation effects evidence.

In assessing the strength of the evidence, it is important to note that different types of studies can provide different types of information, each with different associated uncertainties

(U.S. EPA, 2013, Chapter 9, section 9.2). Controlled chamber studies are the best method for isolating or characterizing the role of O₃ in inducing the observed plant effects, and in assessing plant response to O₃ at the finer scales (U.S. EPA, 2013, Chapter 9, section 9.3). Recent controlled studies have focused on a variety of plant responses to O₃ including: 1) the underlying mechanisms as they relate to growth, productivity and carbon storage including: reduced carbon dioxide uptake due to stomatal closure (U.S. EPA 2013, section 9.3.2.1); 2) the upregulation of genes associated with plant defense, signaling, hormone synthesis and secondary metabolism (U.S. EPA 2013, section 9.3.3.2); 3) the down regulation of genes related to photosynthesis and general metabolism (U.S. EPA 2013, section 9.3.3.2); 4) the loss of carbon assimilation capacity due to declines in the quantity and activity of key proteins and enzymes (U.S. EPA, 2013, section 9.3.5.1); and 5) the negative impacts on the efficiency of the photosynthetic light reactions (U.S. EPA, 2013, section 9.3.5.1). As described in the ISA, these new studies “have increased knowledge of the molecular, biochemical and cellular mechanisms occurring in plants in response to O₃”, adding “to the understanding of the basic biology of how plants are affected by oxidative stress...” (U.S. EPA, 2013, p. 9-11). The ISA further concluded that controlled studies “have clearly shown that exposure to O₃ is causally linked to visible foliar injury, decreased photosynthesis, changes in reproduction, and decreased growth” in many species of vegetation (U.S. EPA 2013, p. 1-15).

Such effects at the plant scale can also be linked to an array of effects at larger spatial scales. For example, recent field studies at larger spatial scales, together with previously available evidence, support the controlled exposure study results and indicate that “ambient O₃ exposures can affect ecosystem productivity, crop yield, water cycling, and ecosystem community composition” (U.S. EPA, 2013, p. 1-15; Chapter 9, section 9.4).

The ISA summarizes the coherence across the full range of effects, from the least serious to the most serious, as follows (U.S. EPA, 2013, p. 1-8):

The welfare effects of O₃ can be observed across spatial scales, starting at the subcellular and cellular level, then the whole plant and finally, ecosystem-level processes. Ozone effects at small spatial scales, such as the leaf of an individual plant, can result in effects along a continuum of larger spatial scales. These effects include altered rates of leaf gas exchange, growth, and reproduction at the individual plant level, and can result in broad changes in ecosystems, such as productivity, carbon storage, water cycling, nutrient cycling, and community composition.

Based on its assessment of this extensive body of science, the ISA determined that, with respect to vegetation and ecosystems, a causal relationship exists between exposure to O₃ in ambient air and visible foliar injury effects on vegetation, reduced vegetation growth, reduced

productivity in terrestrial ecosystems, reduced yield and quality of agricultural crops and alteration of below-ground biogeochemical cycles (U.S. EPA 2013, Table 1-2). Additionally, the ISA determined that a likely to be causal relationship exists between exposures to O₃ in ambient air and reduced carbon sequestration in terrestrial ecosystems, alteration of terrestrial ecosystem water cycling and alteration of terrestrial community composition (U.S. EPA, 2013, Table 1-2). With regard to the relationship between O₃ and radiative forcing and climate change, the ISA determined that there is a causal relationship between changes in tropospheric O₃ concentrations and radiative forcing, and likely to be a causal relationship between changes in tropospheric O₃ concentrations and effects on climate (U.S. EPA, 2013, p. 1-13, and Table 1-3). From this set of effects that the ISA has concluded to be causally or likely causally related to O₃ in ambient air, we focus the discussion in the PA primarily on: 1) impacts on tree growth, productivity and carbon storage; 2) crop yield loss; 3) visible foliar injury. Each of these discussions also includes where appropriate, a discussion of any known or anticipated impacts that such individual plant or species level effects could have at larger scales, including ecosystems, and on associated ecosystem services.

In considering the available vegetation effects evidence, we make note of several important contextual features that frame our understanding of the science and how it informs our evaluation of the adequacy of the protection afforded by the current secondary NAAQS. First, we acknowledge that under natural conditions, a variety of factors can either mitigate or exacerbate the predicted O₃-plant interactions and are recognized sources of uncertainty and variability. These include: 1) multiple genetically influenced determinants of O₃ sensitivity; 2) changing sensitivity to O₃ across vegetative growth stages; 3) co-occurring stressors and/or modifying environmental factors (U.S. EPA, 2013, section 9.4.8).

Second, we acknowledge that the species that have been studied for O₃ sensitivity represent only a fraction of the tens of thousands of plant species that grow in the U.S. (USDA NRCS, 2014)¹, and that these species were typically selected because of their commercial importance (e.g., commodity crop or timber species) or because of observed O₃-induced visible foliar injury in the field. Of the species known to be sensitive to O₃ for foliar injury, 66 species have been identified on National Park Service (NPS) and U.S. Fish and Wildlife Service lands² and a subset of these are used in the USFS biomonitoring program (discussed in section 5.4 below). A number of these species have also been identified as important to tribal cultural practices (see Appendix 5-A). Appendix 7J of the 2007 Staff Paper showed that no state in the

¹ USDA NRCS. 2014. The PLANTS Database (<http://plants.usda.gov>, 3 January 2014). National Plant Data Team, Greensboro, NC 27401-4901 USA.

² See <http://www2.nature.nps.gov/air/Pubs/pdf/flag/NPSozonesensppFLAG06.pdf>

lower 48 states had less than seven known O₃-sensitive plant species, with the majority of states having between 11 and 30 (see Appendix 7J-2 in U.S. EPA, 2007). We would not expect this information to have changed since the previous review because there has been very little change in the list of sensitive species and the occurrence of any of these plant species within a state would not be expected to change. With respect to agricultural species, a number of important commodity crops such as soybean and additional fruit and vegetable species such as lettuce have been shown to be sensitive to O₃ for either foliar injury or yield loss (U.S. EPA, 2013, section 9.4.4.1; Abt Associates, Inc., 1995).

Third, we acknowledge that out of the group of species known to be sensitive to O₃, we have chosen to focus primarily on species for which we have robust exposure-response (E-R) functions for biomass loss and yield loss using the W126 form (i.e., 11 tree and 10 crop species) in order to be able to quantitatively relate predicted changes in O₃ to predicted changes in plant exposures, responses and associated risks.³ However, while we recognize that this small group represents only a fraction of all species known or anticipated to be sensitive to O₃ in the U.S., we also note, as did CASAC, that among the studied species, there is a fairly large range of O₃ sensitivities represented, so that it could be reasonable to assume that other non-studied species might have sensitivities that fall within or near this range. Specifically, CASAC states “[i]t should not be assumed that species of unknown sensitivity are tolerant to ozone. It is more appropriate to assume that the sensitivity of species without E-R functions might be similar to the range of sensitivity for those species with E-R functions” (Frey, 2014, p. 11).

Fourth, we acknowledge that in addition to the well-studied effects of biomass loss in trees and crops and visible foliar injury in bioindicator plants that we can quantify, numerous other more subtle and less easily observed effects occur along the continuum of spatial scales that lead to ecosystem effects. While these effects are more difficult to quantify, we acknowledge that any secondary standard set to protect the public welfare against the known and quantifiable adverse effects to vegetation should also consider the anticipated, but currently unquantifiable, potential adverse effects on vegetation, ecosystems and associated services.

Finally, we further acknowledge that in light of the above, when considering the available evidence, we seek to find the right balance between placing weight on the associated uncertainties and limitations of the evidence and placing weight on its well-established strength, coherence and consistency. In so doing, we note that CASAC, in commenting on section 6.7 which describes key uncertainties and future research areas, states that “[w]hile these scientific research priorities will enhance future scientific reviews of the ozone primary and secondary

³ There is an E-R function available for a 12th tree species (cottonwood), but this E-R function is considered less robust because it is based on the results of a single gradient study (Gregg et al., 2003).

standards, we also make clear that there is sufficient scientific evidence, and sufficient confidence in the available research results, to support the advice we have given above for this review cycle of the primary and secondary standards” (Frey, 2014, p. iv).

- **Does the current evidence continue to support a cumulative, seasonal exposure index as a biological-relevant and appropriate metric for assessment of the evidence and exposure/risk information?**

In this review, the ISA assessment of the full body of currently available evidence stated the following regarding biological indices (U.S. EPA, 2013, p. 2-44):

The main conclusions from the 1996 and 2006 O₃ AQCDs [Air Quality Criteria Documents] regarding indices based on ambient exposure remain valid. These key conclusions can be restated as follows:

- *ozone effects in plants are cumulative;*
- *higher O₃ concentrations appear to be more important than lower concentrations in eliciting a response;*
- *plant sensitivity to O₃ varies with time of day and plant development stage;*
- *quantifying exposure with indices that cumulate hourly O₃ concentrations and preferentially weight the higher concentrations improves the explanatory power of exposure/response models for growth and yield, over using indices based on mean and peak exposure values.*

The long-standing body of available evidence upon which these conclusions are based, provides a wealth of information on aspects of O₃ exposure that are important in influencing plant response. Specifically, a variety of “factors with known or suspected bearing on the exposure-response relationship, including concentration, time of day, respite time, frequency of peak occurrence, plant phenology, predisposition, etc.,” have been identified (U.S. EPA, 2013, section 9.5.2). In addition, the importance of the duration of the exposure and the relatively greater importance of higher concentrations over lower in determining plant response to O₃ have been consistently well documented (U.S. EPA, 2013, section 9.5.3). Much of this evidence was assessed in the 1996 Criteria Document (CD) (U.S. EPA, 1996), while more recent work substantiating this evidence is assessed in the subsequent 2006 CD and 2013 ISA.

Understanding of the biological basis for plant response to O₃ exposure led to the development of a large number of “mathematical approaches for summarizing ambient air quality information in biologically meaningful forms for O₃ vegetation effects assessment purposes ...” (U.S. EPA, 2013, section 9.5.3), including those that cumulate exposures over some specified period while weighting higher concentrations more than lower (U.S. EPA, 2013, section 9.5.2). As with any summary statistic, these exposure indices retain information on

Exhibit 14



Integrated Science Assessment for Ozone and Related Photochemical Oxidants





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Integrated Science Assessment for Ozone and Related Photochemical Oxidants

National Center for Environmental Assessment-RTP Division
Office of Research and Development
U.S. Environmental Protection Agency
Research Triangle Park, NC

with increases in respiratory symptoms and asthma medication use in children with asthma, respiratory-related hospital admissions, and asthma-related ED visits. Although O₃ was consistently associated with nonaccidental and cardiopulmonary mortality, the contribution of respiratory causes to these findings was uncertain.

Collectively, there is a vast amount of evidence spanning several decades that demonstrated that exposure to O₃ induces a range of respiratory effects. The majority of this evidence was derived from studies investigating short-term exposure (i.e., hours to weeks) to O₃. The combined evidence across disciplines led to the causal relationship between short-term O₃ exposure and respiratory effects reported in the 2006 O₃ AQCD.

Mechanistic evidence for the effects of O₃ on the respiratory system was characterized in the 1996 O₃ AQCD ([U.S. EPA, 1996a](#)), which identified O₃-induced changes in a variety of lung lipid species whose numerous biologically active metabolites, in turn, can affect host defenses, lung function, and the immune system. As summarized in [Section 2.4](#) and fully characterized in [Chapter 5](#), key events in the toxicity pathway of O₃ have been identified in humans and animal models. They include formation of secondary oxidation products, activation of neural reflexes, initiation of inflammation, alteration of epithelial barrier function, sensitization of bronchial smooth muscle, modification of innate/adaptive immunity, airways remodeling, and systemic inflammation and oxidative/nitrosative stress.

2.5.2 Summary of Causal Determinations

Recent studies support or build upon the strong body of evidence presented in the 1996 and 2006 O₃ AQCDs that *short-term O₃ exposure is causally associated with respiratory health effects*. Recent controlled human exposure studies demonstrate statistically significant group mean decreases in pulmonary function to exposures as low as 60-70 ppb O₃ in young, healthy adults, and are supported by the strong, cumulative evidence from epidemiologic studies. Equally strong evidence demonstrated associations of ambient O₃ with respiratory hospital admissions and ED visits across the U.S., Europe, and Canada. Most effect estimates ranged from a 1.6 to 5.4% increase in daily respiratory-related ED visits or hospital admissions in all-year analyses for unit increases¹ in ambient O₃ concentrations. Several multicity studies and a multicontinent study reported associations between short-term increases in ambient O₃ concentrations and increases in respiratory mortality. This evidence is supported by a large body of individual-level epidemiologic panel studies that demonstrate associations of ambient O₃ with respiratory symptoms in children with asthma. Further support is provided by recent studies that found O₃-associated increases in indicators of airway inflammation and oxidative stress in children with asthma. Across respiratory endpoints, evidence indicates antioxidant capacity may modify the risk of respiratory morbidity associated with O₃ exposure. The potentially elevated risk of populations with diminished antioxidant capacity and the reduced

¹ Effect estimates were standardized to a 40-, 30-, and 20-ppb unit increase for 1-h max, 8-h max, and 24-h avg O₃.

risk of populations with enhanced antioxidant capacity identified in epidemiologic studies is strongly supported by similar findings from controlled human exposure studies and by evidence that characterizes O₃-induced decreases in intracellular antioxidant levels as a mode of action for downstream effects. By demonstrating O₃-induced airway hyperresponsiveness, decreased pulmonary function, allergic responses, lung injury, impaired host defense, and airway inflammation, toxicological studies have characterized O₃ modes of action and provided biological plausibility for epidemiologic associations of ambient O₃ concentrations with lung function and respiratory symptoms, hospital admissions, ED visits, and mortality. Together, the evidence integrated across controlled human exposure, epidemiologic, and toxicological studies and across the spectrum of respiratory health endpoints continues to demonstrate that there **is a causal relationship between short-term O₃ exposure and respiratory health effects.**

The epidemiologic evidence for a relationship between *long-term O₃ exposure and respiratory health effects* (including respiratory symptoms, new-onset asthma, and respiratory mortality) is contributed by recent studies that evaluate the associations between long-term exposure to O₃ and respiratory effects that demonstrate interactions between exercise or different genetic variants and both new-onset asthma in children and increased respiratory symptom effects in individuals with asthma. While the evidence is limited, a U.S. multicomunity prospective cohort demonstrates that asthma risk is affected by interactions among genetic variability, environmental O₃ exposure, and behavior. The evidence relating new-onset asthma to long-term O₃ exposure is supported by toxicological studies of asthma in monkeys. This nonhuman primate evidence of O₃-induced changes supports the biologic plausibility of long-term exposure to O₃ contributing to the effects of asthma in children. Early life O₃ exposure can alter airway development and lead to the development of asthma. Other recent epidemiologic studies provide coherent evidence for long-term O₃ exposure and respiratory effects such as first asthma hospitalization, respiratory symptoms in asthmatics, and respiratory mortality. Generally, the epidemiologic and toxicological evidence provides a compelling case that supports the hypothesis that a relationship exists between long-term exposure to ambient O₃ and measures of respiratory health effects and mortality. The evidence for short-term exposure to O₃ and effects on respiratory endpoints provides coherence and biological plausibility for the effects of long-term exposure to O₃. Building upon that evidence, the more recent epidemiologic evidence, combined with toxicological studies in rodents and nonhuman primates, provides biologically plausible evidence that there **is likely to be a causal relationship between long-term exposure to O₃ and respiratory health effects.**

In past O₃ AQCDs the effects of *short-term exposure to O₃ on the cardiovascular system* could not be thoroughly evaluated due to the paucity of information available. However, studies investigating O₃-induced cardiovascular events have advanced in the last two decades. Animal toxicological studies, although limited in number, demonstrate O₃-induced cardiovascular effects; specifically enhanced ischemia/reperfusion (I/R) injury, disrupted NO-induced vascular reactivity, decreased cardiac function, and increased heart rate variability (HRV). These effects

are consistent with cardiovascular system effects observed after long-term O₃ exposure, such as increased vascular disease. These effects may, in part, correspond to the alteration of the autonomic nervous system or to the development and maintenance of systemic oxidative stress and a proinflammatory environment that can result from pulmonary inflammation. Controlled human exposure studies provide some coherence with the evidence from animal toxicological studies, by demonstrating increases and decreases in HRV following relatively low (120 ppb during rest) and high (300 ppb with exercise) O₃ exposures, respectively. Controlled human exposure studies also support the animal toxicology studies by demonstrating O₃-induced effects on blood biomarkers of systemic inflammation and oxidative stress as well as changes in biomarkers suggestive of a pro-thrombogenic response to O₃. The experimental evidence provides initial biological plausibility for the consistently positive associations observed across multiple epidemiologic studies of short-term O₃ exposure and cardiovascular mortality. However, epidemiologic studies generally do not observe associations between short-term exposure to O₃ and cardiovascular morbidity; studies of cardiovascular-related hospital admissions and ED visits and other various cardiovascular effects did not find consistent evidence of a relationship with O₃ exposure. The lack of coherence between the results from studies that examined associations between short-term O₃ exposure and cardiovascular morbidity and subsequently cardiovascular mortality complicate the interpretation of the overall evidence for O₃-induced cardiovascular effects. Overall, animal toxicological studies demonstrate O₃-induced cardiovascular effects, and support to the strong body of evidence indicating O₃-induced cardiovascular mortality. Animal toxicological and controlled human exposure studies provide evidence for biologically plausible mechanisms underlying these O₃-induced cardiovascular effects. However, a lack of coherence with epidemiologic studies of cardiovascular morbidity remains an important uncertainty. Taken together, the overall body of evidence across disciplines indicates that **there is likely to be a causal relationship between short-term exposures to O₃ and cardiovascular effects.**

The 2006 O₃ AQCD concluded that the overall body of evidence was highly suggestive that short-term exposure to O₃ directly or indirectly contributes to nonaccidental and cardiopulmonary-related mortality, but additional research was needed to more fully establish underlying mechanisms by which such effects occur. The evaluation of recent multicity studies and a multicontinent study that examined the association between *short-term increases in ambient O₃ concentration and mortality* found evidence that supports the conclusions of the 2006 O₃ AQCD. These recent studies reported consistent positive associations between short-term increases in ambient O₃ concentration and total (nonaccidental) mortality, with associations being stronger during the warm season, as well as provided additional support for associations between O₃ concentrations and cardiovascular mortality being similar or larger in magnitude compared to respiratory mortality. Additionally, these new studies examined previously identified areas of uncertainty in the O₃-mortality relationship, and provide additional evidence supporting an association between short-term O₃ exposure and mortality. Taken together, the body of evidence indicates

that there **is likely to be a causal relationship between short-term O₃ exposures and total mortality.**

The 2006 O₃ AQCD concluded that an insufficient amount of evidence existed to suggest a causal relationship between *long-term O₃ exposure and mortality* ([U.S. EPA, 2006b](#)). A synthesis of the recent and earlier evidence reveals that the strongest evidence for an association between long-term exposure to ambient O₃ concentrations and mortality is derived from associations for respiratory mortality that remained robust after adjusting for PM_{2.5} concentrations. There is inconsistent evidence for an association between long-term exposure to ambient O₃ and cardiopulmonary mortality, with several analyses from the American Cancer Society (ACS) cohort reporting some positive associations, while other studies reported no association. There is generally limited evidence for an association with long-term exposure to ambient O₃ and total mortality. The findings for respiratory mortality are consistent and coherent with the evidence from epidemiologic, controlled human exposure, and animal toxicological studies for the effects of short- and long-term exposure to O₃ on respiratory effects. Respiratory mortality is a relatively small portion of total mortality [about 7.6% of all deaths in 2010 were due to respiratory causes ([Murphy et al., 2012](#))], thus it is not surprising that the respiratory mortality signal may be difficult to detect in studies of cardiopulmonary or total mortality. Based on the recent evidence for respiratory mortality along with limited evidence for total and cardiopulmonary mortality, the evidence **is suggestive of a causal relationship between long-term O₃ exposures and total mortality.**

In past O₃ AQCDs the effects of *long-term exposure to O₃ on the cardiovascular system* could not be thoroughly evaluated due to the paucity of information available. However, studies investigating O₃-induced cardiovascular events have advanced in the last two decades. Animal toxicological studies provide evidence for long-term O₃ exposure leading to cardiovascular morbidity, including increased vascular disease. There is limited, inconsistent evidence for cardiovascular morbidity in epidemiologic studies examining long-term exposure to O₃. Overall, animal toxicological studies provide some evidence for O₃-induced cardiovascular effects, but the effects observed were not consistently supported by controlled human exposure studies or epidemiologic studies. Thus, the overall body of evidence across disciplines **is suggestive of a causal relationship between long-term exposures to O₃ and cardiovascular effects.**

In the 2006 O₃ AQCD, there were a number of health effects for which an insufficient amount of evidence existed to adequately characterize the relationships with exposure to O₃. However, recent evidence suggests that O₃ may impart health effects through exposure durations and biological mechanisms not previously considered. For example, recent toxicological studies add to earlier evidence that *short- and long-term exposures to O₃ can produce a range of effects on the central nervous system and behavior*. Additionally, an epidemiologic study demonstrated that long-term exposure to O₃ affects memory in humans as well. Together the evidence from studies of short- and long-term exposure to O₃ **is suggestive of a causal relationship between O₃ exposure and central nervous system effects.** There is also limited though positive toxicological evidence for *O₃-induced*

developmental effects. Limited epidemiologic evidence exists for an association of O₃ concentration with decreased sperm concentration and associations with reduced birth weight and restricted fetal growth. Overall, the evidence **is suggestive of a causal relationship between long-term exposures to O₃ and reproductive and developmental effects.**

These causal determinations are summarized in [Table 2-1](#), along with the conclusions from the previous NAAQS review. Special emphasis and additional details are provided in [Table 2-1](#) for respiratory health outcomes, for which there is the strongest body of evidence.

Table 2-1 Summary of evidence from epidemiologic, controlled human exposure, and animal toxicological studies on the health effects associated with short- and long-term exposure to O₃.

Health Outcome	Conclusions from 2006 O ₃ AQCD	Conclusions from this ISA
Short-Term Exposure to O₃		
Respiratory effects	The overall evidence supports a causal relationship between acute ambient O ₃ exposures and increased respiratory morbidity outcomes.	Evidence integrated across controlled human exposure, epidemiologic, and toxicological studies and across the spectrum of respiratory health endpoints continues to demonstrate that there is a causal relationship between short-term O₃ exposure and respiratory health effects.
Lung function	Results from controlled human exposure studies and animal toxicological studies provide clear evidence of causality for the associations observed between acute (≤ 24 h) O ₃ exposure and relatively small, but statistically significant declines in lung function observed in numerous recent epidemiologic studies. Declines in lung function are particularly noted in children, asthmatics, and adults who work or exercise outdoors.	Recent controlled human exposure studies demonstrate group mean decreases in FEV ₁ in the range of 2 to 3% with 6.6 hour exposures to as low as 60 ppb O ₃ . The collective body of epidemiologic evidence demonstrates associations between short-term ambient O ₃ exposure and decrements in lung function, particularly in children with asthma, children, and adults who work or exercise outdoors.
Airway hyperresponsiveness	Evidence from human clinical and animal toxicological studies clearly indicate that acute exposure to O ₃ can induce airway hyperreactivity, thus likely placing atopic asthmatics at greater risk for more prolonged bouts of breathing difficulties due to airway constriction in response to various airborne allergens or other triggering stimuli.	A limited number of studies have observed airway hyperresponsiveness in rodents and guinea pigs after exposure to less than 300 ppb O ₃ . As previously reported in the 2006 O ₃ AQCD, increased airway responsiveness has been demonstrated at 80 ppb in young, healthy adults, and at 50 ppb in certain strains of rats.

Health Outcome	Conclusions from 2006 O ₃ AQCD	Conclusions from this ISA
Pulmonary inflammation, injury and oxidative stress	The extensive human clinical and animal toxicological evidence, together with the limited available epidemiologic evidence, is clearly indicative of a causal role for O ₃ in inflammatory responses in the airways.	Epidemiologic studies provided new evidence for associations of ambient O ₃ with mediators of airway inflammation and oxidative stress and indicate that higher antioxidant levels may reduce pulmonary inflammation associated with O ₃ exposure. Generally, these studies had mean 8-h max O ₃ concentrations less than 73 ppb . Recent controlled human exposure studies show O ₃ -induced inflammatory responses at 60 ppb, the lowest concentration evaluated.
Respiratory symptoms and medication use	Young healthy adult subjects exposed in clinical studies to O ₃ concentrations \geq 80 ppb for 6 to 8 h during moderate exercise exhibit symptoms of cough and pain on deep inspiration. The epidemiologic evidence shows significant associations between acute exposure to ambient O ₃ and increases in a wide variety of respiratory symptoms (e.g., cough, wheeze, production of phlegm, and shortness of breath) and medication use in asthmatic children.	The collective body of epidemiologic evidence demonstrates positive associations between short-term exposure to ambient O ₃ and respiratory symptoms (e.g., cough, wheeze, and shortness of breath) in children with asthma. Generally, these studies had mean 8-h max O ₃ concentrations less than 69 ppb .
Lung host defenses	Toxicological studies provided extensive evidence that acute O ₃ exposures as low as 80 to 500 ppb can cause increases in susceptibility to infectious diseases due to modulation of lung host defenses. A single controlled human exposure study found decrements in the ability of alveolar macrophages to phagocytize microorganisms upon exposure to 80 to 100 ppb O ₃ .	Recent controlled human exposure studies demonstrate the increased expression of cell surface markers and alterations in sputum leukocyte markers related to innate adaptive immunity with short-term O ₃ exposures of 80-400 ppb . Recent studies demonstrating altered immune responses and natural killer cell function build on prior evidence that O ₃ can affect multiple aspects of innate and acquired immunity with short-term O ₃ exposures as low as 80 ppb .
Allergic and asthma related responses	Previous toxicological evidence indicated that O ₃ exposure skews immune responses toward an allergic phenotype, and enhances the development and severity of asthma-related responses such as AHR.	Recent controlled human exposure studies demonstrate enhanced allergic cytokine production in atopic individuals and asthmatics, increased IgE receptors in atopic asthmatics, and enhanced markers of innate immunity and antigen presentation in health subjects or atopic asthmatics with short-term exposure to 80-400 ppb O ₃ , all of which may enhance allergy and/or asthma. Further evidence for O ₃ -induced allergic skewing is provided by a few recent studies in rodents using exposure concentrations as low as 200 ppb .

Health Outcome	Conclusions from 2006 O ₃ AQCD	Conclusions from this ISA
Respiratory Hospital admissions, ED visits, and physician visits	Aggregate population time-series studies observed that ambient O ₃ concentrations are positively and robustly associated with respiratory-related hospitalizations and asthma ED visits during the warm season.	Consistent, positive associations of ambient O ₃ with respiratory hospital admissions and ED visits in the U.S., Europe, and Canada with supporting evidence from single city studies. Generally, these studies had mean 8-h max O ₃ concentrations less than 60 ppb .
Respiratory Mortality	Aggregate population time-series studies specifically examining mortality from respiratory causes were limited in number and showed inconsistent associations between acute exposure to ambient O ₃ exposure and respiratory mortality.	Recent multicity time-series studies and a multicontinent study consistently demonstrated associations between ambient O ₃ and respiratory-related mortality visits across the U.S., Europe, and Canada with supporting evidence from single city studies. Generally, these studies had mean 8-h max O ₃ concentrations less than 63 ppb .
Cardiovascular effects	The limited evidence is highly suggestive that O ₃ directly and/or indirectly contributes to cardiovascular-related morbidity, but much remains to be done to more fully substantiate the association.	The overall body of evidence across disciplines indicates that there is likely to be a causal relationship for short-term exposures to O₃ and cardiovascular effects .
Central nervous system effects	Toxicological studies report that acute exposures to O ₃ are associated with alterations in neurotransmitters, motor activity, short- and long-term memory, sleep patterns, and histological signs of neurodegeneration.	Together the evidence from studies of short-term exposure to O ₃ is suggestive of a causal relationship between O₃ exposure and CNS effects .
Total Mortality	The evidence is highly suggestive that O ₃ directly or indirectly contributes to non-accidental and cardiopulmonary-related mortality.	Taken together, the body of evidence indicates that there is likely to be a causal relationship between short-term exposures to O₃ and total mortality .
Long-term Exposure to O₃		
Respiratory effects	The current evidence is suggestive but inconclusive for respiratory health effects from long-term O ₃ exposure.	Recent epidemiologic evidence, combined with toxicological studies in rodents and non-human primates, provides biologically plausible evidence that there is likely to be a causal relationship between long-term exposure to O₃ and respiratory health effects .
New onset asthma	No studies examining this outcome were evaluated in the 2006 O ₃ AQCD.	Evidence that different genetic variants (HMOX, GST, ARG), in combination with O ₃ exposure, are related to new onset asthma. These associations were observed when subjects living in areas where the mean annual 8-h max O ₃ concentration was 55.2 ppb , compared to those who lived where it was 38.4 ppb .

Health Outcome	Conclusions from 2006 O ₃ AQCD	Conclusions from this ISA
Asthma hospital admissions	No studies examining this outcome were evaluated in the 2006 O ₃ AQCD.	Chronic O ₃ exposure was related to first childhood asthma hospital admissions in a positive concentration-response relationship. Generally, these studies had mean annual 8-h max O ₃ concentrations less than 41 ppb .
Pulmonary structure and function	Epidemiologic studies observed that reduced lung function growth in children was associated with seasonal exposure to O ₃ ; however, cohort studies of annual or multiyear O ₃ exposure observed little clear evidence for impacts of longer-term, relatively low-level O ₃ exposure on lung function development in children. Animal toxicological studies reported chronic O ₃ -induced structural alterations, some of which were irreversible, in several regions of the respiratory tract including the centriacinar region. Morphologic evidence from studies using exposure regimens that mimic seasonal exposure patterns report increased lung injury compared to conventional chronic stable exposures.	Evidence for pulmonary function effects is inconclusive, with some new epidemiologic studies observing positive associations (mean annual 8-h max O ₃ concentrations less than 65 ppb). Information from toxicological studies indicates that long-term exposure during development among infant monkeys (500 ppb) and adult rodents (> 120 ppb) can result in irreversible morphological changes in the lung, which in turn can influence pulmonary function.
Pulmonary inflammation, injury and oxidative stress	Extensive human clinical and animal toxicological evidence, together with limited epidemiologic evidence available, suggests a causal role for O ₃ in inflammatory responses in the airways.	Several epidemiologic studies (mean 8-h max O ₃ concentrations less than 69 ppb) and toxicology studies (as low as 500 ppb) add to observations of O ₃ -induced inflammation and injury.
Lung host defenses	Toxicological studies provided evidence that chronic O ₃ exposure as low as 100 ppb can cause increases in susceptibility to infectious diseases due to modulation of lung host defenses, but do not cause greater effects on infectivity than short exposures.	Consistent with decrements in host defenses observed in rodents exposed to 100 ppb O ₃ , recent evidence demonstrates a decreased ability to respond to pathogenic signals in infant monkeys exposed to 500 ppb O ₃ .
Allergic responses	Limited epidemiologic evidence supported an association between ambient O ₃ and allergic symptoms. Little if any information was available from toxicological studies.	Evidence relates positive outcomes of allergic response and O ₃ exposure but with variable strength for the effect estimates; exposure to O ₃ may increase total IgE in adult asthmatics. Allergic indicators in monkeys were increased by exposure to O ₃ concentrations of 500 ppb .
Respiratory mortality	Studies of cardio-pulmonary mortality were insufficient to suggest a causal relationship between chronic O ₃ exposure and increased risk for mortality in humans.	A single study demonstrated that exposure to O ₃ (long-term mean O ₃ less than 104 ppb) elevated the risk of death from respiratory causes and this effect was robust to the inclusion of PM _{2.5} .

Health Outcome	Conclusions from 2006 O ₃ AQCD	Conclusions from this ISA
Cardiovascular Effects	No studies examining this outcome were evaluated in the 2006 O ₃ AQCD.	The overall body of evidence across disciplines is suggestive of a causal relationship for long-term exposures to O₃ and cardiovascular effects.
Reproductive and developmental effects	Limited evidence for a relationship between air pollution and birth-related health outcomes, including mortality, premature births, low birth weights, and birth defects, with little evidence being found for O ₃ effects.	Overall, the evidence is suggestive of a causal relationship between long-term exposures to O₃ and reproductive and developmental effects.
Central nervous system effects	Toxicological studies reported that acute exposures to O ₃ are associated with alterations in neurotransmitters, motor activity, short and long term memory, sleep patterns, and histological signs of neurodegeneration. Evidence regarding chronic exposure and neurobehavioral effects was not available.	Together the evidence from studies of long-term exposure to O ₃ is suggestive of a causal relationship between O₃ exposure and CNS effects.
Cancer	Little evidence for a relationship between chronic O ₃ exposure and increased risk of lung cancer.	Overall, the evidence is inadequate to determine if a causal relationship exists between ambient O₃ exposures and cancer.
Total Mortality	There is little evidence to suggest a causal relationship between chronic O ₃ exposure and increased risk for mortality in humans.	Collectively, the evidence is suggestive of a causal relationship between long-term O₃ exposures and total mortality.

2.5.3 Integrated Synthesis of Evidence for Health Effects

This section integrates the evidence for respiratory and cardiovascular effects (including mortality) across scientific disciplines and both short- and long-term exposure periods. Here, the complete body of evidence from both previous and the current NAAQS reviews is synthesized for the broad range of respiratory and cardiovascular effects associated with exposure to O₃.

2.5.3.1 Respiratory Effects

Building on evidence evaluated in previous O₃ AQCDs, recent evidence confirms and extends that O₃ is associated with a *broad range of respiratory effects, including altered development of the respiratory tract*. Recent animal toxicological studies of long-term exposure to O₃ occurring throughout various lifestages in monkeys, beginning with prenatal and early life exposures, provide novel evidence for effects on the development of the respiratory system, including ultrastructural changes in bronchiole development, increased offspring airway hyper-reactivity ([Section 7.4.8](#)),

9 ENVIRONMENTAL EFFECTS: OZONE EFFECTS ON VEGETATION AND ECOSYSTEMS

9.1 Introduction

This chapter synthesizes and evaluates the relevant science to help form the scientific foundation for the review of a vegetation- and ecologically-based secondary NAAQS for O₃. The secondary NAAQS are based on welfare effects. The Clean Air Act (CAA) definition of welfare effects includes, but is not limited to, effects on soils, water, wildlife, vegetation, visibility, weather, and climate, as well as effects on materials, economic values, and personal comfort and well-being. The effects of O₃ as a greenhouse gas and its direct effects on climate are discussed in [Chapter 10](#) of this document.

The intent of the ISA, according to the CAA, is to “accurately reflect the latest scientific knowledge expected from the presence of [a] pollutant in ambient air” (42 U.S.C.7408 and 42 U.S.C.7409). This chapter of the ISA includes scientific research from biogeochemistry, soil science, plant physiology, and ecology conducted at multiple levels of biological organization (e.g., molecular, organ, organism, population, community, ecosystem). Key information and judgments formerly found in the AQCDs regarding O₃ effects on vegetation and ecosystems are found in this chapter. This chapter of the O₃ ISA serves to update and revise Chapter 9 and AX9 of the 2006 O₃ AQCD ([U.S. EPA, 2006b](#)).

Numerous studies of the effects of O₃ on vegetation and ecosystems were reviewed in the 2006 O₃ AQCD. That document concluded that the effects of ambient O₃ on vegetation and ecosystems appear to be widespread across the U.S., and experimental studies demonstrated plausible mechanisms for these effects. Ozone effect studies published from 2005 to July 2011 are reviewed in this document in the context of the previous O₃ AQCDs. From 2005 to 2011, some areas have had very little new research published and the reader is referred back to sections of the 2006 O₃ AQCD for a more comprehensive discussion of those subjects. This chapter is focused on studies of vegetation and ecosystems that occur in the U.S. and that provide information on endpoints or processes most relevant to the review of the secondary standard. Many studies have been published about vegetation and ecosystems outside of the U.S. and North America, largely in Europe and Asia. This document includes discussion of studies of vegetation and ecosystems outside of North America only if those studies contribute to the general understanding of O₃ effects across species and ecosystems. For example, studies outside North America are discussed that consider physiological and biochemical processes that contribute to the understanding of effects of O₃ across species. Also, ecosystem studies outside of North America that contribute to the understanding of O₃ effects on general ecosystem processes are discussed in the chapter.

Sections of this chapter first discuss exposure methods, followed by effects on vegetation and ecosystems at various levels of biological organization and ends with policy-relevant discussions of exposure indices and exposure-response. [Figure 9-1](#) is a simplified illustrative diagram of the major endpoints O_3 may affect. First, [Section 9.2](#) presents a brief overview of various methodologies that have been, and continue to be, central to quantifying O_3 effects on vegetation (see AX9.1 of the 2006 O_3 AQCD for more detailed discussion) ([U.S. EPA, 2006b](#)). [Section 9.3](#) through [Section 9.4](#) begin with a discussion of effects at the cellular and subcellular level followed by consideration of the O_3 effects on plant and ecosystem processes ([Figure 9-1](#)). In [Section 9.3](#) research is reviewed from the molecular to the biochemical and physiological levels in impacted plants, offering insight into the mode of action of O_3 . [Section 9.4](#) provides a review of the effects of O_3 exposure on major endpoints at the whole plant scale including growth, reproduction, visible foliar injury and leaf gas exchange in woody and herbaceous plants in the U.S., as well as a brief discussion of O_3 effects on agricultural crop yield and quality. [Section 9.4](#) also integrates the effects of O_3 on individual plants in a discussion of available research for assessing the effect of O_3 on ecosystems, along with available studies that could inform assessments of various ecosystem services (see [Section 9.4.1.2](#)). The development of indices of O_3 exposure and dose modeling is discussed in [Section 9.5](#). Finally, exposure-response relationships for a number of tree species, native vegetation, and crop species and cultivars are reviewed, tabulated, and compared in [Section 9.6](#) to form the basis for an assessment of the potential risk to vegetation from current ambient levels of O_3 .

Exhibit 15

COMPARISON OF GROWTH AREAS AND EMISSIONS

1970-2015

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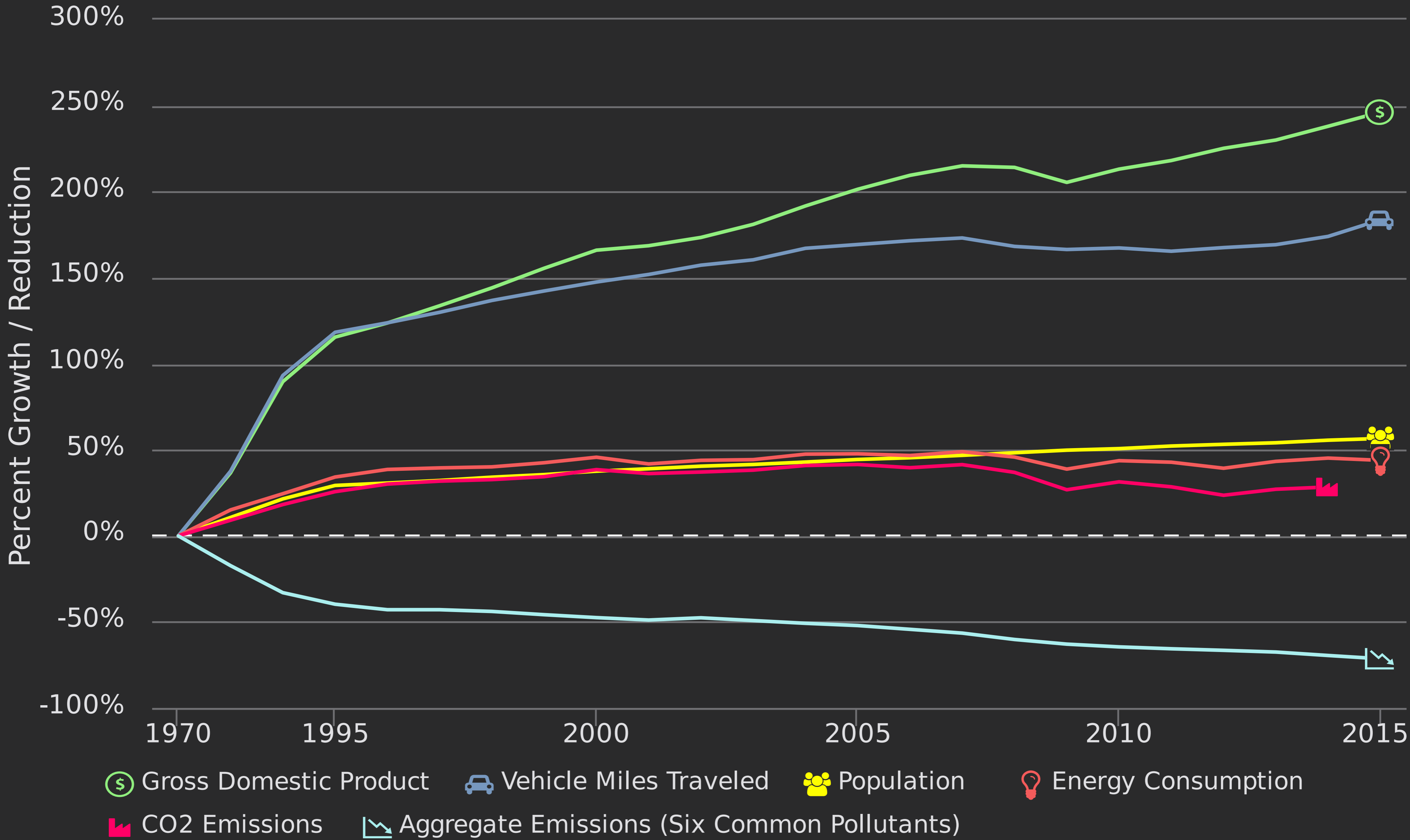


Exhibit 16



July 5, 2017

E. Scott Pruitt
Administrator
U.S. Environmental Protection Agency
William Jefferson Clinton Building
1200 Pennsylvania Avenue, NW
Mail Code: 1101A
Washington, DC 20460

Re: Request for Stay Pending Judicial Review of Extension of Deadline for Promulgating Initial Area Designations Under 2015 Ozone National Ambient Air Quality Standards, Announced by Letters to State Governors on June 6, 2017, and at 82 FR 29,246 (June 28, 2017)

BY MAIL AND E-MAIL

Dear Administrator Pruitt:

This is a petition to stay, pending judicial review, the effectiveness of the final action taken by EPA extending the deadline for promulgating initial area designations for the 2015 ozone national ambient air quality standards (“designations delay”), announced in letters to state governors dated June 6, 2017, *e.g.*, Letter from Scott Pruitt, Adm’r, EPA, to Doug Doucey, Gov. of Ariz., at 1, *available at* https://www.epa.gov/sites/production/files/2017-06/documents/az_ducey_6-6-17.pdf (“EPA Delay Letter”), and in a Federal Register notice published at 82 FR 29,246 (June 28, 2017) (“Second Announcement”) and titled “Extension of Deadline for Promulgating Designations for the 2015 Ozone National Ambient Air Quality Standards.” This action delays the legal deadline for EPA to promulgate designations for the entire country by a year, thus delaying the trigger date for attainment deadlines and for imposition of pollution controls that will significantly benefit public health and the environment. As explained below, EPA’s decision to take this action failed to comply with statutory requirements and was irrational.

The parties submitting this petition are American Lung Association, 55 W. Wacker Dr., Suite 1150, Chicago, IL 60601; Clean Air Council, 135 S. 19th St., Suite 300, Philadelphia, PA 19103; National Parks Conservation Association, 777 6th St. NW, Suite 700, Washington, DC 20001; Natural Resources Defense Council (NRDC), 40 W. 20th St., 11th floor, New York, NY 10011; Physicians for Social Responsibility, 1111 14th St. NW, Suite 700, Washington, DC 20005;

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Sierra Club, 2101 Webster St., Suite 1300, Oakland, CA 94612; and West Harlem Environmental Action, 1854 Amsterdam Ave., 2nd floor, New York, NY 10031 (collectively, "Petitioners").

We request that the agency act immediately on this request. Because of the rule's illegality and harmful effects, we (potentially along with others) intend to file a petition for review in the U.S. Court of Appeals for the District of Columbia Circuit shortly and to seek a stay from the Court. EPA's authority to stay Clean Air Act actions is unclear. To the extent EPA has such authority, and to the extent that it may be deemed necessary for any reason for Petitioners to submit such a request to EPA before seeking a stay pending judicial review from the Court, this is a petition requesting that EPA stay, pending judicial review, the effectiveness of the designations delay. Please inform the undersigned counsel by 6:00 p.m., on July 11, 2017, whether the agency will grant our request for a stay.

ARGUMENT

A stay of the designations delay is warranted because (1) Petitioners are likely to prevail on the merits of their claim; (2) Petitioners will suffer irreparable harm without a stay; (3) a stay will not substantially harm other parties; and (4) the public interest favors a stay. *See generally Wash. Metro Area Transit Comm'n v. Holiday Tours, Inc.*, 559 F.2d 841 (D.C. Cir. 1977).

I. PETITIONERS ARE LIKELY TO SUCCEED ON THE MERITS OF THEIR CLAIM BECAUSE THE DESIGNATIONS DELAY IS ILLEGAL AND ARBITRARY.

The sole bases for making designations are whether an area fails to meet the standards (a simple numerical comparison based on data EPA holds) and whether it contributes to another area's failure to meet the standards: if an area meets either condition, it is a nonattainment area; otherwise, it is an attainment area. 42 U.S.C. § 7407(d)(1)(A)(i)-(ii) (defining "nonattainment" and "attainment" area). EPA is required to promulgate designations "as expeditiously as practicable," with an outer deadline of two years from revision of standards. *Id.* § 7407(d)(1)(B)(i). The sole basis for extending that deadline (for up to a year) is where EPA "has insufficient information to promulgate the designations." *Id.* EPA's rationale for delaying designations in every area of the country by a year is untethered from those statutory considerations, unexplained, and baseless.

EPA's justification for the designations delay has nothing to do with the relevant statutory factors for extending the deadline for issuing designations. Congress allowed only one basis for EPA to extend its deadline for promulgating designations: where EPA "has insufficient information to promulgate the designations." *Id.* The information needed to promulgate designations is information about air quality at air quality monitoring sites and information

about what areas affect air quality in nearby areas with monitors that violate the standards, so that EPA can set the boundaries of nonattainment areas. *See id.* § 7407(d)(1)(A)(i)-(iii); *see also* Memorandum on Area Designations for the 2015 Ozone National Ambient Air Quality Standards, from Janet McCabe, Acting Ass't Adm'r, to Reg'l Adm'rs at 3-7 (Feb. 25, 2016), <https://www.epa.gov/sites/production/files/2016-02/documents/ozone-designations-guidance-2015.pdf> (“Designations Memo”) (describing EPA’s intended practice); *Miss. Comm’n on Env’tl. Quality v. EPA*, 790 F.3d 138, 147, 149, 158-59 (D.C. Cir. 2015) (describing EPA’s historical practice). Thus, the only factors relevant for EPA to justify extending the designations deadline relate to information about air quality conditions and areas that contribute to harmful air quality. Yet the Delay Letter and Second Announcement illegally seek to justify the designations delay based on considerations unrelated to these factors. *See Massachusetts v. EPA*, 549 U.S. 497, 535 (2007) (“EPA must ground its reasons for action or inaction in the statute.”).

EPA principally relies on the irrelevant fact that it is reviewing the standards themselves. That review is unrelated to the sole statutory ground for delaying designations. In the Delay Letter, the Agency says that, “[a]s part of the review process,” it is examining purported “issues that could undermine associated compliance efforts,” which are background ozone levels,¹ ozone originating abroad, and “exceptional events demonstrations.” EPA Delay Letter 1. Similarly, in the Second Announcement, EPA says it is “evaluating a host of complex issues regarding the 2015 ozone NAAQS and its implementation,” citing background ozone levels and ozone originating abroad, which purportedly prevent it from even determining if it has insufficient information to allow it to make designations. 82 FR 29,247/2. EPA frames the designations delay as purportedly justified “[i]n light of the analyses currently underway at the agency.” *Id.* 29,247/3. But review of the standards or issues about implementing them says nothing about the sufficiency of the information about air quality that is needed to make designations under these still-effective standards. *See, e.g., Motor Vehicle Mfrs. Ass’n v. State Farm Mut. Auto. Ins.*, 463 U.S. 29, 43 (1983).

Even if the review were relevant, the statute does not allow EPA to delay designations simply because it might have insufficient information. Congress requires EPA to promulgate designations “as expeditiously as practicable” and authorized delay only where EPA “has insufficient information.” 42 U.S.C. § 7407(d)(1)(B)(i) (emphasis added). Congress thus mandated EPA to move swiftly, delaying only if it actually possesses—as opposed to might possess—insufficient information. Because EPA has not found the information it has is insufficient, it cannot lawfully or rationally grant itself an extension under § 7407(d)(1)(B)(i). *See*

¹ *See* 80 FR 65,292, 65,328/1 (Oct. 26, 2015) (“U.S. background” means ozone “that would exist even in the absence of any manmade emissions within the United States”).

Chamber of Commerce v. SEC, 412 F.3d 133, 143 (D.C. Cir. 2005) (where Congress requires agency to make certain assessment, agency must do so).

The designations delay is also fundamentally arbitrary because EPA has given no explanation of how the information it has is insufficient to promulgate designations. It has entirely failed to explain how the purported “issues” implicated by its review of the 2015 standards affect designations. It identifies no way in which “background ozone levels” or “international transport” are relevant to designations, nor can it. 82 FR 29,247/2. Indeed, the Act specifically deals with international transport at the post-designation stage, during actual implementation. *See* 42 U.S.C. § 7509a(a)-(b). Addressing it at the designation stage would thus conflict with Congress’s careful implementation design. EPA’s attempt to justify the designations delay based on speculation that it might find new approaches for states to comply with the standards is utterly irrelevant to the issue of whether communities are currently meeting or violating the standards. 82 FR 29,247/2-3 (discussing new “Ozone Cooperative Compliance Task Force” (emphasis added)). Because EPA has failed to provide the required explanation for how the evidence before it supports the designations delay, the designations delay is arbitrary. *See American Lung Ass’n v. EPA*, 134 F.3d 388, 392 (D.C. Cir. 1998) (“Where, as here, Congress has delegated to an administrative agency the critical task of assessing the public health and the power to make decisions of national import in which individuals’ lives and welfare hang in the balance, that agency has the heaviest of obligations to explain and expose every step of its reasoning.”).

Even if EPA’s stated bases for wanting more time were statutorily relevant, they are meritless. EPA claims it cannot even tell if the information it has is insufficient, 82 FR 29,247/2, but it hasn’t provided any explanation, much less a rational one, about why it cannot assess the information it has when it has all the information the statute and its own guidance say EPA needs. *See* Letter from Dannel Malloy, Gov. of Conn., to Scott Pruitt, Adm’r, EPA, at 3 (June 20, 2017) (“There is nothing missing from past information used by EPA to designate areas after previous revisions to the ozone NAAQS.”).

Even if some information for some areas were insufficient—a claim EPA has not supported—EPA has not explained why a one-year extension for all areas is justified. The Act instructs EPA to make designations “as expeditiously as practicable,” and the length of any extension beyond the two-year outer deadline is limited to being “up to one year.” 42 U.S.C. § 7407(d)(1)(B)(i) (emphasis added). Thus, a one-year extension is not a default: it is an outer bound, with EPA obligated to move as quickly as practicable to promulgate designations. Here, EPA has given no explanation why a full year’s extension is reasonable or necessary anywhere, let alone for every single area of the nation. That is arbitrary. *See American Lung*, 134 F.3d at 392. Further, because EPA failed to address the statutory requirement for expedition, the

designations delay is unlawful and arbitrary. *Public Citizen v. FMCSA*, 374 F.3d 1209, 1216 (D.C. Cir. 2004) (“the complete absence of any discussion of a statutorily mandated factor leaves us with no alternative but to conclude that the agency failed to take account of this statutory limit on its authority, making the agency’s reasoning arbitrary and capricious.”).

EPA has also failed to explain why the information it has is insufficient to allow it to make any designations. EPA claims that its review of certain issues relating to the standards and their implementation, like background ozone levels, international transport of ozone pollution, and exceptional events, affects its ability to make designations. 82 FR 29,247/2-3. Even if that were true—and it patently is not—EPA has not explained why those issues are relevant over every square inch of the nation.

To the contrary, far from claiming that background ozone levels are high enough to matter for regulatory purposes everywhere in the country, EPA has made clear that “the locations that are most strongly influenced by background [ozone] are relatively limited in scope, i.e., rural areas in the intermountain western U.S.” EPA-HQ-OAR-2008-0699-4309 at 347 (EPA Response to Comments on establishment of 2015 standards). The same holds for international transport. 80 FR at 65,328/2. Neither has EPA shown that all—or even most—areas of the country claim to experience exceedances of the standards resulting from exceptional events. EPA has thus failed to explain why it chose to delay designations for the entire country.

If there were insufficient information for certain areas, EPA had a time-tested alternative to a national delay, but arbitrarily failed to consider it. In prior designations, EPA has routinely issued designations for certain areas while deferring designations for others for which it lacked sufficient information. It was arbitrary for EPA not even to consider this option of making some designations but deferring others. *E.g., Del. Dep’t of Nat. Res. v. EPA*, 785 F.3d 1, 17-18 (D.C. Cir. 2015) (where EPA failed to consider “reasonable alternatives” to uniform national rule, “its action was not rational and must, therefore, be set aside”).

II. ABSENT A STAY, THE DESIGNATIONS DELAY WILL IRREPARABLY HARM PETITIONERS.

Judicial review of the designations delay will extend beyond October 1, 2017. Even expedited cases often take over a year to be decided. For example, a challenge to an EPA action was filed in April 2013, expedited in June 2013, and decided in April 2014. *Sierra Club v. EPA*, No. 13-1112 (D.C. Cir.) (petition filed Apr. 5, 2013); Order, *NRDC v. EPA*, No. 10-1371 (consolidated with No. 13-1112) (D.C. Cir. June 11, 2013) (granting request for expedited consideration); *NRDC v. EPA*, 749 F.3d 1055 (D.C. Cir. 2014) (decision in consolidated cases issued April 18, 2014). Thus, without a stay, litigation over the designations delay will likely last deep into 2018, and easily even until October 2018, after EPA’s new deadline for promulgating

designations. Without a stay pending judicial review, Petitioners' members face a substantial likelihood of irreparable harm resulting from the designations delay.

By delaying designations, EPA delays pollution controls that otherwise would result from implementing the 2015 standards in communities where Petitioners' members live, work, and enjoy recreation. Such communities include areas currently designated as attainment under the 2008 standards but whose ozone levels violate the 2015 standards; because of the designations delay, they will not be designated nonattainment and thus will lack the anti-pollution protections that a nonattainment designation would accord them. These important protections include measures states adopt into their implementation plans to limit emissions of ozone-forming chemicals sufficiently for the area to come into attainment, as well as attainment deadlines, and nonattainment new source review for new or modified major sources of pollution, like factories and power plants.

The delay in implementing pollution controls irreparably harms Petitioners' members in several ways. EPA itself admits that compliance with the 2015 standards will prevent health-related harms, including premature deaths, asthma attacks in children, and health harms resulting in lost work days and missed school days for children. EPA, EPA-452/R-15-007, *Regulatory Impact Analysis of the Final Revisions to the National Ambient Air Quality Standards for Ground-Level Ozone* at ES-16, tbl.ES-6 (2015) ("RIA"); see also *id.* ES-2 to -3. Petitioners' members living, working, and recreating in areas attaining the 2008 standards, but with ozone levels that violate the 2015 standards—and Petitioners have tens of thousands of members residing in such areas—routinely find their ability to breathe impaired and their ability to work and their children's ability to attend school impeded. Their health and wellbeing are threatened in serious ways.

Because the attainment deadlines run from the date of designation, the ozone pollution levels in these areas will be allowed to remain at dangerously elevated levels for an additional year because of EPA's year-long designations delay absent a stay of EPA's unlawful action. Petitioners' members living in such areas, who experience severe harms to their health and wellbeing because of ozone pollution, will thus be harmed both by the delay in mandatory pollution reductions and by the additional year that they will have to endure dangerous ozone levels.

Even in areas violating both standards, the designations delay means Petitioners' members there will receive neither the benefits of pollution reductions designed to drive compliance with the new, more protective 2015 standards nor the benefit of actual compliance with those standards as soon as they would absent the designations delay. Accordingly, the harms described above affect even more of Petitioners' members than just those living in areas attaining the 2008 standards but violating the 2015 standards.

Particular harms will affect Petitioners' members who live, work, and recreate in areas that will evade nonattainment new source review for ozone because of EPA's designations delay. Were such areas designated nonattainment, plants under development that would be subject to nonattainment new source review would have to limit their emissions of ozone-forming compounds to the very stringent "lowest achievable emissions rate," and they would have to secure reductions of more ozone-forming pollution from other sources in the area than they would add to the air. By the time nonattainment designations are made, many of these new source additions or expansions will likely already be approved and potentially even constructed, having successfully evaded the controls that would apply absent EPA's illegal delay.

These human health harms resulting directly from EPA's designations delay are irreparable, for ultimate success on the merits cannot undo them: no court order can enable EPA to raise the dead, undo asthma attacks, or restore a missed day in the classroom or at work. See *Wisc. Gas Co. v. FERC*, 758 F.2d 669, 674 (D.C. Cir. 1985). Petitioners' members, their families, and, for medical professionals, their patients have demonstrated heightened vulnerability to ozone pollution—they already experience serious harms from it. The harms to them unless EPA's designations delay is stayed are "likely," as well as "certain and great," "actual and not theoretical," "beyond remediation," and so "imminent that there is a clear and present need for equitable relief to prevent irreparable harm." *League of Women Voters v. Newby*, 838 F.3d 1, 6-8 (D.C. Cir. 2016). Even the mere potential of exposure to harmful substances can constitute irreparable harm, and Petitioners have shown far more than that. See *Nat'l Ass'n of Farmworkers Orgs. v. Marshall*, 628 F.2d 604, 613-14 (D.C. Cir. 1980) (potential that children will be exposed to substances that could harm their health constitutes irreparable harm).

Additionally, for the same reasons, the designations delay is substantially likely to cause irreparable environmental harms. Ozone also damages vegetation and forested ecosystems, causing or contributing to widespread stunting of plant growth, tree deaths, visible leaf injury, reduced carbon storage, and damaging entire ecosystems. EPA-HQ-OAR-2008-0699-0404 at 5-2 to -3; EPA-HQ-OAR-2008-0699-0405 at 9-1; 80 FR 65,370/1-2, 65,377/3. EPA acknowledges that, "[i]n terms of forest productivity and ecosystem diversity, ozone may be the pollutant with the greatest potential for region-scale forest impacts." RIA 7-3. Such widespread vegetation and ecosystem losses are the type of environmental harms that are irreparable, as they cannot "be adequately remedied by money damages" and are of "permanent or at least of long duration." *Amoco Production Co. v. Village of Gambell, Alaska*, 480 U.S. 531, 545 (1987) ("If such injury is sufficiently likely...the balance of harms will usually favor the issuance of an injunction to protect the environment.").

III. A STAY WILL NOT HARM OTHER PARTIES.

As the agency responsible for the proper execution of the Clean Air Act, EPA cannot be substantially harmed by a stay that would prevent it from giving effect to a rule that contradicts the Act and is arbitrary. *See Nat'l Ass'n of Farmworkers*, 628 F.2d at 615 (“consequences [that] are no different from [agency’s] burdens under the statutory scheme” “do not constitute substantial harm for the purpose of delaying injunctive relief”). For the same reason, industry and states are also not cognizably harmed by having to comply with the ozone implementation program Congress enacted. History shows this country can have both economic growth and air pollution reductions. *See, e.g.*, https://gispub.epa.gov/air/trendsreport/2016/#econ_growth_cleaner_air_ (from 1970-2015, emissions of the six pollutants most directly limited by national ambient air quality standards down 71%, and gross domestic product up nearly 250%). Some states will have to undertake planning for pollution control, and some industrial pollution sources will have to control their emissions, but that is not a cognizable harm. *See Fla. Power & Light Co. v. EPA*, 145 F.3d 1414, 1421 (D.C. Cir. 1998) (petitioner power company will not be harmed by participation in additional administrative and judicial proceedings).

Moreover, a stay would not prevent EPA from reviewing the 2015 standards or their implementation. The Act plainly contemplates that EPA will implement ozone standards even as it reviews them. Indeed, the Act puts the ozone standards on an implementation schedule that can last up to 20 years, while requiring EPA to review standards at least every 5 years. *Whitman v. Am. Trucking Ass'ns*, 531 U.S. 457, 485 (2001) (“Congress knew” EPA could review ozone standards at any time, but still established implementation framework “reaching...far into the future” (discussing 42 U.S.C. §§ 7409(d)(1), 7511(a), and various provisions in 7511a). Thus, EPA would not experience harm from staying the designations delay.

IV. THE PUBLIC INTEREST DECISIVELY FAVORS STAYING THE DESIGNATIONS DELAY.

EPA projects that compliance with the 2015 standards will realize significant health benefits. In the areas of the country outside California, EPA estimates that compliance with the standards will result in upwards of 600 lives saved, over 250 heart attacks avoided, about 1,000 hospital admissions or emergency room visits prevented, 230,000 asthma attacks in children not occurring, and 160,000 school loss days for children averted. RIA at ES-16 tbl.ES-6. The economic value of these benefits substantially outweighs the costs of achieving them, moreover. *Id.* at ES-15 tbl.ES-5. In making its estimate of public health benefits, EPA assumed designations would be completed in late 2017. *See id.* at ES-2 to -3. These health benefits would come on top of the public health gains from achieving the 2008 standards and from several other pollution reduction requirements EPA put in place around the time it finalized the 2015 standards. *Id.* 6-1

("The benefits...are estimated as being incremental to attaining the existing standard of 75 ppb.... These estimated benefits are incremental to the benefits estimated for several recent rules....").

These protections are already years overdue. EPA's deadline for reviewing and revising the 2008 standards fell in March 2013. *See* 42 U.S.C. § 7409(d)(1) (five-year review cycle); 73 FR 16,436 (2008) (standards promulgated in March 2008). EPA acted over 18 months late, and only after being sued (a suit in which it requested still more time to finalize its review and revision of the 2008 standards). Order 1-2, *Sierra Club v. EPA*, No. 13-cv-2809 (N.D. Cal. Apr. 30, 2014) (rejecting deadline EPA sought for finishing rulemaking).

The designations delay means that Congress's carefully refined ozone nonattainment provisions will not engage as quickly as EPA assumed in its analysis. The pollution reductions they are specifically designed to assure will not occur as quickly, either. *See South Coast Air Quality Mgmt. Dist. v. EPA*, 472 F.3d 882, 887-88 (D.C. Cir. 2006), *amended in other parts* 489 F.3d 1245 (D.C. Cir. 2007) (describing basis for and functioning of ozone nonattainment provisions of Clean Air Act). Thus, compliance with them will be delayed, and Congress's promise to all residents of this country that they will have safe air to breathe will go unfulfilled even longer than it already has. Significant numbers of lives will be worsened as a result, even though they need not be. That cannot be consistent with the public interest, which Congress established as curbing air pollution. *Union Elec. v. EPA*, 427 U.S. 246, 256 (1976) (Clean Air Act is "a drastic remedy to what was perceived as a serious and otherwise uncheckable problem of air pollution").

CONCLUSION

For the foregoing reasons, a stay of the designations delay is warranted.

As delay of relief promises irreparable harm to Petitioners, we respectfully request that you inform the undersigned counsel by 6:00 p.m., on July 11, 2017, whether the agency will grant our request for a stay.

DATED: July 5, 2017

Respectfully submitted,

/s/Scott Strand (with permission)

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/s/Seth L. Johnson

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Resources Defense Council, Physicians for Social
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Environmental Action*

/s/Ann Brewster Weeks (with permission)

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Counsel for Clean Air Council

Exhibit 17



July 10, 2017

E. Scott Pruitt
Administrator
U.S. Environmental Protection Agency
William Jefferson Clinton Building
1200 Pennsylvania Avenue, NW
Mail Code: 1101A
Washington, DC 20460

Re: Additional Petitioners Requesting for Stay Pending Judicial Review of Extension of Deadline for Promulgating Initial Area Designations Under 2015 Ozone National Ambient Air Quality Standards, Announced by Letters to State Governors on June 6, 2017, and at 82 FR 29,246 (June 28, 2017)

BY MAIL AND E-MAIL

Dear Administrator Pruitt:

On July 5, 2017, American Lung Association, Clean Air Council, National Parks Conservation Association, Natural Resources Defense Council, Physicians for Social Responsibility, Sierra Club, and West Harlem Environmental Action petitioned you to stay, pending judicial review, the effectiveness of the final action taken by EPA extending the deadline for promulgating initial area designations for the 2015 ozone national ambient air quality standards (“designations delay”), announced in letters to state governors dated June 6, 2017, *e.g.*, Letter from Scott Pruitt, Adm’r, EPA, to Doug Ducey, Gov. of Ariz., at 1, *available at* https://www.epa.gov/sites/production/files/2017-06/documents/az_ducey_6-6-17.pdf, and in a Federal Register notice published at 82 FR 29,246 (June 28, 2017) and titled “Extension of Deadline for Promulgating Designations for the 2015 Ozone National Ambient Air Quality Standards.” The following additional parties hereby join the previously filed petition: American Public Health Association, 800 I St. NW, Washington, DC 20001; and American Thoracic Society, 25 Broadway, 18th floor, New York, NY 10004.

DATED: July 10, 2017

Respectfully submitted,

/s/Seth L. Johnson

Seth L. Johnson

Laura Dumais

David S. Baron

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*Counsel for American Public Health Association
and American Thoracic Society*

Exhibit 18

July 10, 2017

E. Scott Pruitt
Administrator
U.S. Environmental Protection Agency
William Jefferson Clinton Building
1200 Pennsylvania Avenue, NW
Mail Code: 1101A
Washington, DC 20460

ELECTRONICALLY TRANSMITTED

Re: Environmental Defense Fund Joining Request to Stay Pending Judicial Review *Extension of Deadline for Promulgating Designations for the 2015 Ozone National Ambient Air Quality Standards*, 82 Fed. Reg. 29,246 (June 28, 2017)

Dear Administrator Pruitt:

On July 5, 2017, American Lung Association, Clean Air Council, National Parks Conservation Association, Natural Resources Defense Council, Physicians for Social Responsibility, Sierra Club, and West Harlem Environmental Action petitioned you to stay, pending judicial review, the effectiveness of the final action taken by EPA extending the deadline for promulgating initial area designations for the 2015 ozone national ambient air quality standards (“designations delay”), announced in letters to state governors dated June 6, 2017, e.g., Letter from Scott Pruitt, Adm’r, EPA, to Doug Ducey, Gov. of Ariz., at 1, *available at* https://www.epa.gov/sites/production/files/2017-06/documents/az_ducey_6-6-17.pdf, and in a Federal Register notice published at 82 Fed. Reg. 29,246 (June 28, 2017) and titled “Extension of Deadline for Promulgating Designations for the 2015 Ozone National Ambient Air Quality Standards.” On July 10, 2017, the American Public Health Association and American Thoracic Society wrote you indicating their intention to join the stay petition.

Environmental Defense Fund (EDF) hereby joins the July 5 petition. As explained in the petition, EPA’s decision to delay the ozone designations failed to comply with statutory requirements and was irrational. EPA’s action harms EDF members who live, work, and recreate in areas that will now evade nonattainment requirements for ozone because of EPA’s designations delay. You should immediately stay that unlawful and harmful action.

DATED: July 10, 2017

Respectfully submitted,

Peter Zalzal
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Exhibit 19



July 11, 2017

Transmitted by electronic mail

E. Scott Pruitt, Administrator
U.S. Environmental Protection Agency
William Jefferson Clinton Building
1200 Pennsylvania Avenue, N.W.
Mail Code: 1101A
Washington, D.C. 20460

RE: The Ohio Environmental Council Joining Administrative Petition to Stay, Pending Judicial Review, the *Extension of Deadline for Promulgating Designations for the 2015 Ozone National Ambient Air Quality Standards*, 82 Fed. Reg. 29,249 (June 28, 2017).

Dear Mr. Pruitt:

On July 5, 2017, American Lung Association, Clean Air Council, National Parks Conservation Association, Natural Resources Defense Council, Physicians for Social Responsibility, Sierra Club, and West Harlem Environmental Action petitioned you to stay, pending judicial review, the effectiveness of the final action taken by EPA extending the deadline for promulgating initial area designations for the 2015 ozone National Ambient Air Quality Standards (“designations delay”), first announced in letters to state governors dated June 6, 2017, e.g., Letter from Scott Pruitt, Adm’r, EPA to Doug Ducey, Gov. of Ariz., at 1, *available at*:

https://www.epa.gov/sites/production/files/2017-06/documents/az_ducey_6-6-17.pdf, and late published at 82 Fed. Reg. 29,246 (June 28, 2017), entitled *Extension of Deadline for Promulgating Designations for the 2015 Ozone National Ambient Air Quality Standards*. On July 10, 2017, the American Public Health Association, American Thoracic Society, and Environmental Defense Fund wrote you indicating their intention to join the stay petition.

The Ohio Environmental Council hereby also joins the July 5, 2017 petition. As explained in the petition, EPA’s decision to delay the ozone designations failed to

comply with statutory requirements and was capricious and irrational. Your designations delay action must be immediately stayed.

DATED: July 11, 2017

Respectfully submitted,



Ann Brewster Weeks
Legal Director, CATF
aweeks@catf.us
(617) 359-4077

Counsel to:
The Ohio Environmental Council

Cc: Trent Dougherty, The OEC
Kevin Minoli, U.S. EPA
Denise Scott, U.S. EPA

Exhibit 20

DECLARATION OF PETER ADLER

I, Peter Adler, hereby declare as follows:

1. I am currently a member of the Natural Resources Defense Council (“NRDC”). I have been a member for one year.

2. I live in Salt Lake City, in Salt Lake County. I have lived at my current address for 9 years.

3. When the air quality in my community is poor, it is noticeable to me. Specifically, in the winter, the air inversion that happens over Salt Lake is particularly noticeable to me. I make a point to always check the air quality online when I notice these inversions, and to plan my outdoor activity accordingly. In some winters, the cumulate number of days with poor air quality may approach six weeks, and I avoid exercising outdoors on those days.

4. My wife is a physician, and we have a six year old child. Accordingly, I am very concerned about air pollution and other forms of pollution that impact public health. We love Salt Lake City, but when we think about whether or not we could stay here forever, the poor air quality is one of the main reasons we think about leaving. In fact, we have friends who have left the area because of the poor air quality.

5. My wife studied the impacts of air pollution when she was in medical school, and has relayed information to me about the dangers of various pollutants. In particular, I’m aware of the risks to the heart, lungs, and the inflammatory response that air pollution can have on the body. I’m aware that inhaling elevated

concentrations of particulate matter and ozone pollution can cause immediate injury to a person, as well as increase that person's risk of developing serious and even life-threatening illnesses. For these reasons, I am very concerned about the health threat posed to my health and the health of my family and community by particulate pollution and ozone pollution.

6. My family and I are very active. I run, bike, and ski outside, and I'm often outside with my six year old. When the air quality is bad, I limit my time outdoors and also the time my child spends outdoors. On bad air quality days, I don't run or bike outside, and I don't take my child to the park.

7. I believe that regulating particulate pollution and ozone pollution will help reduce the amount of air pollution to which I am exposed. I am concerned that if the U.S. EPA weakens air quality standards or rules enforcing these standards, that there will be a negative impact on my health and the health of my community. I support litigation by NRDC to ensure that requirements of the Clean Air Act for controlling air pollution like particulate pollution and ozone pollution are fully and expeditiously implemented so as to protect my health.

I declare that the foregoing is true and correct to the best of my knowledge, information, and belief. Executed in Salt Lake City, Utah on June 23, 2017

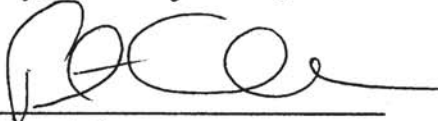

Peter Adler

Exhibit 21

DECLARATION OF RHONDA ANDERSON

I, Rhonda Anderson, hereby declare and state:

1. I am a member of the Sierra Club. I have been a member for 17 years. I also work for the Club as an organizer in its Detroit office, in Midtown.

2. I live in Detroit, Michigan. I grew up here, and have lived in the city my entire life. I currently live in the Davison Dexter area. I previously lived in Midtown, and I grew up in River Rouge. The River Rouge area where I grew up is the most polluted area in the state. It has operating coal-fired power plants, a coke battery plant, an auto plant, steel mills, a tar sands oil refinery, mineral processors, asphalt plants, and others, all of which emit air pollutants. I have no plans to move out of the Detroit area. I love this city. I think we're some of the strongest, most resilient people around.

3. I'm 66, and I have grandchildren and great-grandchildren. Two of my grandchildren live with me; along with one of my daughters, who has very bad asthma; and my son. When my daughter was pregnant with my granddaughter who was born in March 2015, her asthma got worse and I had to take her to the emergency room because of it. My two newest grandchildren—one lives with me, and both just turned 2-years-old—have already been diagnosed with asthma. So many young and very young children, especially African American ones like mine, have asthma here that it almost seems normal for children to have it. That is an unnatural norm, and it's just morally wrong.

4. I have had asthma as long as I can remember. I had asthma so bad and stayed in the hospital so much that they put my bed in the hallway by the nurse's station. As an adult, I've also sometimes had to go to the hospital because of my asthma. We're really dependent on

inhalers and medication because the air here is so bad. I usually use my inhaler a few times a week. If I have a cold, it makes things much worse.

5. Two of my granddaughters also use nebulizers, including one who lives with me. One of my granddaughters who lives in the Detroit metro area had to go to the hospital for an asthma attack, and had to stay in the hospital overnight last summer.

6. I've spent what seems like days at emergency rooms with my children and now grandchildren because of asthma. Going to the hospital for an asthma attack is a difficult experience. You have to go to the emergency room, which is almost always crowded and does not operate on a first come, first served basis. You know to be patient, but your child is sick and you want them to be seen right away. It's very upsetting. Often, I have spent no less than 4 hours waiting, all the while wondering when my child will be able to see the doctor. Once we are called, we get sent to a second crowded room with breathing machines, and every station is filled with a child having an asthma attack. It is very stressful and there is nothing that can take my mind off the fact that I have a sick child that I cannot help.

7. I spend a lot of time outdoors both for exercise and for work. Along with my daughters and their two infants, I walk around Palmer Park about twice a week for exercise. When we aren't walking there, we're walking around the neighborhood about 3 times a week. At work, I walk around outside daily. Also, as part of my organizing, I go to the River Rouge area regularly—at least 3-4 times a week. I lead tours there as part of my work and participate in outdoor events, especially in the summer.

8. I know from my work and my personal experience that we have bad air here and the impacts of bad air. I'm most definitely concerned about impact of ozone pollution on me and my family. My aunt died in my grandmother's arms from an asthma attack. I know many people

who have died from asthma attacks, including young adults. I am aware that people with asthma and older adults are especially vulnerable to ozone air pollution.

9. I know from my work in the community that there are proposals to build new roads and bridges connecting Detroit to Canada, to serve trade. There is already a lot of truck traffic here as trucks drive through Detroit to get to and from Canada, I am concerned about more trucks making for more air pollution. I also am aware that there is a plan to build a scrap metal yard just south of Detroit in Melvindale. I'm worried that the scrap metal yard will increase air pollution in the area both by its operations and through increased traffic to the yard.

10. I am aware that ozone pollution levels in the Detroit area are higher than allowed under EPA's new ozone clean air standards. I am also aware that EPA delayed making air quality designations for the new standard. I'm most definitely concerned that any delay in implementing the standard threatens my health and my family's health. I don't know how much more we can bear. We have to take on so much as is. Delayed cleanup of the air harms me because it allows the air to stay dirty for longer. Being already burdened by asthma, my family and I are especially endangered. I support the Sierra Club's efforts to make sure my health and my family's health are protected against pollution through the timely and full implementation of the Clean Air Act.

I declare under penalty of perjury that the foregoing is true and correct.

Executed on June 30, 2017.

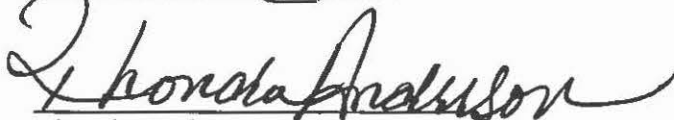

Rhonda Anderson

Exhibit 22

DECLARATION OF Georges C. Benjamin, MD

I, Georges C. Benjamin, MD declare as follows:

1. I am Georges C. Benjamin, MD, the Executive Director of the American Public Health Association. I have worked at the American Public Health Association since December 2002. In that capacity, I am required to be familiar with the organization's structure, function, purpose, and membership.

2. APHA is incorporated in Massachusetts and headquartered in Washington, DC. APHA has 54 state and regional Affiliates representing all 50 states, the District of Columbia and Puerto Rico. APHA is recognized as a not-for-profit corporation under section 501(c)(3) of the United States Internal Revenue Code.

3. The American Public Health Association champions the health of all people and all communities and strengthens the profession of public health, shares the latest research and information, promotes best practices and advocates for public health policies grounded in research. APHA represents over 20,000 individual members and is the only organization that combines a 140-plus year perspective and a broad-based member community with an interest in improving the public's health. APHA has long advocated in support of the Clean Air Act and for strong public health protections from ozone and other dangerous air pollutants.

4. APHA has members and Affiliate members throughout the country residing in counties with ozone levels that are below the 2008 national ambient air quality standard ("NAAQS") for ozone of 0.075 parts per million ("ppm") averaged over eight hours, but above 0.070 ppm, the level that EPA adopted as the standard in October 1, 2015, because of the scientific evidence of harm to human health above that level. The harm to human health includes

increased risk of asthma attacks, hospitalization and premature death. The individuals residing in these areas would benefit from the cleanup required to meet the more protective ozone standards.

5. APHA also has members and Affiliate members throughout the country that reside in areas exceeding the both the 2008 and 2015 standards for ozone. These individuals would also benefit from implementation of the 2015 ozone standards.

6. APHA members living in the above-named and other areas have a strong interest in full, timely, and effective implementation of the Clean Air Act requirements designed to protect them from unhealthy levels of pollution in the air they breathe. EPA's delayed implementation of the 2015 NAAQS for ozone by extending the Clean Air Act's statutory deadline for making appropriate determinations of attainment and nonattainment denies APHA's members and their patients the protections guaranteed under the Clean Air Act. This delayed implementation harms APHA's members and patients living in the above-named and other areas by allowing increased, prolonged exposure to harmful pollution that causes serious health consequences like asthma attacks, hospitalization, and, likely, premature death.

I declare under penalty of perjury that the foregoing is true and correct.

Executed in Washington, D.C. on June 29, 2017.

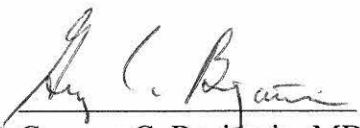

Georges C. Benjamin. MD

Exhibit 23

DECLARATION OF JOSHUA BERMAN

1. I am a Senior Attorney with Sierra Club in Washington, DC. I have worked as an attorney at the Sierra Club since 2011, first as an Associate Attorney, from 2013 to June 2017 as a Staff Attorney, and since June 2017 as a Senior Attorney. I have a law degree from Yale Law School, and a Master's degree in Environmental Science from the Yale School of Forestry and Environmental Studies. I attended Yale College as an undergraduate, where I received my Bachelor's degree in mathematics. After I graduated from Yale Law School, I clerked for a judge of the United States Court of Appeals for the Fourth Circuit in Charleston, West Virginia. I was admitted to the California Bar in 2009, and to the District of Columbia Bar in 2012.
2. As a Senior Attorney, I am a member of Sierra Club's Environmental Law Program. As part of my job, I perform a broad variety of advocacy activities to promote protection of public health and the environment from air pollution.
3. My work includes advocacy on federal rulemakings and actions related to ground-level ozone pollution. I have been involved with this work since 2014, and since 2013, I have also been working on implementation of ozone standards at the state level. Some of my responsibilities in this area include helping to coordinate and plan the organization's legal advocacy with respect to these rulemakings; participating in the drafting of comments on these rulemakings; collecting, reviewing, and analyzing air quality monitoring data; and working with members and outside legal counsel to assist in and coordinate advocacy and litigation efforts related to our work on national ozone standards and their implementation. My work also encompasses involvement in state-level rulemakings about ozone pollution.
4. My position requires me to be familiar with Sierra Club's purpose and mission, its activities related to ozone pollution (among other things), and the nature and scope of its membership. As part of my job, I have occasion to speak to members of the Club around the country in relation to my work on ozone pollution.
5. Sierra Club is a nonprofit, membership organization founded in 1892 to promote the conservation of natural resources, and the enjoyment and protection of the natural environment. For decades the Sierra Club has worked to promote a clean and healthy environment for its members, including preserving and improving air quality. Since the passage of the Clean Air Act, Sierra Club has worked to strengthen and fully implement our mission, including by engaging in public education, advocacy, and litigation for full and effective implementation of the Clean Air Act's protections. Among other things, Sierra Club has successfully opposed past attempts by EPA to weaken or waive Clean Air Act protections. *See, e.g., Sierra Club v. EPA*, 705 F.3d 458 (D.C. Cir. 2013) (vacating EPA rules establishing loopholes in air permitting requirements); *Sierra Club v. EPA*, 551 F.3d 1019, 1028 (D.C. Cir. 2008) (holding unlawful EPA-created exemption allowing

sources to exceed air toxics standards during periods of startup, shutdown, and malfunction); *Sierra Club v. EPA*, 129 F.3d 137, 143 (D.C. Cir. 1997) (holding unlawful EPA exemption for nonattainment areas from transportation conformity control requirement).

6. Consistent with its mission, Sierra Club works to ensure that National Ambient Air Quality Standards (“standards”) are fully adequate to protect public health and welfare, as the Clean Air Act requires. This work will be a priority for us for the foreseeable future. For example, along with other environmental and public health groups, we filed litigation that established the deadlines for EPA to complete legally mandated reviews that led to both the 2008 ozone standard and the new, 2015 ozone standard. *Sierra Club, et al. v. EPA*, No. 13-cv-2809 (N.D. Cal. filed June 19, 2013); *American Lung Association, et al. v. Whitman*, No. 03-cv-0778 (D.D.C. filed Mar. 31, 2003). We, along with others, have since filed a petition for judicial review of the 2015 ozone standard and intervened to defend against suits that seek to weaken it. *Sierra Club, et al. v. EPA*, No. 15-1490 (D.C. Cir. filed Dec. 23, 2015); Motion of Health and Environmental Organizations to Intervene on Behalf of Respondent, *Murray Energy Corp. v. EPA*, No. 15-1385 (D.C. Cir. Nov. 24, 2015). Further, sometimes also with others, Sierra Club regularly files suits to ensure that state and federal rules for effectuating compliance with ozone standards are put in place and meet the requirements of the Clean Air Act. *See, e.g., Miss. Comm’n on Env’tl. Quality v. EPA*, 790 F.3d 138 (D.C. Cir. 2015) (Sierra Club was petitioner in challenge to EPA area air quality designations under 2008 ozone standard); *South Coast Air Quality Mgmt. Dist. v. EPA*, 472 F.3d 882 (D.C. Cir. 2006) (Sierra Club was petitioner in challenge to EPA rules for implementing prior ozone standard); *Sierra Club v. McCarthy*, No. 14-cv-3198 (N.D. Cal. filed July 15, 2014) (deadline suit to compel EPA action on various state plans to implement 2008 ozone standard); *see also Sierra Club v. McCarthy*, No. 13-cv-3953 (N.D. Cal. filed Aug. 26, 2013) (deadline suit to compel EPA to make initial area designations under 2010 sulfur dioxide standard). At the state level, too, Sierra Club, along with others, files suits seeking to ensure that plans to comply with the standard are as strong as they need to be under the Clean Air Act. *E.g., Sierra Club, Md. Chapter, et al. v. Md. Dep’t of the Env’t*, No. CAL15-40282 (Md. Prince George’s County Cir. Ct. filed Dec. 29, 2015).
7. We also participate in administrative proceedings under which EPA establishes the standard, its implementing regulations, and initial area designations. Along with others, we filed extensive comments urging EPA to establish ozone standards that would comply with the Clean Air Act’s health- and welfare-based requirements by providing greater protection than the standards EPA ultimately set. *E.g., EPA-HQ-OAR-2008-0699-2720* (comments on what became 2015 ozone standard); *EPA-HQ-OAR-2005-0172-4261* (comments on what became 2008 ozone standard). We participated in the designations process for the 2008 ozone standard by filing comments. *E.g., EPA-HQ-OAR-2008-0476-0420*. We also participate at the administrative level in various states’ development of plans and programs

to control ozone pollution and in EPA rulemakings to approve or disapprove such plans and programs, as well as in state development of “exceptional event” petitions, through which a state seeks to exclude certain air quality monitoring data from use for regulatory purposes.

8. Along with other groups, we have repeatedly advocated for a more protective ozone standard than the ones adopted in 2008 (0.075 parts per million (“ppm”)) and in 2015 (0.070 ppm), 40 C.F.R. §§ 50.15(b), .19(b). As described above, we filed lengthy, detailed comments calling for a standard with level no higher than 0.060 ppm and have challenged the 2015 ozone standards in court as being insufficiently protective of human health and the environment to satisfy the Clean Air Act. In our comments, we described extensive evidence that ozone causes asthma attacks, hospitalizations and emergency room visits because of breathing problems, and death at levels below 0.070 ppm. EPA’s own science advisors—the Clean Air Scientific Advisory Committee (“CASAC”)—unanimously advised the agency that, at 0.070 ppm, “there is substantial scientific certainty of a variety of adverse effects.” CASAC Consensus Responses 8, June 26, 2014, EPA-HQ-OAR-2008-0699-0190. CASAC recommended EPA consider a range down to 0.060 ppm to protect public health. *Id.* Both EPA and CASAC strongly agreed that the 2008 standard of 0.075 ppm is not adequate to protect public health, and EPA in 2015 revised the health standard to 0.070 ppm. *See, e.g.*, 80 Fed. Reg. 65,292, 65,346 (Oct. 26, 2015).
9. As a result of my work, I am familiar with the Clean Air Act’s provisions and EPA rules for implementing the national ambient air quality standards for ozone, and with the history of implementation of ozone standards. My familiarity includes the requirements for EPA to promulgate initial area air quality designations, and the consequences that flow from designations. I am also familiar with the data available on EPA’s website and the calculations EPA prescribes to determine whether air quality monitoring data demonstrate compliance with the ozone standards. I regularly examine and analyze such data.
10. EPA requires states to establish and maintain networks of air quality monitors to determine the levels of ozone in the air by taking samples of air and analyzing it. *See* 40 C.F.R. pts.53, 58. States must report the resulting data to EPA. *Id.* § 58.15.
11. For ozone, at least one air quality monitoring “site for each [Metropolitan Statistical Area] MSA, or [Combined Statistical Area] CSA if multiple MSAs are involved, must be designed to record the maximum concentration for that particular metropolitan area.” *Id.* pt.58 app.D, § 4(b); *see also id.* § 2(e) (defining “MSA” and “CSA” as the relevant “statistical-based definitions for metropolitan areas provided by the Office of Management and Budget and the Census Bureau”). EPA uses this data to identify the areas of the country that violate the standards. Memorandum on Area Designations for the 2015 Ozone National Ambient Air Quality Standards, from Janet McCabe, Acting Ass’t Admin’r, to

Reg'l Admin'rs 3 (Feb. 25, 2016), <https://www.epa.gov/sites/production/files/2016-02/documents/ozone-designations-guidance-2015.pdf> (“Designations Memo”). EPA then “designates” such areas as “nonattainment” areas. Typically, EPA uses counties as the bases for identifying those violating areas. *See id.* at 7 (“EPA generally believes it is appropriate to include the entire violating...county in an ozone nonattainment area”).

12. EPA also designates as “nonattainment” those areas near communities with violating monitors when those areas contribute to the violation. 42 U.S.C. § 7407(d)(1)(A)(i); Designations Memo at 6-7. EPA has done so repeatedly for prior ozone standards, and based on that experience, provides detailed guidance on how it intends to proceed for these standards. Designations Memo at 5-7, attach.3. Where an area has been designated nonattainment under multiple prior ozone standards, the boundaries of the area often include the same counties (or portions of counties). *See* <https://www3.epa.gov/airquality/greenbook/hbca.html> (listing counties within current and former nonattainment areas under 2008 ozone standards); <https://www3.epa.gov/airquality/greenbook/gbca.html> (same, for 1997 ozone standards); <https://www3.epa.gov/airquality/greenbook/obca.html> (same, for 1979 ozone standards).
13. We used the air quality monitoring data available on EPA’s own website in June 2017 to calculate the “design values” that EPA uses to determine whether an area is in compliance with the 2015 standards. To do so, we collected data on ozone levels from <https://www.epa.gov/outdoor-air-quality-data/monitor-values-report> for all air quality monitors for the years 2014-2016.¹ EPA reports, in parts per million (ppm) the fourth-highest daily maximum 8-hour average ozone level for each monitor for each year. We took the 3-year average of the fourth-highest daily maximum 8-hour average ozone level for each monitor over that 3-year period and truncated the resulting figure to three decimal places. *See* 40 C.F.R. § 50.19(b), (d); *id.* pt.50 app.U, § 3(e). This is called the “design value.” *Id.* pt.50 app.U, § 1(c). If it is above 0.070 ppm, the monitor is in violation of the 2015 standard based on its 2016 design value. *Id.* § 50.19(b), (d). We followed EPA’s rules on “data completeness,” which governs whether a monitor has adequate amounts of data to calculate a “valid design value.” *Id.* pt.50 app.U, § 4(b)-(c).
14. The data were analyzed twice, once with data marked as affected by “exceptional events” excluded and once with such data included. Whether a particular design value exceeded 0.070 ppm changed only in a few instances, none material to the following discussion.

¹ We used 2016 as the final year of analysis because states must certify by May 1 that the air quality monitoring data they submitted to EPA for the preceding year is complete, accurate, and quality-assured. 40 C.F.R. § 58.15. Thus, 2016 is the most recent year for which EPA must have complete, accurate, and quality-assured air quality monitoring data.

15. Consistent with EPA's practice, we assigned as each county's design value the highest valid design value calculated for a monitoring site in the county. *See, e.g.*, https://www.epa.gov/sites/production/files/2016-07/ozone_designvalues_20132015_final_07_29_16.xlsx tbl.4 ("The design value listed for each county is the highest among sites with valid design values."). We refer to these design values as "preliminary 2016 design values," as EPA has yet to post its official calculations of such design values. As noted above, the air quality monitoring data for 2016 was due to EPA, certified complete, accurate, and quality-assured, by May 1, 2017. The complete list of monitors with valid design values and of county-level design values, with exceptional events excluded from the calculation (thus making them potentially lower than they would be with all data included), is attached to my declaration.
16. As noted in the declarations filed in this matter, Sierra Club and the other petitioners have over a million members throughout the United States.
17. The declarations show that petitioners' members live, work, and recreate in numerous communities and areas that would very likely or almost certainly be designated nonattainment under the 2015 ozone standard if EPA made designations by October 1, 2017. Per the analysis discussed above of EPA's air quality monitoring data, the most recent certified data available show that all these areas have, and most historically have had, design values that exceed the level of 0.070 ppm that EPA in 2015 found causes human health and welfare harms. These include Pinal County, AZ (0.071 ppm); Yuma County, AZ (0.074 ppm); Alameda County, CA (0.074 ppm); Amador County, CA (0.073 ppm); Los Angeles County, CA (0.096 ppm); Mariposa County, CA (0.075 ppm); Tuolumne County, CA (0.079 ppm); Hartford County, CT (0.074 ppm); DeKalb County, GA (0.071 ppm); Fulton County, GA (0.075 ppm); Gwinnett County, GA (0.072 ppm); Cook County, IL (0.072 ppm); Jefferson County, KY (0.074 ppm); East Baton Rouge Parish, LA (0.072 ppm); Baltimore County, MD (0.072 ppm); Macomb County, MI (0.072 ppm); Wayne County, MI (0.072 ppm); St. Louis County, MO (0.071 ppm); Clark County, NV (0.074 ppm); Franklin County, OH (0.071 ppm); Hamilton County, OH (0.072 ppm); Lake County, OH (0.075 ppm); Warren County, OH (0.072 ppm); Philadelphia County, PA (0.075 ppm); Bexar County, TX (0.073 ppm); El Paso County, TX (0.071 ppm); Davis County, UT (0.074 ppm); Salt Lake County, UT (0.075 ppm); Weber County, UT (0.072 ppm); Arlington County, VA (0.072 ppm); and Milwaukee County, WI (0.071 ppm). *Compare* Declarations, *with* attach.1, *and* https://www.epa.gov/sites/production/files/2016-07/ozone_designvalues_20132015_final_07_29_16.xlsx tbl.6 (giving 10-year trends for each monitor's design value through its 2013-15 design value). These are just some of the counties with preliminary 2016 design values violating the 2015 standards.
18. State recommendations for nonattainment area designations included nearly all of the above-listed areas, as well as others. For example, Arizona recommended that portions of

Pinal and Yuma Counties, as well as others, be designated nonattainment, based on 2015 design values. <https://www.epa.gov/sites/production/files/2016-11/documents/az-rec-enc-1.pdf> at 1. California recommended that Alameda, Amador, Los Angeles, Mariposa, and Tuolumne Counties, as well as others, be designated nonattainment, based on 2015 design values. <https://www.epa.gov/sites/production/files/2016-11/documents/ca-rec-enclosures.pdf> at 5-12 & fig.1, tbl.1. Connecticut recommended that all counties in Connecticut be designated nonattainment, based on 2015 design values. <https://www.epa.gov/sites/production/files/2016-11/documents/ct-rec-tds.pdf> at 1, 6. The District of Columbia recommended that it be designated nonattainment, based on preliminary 2016 design values. <https://www.epa.gov/sites/production/files/2016-11/documents/dc-rec.pdf> attach.A 2. Georgia recommended that DeKalb, Fulton, and Gwinnett Counties, as well as others, be designated nonattainment based on 2015 design values and preliminary 2016 design values. <https://www.epa.gov/sites/production/files/2016-11/documents/ga-rec.pdf> at 1. Illinois recommended that Cook County, as well as others, be designated nonattainment based on 2015 design values. <https://www.epa.gov/sites/production/files/2016-11/documents/il-rec.pdf> at 1-2. Louisiana recommended that East Baton Rouge Parish, as well as others, be designated nonattainment based on 2015 design values. <https://www.epa.gov/sites/production/files/2016-11/documents/la-rec.pdf> at 1, 3. Maryland recommended that Baltimore and Charles Counties and Baltimore City, as well as others, be designated nonattainment based on preliminary 2016 design values. See https://www.epa.gov/sites/production/files/2017-05/documents/md_recommendations.pdf at 1 (recommending that nonattainment areas remain the same as nonattainment areas under 2008 standards); <https://www3.epa.gov/airquality/greenbook/hbcs.html#MD> (listing counties designated nonattainment under 2008 standards). Michigan recommended that Macomb and Wayne Counties, as well as others, be designated nonattainment based on 2015 design values. <https://www.epa.gov/sites/production/files/2016-11/documents/mi-rec.pdf> at 1. Missouri recommended that St. Louis County, as well as others, be designated nonattainment based on 2015 design values. <https://www.epa.gov/sites/production/files/2016-11/documents/mo-rec.pdf> at 1, 8-9. Nevada recommended that portions of Clark County be designated nonattainment based on 2015 design values. <https://www.epa.gov/sites/production/files/2016-11/documents/nv-rec.pdf> attach.D 1-1, 2-3 & tbl.2-2. Ohio recommended that Franklin, Hamilton, Lake, Summit, and Warren Counties, as well as others, be designated nonattainment based on 2015 design values and preliminary 2016 design values. <https://www.epa.gov/sites/production/files/2016-11/documents/oh-rec.pdf> at 1-2. Pennsylvania recommended that Philadelphia County, as well as others, be designated nonattainment based on 2015 design values. <https://www.epa.gov/sites/production/files/2016-11/documents/pa-rec-app-a.pdf> at 1-5 tbl.1. Texas recommended that Bexar and El Paso Counties, as well as others, be designated nonattainment based on 2015 design values.

<https://www.epa.gov/sites/production/files/2016-11/documents/tx-rec.pdf> at A-1, B-1 to -2. Utah recommended that Davis and Salt Lake Counties and portions of Weber County, as well as others, be designated nonattainment based on 2015 design values and preliminary 2016 design values. <https://www.epa.gov/sites/production/files/2016-11/documents/ut-rec-tds.pdf> at 11-12 & tbls.1, 30-31, 34-35 & tbl.5, 54-57.

19. The declarations also show that petitioners' members live, work, and recreate in communities and areas that are currently designated attainment under the 2008 ozone standard, but have preliminary 2016 design values above 0.070 ppm. Examples of these areas currently designated attainment, but with preliminary 2016 design values above 0.070 ppm include Yuma County, AZ; Amador County, CA; Tuolumne County, CA; DeKalb County, GA; Fulton County, GA; Gwinnett County, GA; Jefferson County, KY; East Baton Rouge Parish, LA; Macomb County, MI; Wayne County, MI; Clark County, NV; Franklin County, OH; Hamilton County, OH; Lake County, OH; Warren County, OH; Bexar County, TX; El Paso County, TX; Davis County, UT; Salt Lake County, UT; Weber County, UT; and Milwaukee County, WI. *Compare* Declarations, *with* attach.1, *and* <https://www3.epa.gov/airquality/greenbook/hnca.html> (listing all counties currently designated nonattainment under 2008 standard) (last updated June 20, 2017). EPA takes the position that the only currently effective ozone standards are the 2008 and 2015 standards, and that the only current legally effective designations are those for the 2008 standards. *See* 80 Fed. Reg. at 65,437; 80 Fed. Reg. 12,264, 12,296-97 & n.75 (Mar. 6, 2015).
20. The declarations show petitioners' members' live, work, and recreate in communities and areas referred to in Paragraph 19 that are "maintenance" areas under the 2008 standards, meaning they formerly were designated nonattainment under the 2008 standards, but have since been redesignated attainment. The areas listed above in Paragraph 19 that fall into this category (and there are others) are DeKalb County, GA; Fulton County, GA; Gwinnett County, GA; East Baton Rouge Parish, LA; Franklin County, OH; Hamilton County, OH; Lake County, OH; and Warren County, OH. *Compare* Declarations, *with* attach.1, *and* <https://www3.epa.gov/airquality/greenbook/hmca.html> (listing all "maintenance" counties under 2008 standard) (last updated June 20, 2017).
21. Other communities and areas referred to in Paragraph 19 have never been designated nonattainment under the 2008 ozone standards. The areas listed above in Paragraph 19 that fall into this category (and there are others) are Yuma County, AZ; Amador County, CA; Tuolumne County, CA; Jefferson County, KY; Macomb County, MI; Wayne County, MI; Clark County, NV; Bexar County, TX; El Paso County, TX; Davis County, UT; Salt Lake County, UT; Weber County, UT; and Milwaukee County, WI. *Compare* Declarations, *with* attach.1, *and* <https://www3.epa.gov/airquality/greenbook/hbca.html> (listing all counties that have ever been designated nonattainment under 2008 standard) (last updated June 20, 2017).

22. Moreover, the declarations also show that petitioners' members live, work, and recreate in communities and areas that have preliminary 2016 design values above 0.070 ppm and that are currently designated nonattainment under the 2008 standards but that have received "Clean Data Determinations" from EPA under that standard. EPA issues these "Clean Data Determinations" when an area has attained a standard but has not satisfied the additional statutory criteria necessary to allow it to be redesignated to attainment, *see* 42 U.S.C. § 7407(d)(3)(E) (establishing five criteria an area must meet before being redesignated attainment, including a demonstration that the attainment results from permanent and enforceable emission reductions). The regulatory effect of such a Determination is that the area is exempt from undertaking various efforts to require pollution reductions. 40 C.F.R. § 51.1118. Accordingly, pollution control requirements in such areas are weakened. Examples of areas with Clean Data Determinations under the 2008 standard but preliminary 2016 design values above 0.070 ppm include Alameda and Mariposa Counties, CA; Baltimore County, MD; and St. Louis County, MO. *Compare* Declarations, *with* <https://www3.epa.gov/airquality/greenbook/hfr2rpt5.html> (listing all proposed and finalized Clean Data Determinations under 2008 standard) (last updated June 20, 2017), *and* <https://www3.epa.gov/airquality/greenbook/hnca.html> (listing all counties currently designated nonattainment under 2008 standard) (last updated June 20, 2017).
23. In addition, the declarations show petitioners' members live, work, and recreate in communities and areas that may not have preliminary 2016 design values violating the 2015 standards, but that EPA has historically considered nonattainment areas because of their contribution to historically violating monitors in nearby communities and areas, including nearby communities and areas that now have preliminary 2016 design values violating the 2015 standards. *See* 42 U.S.C. § 7407(d)(1)(A)(i). It is likely that such areas would be designated nonattainment under the 2015 standards as well. Many such areas have preliminary 2016 design values above the 0.060 ppm value that we and other scientific and medical groups urged EPA was requisite to protect public health with an adequate margin of safety. Such areas include Washington, DC (0.070 ppm), Charles County, MD (0.070 ppm), and Prince William County, VA (0.065 ppm) (part of area that includes Arlington County, VA (0.072 ppm)); Douglas County, GA (0.068 ppm) (part of area that includes Fulton County, GA (0.075 ppm)); Baltimore City, MD (0.066 ppm) (part of area that includes Baltimore County, MD (0.072 ppm)); New York County, NY (0.069 ppm) (part of area that includes Richmond County, NY (0.076 ppm)); and Cuyahoga County, OH (0.069 ppm), and Summit County, OH (0.061 ppm) (part of area that includes Lake County, OH (0.075 ppm)). *Compare* Declarations, *with* attach.1, *and* <https://www3.epa.gov/airquality/greenbook/hbca.html> (listing all counties that have ever been designated nonattainment under 2008 standard) (last updated June 20, 2017), *and* <https://www3.epa.gov/airquality/greenbook/gbca.html> (same, under 1997 ozone standard) (last updated June 20, 2017), *and* <https://www3.epa.gov/airquality/greenbook/obca.html> (same, under 1979 ozone standard) (last updated June 20, 2017).

24. EPA and CASAC's identification of harms associated with exposure to ground-level ozone indicates that petitioners' members who live in the areas discussed in Paragraphs 17-23—as well as in other areas with ozone levels at or above levels EPA itself and CASAC have identified as harmful to people and the environment—are justified in their concerns about the health and welfare threats posed to them and their families by ozone pollution and the threat of increased ozone pollution in their communities and the places where they work and enjoy outdoor recreation. They have a strong interest in the timely, full, and effective implementation of the Clean Air Act's programs designed to protect them and their families against harmful ozone levels.
25. Moreover, the pollution reductions needed to attain the 2015 ozone standards can bring other benefits. For example, for ozone nonattainment areas, the Act's nonattainment provisions require reductions in both volatile organic compounds (VOCs) and oxides of nitrogen. *See, e.g.*, 42 U.S.C. § 7511a(b)(1)(A)(i). As well as contributing to ozone formation, VOCs include hazardous air pollutants, like benzene, formaldehyde, or toluene. *See* 40 C.F.R. § 51.100(s) (defining VOC as “any compound of carbon, excluding [certain compounds], which participates in atmospheric photochemical reactions”); <https://pubchem.ncbi.nlm.nih.gov/compound/benzene#section=Use-and-Manufacturing> (last visited July 1, 2017) (describing benzene as a VOC); <https://www.epa.gov/indoor-air-quality-iaq/technical-overview-volatile-organic-compounds> (last updated Apr. 12, 2017) (giving formaldehyde and toluene as examples of VOCs); 42 U.S.C. § 7412(b)(1) (listing all three compounds as hazardous air pollutants). Thus, reducing their emissions can benefit human health beyond just any resulting reductions in ozone levels.
26. I am aware that EPA extended the deadline for making all initial area designations under the 2015 ozone standards by one year, announcing the extension by letters to state governors and by a Federal Register notice. This designation delay weakens human health protections and environmental protections in the communities listed in Paragraphs 17-23 and others. The Clean Air Act contains detailed provisions designed to bring designated nonattainment areas into attainment no later than fixed deadlines, but these provisions only apply for a pollutant in an area when that area is designated nonattainment for that pollutant. The delay of designations thus means those provisions do not apply. Accordingly, protections are delayed, forcing people—including petitioners' members—in the area to breathe in and suffer from the health threats of unsafe higher levels of ozone for longer. As the Declarations describe, petitioners' members include people who are at elevated risk of experiencing severe harms as a result of ozone pollution.
27. Delay is especially dangerous in areas that would virtually certainly be designated nonattainment for ozone under the 2015 standards and are currently designated attainment under the 2008 standards. Such areas include Fulton County, GA; Wayne County, MI; Bexar County, TX; and Salt Lake County, UT. In such areas, statutory new source review

permitting does not currently apply but will apply immediately upon designation as nonattainment. 40 C.F.R. pt.51, app.S §§ I, IV. Under new source review, proponents of “major” new or modified stationary sources (for example, power plants or factories) are subject to very stringent emissions controls and would have to secure “offsets” that would actually reduce emissions of ozone-forming pollution within the nonattainment area. 42 U.S.C. §§ 7503, 7511a(a)(4), (b)(5), (c), (c)(10), (d), (d)(2), (e), (e)(1), 7602(j). This new source review control is extremely important for controlling ozone pollution because it secures reductions in emissions of ozone-forming pollution, while also ensuring that the new source’s emissions are very well controlled. Currently, however, proponents of “major” new or modified stationary sources are subject to the Act’s “prevention of significant deterioration” permitting program, 42 U.S.C. §§ 7470-7479, which is less stringent. *New York v. EPA*, 413 F.3d 3, 13 (D.C. Cir. 2005). Thus, delaying designations delays the applicability of enhanced protections against emissions of ozone-forming pollutants in areas where petitioners’ members live, work, and recreate. As the Declarations describe, these areas host large industrial sources like refineries and cement plants. Without EPA promulgating area air quality designations, when such sources seek to expand, they will evade new source review and instead be subject to less protective requirements under the prevention of significant deterioration permitting program. Petitioners’ members in such areas, as well as those affected by the downwind emissions from such areas, are thus deprived of protection against harmful emissions every day that EPA does not promulgate area air quality designations.

28. In areas that are virtually certain to be designated nonattainment for ozone under the 2015 standards and that are not “maintenance” (*i.e.*, were not initially designated nonattainment and then secured redesignation as attainment) under the 2008 standards, the designations delay also pushes back application of the Act’s conformity requirement. Such areas include Wayne County, MI; Bexar County, TX; and Salt Lake County, UT. The conformity requirement applies in all areas that are nonattainment or maintenance under an ozone standard that remains in effect—the 2008 and 2015 standards, per EPA. 42 U.S.C. § 7506(c)(5); *see supra* Paragraph 19. The conformity requirement limits ozone pollution by authorizing transportation plans, programs, and projects, as well as other federally supported or licensed projects, to proceed only if their proponent shows, among other things, that they will not cause or contribute to a new violation or worsen an existing violation of any standard in any area. 42 U.S.C. § 7506(c)(1)-(2). New road and highway projects that go into regional transportation plans and programs typically make attainment of ozone standards harder because these projects lead to more driving and increased emissions of ozone-forming pollution. Similarly, the general conformity requirement helps protect against emissions of ozone-forming pollution that can cause or contribute to ozone pollution. By delaying designations under the 2015 standards, EPA delays the applicability of the conformity requirement in areas that will be designated nonattainment under the 2015 standards and that have never been designated nonattainment under the 2008

standard. It thus delays important protections against ozone pollution resulting from road and highway projects, as well as from other projects requiring federal approval. This harms petitioners' members in such areas.

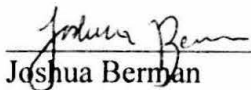
29. Even in areas that are currently designated nonattainment under the 2008 standards but violate the 2015 standards, EPA's designations delay reduces protections against ozone pollution. Currently, areas described in Paragraph 22, which have "Clean Data Determinations" under the 2008 standards, are exempt from developing additional plans to reduce ozone-forming pollution. Designation as nonattainment under the 2015 standards would again require them to develop pollution-reduction plans and make them face increases in stringency of controls should they fail to attain timely. Thus, the designations delay deprives petitioners' members in areas like Baltimore, MD, and Mariposa County, CA, of important health protections to which they are legally entitled.
30. Petitioners' members in areas that are still subject to all statutory protections and planning requirements under the 2008 standards, like Hartford, CT, also lose protections because of EPA's designations delay. They currently face no fixed attainment deadline for the 2015 standards, and the nonattainment planning the Act requires them to undertake focuses on attaining the 2008 standards, not the more protective 2015 standards. When EPA promulgates designations under the 2015 standards, they will be subject to attainment deadlines and planning requirements for the 2015 standards. These mandatory protections drive pollution reductions. *See, e.g.*, 42 U.S.C. §§ 7502(c)(1) (requiring state plans to provide for attainment of standards), 7511a (requiring specific anti-pollution measures in ozone nonattainment areas, including more stringent measures for areas that fail to timely achieve attainment of standards). Accordingly, the designations delay extends the period of time during which petitioners' members in these areas will go without vital human health protections, just as it does in every area that will be designated nonattainment under the 2015 standards.
31. The designations delay also likely postpones health protections for petitioners' members living, working, and enjoying recreation in the communities described in Paragraph 23, communities that, though their county-level preliminary 2016 design value may not violate the 2015 standards, are likely to be designated nonattainment as EPA has historically placed them in the same nonattainment area as other counties with preliminary 2016 design values violating the 2015 standards. Such communities would likely be designated nonattainment and thus be subject to all the protections any nonattainment area receives. These protections likely would lower pollution levels both of ozone and of VOCs, many of which are toxic. Moreover, many of the areas described in Paragraph 23 have preliminary 2016 design values above the 0.060 ppm level that formed the bottom end of the range CASAC recommended as requisite to protect public health and at which we called for EPA to set the health standard to protect public health. Area designations under the 2015 ozone

standards would thus likely drive pollution reductions in such areas and benefit the health and wellbeing of petitioners' members living, working, and recreating in them.

32. The weakening and removal of statutorily required protections described in Paragraphs 24-31 that results from EPA's designations delay does and will put the health of petitioners' members in areas like those discussed in Paragraphs 17-23 at risk and prolong their exposure to harmful levels of ozone pollution. Their welfare interests will also be injured because their enjoyment of activities and the natural world will further diminish due to their reasonable concerns about the harmful effects of the ozone pollution allowed by EPA's elimination and weakening of legally mandated protections.
33. Petitioners are also harmed because EPA deprived them of notice of the designations delay and an opportunity to comment on it. As discussed above and in the Declarations, Sierra Club, along with others, regularly participates in public comment processes on EPA actions that affect their members' health and wellbeing. Had EPA afforded them the opportunity on the designations delay, they would have explained that EPA had no lawful or rational basis for delay.
34. As the Declarations describe, bolstered by the scientific understanding of ozone pollution's effects on people and the environment, the harms petitioners' members face from ozone pollution are severe. Delays in EPA's actions that are supposed to secure pollution reductions will fail to reduce pollutions levels, and can even lead to increased pollution levels, which exacerbates and prolongs the harms petitioners' members already endure. Those harms and the EPA delay that causes them cannot be undone once they occur: delays in EPA's actions can only be mitigated, not fully remedied, once they happen, and no one can erase the fact that petitioners' members will have experienced serious health harms that may include asthma attacks and hospital visits.

I declare under penalty of perjury that the foregoing is true and correct.

Executed on July 6, 2017.



Joshua Berman

Attachment 1

State Name	County Name	CBSA Name	EPA Region	AQS Site ID	2014 Percent Complete	2015 Percent Complete	2016 Percent Complete	2014-2016 Average Percent Complete	2014 4th Highest Daily Max Value	2015 4th Highest Daily Max Value	2016 4th Highest Daily Max Value	Design Value 2014-2016	Meets NAAQS?
Alabama	Baldwin	Daphne-Fairhope-Foley, AL	4	010030010	96	91	95	94	0.070	0.063	0.062	0.065	Yes
Alabama	Colbert	Florence-Muscle Shoals, AL	4	010331002	100	97	97	98	0.059	0.057	0.061	0.059	Yes
Alabama	DeKalb		4	010499991	99	98	92	96	0.062	0.065	0.064	0.063	Yes
Alabama	Etowah	Gadsden, AL	4	010550011	99	97	98	98	0.059	0.060	0.064	0.061	Yes
Alabama	Houston	Dothan, AL	4	010690004	99	98	97	98	0.059	0.061	0.059	0.059	Yes
Alabama	Jefferson	Birmingham-Hoover, AL	4	010736002	98	93	98	96	0.063	0.073	0.070	0.068	Yes
Alabama	Madison	Huntsville, AL	4	010890014	100	98	99	99	0.064	0.063	0.066	0.064	Yes
Alabama	Mobile	Mobile, AL	4	010972005	97	97	98	97	0.070	0.063	0.063	0.065	Yes
Alabama	Montgomery	Montgomery, AL	4	011011002	98	98	100	99	0.062	0.063	0.063	0.062	Yes
Alabama	Morgan	Decatur, AL	4	011030011	98	99	99	99	0.062	0.063	0.067	0.064	Yes
Alabama	Russell	Columbus, GA-AL	4	011130002	98	99	94	97	0.058	0.062	0.066	0.062	Yes
Alabama	Shelby	Birmingham-Hoover, AL	4	011170004	99	96	99	98	0.063	0.065	0.073	0.067	Yes
Alabama	Sumter		4	011190003	97	89	93	93	0.058	0.057	0.058	0.057	Yes
Alabama	Tuscaloosa	Tuscaloosa, AL	4	011250010	97	97	100	98	0.057	0.062	0.061	0.060	Yes
Alaska	Denali		10	020680003	100	100	95	98	0.058	0.054	0.048	0.053	Yes
Alaska	Fairbanks North Star	Fairbanks, AK	10	020900034	99	98	99	99	0.044	0.045	0.039	0.042	Yes
Arizona	Cochise	Sierra Vista-Douglas, AZ	9	040038001	97	99	98	98	0.068	0.065	0.064	0.065	Yes
Arizona	Coconino	Flagstaff, AZ	9	040051008	99	99	99	99	0.073	0.070	0.064	0.069	Yes
Arizona	Gila	Payson, AZ	9	040070010	100	96	99	98	0.072	0.073	0.070	0.071	No
Arizona	La Paz		9	040128000	95	96	82	91	0.071	0.070	0.067	0.069	Yes
Arizona	Maricopa	Phoenix-Mesa-Scottsdale, AZ	9	040132005	98	98	100	99	0.080	0.077	0.074	0.077	No
Arizona	Navajo	Show Low, AZ	9	040170119	99	86	96	94	0.068	0.061	0.063	0.064	Yes
Arizona	Pima	Tucson, AZ	9	040190021	100	100	99	100	0.069	0.066	0.069	0.068	Yes
Arizona	Pinal	Phoenix-Mesa-Scottsdale, AZ	9	040218001	98	98	98	98	0.068	0.074	0.072	0.071	No
Arizona	Yavapai	Prescott, AZ	9	040258033	99	90	94	94	0.077	0.067	0.064	0.069	Yes
Arizona	Yuma	Yuma, AZ	9	040278011	98	92	100	97	0.078	0.077	0.067	0.074	No
Arkansas	Clark	Arkadelphia, AR	6	050199991	99	90	96	95	0.059	0.060	0.055	0.058	Yes
Arkansas	Crittenden	Memphis, TN-MS-AR	6	050350005	100	100	99	100	0.067	0.066	0.070	0.067	Yes
Arkansas	Newton	Harrison, AR	6	051010002	100	100	100	100	0.062	0.061	0.056	0.059	Yes
Arkansas	Polk		6	051130003	97	99	100	99	0.063	0.065	0.060	0.062	Yes
Arkansas	Pulaski	Little Rock-North Little Rock-Conway, AR	6	051191002	100	100	100	100	0.065	0.065	0.063	0.064	Yes
Arkansas	Washington	Fayetteville-Springdale-Rogers, AR-MO	6	051430006	99	100	100	100	0.062	0.061	0.058	0.060	Yes
California	Alameda	San Francisco-Oakland-Hayward, CA	9	060010007	99	99	98	99	0.076	0.074	0.072	0.074	No
California	Amador		9	060050002	98	100	100	99	0.074	0.074	0.071	0.073	No
California	Butte	Chico, CA	9	060070007	99	100	99	99	0.074	0.075	0.077	0.075	No
California	Calaveras		9	060090001	99	100	100	100	0.071	0.081	0.077	0.076	No
California	Colusa		9	060111002	99	94	97	97	0.061	0.064	0.065	0.063	Yes
California	Contra Costa	San Francisco-Oakland-Hayward, CA	9	060132007	98	99	98	98	0.072	0.074	0.062	0.069	Yes
California	El Dorado	Sacramento-Roseville-Arden-Arcade, CA	9	060170010	99	99	100	99	0.082	0.080	0.093	0.085	No
California	Fresno	Fresno, CA	9	060195001	89	90	93	91	0.097	0.093	0.092	0.094	No
California	Glenn		9	060210003	99	98	96	98	0.067	0.064	0.061	0.064	Yes
California	Imperial	El Centro, CA	9	060250005	91	99	97	96	0.078	0.077	0.074	0.076	No
California	Kern	Bakersfield, CA	9	060292012	82	87	97	89	0.087	0.097	0.088	0.090	No
California	Kings	Hanford-Corcoran, CA	9	060311004	79	92	95	89	0.086	0.085	0.083	0.084	No
California	Lake	Clearlake, CA	9	060333001	96	98	97	97	0.060	0.058	0.057	0.058	Yes
California	Los Angeles	Los Angeles-Long Beach-Anaheim, CA	9	060376012	96	95	98	96	0.097	0.091	0.100	0.096	No
California	Madera	Madera, CA	9	060392010	84	89	99	91	0.082	0.083	0.084	0.083	No
California	Marin	San Francisco-Oakland-Hayward, CA	9	060410001	99	98	99	99	0.064	0.063	0.056	0.061	Yes
California	Mariposa		9	060430006	83	96	85	88	0.077	0.071	0.077	0.075	No
California	Mendocino	Ukiah, CA	9	060450008	98	91	93	94	0.052	0.053	0.048	0.051	Yes

State Name	County Name	CBSA Name	EPA Regi on	AQS Site ID	2014 Percent Complete	2015 Percent Complete	2016 Percent Complete	2014-2016 Average Percent Complete	2014 4th Highest Daily Max Value	2015 4th Highest Daily Max Value	2016 4th Highest Daily Max Value	Design Value 2014-2016	Meets NAAQS?
California	Merced	Merced, CA	9	060470003	92	86	97	92	0.082	0.083	0.082	0.082	No
California	Monterey	Salinas, CA	9	060530008	99	99	100	99	0.062	0.062	0.058	0.060	Yes
California	Napa	Napa, CA	9	060550003	99	98	99	99	0.062	0.066	0.058	0.062	Yes
California	Nevada	Truckee-Grass Valley, CA	9	060570005	99	98	100	99	0.081	0.084	0.086	0.083	No
California	Orange	Los Angeles-Long Beach-Anaheim, CA	9	060592022	99	95	98	97	0.078	0.075	0.079	0.077	No
California	Placer	Sacramento--Roseville--Arden-Arcade, CA	9	060610006	99	95	96	97	0.083	0.073	0.084	0.080	No
California	Riverside	Riverside-San Bernardino-Ontario, CA	9	060651016	97	92	86	92	0.098	0.097	0.097	0.097	No
California	Sacramento	Sacramento--Roseville--Arden-Arcade, CA	9	060670012	98	95	97	97	0.081	0.081	0.088	0.083	No
California	San Benito	San Jose-Sunnyvale-Santa Clara, CA	9	060690003	99	92	99	97	0.069	0.066	0.072	0.069	Yes
California	San Bernardino	Riverside-San Bernardino-Ontario, CA	9	060710005	100	98	98	99	0.102	0.107	0.116	0.108	No
California	San Diego	San Diego-Carlsbad, CA	9	060731006	97	97	98	97	0.080	0.079	0.084	0.081	No
California	San Francisco	San Francisco-Oakland-Hayward, CA	9	060750005	99	99	99	99	0.052	0.050	0.046	0.049	Yes
California	San Joaquin	Stockton-Lodi, CA	9	060773005	95	96	98	96	0.080	0.077	0.080	0.079	No
California	San Luis Obispo	San Luis Obispo-Paso Robles-Arroyo Grande, CA	9	060798005	98	95	96	96	0.073	0.072	0.074	0.073	No
California	San Mateo	San Francisco-Oakland-Hayward, CA	9	060811001	98	98	99	98	0.064	0.059	0.056	0.059	Yes
California	Santa Barbara	Santa Maria-Santa Barbara, CA	9	060831025	99	99	97	98	0.070	0.067	0.064	0.067	Yes
California	Santa Clara	San Jose-Sunnyvale-Santa Clara, CA	9	060852006	100	100	97	99	0.073	0.071	0.068	0.070	Yes
California	Santa Cruz	Santa Cruz-Watsonville, CA	9	060870007	99	100	98	99	0.062	0.057	0.052	0.057	Yes
California	Shasta	Redding, CA	9	060890004	99	99	98	99	0.072	0.066	0.072	0.070	Yes
California	Siskiyou		9	060932001	99	99	98	99	0.061	0.061	0.055	0.059	Yes
California	Solano	Vallejo-Fairfield, CA	9	060953003	99	100	100	100	0.066	0.068	0.067	0.067	Yes
California	Sonoma	Santa Rosa, CA	9	060971003	99	97	93	96	0.062	0.059	0.055	0.058	Yes
California	Stanislaus	Modesto, CA	9	060990006	79	88	98	88	0.081	0.085	0.085	0.083	No
California	Sutter	Yuba City, CA	9	061010004	92	99	97	96	0.075	0.072	0.080	0.075	No
California	Tehama	Red Bluff, CA	9	061030004	99	99	97	98	0.076	0.076	0.087	0.079	No
California	Tulare	Visalia-Porterville, CA	9	061070009	96	96	97	96	0.089	0.088	0.092	0.089	No
California	Tuolumne	Sonora, CA	9	061090005	91	99	96	95	0.075	0.076	0.088	0.079	No
California	Ventura	Oxnard-Thousand Oaks-Ventura, CA	9	061112002	99	100	99	99	0.081	0.074	0.077	0.077	No
California	Yolo	Sacramento--Roseville--Arden-Arcade, CA	9	061131003	99	100	99	99	0.066	0.070	0.071	0.069	Yes
Colorado	Adams	Denver-Aurora-Lakewood, CO	8	080013001	97	96	97	97	0.067	0.069	0.066	0.067	Yes
Colorado	Arapahoe	Denver-Aurora-Lakewood, CO	8	080050006	100	98	99	99	0.067	0.068	0.066	0.067	Yes
Colorado	Denver	Denver-Aurora-Lakewood, CO	8	080310026	99	94	98	97	0.066	0.071	0.069	0.068	Yes
Colorado	Douglas	Denver-Aurora-Lakewood, CO	8	080350004	99	99	97	98	0.074	0.081	0.078	0.077	No
Colorado	El Paso	Colorado Springs, CO	8	080410013	100	100	99	100	0.064	0.067	0.069	0.066	Yes
Colorado	Garfield	Glenwood Springs, CO	8	080450012	93	91	89	91	0.061	0.068	0.060	0.063	Yes
Colorado	Gunnison		8	080519991	99	97	98	98	0.063	0.068	0.062	0.064	Yes
Colorado	Jefferson	Denver-Aurora-Lakewood, CO	8	080590011	99	98	100	99	0.076	0.081	0.083	0.080	No
Colorado	La Plata	Durango, CO	8	080677001	93	98	92	94	0.067	0.068	0.071	0.068	Yes
Colorado	Larimer	Fort Collins, CO	8	080690011	96	100	99	98	0.074	0.075	0.076	0.075	No
Colorado	Mesa	Grand Junction, CO	8	080770020	99	99	98	99	0.062	0.065	0.063	0.063	Yes
Colorado	Montezuma		8	080830101	99	98	100	99	0.065	0.066	0.066	0.065	Yes
Colorado	Rio Blanco		8	081030006	96	96	97	96	0.062	0.066	0.061	0.063	Yes
Colorado	Weld	Greeley, CO	8	081230009	99	97	97	98	0.070	0.073	0.067	0.070	Yes
Connecticut	Fairfield	Bridgeport-Stamford-Norwalk, CT	1	090019003	97	96	86	93	0.081	0.087	0.081	0.083	No
Connecticut	Hartford	Hartford-West Hartford-East Hartford, CT	1	090031003	99	99	96	98	0.077	0.075	0.072	0.074	No
Connecticut	Litchfield	Torrington, CT	1	090050005	97	96	97	97	0.068	0.076	0.074	0.072	No
Connecticut	Middlesex	Hartford-West Hartford-East Hartford, CT	1	090070007	98	99	95	97	0.080	0.078	0.080	0.079	No
Connecticut	New Haven	New Haven-Milford, CT	1	090099002	99	98	95	97	0.069	0.081	0.080	0.076	No
Connecticut	New London	Norwich-New London, CT	1	090110124	99	99	98	99	0.065	0.077	0.075	0.072	No
Connecticut	Tolland	Hartford-West Hartford-East Hartford, CT	1	090131001	97	99	96	97	0.077	0.072	0.072	0.073	No

State Name	County Name	CBSA Name	EPA Region	AQS Site ID	2014 Percent Complete	2015 Percent Complete	2016 Percent Complete	2014-2016 Average Percent Complete	2014 4th Highest Daily Max Value	2015 4th Highest Daily Max Value	2016 4th Highest Daily Max Value	Design Value 2014-2016	Meets NAAQS?
Connecticut	Windham	Worcester, MA-CT	1	090159991	99	97	99	98	0.067	0.070	0.067	0.068	Yes
Delaware	Kent	Dover, DE	3	100010002	100	100	99	100	0.066	0.066	0.068	0.066	Yes
Delaware	New Castle	Philadelphia-Camden-Wilmington, PA-NJ-DE-MD	3	100031010	74	91	99	88	0.074	0.071	0.078	0.074	No
Delaware	Sussex	Salisbury, MD-DE	3	100051003	98	100	93	97	0.067	0.070	0.070	0.069	Yes
District Of Columbia	District of Columbia	Washington-Arlington-Alexandria, DC-VA-MD-WV	3	110010043	99	99	97	98	0.068	0.072	0.072	0.070	Yes
Florida	Alachua	Gainesville, FL	4	120013011	96	97	96	96	0.060	0.056	0.060	0.058	Yes
Florida	Baker	Jacksonville, FL	4	120030002	99	99	99	99	0.059	0.058	0.061	0.059	Yes
Florida	Bay	Panama City, FL	4	120050006	99	99	99	99	0.065	0.061	0.062	0.062	Yes
Florida	Brevard	Palm Bay-Melbourne-Titusville, FL	4	120094001	85	100	100	95	0.063	0.059	0.061	0.061	Yes
Florida	Broward	Miami-Fort Lauderdale-West Palm Beach, FL	4	120118002	100	99	98	99	0.063	0.063	0.061	0.062	Yes
Florida	Collier	Naples-Immokalee-Marco Island, FL	4	120210004	99	100	99	99	0.058	0.057	0.058	0.057	Yes
Florida	Columbia	Lake City, FL	4	120230002	86	80	93	86	0.060	0.058	0.061	0.059	Yes
Florida	Escambia	Pensacola-Ferry Pass-Brent, FL	4	120330004	100	93	91	95	0.069	0.062	0.063	0.064	Yes
Florida	Flagler	Deltona-Daytona Beach-Ormond Beach, FL	4	120350004	99	100	99	99	0.063	0.058	0.059	0.060	Yes
Florida	Highlands	Sebring, FL	4	120550003	96	98	99	98	0.060	0.058	0.063	0.060	Yes
Florida	Hillsborough	Tampa-St. Petersburg-Clearwater, FL	4	120570081	98	99	98	98	0.071	0.068	0.066	0.068	Yes
Florida	Holmes		4	120590004	100	100	99	100	0.060	0.060	0.060	0.060	Yes
Florida	Indian River	Sebastian-Vero Beach, FL	4	120619991	98	88	91	92	0.064	0.058	0.062	0.061	Yes
Florida	Lake	Orlando-Kissimmee-Sanford, FL	4	120690002	100	100	87	96	0.067	0.059	0.064	0.063	Yes
Florida	Lee	Cape Coral-Fort Myers, FL	4	120712002	98	98	99	98	0.063	0.058	0.060	0.060	Yes
Florida	Leon	Tallahassee, FL	4	120730012	99	94	94	96	0.059	0.061	0.061	0.060	Yes
Florida	Liberty		4	120779991	96	95	96	96	0.057	0.056	0.056	0.056	Yes
Florida	Manatee	North Port-Sarasota-Bradenton, FL	4	120814013	95	88	96	93	0.064	0.060	0.059	0.061	Yes
Florida	Marion	Ocala, FL	4	120830003	94	100	100	98	0.064	0.058	0.062	0.061	Yes
Florida	Martin	Port St. Lucie, FL	4	120850007	97	100	98	98	0.061	0.060	0.062	0.061	Yes
Florida	Miami-Dade	Miami-Fort Lauderdale-West Palm Beach, FL	4	120860027	97	99	92	96	0.063	0.062	0.062	0.062	Yes
Florida	Okaloosa	Crestview-Fort Walton Beach-Destin, FL	4	120910002	100	100	100	100	0.067	0.059	0.060	0.062	Yes
Florida	Orange	Orlando-Kissimmee-Sanford, FL	4	120952002	97	100	100	99	0.063	0.059	0.066	0.062	Yes
Florida	Osceola	Orlando-Kissimmee-Sanford, FL	4	120972002	97	99	100	99	0.064	0.061	0.064	0.063	Yes
Florida	Pasco	Tampa-St. Petersburg-Clearwater, FL	4	121012001	98	99	95	97	0.064	0.060	0.063	0.062	Yes
Florida	Pinellas	Tampa-St. Petersburg-Clearwater, FL	4	121030004	97	100	97	98	0.063	0.059	0.063	0.061	Yes
Florida	Polk	Lakeland-Winter Haven, FL	4	121056005	96	99	99	98	0.063	0.062	0.064	0.063	Yes
Florida	Santa Rosa	Pensacola-Ferry Pass-Brent, FL	4	121130015	100	99	100	100	0.070	0.061	0.061	0.064	Yes
Florida	Sarasota	North Port-Sarasota-Bradenton, FL	4	121151005	98	95	98	97	0.064	0.062	0.062	0.062	Yes
Florida	Seminole	Orlando-Kissimmee-Sanford, FL	4	121171002	100	98	96	98	0.060	0.060	0.063	0.061	Yes
Florida	St. Lucie	Port St. Lucie, FL	4	121110013	98	93	99	97	0.062	0.059	0.063	0.061	Yes
Florida	Volusia	Deltona-Daytona Beach-Ormond Beach, FL	4	121275002	93	100	97	97	0.062	0.057	0.059	0.059	Yes
Georgia	Bibb	Macon, GA	4	130210012	95	100	98	98	0.065	0.062	0.070	0.065	Yes
Georgia	Chatham	Savannah, GA	4	130510021	98	98	90	95	0.057	0.058	0.058	0.057	Yes
Georgia	Chattooga	Summerville, GA	4	130550001	100	78	98	92	0.060	0.063	0.065	0.062	Yes
Georgia	Clarke	Athens-Clarke County, GA	4	130590002	97	87	100	95	0.063	0.061	0.069	0.064	Yes
Georgia	Columbia	Augusta-Richmond County, GA-SC	4	130730001	98	98	98	98	0.062	0.059	0.062	0.061	Yes
Georgia	Coweta	Atlanta-Sandy Springs-Roswell, GA	4	130770002	98	96	100	98	0.067	0.066	0.066	0.066	Yes
Georgia	Dawson	Atlanta-Sandy Springs-Roswell, GA	4	130850001	100	98	98	99	0.066	0.063	0.067	0.065	Yes
Georgia	DeKalb	Atlanta-Sandy Springs-Roswell, GA	4	130890002	99	98	95	97	0.070	0.071	0.074	0.071	No
Georgia	Douglas	Atlanta-Sandy Springs-Roswell, GA	4	130970004	96	95	99	97	0.065	0.070	0.071	0.068	Yes
Georgia	Fulton	Atlanta-Sandy Springs-Roswell, GA	4	131210055	97	100	98	98	0.073	0.077	0.075	0.075	No
Georgia	Glynn	Brunswick, GA	4	131270006	99	100	91	97	0.057	0.055	0.057	0.056	Yes
Georgia	Gwinnett	Atlanta-Sandy Springs-Roswell, GA	4	131350002	100	100	99	100	0.068	0.071	0.078	0.072	No
Georgia	Henry	Atlanta-Sandy Springs-Roswell, GA	4	131510002	99	98	99	99	0.075	0.070	0.078	0.074	No

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Georgia	Murray	Dalton, GA	4	132130003	91	97	98	95	0.067	0.063	0.067	0.065	Yes
Georgia	Muscogee	Columbus, GA-AL	4	132150008	100	89	97	95	0.061	0.062	0.065	0.062	Yes
Georgia	Paulding	Atlanta-Sandy Springs-Roswell, GA	4	132230003	97	98	93	96	0.059	0.065	0.067	0.063	Yes
Georgia	Pike	Atlanta-Sandy Springs-Roswell, GA	4	132319991	99	100	96	98	0.066	0.068	0.071	0.068	Yes
Georgia	Richmond	Augusta-Richmond County, GA-SC	4	132450091	97	98	98	98	0.061	0.060	0.065	0.062	Yes
Georgia	Rockdale	Atlanta-Sandy Springs-Roswell, GA	4	132470001	99	99	97	98	0.079	0.068	0.076	0.074	No
Georgia	Sumter	Americus, GA	4	132611001	99	100	99	99	0.059	0.057	0.065	0.060	Yes
Hawaii	Honolulu	Urban Honolulu, HI	9	150031004	92	95	92	93	0.054	0.048	0.047	0.049	Yes
Idaho	Ada	Boise City, ID	10	160010017	78	98	99	92	0.065	0.064	0.072	0.067	Yes
Idaho	Butte	Idaho Falls, ID	10	160230101	97	96	96	96	0.062	0.061	0.058	0.060	Yes
Illinois	Adams	Quincy, IL-MO	5	170010007	98	100	100	99	0.061	0.064	0.061	0.062	Yes
Illinois	Champaign	Champaign-Urbana, IL	5	170191001	94	94	95	94	0.068	0.065	0.066	0.066	Yes
Illinois	Clark	Chicago-Naperville-Elgin, IL-IN-WI	5	170230001	98	97	95	97	0.063	0.064	0.066	0.064	Yes
Illinois	Cook	Chicago-Naperville-Elgin, IL-IN-WI	5	170317002	95	96	96	96	0.072	0.070	0.076	0.072	No
Illinois	DuPage	Chicago-Naperville-Elgin, IL-IN-WI	5	170436001	91	98	86	92	0.064	0.067	0.074	0.068	Yes
Illinois	Effingham	Effingham, IL	5	170491001	98	94	100	97	0.063	0.064	0.066	0.064	Yes
Illinois	Hamilton	St. Louis, MO-IL	5	170650002	100	99	86	95	0.063	0.064	0.068	0.065	Yes
Illinois	Jersey	St. Louis, MO-IL	5	170831001	100	86	93	93	0.065	0.067	0.074	0.068	Yes
Illinois	Jo Daviess	Chicago-Naperville-Elgin, IL-IN-WI	5	170859991	98	86	94	93	0.067	0.062	0.067	0.065	Yes
Illinois	Kane	Chicago-Naperville-Elgin, IL-IN-WI	5	170890005	100	99	95	98	0.066	0.065	0.074	0.068	Yes
Illinois	Lake	Chicago-Naperville-Elgin, IL-IN-WI	5	170971007	99	99	100	99	0.073	0.070	0.077	0.073	No
Illinois	Macon	Decatur, IL	5	171150013	100	100	100	100	0.067	0.066	0.066	0.066	Yes
Illinois	Macoupin	St. Louis, MO-IL	5	171170002	100	99	94	98	0.063	0.064	0.067	0.064	Yes
Illinois	Madison	St. Louis, MO-IL	5	171193007	98	99	88	95	0.070	0.069	0.075	0.071	No
Illinois	McHenry	Chicago-Naperville-Elgin, IL-IN-WI	5	1711110001	100	94	98	97	0.067	0.064	0.073	0.068	Yes
Illinois	McLean	Bloomington, IL	5	171132003	99	100	100	100	0.066	0.063	0.065	0.064	Yes
Illinois	Peoria	Peoria, IL	5	171430024	99	97	98	98	0.064	0.060	0.068	0.064	Yes
Illinois	Randolph	Chicago-Naperville-Elgin, IL-IN-WI	5	171570001	98	98	94	97	0.071	0.065	0.066	0.067	Yes
Illinois	Rock Island	Davenport-Moline-Rock Island, IA-IL	5	171613002	95	100	92	96	0.062	0.060	0.064	0.062	Yes
Illinois	Saint Clair	St. Louis, MO-IL	5	171630010	98	100	96	98	0.067	0.066	0.073	0.068	Yes
Illinois	Sangamon	Springfield, IL	5	171670014	96	100	98	98	0.059	0.064	0.068	0.063	Yes
Illinois	Will	Chicago-Naperville-Elgin, IL-IN-WI	5	171971011	89	98	100	96	0.064	0.064	0.064	0.064	Yes
Illinois	Winnebago	Rockford, IL	5	172012001	90	98	98	95	0.070	0.066	0.070	0.068	Yes
Indiana	Allen	Fort Wayne, IN	5	180030004	99	96	92	96	0.063	0.059	0.069	0.063	Yes
Indiana	Bartholomew	Columbus, IN	5	180050007	99	99	96	98	0.067	0.066	0.071	0.068	Yes
Indiana	Boone	Indianapolis-Carmel-Anderson, IN	5	180110001	99	95	98	97	0.066	0.064	0.070	0.066	Yes
Indiana	Carroll	Lafayette-West Lafayette, IN	5	180150002	93	98	98	96	0.064	0.064	0.066	0.064	Yes
Indiana	Clark	Louisville/Jefferson County, KY-IN	5	180190008	93	96	98	96	0.066	0.074	0.072	0.070	Yes
Indiana	Delaware	Muncie, IN	5	180350010	99	98	86	94	0.054	0.058	0.067	0.059	Yes
Indiana	Elkhart	Elkhart-Goshen, IN	5	180390007	98	99	99	99	0.055	0.058	0.072	0.061	Yes
Indiana	Floyd	Louisville/Jefferson County, KY-IN	5	180431004	98	84	96	93	0.068	0.067	0.073	0.069	Yes
Indiana	Greene	Indianapolis-Carmel-Anderson, IN	5	180550001	99	99	99	99	0.064	0.067	0.069	0.066	Yes
Indiana	Hamilton	Indianapolis-Carmel-Anderson, IN	5	180570006	99	99	99	99	0.058	0.063	0.069	0.063	Yes
Indiana	Hendricks	Indianapolis-Carmel-Anderson, IN	5	180630004	99	99	99	99	0.057	0.055	0.068	0.060	Yes
Indiana	Huntington	Huntington, IN	5	180690002	95	97	98	97	0.056	0.054	0.066	0.058	Yes
Indiana	Jackson	Seymour, IN	5	180710001	97	90	99	95	0.064	0.064	0.070	0.066	Yes
Indiana	Johnson	Indianapolis-Carmel-Anderson, IN	5	180810002	95	97	99	97	0.056	0.063	0.062	0.060	Yes
Indiana	Knox	Vincennes, IN	5	180839991	95	97	99	97	0.062	0.063	0.071	0.065	Yes
Indiana	Lake	Chicago-Naperville-Elgin, IL-IN-WI	5	180890022	96	93	97	95	0.067	0.064	0.070	0.067	Yes
Indiana	LaPorte	Michigan City-La Porte, IN	5	180910010	98	99	99	99	0.061	0.061	0.068	0.063	Yes

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Indiana	Madison	Indianapolis-Carmel-Anderson, IN	5	180950010	93	98	99	97	0.054	0.054	0.064	0.057	Yes
Indiana	Marion	Indianapolis-Carmel-Anderson, IN	5	180970050	99	98	99	99	0.066	0.069	0.073	0.069	Yes
Indiana	Morgan	Indianapolis-Carmel-Anderson, IN	5	181090005	98	97	99	98	0.064	0.063	0.065	0.064	Yes
Indiana	Perry		5	181230009	94	99	99	97	0.067	0.067	0.069	0.067	Yes
Indiana	Porter	Chicago-Naperville-Elgin, IL-IN-WI	5	181270024	95	99	99	98	0.071	0.066	0.070	0.069	Yes
Indiana	Posey	Evansville, IN-KY	5	181290003	98	99	98	98	0.065	0.067	0.068	0.066	Yes
Indiana	Shelby	Indianapolis-Carmel-Anderson, IN	5	181450001	99	99	99	99	0.062	0.059	0.065	0.062	Yes
Indiana	St. Joseph	South Bend-Mishawaka, IN-MI	5	181410015	99	99	90	96	0.067	0.066	0.072	0.068	Yes
Indiana	Vanderburgh	Evansville, IN-KY	5	181630021	98	99	98	98	0.068	0.072	0.072	0.070	Yes
Indiana	Vigo	Terre Haute, IN	5	181670018	98	99	99	99	0.062	0.064	0.069	0.065	Yes
Indiana	Wabash	Wabash, IN	5	181699991	90	96	92	93	0.066	0.068	0.071	0.068	Yes
Indiana	Warrick	Evansville, IN-KY	5	181730008	98	99	99	99	0.065	0.069	0.070	0.068	Yes
Iowa	Bremer	Waterloo-Cedar Falls, IA	7	190170011	99	100	99	99	0.061	0.059	0.062	0.060	Yes
Iowa	Clinton	Clinton, IA	7	190450021	99	99	99	99	0.065	0.061	0.063	0.063	Yes
Iowa	Harrison	Omaha-Council Bluffs, NE-IA	7	190850007	97	98	96	97	0.063	0.061	0.063	0.062	Yes
Iowa	Linn	Cedar Rapids, IA	7	191130028	100	95	93	96	0.059	0.063	0.063	0.061	Yes
Iowa	Montgomery		7	191370002	99	95	99	98	0.059	0.060	0.062	0.060	Yes
Iowa	Palo Alto		7	191471002	95	98	93	95	0.063	0.064	0.058	0.061	Yes
Iowa	Polk	Des Moines-West Des Moines, IA	7	191530030	100	98	99	99	0.061	0.058	0.061	0.060	Yes
Iowa	Scott	Davenport-Moline-Rock Island, IA-IL	7	191630014	99	99	99	99	0.064	0.063	0.064	0.063	Yes
Iowa	Story	Ames, IA	7	191690011	100	99	98	99	0.062	0.059	0.060	0.060	Yes
Iowa	Van Buren		7	191770006	90	99	97	95	0.063	0.057	0.061	0.060	Yes
Iowa	Warren	Des Moines-West Des Moines, IA	7	191810022	99	100	99	99	0.060	0.059	0.057	0.058	Yes
Kansas	Johnson	Kansas City, MO-KS	7	200910010	100	100	100	100	0.062	0.062	0.058	0.060	Yes
Kansas	Leavenworth	Kansas City, MO-KS	7	201030003	99	100	98	99	0.068	0.060	0.062	0.063	Yes
Kansas	Neosho		7	201330003	98	99	99	99	0.062	0.061	0.061	0.061	Yes
Kansas	Sedgwick	Wichita, KS	7	201730010	99	100	100	100	0.069	0.064	0.062	0.065	Yes
Kansas	Shawnee	Topeka, KS	7	201770013	100	99	99	99	0.064	0.062	0.063	0.063	Yes
Kansas	Sumner	Wichita, KS	7	201910002	100	100	97	99	0.067	0.065	0.062	0.064	Yes
Kansas	Trego		7	201950001	96	95	98	96	0.068	0.063	0.058	0.063	Yes
Kansas	Wyandotte	Kansas City, MO-KS	7	202090021	95	98	100	98	0.063	0.063	0.064	0.063	Yes
Kentucky	Bell	Middlesborough, KY	4	210130002	96	99	99	98	0.062	0.059	0.062	0.061	Yes
Kentucky	Boone	Cincinnati, OH-KY-IN	4	210150003	100	99	98	99	0.062	0.062	0.065	0.063	Yes
Kentucky	Boyd	Huntington-Ashland, WV-KY-OH	4	210190017	100	98	98	99	0.065	0.069	0.066	0.066	Yes
Kentucky	Bullitt	Louisville/Jefferson County, KY-IN	4	210290006	90	98	96	95	0.065	0.067	0.067	0.066	Yes
Kentucky	Campbell	Cincinnati, OH-KY-IN	4	210373002	98	99	99	99	0.071	0.071	0.069	0.070	Yes
Kentucky	Carter		4	210430500	98	96	99	98	0.060	0.061	0.065	0.062	Yes
Kentucky	Christian	Clarksville, TN-KY	4	210470006	99	95	98	97	0.065	0.062	0.061	0.062	Yes
Kentucky	Daviess	Owensboro, KY	4	210590005	97	98	96	97	0.064	0.065	0.068	0.065	Yes
Kentucky	Edmonson	Bowling Green, KY	4	210610501	98	97	84	93	0.065	0.063	0.065	0.064	Yes
Kentucky	Fayette	Lexington-Fayette, KY	4	210670012	98	95	98	97	0.065	0.071	0.065	0.067	Yes
Kentucky	Greenup	Huntington-Ashland, WV-KY-OH	4	210890007	99	98	100	99	0.061	0.066	0.063	0.063	Yes
Kentucky	Hancock	Owensboro, KY	4	210910012	96	99	97	97	0.066	0.071	0.067	0.068	Yes
Kentucky	Hardin	Elizabethtown-Fort Knox, KY	4	210930006	90	99	98	96	0.062	0.066	0.068	0.065	Yes
Kentucky	Henderson	Evansville, IN-KY	4	211010014	98	98	98	98	0.069	0.067	0.072	0.069	Yes
Kentucky	Jefferson	Louisville/Jefferson County, KY-IN	4	211110067	99	100	99	99	0.070	0.076	0.076	0.074	No
Kentucky	Jessamine	Lexington-Fayette, KY	4	211130001	100	99	99	99	0.064	0.066	0.066	0.065	Yes
Kentucky	Livingston	Paducah, KY-IL	4	211390003	99	99	98	99	0.065	0.063	0.068	0.065	Yes
Kentucky	McCracken	Paducah, KY-IL	4	211451024	99	99	99	99	0.065	0.063	0.063	0.063	Yes
Kentucky	Morgan		4	211759991	90	98	100	96	0.064	0.064	0.066	0.064	Yes

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Kentucky	Oldham	Louisville/Jefferson County, KY-IN	4	211850004	100	77	97	91	0.068	0.073	0.069	0.070	Yes
Kentucky	Perry		4	211930003	99	98	93	97	0.061	0.057	0.058	0.058	Yes
Kentucky	Pike		4	211950002	100	98	99	99	0.063	0.058	0.061	0.060	Yes
Kentucky	Pulaski	Somerset, KY	4	211990003	100	98	95	98	0.063	0.062	0.063	0.062	Yes
Kentucky	Simpson		4	212130004	100	84	99	94	0.063	0.067	0.063	0.064	Yes
Kentucky	Trigg	Clarksville, TN-KY	4	212219991	93	96	97	95	0.066	0.060	0.063	0.063	Yes
Kentucky	Warren	Bowling Green, KY	4	212270009	98	86	99	94	0.063	0.061	0.062	0.062	Yes
Kentucky	Washington		4	212299991	99	98	95	97	0.065	0.064	0.065	0.064	Yes
Louisiana	Ascension	Baton Rouge, LA	6	220050004	97	99	98	98	0.069	0.074	0.071	0.071	No
Louisiana	Bossier	Shreveport-Bossier City, LA	6	220150008	98	98	96	97	0.066	0.065	0.065	0.065	Yes
Louisiana	Caddo	Shreveport-Bossier City, LA	6	220170001	98	99	98	98	0.065	0.066	0.062	0.064	Yes
Louisiana	Calcasieu	Lake Charles, LA	6	220190002	96	99	99	98	0.067	0.072	0.065	0.068	Yes
Louisiana	East Baton Rouge	Baton Rouge, LA	6	220330003	97	100	98	98	0.075	0.073	0.068	0.072	No
Louisiana	Iberville	Baton Rouge, LA	6	220470009	98	98	99	98	0.061	0.069	0.064	0.064	Yes
Louisiana	Jefferson	New Orleans-Metairie, LA	6	220511001	98	99	99	99	0.071	0.068	0.067	0.068	Yes
Louisiana	Lafayette	Lafayette, LA	6	220550007	100	99	99	99	0.068	0.067	0.063	0.066	Yes
Louisiana	Lafourche	Houma-Thibodaux, LA	6	220570004	98	96	98	97	0.067	0.064	0.065	0.065	Yes
Louisiana	Livingston	Baton Rouge, LA	6	220630002	99	98	94	97	0.073	0.070	0.067	0.070	Yes
Louisiana	Ouachita	Monroe, LA	6	220730004	94	92	87	91	0.059	0.060	0.060	0.059	Yes
Louisiana	Pointe Coupee	Baton Rouge, LA	6	220770001	97	100	99	99	0.071	0.069	0.065	0.068	Yes
Louisiana	St. Bernard	New Orleans-Metairie, LA	6	220870004	100	100	92	97	0.071	0.063	0.065	0.066	Yes
Louisiana	St. James	New Orleans-Metairie, LA	6	220930002	97	98	99	98	0.067	0.066	0.063	0.065	Yes
Louisiana	St. John the Baptist	New Orleans-Metairie, LA	6	220950002	96	100	99	98	0.068	0.067	0.064	0.066	Yes
Louisiana	St. Tammany	New Orleans-Metairie, LA	6	221030002	99	99	99	99	0.074	0.067	0.064	0.068	Yes
Louisiana	West Baton Rouge	Baton Rouge, LA	6	221210001	100	99	99	99	0.066	0.066	0.066	0.066	Yes
Maine	Androscoggin	Lewiston-Auburn, ME	1	230010014	99	82	99	93	0.065	0.058	0.057	0.060	Yes
Maine	Aroostook		1	230039991	96	99	98	98	0.051	0.055	0.052	0.052	Yes
Maine	Cumberland	Portland-South Portland, ME	1	230052003	100	99	100	100	0.066	0.064	0.065	0.065	Yes
Maine	Hancock		1	230090102	97	86	99	94	0.065	0.069	0.066	0.066	Yes
Maine	Kennebec	Augusta-Waterville, ME	1	230112005	98	99	95	97	0.057	0.063	0.059	0.059	Yes
Maine	Knox		1	230130004	99	94	97	97	0.061	0.067	0.063	0.063	Yes
Maine	Penobscot	Bangor, ME	1	230194008	99	100	99	99	0.054	0.063	0.057	0.058	Yes
Maine	Washington		1	230290019	89	98	99	95	0.054	0.062	0.057	0.057	Yes
Maine	York	Portland-South Portland, ME	1	230310040	97	99	99	98	0.061	0.062	0.061	0.061	Yes
Maryland	Baltimore	Baltimore-Columbia-Towson, MD	3	240051007	91	92	95	93	0.067	0.078	0.073	0.072	No
Maryland	Baltimore (City)	Baltimore-Columbia-Towson, MD	3	245100054	95	77	98	90	0.060	0.072	0.067	0.066	Yes
Maryland	Calvert	Washington-Arlington-Alexandria, DC-VA-MD-WV	3	240090011	99	95	99	98	0.070	0.067	0.068	0.068	Yes
Maryland	Carroll	Baltimore-Columbia-Towson, MD	3	240130001	100	98	99	99	0.064	0.070	0.066	0.066	Yes
Maryland	Cecil	Philadelphia-Camden-Wilmington, PA-NJ-DE-MD	3	240150003	95	89	98	94	0.074	0.074	0.075	0.074	No
Maryland	Charles	Washington-Arlington-Alexandria, DC-VA-MD-WV	3	240170010	96	98	99	98	0.070	0.068	0.073	0.070	Yes
Maryland	Dorchester	Cambridge, MD	3	240199991	95	100	90	95	0.065	0.065	0.068	0.066	Yes
Maryland	Frederick	Washington-Arlington-Alexandria, DC-VA-MD-WV	3	240210037	96	96	98	97	0.063	0.070	0.066	0.066	Yes
Maryland	Garrett		3	240230002	98	95	91	95	0.063	0.067	0.066	0.065	Yes
Maryland	Harford	Baltimore-Columbia-Towson, MD	3	240251001	100	99	98	99	0.067	0.074	0.077	0.072	No
Maryland	Kent		3	240290002	99	92	96	96	0.068	0.072	0.069	0.069	Yes
Maryland	Montgomery	Washington-Arlington-Alexandria, DC-VA-MD-WV	3	240313001	98	99	99	99	0.064	0.072	0.065	0.067	Yes
Maryland	Prince George's	Washington-Arlington-Alexandria, DC-VA-MD-WV	3	240338003	97	98	100	98	0.069	0.069	0.073	0.070	Yes
Maryland	Washington	Hagerstown-Martinsburg, MD-WV	3	240430009	97	98	98	98	0.061	0.067	0.068	0.065	Yes
Massachusetts	Bristol	Providence-Warwick, RI-MA	1	250051004	98	99	100	99	0.060	0.070	0.076	0.068	Yes
Massachusetts	Essex	Boston-Cambridge-Newton, MA-NH	1	250092006	99	98	97	98	0.063	0.065	0.067	0.065	Yes

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Massachusetts	Franklin	Greenfield Town, MA	1	250112005	98	97	99	98	0.058	0.064	0.068	0.063	Yes
Massachusetts	Hampden	Springfield, MA	1	250130008	94	100	96	97	0.065	0.070	0.076	0.070	Yes
Massachusetts	Hampshire	Springfield, MA	1	250154002	99	96	94	96	0.068	0.071	0.072	0.070	Yes
Massachusetts	Middlesex	Boston-Cambridge-Newton, MA-NH	1	250170009	98	99	97	98	0.064	0.061	0.066	0.063	Yes
Massachusetts	Norfolk	Boston-Cambridge-Newton, MA-NH	1	250213003	94	94	98	95	0.065	0.067	0.070	0.067	Yes
Massachusetts	Plymouth	Boston-Cambridge-Newton, MA-NH	1	250230005	99	96	98	98	0.060	0.065	0.067	0.064	Yes
Massachusetts	Suffolk	Boston-Cambridge-Newton, MA-NH	1	250250042	100	99	99	99	0.054	0.056	0.058	0.056	Yes
Massachusetts	Worcester	Worcester, MA-CT	1	250270015	96	99	99	98	0.065	0.063	0.066	0.064	Yes
Michigan	Allegan	Holland, MI	5	260050003	97	99	97	98	0.077	0.072	0.076	0.075	No
Michigan	Benzie	Traverse City, MI	5	260190003	95	99	92	95	0.069	0.067	0.072	0.069	Yes
Michigan	Berrien	Niles-Benton Harbor, MI	5	260210014	99	99	98	99	0.073	0.072	0.078	0.074	No
Michigan	Cass	South Bend-Mishawaka, IN-MI	5	260270003	89	98	99	95	0.066	0.068	0.077	0.070	Yes
Michigan	Chippewa	Sault Ste. Marie, MI	5	260330901	93	92	92	92	0.056	0.059	0.062	0.059	Yes
Michigan	Clinton	Lansing-East Lansing, MI	5	260370001	98	99	98	98	0.066	0.064	0.073	0.067	Yes
Michigan	Genesee	Flint, MI	5	260492001	99	98	99	99	0.068	0.067	0.073	0.069	Yes
Michigan	Huron		5	260630007	90	99	99	96	0.066	0.067	0.072	0.068	Yes
Michigan	Ingham	Lansing-East Lansing, MI	5	260650012	99	99	93	97	0.065	0.064	0.073	0.067	Yes
Michigan	Kalamazoo	Kalamazoo-Portage, MI	5	260770008	99	96	98	98	0.067	0.067	0.074	0.069	Yes
Michigan	Kent	Grand Rapids-Wyoming, MI	5	260810020	97	100	100	99	0.066	0.067	0.075	0.069	Yes
Michigan	Lenawee	Adrian, MI	5	260910007	93	99	98	97	0.068	0.065	0.069	0.067	Yes
Michigan	Macomb	Detroit-Warren-Dearborn, MI	5	260990009	98	99	99	99	0.071	0.072	0.075	0.072	No
Michigan	Manistee		5	261010922	97	99	98	98	0.068	0.067	0.070	0.068	Yes
Michigan	Mason	Ludington, MI	5	261050007	99	99	98	99	0.070	0.066	0.074	0.070	Yes
Michigan	Missaukee	Cadillac, MI	5	261130001	100	98	99	99	0.063	0.064	0.074	0.067	Yes
Michigan	Muskegon	Muskegon, MI	5	261210039	99	96	97	97	0.075	0.074	0.076	0.075	No
Michigan	Oakland	Detroit-Warren-Dearborn, MI	5	261250001	97	94	99	97	0.067	0.066	0.075	0.069	Yes
Michigan	Ottawa	Grand Rapids-Wyoming, MI	5	261390005	96	87	89	91	0.071	0.065	0.074	0.070	Yes
Michigan	Schoolcraft		5	261530001	98	99	99	99	0.067	0.070	0.075	0.070	Yes
Michigan	St. Clair	Detroit-Warren-Dearborn, MI	5	261470005	99	98	99	99	0.071	0.075	0.073	0.073	No
Michigan	Tuscola		5	261579991	97	95	98	97	0.063	0.064	0.071	0.066	Yes
Michigan	Washtenaw	Ann Arbor, MI	5	261619991	89	92	92	91	0.067	0.064	0.074	0.068	Yes
Michigan	Wayne	Detroit-Warren-Dearborn, MI	5	261630019	96	97	95	96	0.073	0.070	0.074	0.072	No
Michigan	Wexford	Cadillac, MI	5	261659991	97	99	95	97	0.066	0.064	0.071	0.067	Yes
Minnesota	Anoka	Minneapolis-St. Paul-Bloomington, MN-WI	5	270031002	100	98	99	99	0.064	0.064	0.061	0.063	Yes
Minnesota	Becker		5	270052013	100	99	99	99	0.061	0.062	0.059	0.060	Yes
Minnesota	Crow Wing	Brainerd, MN	5	270353204	100	99	98	99	0.057	0.060	0.060	0.059	Yes
Minnesota	Goodhue	Red Wing, MN	5	270495302	100	99	100	100	0.063	0.060	0.061	0.061	Yes
Minnesota	Hennepin	Minneapolis-St. Paul-Bloomington, MN-WI	5	270530962	99	99	100	99	0.060	0.053	0.056	0.056	Yes
Minnesota	Lake		5	270750005	97	97	94	96	0.056	0.056	0.058	0.056	Yes
Minnesota	Lyon	Marshall, MN	5	270834210	100	98	100	99	0.062	0.067	0.057	0.062	Yes
Minnesota	Mille Lacs	Minneapolis-St. Paul-Bloomington, MN-WI	5	270953051	100	100	97	99	0.060	0.059	0.061	0.060	Yes
Minnesota	Olmsted	Rochester, MN	5	271095008	100	100	98	99	0.062	0.059	0.062	0.061	Yes
Minnesota	Saint Louis	Duluth, MN-WI	5	271370034	100	99	79	93	0.057	0.056	0.052	0.055	Yes
Minnesota	Scott	Minneapolis-St. Paul-Bloomington, MN-WI	5	271390505	99	98	99	99	0.061	0.060	0.061	0.060	Yes
Minnesota	Stearns	St. Cloud, MN	5	271453052	100	95	100	98	0.062	0.060	0.058	0.060	Yes
Minnesota	Washington	Minneapolis-St. Paul-Bloomington, MN-WI	5	271636016	98	97	99	98	0.061	0.058	0.060	0.059	Yes
Minnesota	Wright	Minneapolis-St. Paul-Bloomington, MN-WI	5	271713201	100	96	98	98	0.064	0.059	0.061	0.061	Yes
Mississippi	Bolivar	Cleveland, MS	4	280110001	97	99	98	98	0.060	0.063	0.064	0.062	Yes
Mississippi	DeSoto	Memphis, TN-MS-AR	4	280330002	100	99	88	96	0.067	0.061	0.066	0.064	Yes
Mississippi	Hancock	Gulfport-Biloxi-Pascagoula, MS	4	280450003	99	96	94	96	0.069	0.061	0.061	0.063	Yes

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Mississippi	Harrison	Gulfport-Biloxi-Pascagoula, MS	4	280470008	89	98	98	95	0.073	0.067	0.063	0.067	Yes
Mississippi	Hinds	Jackson, MS	4	280490020	97	93	90	93	0.064	0.061	0.060	0.061	Yes
Mississippi	Jackson	Gulfport-Biloxi-Pascagoula, MS	4	280590006	97	98	98	98	0.075	0.065	0.062	0.067	Yes
Mississippi	Lauderdale	Meridian, MS	4	280750003	98	98	86	94	0.058	0.056	0.058	0.057	Yes
Mississippi	Lee	Tupelo, MS	4	280810005	99	96	97	97	0.059	0.058	0.061	0.059	Yes
Mississippi	Yalobusha		4	281619991	88	87	99	91	0.060	0.056	0.057	0.057	Yes
Missouri	Andrew	St. Joseph, MO-KS	7	290030001	99	99	100	99	0.064	0.064	0.062	0.063	Yes
Missouri	Boone	Columbia, MO	7	290190011	100	99	100	100	0.064	0.063	0.065	0.064	Yes
Missouri	Callaway	Jefferson City, MO	7	290270002	99	100	100	100	0.064	0.065	0.064	0.064	Yes
Missouri	Cass	Kansas City, MO-KS	7	290370003	100	100	99	100	0.065	0.065	0.061	0.063	Yes
Missouri	Cedar		7	290390001	99	99	99	99	0.065	0.060	0.060	0.061	Yes
Missouri	Clay	Kansas City, MO-KS	7	290470006	100	100	99	100	0.066	0.068	0.069	0.067	Yes
Missouri	Clinton	Kansas City, MO-KS	7	290490001	99	100	99	99	0.064	0.068	0.069	0.067	Yes
Missouri	Greene	Springfield, MO	7	290770042	99	100	99	99	0.060	0.061	0.059	0.060	Yes
Missouri	Jasper	Joplin, MO	7	290970004	100	98	100	99	0.065	0.061	0.059	0.061	Yes
Missouri	Jefferson	St. Louis, MO-IL	7	290990019	99	100	99	99	0.072	0.069	0.070	0.070	Yes
Missouri	Lincoln	St. Louis, MO-IL	7	291130003	100	99	99	99	0.067	0.065	0.065	0.065	Yes
Missouri	Monroe		7	291370001	100	100	100	100	0.060	0.058	0.059	0.059	Yes
Missouri	Perry		7	291570001	100	99	100	100	0.067	0.067	0.069	0.067	Yes
Missouri	Saint Charles	St. Louis, MO-IL	7	291831002	100	90	99	96	0.072	0.070	0.075	0.072	No
Missouri	Saint Louis	St. Louis, MO-IL	7	291890014	99	100	99	99	0.072	0.069	0.073	0.071	No
Missouri	Sainte Genevieve		7	291860005	100	100	98	99	0.069	0.063	0.067	0.066	Yes
Missouri	St. Louis City	St. Louis, MO-IL	7	295100085	99	99	100	99	0.066	0.063	0.068	0.065	Yes
Missouri	Taney	Branson, MO	7	292130004	100	100	99	100	0.058	0.059	0.056	0.057	Yes
Montana	Fergus		8	300270006	91	92	97	93	0.056	0.056	0.055	0.055	Yes
Montana	Flathead	Kalispell, MT	8	300298001	91	98	93	94	0.052	0.057	0.050	0.053	Yes
Montana	Lewis and Clark	Helena, MT	8	300490004	100	92	100	97	0.058	0.058	0.055	0.057	Yes
Montana	Missoula	Missoula, MT	8	300630024	82	99	98	93	0.053	0.055	0.051	0.053	Yes
Montana	Phillips		8	300710010	89	98	99	95	0.052	0.061	0.053	0.055	Yes
Montana	Richland		8	300830001	95	94	92	94	0.051	0.058	0.057	0.055	Yes
Montana	Rosebud		8	300870001	100	82	98	93	0.055	0.056	0.057	0.056	Yes
Nebraska	Douglas	Omaha-Council Bluffs, NE-IA	7	310550019	99	100	93	97	0.063	0.062	0.062	0.062	Yes
Nebraska	Knox		7	311079991	97	95	99	97	0.063	0.063	0.064	0.063	Yes
Nebraska	Lancaster	Lincoln, NE	7	311090016	98	97	97	97	0.061	0.061	0.058	0.060	Yes
Nevada	Carson City	Carson City, NV	9	325100020	100	100	99	100	0.068	0.068	0.066	0.067	Yes
Nevada	Churchill	Fallon, NV	9	320010002	98	94	95	96	0.065	0.068	0.067	0.066	Yes
Nevada	Clark	Las Vegas-Henderson-Paradise, NV	9	320030075	97	99	96	97	0.079	0.071	0.073	0.074	No
Nevada	Lyon	Fernley, NV	9	320190006	99	100	95	98	0.067	0.071	0.068	0.068	Yes
Nevada	Washoe	Reno, NV	9	320310016	95	94	95	95	0.071	0.073	0.069	0.071	No
Nevada	White Pine		9	320330101	93	97	97	96	0.064	0.066	0.063	0.064	Yes
New Hampshire	Belknap	Laconia, NH	1	330012004	99	99	99	99	0.061	0.055	0.060	0.058	Yes
New Hampshire	Cheshire	Keene, NH	1	330050007	100	99	100	100	0.060	0.059	0.066	0.061	Yes
New Hampshire	Coos	Berlin, NH-VT	1	330074001	99	98	99	99	0.065	0.071	0.066	0.067	Yes
New Hampshire	Grafton	Claremont-Lebanon, NH-VT	1	330090010	99	99	100	99	0.062	0.054	0.055	0.057	Yes
New Hampshire	Hillsborough	Manchester-Nashua, NH	1	330115001	99	100	100	100	0.069	0.066	0.069	0.068	Yes
New Hampshire	Merrimack	Concord, NH	1	330131007	98	100	100	99	0.061	0.062	0.062	0.061	Yes
New Hampshire	Rockingham	Boston-Cambridge-Newton, MA-NH	1	330150016	99	97	99	98	0.070	0.065	0.067	0.067	Yes
New Jersey	Atlantic	Atlantic City-Hammonton, NJ	2	340010006	99	96	99	98	0.061	0.068	0.063	0.064	Yes
New Jersey	Bergen	New York-Newark-Jersey City, NY-NJ-PA	2	340030006	97	98	99	98	0.073	0.076	0.072	0.073	No
New Jersey	Camden	Philadelphia-Camden-Wilmington, PA-NJ-DE-MD	2	340070002	100	100	100	100	0.068	0.079	0.076	0.074	No

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New Jersey	Cumberland	Vineland-Bridgeton, NJ	2	340110007	100	99	100	100	0.067	0.068	0.068	0.067	Yes
New Jersey	Essex	New York-Newark-Jersey City, NY-NJ-PA	2	340130003	99	99	100	99	0.070	0.072	0.068	0.070	Yes
New Jersey	Gloucester	Philadelphia-Camden-Wilmington, PA-NJ-DE-MD	2	340150002	84	96	95	92	0.070	0.076	0.074	0.073	No
New Jersey	Hudson	New York-Newark-Jersey City, NY-NJ-PA	2	340170006	99	100	100	100	0.072	0.077	0.067	0.072	No
New Jersey	Hunterdon	New York-Newark-Jersey City, NY-NJ-PA	2	340190001	100	100	100	100	0.065	0.073	0.073	0.070	Yes
New Jersey	Mercer	Trenton, NJ	2	340219991	100	99	97	99	0.071	0.075	0.074	0.073	No
New Jersey	Middlesex	New York-Newark-Jersey City, NY-NJ-PA	2	340230011	97	68	95	87	0.071	0.077	0.074	0.074	No
New Jersey	Monmouth	New York-Newark-Jersey City, NY-NJ-PA	2	340250005	98	98	100	99	0.064	0.077	0.068	0.069	Yes
New Jersey	Morris	New York-Newark-Jersey City, NY-NJ-PA	2	340273001	97	99	95	97	0.068	0.070	0.068	0.068	Yes
New Jersey	Ocean	New York-Newark-Jersey City, NY-NJ-PA	2	340290006	100	100	89	96	0.072	0.075	0.071	0.072	No
New Jersey	Passaic	New York-Newark-Jersey City, NY-NJ-PA	2	340315001	98	96	96	97	0.067	0.071	0.068	0.068	Yes
New Jersey	Warren	Allentown-Bethlehem-Easton, PA-NJ	2	340410007	99	100	100	100	0.060	0.066	0.065	0.063	Yes
New Mexico	Bernalillo	Albuquerque, NM	6	350010023	98	97	97	97	0.064	0.065	0.067	0.065	Yes
New Mexico	Dona Ana	Las Cruces, NM	6	350130021	91	98	99	96	0.072	0.074	0.070	0.072	No
New Mexico	Eddy	Carlsbad-Artesia, NM	6	350151005	92	96	98	95	0.072	0.067	0.063	0.067	Yes
New Mexico	Lea	Hobbs, NM	6	350250008	95	98	97	97	0.068	0.067	0.065	0.066	Yes
New Mexico	Rio Arriba	Espanola, NM	6	350390026	97	98	98	98	0.065	0.064	0.063	0.064	Yes
New Mexico	San Juan	Farmington, NM	6	350450018	92	99	94	95	0.063	0.068	0.067	0.066	Yes
New Mexico	Sandoval	Albuquerque, NM	6	350431001	90	97	98	95	0.062	0.066	0.064	0.064	Yes
New Mexico	Santa Fe	Santa Fe, NM	6	350490021	99	99	99	99	0.064	0.062	0.064	0.063	Yes
New Mexico	Valencia	Albuquerque, NM	6	350610008	97	97	98	97	0.064	0.064	0.065	0.064	Yes
New York	Albany	Albany-Schenectady-Troy, NY	2	360010012	93	99	98	97	0.061	0.063	0.068	0.064	Yes
New York	Bronx	New York-Newark-Jersey City, NY-NJ-PA	2	360050133	100	100	100	100	0.070	0.072	0.070	0.070	Yes
New York	Chautauqua	Jamestown-Dunkirk-Fredonia, NY	2	360130006	95	97	100	97	0.066	0.071	0.069	0.068	Yes
New York	Dutchess	New York-Newark-Jersey City, NY-NJ-PA	2	360270007	97	97	96	97	0.068	0.067	0.071	0.068	Yes
New York	Erie	Buffalo-Cheektowaga-Niagara Falls, NY	2	360290002	95	100	96	97	0.063	0.071	0.074	0.069	Yes
New York	Essex		2	360310003	95	90	93	93	0.060	0.067	0.068	0.065	Yes
New York	Franklin	Malone, NY	2	360337003	94	93	86	91	0.054	0.071	0.047	0.057	Yes
New York	Hamilton		2	360410005	97	98	95	97	0.058	0.062	0.061	0.060	Yes
New York	Herkimer	Utica-Rome, NY	2	360430005	98	99	96	98	0.057	0.061	0.071	0.063	Yes
New York	Jefferson	Watertown-Fort Drum, NY	2	360450002	99	98	88	95	0.059	0.065	0.067	0.063	Yes
New York	Monroe	Rochester, NY	2	360551007	98	98	97	98	0.058	0.065	0.067	0.063	Yes
New York	New York	New York-Newark-Jersey City, NY-NJ-PA	2	360610135	97	100	100	99	0.065	0.071	0.071	0.069	Yes
New York	Niagara	Buffalo-Cheektowaga-Niagara Falls, NY	2	360631006	83	98	97	93	0.061	0.067	0.070	0.066	Yes
New York	Onondaga	Syracuse, NY	2	360671015	96	96	100	97	0.063	0.063	0.067	0.064	Yes
New York	Orange	New York-Newark-Jersey City, NY-NJ-PA	2	360715001	99	99	96	98	0.062	0.072	0.064	0.066	Yes
New York	Oswego	Syracuse, NY	2	360750003	98	99	85	94	0.058	0.063	0.061	0.060	Yes
New York	Putnam	New York-Newark-Jersey City, NY-NJ-PA	2	360790005	100	99	98	99	0.066	0.069	0.071	0.068	Yes
New York	Queens	New York-Newark-Jersey City, NY-NJ-PA	2	360810124	92	99	95	95	0.063	0.073	0.071	0.069	Yes
New York	Richmond	New York-Newark-Jersey City, NY-NJ-PA	2	360850067	96	100	98	98	0.072	0.079	0.077	0.076	No
New York	Rockland	New York-Newark-Jersey City, NY-NJ-PA	2	360870005	100	100	98	99	0.068	0.077	0.073	0.072	No
New York	Saratoga	Albany-Schenectady-Troy, NY	2	360910004	100	100	98	99	0.061	0.061	0.067	0.063	Yes
New York	Steuben	Corning, NY	2	361010003	91	85	94	90	0.058	0.059	0.062	0.059	Yes
New York	Suffolk	New York-Newark-Jersey City, NY-NJ-PA	2	361030004	95	99	95	96	0.064	0.076	0.078	0.072	No
New York	Tompkins	Ithaca, NY	2	361099991	95	96	98	96	0.059	0.064	0.066	0.063	Yes
New York	Wayne	Rochester, NY	2	361173001	90	98	98	95	0.064	0.061	0.067	0.064	Yes
New York	Westchester	New York-Newark-Jersey City, NY-NJ-PA	2	361192004	100	100	100	100	0.074	0.073	0.075	0.074	No
North Carolina	Alexander	Hickory-Lenoir-Morganton, NC	4	370030005	99	97	99	98	0.064	0.065	0.066	0.065	Yes
North Carolina	Avery		4	370119991	94	96	90	93	0.062	0.062	0.069	0.064	Yes
North Carolina	Buncombe	Asheville, NC	4	370210030	100	99	99	99	0.064	0.065	0.062	0.063	Yes

State Name	County Name	CBSA Name	EPA Region	AQS Site ID	2014 Percent Complete	2015 Percent Complete	2016 Percent Complete	2014-2016 Average Percent Complete	2014 4th Highest Daily Max Value	2015 4th Highest Daily Max Value	2016 4th Highest Daily Max Value	Design Value 2014-2016	Meets NAAQS?
North Carolina	Caldwell	Hickory-Lenoir-Morganton, NC	4	370270003	98	96	100	98	0.061	0.065	0.066	0.064	Yes
North Carolina	Caswell		4	370330001	100	98	97	98	0.067	0.060	0.064	0.063	Yes
North Carolina	Cumberland	Fayetteville, NC	4	370510008	99	97	99	98	0.061	0.060	0.064	0.061	Yes
North Carolina	Durham	Durham-Chapel Hill, NC	4	370630015	100	96	98	98	0.062	0.061	0.063	0.062	Yes
North Carolina	Forsyth	Winston-Salem, NC	4	370670030	99	99	98	99	0.067	0.068	0.070	0.068	Yes
North Carolina	Graham		4	370750001	99	94	100	98	0.063	0.065	0.066	0.064	Yes
North Carolina	Granville	Oxford, NC	4	370770001	100	100	100	100	0.065	0.063	0.065	0.064	Yes
North Carolina	Guilford	Greensboro-High Point, NC	4	370810013	100	99	99	99	0.065	0.064	0.068	0.065	Yes
North Carolina	Haywood	Asheville, NC	4	370870035	91	93	99	94	0.067	0.065	0.067	0.066	Yes
North Carolina	Johnston	Raleigh, NC	4	371010002	99	99	99	99	0.064	0.065	0.066	0.065	Yes
North Carolina	Lee	Sanford, NC	4	371050002	99	100	98	99	0.064	0.060	0.064	0.062	Yes
North Carolina	Lenoir	Kinston, NC	4	371070004	100	97	90	96	0.065	0.062	0.063	0.063	Yes
North Carolina	Lincoln	Charlotte-Concord-Gastonia, NC-SC	4	371090004	99	97	98	98	0.064	0.068	0.069	0.067	Yes
North Carolina	Macon		4	371139991	95	94	94	94	0.063	0.062	0.061	0.062	Yes
North Carolina	Martin		4	371170001	100	96	92	96	0.061	0.059	0.061	0.060	Yes
North Carolina	Mecklenburg	Charlotte-Concord-Gastonia, NC-SC	4	371190041	100	100	99	100	0.065	0.073	0.070	0.069	Yes
North Carolina	Montgomery		4	371239991	100	96	87	94	0.062	0.059	0.062	0.061	Yes
North Carolina	New Hanover	Wilmington, NC	4	371290002	99	97	97	98	0.063	0.057	0.060	0.060	Yes
North Carolina	Person	Durham-Chapel Hill, NC	4	371450003	100	100	100	100	0.065	0.061	0.063	0.063	Yes
North Carolina	Pitt	Greenville, NC	4	371470006	95	97	92	95	0.063	0.060	0.065	0.062	Yes
North Carolina	Rockingham	Greensboro-High Point, NC	4	371570099	100	100	99	100	0.065	0.066	0.067	0.066	Yes
North Carolina	Rowan	Charlotte-Concord-Gastonia, NC-SC	4	371590021	100	98	100	99	0.064	0.066	0.066	0.065	Yes
North Carolina	Swain		4	371730007	95	92	98	95	0.060	0.063	0.061	0.061	Yes
North Carolina	Union	Charlotte-Concord-Gastonia, NC-SC	4	371790003	100	97	98	98	0.067	0.067	0.070	0.068	Yes
North Carolina	Wake	Raleigh, NC	4	371830014	100	97	97	98	0.063	0.065	0.069	0.065	Yes
North Carolina	Yancey		4	371990004	92	90	95	92	0.061	0.064	0.070	0.065	Yes
North Dakota	Billings		8	380070002	99	99	99	99	0.055	0.064	0.056	0.058	Yes
North Dakota	Burke		8	380130004	97	97	99	98	0.058	0.065	0.055	0.059	Yes
North Dakota	Dunn		8	380250003	98	100	99	99	0.057	0.063	0.056	0.058	Yes
North Dakota	McKenzie		8	380530002	95	96	100	97	0.056	0.061	0.056	0.057	Yes
North Dakota	Oliver	Bismarck, ND	8	380650002	89	100	100	96	0.060	0.064	0.054	0.059	Yes
North Dakota	Williams	Williston, ND	8	381050003	97	99	98	98	0.056	0.059	0.053	0.056	Yes
Ohio	Allen	Lima, OH	5	390030009	99	99	99	99	0.066	0.064	0.068	0.066	Yes
Ohio	Ashtabula	Ashtabula, OH	5	390071001	99	98	99	99	0.069	0.070	0.072	0.070	Yes
Ohio	Butler	Cincinnati, OH-KY-IN	5	390170004	99	94	95	96	0.070	0.070	0.076	0.072	No
Ohio	Clark	Springfield, OH	5	390230001	100	100	100	100	0.065	0.071	0.071	0.069	Yes
Ohio	Clermont	Cincinnati, OH-KY-IN	5	390250022	99	98	99	99	0.068	0.070	0.073	0.070	Yes
Ohio	Clinton	Wilmington, OH	5	390271002	99	100	98	99	0.070	0.070	0.071	0.070	Yes
Ohio	Cuyahoga	Cleveland-Elyria, OH	5	390350034	99	99	94	97	0.071	0.068	0.070	0.069	Yes
Ohio	Delaware	Columbus, OH	5	390410002	99	100	98	99	0.066	0.068	0.067	0.067	Yes
Ohio	Fayette	Washington Court House, OH	5	390479991	93	97	99	96	0.069	0.070	0.067	0.068	Yes
Ohio	Franklin	Columbus, OH	5	390490029	100	100	99	100	0.070	0.071	0.072	0.071	No
Ohio	Geauga	Cleveland-Elyria, OH	5	390550004	100	100	99	100	0.065	0.073	0.077	0.071	No
Ohio	Greene	Dayton, OH	5	390570006	100	100	100	100	0.066	0.071	0.069	0.068	Yes
Ohio	Hamilton	Cincinnati, OH-KY-IN	5	390610006	97	98	99	98	0.071	0.072	0.075	0.072	No
Ohio	Jefferson	Weirton-Stuebenville, WV-OH	5	390810017	99	100	100	100	0.067	0.066	0.062	0.065	Yes
Ohio	Knox	Mount Vernon, OH	5	390830002	100	97	99	99	0.066	0.071	0.066	0.067	Yes
Ohio	Lake	Cleveland-Elyria, OH	5	390850003	100	98	100	99	0.075	0.074	0.076	0.075	No
Ohio	Lawrence	Huntington-Ashland, WV-KY-OH	5	390870012	98	97	96	97	0.062	0.069	0.070	0.067	Yes
Ohio	Licking	Columbus, OH	5	390890005	100	99	99	99	0.066	0.068	0.067	0.067	Yes

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Ohio	Lorain	Cleveland-Elyria, OH	5	390930018	99	98	98	98	0.067	0.062	0.070	0.066	Yes
Ohio	Lucas	Toledo, OH	5	390950024	78	93	100	90	0.070	0.063	0.070	0.067	Yes
Ohio	Madison	Columbus, OH	5	390970007	95	100	99	98	0.069	0.069	0.068	0.068	Yes
Ohio	Mahoning	Youngstown-Warren-Boardman, OH-PA	5	390990013	93	98	96	96	0.066	0.069	0.054	0.063	Yes
Ohio	Medina	Cleveland-Elyria, OH	5	391030004	99	100	95	98	0.064	0.063	0.066	0.064	Yes
Ohio	Miami	Dayton, OH	5	391090005	100	99	99	99	0.066	0.068	0.069	0.067	Yes
Ohio	Montgomery	Dayton, OH	5	391130037	99	100	100	100	0.069	0.070	0.072	0.070	Yes
Ohio	Noble		5	391219991	97	98	93	96	0.065	0.066	0.068	0.066	Yes
Ohio	Portage	Akron, OH	5	391331001	99	100	99	99	0.061	0.064	0.059	0.061	Yes
Ohio	Preble		5	391351001	100	100	100	100	0.065	0.067	0.069	0.067	Yes
Ohio	Stark	Canton-Massillon, OH	5	391510016	94	93	98	95	0.065	0.072	0.072	0.069	Yes
Ohio	Summit	Akron, OH	5	391530020	100	99	99	99	0.058	0.065	0.061	0.061	Yes
Ohio	Trumbull	Youngstown-Warren-Boardman, OH-PA	5	391550011	90	100	100	97	0.065	0.070	0.071	0.068	Yes
Ohio	Warren	Cincinnati, OH-KY-IN	5	391650007	100	100	97	99	0.071	0.071	0.074	0.072	No
Ohio	Washington	Marietta, OH	5	391670004	93	100	100	98	0.063	0.068	0.064	0.065	Yes
Ohio	Wood	Toledo, OH	5	391730003	100	98	100	99	0.063	0.062	0.066	0.063	Yes
Oklahoma	Adair		6	400019009	98	90	97	95	0.063	0.062	0.058	0.061	Yes
Oklahoma	Canadian	Oklahoma City, OK	6	400170101	93	96	89	93	0.068	0.066	0.062	0.065	Yes
Oklahoma	Cherokee	Tahlequah, OK	6	400219002	100	97	95	97	0.061	0.061	0.058	0.060	Yes
Oklahoma	Cleveland	Oklahoma City, OK	6	400270049	99	100	99	99	0.068	0.065	0.065	0.066	Yes
Oklahoma	Comanche	Lawton, OK	6	400310651	100	98	100	99	0.069	0.066	0.061	0.065	Yes
Oklahoma	Creek	Tulsa, OK	6	400370144	95	95	93	94	0.066	0.063	0.064	0.064	Yes
Oklahoma	Dewey		6	400430860	92	92	93	92	0.067	0.065	0.065	0.065	Yes
Oklahoma	Kay	Ponca City, OK	6	400719010	96	88	97	94	0.069	0.063	0.059	0.063	Yes
Oklahoma	Mayes		6	400979014	98	97	100	98	0.060	0.065	0.061	0.062	Yes
Oklahoma	McClain	Oklahoma City, OK	6	400871073	99	98	99	99	0.069	0.065	0.065	0.066	Yes
Oklahoma	Oklahoma	Oklahoma City, OK	6	401091037	98	93	91	94	0.070	0.068	0.068	0.068	Yes
Oklahoma	Ottawa	Miami, OK	6	401159004	96	93	95	95	0.055	0.056	0.053	0.054	Yes
Oklahoma	Pittsburg	McAlester, OK	6	401210415	93	100	99	97	0.062	0.060	0.059	0.060	Yes
Oklahoma	Sequoyah	Fort Smith, AR-OK	6	401359021	88	99	90	92	0.060	0.062	0.058	0.060	Yes
Oklahoma	Tulsa	Tulsa, OK	6	401431127	100	95	96	97	0.065	0.068	0.062	0.065	Yes
Oregon	Clackamas	Portland-Vancouver-Hillsboro, OR-WA	10	410050004	99	96	99	98	0.062	0.069	0.064	0.065	Yes
Oregon	Columbia	Portland-Vancouver-Hillsboro, OR-WA	10	410090004	99	97	99	98	0.053	0.058	0.051	0.054	Yes
Oregon	Jackson	Medford, OR	10	410290201	98	91	85	91	0.059	0.069	0.049	0.059	Yes
Oregon	Lane	Eugene, OR	10	410390060	99	99	99	99	0.058	0.068	0.057	0.061	Yes
Oregon	Lane	Eugene, OR	10	410391007	99	99	99	99	0.058	0.071	0.056	0.061	Yes
Oregon	Marion	Salem, OR	10	410470004	94	79	98	90	0.066	0.065	0.065	0.065	Yes
Oregon	Multnomah	Portland-Vancouver-Hillsboro, OR-WA	10	410510080	99	99	96	98	0.055	0.057	0.055	0.055	Yes
Oregon	Umatilla	Hermiston-Pendleton, OR	10	410591003	95	98	89	94	0.064	0.070	0.063	0.065	Yes
Oregon	Washington	Portland-Vancouver-Hillsboro, OR-WA	10	410671004	98	99	98	98	0.057	0.063	0.058	0.059	Yes
Pennsylvania	Adams	Gettysburg, PA	3	420019991	94	97	97	96	0.063	0.067	0.071	0.067	Yes
Pennsylvania	Allegheny	Pittsburgh, PA	3	420031008	99	93	86	93	0.071	0.074	0.067	0.070	Yes
Pennsylvania	Armstrong	Pittsburgh, PA	3	420050001	100	100	93	98	0.068	0.070	0.068	0.068	Yes
Pennsylvania	Beaver	Pittsburgh, PA	3	420070002	98	98	95	97	0.069	0.070	0.069	0.069	Yes
Pennsylvania	Berks	Reading, PA	3	420110011	98	99	99	99	0.068	0.071	0.068	0.069	Yes
Pennsylvania	Blair	Altoona, PA	3	420130801	98	97	77	91	0.060	0.069	0.062	0.063	Yes
Pennsylvania	Bradford	Sayre, PA	3	420150011	100	100	97	99	0.051	0.058	0.057	0.055	Yes
Pennsylvania	Bucks	Philadelphia-Camden-Wilmington, PA-NJ-DE-MD	3	420170012	99	96	93	96	0.071	0.082	0.074	0.075	No
Pennsylvania	Cambria	Johnstown, PA	3	420210011	100	98	96	98	0.060	0.065	0.062	0.062	Yes
Pennsylvania	Centre	State College, PA	3	420279991	100	100	98	99	0.064	0.068	0.065	0.065	Yes

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Pennsylvania	Chester	Philadelphia-Camden-Wilmington, PA-NJ-DE-MD	3	420290100	94	97	96	96	0.071	0.068	0.072	0.070	Yes
Pennsylvania	Clearfield	DuBois, PA	3	420334000	91	97	93	94	0.060	0.068	0.066	0.064	Yes
Pennsylvania	Dauphin	Harrisburg-Carlisle, PA	3	420431100	93	99	100	97	0.063	0.068	0.065	0.065	Yes
Pennsylvania	Delaware	Philadelphia-Camden-Wilmington, PA-NJ-DE-MD	3	420450002	98	96	97	97	0.073	0.074	0.070	0.072	No
Pennsylvania	Elk		3	420479991	100	93	91	95	0.064	0.067	0.067	0.066	Yes
Pennsylvania	Erie	Erie, PA	3	420490003	90	100	98	96	0.065	0.066	0.065	0.065	Yes
Pennsylvania	Franklin	Chambersburg-Waynesboro, PA	3	420550001	98	99	92	96	0.063	0.059	0.058	0.060	Yes
Pennsylvania	Greene		3	420590002	99	99	91	96	0.065	0.071	0.067	0.067	Yes
Pennsylvania	Indiana	Indiana, PA	3	420630004	100	100	98	99	0.068	0.073	0.066	0.069	Yes
Pennsylvania	Lackawanna	Scranton--Wilkes-Barre--Hazleton, PA	3	420690101	100	99	92	97	0.061	0.069	0.068	0.066	Yes
Pennsylvania	Lancaster	Lancaster, PA	3	420710007	100	100	93	98	0.066	0.071	0.064	0.067	Yes
Pennsylvania	Lawrence	New Castle, PA	3	420730015	100	98	96	98	0.068	0.069	0.066	0.067	Yes
Pennsylvania	Lebanon	Lebanon, PA	3	420750100	100	99	99	99	0.067	0.074	0.067	0.069	Yes
Pennsylvania	Lehigh	Allentown-Bethlehem-Easton, PA-NJ	3	420770004	96	98	89	94	0.068	0.070	0.067	0.068	Yes
Pennsylvania	Luzerne	Scranton--Wilkes-Barre--Hazleton, PA	3	420791101	100	97	95	97	0.060	0.067	0.065	0.064	Yes
Pennsylvania	Lycoming	Williamsport, PA	3	420810100	100	100	99	100	0.062	0.065	0.063	0.063	Yes
Pennsylvania	Mercer	Youngstown-Warren-Boardman, OH-PA	3	420850100	98	78	99	92	0.071	0.066	0.069	0.068	Yes
Pennsylvania	Monroe	East Stroudsburg, PA	3	420890002	99	100	95	98	0.060	0.067	0.068	0.065	Yes
Pennsylvania	Montgomery	Philadelphia-Camden-Wilmington, PA-NJ-DE-MD	3	420910013	99	99	93	97	0.072	0.073	0.066	0.070	Yes
Pennsylvania	Northampton	Allentown-Bethlehem-Easton, PA-NJ	3	420950025	98	99	93	97	0.067	0.070	0.069	0.068	Yes
Pennsylvania	Philadelphia	Philadelphia-Camden-Wilmington, PA-NJ-DE-MD	3	421010024	94	100	97	97	0.072	0.079	0.074	0.075	No
Pennsylvania	Tioga		3	421174000	97	96	100	98	0.058	0.065	0.062	0.061	Yes
Pennsylvania	Washington	Pittsburgh, PA	3	421250005	100	98	98	99	0.065	0.072	0.069	0.068	Yes
Pennsylvania	Westmoreland	Pittsburgh, PA	3	421290008	99	96	100	98	0.064	0.069	0.072	0.068	Yes
Pennsylvania	York	York-Hanover, PA	3	421330008	99	100	99	99	0.063	0.068	0.067	0.066	Yes
Puerto Rico	Catano	San Juan-Carolina-Caguas, PR	2	720330008	92	95	94	94	0.020	0.029	0.033	0.027	Yes
Rhode Island	Kent	Providence-Warwick, RI-MA	1	440030002	98	98	96	97	0.067	0.070	0.075	0.070	Yes
Rhode Island	Providence	Providence-Warwick, RI-MA	1	440071010	98	98	97	98	0.064	0.071	0.071	0.068	Yes
Rhode Island	Washington	Providence-Warwick, RI-MA	1	440090007	98	99	99	99	0.063	0.077	0.071	0.070	Yes
South Carolina	Aiken	Augusta-Richmond County, GA-SC	4	450030003	96	98	99	98	0.062	0.057	0.062	0.060	Yes
South Carolina	Anderson	Greenville-Anderson-Mauldin, SC	4	450070005	99	96	100	98	0.060	0.063	0.062	0.061	Yes
South Carolina	Berkeley	Charleston-North Charleston, SC	4	450150002	97	93	85	92	0.060	0.054	0.058	0.057	Yes
South Carolina	Chesterfield		4	450250001	99	96	91	95	0.059	0.059	0.063	0.060	Yes
South Carolina	Darlington	Florence, SC	4	450310003	98	96	97	97	0.063	0.061	0.063	0.062	Yes
South Carolina	Edgefield	Augusta-Richmond County, GA-SC	4	450370001	94	93	96	94	0.055	0.061	0.062	0.059	Yes
South Carolina	Greenville	Greenville-Anderson-Mauldin, SC	4	450450016	98	96	95	96	0.062	0.067	0.066	0.065	Yes
South Carolina	Oconee	Seneca, SC	4	450730001	96	98	96	97	0.064	0.060	0.065	0.063	Yes
South Carolina	Pickens	Greenville-Anderson-Mauldin, SC	4	450770002	98	97	96	97	0.062	0.064	0.067	0.064	Yes
South Carolina	Richland	Columbia, SC	4	450791001	98	99	99	99	0.065	0.062	0.070	0.065	Yes
South Carolina	York	Charlotte-Concord-Gastonia, NC-SC	4	450910006	94	94	99	96	0.056	0.061	0.061	0.059	Yes
South Dakota	Brookings	Brookings, SD	8	460110003	99	99	93	97	0.061	0.063	0.061	0.061	Yes
South Dakota	Custer	Rapid City, SD	8	460330132	98	99	98	98	0.057	0.059	0.060	0.058	Yes
South Dakota	Jackson		8	460710001	99	99	100	99	0.057	0.057	0.060	0.058	Yes
South Dakota	Meade	Rapid City, SD	8	460930001	98	100	98	99	0.056	0.059	0.058	0.057	Yes
South Dakota	Minnehaha	Sioux Falls, SD	8	460990008	96	99	98	98	0.066	0.061	0.064	0.063	Yes
South Dakota	Union	Sioux City, IA-NE-SD	8	461270001	100	100	98	99	0.062	0.061	0.060	0.061	Yes
Tennessee	Anderson	Knoxville, TN	4	470010101	94	98	89	94	0.060	0.065	0.066	0.063	Yes
Tennessee	Blount	Knoxville, TN	4	470090101	99	98	96	98	0.064	0.068	0.070	0.067	Yes
Tennessee	Claiborne		4	470259991	98	99	93	97	0.065	0.061	0.064	0.063	Yes
Tennessee	Davidson	Nashville-Davidson--Murfreesboro--Franklin, TN	4	470370026	98	99	100	99	0.071	0.064	0.068	0.067	Yes

State Name	County Name	CBSA Name	EPA Regi on	AQS Site ID	2014 Percent Complete	2015 Percent Complete	2016 Percent Complete	2014-2016 Average Percent Complete	2014 4th Highest Daily Max Value	2015 4th Highest Daily Max Value	2016 4th Highest Daily Max Value	Design Value 2014-2016	Meets NAAQS?
Tennessee	DeKalb		4	470419991	98	96	96	97	0.062	0.061	0.063	0.062	Yes
Tennessee	Hamilton	Chattanooga, TN-GA	4	470654003	99	99	99	99	0.067	0.068	0.069	0.068	Yes
Tennessee	Jefferson	Morristown, TN	4	470890002	99	100	97	99	0.067	0.070	0.068	0.068	Yes
Tennessee	Knox	Knoxville, TN	4	470931020	98	99	100	99	0.063	0.066	0.070	0.066	Yes
Tennessee	Loudon	Knoxville, TN	4	471050108	98	96	95	96	0.067	0.069	0.073	0.069	Yes
Tennessee	Sevier	Sevierville, TN	4	471550101	97	98	96	97	0.068	0.067	0.070	0.068	Yes
Tennessee	Shelby	Memphis, TN-MS-AR	4	471570021	99	98	98	98	0.067	0.065	0.071	0.067	Yes
Tennessee	Sullivan	Kingsport-Bristol-Bristol, TN-VA	4	471632002	99	97	98	98	0.065	0.065	0.068	0.066	Yes
Tennessee	Sumner	Nashville-Davidson--Murfreesboro--Franklin, TN	4	471650007	99	100	99	99	0.066	0.067	0.068	0.067	Yes
Tennessee	Williamson	Nashville-Davidson--Murfreesboro--Franklin, TN	4	471870106	96	98	99	98	0.063	0.061	0.061	0.061	Yes
Tennessee	Wilson	Nashville-Davidson--Murfreesboro--Franklin, TN	4	471890103	97	98	91	95	0.064	0.061	0.067	0.064	Yes
Texas	Bell	Killeen-Temple, TX	6	480271045	96	100	99	98	0.067	0.072	0.064	0.067	Yes
Texas	Bexar	San Antonio-New Braunfels, TX	6	480290052	95	96	96	96	0.072	0.080	0.069	0.073	No
Texas	Brazoria	Houston-The Woodlands-Sugar Land, TX	6	480391004	99	98	98	98	0.071	0.086	0.069	0.075	No
Texas	Brewster		6	480430101	95	95	96	95	0.063	0.063	0.061	0.062	Yes
Texas	Cameron	Brownsville-Harlingen, TX	6	480611023	93	98	98	96	0.059	0.058	0.055	0.057	Yes
Texas	Collin	Dallas-Fort Worth-Arlington, TX	6	480850005	98	98	98	98	0.074	0.077	0.073	0.074	No
Texas	Dallas	Dallas-Fort Worth-Arlington, TX	6	481130075	86	98	99	94	0.070	0.079	0.067	0.072	No
Texas	Denton	Dallas-Fort Worth-Arlington, TX	6	481210034	100	97	98	98	0.077	0.088	0.076	0.080	No
Texas	El Paso	El Paso, TX	6	481410037	94	98	98	97	0.070	0.072	0.071	0.071	No
Texas	Ellis	Dallas-Fort Worth-Arlington, TX	6	481390016	98	99	100	99	0.062	0.068	0.060	0.063	Yes
Texas	Galveston	Houston-The Woodlands-Sugar Land, TX	6	481671034	96	96	97	96	0.071	0.084	0.074	0.076	No
Texas	Gregg	Longview, TX	6	481830001	100	99	99	99	0.066	0.068	0.065	0.066	Yes
Texas	Harris	Houston-The Woodlands-Sugar Land, TX	6	482010024	96	99	98	98	0.068	0.095	0.074	0.079	No
Texas	Harrison	Marshall, TX	6	482030002	98	99	98	98	0.066	0.062	0.059	0.062	Yes
Texas	Hidalgo	McAllen-Edinburg-Mission, TX	6	482150043	94	93	99	95	0.056	0.058	0.052	0.055	Yes
Texas	Hood	Dallas-Fort Worth-Arlington, TX	6	482210001	99	99	99	99	0.073	0.073	0.063	0.069	Yes
Texas	Hunt	Dallas-Fort Worth-Arlington, TX	6	482311006	97	99	99	98	0.062	0.062	0.058	0.060	Yes
Texas	Jefferson	Beaumont-Port Arthur, TX	6	482450022	96	98	90	95	0.067	0.068	0.069	0.068	Yes
Texas	Johnson	Dallas-Fort Worth-Arlington, TX	6	482510003	99	97	97	98	0.071	0.073	0.072	0.072	No
Texas	Kaufman	Dallas-Fort Worth-Arlington, TX	6	482570005	99	99	99	99	0.062	0.064	0.057	0.061	Yes
Texas	McLennan	Waco, TX	6	483091037	96	99	99	98	0.063	0.068	0.060	0.063	Yes
Texas	Montgomery	Houston-The Woodlands-Sugar Land, TX	6	483390078	99	99	99	99	0.072	0.073	0.071	0.072	No
Texas	Navarro	Corsicana, TX	6	483491051	99	98	97	98	0.060	0.064	0.060	0.061	Yes
Texas	Nueces	Corpus Christi, TX	6	483550025	96	97	97	97	0.065	0.065	0.062	0.064	Yes
Texas	Nueces	Corpus Christi, TX	6	483550026	97	98	92	96	0.066	0.062	0.061	0.063	Yes
Texas	Orange	Beaumont-Port Arthur, TX	6	483611001	99	95	99	98	0.063	0.062	0.058	0.061	Yes
Texas	Parker	Dallas-Fort Worth-Arlington, TX	6	483670081	98	100	100	99	0.072	0.079	0.068	0.073	No
Texas	Polk		6	483739991	90	90	93	91	0.065	0.063	0.056	0.061	Yes
Texas	Randall	Amarillo, TX	6	483819991	96	99	92	96	0.066	0.064	0.064	0.064	Yes
Texas	Rockwall	Dallas-Fort Worth-Arlington, TX	6	483970001	98	99	96	98	0.066	0.071	0.061	0.066	Yes
Texas	Smith	Tyler, TX	6	484230007	100	99	98	99	0.066	0.066	0.063	0.065	Yes
Texas	Tarrant	Dallas-Fort Worth-Arlington, TX	6	484393009	98	98	96	97	0.073	0.079	0.075	0.075	No
Texas	Travis	Austin-Round Rock, TX	6	484530014	96	100	98	98	0.062	0.073	0.064	0.066	Yes
Texas	Victoria	Victoria, TX	6	484690003	98	96	94	96	0.062	0.070	0.064	0.065	Yes
Texas	Webb	Laredo, TX	6	484790016	93	96	97	95	0.057	0.056	0.050	0.054	Yes
Utah	Box Elder	Ogden-Clearfield, UT	8	490030003	86	92	98	92	0.067	0.068	0.067	0.067	Yes
Utah	Carbon	Price, UT	8	490071003	98	99	95	97	0.064	0.069	0.067	0.066	Yes
Utah	Davis	Ogden-Clearfield, UT	8	490110004	99	54	99	84	0.074	0.073	0.076	0.074	No
Utah	Duchesne		8	490137011	39	98	98	78	0.067	0.066	0.085	0.072	No

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Utah	Salt Lake	Salt Lake City, UT	8	490353006	99	99	84	94	0.072	0.081	0.074	0.075	No
Utah	San Juan		8	490370101	95	99	99	98	0.064	0.065	0.064	0.064	Yes
Utah	Uintah	Vernal, UT	8	490472003	0	98	99	66	0.079	0.068	0.096	0.081	No
Utah		Provo-Orem, UT	8	490495010	97	94	99	97	0.076	0.071	0.072	0.073	No
Utah	Washington	St. George, UT	8	490530007	95	100	99	98	0.066	0.069	0.062	0.065	Yes
Utah	Weber	Ogden-Clearfield, UT	8	490571003	99	94	98	97	0.070	0.074	0.073	0.072	No
Vermont	Bennington	Bennington, VT	1	500030004	95	96	97	96	0.061	0.063	0.067	0.063	Yes
Vermont	Chittenden	Burlington-South Burlington, VT	1	500070007	94	95	99	96	0.059	0.066	0.060	0.061	Yes
Virginia	Arlington	Washington-Arlington-Alexandria, DC-VA-MD-WV	3	510130020	99	99	100	99	0.071	0.073	0.072	0.072	No
Virginia	Charles	Richmond, VA	3	510360002	93	94	98	95	0.066	0.059	0.065	0.063	Yes
Virginia	Chesterfield	Richmond, VA	3	510410004	86	88	99	91	0.061	0.063	0.063	0.062	Yes
Virginia	Fairfax	Washington-Arlington-Alexandria, DC-VA-MD-WV	3	510590030	92	97	98	96	0.065	0.072	0.073	0.070	Yes
Virginia	Fauquier	Washington-Arlington-Alexandria, DC-VA-MD-WV	3	510610002	98	97	99	98	0.059	0.056	0.063	0.059	Yes
Virginia	Frederick	Winchester, VA-WV	3	510690010	99	96	99	98	0.059	0.061	0.065	0.061	Yes
Virginia	Giles	Blacksburg-Christiansburg-Radford, VA	3	510719991	98	99	93	97	0.062	0.063	0.062	0.062	Yes
Virginia	Hampton City	Virginia Beach-Norfolk-Newport News, VA-NC	3	516500008	99	99	97	98	0.061	0.065	0.068	0.064	Yes
Virginia	Hanover	Richmond, VA	3	510850003	93	100	96	96	0.062	0.061	0.065	0.062	Yes
Virginia	Loudoun	Washington-Arlington-Alexandria, DC-VA-MD-WV	3	511071005	99	93	98	97	0.063	0.071	0.068	0.067	Yes
Virginia	Madison		3	511130003	98	98	99	98	0.060	0.063	0.066	0.063	Yes
Virginia	Prince Edward		3	511479991	91	99	95	95	0.063	0.057	0.060	0.060	Yes
Virginia	Prince William	Washington-Arlington-Alexandria, DC-VA-MD-WV	3	511530009	99	99	99	99	0.062	0.067	0.067	0.065	Yes
Virginia	Roanoke	Roanoke, VA	3	511611004	93	99	93	95	0.060	0.062	0.064	0.062	Yes
Virginia	Rockbridge		3	511630003	99	99	99	99	0.058	0.056	0.060	0.058	Yes
Virginia	Rockingham	Harrisonburg, VA	3	511650003	97	100	98	98	0.058	0.060	0.062	0.060	Yes
Virginia	Stafford	Washington-Arlington-Alexandria, DC-VA-MD-WV	3	511790001	100	100	90	97	0.062	0.063	0.066	0.063	Yes
Virginia	Suffolk City	Virginia Beach-Norfolk-Newport News, VA-NC	3	518000005	96	99	97	97	0.063	0.060	0.061	0.061	Yes
Virginia	Wythe		3	511970002	99	98	97	98	0.064	0.058	0.062	0.061	Yes
Washington	Clark	Portland-Vancouver-Hillsboro, OR-WA	10	530110011	99	86	92	92	0.057	0.065	0.055	0.059	Yes
Washington	King	Seattle-Tacoma-Bellevue, WA	10	530330023	97	99	90	95	0.067	0.074	0.061	0.067	Yes
Washington	Skagit	Mount Vernon-Anacortes, WA	10	530570020	93	99	99	97	0.048	0.050	0.052	0.050	Yes
Washington	Spokane	Spokane-Spokane Valley, WA	10	530630046	92	98	99	96	0.060	0.063	0.056	0.059	Yes
Washington	Thurston	Olympia-Tumwater, WA	10	530670005	97	98	97	97	0.056	0.058	0.058	0.057	Yes
Washington	Whatcom	Bellingham, WA	10	530730005	85	99	99	94	0.047	0.048	0.045	0.046	Yes
West Virginia	Berkeley	Hagerstown-Martinsburg, MD-WV	3	540030003	92	98	96	95	0.060	0.066	0.064	0.063	Yes
West Virginia	Cabell	Huntington-Ashland, WV-KY-OH	3	540110006	99	98	99	99	0.063	0.066	0.065	0.064	Yes
West Virginia	Gilmer		3	540219991	97	96	93	95	0.058	0.057	0.062	0.059	Yes
West Virginia	Greenbrier		3	540250003	97	98	97	97	0.060	0.057	0.062	0.059	Yes
West Virginia	Monongalia	Morgantown, WV	3	540610003	97	97	96	97	0.064	0.069	0.059	0.064	Yes
West Virginia	Ohio	Wheeling, WV-OH	3	540690010	100	98	100	99	0.066	0.069	0.069	0.068	Yes
West Virginia	Tucker		3	540939991	99	98	95	97	0.059	0.062	0.065	0.062	Yes
West Virginia	Wood	Parkersburg-Vienna, WV	3	541071002	96	99	98	98	0.067	0.071	0.067	0.068	Yes
Wisconsin	Ashland		5	550030010	95	97	96	96	0.056	0.057	0.062	0.058	Yes
Wisconsin	Brown	Green Bay, WI	5	550090026	97	99	98	98	0.066	0.065	0.068	0.066	Yes
Wisconsin	Columbia	Madison, WI	5	550210015	99	99	98	99	0.069	0.064	0.070	0.067	Yes
Wisconsin	Dane	Madison, WI	5	550250041	99	100	100	100	0.066	0.064	0.066	0.065	Yes
Wisconsin	Dodge	Beaver Dam, WI	5	550270001	99	98	98	98	0.071	0.066	0.068	0.068	Yes
Wisconsin	Door		5	550290004	98	97	99	98	0.065	0.074	0.077	0.072	No
Wisconsin	Eau Claire	Eau Claire, WI	5	550350014	99	95	98	97	0.061	0.059	0.064	0.061	Yes
Wisconsin	Fond du Lac	Fond du Lac, WI	5	550390006	99	99	99	99	0.067	0.065	0.066	0.066	Yes
Wisconsin	Forest		5	550410007	93	98	93	95	0.061	0.063	0.065	0.063	Yes

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Wisconsin	Jefferson	Watertown-Fort Atkinson, WI	5	550550009	99	99	100	99	0.071	0.065	0.071	0.069	Yes
Wisconsin	Kenosha	Chicago-Naperville-Elgin, IL-IN-WI	5	550590019	100	99	100	100	0.076	0.075	0.080	0.077	No
Wisconsin	Kewaunee	Green Bay, WI	5	550610002	100	100	99	100	0.065	0.070	0.072	0.069	Yes
Wisconsin	La Crosse	La Crosse-Onalaska, WI-MN	5	550630012	96	98	99	98	0.063	0.061	0.063	0.062	Yes
Wisconsin	Manitowoc	Manitowoc, WI	5	550710007	98	99	99	99	0.066	0.077	0.074	0.072	No
Wisconsin	Marathon	Wausau, WI	5	550730012	90	95	98	94	0.064	0.063	0.068	0.065	Yes
Wisconsin	Milwaukee	Milwaukee-Waukesha-West Allis, WI	5	550790085	99	100	100	100	0.069	0.068	0.077	0.071	No
Wisconsin	Outagamie	Appleton, WI	5	550870009	99	99	100	99	0.070	0.066	0.066	0.067	Yes
Wisconsin	Ozaukee	Milwaukee-Waukesha-West Allis, WI	5	550890009	98	99	100	99	0.070	0.071	0.079	0.073	No
Wisconsin	Rock	Janesville-Beloit, WI	5	551050030	99	100	100	100	0.072	0.064	0.072	0.069	Yes
Wisconsin	Sauk	Baraboo, WI	5	551110007	99	93	99	97	0.064	0.063	0.066	0.064	Yes
Wisconsin	Sheboygan	Sheboygan, WI	5	551170006	98	99	98	98	0.072	0.081	0.085	0.079	No
Wisconsin	Taylor		5	551199991	98	94	95	96	0.062	0.057	0.064	0.061	Yes
Wisconsin	Vilas		5	551250001	91	93	96	93	0.061	0.060	0.063	0.061	Yes
Wisconsin	Walworth	Whitewater-Elkhorn, WI	5	551270005	98	100	100	99	0.073	0.067	0.072	0.070	Yes
Wisconsin	Waukesha	Milwaukee-Waukesha-West Allis, WI	5	551330027	98	97	100	98	0.067	0.066	0.067	0.066	Yes
Wyoming	Albany	Laramie, WY	8	560019991	93	96	91	93	0.065	0.064	0.064	0.064	Yes
Wyoming	Campbell	Gillette, WY	8	560050456	93	89	97	93	0.059	0.062	0.060	0.060	Yes
Wyoming	Carbon		8	560070100	97	81	98	92	0.062	0.059	0.060	0.060	Yes
Wyoming	Converse		8	560090008	98	96	98	97	0.058	0.060	0.060	0.059	Yes
Wyoming	Fremont	Riverton, WY	8	560130099	98	98	98	98	0.065	0.062	0.062	0.063	Yes
Wyoming	Laramie	Cheyenne, WY	8	560210100	89	95	94	93	0.065	0.063	0.061	0.063	Yes
Wyoming	Natrona	Casper, WY	8	560250100	98	99	95	97	0.061	0.060	0.061	0.060	Yes
Wyoming	Sublette		8	560359991	91	87	98	92	0.062	0.065	0.064	0.063	Yes
Wyoming	Sweetwater	Rock Springs, WY	8	560370300	97	87	97	94	0.063	0.071	0.064	0.066	Yes
Wyoming	Teton	Jackson, WY-ID	8	560391011	96	88	97	94	0.060	0.062	0.060	0.060	Yes
Wyoming	Uinta	Evanston, WY	8	560410101	96	98	98	97	0.059	0.066	0.060	0.061	Yes
Wyoming	Weston		8	560450003	98	100	95	98	0.059	0.061	0.060	0.060	Yes
<p>1. The level of the 2015 8-hour ozone NAAQS is 0.070 parts per million (ppm). The design value is the 3-year average of the annual 4th highest daily maximum 8-hour ozone concentration. The design value listed for each county is the highest among sites with valid design values.</p>													
<p>2. The design values shown here are computed using Federal Reference Method or equivalent data reported by State, Tribal, and Local monitoring agencies to EPA's Air Quality System (AQS) as of June 24, 2016. Concentrations flagged by State, Tribal, or Local monitoring agencies as having been affected by an exceptional event (e.g., wildfire, volcanic eruption) and concurred by the associated EPA Regional Office are not included in these calculations.</p>													

Attachment 2

State Name	County Name	Street Address	CBSA Name	CSA Name	EPA Region	AQS Site ID	Local Site Name
Alabama	Baldwin	FAIRHOPE HIGH SCHOOL, FAIRHOPE, ALABAMA	Daphne-Fairhope-Foley, AL	Mobile-Daphne-Fairhope, AL	4	010030010	FAIRHOPE, Alabama
Alabama	Colbert	WILSON DAM RD AND 2ND ST.	Florence-Muscle Shoals, AL		4	010331002	MUSCLE SHOALS
Alabama	DeKalb	Sand Mountain Alabama Agricultural Experiment Station, Crossville, AL 35962			4	010499991	Sand Mountain
Alabama	Elmore	DEWBERRY TRAIL, ELMORE CO., WETUMPKA, ALABAMA	Montgomery, AL		4	010510001	DBT, WETUMPKA
Alabama	Etowah	1450 PARKER ANDERSON LANE, SOUTHSIDE, AL 35907	Gadsden, AL		4	010550011	SOUTHSIDE
Alabama	Houston	161 BUFORD LANE	Dothan, AL	Dothan-Enterprise-Ozark, AL	4	010690004	DOTHAN
Alabama	Jefferson	NO. B'HAM,SOU R.R., 3009 28TH ST. NO.	Birmingham-Hoover, AL	Birmingham-Hoover-Talladega, AL	4	010730023	North Birmingham
Alabama	Jefferson	FAIRFIELD, PFD, 5229 COURT B	Birmingham-Hoover, AL	Birmingham-Hoover-Talladega, AL	4	010731003	Fairfield
Alabama	Jefferson	ROUTE 8 MCADORY	Birmingham-Hoover, AL	Birmingham-Hoover-Talladega, AL	4	010731005	McAdory
Alabama	Jefferson	201 ASHVILLE ROAD	Birmingham-Hoover, AL	Birmingham-Hoover-Talladega, AL	4	010731010	Leeds
Alabama	Jefferson	3425 TAMASSEE LANE, HOOVER, AL 35226	Birmingham-Hoover, AL	Birmingham-Hoover-Talladega, AL	4	010732006	
Alabama	Jefferson	10005 CORNER SCHOOL ROAD	Birmingham-Hoover, AL	Birmingham-Hoover-Talladega, AL	4	010735003	
Alabama	Jefferson	TARRANT, ELEM. SCH., 1269 PORTLAND STREE	Birmingham-Hoover, AL	Birmingham-Hoover-Talladega, AL	4	010736002	
Alabama	Madison	2201 AIRPORT ROAD	Huntsville, AL	Huntsville-Decatur-Albertville, AL	4	010890014	HUNTSVILLE OLD AIRPORT
Alabama	Madison	1130 Capshaw Road	Huntsville, AL	Huntsville-Decatur-Albertville, AL	4	010890022	HUNTSVILLE CAPSHAW ROAD
Alabama	Mobile	CHICKASAW, MOBILE CO., ALABAMA	Mobile, AL	Mobile-Daphne-Fairhope, AL	4	010970003	CHICKASAW
Alabama	Mobile	BAY RD. ,MOBILE AL.	Mobile, AL	Mobile-Daphne-Fairhope, AL	4	010972005	BAY ROAD
Alabama	Montgomery	1350 COLISEUM BLVD, MONTGOMERY, ALABAMA 36110	Montgomery, AL		4	011011002	MOMS, ADEM
Alabama	Morgan	P.O. BOX 2224 WALLACE DEVELOPMENT CENTER. DECATUR, ALABAMA	Decatur, AL	Huntsville-Decatur-Albertville, AL	4	011030011	DECATUR, Alabama
Alabama	Russell	9 WOODLAND DRIVE (SCHOOL) LADONIA, AL 36869	Columbus, GA-AL	Columbus-Auburn-Opelika, GA-AL	4	011130002	LADONIA, PHENIX CITY
Alabama	Shelby	HELENA, BEARDEN FARM	Birmingham-Hoover, AL	Birmingham-Hoover-Talladega, AL	4	011170004	HELENA
Alabama	Sumter	NNE of Ward Post Office, Sumter Co., Alabama			4	011190003	Ward, Sumter Co.
Alabama	Tuscaloosa	11690 SOUTHFORK DR. DUNCANVILLE, AL_35456	Tuscaloosa, AL		4	011250010	DUNCANVILLE, TUSCALOOSA
Alaska	Denali	DENALI NATIONAL PARK			10	020680003	
Alaska	Fairbanks North Star	809 Pioneer Road	Fairbanks, AK		10	020900034	NCORE
Arizona	Cochise	CHIRICAHUA NATIONAL MOUMENT	Sierra Vista-Douglas, AZ		9	040038001	Chiricahua National Monument
Arizona	Coconino	755 W BONITO	Flagstaff, AZ		9	040051008	FLAGSTAFF MIDDLE SCHOOL
Arizona	Coconino	GRAND CANYON NATIONAL PARK, W RIM DRIVE	Flagstaff, AZ		9	040058001	Grand Canyon National Park, The Abyss
Arizona	Gila	SOUTH OF SR88	Payson, AZ		9	040070010	TONTO NM
Arizona	La Paz	ALAMO LAKE STATE PARK			9	040128000	ALAMO LAKE

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Arizona	Maricopa	3847 W EARLL DR-WEST PHOENIX STATION	Phoenix-Mesa-Scottsdale, AZ		9	040130019	WEST PHOENIX
Arizona	Maricopa	310 S BROOKS, MESA	Phoenix-Mesa-Scottsdale, AZ		9	040131003	MESA
Arizona	Maricopa	601 E BUTLER DR & N 6TH ST,PHOENIX	Phoenix-Mesa-Scottsdale, AZ		9	040131004	NORTH PHOENIX
Arizona	Maricopa	4530 E MCKELLIPS RD-FALCON FIELD STATION	Phoenix-Mesa-Scottsdale, AZ		9	040131010	FALCON FIELD
Arizona	Maricopa	6000 W OLIVE AVE, GLENDALE	Phoenix-Mesa-Scottsdale, AZ		9	040132001	GLENDALE
Arizona	Maricopa	25000 N WINDY WALK-PINNACLE PEAK STATION	Phoenix-Mesa-Scottsdale, AZ		9	040132005	PINNACLE PEAK
Arizona	Maricopa	1645 E ROOSEVELT ST-CENTRAL PHOENIX STN	Phoenix-Mesa-Scottsdale, AZ		9	040133002	CENTRAL PHOENIX
Arizona	Maricopa	2857 N MILLER RD-S SCOTTSDALE STN	Phoenix-Mesa-Scottsdale, AZ		9	040133003	SOUTH SCOTTSDALE
Arizona	Maricopa	33 W TAMARISK AVE, PHOENIX	Phoenix-Mesa-Scottsdale, AZ		9	040134003	SOUTH PHOENIX
Arizona	Maricopa	275 S ELLIS, CHANDLER	Phoenix-Mesa-Scottsdale, AZ		9	040134004	WEST CHANDLER
Arizona	Maricopa	1525 S COLLEGE AVE, TEMPE	Phoenix-Mesa-Scottsdale, AZ		9	040134005	TEMPE
Arizona	Maricopa	37019 N. LAVA LANE. CAVE CREEK AZ	Phoenix-Mesa-Scottsdale, AZ		9	040134008	CAVE CREEK
Arizona	Maricopa	16825 N. DYSART	Phoenix-Mesa-Scottsdale, AZ		9	040134010	DYSART
Arizona	Maricopa	26453 W. MC85	Phoenix-Mesa-Scottsdale, AZ		9	040134011	BUCKEYE
Arizona	Maricopa	4208 W. PECOS LAVEEN, AZ 85339 (GILA CROSSING NORTH ELEM SCH)	Phoenix-Mesa-Scottsdale, AZ		9	040137003	St Johns Air Monitoring Site
Arizona	Maricopa	10844 EAST OSBORN ROAD SCOTTSDALE AZ 85256	Phoenix-Mesa-Scottsdale, AZ		9	040137020	Senior Center Air Monitoring Station
Arizona	Maricopa	15115 BEELINE HIGHWAY, SCOTTSDALE, AZ 86256	Phoenix-Mesa-Scottsdale, AZ		9	040137021	Red Mountain Air Monitoring Station
Arizona	Maricopa	3250 NORTH STAPLEY DRIVE, MESA, AZ 85203	Phoenix-Mesa-Scottsdale, AZ		9	040137022	Lehi Air Monitoring Station
Arizona	Maricopa	4827 NORTH COUNTRY CLUB DRIVE, SCOTTSDALE, AZ 85256	Phoenix-Mesa-Scottsdale, AZ		9	040137024	High School Air Monitoring Station
Arizona	Maricopa	7 SPRINGS RD-FAA RADAR STN-TONTO NF	Phoenix-Mesa-Scottsdale, AZ		9	040139508	HUMBOLDT MOUNTAIN
Arizona	Maricopa	USERY PASS RD/BUSH HWY	Phoenix-Mesa-Scottsdale, AZ		9	040139702	BLUE POINT-SHERIFF STATION-TONTO NF-SALT RIVER RECREATION AREA
Arizona	Maricopa	16426 E PALISADES BLVD, FOUNTAIN HILLS, AZ	Phoenix-Mesa-Scottsdale, AZ		9	040139704	FOUNTAIN HILLS
Arizona	Maricopa	25608 N FOREST RD., RIO VERDE, AZ	Phoenix-Mesa-Scottsdale, AZ		9	040139706	RIO VERDE
Arizona	Maricopa	4530 N 17TH AVENUE	Phoenix-Mesa-Scottsdale, AZ		9	040139997	JLG SUPERSITE
Arizona	Navajo	PET FOR NAT PARK, NEAR OLD SW ENTERANCE ON OLD ROUTE 180	Show Low, AZ		9	040170119	Petrified Forest National Park, South Entrance
Arizona	Pima	3905 S. OLD SPANISH TRAIL, SAGUARO PARK EAST	Tucson, AZ	Tucson-Nogales, AZ	9	040190021	SAGUARO PARK
Arizona	Pima	1237 S. BEVERLY, TUCSON	Tucson, AZ	Tucson-Nogales, AZ	9	040191011	22ND & CRAYCROFT
Arizona	Pima	12101 N CAMINO DE OESTE, TUCSON	Tucson, AZ	Tucson-Nogales, AZ	9	040191018	TANGERINE
Arizona	Pima	11330 S HOUGHTON, TUCSON	Tucson, AZ	Tucson-Nogales, AZ	9	040191020	FAIRGROUNDS

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Arizona	Pima	400 W RIVER ROAD	Tucson, AZ	Tucson-Nogales, AZ	9	040191028	CHILDREN'S PARK NCore
Arizona	Pima	601 NORTH LA CANADA DRIVE	Tucson, AZ	Tucson-Nogales, AZ	9	040191030	GREEN VALLEY - REPLACES SITE 0007 245 W ESPERANZA
Arizona	Pima	710 W. MICHIGAN ST.	Tucson, AZ	Tucson-Nogales, AZ	9	040191032	ROSE ELEMENTARY
Arizona	Pima	9597 N. COACHLINE BLVD	Tucson, AZ	Tucson-Nogales, AZ	9	040191034	COACHLINE
Arizona	Pinal	305 E. SUPERSTITION BLVD, APACHE JUNCTION	Phoenix-Mesa-Scottsdale, AZ		9	040213001	A J MAINTENANCE YARD
Arizona	Pinal	660 W. AERO DR. CASA GRANDE	Phoenix-Mesa-Scottsdale, AZ		9	040213003	CASA GRANDE AIRPORT
Arizona	Pinal	WATER WELL #2 PINAL AIR PARK RD MARANA	Phoenix-Mesa-Scottsdale, AZ		9	040213007	PINAL AIR PARK
Arizona	Pinal	35 PIMA STREET, SACATON	Phoenix-Mesa-Scottsdale, AZ		9	040217001	Sacaton Air Monitoring Site
Arizona	Pinal	10 S QUEEN ANN QUEEN VALLEY	Phoenix-Mesa-Scottsdale, AZ		9	040218001	QUEEN VALLEY
Arizona	Yavapai	330 Grove Avenue	Prescott, AZ		9	040258033	PRESCOTT COLLEGE AQD
Arizona	Yuma	2323 S ARIZONA AVE	Yuma, AZ		9	040278011	YUMA SUPERSITE
Arkansas	Clark	Lower Lake Recreation Area, Caddo Valley, AR 71923	Arkadelphia, AR		6	050199991	Caddo Valley
Arkansas	Crittenden	LH POLK AND COLONIAL DRIVE	Memphis, TN-MS-AR	Memphis-Forrest City, TN-MS-AR	6	050350005	MARION
Arkansas	Newton	HWY 16	Harrison, AR		6	051010002	DEER
Arkansas	Polk	463 POLK 631			6	051130003	EAGLE MOUNTAIN
Arkansas	Pulaski	PIKE AVE AT RIVER ROAD	Little Rock-North Little Rock-Conway, AR	Little Rock-North Little Rock, AR	6	051190007	PARR
Arkansas	Pulaski	REMOUNT ROAD N. LITTLE ROCK, ARKANSAS	Little Rock-North Little Rock-Conway, AR	Little Rock-North Little Rock, AR	6	051191002	NLR AIRPORT
Arkansas	Washington	600 SOUTH OLD MISSOURI ROAD	Fayetteville-Springdale-Rogers, AR-MO		6	051430005	SPRINGDALE
Arkansas	Washington	429 Ernest Lancaster Dr.	Fayetteville-Springdale-Rogers, AR-MO		6	051430006	Fayetteville Airport
California	Alameda	793 Rincon Ave.	San Francisco-Oakland-Hayward, CA	San Jose-San Francisco-Oakland, CA	9	060010007	Livermore
California	Alameda	9925 International Blvd	San Francisco-Oakland-Hayward, CA	San Jose-San Francisco-Oakland, CA	9	060010009	Oakland
California	Alameda	1100 21st Street	San Francisco-Oakland-Hayward, CA	San Jose-San Francisco-Oakland, CA	9	060010011	Oakland West
California	Alameda	3466 LA MESA DR.	San Francisco-Oakland-Hayward, CA	San Jose-San Francisco-Oakland, CA	9	060012001	Hayward
California	Amador	201 CLINTON ROAD, JACKSON, CA 95642			9	060050002	Jackson-Clinton Road
California	Butte	4405 AIRPORT RD-PARADISE CA	Chico, CA		9	060070007	Paradise - Airport
California	Butte	984 East Avenue, Chico	Chico, CA		9	060070008	Chico-East Avenue
California	Calaveras	501 GOLD STRIKE ROAD, SAN ANDREAS			9	060090001	San Andreas-Gold Strike Road
California	Colusa	100 SUNRISE BLVD., COLUSA			9	060111002	Colusa-Sunrise Blvd

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California	Contra Costa	2956-A TREAT BOULEVARD	San Francisco-Oakland-Hayward, CA	San Jose-San Francisco-Oakland, CA	9	060130002	Concord
California	Contra Costa	5551 BETHEL ISLAND RD	San Francisco-Oakland-Hayward, CA	San Jose-San Francisco-Oakland, CA	9	060131002	Bethel Island
California	Contra Costa	1865 D RUMRILL BLVD, San Pablo	San Francisco-Oakland-Hayward, CA	San Jose-San Francisco-Oakland, CA	9	060131004	San Pablo
California	Contra Costa	9885 Alcosta BLVD	San Francisco-Oakland-Hayward, CA	San Jose-San Francisco-Oakland, CA	9	060132007	San Ramon
California	El Dorado	3111 GOLD NUGGET WAY, PLACERVILLE, CA	Sacramento--Roseville--Arden-Arcade, CA	Sacramento-Roseville, CA	9	060170010	Placerville-Gold Nugget Way
California	El Dorado	1400 AMERICAN RIVER TRAIL, COOL	Sacramento--Roseville--Arden-Arcade, CA	Sacramento-Roseville, CA	9	060170020	Cool
California	Fresno	4706 E. DRUMMOND ST., FRESNO	Fresno, CA	Fresno-Madera, CA	9	060190007	Fresno-Drummond
California	Fresno	3727 N FIRST ST, FRESNO	Fresno, CA	Fresno-Madera, CA	9	060190011	Fresno - Garland
California	Fresno	SIERRA SKYPARK#2-BLYTHE & CHNNLT, FRESNO	Fresno, CA	Fresno-Madera, CA	9	060190242	Fresno-Sky Park
California	Fresno	32650 West Adams Avenue Tranquillity CA 93668	Fresno, CA	Fresno-Madera, CA	9	060192009	Tranquillity
California	Fresno	9240 S. RIVERBEND, PARLIER 93648	Fresno, CA	Fresno-Madera, CA	9	060194001	Parlier
California	Fresno	908 N VILLA AVE, CLOVIS	Fresno, CA	Fresno-Madera, CA	9	060195001	Clovis-Villa
California	Glenn	720 N Colusa Street, Willows			9	060210003	Willows-Colusa Street
California	Humboldt	717 SOUTH AVENUE	Eureka-Arcata-Fortuna, CA		9	060231004	Jacobs
California	Humboldt	170 meters SE of Donna Dr. & Humboldt Hill Rd., Eureka, CA	Eureka-Arcata-Fortuna, CA		9	060231005	Humboldt Hill
California	Imperial	1029 ETHEL ST, CALEXICO HIGH SCHOOL	El Centro, CA		9	060250005	Calexico-Ethel Street
California	Imperial	150 9TH ST., EL CENTRO	El Centro, CA		9	060251003	El Centro-9th Street
California	Imperial	7711 ENGLISH ROAD, NILAND	El Centro, CA		9	060254004	Niland-English Road
California	Kern	JOHNSON FARM, EDISON, CA. 93320	Bakersfield, CA		9	060290007	Edison
California	Kern	755 Stanislaus St., Maricopa CA 93352	Bakersfield, CA		9	060290008	Maricopa
California	Kern	923 POOLE STREET, MOJAVE, CA 93501	Bakersfield, CA		9	060290011	Mojave
California	Kern	5558 California Ave., Bakersfield CA 93309	Bakersfield, CA		9	060290014	Bakersfield-California
California	Kern	3311 Manor St, Oildale CA 93308	Bakersfield, CA		9	060290232	Oildale
California	Kern	2000 South Union Ave. Bakersfield CA 93307	Bakersfield, CA		9	060292012	Bakersfield-Muni
California	Kern	19405 BUENA VISTA BLVD, ARVIN, CA., 93203	Bakersfield, CA		9	060295002	Arvin-Di Giorgio
California	Kern	548 WALKER ST., SHAFTER, CA., 93263	Bakersfield, CA		9	060296001	Shafter
California	Kings	807 SOUTH IRWIN ST., HANFORD	Hanford-Corcoran, CA	Visalia-Porterville-Hanford, CA	9	060311004	Hanford-Irwin
California	Lake	905 LAKEPORT BLVD., LAKEPORT	Clearlake, CA		9	060333001	Lakeport-Lakeport Blvd.
California	Los Angeles	803 N. LOREN AVE., AZUSA	Los Angeles-Long Beach-Anaheim, CA	Los Angeles-Long Beach, CA	9	060370002	Azusa
California	Los Angeles	840 LAUREL, GLENDORA	Los Angeles-Long Beach-Anaheim, CA	Los Angeles-Long Beach, CA	9	060370016	Glendora
California	Los Angeles	VA HOSPITAL, WEST LOS ANGELES	Los Angeles-Long Beach-Anaheim, CA	Los Angeles-Long Beach, CA	9	060370113	West Los Angeles
California	Los Angeles	1630 N MAIN ST, LOS ANGELES	Los Angeles-Long Beach-Anaheim, CA	Los Angeles-Long Beach, CA	9	060371103	Los Angeles-North Main Street

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California	Los Angeles	18330 GAULT ST., RESEDA	Los Angeles-Long Beach-Anaheim, CA	Los Angeles-Long Beach, CA	9	060371201	Reseda
California	Los Angeles	700 North Bullis Road	Los Angeles-Long Beach-Anaheim, CA	Los Angeles-Long Beach, CA	9	060371302	Compton
California	Los Angeles	4144 SAN GABRIEL RIVER PKWY, PICO RIVERA	Los Angeles-Long Beach-Anaheim, CA	Los Angeles-Long Beach, CA	9	060371602	Pico Rivera #2
California	Los Angeles	924 N. GAREY AVE., POMONA	Los Angeles-Long Beach-Anaheim, CA	Los Angeles-Long Beach, CA	9	060371701	Pomona
California	Los Angeles	752 S. WILSON AVE., PASADENA	Los Angeles-Long Beach-Anaheim, CA	Los Angeles-Long Beach, CA	9	060372005	Pasadena
California	Los Angeles	2425 Webster St., Long Beach, CA	Los Angeles-Long Beach-Anaheim, CA	Los Angeles-Long Beach, CA	9	060374006	Long Beach (Hudson)
California	Los Angeles	7201 W. WESTCHESTER PARKWAY	Los Angeles-Long Beach-Anaheim, CA	Los Angeles-Long Beach, CA	9	060375005	LAX Hastings
California	Los Angeles	22224 PLACERITA CANYON RD, SANTA CLARITA	Los Angeles-Long Beach-Anaheim, CA	Los Angeles-Long Beach, CA	9	060376012	Santa Clarita
California	Los Angeles	43301 DIVISION ST., LANCASTER, CA	Los Angeles-Long Beach-Anaheim, CA	Los Angeles-Long Beach, CA	9	060379033	Lancaster-Division Street
California	Madera	RD. 29 1/2 NO. OF AVE 8 MADERA COUNTY	Madera, CA	Fresno-Madera, CA	9	060390004	Madera-Pump Yard
California	Madera	28261 Avenue 14 Madera CA 93638	Madera, CA	Fresno-Madera, CA	9	060392010	Madera-City
California	Marin	534 4TH ST.	San Francisco-Oakland-Hayward, CA	San Jose-San Francisco-Oakland, CA	9	060410001	San Rafael
California	Mariposa	TURTLEBACK DOME, YOSEMITE NATL' PK 95389			9	060430003	Yosemite National Park, Turtleback Dome
California	Mariposa	6440 JERSEYDALE ROAD, JERSEYDALE			9	060430006	Jerseydale
California	Mendocino	306 E. GOBBI STREET, UKIAH, CA	Ukiah, CA		9	060450008	Ukiah-Gobbi Street
California	Merced	385 S. COFFEE AVENUE, MERCED, CA 95340	Merced, CA	Modesto-Merced, CA	9	060470003	Merced-Coffee
California	Monterey	35 Ford Road	Salinas, CA		9	060530002	Carmel Valley
California	Monterey	415 Pearl Street	Salinas, CA		9	060530008	King City 2
California	Monterey	867 E. LAUREL Dr	Salinas, CA		9	060531003	Salinas 3
California	Napa	2552 JEFFERSON AVE.	Napa, CA	San Jose-San Francisco-Oakland, CA	9	060550003	Napa
California	Nevada	200 LITTON DR., GRASS VALLEY, CA	Truckee-Grass Valley, CA	Sacramento-Roseville, CA	9	060570005	Grass Valley-Litton Building
California	Orange	1630 W. PAMPAS LANE	Los Angeles-Long Beach-Anaheim, CA	Los Angeles-Long Beach, CA	9	060590007	Anaheim
California	Orange	2850 MESA VERDE DR EAST, COSTA MESA	Los Angeles-Long Beach-Anaheim, CA	Los Angeles-Long Beach, CA	9	060591003	Costa Mesa
California	Orange	26081 VIA PERA, MISSION VIEJO, CA 92691	Los Angeles-Long Beach-Anaheim, CA	Los Angeles-Long Beach, CA	9	060592022	Mission Viejo
California	Orange	621 W. LAMBERT, LA HABRA	Los Angeles-Long Beach-Anaheim, CA	Los Angeles-Long Beach, CA	9	060595001	La Habra
California	Placer	11645 Atwood Street, Auburn	Sacramento--Roseville--Arden-Arcade, CA	Sacramento-Roseville, CA	9	060610003	Auburn-Atwood

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California	Placer	CITY HALL-33 S MAIN ST, COLFAX	Sacramento--Roseville--Arden-Arcade, CA	Sacramento-Roseville, CA	9	060610004	Colfax-City Hall
California	Placer	151 NO SUNRISE BLVD, ROSEVILLE, CA	Sacramento--Roseville--Arden-Arcade, CA	Sacramento-Roseville, CA	9	060610006	Roseville-N Sunrise Ave
California	Placer	221 Fairway Drive, Tahoe City, CA 96145	Sacramento--Roseville--Arden-Arcade, CA	Sacramento-Roseville, CA	9	060611004	Tahoe City-Fairway Drive
California	Placer	1445 1st Street, Lincoln	Sacramento--Roseville--Arden-Arcade, CA	Sacramento-Roseville, CA	9	060612002	Lincoln-1st Street
California	Riverside	12705 Pechanga Rd., Temecula, CA 92592	Riverside-San Bernardino-Ontario, CA	Los Angeles-Long Beach, CA	9	060650009	Pechanga
California	Riverside	200 S. HATHAWAY ST., BANNING CA	Riverside-San Bernardino-Ontario, CA	Los Angeles-Long Beach, CA	9	060650012	Banning Airport
California	Riverside	33700 Borel Road, Winchester, CA 92596	Riverside-San Bernardino-Ontario, CA	Los Angeles-Long Beach, CA	9	060650016	Temecula
California	Riverside	12160 Santiago Rd. Banning, CA 92220	Riverside-San Bernardino-Ontario, CA	Los Angeles-Long Beach, CA	9	060651016	Morongo Air Monitoring Station
California	Riverside	46-990 JACKSON ST., INDIO	Riverside-San Bernardino-Ontario, CA	Los Angeles-Long Beach, CA	9	060652002	Indio
California	Riverside	FS-590 RACQUET CLUB AVE, PALM SPRINGS	Riverside-San Bernardino-Ontario, CA	Los Angeles-Long Beach, CA	9	060655001	Palm Springs
California	Riverside	237 1/2 N. 'D' ST., PERRIS	Riverside-San Bernardino-Ontario, CA	Los Angeles-Long Beach, CA	9	060656001	Perris
California	Riverside	5888 MISSION BLVD., RUBIDOUX	Riverside-San Bernardino-Ontario, CA	Los Angeles-Long Beach, CA	9	060658001	Rubidoux
California	Riverside	5130 POINSETTIA PLACE	Riverside-San Bernardino-Ontario, CA	Los Angeles-Long Beach, CA	9	060658005	Mira Loma (Van Buren)
California	Riverside	506 W FLINT ST, LAKE ELSINORE	Riverside-San Bernardino-Ontario, CA	Los Angeles-Long Beach, CA	9	060659001	Lake Elsinore
California	Riverside	495 W MURPHY STREET, BLYTHE	Riverside-San Bernardino-Ontario, CA	Los Angeles-Long Beach, CA	9	060659003	Blythe-445 W Murphy Street
California	Sacramento	7823 BLACKFOOT WAY, NORTH HIGHLANDS	Sacramento--Roseville--Arden-Arcade, CA	Sacramento-Roseville, CA	9	060670002	North Highlands-Blackfoot
California	Sacramento	DEL PASO-2701 AVALON DR, SACRAMENTO	Sacramento--Roseville--Arden-Arcade, CA	Sacramento-Roseville, CA	9	060670006	Sacramento-Del Paso Manor
California	Sacramento	1309 T ST., SACRAMENTO, CA. 95814	Sacramento--Roseville--Arden-Arcade, CA	Sacramento-Roseville, CA	9	060670010	Sacramento-1309 T Street
California	Sacramento	12490 BRUCEVILLE RD, ELK GROVE, CA	Sacramento--Roseville--Arden-Arcade, CA	Sacramento-Roseville, CA	9	060670011	Elk Grove-Bruceville
California	Sacramento	50 NATOMA STREET, FOLSOM	Sacramento--Roseville--Arden-Arcade, CA	Sacramento-Roseville, CA	9	060670012	Folsom-Natoma St.
California	Sacramento	68 GOLDENLAND COURT, SACRAMENTO, CA 95834	Sacramento--Roseville--Arden-Arcade, CA	Sacramento-Roseville, CA	9	060670014	Sacramento-Goldenland Ct.
California	Sacramento	7520 SLOUGHHOUSE ROAD, SLOUGHHOUSE	Sacramento--Roseville--Arden-Arcade, CA	Sacramento-Roseville, CA	9	060675003	Sloughhouse
California	San Benito	1979 Fairview Rd	San Jose-Sunnyvale-Santa Clara, CA	San Jose-San Francisco-Oakland, CA	9	060690002	Hollister

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California	San Benito	Northeast entrance, Pinnacles National Park	San Jose-Sunnyvale-Santa Clara, CA	San Jose-San Francisco-Oakland, CA	9	060690003	Pinnacles National Park
California	San Bernardino	200 E. BUENA VISTA, BARSTOW	Riverside-San Bernardino-Ontario, CA	Los Angeles-Long Beach, CA	9	060710001	Barstow
California	San Bernardino	LAKE GREGORY-LAKE DR, CRESTLINE	Riverside-San Bernardino-Ontario, CA	Los Angeles-Long Beach, CA	9	060710005	Crestline
California	San Bernardino	BEEKLEY & PHELAN RDS, PHELAN	Riverside-San Bernardino-Ontario, CA	Los Angeles-Long Beach, CA	9	060710012	Phelan-Beekley Road & Phelan Road
California	San Bernardino	14306 PARK AVE., VICTORVILLE, CA	Riverside-San Bernardino-Ontario, CA	Los Angeles-Long Beach, CA	9	060710306	Victorville-Park Avenue
California	San Bernardino	1350 SAN BERNARDINO RD., UPLAND	Riverside-San Bernardino-Ontario, CA	Los Angeles-Long Beach, CA	9	060711004	Upland
California	San Bernardino	Corner of Athol and Telescope, Trona	Riverside-San Bernardino-Ontario, CA	Los Angeles-Long Beach, CA	9	060711234	Trona - Athol/Telescope #2
California	San Bernardino	14360 ARROW BLVD., FONTANA	Riverside-San Bernardino-Ontario, CA	Los Angeles-Long Beach, CA	9	060712002	Fontana
California	San Bernardino	17288 OLIVE ST., HESPERIA	Riverside-San Bernardino-Ontario, CA	Los Angeles-Long Beach, CA	9	060714001	Hesperia-Olive Street
California	San Bernardino	500 N. DEARBORN, REDLANDS, CA. 92373	Riverside-San Bernardino-Ontario, CA	Los Angeles-Long Beach, CA	9	060714003	Redlands
California	San Bernardino	JOSHUA TREE NATIONAL MONUMENT	Riverside-San Bernardino-Ontario, CA	Los Angeles-Long Beach, CA	9	060719002	Joshua Tree National Park - Black Rock
California	San Bernardino	24302 4TH ST., SAN BERNARDINO, CA.	Riverside-San Bernardino-Ontario, CA	Los Angeles-Long Beach, CA	9	060719004	San Bernardino
California	San Diego	80 E. 'J' ST., CHULA VISTA	San Diego-Carlsbad, CA		9	060730001	Chula Vista
California	San Diego	225 9th Street	San Diego-Carlsbad, CA		9	060731001	Del Mar
California	San Diego	2300 VICTORIA DR., ALPINE	San Diego-Carlsbad, CA		9	060731006	Alpine
California	San Diego	21441-W B STREET	San Diego-Carlsbad, CA		9	060731008	Camp Pendleton
California	San Diego	1110 BEARDSLEY STREET, SAN DIEGO, CA 92112	San Diego-Carlsbad, CA		9	060731010	San Diego - Beardsley Street
California	San Diego	RICHARD J DONOVAN CORRECTIONAL FACILITY, OTAY MESA, CA	San Diego-Carlsbad, CA		9	060731014	Donovan
California	San Diego	6125A KEARNY VILLA RD., SAN DIEGO	San Diego-Carlsbad, CA		9	060731016	Kearny Villa Rd.
California	San Diego	10537 FLOYD SMITH DRIVE	San Diego-Carlsbad, CA		9	060731018	El Cajon - Floyd Smith Dr.
California	San Francisco	10 ARKANSAS ST.	San Francisco-Oakland-Hayward, CA	San Jose-San Francisco-Oakland, CA	9	060750005	San Francisco
California	San Joaquin	HAZELTON-HD, STOCKTON	Stockton-Lodi, CA	San Jose-San Francisco-Oakland, CA	9	060771002	Stockton-Hazelton
California	San Joaquin	5749 S. TRACY BLVD., TRACY	Stockton-Lodi, CA	San Jose-San Francisco-Oakland, CA	9	060773005	Tracy-Airport
California	San Luis Obispo	235 SANTA FE AVENUE, PASO ROBLES, CA	San Luis Obispo-Paso Robles-Arroyo Grande, CA		9	060790005	Paso Robles
California	San Luis Obispo	3220 SOUTH HIGUERA STREET, SAN LUIS OBISPO	San Luis Obispo-Paso Robles-Arroyo Grande, CA		9	060792006	San Luis Obispo
California	San Luis Obispo	MORRO BAY BLVD & KERN AVE, MORRO BAY	San Luis Obispo-Paso Robles-Arroyo Grande, CA		9	060793001	Morro Bay

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California	San Luis Obispo	NIPOMO REGIONAL PARK, NIPOMO, CA.	San Luis Obispo-Paso Robles-Arroyo Grande, CA		9	060794002	Nipomo Regional Park (NRP)
California	San Luis Obispo	5599 Traffic Way, Atascadero CA	San Luis Obispo-Paso Robles-Arroyo Grande, CA		9	060798002	Atascadero
California	San Luis Obispo	3601 GILLIS CANYON ROAD	San Luis Obispo-Paso Robles-Arroyo Grande, CA		9	060798005	Red Hills
California	San Luis Obispo	9640 CARRIZO HIGHWAY	San Luis Obispo-Paso Robles-Arroyo Grande, CA		9	060798006	Carrizo Plains
California	San Mateo	897 BARRON AVE.	San Francisco-Oakland-Hayward, CA	San Jose-San Francisco-Oakland, CA	9	060811001	Redwood City
California	Santa Barbara	EL CAPITAN ST PRK, HWY 10	Santa Maria-Santa Barbara, CA		9	060830008	El Capitan
California	Santa Barbara	700 E. CANON PERDIDO	Santa Maria-Santa Barbara, CA		9	060830011	Santa Barbara
California	Santa Barbara	906 S BROADWAY - SANTA MARIA	Santa Maria-Santa Barbara, CA		9	060831008	Santa Maria
California	Santa Barbara	HS & P FACILITY-500 M SW, LOMPOC	Santa Maria-Santa Barbara, CA		9	060831013	Lompoc HSP
California	Santa Barbara	PARADISE RD-LOS PADRES National Forest	Santa Maria-Santa Barbara, CA		9	060831014	Paradise Road
California	Santa Barbara	GTC B-HWY 101 NEAR NOJOQUI PASS, GAVIOTA	Santa Maria-Santa Barbara, CA		9	060831018	Nojoqui
California	Santa Barbara	GOBERNADOR RD, CARPINTERIA	Santa Maria-Santa Barbara, CA		9	060831021	Carpinteria
California	Santa Barbara	LFC #1-LAS FLORES CANYON	Santa Maria-Santa Barbara, CA		9	060831025	Los Flores Canyon #1
California	Santa Barbara	128 S 'H' ST, LOMPOC	Santa Maria-Santa Barbara, CA		9	060832004	Lompoc H Street
California	Santa Barbara	380 N FAIRVIEW AVENUE, GOLETA	Santa Maria-Santa Barbara, CA		9	060832011	Goleta
California	Santa Barbara	900 AIRPORT RD., SANTA YNEZ	Santa Maria-Santa Barbara, CA		9	060833001	Santa Ynez
California	Santa Barbara	STS POWER PLANT, VANDENBERG AFB	Santa Maria-Santa Barbara, CA		9	060834003	VAFB South Base
California	Santa Clara	9TH & PRINCEVILLE	San Jose-Sunnyvale-Santa Clara, CA	San Jose-San Francisco-Oakland, CA	9	060850002	Gilroy
California	Santa Clara	158B JACKSON ST	San Jose-Sunnyvale-Santa Clara, CA	San Jose-San Francisco-Oakland, CA	9	060850005	San Jose - Jackson
California	Santa Clara	306 UNIVERSITY AVE.	San Jose-Sunnyvale-Santa Clara, CA	San Jose-San Francisco-Oakland, CA	9	060851001	Los Gatos
California	Santa Clara	13030 MURPHY AVE.	San Jose-Sunnyvale-Santa Clara, CA	San Jose-San Francisco-Oakland, CA	9	060852006	San Martin
California	Santa Cruz	960 Bostwick Lane	Santa Cruz-Watsonville, CA	San Jose-San Francisco-Oakland, CA	9	060870007	Santa Cruz
California	Shasta	HLTH CTR-2630 BRESLAUER WAY, REDDING	Redding, CA	Redding-Red Bluff, CA	9	060890004	Redding - Health Department
California	Shasta	2220 NORTH STREET, ANDERSON, CA 96007	Redding, CA	Redding-Red Bluff, CA	9	060890007	Anderson - North Street
California	Shasta	13791 Lake Blvd, Shasta Lake, CA	Redding, CA	Redding-Red Bluff, CA	9	060890009	Shasta Lake - Lake Blvd
California	Shasta	MANZANITA LAKE RS, LASSEN VOLCANIC NP	Redding, CA	Redding-Red Bluff, CA	9	060893003	Lassen Volcanic National Park
California	Siskiyou	530 FOOTHILL DR., YREKA			9	060932001	Yreka
California	Solano	304 TUOLUMNE ST.	Vallejo-Fairfield, CA	San Jose-San Francisco-Oakland, CA	9	060950004	Vallejo
California	Solano	1010 CHADBOURNE RD	Vallejo-Fairfield, CA	San Jose-San Francisco-Oakland, CA	9	060950005	Fairfield

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California	Solano	2012 ULATIS DRIVE, VACAVILLE	Vallejo-Fairfield, CA	San Jose-San Francisco-Oakland, CA	9	060953003	Vacaville-Ulatis Drive
California	Sonoma	103 Morris Street,	Santa Rosa, CA	San Jose-San Francisco-Oakland, CA	9	060970004	Sebastopol
California	Sonoma	MUNICIPAL AIRPORT-200A HEIDELBERG WAY, HEALDSBURG	Santa Rosa, CA	San Jose-San Francisco-Oakland, CA	9	060971003	Healdsburg-Municipal Airport
California	Stanislaus	814 14TH ST., MODESTO	Modesto, CA	Modesto-Merced, CA	9	060990005	Modesto-14th Street
California	Stanislaus	900 S MINARET STREET, TURLOCK, CA	Modesto, CA	Modesto-Merced, CA	9	060990006	Turlock
California	Sutter	773 ALMOND ST, YUBA CITY	Yuba City, CA	Sacramento-Roseville, CA	9	061010003	Yuba City
California	Sutter	TOP OF SOUTH BUTTE,SUTTER BUTTES, CA	Yuba City, CA	Sacramento-Roseville, CA	9	061010004	Sutter Buttes (seasonal)
California	Tehama	OLD FIRE LOOKOUT ATOP TUSCAN BUTTE	Red Bluff, CA	Redding-Red Bluff, CA	9	061030004	Tuscan Butte (seasonal)
California	Tehama	1834 Walnut Street, Red Bluff, CA 96080	Red Bluff, CA	Redding-Red Bluff, CA	9	061030007	Red Bluff-Walnut St. District Office
California	Tulare	SEQUOIA & KINGS CANYON NP	Visalia-Porterville, CA	Visalia-Porterville-Hanford, CA	9	061070009	Sequoia and Kings Canyon National Parks, Ash Mountain
California	Tulare	310 N CHURCH ST, VISALIA	Visalia-Porterville, CA	Visalia-Porterville-Hanford, CA	9	061072002	Visalia-Church
California	Tulare	1839 South Newcomb Street Porterville, CA 93257	Visalia-Porterville, CA	Visalia-Porterville-Hanford, CA	9	061072010	Porterville
California	Tuolumne	251 S BARRETTA, SONORA, CA 95370	Sonora, CA		9	061090005	Sonora-Barretta Street
California	Ventura	2323 Moorpark Road, Thousand Oaks, CA 91360	Oxnard-Thousand Oaks-Ventura, CA	Los Angeles-Long Beach, CA	9	061110007	Thousand Oaks
California	Ventura	3301 PACIFIC AVENUE, PIRU, CA 93040	Oxnard-Thousand Oaks-Ventura, CA	Los Angeles-Long Beach, CA	9	061110009	Piru - Pacific
California	Ventura	1201 E. OJAI AVENUE, OJAI, CA 93023	Oxnard-Thousand Oaks-Ventura, CA	Los Angeles-Long Beach, CA	9	061111004	Ojai - East Ojai Ave
California	Ventura	5400 COCHRAN STREET, SIMI VALLEY, CA 93063	Oxnard-Thousand Oaks-Ventura, CA	Los Angeles-Long Beach, CA	9	061112002	Simi Valley-Cochran Street
California	Ventura	545 CENTRAL AVENUE, OXNARD, CA 93030	Oxnard-Thousand Oaks-Ventura, CA	Los Angeles-Long Beach, CA	9	061113001	El Rio-Rio Mesa School #2
California	Yolo	UC DAVIS-CAMPUS, CAMPBELL ROAD WEST OF HIWAY 113 & SOUTH OF HUTCHISON DRIVE.	Sacramento--Roseville--Arden-Arcade, CA	Sacramento-Roseville, CA	9	061130004	Davis-UCD Campus
California	Yolo	41929 E. GIBSON ROAD, WOODLAND	Sacramento--Roseville--Arden-Arcade, CA	Sacramento-Roseville, CA	9	061131003	Woodland-Gibson Road
Colorado	Adams	3174 E. 78TH AVE.	Denver-Aurora-Lakewood, CO	Denver-Aurora, CO	8	080013001	Welby
Colorado	Arapahoe	36001 E. Quincy Ave.	Denver-Aurora-Lakewood, CO	Denver-Aurora, CO	8	080050006	Aurora East
Colorado	Denver	2105 BROADWAY	Denver-Aurora-Lakewood, CO	Denver-Aurora, CO	8	080310002	DENVER - CAMP
Colorado	Denver	4545 Navajo St.	Denver-Aurora-Lakewood, CO	Denver-Aurora, CO	8	080310026	La Casa
Colorado	Douglas	11500 N. Roxborough Park Rd.	Denver-Aurora-Lakewood, CO	Denver-Aurora, CO	8	080350004	Chatfield State Park
Colorado	El Paso	ROAD 640, USAF ACADEMY	Colorado Springs, CO		8	080410013	U.S. AIR FORCE ACADEMY
Colorado	El Paso	101 BANKS PL.	Colorado Springs, CO		8	080410016	MANITOU SPRINGS
Colorado	Garfield	195 W. 14th St.	Glenwood Springs, CO	Edwards-Glenwood Springs, CO	8	080450012	Rifle-Health Dept

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Colorado	Gunnison	Gunnison National Forest, Crested Butte, CO 81224			8	080519991	Gothic
Colorado	Jefferson	12400 W. HWY 285	Denver-Aurora-Lakewood, CO	Denver-Aurora, CO	8	080590005	WELCH
Colorado	Jefferson	16600 W COLO #128	Denver-Aurora-Lakewood, CO	Denver-Aurora, CO	8	080590006	ROCKY FLATS-N
Colorado	Jefferson	2054 QUAKER ST.	Denver-Aurora-Lakewood, CO	Denver-Aurora, CO	8	080590011	NATIONAL RENEWABLE ENERGY LABS - NREL
Colorado	Jefferson	26137 Conifer Road	Denver-Aurora-Lakewood, CO	Denver-Aurora, CO	8	080590013	Aspen Park
Colorado	La Plata	Weminuche Wilderness Area - Shamrock Station	Durango, CO		8	080671004	
Colorado	La Plata	1 MI. NE OF IGNACIO ON COUNTY RD. 517	Durango, CO		8	080677001	LOCATED IN PINE RIVER VALLEY, THE MOST DENSELY POPULATED AREA.
Colorado	La Plata	7571 HWY. 5505	Durango, CO		8	080677003	SITE IS LOCATED ON THE EASTERN RIM OF THE ANIMAS RIVER VALLEY.
Colorado	Larimer	ROCKY MOUNTAIN NP	Fort Collins, CO		8	080690007	Rocky Mountain National Park, Long's Peak
Colorado	Larimer	3416 LA PORTE AVE.	Fort Collins, CO		8	080690011	FORT COLLINS - WEST
Colorado	Larimer	708 S. Mason St.	Fort Collins, CO		8	080691004	Fort Collins - CSU - S. Mason
Colorado	Mesa	865 Rapid Creek Rd.	Grand Junction, CO		8	080770020	Palisade-Water Treatment
Colorado	Montezuma	106 W. North Street			8	080830006	Cortez - Health Dept
Colorado	Montezuma	MESA VERDE NATIONAL PARK, COLORADO			8	080830101	Mesa Verde National Park, Resource Management Area
Colorado	Rio Blanco	Meecker, Colorado Plant Science Building			8	081030005	
Colorado	Rio Blanco	Rangely Colorado, Golf Course			8	081030006	Rangely, Golf Course
Colorado	Weld	3101 35TH AVE.	Greeley, CO	Denver-Aurora, CO	8	081230009	Greeley - Weld County Tower
Connecticut	Fairfield	Tods Driftway (see coordinates)	Bridgeport-Stamford-Norwalk, CT	New York-Newark, NY-NJ-CT-PA	1	090010017	Greenwich Point Park
Connecticut	Fairfield	White St at 8th Ave, parking garage roof	Bridgeport-Stamford-Norwalk, CT	New York-Newark, NY-NJ-CT-PA	1	090011123	Western Conn State Univ
Connecticut	Fairfield	USCG LIGHTHOUSE , PROSPECT STREET	Bridgeport-Stamford-Norwalk, CT	New York-Newark, NY-NJ-CT-PA	1	090013007	
Connecticut	Fairfield	SHERWOOD ISLAND STATE PARK	Bridgeport-Stamford-Norwalk, CT	New York-Newark, NY-NJ-CT-PA	1	090019003	Sherwood Island Connector (see coordinates)
Connecticut	Hartford	Remington Road (see coordinates)	Hartford-West Hartford-East Hartford, CT	Hartford-West Hartford, CT	1	090031003	McAuliffe Park
Connecticut	Litchfield	MOHAWK MOUNTAIN ROAD (see coordinates)	Torrington, CT	New York-Newark, NY-NJ-CT-PA	1	090050005	Mohawk Mt-Cornwall
Connecticut	Middlesex	CONN. VALLEY HOSP., SHEW HALL, EASTERN D	Hartford-West Hartford-East Hartford, CT	Hartford-West Hartford, CT	1	090070007	

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Connecticut	New Haven	IJAMES STREET	New Haven-Milford, CT	New York-Newark, NY-NJ-CT-PA	1	090090027	Criscuolo Park-New Haven
Connecticut	New Haven	Meigs Point, Hammonasset State Park	New Haven-Milford, CT	New York-Newark, NY-NJ-CT-PA	1	090099002	Hammonasset State Park
Connecticut	New London	141 Smith Street, Groton	Norwich-New London, CT	Hartford-West Hartford, CT	1	090110124	Fort Griswold Park
Connecticut	Tolland	ROUTE 190, SHENIPSIT STATE FOREST	Hartford-West Hartford-East Hartford, CT	Hartford-West Hartford, CT	1	090131001	
Connecticut	Windham	80 Ayers Rd, Abington, CT 06230	Worcester, MA-CT	Boston-Worcester-Providence, MA-RI-NH-CT	1	090159991	Abington
Delaware	Kent	STATE ROAD 384, KILLENS POND RD	Dover, DE	Philadelphia-Reading-Camden, PA-NJ-DE-MD	3	100010002	PROPERTY OF KILLENS POND STATE PARK; BEHIND FARM BUILDINGS
Delaware	New Castle	LUMS POND STATE PARK	Philadelphia-Camden-Wilmington, PA-NJ-DE-MD	Philadelphia-Reading-Camden, PA-NJ-DE-MD	3	100031007	
Delaware	New Castle	BRANDYWINE CREEK STATE PARK	Philadelphia-Camden-Wilmington, PA-NJ-DE-MD	Philadelphia-Reading-Camden, PA-NJ-DE-MD	3	100031010	BCSP
Delaware	New Castle	BELLEVUE STATE PARK	Philadelphia-Camden-Wilmington, PA-NJ-DE-MD	Philadelphia-Reading-Camden, PA-NJ-DE-MD	3	100031013	BELLEVUE STATE PARK, FIELD IN SE PORTION OF PARK
Delaware	New Castle	MLK BLVD AND JUSTISON ST.	Philadelphia-Camden-Wilmington, PA-NJ-DE-MD	Philadelphia-Reading-Camden, PA-NJ-DE-MD	3	100032004	MLK CORNER OF MLK BLVD AND JUSTISON ST
Delaware	Sussex	350 VIRGINIA AVE SEAFORD	Salisbury, MD-DE		3	100051002	Seaford Shipley State Service Center
Delaware	Sussex	UNIV. OF DE COLLEGE OF MARINE STUDIES	Salisbury, MD-DE		3	100051003	Lewes SPM SITE, NEAR UD ACID RAIN/MERCURY COLLECTORS
District Of Columbia	District of Columbia	2500 1ST STREET, N.W. WASHINGTON DC	Washington-Arlington-Alexandria, DC-VA-MD-WV	Washington-Baltimore-Arlington, DC-MD-VA-WV-PA	3	110010043	MCMILLAN NCore-PAMS
District Of Columbia	District of Columbia	301 Van Buren Street, N.W. Washington DC 20012	Washington-Arlington-Alexandria, DC-VA-MD-WV	Washington-Baltimore-Arlington, DC-MD-VA-WV-PA	3	110010050	Takoma Rec Center
Florida	Alachua	918 SE 119TH AVE	Gainesville, FL	Gainesville-Lake City, FL	4	120013011	LOCATED IN PAYNES PRAIRIE STATE PARK, NATURE AND WILDLIFE PRESERVE
Florida	Baker	HWY 90 OLUSTEE FOREST SERVICE OFFICE	Jacksonville, FL	Jacksonville-St. Marys-Palatka, FL-GA	4	120030002	OLUSTEE
Florida	Bay	4607 STATE PARK LN PCB,FL	Panama City, FL		4	120050006	ST.ANDREWS STATE PARK, PANAMA CITY BEACH
Florida	Brevard	401 FLORIDA AVE	Palm Bay-Melbourne-Titusville, FL		4	120090007	LOCATED IN CITY PARK ADJACENT TO SOFTBALL AND SOCCER FIELDS

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Florida	Brevard	400 SOUTH 4TH STREET	Palm Bay-Melbourne-Titusville, FL		4	120094001	
Florida	Broward	3211 College Ave, Davie, FL 33314	Miami-Fort Lauderdale-West Palm Beach, FL	Miami-Fort Lauderdale-Port St. Lucie, FL	4	120110033	Vista View Park
Florida	Broward	1951 NE 48TH ST	Miami-Fort Lauderdale-West Palm Beach, FL	Miami-Fort Lauderdale-Port St. Lucie, FL	4	120112003	Pompano Highlands
Florida	Broward	7000 N. OCEAN DRIVE	Miami-Fort Lauderdale-West Palm Beach, FL	Miami-Fort Lauderdale-Port St. Lucie, FL	4	120118002	
Florida	Collier	7800 IMMOKALEE ROAD NAPLES 34119	Naples-Immokalee-Marco Island, FL	Cape Coral-Fort Myers-Naples, FL	4	120210004	LAURAL OAKS ELEMENTARY
Florida	Columbia	751 SE SYCAMORE TERRACE	Lake City, FL	Gainesville-Lake City, FL	4	120230002	LOCATED AT LAKE CITY'S VETERAN'S DOMICILE
Florida	Duval	13333 LANIER RD.	Jacksonville, FL	Jacksonville-St. Marys-Palatka, FL-GA	4	120310077	
Florida	Duval	13600 William Davis Parkway.	Jacksonville, FL	Jacksonville-St. Marys-Palatka, FL-GA	4	120310100	
Florida	Escambia	ELLYSON INDUSTRIAL PARK-COPTER ROAD	Pensacola-Ferry Pass-Brent, FL		4	120330004	Ellyson Industrial Park
Florida	Escambia	NAS PENSACOLA	Pensacola-Ferry Pass-Brent, FL		4	120330018	
Florida	Flagler	208 Sawgrass Rd	Deltona-Daytona Beach-Ormond Beach, FL	Orlando-Deltona-Daytona Beach, FL	4	120350004	Flagler
Florida	Highlands	123 MAIN DRIVE VENUS FL 33960	Sebring, FL		4	120550003	ARCHBOLD BIOLOGICAL STATION PROPERTY. NORTH END OF LOOP
Florida	Hillsborough	2401 19th Avenue Northwest	Tampa-St. Petersburg-Clearwater, FL		4	120570081	
Florida	Hillsborough	COAST GUARD STATION DAVIS ISLAND	Tampa-St. Petersburg-Clearwater, FL		4	120571035	
Florida	Hillsborough	5121 GANDY BLVD	Tampa-St. Petersburg-Clearwater, FL		4	120571065	
Florida	Hillsborough	1167 NORTH DOVER ROAD	Tampa-St. Petersburg-Clearwater, FL		4	120573002	SYDNEY
Florida	Holmes	TRI CO AIRPORT RD (CO RD 162)			4	120590004	SITE IS ON TRI CO AIRPORT LAND IN A RURAL AREA
Florida	Indian River	Sebastian Inlet State Recreation Area, Vero Beach, FL 32963	Sebastian-Vero Beach, FL	Miami-Fort Lauderdale-Port St. Lucie, FL	4	120619991	Indian River Lagoon
Florida	Lake	1901 JOHNS LAKE RD	Orlando-Kissimmee-Sanford, FL	Orlando-Deltona-Daytona Beach, FL	4	120690002	LOCATED BY THE LAKE NOT RESRICTED
Florida	Lee	5505 ROSE GARDEN RD CAPE COREL FL 33914	Cape Coral-Fort Myers, FL	Cape Coral-Fort Myers-Naples, FL	4	120712002	INSIDE THE ROTARY PARK MAIN BLDG

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Florida	Lee	INTERSECTION OF SCHOOL ST AND BAY ST	Cape Coral-Fort Myers, FL	Cape Coral-Fort Myers-Naples, FL	4	120713002	THE SITE IS LOCATED AT THE BAY OAKS OPERATED BY THE LEE COUNTY
Florida	Leon	110 CENTURY PARK CIRCLE WEST	Tallahassee, FL	Tallahassee-Bainbridge, FL-GA	4	120730012	Tallahassee Community College
Florida	Liberty	Apalachicola National Forest, Bristol, FL 32321			4	120779991	Sumatra
Florida	Manatee	PORT MANATEE, REEDER RD & SOUTH DOCK ST.	North Port-Sarasota-Bradenton, FL	North Port-Sarasota, FL	4	120813002	
Florida	Manatee	5502 33RD AVE DRIVE W. (G T BRAY PARK)	North Port-Sarasota-Bradenton, FL	North Port-Sarasota, FL	4	120814012	G.T. BRAY PARK
Florida	Manatee	5511 39TH STREET EAST	North Port-Sarasota-Bradenton, FL	North Port-Sarasota, FL	4	120814013	39TH STREET SITE
Florida	Marion	SE 17TH STREET & SE 30TH AVENUE	Ocala, FL		4	120830003	LOCATED AT THE YMCA
Florida	Marion	692 NW 30TH AVE	Ocala, FL		4	120830004	LOCATED AT THE SHERIFF'S DEPT IMPOUND
Florida	Martin	950 SE MONTEREY RD	Port St. Lucie, FL	Miami-Fort Lauderdale-Port St. Lucie, FL	4	120850007	
Florida	Miami-Dade	ROSENSTIEL SCHOOL	Miami-Fort Lauderdale-West Palm Beach, FL	Miami-Fort Lauderdale-Port St. Lucie, FL	4	120860027	
Florida	Miami-Dade	19590 OLD CUTLER RD-PERDUE MED. CENTER	Miami-Fort Lauderdale-West Palm Beach, FL	Miami-Fort Lauderdale-Port St. Lucie, FL	4	120860029	
Florida	Okaloosa	720 Lovejoy Rd	Crestview-Fort Walton Beach-Destin, FL		4	120910002	
Florida	Orange	7055 WINEGARD RD., ORLANDO	Orlando-Kissimmee-Sanford, FL	Orlando-Deltona-Daytona Beach, FL	4	120950008	
Florida	Orange	MORRIS BLVD.	Orlando-Kissimmee-Sanford, FL	Orlando-Deltona-Daytona Beach, FL	4	120952002	WINTER PARK
Florida	Osceola	8706 WEST 192, KISSIMMEE	Orlando-Kissimmee-Sanford, FL	Orlando-Deltona-Daytona Beach, FL	4	120972002	OSCEOLA CO. FIRE STATION NEAR WALT DISNEY WORLD WEST GATE
Florida	Pasco	30908 WARDER ROAD	Tampa-St. Petersburg-Clearwater, FL		4	121010005	LOCATED ON FDOT COMPOUND
Florida	Pasco	3452 DARLINGTON RD., HOLIDAY	Tampa-St. Petersburg-Clearwater, FL		4	121012001	
Florida	Pinellas	2435 SHARKEY RD.CLEARWATER	Tampa-St. Petersburg-Clearwater, FL		4	121030004	
Florida	Pinellas	7200-22 AVENUE NORTH	Tampa-St. Petersburg-Clearwater, FL		4	121030018	Azalea Park
Florida	Pinellas	COUNTY ROAD 77 BOOKER CK PARK	Tampa-St. Petersburg-Clearwater, FL		4	121035002	
Florida	Polk	2727 SHEPHERD RD (JAMES W. SIKES ELM SCH	Lakeland-Winter Haven, FL		4	121056005	

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Florida	Polk	1015 SIKES BLVD., LAKELAND	Lakeland-Winter Haven, FL		4	121056006	Baptist Childrens' Home
Florida	St. Lucie	1420 E Midway rd	Port St. Lucie, FL	Miami-Fort Lauderdale-Port St. Lucie, FL	4	121110013	
Florida	Santa Rosa	1500 WOODLAWN WAY	Pensacola-Ferry Pass-Brent, FL		4	121130015	
Florida	Sarasota	450 MICINLEY DR.	North Port-Sarasota-Bradenton, FL	North Port-Sarasota, FL	4	121151005	
Florida	Sarasota	4570 17TH STREET	North Port-Sarasota-Bradenton, FL	North Port-Sarasota, FL	4	121151006	COUNTY PARK
Florida	Sarasota	201 SOUTH JACKSON ROAD VENICE FLORIDA	North Port-Sarasota-Bradenton, FL	North Port-Sarasota, FL	4	121152002	
Florida	Seminole	COUNTY HOMES RD. @ US 17-92	Orlando-Kissimmee-Sanford, FL	Orlando-Deltona-Daytona Beach, FL	4	121171002	Seminole Community College
Florida	Volusia	5200 SPRUCE CREEK RD.	Deltona-Daytona Beach-Ormond Beach, FL	Orlando-Deltona-Daytona Beach, FL	4	121272001	
Florida	Volusia	1185-A DUNN AVE., DAYTONA BEACH	Deltona-Daytona Beach-Ormond Beach, FL	Orlando-Deltona-Daytona Beach, FL	4	121275002	DAYTONA BLIND SERVICES
Florida	Wakulla	COUNTY RD 59	Tallahassee, FL	Tallahassee-Bainbridge, FL-GA	4	121290001	WITHIN ST. MARKS WILDLIFE REFUGE AT THE WORK STATION
Georgia	Bibb	Georgia Forestry Commission, 5645 Riggins Mill Road, Dry Branch, Georgia, 31020	Macon, GA	Macon-Warner Robins, GA	4	130210012	Macon SE
Georgia	Chatham	AMERICAN RED CROSS, 2500 E. PRESIDENT STREET, BLDG-A, SAVANNAH, GA, 31404	Savannah, GA	Savannah-Hinesville-Statesboro, GA	4	130510021	Savannah-E. President Street
Georgia	Chattooga	DNR Fish Hatchery, 231 Fish Hatchery Road, Summerville, Georgia, 30747	Summerville, GA	Rome-Summerville, GA	4	130550001	Summerville-DNR Fish Hatchery
Georgia	Clarke	FIRE STATION #7, 2350 BARNETT SHOALS RD, ATHENS, GA 30603	Athens-Clarke County, GA	Atlanta--Athens-Clarke County--Sandy Springs, GA	4	130590002	FIRE STATION # 7
Georgia	Cobb	GA National Guard, 1901 McCollum Parkway, Kennesaw, Georgia, 30144	Atlanta-Sandy Springs-Roswell, GA	Atlanta--Athens-Clarke County--Sandy Springs, GA	4	130670003	Kennesaw-National Guard
Georgia	Columbia	Riverside Park, 4431 Hardy McManus Road, Evans, Georgia, 30809	Augusta-Richmond County, GA-SC		4	130730001	Evans-Riverside Park
Georgia	Coweta	UNIVERSITY OF W. GA AT NEWNAN, 7 Solar Circle, Newnan, Georgia, 30265	Atlanta-Sandy Springs-Roswell, GA	Atlanta--Athens-Clarke County--Sandy Springs, GA	4	130770002	Newnan
Georgia	Dawson	GA FORESTRY COMMISSION, 4500 Georgia Highway 53 East, Dawsonville, Georgia, 30534	Atlanta-Sandy Springs-Roswell, GA	Atlanta--Athens-Clarke County--Sandy Springs, GA	4	130850001	Dawsonville, Georgia Forestry Commission
Georgia	DeKalb	2390-B Wildcat Road, Decatur GA 30034	Atlanta-Sandy Springs-Roswell, GA	Atlanta--Athens-Clarke County--Sandy Springs, GA	4	130890002	South DeKalb
Georgia	Douglas	Douglas County Water Authority, 7725 W. Strickland St., Douglasville, Georgia 30134	Atlanta-Sandy Springs-Roswell, GA	Atlanta--Athens-Clarke County--Sandy Springs, GA	4	130970004	W. Strickland Street
Georgia	Fulton	935 East Confederate Ave., Atlanta, Georgia, 30316	Atlanta-Sandy Springs-Roswell, GA	Atlanta--Athens-Clarke County--Sandy Springs, GA	4	131210055	Confederate Avenue
Georgia	Glynn	RISLEY MIDDLE SCHOOL, 2900 ALBANY ST., BRUNSWICK, GA 31520	Brunswick, GA		4	131270006	Risley Middle School

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Georgia	Gwinnett	Gwinnett Technical College, 5150 Sugarloaf Parkway, Lawrenceville, Georgia, 30043	Atlanta-Sandy Springs-Roswell, GA	Atlanta--Athens-Clarke County--Sandy Springs, GA	4	131350002	GWINNETT TECH
Georgia	Henry	Henry County Extension Office, 86 Work Camp Road, McDonough, Georgia, 30253	Atlanta-Sandy Springs-Roswell, GA	Atlanta--Athens-Clarke County--Sandy Springs, GA	4	131510002	McDonough-County Extension Office
Georgia	Murray	FORT MOUNTAIN, STATE HWY 52, COHUTTA OVERLOOK, CHATSWORTH, GA 30705	Dalton, GA	Chattanooga-Cleveland-Dalton, TN-GA-AL	4	132130003	Fort Mountain
Georgia	Muscogee	Columbus Airport, 3100 Thruway Drive, Columbus, Georgia, 31909	Columbus, GA-AL	Columbus-Auburn-Opelika, GA-AL	4	132150008	Columbus-Airport
Georgia	Paulding	King Farm, 160 Ralph King Path, Rockmart, Georgia, 30153	Atlanta-Sandy Springs-Roswell, GA	Atlanta--Athens-Clarke County--Sandy Springs, GA	4	132230003	Yorkville, King Farm
Georgia	Pike	Georgia Station Georgia Agricultural Experiment Station, Williamson, GA 30292	Atlanta-Sandy Springs-Roswell, GA	Atlanta--Athens-Clarke County--Sandy Springs, GA	4	132319991	Georgia Station
Georgia	Richmond	Bungalow Road Elem School, 2216 BUNGALOW RD, AUGUSTA GA 30906	Augusta-Richmond County, GA-SC		4	132450091	Bungalow Road
Georgia	Rockdale	Monastery of the Holy Spirit, 2625 Georgia 212, Conyers, Georgia, 30094	Atlanta-Sandy Springs-Roswell, GA	Atlanta--Athens-Clarke County--Sandy Springs, GA	4	132470001	Monastery
Georgia	Sumter	Leslie Community Center, N Bass S/E Allen St., Leslie, Georgia, 31764	Americus, GA		4	132611001	Leslie-Union High School
Hawaii	Honolulu	2052 LAUWILIWILI ST	Urban Honolulu, HI		9	150030010	Kapolei
Hawaii	Honolulu	1039 SAND ISLAND PARKWAY	Urban Honolulu, HI		9	150031004	Sand Island
Idaho	Ada	520 S. EAGLE ROAD, MERIDIAN	Boise City, ID	Boise City-Mountain Home-Ontario, ID-OR	10	160010010	St. Luke's Meridian
Idaho	Ada	401 EAST LINDEN ST. BOISE, ID 83706	Boise City, ID	Boise City-Mountain Home-Ontario, ID-OR	10	160010017	SAMPLER LOCATED ON SCHOOL ROOF-TOP IN SUBURBAN MOSTLY RESDNT'L AREA
Idaho	Butte	CRATERS OF THE MOON NATIONAL MON, IDAHO	Idaho Falls, ID	Idaho Falls-Rexburg-Blackfoot, ID	10	160230101	Craters of the Moon National Monument, Visitor Center
Illinois	Adams	1301 S. 48TH ST.	Quincy, IL-MO	Quincy-Hannibal, IL-MO	5	170010007	JOHN WOOD COMMUNITY COLLEGE
Illinois	Champaign	NORTH THOMAS ST.	Champaign-Urbana, IL		5	170190007	BOOKER T. WASHINGTON ES
Illinois	Champaign	TWP RD 500 E.	Champaign-Urbana, IL		5	170191001	ISWS CLIMATE STATION
Illinois	Clark	416 S. STATE ST. HWY 1/ WEST UNION			5	170230001	416 S. State St. Hwy 1- West Union
Illinois	Cook	4500 W. 123RD ST.	Chicago-Naperville-Elgin, IL-IN-WI	Chicago-Naperville, IL-IN-WI	5	170310001	VILLAGE GARAGE
Illinois	Cook	3300 E. CHELTENHAM PL.	Chicago-Naperville-Elgin, IL-IN-WI	Chicago-Naperville, IL-IN-WI	5	170310032	SOUTH WATER FILTRATION PLANT
Illinois	Cook	7801 LAWNSDALE	Chicago-Naperville-Elgin, IL-IN-WI	Chicago-Naperville, IL-IN-WI	5	170310076	COM ED MAINTENANCE BLDG

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Illinois	Cook	6545 W. HURLBUT ST.	Chicago-Naperville-Elgin, IL-IN-WI	Chicago-Naperville, IL-IN-WI	5	170311003	TAFT HS
Illinois	Cook	729 HOUSTON	Chicago-Naperville-Elgin, IL-IN-WI	Chicago-Naperville, IL-IN-WI	5	170311601	COOK COUNTY TRAILER
Illinois	Cook	4743 MANNHEIM RD.	Chicago-Naperville-Elgin, IL-IN-WI	Chicago-Naperville, IL-IN-WI	5	170313103	IEPA TRAILER
Illinois	Cook	1820 S. 51ST AVE.	Chicago-Naperville-Elgin, IL-IN-WI	Chicago-Naperville, IL-IN-WI	5	170314002	COOK COUNTY TRAILER
Illinois	Cook	9511 W. HARRISON ST	Chicago-Naperville-Elgin, IL-IN-WI	Chicago-Naperville, IL-IN-WI	5	170314007	REGIONAL OFFICE BUILDING
Illinois	Cook	750 DUNDEE ROAD	Chicago-Naperville-Elgin, IL-IN-WI	Chicago-Naperville, IL-IN-WI	5	170314201	NORTHBROOK WATER PLANT
Illinois	Cook	531 E. LINCOLN	Chicago-Naperville-Elgin, IL-IN-WI	Chicago-Naperville, IL-IN-WI	5	170317002	WATER PLANT
Illinois	DuPage	RT. 53	Chicago-Naperville-Elgin, IL-IN-WI	Chicago-Naperville, IL-IN-WI	5	170436001	MORTON ARBORETUM
Illinois	Effingham	ROUTE 45 SOUTH	Effingham, IL		5	170491001	CENTRAL JR HIGH
Illinois	Hamilton	STATE ROUTE 14			5	170650002	TEN MILE CREEK DNR OFFICE
Illinois	Jersey	LIBERTY ST. & COUNTY RD.	St. Louis, MO-IL	St. Louis-St. Charles-Farmington, MO-IL	5	170831001	ILLINI JR HIGH
Illinois	Jo Daviess	10952 E. Parker Rd, Stockton, IL 61085			5	170859991	Stockton
Illinois	Kane	665 DUNDEE RD.	Chicago-Naperville-Elgin, IL-IN-WI	Chicago-Naperville, IL-IN-WI	5	170890005	LARSEN JUNIOR HIGH
Illinois	Lake	ILLINOIS BEACH STATE PARK	Chicago-Naperville-Elgin, IL-IN-WI	Chicago-Naperville, IL-IN-WI	5	170971007	CAMP LOGAN TRAILER
Illinois	McHenry	FIRST ST. & THREE OAKS RD.	Chicago-Naperville-Elgin, IL-IN-WI	Chicago-Naperville, IL-IN-WI	5	171110001	CARY GROVE HS
Illinois	McLean	MAIN & GREGORY	Bloomington, IL	Bloomington-Pontiac, IL	5	171132003	ISU HARRIS PHYSICAL PLANT
Illinois	Macon	2200 N. 22ND	Decatur, IL		5	171150013	IEPA TRAILER
Illinois	Macoupin	HEATON & DUBOIS	St. Louis, MO-IL	St. Louis-St. Charles-Farmington, MO-IL	5	171170002	IEPA TRAILER
Illinois	Madison	409 MAIN ST.	St. Louis, MO-IL	St. Louis-St. Charles-Farmington, MO-IL	5	171190008	CLARA BARTON SCHOOL
Illinois	Madison	200 W. DIVISION	St. Louis, MO-IL	St. Louis-St. Charles-Farmington, MO-IL	5	171191009	SOUTHWEST CABLE TV
Illinois	Madison	54 N. WALCOTT	St. Louis, MO-IL	St. Louis-St. Charles-Farmington, MO-IL	5	171193007	WATER PLANT
Illinois	Madison	5403 State Road 160, Highland, IL 62249	St. Louis, MO-IL	St. Louis-St. Charles-Farmington, MO-IL	5	171199991	Alhambra
Illinois	Peoria	HURLBURT & MACARTHUR	Peoria, IL	Peoria-Canton, IL	5	171430024	FIRESTATION
Illinois	Peoria	508 E. GLEN AVE.	Peoria, IL	Peoria-Canton, IL	5	171431001	PEORIA HEIGHTS HS
Illinois	Randolph	HICKORY GROVE & FALLVIEW			5	171570001	IEPA TRAILER
Illinois	Rock Island	32 RODMAN AVE.	Davenport-Moline-Rock Island, IA-IL	Davenport-Moline, IA-IL	5	171613002	ROCK ISLAND ARSENAL

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Illinois	Saint Clair	13TH & TUDOR	St. Louis, MO-IL	St. Louis-St. Charles-Farmington, MO-IL	5	171630010	IEPA-RAPS TRAILER
Illinois	Sangamon	Illinois Building State Fairgrounds	Springfield, IL	Springfield-Jacksonville-Lincoln, IL	5	171670014	Illinois Building State Fairgrounds
Illinois	Will	36400 S. ESSEX RD.	Chicago-Naperville-Elgin, IL-IN-WI	Chicago-Naperville, IL-IN-WI	5	171971011	COM ED TRAINING CENTER
Illinois	Winnebago	1405 MAPLE AVE.	Rockford, IL	Rockford-Freeport-Rochelle, IL	5	172012001	MAPLE ELEMENTARY SCHOOL
Indiana	Allen	14600 AMSTUTZ RD., LEO HS	Fort Wayne, IN	Fort Wayne-Huntington-Auburn, IN	5	180030002	Leo High School
Indiana	Allen	2022 NORTH BEACON	Fort Wayne, IN	Fort Wayne-Huntington-Auburn, IN	5	180030004	Ft. Wayne- Beacon St.
Indiana	Bartholomew	Hope-Hauser Jr-Sr High School, 9404 N. 775 E.	Columbus, IN	Indianapolis-Carmel-Muncie, IN	5	180050007	Hope- Hauser Jr-Sr High School
Indiana	Boone	3900 E. 300 S, WHITESTOWN	Indianapolis-Carmel-Anderson, IN	Indianapolis-Carmel-Muncie, IN	5	180110001	Perry Worth ELEMENTRY SCHOOL, WEST OF WHITESTOWN
Indiana	Brown	4831 Helmsburg Rd.- Helmsburg	Indianapolis-Carmel-Anderson, IN	Indianapolis-Carmel-Muncie, IN	5	180130001	Helmsburg
Indiana	Carroll	481 S. 150 W. / FLORA AIRPORT	Lafayette-West Lafayette, IN	Lafayette-West Lafayette-Frankfort, IN	5	180150002	Flora-Flora Airport
Indiana	Clark	12500 St. Rd. 62-Charlestown State Park/ Indiana Armory	Louisville/Jefferson County, KY-IN	Louisville/Jefferson County-- Elizabethtown--Madison, KY-IN	5	180190008	Charlestown State Park- 1051.8 meters East of SR 62/ Indiana armory
Indiana	Delaware	700 W. STATE ST./ ALBANY ELEMENTARY	Muncie, IN	Indianapolis-Carmel-Muncie, IN	5	180350010	Albany- Albany Elem. Sch.
Indiana	Elkhart	705 INDIANA AVE./ BRISTOL ELEMENTARY	Elkhart-Goshen, IN	South Bend-Elkhart-Mishawaka, IN-MI	5	180390007	Bristol- Bristol Elem. Sch.
Indiana	Floyd	2230 GREEN VALLEY ROAD/GREEN VALLEY ELEMENTARY SCHOOL	Louisville/Jefferson County, KY-IN	Louisville/Jefferson County-- Elizabethtown--Madison, KY-IN	5	180431004	New Albany- Green Valley Elem. Sch.
Indiana	Greene	2500 S. 275 W, PLUMMER			5	180550001	Plummer, 2500 S. W- Citizens gas Plummer maintenance facility
Indiana	Hamilton	Noblesville- 9900 E. 191st St.	Indianapolis-Carmel-Anderson, IN	Indianapolis-Carmel-Muncie, IN	5	180570006	Our Lady of Grace- Noblesville
Indiana	Hendricks	Avon- 7203 E. US HIGHWAY 36, Avon Transportation Cntr.- 255 S. Avon Ave.(updated 2016)	Indianapolis-Carmel-Anderson, IN	Indianapolis-Carmel-Muncie, IN	5	180630004	AVON- 255 S. SR 267 (also 255 S. Avon Ave.) Avon, IN
Indiana	Huntington	423 WEST VINE STREET - ROANOKE	Huntington, IN	Fort Wayne-Huntington-Auburn, IN	5	180690002	Roanoke- Roanoke Elem. School
Indiana	Jackson	225 W & 300 N, BROWNSTOWN	Seymour, IN	Indianapolis-Carmel-Muncie, IN	5	180710001	Brownstown- 225 W & 200 N. Water facility
Indiana	Johnson	200 W. PEARL ST., TRAFALGAR	Indianapolis-Carmel-Anderson, IN	Indianapolis-Carmel-Muncie, IN	5	180810002	Indian Creek Elementary School in Trafalgar, DUE SOUTH OF INDIANAPOLIS
Indiana	Knox	Southwest Purdue Agricultural Center, Vincennes, IN 47591	Vincennes, IN		5	180839991	Vincennes

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Indiana	Lake	201 MISSISSIPPI ST., IITRI BUNKER	Chicago-Naperville-Elgin, IL-IN-WI	Chicago-Naperville, IL-IN-WI	5	180890022	Gary-IITRI/ 1219.5 meters east of Tennessee St.- old ammunition bunker
Indiana	Lake	1300 141 ST STREET	Chicago-Naperville-Elgin, IL-IN-WI	Chicago-Naperville, IL-IN-WI	5	180892008	HAMMOND CAAP-Hammond- 141st St.
Indiana	LaPorte	2011 E. LINCOLNWAY LaPorte- E. Lincolnway	Michigan City-La Porte, IN	Chicago-Naperville, IL-IN-WI	5	180910010	LAPORTE OZONE SITE AT WATER TREATMENT PLANT
Indiana	Madison	Emporia EAST ELEM. SCH., 893 E. US 36, PENDLETON	Indianapolis-Carmel-Anderson, IN	Indianapolis-Carmel-Muncie, IN	5	180950010	SCHOOL LOCATED ON THE SW CORNER OF US 36 AND IND 109
Indiana	Marion	5753 Glenn Rd.- FORT HARRISON STATE PARK	Indianapolis-Carmel-Anderson, IN	Indianapolis-Carmel-Muncie, IN	5	180970050	Indpls.- Ft. Harrison
Indiana	Marion	1321 SOUTH HARDING	Indianapolis-Carmel-Anderson, IN	Indianapolis-Carmel-Muncie, IN	5	180970057	Indpls- Harding St.
Indiana	Marion	NAVAL AVIONICS CENTER, 6125 E. 16TH ST.	Indianapolis-Carmel-Anderson, IN	Indianapolis-Carmel-Muncie, IN	5	180970073	Indpls.- E. 16th St.
Indiana	Marion	3120 E. 30TH ST., WASHINGTON PARK	Indianapolis-Carmel-Anderson, IN	Indianapolis-Carmel-Muncie, IN	5	180970078	Indpls- Washington Park/ in parking lot next to police station
Indiana	Marion	1650 Ludlow Ave.- Indianapolis- I 70	Indianapolis-Carmel-Anderson, IN	Indianapolis-Carmel-Muncie, IN	5	180970087	Indpls.- I 70
Indiana	Morgan	135 S. CHESTNUT, MONROVIA HIGH SCHOOL	Indianapolis-Carmel-Anderson, IN	Indianapolis-Carmel-Muncie, IN	5	181090005	Monrovia- Monrovia HS.
Indiana	Perry	19856 OLD ST RD 37/PERRY CENTRAL HS/ LEOPOLD			5	181230009	Leopold- Perry Central HS
Indiana	Porter	84 DIANA RD/ WATER TREATMENT PLANT	Chicago-Naperville-Elgin, IL-IN-WI	Chicago-Naperville, IL-IN-WI	5	181270024	Ogden Dunes- Water Treatment Plant
Indiana	Porter	1000 WESLEY ST./ VALPARAISO WATER DEPT.	Chicago-Naperville-Elgin, IL-IN-WI	Chicago-Naperville, IL-IN-WI	5	181270026	VALPARAISO
Indiana	Posey	2027 S. ST. PHILLIPS RD., Evansville	Evansville, IN-KY		5	181290003	ST. PHILLIPS- St. Phillips road CAAP trailer
Indiana	St. Joseph	25601 STATE RD. 4, North Liberty/ POTATO CREEK STATE PARK	South Bend-Mishawaka, IN-MI	South Bend-Elkhart-Mishawaka, IN-MI	5	181410010	Potato Creek State Park
Indiana	St. Joseph	2335 SHIELDS DR/ SOUTH BEND CAAP 2	South Bend-Mishawaka, IN-MI	South Bend-Elkhart-Mishawaka, IN-MI	5	181410015	South Bend-Shields Dr.
Indiana	St. Joseph	Granger- Beckley St. 12441 Beckley. St., Granger	South Bend-Mishawaka, IN-MI	South Bend-Elkhart-Mishawaka, IN-MI	5	181410016	Granger-Beckley St.
Indiana	Shelby	4740 W. 600 N, FAIRLAND	Indianapolis-Carmel-Anderson, IN	Indianapolis-Carmel-Muncie, IN	5	181450001	TRITON Middle SCHOOL, NORTH OF FAIRLAND
Indiana	Vanderburgh	14940 OLD STATE ROAD/ SCOTT ELEMENTARY	Evansville, IN-KY		5	181630013	Inglefield/ Scott School
Indiana	Vanderburgh	Evansville-Buena Vista-1110 W. Buena Vista Rd.	Evansville, IN-KY		5	181630021	Evansville- Buena Vista
Indiana	Vigo	Terre Haute- Lafayette Ave. 961 N. LAFAYETTE AVE.	Terre Haute, IN		5	181670018	TERRE HAUTE CAAP/ McLean High School

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Indiana	Vigo	7597 N. STEVENSON RD/ SANDCUT	Terre Haute, IN		5	181670024	Sandcut/ SITE LOCATED BY HOME BEHIND SHED.
Indiana	Wabash	Hamilton Rd, Lagro, IN 46941	Wabash, IN		5	181699991	Salamonie Reservoir
Indiana	Warrick	300 N. 1ST ST./ BOONVILLE HIGH SCHOOL	Evansville, IN-KY		5	181730008	Boonville- Boonville HS
Indiana	Warrick	5244 STATE RD 68/ TECUMSEH HIGH SCHOOL	Evansville, IN-KY		5	181730009	Lynnville- Tecumseh HS
Indiana	Warrick	DAYVILLE- 3488 EBLE RD, Newburgh	Evansville, IN-KY		5	181730011	Dayville
Iowa	Bremer	WAVERLY AIRPORT	Waterloo-Cedar Falls, IA		7	190170011	WAVERLY AIRPORT SITE
Iowa	Clinton	ROOSEVELT ST.	Clinton, IA	Davenport-Moline, IA-IL	7	190450021	CLINTON, RAINBOW PARK
Iowa	Harrison	206 Polk St, Pisgah, IA	Omaha-Council Bluffs, NE-IA	Omaha-Council Bluffs-Fremont, NE-IA	7	190850007	
Iowa	Harrison	1575 HWY 183	Omaha-Council Bluffs, NE-IA	Omaha-Council Bluffs-Fremont, NE-IA	7	190851101	PISGAH, HIGHWAY MAINTENANCE
Iowa	Linn	6301 KIRKWOOD BLVD SW (IOWA HALL)	Cedar Rapids, IA	Cedar Rapids-Iowa City, IA	7	191130028	KIRKWOOD
Iowa	Linn	408 E. LINN ST. COGGON, IOWA	Cedar Rapids, IA	Cedar Rapids-Iowa City, IA	7	191130033	COGGON ELEMENTARY SCHOOL BLDG. NORTHERN LIMITS OF LINN COUNTY
Iowa	Linn	500 11TH ST NW	Cedar Rapids, IA	Cedar Rapids-Iowa City, IA	7	191130040	Public Health
Iowa	Montgomery	2780 VIKING LAKE ROAD			7	191370002	VIKING LAKE STATE PARK
Iowa	Palo Alto	IOWA LAKES COMMUNITY COLLEGE			7	191471002	EMMETSBURG, IOWA LAKES COMMUNITY COLL.
Iowa	Polk	1907 CARPENTER, DES MOINES IOWA	Des Moines-West Des Moines, IA	Des Moines-Ames-West Des Moines, IA	7	191530030	CARPENTER
Iowa	Scott	SCOTT COUNTY PARK	Davenport-Moline-Rock Island, IA-IL	Davenport-Moline, IA-IL	7	191630014	SCOTT COUNTY PARK
Iowa	Scott	10TH ST. & VINE ST. DAVENPORT	Davenport-Moline-Rock Island, IA-IL	Davenport-Moline, IA-IL	7	191630015	DAVENPORT, JEFFERSON SCH.
Iowa	Story	105 GREENE STREET-CITY HALL	Ames, IA	Des Moines-Ames-West Des Moines, IA	7	191690011	SLATER CITY HALL
Iowa	Van Buren	24430 LACEY TRAIL			7	191770006	LAKE SUGEMA STATE PARK II
Iowa	Warren	1650 118TH AVENUE, INDIANOLA	Des Moines-West Des Moines, IA	Des Moines-Ames-West Des Moines, IA	7	191810022	GRAVEL ROAD IN LAKE AQUABI STATE PARK
Kansas	Johnson	13899 W 159TH	Kansas City, MO-KS	Kansas City-Overland Park-Kansas City, MO-KS	7	200910010	HERITAGE PARK

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Kansas	Leavenworth	2010 METROPOLITAN	Kansas City, MO-KS	Kansas City-Overland Park-Kansas City, MO-KS	7	201030003	Leavenworth
Kansas	Neosho	1500 WEST SEVENTH			7	201330003	CHANUTE
Kansas	Sedgwick	HEALTH DEPT., 1900 EAST 9TH ST.	Wichita, KS	Wichita-Arkansas City-Winfield, KS	7	201730010	WICHITA HD
Kansas	Sedgwick	12831 W. 117N	Wichita, KS	Wichita-Arkansas City-Winfield, KS	7	201730018	Sedgwick Ozone
Kansas	Shawnee	2501 RANDOLPH AVENUE	Topeka, KS		7	201770013	KNI
Kansas	Sumner	707 E 119TH ST SOUTH, PECK COMMUNITY BLDG	Wichita, KS	Wichita-Arkansas City-Winfield, KS	7	201910002	PECK
Kansas	Trego	CEDAR BLUFF RESERVOIR, PRONGHORN & MULEY			7	201950001	CEDAR BLUFF
Kansas	Wyandotte	1210 N. 10TH ST., JFK RECREATION CENTER	Kansas City, MO-KS	Kansas City-Overland Park-Kansas City, MO-KS	7	202090021	JFK
Kentucky	Bell	1420 DORCHESTER AVENUE	Middlesborough, KY		4	210130002	MIDDLESBORO
Kentucky	Boone	KY 338 & LOWER RIVER ROAD	Cincinnati, OH-KY-IN	Cincinnati-Wilmington-Maysville, OH-KY-IN	4	210150003	EAST BEND
Kentucky	Boyd	FIVCO HEALTH DEPARTMENT, 2924 HOLT STREET	Huntington-Ashland, WV-KY-OH	Charleston-Huntington-Ashland, WV-OH-KY	4	210190017	ASHLAND PRIMARY (FIVCO)
Kentucky	Bullitt	SECOND & CARPENTER STREETS	Louisville/Jefferson County, KY-IN	Louisville/Jefferson County--Elizabethtown--Madison, KY-IN	4	210290006	SHEPHERDSVILLE
Kentucky	Campbell	524A JOHN'S HILL ROAD	Cincinnati, OH-KY-IN	Cincinnati-Wilmington-Maysville, OH-KY-IN	4	210373002	NORTHERN KENTUCKY UNIVERSITY (NKU)
Kentucky	Carter	CAMP ROBERT WEBB, 1486 CAMP WEBB ROAD			4	210430500	GRAYSON LAKE
Kentucky	Christian	WILLIAMSON RESIDENCE, 10800 PILOT ROCK ROAD	Clarksville, TN-KY		4	210470006	HOPKINSVILLE
Kentucky	Daviess	716 PLEASANT VALLEY ROAD	Owensboro, KY		4	210590005	OWENSBORO PRIMARY
Kentucky	Edmonson	MAMMOTH CAVE NP - ALFRED COOK ROAD	Bowling Green, KY	Bowling Green-Glasgow, KY	4	210610501	Mammoth Cave National Park, Houchin Meadow
Kentucky	Fayette	FAYETTE COUNTY HEALTH DEPT, 650 NEWTOWN PIKE	Lexington-Fayette, KY	Lexington-Fayette--Richmond--Frankfort, KY	4	210670012	LEXINGTON PRIMARY
Kentucky	Greenup	SCOTT STREET & CENTER AVENUE	Huntington-Ashland, WV-KY-OH	Charleston-Huntington-Ashland, WV-OH-KY	4	210890007	WORTHINGTON
Kentucky	Hancock	2ND STREET & CAROLINE STREET	Owensboro, KY		4	210910012	LEWISPORT
Kentucky	Hardin	AMERICAN LEGION PARK, 801 NORTH MILES STREET	Elizabethtown-Fort Knox, KY	Louisville/Jefferson County--Elizabethtown--Madison, KY-IN	4	210930006	ELIZABETHTOWN
Kentucky	Henderson	BASKETT FIRE DEPARTMENT, 7492 DR. HODGE ROAD	Evansville, IN-KY		4	211010014	BASKETT
Kentucky	Jefferson	7601 BARDSTOWN RD, BATES ELEMENTARY SCH	Louisville/Jefferson County, KY-IN	Louisville/Jefferson County--Elizabethtown--Madison, KY-IN	4	211110027	Bates
Kentucky	Jefferson	7201 WATSON LN, WATSON LN ELEMENTARY SCH	Louisville/Jefferson County, KY-IN	Louisville/Jefferson County--Elizabethtown--Madison, KY-IN	4	211110051	Watson Lane
Kentucky	Jefferson	2730 CANNONS LANE, BOWMAN FIELD	Louisville/Jefferson County, KY-IN	Louisville/Jefferson County--Elizabethtown--Madison, KY-IN	4	211110067	CANNONS LANE

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Kentucky	Jessamine	KYTC MAINTENANCE GARAGE, 260 WILSON DRIVE	Lexington-Fayette, KY	Lexington-Fayette--Richmond--Frankfort, KY	4	211130001	NICHOLASVILLE
Kentucky	Livingston	LIVINGSTON CO ROAD DEPT, 706 STATE DRIVE	Paducah, KY-IL	Paducah-Mayfield, KY-IL	4	211390003	SMITHLAND
Kentucky	McCracken	JACKSON PURCHASE RECC, 2901 POWELL STREET	Paducah, KY-IL	Paducah-Mayfield, KY-IL	4	211451024	JACKSON PURCHASE (PADUCAH PRIMARY)
Kentucky	Morgan	State Highway 437, West Liberty, KY 41472			4	211759991	Crockett
Kentucky	Oldham	KYTC MAINTENANCE FACILITY, 1601 SOUTH HWY 393	Louisville/Jefferson County, KY-IN	Louisville/Jefferson County--Elizabethtown--Madison, KY-IN	4	211850004	BUCKNER
Kentucky	Perry	PERRY COUNTY HORSE PARK, 354 PERRY PARK ROAD			4	211930003	HAZARD
Kentucky	Pike	KYTC DISTRICT OFFICE, 109 LORAIN STREET			4	211950002	PIKEVILLE PRIMARY
Kentucky	Pulaski	SOMERSET GAS CO WAREHOUSE, 305 CLIFTY STREET	Somerset, KY		4	211990003	SOMERSET
Kentucky	Simpson	KYTC MAINTENANCE FACILITY, 573 HARDING ROAD			4	212130004	FRANKLIN
Kentucky	Trigg	5720 Old Dover Rd, Cadiz, KY 42211	Clarksville, TN-KY		4	212219991	Cadiz
Kentucky	Warren	226 SUNSET STREET	Bowling Green, KY	Bowling Green-Glasgow, KY	4	212270009	ED SPEAR PARK (SMITHS GROVE)
Kentucky	Washington	542 Wesley-Miller Rd, Harrodsburg, KY 40330			4	212299991	Mackville
Louisiana	Ascension	11153 Kling Road	Baton Rouge, LA		6	220050004	Dutchtown
Louisiana	Bossier	1425 Airport Drive	Shreveport-Bossier City, LA		6	220150008	Shreveport / Airport
Louisiana	Caddo	HAGOOD ROAD	Shreveport-Bossier City, LA		6	220170001	Dixie
Louisiana	Calcasieu	HIGHWAY 27 AND HIGHWAY 108	Lake Charles, LA		6	220190002	Carlyss
Louisiana	Calcasieu	2284 Paul Bellow Road	Lake Charles, LA		6	220190009	Vinton
Louisiana	East Baton Rouge	EAST END OF ASTER LANE	Baton Rouge, LA		6	220330003	LSU
Louisiana	East Baton Rouge	1061-A Leesville Ave	Baton Rouge, LA		6	220330009	Capitol
Louisiana	East Baton Rouge	11245 Port Hudson-Pride Rd. Zachary, La	Baton Rouge, LA		6	220330013	Pride
Louisiana	Iberville	65180 Belleview Road	Baton Rouge, LA		6	220470009	Bayou Plaquemine
Louisiana	Iberville	HIGHWAY 171, CARVILLE	Baton Rouge, LA		6	220470012	Carville
Louisiana	Jefferson	West Temple Pl	New Orleans-Metairie, LA	New Orleans-Metairie-Hammond, LA-MS	6	220511001	Kenner
Louisiana	Lafayette	646 Cajundome	Lafayette, LA	Lafayette-Opelousas-Morgan City, LA	6	220550007	Lafayette / USGS
Louisiana	Lafourche	Nicholls University Farm Highway 1	Houma-Thibodaux, LA		6	220570004	Thibodaux
Louisiana	Livingston	Highway 16, French Settlement	Baton Rouge, LA		6	220630002	French Settlement
Louisiana	Ouachita	5296 Southwest	Monroe, LA	Monroe-Ruston-Bastrop, LA	6	220730004	Monroe / Airport
Louisiana	Pointe Coupee	TED DAVIS RESIDENCE. HIGHWAY 415	Baton Rouge, LA		6	220770001	New Roads
Louisiana	St. Bernard	4101 Mistrot Dr. Meraux, LA 70075	New Orleans-Metairie, LA	New Orleans-Metairie-Hammond, LA-MS	6	220870004	Meraux
Louisiana	St. James	ST. JAMES COURTHOUSE, HWY 44 @ CANAPELLA	New Orleans-Metairie, LA	New Orleans-Metairie-Hammond, LA-MS	6	220930002	Convent
Louisiana	St. John the Baptist	Anthony F. Monica Street	New Orleans-Metairie, LA	New Orleans-Metairie-Hammond, LA-MS	6	220950002	Garyville

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Louisiana	St. Tammany	1421 Hwy 22 W, Madison Ville, LA 70447	New Orleans-Metairie, LA	New Orleans-Metairie-Hammond, LA-MS	6	221030002	Madisonville
Louisiana	West Baton Rouge	1005 Northwest Drive, Port Allen	Baton Rouge, LA		6	221210001	Port Allen
Maine	Androscoggin	ROUTE 9, DURHAM	Lewiston-Auburn, ME	Portland-Lewiston-South Portland, ME	1	230010014	DURHAM FIRE STATION
Maine	Aroostook	8 NORTHERN ROAD, PRESQUE ISLE, ME 04769			1	230031100	MICMAC HEALTH DEPARTMENT
Maine	Aroostook	45 Radar Rd, Ashland, ME 04732			1	230039991	Ashland
Maine	Cumberland	TWO LIGHTS STATE PARK, Near the Obsevation Tower	Portland-South Portland, ME	Portland-Lewiston-South Portland, ME	1	230052003	CETL - Cape Elizabeth Two Lights (State Park)
Maine	Hancock	TOP OF CADILLAC MOUNTAIN			1	230090102	TOP OF CADILLAC MTN (FENCED ENCLOSURE)
Maine	Hancock	567 Eagle Lake Road			1	230090103	MCFARLAND HILL Air Pollutant Research Site
Maine	Kennebec	14 Pray Street	Augusta-Waterville, ME		1	230112005	Gardiner, Pray Street School (GPSS)
Maine	Knox	PORT CLYDE, MARSHALL POINT LIGHTHOUSE			1	230130004	Marshall Point Lighthouse
Maine	Penobscot	SUMMIT OF RIDER BLUFF (WLBZ TRANSMITTER)	Bangor, ME		1	230194008	WLBZ TV Transmitter Building - Summit of Rider Bluff
Maine	Washington	PUBLIC LANDING, Jonesport			1	230290019	Harbor Masters Office; Jonesport Public Landing
Maine	Washington	184 COUNTY ROAD			1	230290032	
Maine	York	34 Town Farm Road, HOLLIS	Portland-South Portland, ME	Portland-Lewiston-South Portland, ME	1	230310038	WBFD - West Buxton (Hollis) Fire Department
Maine	York	Rt. 11, Shapleigh Ball Park, West Newfield Road, Shapleigh, Maine	Portland-South Portland, ME	Portland-Lewiston-South Portland, ME	1	230310040	SBP - Shapleigh Ball Park
Maine	York	OCEAN AVE / PARSONS WAY, KENNEBUNKPORT	Portland-South Portland, ME	Portland-Lewiston-South Portland, ME	1	230312002	KPW - Kennebunkport Parson'd Way
Maryland	Baltimore	Padonia Elementary School, 9834 Greenside Drive	Baltimore-Columbia-Towson, MD	Washington-Baltimore-Arlington, DC-MD-VA-WV-PA	3	240051007	Padonia
Maryland	Baltimore	600 Dorsey Avenue	Baltimore-Columbia-Towson, MD	Washington-Baltimore-Arlington, DC-MD-VA-WV-PA	3	240053001	Essex
Maryland	Calvert	350 Stafford Road	Washington-Arlington-Alexandria, DC-VA-MD-WV	Washington-Baltimore-Arlington, DC-MD-VA-WV-PA	3	240090011	Calvert
Maryland	Carroll	1300 W. Old Liberty Road	Baltimore-Columbia-Towson, MD	Washington-Baltimore-Arlington, DC-MD-VA-WV-PA	3	240130001	South Carroll
Maryland	Cecil	Fair Hill Natural Resource Mgmt Area, 4600 Telegraph Road	Philadelphia-Camden-Wilmington, PA-NJ-DE-MD	Philadelphia-Reading-Camden, PA-NJ-DE-MD	3	240150003	Fair Hill Natural Resource Management Area
Maryland	Charles	14320 Oaks Road	Washington-Arlington-Alexandria, DC-VA-MD-WV	Washington-Baltimore-Arlington, DC-MD-VA-WV-PA	3	240170010	Southern Maryland
Maryland	Dorchester	University of Maryland for Environmental and Estuarine Studies	Cambridge, MD	Washington-Baltimore-Arlington, DC-MD-VA-WV-PA	3	240190004	Horn Point

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Maryland	Dorchester	Blackwater National Wildlife Refuge, Cambridge, MD 21613	Cambridge, MD	Washington-Baltimore-Arlington, DC-MD-VA-WV-PA	3	240199991	Blackwater NWR
Maryland	Frederick	Frederick County Airport ,180 E Airport Drive	Washington-Arlington-Alexandria, DC-VA-MD-WV	Washington-Baltimore-Arlington, DC-MD-VA-WV-PA	3	240210037	Frederick Airport
Maryland	Garrett	Piney Run, Frostburg Reservoir, Finzel			3	240230002	Piney Run
Maryland	Harford	Edgewood Chemical Biological Center (APG), Waehli Road	Baltimore-Columbia-Towson, MD	Washington-Baltimore-Arlington, DC-MD-VA-WV-PA	3	240251001	Edgewood
Maryland	Harford	3560 Aldino Road	Baltimore-Columbia-Towson, MD	Washington-Baltimore-Arlington, DC-MD-VA-WV-PA	3	240259001	Aldino
Maryland	Kent	Millington Wildlife Management Area, Massey - Maryland Line Road (Route 330)			3	240290002	Millington
Maryland	Montgomery	Lathrop E. Smith Environmental Education Center, 5110 Meadows Lane	Washington-Arlington-Alexandria, DC-VA-MD-WV	Washington-Baltimore-Arlington, DC-MD-VA-WV-PA	3	240313001	Rockville
Maryland	Prince George's	Howard University's Beltsville Laboratory, 12003 Old Baltimore Pike	Washington-Arlington-Alexandria, DC-VA-MD-WV	Washington-Baltimore-Arlington, DC-MD-VA-WV-PA	3	240330030	HU-Beltsville
Maryland	Prince George's	PG County Equestrian Center, 14900 Pennsylvania Ave.	Washington-Arlington-Alexandria, DC-VA-MD-WV	Washington-Baltimore-Arlington, DC-MD-VA-WV-PA	3	240338003	PG Equestrian Center
Maryland	Prince George's	Powder Mill Rd, Laurel, MD 20708	Washington-Arlington-Alexandria, DC-VA-MD-WV	Washington-Baltimore-Arlington, DC-MD-VA-WV-PA	3	240339991	Beltsville
Maryland	Washington	MD Correctional Institution 18530 Roxbury Road	Hagerstown-Martinsburg, MD-WV	Washington-Baltimore-Arlington, DC-MD-VA-WV-PA	3	240430009	Hagerstown
Maryland	Baltimore (City)	Furley E.S. Recreational Center, 4633 Furley Avenue.	Baltimore-Columbia-Towson, MD	Washington-Baltimore-Arlington, DC-MD-VA-WV-PA	3	245100054	Furley
Massachusetts	Barnstable	FOX BOTTOM AREA	Barnstable Town, MA	Boston-Worcester-Providence, MA-RI-NH-CT	1	250010002	TRURO NATIONAL SEASHORE
Massachusetts	Bristol	659 GLOBE ST	Providence-Warwick, RI-MA	Boston-Worcester-Providence, MA-RI-NH-CT	1	250051004	FALL RIVER
Massachusetts	Bristol	30 SCHOOL ST - HASTINGS MIDDLE SCHOOL	Providence-Warwick, RI-MA	Boston-Worcester-Providence, MA-RI-NH-CT	1	250051006	FAIRHAVEN2
Massachusetts	Dukes	1 HERRING CREEK RD, AQUINNAH(WAMPANOAG TRIBAL SITE)	Vineyard Haven, MA		1	250070001	1 HERRING CREEK RD, AQUINNAH (WAMPANOAG TRIBAL SITE)
Massachusetts	Essex	390 PARKLAND	Boston-Cambridge-Newton, MA-NH	Boston-Worcester-Providence, MA-RI-NH-CT	1	250092006	LYNN WATER TREATMENT PLANT
Massachusetts	Essex	HARBOR STREET	Boston-Cambridge-Newton, MA-NH	Boston-Worcester-Providence, MA-RI-NH-CT	1	250094005	NEWBURYPORT HARBOR ST PARKING LOT
Massachusetts	Essex	685 WASHINGTON STREET	Boston-Cambridge-Newton, MA-NH	Boston-Worcester-Providence, MA-RI-NH-CT	1	250095005	CONSENTINO SCHOOL.
Massachusetts	Franklin	VETERANS FIELD	Greenfield Town, MA	Springfield-Greenfield Town, MA	1	250112005	Greenfield 16 Barr Ave
Massachusetts	Hampden	ANDERSON RD AFB	Springfield, MA	Springfield-Greenfield Town, MA	1	250130008	WESTOVER AFB
Massachusetts	Hampshire	QUABBIN SUMMIT	Springfield, MA	Springfield-Greenfield Town, MA	1	250154002	QUABBIN RES
Massachusetts	Middlesex	11 TECHNOLOGY DRIVE, EPA NEW ENGLAND REGIONAL LABORATORY	Boston-Cambridge-Newton, MA-NH	Boston-Worcester-Providence, MA-RI-NH-CT	1	250170009	USEPA REGION 1 LAB

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Massachusetts	Norfolk	695 HILLSIDE ST Blue Hill Observatory	Boston-Cambridge-Newton, MA-NH	Boston-Worcester-Providence, MA-RI-NH-CT	1	250213003	BLUE HILL OBSERVATORY
Massachusetts	Plymouth	170 Clinton Street(Buckley)	Boston-Cambridge-Newton, MA-NH	Boston-Worcester-Providence, MA-RI-NH-CT	1	250230005	Brockton Buckley
Massachusetts	Suffolk	HARRISON AVE	Boston-Cambridge-Newton, MA-NH	Boston-Worcester-Providence, MA-RI-NH-CT	1	250250042	DUDLEY SQUARE ROXBURY
Massachusetts	Worcester	375 AIRPORT DRIVE	Worcester, MA-CT	Boston-Worcester-Providence, MA-RI-NH-CT	1	250270015	WORCESTER AIRPORT
Massachusetts	Worcester	366 E HARTFORD DR	Worcester, MA-CT	Boston-Worcester-Providence, MA-RI-NH-CT	1	250270024	UXBRIDGE
Michigan	Allegan	966 W 32ND	Holland, MI	Grand Rapids-Wyoming-Muskegon, MI	5	260050003	Holland
Michigan	Benzie	1060 WEST ST., BENZONIA TWP.	Traverse City, MI		5	260190003	
Michigan	Berrien	PAW PAW WWTP, 4689 DEFIELD RD.	Niles-Benton Harbor, MI	South Bend-Elkhart-Mishawaka, IN-MI	5	260210014	Coloma
Michigan	Cass	ROSS BEATTY HIGH SCHOOL, 22721 DIAMOND COVE ST.	South Bend-Mishawaka, IN-MI	South Bend-Elkhart-Mishawaka, IN-MI	5	260270003	Cassopolis
Michigan	Chippewa	LK SUPERIOR STATE UNIV,650 W EASTERDAY A	Sault Ste. Marie, MI		5	260330901	NORTH OF EASTERDAY AVENUE
Michigan	Clinton	8562 E STOLL RD	Lansing-East Lansing, MI	Lansing-East Lansing-Owosso, MI	5	260370001	ROSE LAKE, STOLL RD.(8562 E.)
Michigan	Genesee	WHALEY PARK, 3610 IOWA	Flint, MI	Detroit-Warren-Ann Arbor, MI	5	260490021	
Michigan	Genesee	LAKEVILLE MIDDLE SCHOOL, G11107 WASHBURN RD	Flint, MI	Detroit-Warren-Ann Arbor, MI	5	260492001	Otisville
Michigan	Huron	1172 S.M25,SAND BEACH TWP.			5	260630007	RURAL THUMB AREA OZONE SITE
Michigan	Ingham	220 N PENNSYLVANIA	Lansing-East Lansing, MI	Lansing-East Lansing-Owosso, MI	5	260650012	
Michigan	Kalamazoo	FAIRGROUNDS, 1400 OLMSTEAD RD	Kalamazoo-Portage, MI	Kalamazoo-Battle Creek-Portage, MI	5	260770008	KALAMAZOO FAIRGROUNDS
Michigan	Kent	1179 MONROE NW	Grand Rapids-Wyoming, MI	Grand Rapids-Wyoming-Muskegon, MI	5	260810020	GR-Monroe
Michigan	Kent	10300 14 MILE RD NE #B	Grand Rapids-Wyoming, MI	Grand Rapids-Wyoming-Muskegon, MI	5	260810022	APPROXIMATELY 1/4 MILE SOUTH OF 14 MILE RD
Michigan	Lenawee	6792 RAISEN CENTER HIGHWAY	Adrian, MI	Detroit-Warren-Ann Arbor, MI	5	260910007	6792 RAISIN CENTER HWY, LENAWEE CO.RD.COMM.OWNER, TECUMSEH
Michigan	Macomb	57700 GRATIOT	Detroit-Warren-Dearborn, MI	Detroit-Warren-Ann Arbor, MI	5	260990009	New Haven
Michigan	Macomb	WARREN FIRE STATION 29900 HOOVER AT COMMON	Detroit-Warren-Dearborn, MI	Detroit-Warren-Ann Arbor, MI	5	260991003	
Michigan	Manistee	3031 DOMRES RD.			5	261010922	
Michigan	Mason	525 W US10	Ludington, MI		5	261050007	LOCATED 550 FT NORTH OF US10
Michigan	Missaukee	1769 S JEFFS RD	Cadillac, MI		5	261130001	LOCATED ABOUT 1/4 MILE WEST OF SITE

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Michigan	Muskegon	1340 GREEN CREEK ROAD	Muskegon, MI	Grand Rapids-Wyoming-Muskegon, MI	5	261210039	
Michigan	Oakland	13701 OAK PARK BLVD.	Detroit-Warren-Dearborn, MI	Detroit-Warren-Ann Arbor, MI	5	261250001	Oak Park
Michigan	Ottawa	6981 28TH AVE. GEORGETOWN TWP.	Grand Rapids-Wyoming, MI	Grand Rapids-Wyoming-Muskegon, MI	5	261390005	Jenison
Michigan	St. Clair	2525 DOVE RD	Detroit-Warren-Dearborn, MI	Detroit-Warren-Ann Arbor, MI	5	261470005	Port Huron
Michigan	Schoolcraft	SENEY NAT'L WILDLIFE REFUGE, HCR2, BOX 1			5	261530001	Seney
Michigan	Tuscola	1821 E. Dickerson Rd, Unionville, MI 48767			5	261579991	Unionville
Michigan	Washtenaw	555 TOWNER ST	Ann Arbor, MI	Detroit-Warren-Ann Arbor, MI	5	261610008	TOWNER ST, SOUTH; 2 LANE RESIDENTIAL - HOSPITAL
Michigan	Washtenaw	10070 Strawberry Lake Rd, Dexter, MI 48130	Ann Arbor, MI	Detroit-Warren-Ann Arbor, MI	5	261619991	Ann Arbor
Michigan	Wayne	14700 GODDARD	Detroit-Warren-Dearborn, MI	Detroit-Warren-Ann Arbor, MI	5	261630001	Allen Park
Michigan	Wayne	11600 EAST SEVEN MILE ROAD	Detroit-Warren-Dearborn, MI	Detroit-Warren-Ann Arbor, MI	5	261630019	East 7 Mile
Michigan	Wexford	10637 S 9 Rd, Cadillac, MI 49601	Cadillac, MI		5	261659991	Hoxeyville
Minnesota	Anoka	2660 Fawn Rd.	Minneapolis-St. Paul-Bloomington, MN-WI	Minneapolis-St. Paul, MN-WI	5	270031001	Cedar Creek
Minnesota	Anoka	9399 Lima St	Minneapolis-St. Paul-Bloomington, MN-WI	Minneapolis-St. Paul, MN-WI	5	270031002	Anoka Airport
Minnesota	Becker	26624 NORTH TOWER ROAD			5	270052013	Detroit Lakes
Minnesota	Crow Wing	16384 AIRPORT RD	Brainerd, MN		5	270353204	Brainerd Airport
Minnesota	Goodhue	1235 HIGHWAY 19	Red Wing, MN	Minneapolis-St. Paul, MN-WI	5	270495302	Stanton Air Field
Minnesota	Hennepin	1444 E 18th St	Minneapolis-St. Paul-Bloomington, MN-WI	Minneapolis-St. Paul, MN-WI	5	270530962	Minneapolis Near Roadway NOx
Minnesota	Lake	Fernberg Road			5	270750005	Fernberg Road
Minnesota	Lyon	WEST HIGHWAY 19	Marshall, MN		5	270834210	Marshall Airport
Minnesota	Mille Lacs	16687 Shaw-Bosh-Kung Drive	Minneapolis-St. Paul-Bloomington, MN-WI	Minneapolis-St. Paul, MN-WI	5	270953051	Mille Lacs
Minnesota	Olmsted	1801 9th Ave. SE	Rochester, MN	Rochester-Austin, MN	5	271095008	Ben Franklin School
Minnesota	Saint Louis	VOYAGEURS NATIONAL PARK	Duluth, MN-WI		5	271370034	VOYAGEURS NATIONAL PARK, NEAR SULLIVAN BAY
Minnesota	Saint Louis	1202 EAST UNIVERSITY CIRCLE	Duluth, MN-WI		5	271377550	WDSE
Minnesota	Scott	917 DAKOTA ST.	Minneapolis-St. Paul-Bloomington, MN-WI	Minneapolis-St. Paul, MN-WI	5	271390505	Shakopee
Minnesota	Stearns	1321 University Dr.	St. Cloud, MN	Minneapolis-St. Paul, MN-WI	5	271453052	Talahi School
Minnesota	Washington	St. Croix Trail N	Minneapolis-St. Paul-Bloomington, MN-WI	Minneapolis-St. Paul, MN-WI	5	271636016	
Minnesota	Wright	101 Central Ave. W.	Minneapolis-St. Paul-Bloomington, MN-WI	Minneapolis-St. Paul, MN-WI	5	271713201	St. Michael
Mississippi	Bolivar	213 N. BAYOU AVE.	Cleveland, MS	Cleveland-Indianola, MS	4	280110001	Cleveland
Mississippi	DeSoto	5 EAST SOUTH ST. (HERNANDO)	Memphis, TN-MS-AR	Memphis-Forrest City, TN-MS-AR	4	280330002	Hernando
Mississippi	Hancock	400 Baltic St	Gulfport-Biloxi-Pascagoula, MS		4	280450003	Waveland
Mississippi	Harrison	47 Maple Street	Gulfport-Biloxi-Pascagoula, MS		4	280470008	Gulfport Youth Court

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Mississippi	Hinds	232 East Woodrow Wilson Drive	Jackson, MS	Jackson-Vicksburg-Brookhaven, MS	4	280490020	Jackson NCORE
Mississippi	Jackson	Hospital Road at Co. Health Dept.	Gulfport-Biloxi-Pascagoula, MS		4	280590006	Pascagoula
Mississippi	Lauderdale	Hwy 19 and 53rd Ave	Meridian, MS		4	280750003	Meridian
Mississippi	Lee	West Jackson at Tupelo Airport	Tupelo, MS		4	280810005	TUPELO AIRPORT NEAR OLD NWS OFFICE
Mississippi	Yalobusha	Jamie L. Whitten Plant Materials Center, Coffeetown, MS 38922			4	281619991	Coffeetown
Missouri	Andrew	SAVANAH: 11796 Highway 71, Savannah, MO 64485	St. Joseph, MO-KS	Kansas City-Overland Park-Kansas City, MO-KS	7	290030001	Savannah
Missouri	Boone	FINGER LAKES: 1505 East Peabody Road, Columbia, MO 65202	Columbia, MO	Columbia-Moberly-Mexico, MO	7	290190011	Finger Lakes
Missouri	Callaway	NEW BLOOMFIELD: 2625 Meadow Lane, New Bloomfield, MO, 65063	Jefferson City, MO		7	290270002	New Bloomfield
Missouri	Cass	RICHARDS GEBEUR - SOUTH: 1802 E 203rd Street, Belton, MO 64012	Kansas City, MO-KS	Kansas City-Overland Park-Kansas City, MO-KS	7	290370003	Richard Gebaur-South
Missouri	Cedar	EL DORADO SPRINGS: Highway 97 & Barnes Road, El Dorado Springs, 64744			7	290390001	El Dorado Springs
Missouri	Clay	WATKINS MILL STATE PARK: Watkins Mill Road, Lawson, MO 64062	Kansas City, MO-KS	Kansas City-Overland Park-Kansas City, MO-KS	7	290470003	Watkins Mill State Park
Missouri	Clay	LIBERTY: Highway & County Home Road, Liberty, 64068	Kansas City, MO-KS	Kansas City-Overland Park-Kansas City, MO-KS	7	290470005	Liberty
Missouri	Clay	ROCKY CREEK: 13131 Highway 169 NE, Smithville, MO 64089	Kansas City, MO-KS	Kansas City-Overland Park-Kansas City, MO-KS	7	290470006	Rocky Creek
Missouri	Clinton	TRIMBLE: 7536 SW O Highway, Trimble, MO 64492	Kansas City, MO-KS	Kansas City-Overland Park-Kansas City, MO-KS	7	290490001	Trimble
Missouri	Greene	HILLCREST HIGH SCHOOL: 3319 N Grant, Springfield, MO 65803	Springfield, MO	Springfield-Branson, MO	7	290770036	Hillcrest High School
Missouri	Greene	FELLOWS LAKE: 4208 E Farm Road 66, Springfield, MO 65803	Springfield, MO	Springfield-Branson, MO	7	290770042	Fellows Lake
Missouri	Jasper	ALBA: 20400 Millwood Road, Alba, MO64755	Joplin, MO	Joplin-Miami, MO-OK	7	290970004	Alba
Missouri	Jefferson	ARNOLD WEST: 1709 Lonedell Drive, Arnold, MO 63010	St. Louis, MO-IL	St. Louis-St. Charles-Farmington, MO-IL	7	290990019	Arnold West
Missouri	Lincoln	FOLEY: #7 Wild Horse, Foley, MO 63347	St. Louis, MO-IL	St. Louis-St. Charles-Farmington, MO-IL	7	291130003	Foley
Missouri	Monroe	MARK TWAIN STATE PARK: 20057 State Park Office Road, Stoutville, MO 65283			7	291370001	MTSP
Missouri	Perry	FARRAR: County Road 342, Farrar, MO 63746			7	291570001	
Missouri	Saint Charles	WEST ALTON: General Electric Store, Highway 94, West Alton, MO 63386	St. Louis, MO-IL	St. Louis-St. Charles-Farmington, MO-IL	7	291831002	West Alton
Missouri	Saint Charles	ORCHARD FARM: 2165 Highway V, St. Charles, MO 63301	St. Louis, MO-IL	St. Louis-St. Charles-Farmington, MO-IL	7	291831004	Orchard Farm
Missouri	Sainte Genevieve	BONNE TERRE: 15797 Highway D, Bonne Terre, MO 63628			7	291860005	Bonne Terre

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Missouri	Saint Louis	PACIFIC: 18701 Old Highway 66, Pacific, MO 63039	St. Louis, MO-IL	St. Louis-St. Charles-Farmington, MO-IL	7	291890005	Pacific
Missouri	Saint Louis	MARYLAND HEIGHTS: 13044 Marine Avenue, Maryland Heights, MO 63146	St. Louis, MO-IL	St. Louis-St. Charles-Farmington, MO-IL	7	291890014	Maryland Heights
Missouri	Taney	BRANSON: 251 S.W. Outer Rd, Branson, MO 65616	Branson, MO	Springfield-Branson, MO	7	292130004	Branson
Missouri	St. Louis City	BLAIR STREET: 3247 Blair Street, St. Louis, MO 63107	St. Louis, MO-IL	St. Louis-St. Charles-Farmington, MO-IL	7	295100085	Blair Street
Montana	Fergus	303 East Aztec Drive Lewistown MT 59457			8	300270006	Lewistown
Montana	Flathead	GLACIER NATIONAL PARK	Kalispell, MT		8	300298001	GLACIER NATIONAL PARK
Montana	Lewis and Clark	I-15 Exit 209, then Sperry Dr.	Helena, MT		8	300490004	NCORE - Sieben Flats
Montana	Missoula	BOYD PARK, 3100 WASHBURN Rd.	Missoula, MT		8	300630024	MSLA Boyd Park
Montana	Phillips	2309 Short Oil Road, Malta, MT 59538			8	300710010	Malta
Montana	Powder River	Big Powder River Road East			8	300750001	BROADUS
Montana	Richland	Corner Cnty Roads 335 and 131			8	300830001	Sidney Oil Field
Montana	Rosebud	SR 566, 3 Miles N of Birney			8	300870001	Birney - Tongue river
Nebraska	Douglas	DOUGLAS COUNTY HOSP 42ND & WOOLWORTH	Omaha-Council Bluffs, NE-IA	Omaha-Council Bluffs-Fremont, NE-IA	7	310550019	4102 Woolworth Ave. on Healthcenter Warehouse
Nebraska	Douglas	2411 O ST.	Omaha-Council Bluffs, NE-IA	Omaha-Council Bluffs-Fremont, NE-IA	7	310550028	
Nebraska	Knox	State Spur 54D, Niobrara, NE 68760			7	311079991	Santee Sioux
Nebraska	Lancaster	FIRST & MAPLE , DAVEY	Lincoln, NE	Lincoln-Beatrice, NE	7	311090016	
Nevada	Churchill	280 SOUTH RUSSELL STREET	Fallon, NV		9	320010002	FALLON - WEST END ELEMENTARY SCHOOL
Nevada	Clark	12101 Hwy 91, Nevada Las Vegas, NV 89165	Las Vegas-Henderson-Paradise, NV	Las Vegas-Henderson, NV-AZ	9	320030022	Apex
Nevada	Clark	465 E. OLD MILL ROAD, MESQUITE, NV	Las Vegas-Henderson-Paradise, NV	Las Vegas-Henderson, NV-AZ	9	320030023	Mesquite
Nevada	Clark	4525 NEW FOREST DRIVE	Las Vegas-Henderson-Paradise, NV	Las Vegas-Henderson, NV-AZ	9	320030043	Paul Meyer
Nevada	Clark	7701 DUCHARME AVE	Las Vegas-Henderson-Paradise, NV	Las Vegas-Henderson, NV-AZ	9	320030071	Walter Johnson
Nevada	Clark	333 PAVILION CENTER DRIVE	Las Vegas-Henderson-Paradise, NV	Las Vegas-Henderson, NV-AZ	9	320030073	Palo Verde
Nevada	Clark	6651 W. AZURE AVE	Las Vegas-Henderson-Paradise, NV	Las Vegas-Henderson, NV-AZ	9	320030075	JOE NEAL
Nevada	Clark	4250 Karen Ave	Las Vegas-Henderson-Paradise, NV	Las Vegas-Henderson, NV-AZ	9	320030540	Jerome Mack-NCORE
Nevada	Clark	1005 INDUSTRIAL ROAD	Las Vegas-Henderson-Paradise, NV	Las Vegas-Henderson, NV-AZ	9	320030601	Boulder City
Nevada	Clark	1965 State Hwy 161, Jean, NV	Las Vegas-Henderson-Paradise, NV	Las Vegas-Henderson, NV-AZ	9	320031019	Jean
Nevada	Clark	1301B EAST TONOPAH	Las Vegas-Henderson-Paradise, NV	Las Vegas-Henderson, NV-AZ	9	320032002	JD Smith

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Nevada	Clark	668 Gretta Ln, Indian Springs	Las Vegas-Henderson-Paradise, NV	Las Vegas-Henderson, NV-AZ	9	320037772	Indian Springs
Nevada	Lyon	320 HARDIE LANE	Fernley, NV	Reno-Carson City-Fernley, NV	9	320190006	FERNLEY INTERMEDIATE SCHOOL
Nevada	Washoe	301 A STATE STREET, RENO, NV 89502	Reno, NV	Reno-Carson City-Fernley, NV	9	320310016	Reno3
Nevada	Washoe	4110 DE LUCCI LANE, RENO NV 89502	Reno, NV	Reno-Carson City-Fernley, NV	9	320310020	South Reno
Nevada	Washoe	684A STATE ROUTE 341, RENO NV 89521	Reno, NV	Reno-Carson City-Fernley, NV	9	320310025	Toll
Nevada	Washoe	750 4TH ST, SPARKS, NV 89431	Reno, NV	Reno-Carson City-Fernley, NV	9	320311005	Sparks
Nevada	Washoe	855 ALDER DRIVE, INCLINE VILLAGE, NV 89451	Reno, NV	Reno-Carson City-Fernley, NV	9	320312002	Incline
Nevada	Washoe	325 PATRICIAN DR, LEMMON VALLEY, NV 89506	Reno, NV	Reno-Carson City-Fernley, NV	9	320312009	Lemmon Valley
Nevada	White Pine	GREAT BASIN NATIONAL PARK			9	320330101	Great Basin National Park
Nevada	Carson City	2601 SOUTH CARSON ST (OLD NATIONAL GUARD ARMORY)	Carson City, NV	Reno-Carson City-Fernley, NV	9	325100020	OLD NATIONAL GUARD ARMORY
New Hampshire	Belknap	GREEN STREET, LACONIA	Laconia, NH	Boston-Worcester-Providence, MA-RI-NH-CT	1	330012004	FIELD OFFICE ON THE GROUNDS OF THE FORMER STATE PRISON
New Hampshire	Cheshire	WATER STREET	Keene, NH		1	330050007	WATER STREET
New Hampshire	Coos	MT. WASHINGTON	Berlin, NH-VT		1	330074001	
New Hampshire	Coos	CAMP DODGE, ROUTE 16, GREEN'S GRANT	Berlin, NH-VT		1	330074002	CAMP DODGE, GREENS GRANT
New Hampshire	Grafton	LEBANON AIRPORT	Claremont-Lebanon, NH-VT		1	330090010	LEBANON AIRPORT ROAD
New Hampshire	Grafton	Hubbard Brook Experimental Forest, North Woodstock, NH 03262	Claremont-Lebanon, NH-VT		1	330099991	Woodstock
New Hampshire	Hillsborough	GILSON ROAD	Manchester-Nashua, NH	Boston-Worcester-Providence, MA-RI-NH-CT	1	330111011	GILSON ROAD
New Hampshire	Hillsborough	PACK MONADNOCK SUMMIT	Manchester-Nashua, NH	Boston-Worcester-Providence, MA-RI-NH-CT	1	330115001	MILLER STATE PARK
New Hampshire	Merrimack	HAZEN DRIVE	Concord, NH	Boston-Worcester-Providence, MA-RI-NH-CT	1	330131007	HAZEN DRIVE
New Hampshire	Rockingham	PORTSMOUTH, PEIRCE ISLAND	Boston-Cambridge-Newton, MA-NH	Boston-Worcester-Providence, MA-RI-NH-CT	1	330150014	PORTSMOUTH - PEIRCE ISLAND
New Hampshire	Rockingham	SEACOAST SCIENCE CENTER	Boston-Cambridge-Newton, MA-NH	Boston-Worcester-Providence, MA-RI-NH-CT	1	330150016	SEACOAST SCIENCE CENTER
New Hampshire	Rockingham	150 Pillsbury Rd	Boston-Cambridge-Newton, MA-NH	Boston-Worcester-Providence, MA-RI-NH-CT	1	330150018	MOOSEHILL SCHOOL
New Jersey	Atlantic	Edwin B. Forsythe National Wildlife Refuge Visitor Center, Great Creek Road	Atlantic City-Hammonton, NJ	Philadelphia-Reading-Camden, PA-NJ-DE-MD	2	340010006	Brigantine
New Jersey	Bergen	Overpeck Park, 40 Fort Lee Road	New York-Newark-Jersey City, NY-NJ-PA	New York-Newark, NY-NJ-CT-PA	2	340030006	Leonia
New Jersey	Camden	266 Spruce Street	Philadelphia-Camden-Wilmington, PA-NJ-DE-MD	Philadelphia-Reading-Camden, PA-NJ-DE-MD	2	340070002	Camden Spruce Street

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New Jersey	Camden	Ancora State Hospital, 202 Spring Garden Road	Philadelphia-Camden-Wilmington, PA-NJ-DE-MD	Philadelphia-Reading-Camden, PA-NJ-DE-MD	2	340071001	Ancora State Hospital
New Jersey	Cumberland	Lincoln Avenue and Route 55, Northeast of Millville	Vineland-Bridgeton, NJ	Philadelphia-Reading-Camden, PA-NJ-DE-MD	2	340110007	Millville
New Jersey	Essex	360 Clinton Avenue	New York-Newark-Jersey City, NY-NJ-PA	New York-Newark, NY-NJ-CT-PA	2	340130003	Newark - Firehouse
New Jersey	Gloucester	Clarksboro Shady Rest Home, Shady Lane and County House Road	Philadelphia-Camden-Wilmington, PA-NJ-DE-MD	Philadelphia-Reading-Camden, PA-NJ-DE-MD	2	340150002	Clarksboro
New Jersey	Hudson	Veterans Park on Newark Bay, 25th Street near Park Road	New York-Newark-Jersey City, NY-NJ-PA	New York-Newark, NY-NJ-CT-PA	2	340170006	Bayonne
New Jersey	Hunterdon	Raritan Township Municipal Utilities Authority, 365 Old York Road	New York-Newark-Jersey City, NY-NJ-PA	New York-Newark, NY-NJ-CT-PA	2	340190001	Flemington
New Jersey	Mercer	Athletic Fields, Route 206 South	Trenton, NJ	New York-Newark, NY-NJ-CT-PA	2	340210005	Rider University
New Jersey	Mercer	Washington Crossing State Park, Titusville, NJ 08560	Trenton, NJ	New York-Newark, NY-NJ-CT-PA	2	340219991	Wash. Crossing
New Jersey	Middlesex	Horticultural Farm #3, off Ryder's Lane	New York-Newark-Jersey City, NY-NJ-PA	New York-Newark, NY-NJ-CT-PA	2	340230011	Rutgers University
New Jersey	Monmouth	Edison Science Building, 400 Cedar Avenue	New York-Newark-Jersey City, NY-NJ-PA	New York-Newark, NY-NJ-CT-PA	2	340250005	Monmouth University
New Jersey	Morris	Building #1, Department of Public Works (DPW) off Route 513	New York-Newark-Jersey City, NY-NJ-PA	New York-Newark, NY-NJ-CT-PA	2	340273001	Chester
New Jersey	Ocean	Colliers Mills Wildlife Management Area	New York-Newark-Jersey City, NY-NJ-PA	New York-Newark, NY-NJ-CT-PA	2	340290006	Colliers Mills
New Jersey	Passaic	Ramapo Mountain State Forest, Access Road, off Skyline Drive	New York-Newark-Jersey City, NY-NJ-PA	New York-Newark, NY-NJ-CT-PA	2	340315001	Ramapo
New Jersey	Warren	Columbia Wildlife Management Area, Delaware Road	Allentown-Bethlehem-Easton, PA-NJ	New York-Newark, NY-NJ-CT-PA	2	340410007	Columbia WMA
New Mexico	Bernalillo	4700A SAN MATEO NE	Albuquerque, NM	Albuquerque-Santa Fe-Las Vegas, NM	6	350010023	DEL NORTE HIGH SCHOOL
New Mexico	Bernalillo	201 PROSPERITY SE	Albuquerque, NM	Albuquerque-Santa Fe-Las Vegas, NM	6	350010029	SOUTH VALLEY
New Mexico	Bernalillo	8901 LOWELL NE	Albuquerque, NM	Albuquerque-Santa Fe-Las Vegas, NM	6	350011012	Foothills
New Mexico	Dona Ana	ST LUKES EPISCOPAL CH RT 1 (LA UNION)	Las Cruces, NM	El Paso-Las Cruces, TX-NM	6	350130008	
New Mexico	Dona Ana	680 MCCOMBS, CHAPARRAL, NM	Las Cruces, NM	El Paso-Las Cruces, TX-NM	6	350130020	6ZK 3 MI NORTH OF EL PASO, TX ON EAST SIDE OF FRANKLIN MOUNTAINS
New Mexico	Dona Ana	5935A VALLE VISTA, SUNLAND PARK, NM	Las Cruces, NM	El Paso-Las Cruces, TX-NM	6	350130021	6ZM 2MI FROM MT CRISTO REY WHERE NM, TEX, AND MEXICO JOIN TOGETHER

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New Mexico	Dona Ana	104-2 SANTA TERESA INTERNATIONAL BLVD,NM	Las Cruces, NM	El Paso-Las Cruces, TX-NM	6	350130022	6ZN US-MEXICO BORDER CROSSING. BOTH SIDES UNINHABITED AS OF 1996.
New Mexico	Dona Ana	750 N.SOLANO DRIVE, LAS CRUCES, NM	Las Cruces, NM	El Paso-Las Cruces, TX-NM	6	350130023	6ZQ IN SE CORNER OF NM HIGHWAY DEPT. YARDS IN LAS CRUCES
New Mexico	Eddy	HOLLAND ST,SE OF WATER TANK,CARLSBAD,NM	Carlsbad-Artesia, NM		6	350151005	5ZR ON BLM LAND BORDERING RESIDENTIAL AREA OUTSIDE CARLSBAD CITY LIM
New Mexico	Lea	2320 N. JEFFERSON ST, HOBBS, NEW MEXICO	Hobbs, NM		6	350250008	HOBBS-JEFFERSON
New Mexico	Rio Arriba	21 New Mexico 96, Coyote, NM, 87012	Espanola, NM	Albuquerque-Santa Fe-Las Vegas, NM	6	350390026	
New Mexico	Sandoval	HIGHWAY DEPT. YARD NEAR BERNALILLO	Albuquerque, NM	Albuquerque-Santa Fe-Las Vegas, NM	6	350431001	
New Mexico	San Juan	162 HWAY 544, BLOOMFIELD NM 87413	Farmington, NM		6	350450009	SE CORNER OF NM HIGHWAY DEPT YARD
New Mexico	San Juan	423 HWAY 539, NAVAJO DAM, NM 87419	Farmington, NM		6	350450018	
New Mexico	San Juan	USBR SHIPROCK SUBSTATION (FARMINGTON)	Farmington, NM		6	350451005	
New Mexico	Santa Fe	2001 Aviation Drive, Santa Fe, New Mexico 87507	Santa Fe, NM	Albuquerque-Santa Fe-Las Vegas, NM	6	350490021	
New Mexico	Valencia	1000 W. MAIN ST, LOS LUNAS, NM 87031	Albuquerque, NM	Albuquerque-Santa Fe-Las Vegas, NM	6	350610008	
New York	Albany	LOUDONVILLE RESERVOIR 300 ALBANY SHAKER RD	Albany-Schenectady-Troy, NY	Albany-Schenectady, NY	2	360010012	LOUDONVILLE
New York	Bronx	IS 52 681 KELLY ST	New York-Newark-Jersey City, NY-NJ-PA	New York-Newark, NY-NJ-CT-PA	2	360050110	IS 52
New York	Bronx	200TH STREET AND SOUTHERN BOULVDARD Pfizer Lab	New York-Newark-Jersey City, NY-NJ-PA	New York-Newark, NY-NJ-CT-PA	2	360050133	PFIZER LAB SITE
New York	Chautauqua	STP, Wright Park Drive, DUNKIRK	Jamestown-Dunkirk-Fredonia, NY		2	360130006	DUNKIRK
New York	Dutchess	Forest Road, VILLAGE OF MILLBROOK, Inst of Ecosystem Studies	New York-Newark-Jersey City, NY-NJ-PA	New York-Newark, NY-NJ-CT-PA	2	360270007	MILLBROOK
New York	Erie	AUDUBON GOLF COURSE, 450 MAPLE ROAD	Buffalo-Cheektowaga-Niagara Falls, NY	Buffalo-Cheektowaga, NY	2	360290002	AMHERST
New York	Essex	Wilmington, SUMMIT, WHITEFACE MTN, WEATHER STATION			2	360310002	WHITEFACE SUMMIT
New York	Essex	Wilmington, BASE WHITEFACE MTN, ASRC, SUNY			2	360310003	WHITEFACE BASE

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New York	Essex	Huntington Wildlife Forest, Newcomb, NY 12852			2	360319991	Huntington Wildlife Forest
New York	Franklin	55 Library Road, HOGANSBURG, NY 13655	Malone, NY		2	360337003	Y001
New York	Hamilton	PISECO LAKE AIRPORT			2	360410005	PISECO LAKE
New York	Herkimer	278 Bisby Road, NICKS LAKE CAMPGROUND	Utica-Rome, NY		2	360430005	NICKS LAKE
New York	Jefferson	VAADI ROAD, PERCH RIVER	Watertown-Fort Drum, NY		2	360450002	PERCH RIVER
New York	Monroe	2 YARMOUTH ROAD, RG&E Substation	Rochester, NY	Rochester-Batavia-Seneca Falls, NY	2	360551007	ROCHESTER 2
New York	New York	160 CONVENT AVENUE	New York-Newark-Jersey City, NY-NJ-PA	New York-Newark, NY-NJ-CT-PA	2	360610135	CCNY
New York	Niagara	MIDDLEPORT STP, 3825 NORTH HARTLAND RD	Buffalo-Cheektowaga-Niagara Falls, NY	Buffalo-Cheektowaga, NY	2	360631006	MIDDLEPORT
New York	Onondaga	5895 ENTERPRISE PARKWAY,	Syracuse, NY	Syracuse-Auburn, NY	2	360671015	EAST SYRACUSE
New York	Orange	1175 ROUTE 17K, MONTGOMERY Valley Central HS	New York-Newark-Jersey City, NY-NJ-PA	New York-Newark, NY-NJ-CT-PA	2	360715001	VALLEY CENTRAL HIGH SCHOOL
New York	Oswego	820 COUNTY ROUTE 8	Syracuse, NY	Syracuse-Auburn, NY	2	360750003	FULTON
New York	Putnam	NYSDEC FIELD HQTRS GYPSY TRAIL ROAD	New York-Newark-Jersey City, NY-NJ-PA	New York-Newark, NY-NJ-CT-PA	2	360790005	MT NINHAM
New York	Queens	Queens College 65-30 Kissena Blvd Parking Lot#6	New York-Newark-Jersey City, NY-NJ-PA	New York-Newark, NY-NJ-CT-PA	2	360810124	QUEENS COLLEGE 2
New York	Richmond	SUSAN WAGNER HS, 1200 MANOR RD, near Brielle Ave	New York-Newark-Jersey City, NY-NJ-PA	New York-Newark, NY-NJ-CT-PA	2	360850067	SUSAN WAGNER HS
New York	Rockland	2 South Mountain Road	New York-Newark-Jersey City, NY-NJ-PA	New York-Newark, NY-NJ-CT-PA	2	360870005	Rockland County
New York	Saratoga	SARATOGA NATIONAL HISTORICAL PARK	Albany-Schenectady-Troy, NY	Albany-Schenectady, NY	2	360910004	STILLWATER
New York	Steuben	8301 ACKERSON ROAD, Pinnacle State Park	Corning, NY	Elmira-Corning, NY	2	361010003	PINNACLE STATE PARK
New York	Suffolk	EAST FARMINGDALE WATER DIST., 72 GAZZA BLVD.	New York-Newark-Jersey City, NY-NJ-PA	New York-Newark, NY-NJ-CT-PA	2	361030002	BABYLON
New York	Suffolk	3059 SOUND AVENUE, RIVERHEAD	New York-Newark-Jersey City, NY-NJ-PA	New York-Newark, NY-NJ-CT-PA	2	361030004	RIVERHEAD
New York	Suffolk	57 DIVISION STREET	New York-Newark-Jersey City, NY-NJ-PA	New York-Newark, NY-NJ-CT-PA	2	361030009	HOLTSVILLE
New York	Tompkins	Connecticut Hill Wildlife Management Area, Newfield, NY 14867	Ithaca, NY	Ithaca-Cortland, NY	2	361099991	Connecticut Hill
New York	Wayne	4440 Ridge Road, WAYNE County Occupational CENTER, WILLIAMSON	Rochester, NY	Rochester-Batavia-Seneca Falls, NY	2	361173001	WILLIAMSON
New York	Westchester	WHITE PLAINS PUMP STATION, 240 ORCHARD STREET	New York-Newark-Jersey City, NY-NJ-PA	New York-Newark, NY-NJ-CT-PA	2	361192004	WHITE PLAINS
North Carolina	Alexander	700 Liledoun Road	Hickory-Lenoir-Morganton, NC	Hickory-Lenoir, NC	4	370030005	Taylorville Liledoun
North Carolina	Avery	7510 BLUE RIDGE PARKWAY SPUR			4	370110002	Linville Falls
North Carolina	Avery	Pisgah National Forest, Newland, NC 28657			4	370119991	Cranberry
North Carolina	Buncombe	ROUTE 191 SOUTH BREVARD RD	Asheville, NC	Asheville-Brevard, NC	4	370210030	Bent Creek
North Carolina	Caldwell	219 NUWAY CIRCLE	Hickory-Lenoir-Morganton, NC	Hickory-Lenoir, NC	4	370270003	Lenoir (city)
North Carolina	Carteret	Open Grounds Farm, Beaufort, NC 28516	Morehead City, NC	New Bern-Morehead City, NC	4	370319991	Beaufort

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North Carolina	Caswell	7074 CHERRY GROVE RD, REIDSVILLE			4	370330001	Cherry Grove
North Carolina	Cumberland	7112 Covington Lane	Fayetteville, NC	Fayetteville-Lumberton-Laurinburg, NC	4	370510008	Wade
North Carolina	Durham	801 STADIUM DRIVE	Durham-Chapel Hill, NC	Raleigh-Durham-Chapel Hill, NC	4	370630015	Durham Armory
North Carolina	Edgecombe	7589 NC Hwy 33-NW	Rocky Mount, NC	Rocky Mount-Wilson-Roanoke Rapids, NC	4	370650099	Leggett
North Carolina	Forsyth	1300 BLK. HATTIE AVENUE	Winston-Salem, NC	Greensboro--Winston-Salem--High Point, NC	4	370670022	Hattie Avenue
North Carolina	Forsyth	FRATERNITY CHURCH ROAD	Winston-Salem, NC	Greensboro--Winston-Salem--High Point, NC	4	370670030	Clemmons Middle
North Carolina	Forsyth	3656 PIEDMONT MEMORIAL DRIVE	Winston-Salem, NC	Greensboro--Winston-Salem--High Point, NC	4	370671008	Union Cross
North Carolina	Graham	FOREST ROAD 423 SPUR			4	370750001	Joanna Bald
North Carolina	Granville	800 Central Avenue	Oxford, NC	Raleigh-Durham-Chapel Hill, NC	4	370770001	Butner
North Carolina	Guilford	205 WILOUGHBY BLVD	Greensboro-High Point, NC	Greensboro--Winston-Salem--High Point, NC	4	370810013	Mendenhall School
North Carolina	Haywood	2236 ASHEVILLE ROAD	Asheville, NC	Asheville-Brevard, NC	4	370870008	Waynesville School
North Carolina	Haywood	TOWER BLUE RIDGE PKWY MILE MARKER 410	Asheville, NC	Asheville-Brevard, NC	4	370870035	Frying Pan Mountain
North Carolina	Haywood	6905 Purchase Knob Road	Asheville, NC	Asheville-Brevard, NC	4	370870036	Purchase Knob
North Carolina	Johnston	1338 JACK ROAD	Raleigh, NC	Raleigh-Durham-Chapel Hill, NC	4	371010002	West Johnston Co.
North Carolina	Lee	4110 Blackstone Drive	Sanford, NC	Raleigh-Durham-Chapel Hill, NC	4	371050002	Blackstone
North Carolina	Lenoir	HIGHWAY 70 EAST AND HIGHWAY 58 SOUTH	Kinston, NC		4	371070004	Lenoir Co. Comm. Coll.
North Carolina	Lincoln	1487 RIVERVIEW ROAD	Charlotte-Concord-Gastonia, NC-SC	Charlotte-Concord, NC-SC	4	371090004	Crouse
North Carolina	Macon	USDA Southern Research Station, Coweeta Hydrologic Laboratory, Otto, NC 28763			4	371139991	Coweeta
North Carolina	Martin	1210 Hayes Street			4	371170001	Jamesville School
North Carolina	Mecklenburg	1130 EASTWAY DRIVE	Charlotte-Concord-Gastonia, NC-SC	Charlotte-Concord, NC-SC	4	371190041	Garinger High School
North Carolina	Montgomery	136 Perry Dr, Candor, NC 27229			4	371239991	Candor
North Carolina	New Hanover	6028 HOLLY SHELTER RD	Wilmington, NC		4	371290002	Castle Hayne
North Carolina	Person	SR49	Durham-Chapel Hill, NC	Raleigh-Durham-Chapel Hill, NC	4	371450003	Bushy Fork
North Carolina	Pitt	403 Government Circle	Greenville, NC	Greenville-Washington, NC	4	371470006	Pitt Agri. Center
North Carolina	Rockingham	6371 NC 65 @ BETHANY SCHOOL	Greensboro-High Point, NC	Greensboro--Winston-Salem--High Point, NC	4	371570099	Bethany sch.
North Carolina	Rowan	301 WEST ST & GOLD HILL AVENUE	Charlotte-Concord-Gastonia, NC-SC	Charlotte-Concord, NC-SC	4	371590021	Rockwell
North Carolina	Swain	30 Recreation Park Drive			4	371730002	Bryson City
North Carolina	Swain	1501 Acquoni Rd.			4	371730007	
North Carolina	Union	701 CHARLES STREET	Charlotte-Concord-Gastonia, NC-SC	Charlotte-Concord, NC-SC	4	371790003	Monroe School
North Carolina	Wake	3801 SPRING FOREST RD.	Raleigh, NC	Raleigh-Durham-Chapel Hill, NC	4	371830014	Millbrook School
North Carolina	Yancey	2388 State Highway 128			4	371990004	Mt. Mitchell
North Dakota	Billings	13881 194 EAST			8	380070002	PAINTED CANYON
North Dakota	Burke	8315 HIGHWAY 8, KENMARE			8	380130004	LOSTWOOD NWR

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North Dakota	Burleigh	1810 N 16TH STREET	Bismarck, ND		8	380150003	BISMARCK RESIDENTIAL
North Dakota	Cass	4266 40TH AVE NORTH	Fargo, ND-MN	Fargo-Wahpeton, ND-MN	8	380171004	FARGO NW
North Dakota	Dunn	9610 SEVENTH STREET SW			8	380250003	DUNN CENTER
North Dakota	McKenzie	229 SERVICE RD., WATFORD CITY			8	380530002	TRNP-NU
North Dakota	Mercer	6024 HIGHWAY 200			8	380570004	BEULAH NORTH
North Dakota	Oliver	1575 HIGHWAY 31	Bismarck, ND		8	380650002	HANNOVER
North Dakota	Williams	10th Street West	Williston, ND		8	381050003	Williston
Ohio	Allen	2850 BIBLE ROAD	Lima, OH	Lima-Van Wert-Celina, OH	5	390030009	LIMA BATH
Ohio	Ashtabula	770 LAKE RD.	Ashtabula, OH	Cleveland-Akron-Canton, OH	5	390071001	CONNEAUT
Ohio	Butler	SCHULER AND BENDER	Cincinnati, OH-KY-IN	Cincinnati-Wilmington-Maysville, OH-KY-IN	5	390170004	HAMILTON
Ohio	Butler	1701 Runway Dr.	Cincinnati, OH-KY-IN	Cincinnati-Wilmington-Maysville, OH-KY-IN	5	390170018	MIDDLETOWN
Ohio	Butler	Ecology Research Center, Miami University, Oxford, Ohio 45056	Cincinnati, OH-KY-IN	Cincinnati-Wilmington-Maysville, OH-KY-IN	5	390179991	Oxford
Ohio	Clark	5171 URBANA	Springfield, OH	Dayton-Springfield-Sidney, OH	5	390230001	SPRINGFIELD WELL FIELD
Ohio	Clark	5400 SPANGLER	Springfield, OH	Dayton-Springfield-Sidney, OH	5	390230003	MUD RUN
Ohio	Clermont	2400 CLERMONT CENTER DR.	Cincinnati, OH-KY-IN	Cincinnati-Wilmington-Maysville, OH-KY-IN	5	390250022	BATAVIA
Ohio	Clinton	62 LAUREL DR.	Wilmington, OH	Cincinnati-Wilmington-Maysville, OH-KY-IN	5	390271002	LAUREL OAKS_JVS
Ohio	Cuyahoga	891 E. 152 ST.	Cleveland-Elyria, OH	Cleveland-Akron-Canton, OH	5	390350034	5TH DISTRICT
Ohio	Cuyahoga	E. 14TH & ORANGE	Cleveland-Elyria, OH	Cleveland-Akron-Canton, OH	5	390350060	GT CRAIG
Ohio	Cuyahoga	390 FAIR ST.	Cleveland-Elyria, OH	Cleveland-Akron-Canton, OH	5	390350064	BEREA
Ohio	Cuyahoga	6116 WILSON MILLS	Cleveland-Elyria, OH	Cleveland-Akron-Canton, OH	5	390355002	MAYFIELD
Ohio	Delaware	359 MAIN RD.	Columbus, OH	Columbus-Marion-Zanesville, OH	5	390410002	DELAWARE
Ohio	Fayette	Deer Creek State Park, Mt Sterling, OH 43143	Washington Court House, OH	Columbus-Marion-Zanesville, OH	5	390479991	Deer Creek
Ohio	Franklin	7600 FODOR RD.	Columbus, OH	Columbus-Marion-Zanesville, OH	5	390490029	NEW_ALBANY
Ohio	Franklin	1777 E. BROAD	Columbus, OH	Columbus-Marion-Zanesville, OH	5	390490037	FRANKLIN_PK
Ohio	Franklin	5750 MAPLE CANYON	Columbus, OH	Columbus-Marion-Zanesville, OH	5	390490081	MAPLE_C
Ohio	Geauga	13000 AUBURN	Cleveland-Elyria, OH	Cleveland-Akron-Canton, OH	5	390550004	GEAUGA
Ohio	Greene	541 LEDBETTER RD.,	Dayton, OH	Dayton-Springfield-Sidney, OH	5	390570006	XENIA
Ohio	Hamilton	11590 GROOMS RD	Cincinnati, OH-KY-IN	Cincinnati-Wilmington-Maysville, OH-KY-IN	5	390610006	SYCAMORE
Ohio	Hamilton	6950 RIPPLE RD.	Cincinnati, OH-KY-IN	Cincinnati-Wilmington-Maysville, OH-KY-IN	5	390610010	COLERAIN
Ohio	Hamilton	250 WM. HOWARD TAFT	Cincinnati, OH-KY-IN	Cincinnati-Wilmington-Maysville, OH-KY-IN	5	390610040	TAFT
Ohio	Jefferson	618 LOGAN ST.	Weirton-Steubenville, WV-OH	Pittsburgh-New Castle-Weirton, PA-OH-WV	5	390810017	STEUBEN
Ohio	Knox	WATER PLT. SR. 314	Mount Vernon, OH	Columbus-Marion-Zanesville, OH	5	390830002	CENTERBURG
Ohio	Lake	36010 LAKESHORE	Cleveland-Elyria, OH	Cleveland-Akron-Canton, OH	5	390850003	EASTLAKE
Ohio	Lake	177 MAIN STREET	Cleveland-Elyria, OH	Cleveland-Akron-Canton, OH	5	390850007	JFS (PAINSVILLE)
Ohio	Lawrence	S.R. 141, WILGUS	Huntington-Ashland, WV-KY-OH	Charleston-Huntington-Ashland, WV-OH-KY	5	390870011	WILGUS

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Ohio	Lawrence	460 Commerce Drive	Huntington-Ashland, WV-KY-OH	Charleston-Huntington-Ashland, WV-OH-KY	5	390870012	ODOT (IRONTON)
Ohio	Licking	300 LICKING VIEW DR.	Columbus, OH	Columbus-Marion-Zanesville, OH	5	390890005	HEATH
Ohio	Lorain	4706 DETROIT RD.	Cleveland-Elyria, OH	Cleveland-Akron-Canton, OH	5	390930018	SHEFFIELD
Ohio	Lucas	348 S. ERIE	Toledo, OH	Toledo-Port Clinton, OH	5	390950024	ERIE
Ohio	Lucas	200 SOUTH RIVER RD.	Toledo, OH	Toledo-Port Clinton, OH	5	390950027	WATERVILLE
Ohio	Lucas	1002 N. YONDOTA	Toledo, OH	Toledo-Port Clinton, OH	5	390950034	LOW_SER
Ohio	Madison	940 SR 38 SW	Columbus, OH	Columbus-Marion-Zanesville, OH	5	390970007	LONDON
Ohio	Mahoning	345 OAKHILL	Youngstown-Warren-Boardman, OH-PA	Youngstown-Warren, OH-PA	5	390990013	
Ohio	Medina	BALLASH ROAD	Cleveland-Elyria, OH	Cleveland-Akron-Canton, OH	5	391030004	CHIPPEWA
Ohio	Miami	3825 NORTH S. R. 589	Dayton, OH	Dayton-Springfield-Sidney, OH	5	391090005	MIAMI EAST
Ohio	Montgomery	1401 Harshman Road	Dayton, OH	Dayton-Springfield-Sidney, OH	5	391130037	EASTWOOD
Ohio	Noble	58163 St. Johns Rd. Quaker City, OH 43773			5	391219991	Quaker City
Ohio	Portage	1570 RAVENNA RD.	Akron, OH	Cleveland-Akron-Canton, OH	5	391331001	ROCKWELL
Ohio	Preble	6940 OXFORD GETTYSBURG RD.			5	391351001	NATIONAL TRAIL SCHOOL
Ohio	Stark	515 25TH. ST.	Canton-Massillon, OH	Cleveland-Akron-Canton, OH	5	391510016	MALONE_COL
Ohio	Stark	45 S. WABASH AVENUE, S.R 93	Canton-Massillon, OH	Cleveland-Akron-Canton, OH	5	391510022	BREWSTER (WANDLE)
Ohio	Stark	1175 WEST VINE	Canton-Massillon, OH	Cleveland-Akron-Canton, OH	5	391514005	ALLIANCE
Ohio	Summit	800 PATTERSON AVE.	Akron, OH	Cleveland-Akron-Canton, OH	5	391530020	PATTERSON PARK (PATT_PARK)
Ohio	Trumbull	842 YOUNGSTOWN-KINGSVILLE RD.	Youngstown-Warren-Boardman, OH-PA	Youngstown-Warren, OH-PA	5	391550011	TCSEG
Ohio	Warren	430 SOUTHEAST ST.	Cincinnati, OH-KY-IN	Cincinnati-Wilmington-Maysville, OH-KY-IN	5	391650007	LEBANON
Ohio	Washington	2000 4TH STREET	Marietta, OH	Parkersburg-Marietta-Vienna, WV-OH	5	391670004	MARIETTA_TWP.
Ohio	Wood	347 N DUNBRIDGE	Toledo, OH	Toledo-Port Clinton, OH	5	391730003	BOWLING GREEN
Oklahoma	Adair	South Highway 59, RR1, 1795 Dahlenegah Park Road, Stilwell, Oklahoma			6	400019009	STILWELL
Oklahoma	Caddo	WESTERN DELAWARE TRIBAL COMPLEX			6	400159008	ANADARKO PM2.5
Oklahoma	Canadian	12575 NW 10TH (WATER TOWER)	Oklahoma City, OK	Oklahoma City-Shawnee, OK	6	400170101	OKC WEST-(YUKON)
Oklahoma	Cherokee	P.O. BOX 948 TAHLEQUAH, OK 74464	Tahlequah, OK	Tulsa-Muskogee-Bartlesville, OK	6	400219002	TAHLEQUAH SHELTER
Oklahoma	Cleveland	S. E. 19TH STREET	Oklahoma City, OK	Oklahoma City-Shawnee, OK	6	400270049	MOORE WATER TOWER
Oklahoma	Comanche	2211 NW 25 Lawton, Oklahoma 73502 (LPS: Prof. Dev. Center)	Lawton, OK		6	400310651	LAWTON NORTH
Oklahoma	Creek	MANNFORD WATER PLANT	Tulsa, OK	Tulsa-Muskogee-Bartlesville, OK	6	400370144	MANNFORD
Oklahoma	Dewey	SEILING MUNICIPAL AIRPORT			6	400430860	SEILING MUNICIPAL AIRPORT
Oklahoma	Kay	NORTH OF NEWKIRK OK. ON HWY 77 - EAST HOME ROAD (E0040RD) .90 MILES	Ponca City, OK		6	400719010	NEWKIRK IMPROVE
Oklahoma	McClain	310 E. BURR OAK	Oklahoma City, OK	Oklahoma City-Shawnee, OK	6	400871073	GOLDSBY
Oklahoma	Mayes	CHEROKEE HEIGHTS DRIVE			6	400979014	CHEROKEE HEIGHTS
Oklahoma	Oklahoma	NE 10TH & STONEWALL	Oklahoma City, OK	Oklahoma City-Shawnee, OK	6	401090033	OKC CENTRAL-OSDH
Oklahoma	Oklahoma	12880 A NE 10TH	Oklahoma City, OK	Oklahoma City-Shawnee, OK	6	401090096	CHOCTAW

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Oklahoma	Oklahoma	2501 E. Memorial Rd. (OKLAHOMA CHRISTIAN UNIVERSITY)	Oklahoma City, OK	Oklahoma City-Shawnee, OK	6	401091037	OKC NORTH
Oklahoma	Ottawa	QUAPAW TRIBE'S INDUSTRIAL PARK, MIAMI	Miami, OK	Joplin-Miami, MO-OK	6	401159004	QUAPAW SHELTER
Oklahoma	Pittsburg	104 AIRPORT ROAD	McAlester, OK		6	401210415	McALESTER MUNICIPAL AIRPORT
Oklahoma	Sequoyah	207 Cherokee Boulevard, Roland, OK 74954	Fort Smith, AR-OK		6	401359021	
Oklahoma	Tulsa	1100 SOUTH OSAGE DRIVE	Tulsa, OK	Tulsa-Muskogee-Bartlesville, OK	6	401430137	TULSA NORTH (SKIATOOK)
Oklahoma	Tulsa	502 EAST 144TH. PL.	Tulsa, OK	Tulsa-Muskogee-Bartlesville, OK	6	401430174	TULSA SOUTH
Oklahoma	Tulsa	18707 E. 21ST STREET	Tulsa, OK	Tulsa-Muskogee-Bartlesville, OK	6	401430178	TULSA EAST
Oklahoma	Tulsa	3520 1/2 N. PEORIA	Tulsa, OK	Tulsa-Muskogee-Bartlesville, OK	6	401431127	NORTH TULSA - FIRE STATION#24
Oregon	Clackamas	CARUS TRAILER/SPANGLER RD	Portland-Vancouver-Hillsboro, OR-WA	Portland-Vancouver-Salem, OR-WA	10	410050004	CARUS TRAILER/SPANGLER RD
Oregon	Columbia	SAUVIE ISLAND-SSB/ROUTE 1, BOX 442	Portland-Vancouver-Hillsboro, OR-WA	Portland-Vancouver-Salem, OR-WA	10	410090004	SAUVIE ISLAND-SIS
Oregon	Jackson	7112 RAPP LANE, TALENT	Medford, OR	Medford-Grants Pass, OR	10	410290201	Medford at Talent (South of Medford) - (TAL)
Oregon	Lane	AMAZON PARK/499 E 29TH	Eugene, OR		10	410390060	Eugene - AMAZON PARK (EAP)
Oregon	Lane	79980 DELIGHT VALLEY SCHOOL RD, SAGINAW	Eugene, OR		10	410391007	Eugene - Saginaw
Oregon	Marion	CASCADE JR HIGH 10226 MARION RD TURNER O	Salem, OR	Portland-Vancouver-Salem, OR-WA	10	410470004	Salem - Turner at Cascade School (CJH)
Oregon	Multnomah	SE LAFAYETTE/5824 SE LAFAYETTE	Portland-Vancouver-Hillsboro, OR-WA	Portland-Vancouver-Salem, OR-WA	10	410510080	Portland - SE Lafayette
Oregon	Umatilla	HERMISTON MUNICIPAL AIRPORT, 1600 AIRPORT WAY, HERMISTON	Hermiston-Pendleton, OR		10	410591003	Hermiston - Municipal Airport (HMA)
Oregon	Washington	6745 Bradbury Court, Tualatin, OR (NO2 roadway site)	Portland-Vancouver-Hillsboro, OR-WA	Portland-Vancouver-Salem, OR-WA	10	410670005	
Oregon	Washington	17180 SW Lasich Lane, Sherwood - Lasich and Roy Rogers (SLR)	Portland-Vancouver-Hillsboro, OR-WA	Portland-Vancouver-Salem, OR-WA	10	410671004	Sherwood - Lasich and Roy Rogers (SLR)
Pennsylvania	Adams	747 Winding Rd, Biglerville, PA 17307	Gettysburg, PA	Harrisburg-York-Lebanon, PA	3	420019991	Arendtsville
Pennsylvania	Allegheny	301 39th St, Pittsburgh, PA 15201 (Lawrenceville)	Pittsburgh, PA	Pittsburgh-New Castle-Weirton, PA-OH-WV	3	420030008	Lawrenceville
Pennsylvania	Allegheny	3640 Old Oakdale Rd, McDonald, PA 15057 (South Fayette)	Pittsburgh, PA	Pittsburgh-New Castle-Weirton, PA-OH-WV	3	420030067	South Fayette
Pennsylvania	Allegheny	1500 Pacific Ave, Natrona Heights, PA 15065 (Harrison)	Pittsburgh, PA	Pittsburgh-New Castle-Weirton, PA-OH-WV	3	420031008	Harrison
Pennsylvania	Armstrong	GLADE DR. & NOLTE RD, KITTANNING	Pittsburgh, PA	Pittsburgh-New Castle-Weirton, PA-OH-WV	3	420050001	LAT/LON IS CENTER OF TRAILER
Pennsylvania	Beaver	ROUTE 168 & TOMLINSON ROAD	Pittsburgh, PA	Pittsburgh-New Castle-Weirton, PA-OH-WV	3	420070002	

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Pennsylvania	Beaver	1015 SEBRING ROAD	Pittsburgh, PA	Pittsburgh-New Castle-Weirton, PA-OH-WV	3	420070005	DRIVEWAY TO BAKEY RESIDENCE
Pennsylvania	Beaver	EIGHT STREET AND RIVER ALLEY	Pittsburgh, PA	Pittsburgh-New Castle-Weirton, PA-OH-WV	3	420070014	
Pennsylvania	Berks	Kutztown University Campus	Reading, PA	Philadelphia-Reading-Camden, PA-NJ-DE-MD	3	420110006	Kutztown
Pennsylvania	Berks	1059 Arnold Road	Reading, PA	Philadelphia-Reading-Camden, PA-NJ-DE-MD	3	420110011	Reading Airport
Pennsylvania	Blair	2ND AVE & 7TH ST	Altoona, PA		3	420130801	
Pennsylvania	Bradford	RT 4148 Main St, Monroe, PA 18832	Sayre, PA		3	420150011	Towanda
Pennsylvania	Bucks	ROCKVIEW LANE	Philadelphia-Camden-Wilmington, PA-NJ-DE-MD	Philadelphia-Reading-Camden, PA-NJ-DE-MD	3	420170012	A420170012LAT/LONG POINT IS OF SAMPLING INLET
Pennsylvania	Cambria	MILLER AUTO SHOP 1 MESSENGER ST	Johnstown, PA	Johnstown-Somerset, PA	3	420210011	
Pennsylvania	Centre	PENN STATE UNIVERSITY - ARBORETUM SITE	State College, PA	State College-DuBois, PA	3	420270100	LAT/LON=POINT SW CORNER OF TRAILER
Pennsylvania	Centre	1366 Tadpole Rd, Pennsylvania Furnace, PA 16865	State College, PA	State College-DuBois, PA	3	420279991	Penn State
Pennsylvania	Chester	NEW GARDEN AIRPORT - TOUGHKENAMON	Philadelphia-Camden-Wilmington, PA-NJ-DE-MD	Philadelphia-Reading-Camden, PA-NJ-DE-MD	3	420290100	CHESTER COUNTY TRANSPORT SITE INTO PHILADELPHIA
Pennsylvania	Clearfield	LOCATED NEAR S.B. ELLIOTT STATE PARK	DuBois, PA	State College-DuBois, PA	3	420334000	MOSHANNON STATE FOREST
Pennsylvania	Dauphin	651 Gibson Blvd., Harrisburg, PA 17104	Harrisburg-Carlisle, PA	Harrisburg-York-Lebanon, PA	3	420430401	A420430401LAT/LON POINT IS AT CORNER OF TRAILER
Pennsylvania	Dauphin	SIPE AVE & MAE STREET	Harrisburg-Carlisle, PA	Harrisburg-York-Lebanon, PA	3	420431100	A420431100LAT/LON POINT IS AT CORNER OF TRAILER
Pennsylvania	Delaware	FRONT ST & NORRIS ST	Philadelphia-Camden-Wilmington, PA-NJ-DE-MD	Philadelphia-Reading-Camden, PA-NJ-DE-MD	3	420450002	A420450002LAT/LON POINT IS OF CORNER OF TRAILER
Pennsylvania	Elk	Kane Experimental Forest, Allegheny National Forest, Wilcox, PA 15870			3	420479991	Kane Exp. Forest
Pennsylvania	Erie	10TH AND MARNE STREETS	Erie, PA	Erie-Meadville, PA	3	420490003	
Pennsylvania	Franklin	FOREST ROAD - METHODIST HILL	Chambersburg-Waynesboro, PA	Washington-Baltimore-Arlington, DC-MD-VA-WV-PA	3	420550001	HIGH ELEVATION OZONE SITE
Pennsylvania	Greene	4.8 KM SE OF HOLBROOK			3	420590002	75 KM SSW OF PITTSBURGH RURAL SITE ON A KNOLL WITHIN A LARGE CLEARIN
Pennsylvania	Indiana	PA. DEPT. OF TRANSPORTATION - RT.403 - Strongstown	Indiana, PA	Pittsburgh-New Castle-Weirton, PA-OH-WV	3	420630004	

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Pennsylvania	Lackawanna	WILSON FIRE CO. ERIE & PLEASANT	Scranton--Wilkes-Barre--Hazleton, PA		3	420690101	A420690101LAT/LON POINT IS AT CORNER OF TRAILER
Pennsylvania	Lackawanna	GEORGE ST TROOP AND CITY OF SCRANTON	Scranton--Wilkes-Barre--Hazleton, PA		3	420692006	A420692006LAT/LON POINT IS AT CORNER OF TRAILER
Pennsylvania	Lancaster	ABRAHAM LINCOLN JR HIGH GROFFTOWN RD	Lancaster, PA		3	420710007	A420710007LAT/LON POINT AT CORNER OF TRAILER
Pennsylvania	Lancaster	3545 W. Newport Road	Lancaster, PA		3	420710012	Lancaster DW
Pennsylvania	Lawrence	CROTON ST & JEFFERSON ST.	New Castle, PA	Pittsburgh-New Castle-Weirton, PA-OH-WV	3	420730015	
Pennsylvania	Lebanon	125 Birch Road	Lebanon, PA	Harrisburg-York-Lebanon, PA	3	420750100	Lebanon
Pennsylvania	Lehigh	STATE HOSPITAL REAR 1600 HANOVER AVE	Allentown-Bethlehem-Easton, PA-NJ	New York-Newark, NY-NJ-CT-PA	3	420770004	A420770004LAT/LONG POINT IS OF SAMPLING INLET
Pennsylvania	Luzerne	CHILWICK & WASHINGTON STS	Scranton--Wilkes-Barre--Hazleton, PA		3	420791101	A420791101LAT/LON POINT IS AT CORNER OF TRAILER
Pennsylvania	Lycoming	899 CHERRY STREET	Williamsport, PA	Williamsport-Lock Haven, PA	3	420810100	MONTOURSVILLE
Pennsylvania	Mercer	PA518 (NEW CASTLE ROAD) & PA418	Youngstown-Warren-Boardman, OH-PA	Youngstown-Warren, OH-PA	3	420850100	
Pennsylvania	Mercer	Maurice K Goddard State Park, Sandy Lake, PA 16145	Youngstown-Warren-Boardman, OH-PA	Youngstown-Warren, OH-PA	3	420859991	M.K. Goddard
Pennsylvania	Monroe	HC 1, BOX 95-B NEAR RT 611 & BROOKDALE ROAD	East Stroudsburg, PA	New York-Newark, NY-NJ-CT-PA	3	420890002	SWIFTWATER
Pennsylvania	Montgomery	STATE ARMORY - 1046 BELVOIR RD	Philadelphia-Camden-Wilmington, PA-NJ-DE-MD	Philadelphia-Reading-Camden, PA-NJ-DE-MD	3	420910013	A420910013LAT/LON POINT IS OF CORNER OF TRAILER
Pennsylvania	Northampton	WASHINGTON & CAMBRIA STS. FREEMANSBURG	Allentown-Bethlehem-Easton, PA-NJ	New York-Newark, NY-NJ-CT-PA	3	420950025	LAT/LON POINT IS CENTER OF TRAILER
Pennsylvania	Northampton	17TH AND SPRING GARDEN STREETS	Allentown-Bethlehem-Easton, PA-NJ	New York-Newark, NY-NJ-CT-PA	3	420958000	COMBINED EASTON SITE (420950100) AND EASTON H2S SPECIAL STUDY SITES
Pennsylvania	Philadelphia	1501 E. LYCOMING AVE.	Philadelphia-Camden-Wilmington, PA-NJ-DE-MD	Philadelphia-Reading-Camden, PA-NJ-DE-MD	3	421010004	Air Management Services Laboratory (AMS LAB)
Pennsylvania	Philadelphia	GRANT-ASHTON ROADS PHILA NE AIRPORT	Philadelphia-Camden-Wilmington, PA-NJ-DE-MD	Philadelphia-Reading-Camden, PA-NJ-DE-MD	3	421010024	North East Airport (NEA)
Pennsylvania	Philadelphia	3000 LEWIS ST. (NEAR BATH ST.) Philadelphia, PA. 19137	Philadelphia-Camden-Wilmington, PA-NJ-DE-MD	Philadelphia-Reading-Camden, PA-NJ-DE-MD	3	421010048	North East Waste (NEW)
Pennsylvania	Somerset	Laurel Hill State Park, Rockwood, PA 15557	Somerset, PA	Johnstown-Somerset, PA	3	421119991	Laurel Hill
Pennsylvania	Tioga	TIOGA			3	421174000	PENN STATE OZONE MONITORING SITE
Pennsylvania	Washington	CHARLER01 WASTE TREATMENT PLANT	Pittsburgh, PA	Pittsburgh-New Castle-Weirton, PA-OH-WV	3	421250005	

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Pennsylvania	Washington	MCCARRELL AND FAYETTE STS	Pittsburgh, PA	Pittsburgh-New Castle-Weirton, PA-OH-WV	3	421250200	
Pennsylvania	Washington	HILLMAN STATE PARK - KINGS CREEK ROAD	Pittsburgh, PA	Pittsburgh-New Castle-Weirton, PA-OH-WV	3	421255001	
Pennsylvania	Westmoreland	DONOHOE ROAD - PENN DOT MAINT DIST BLDG	Pittsburgh, PA	Pittsburgh-New Castle-Weirton, PA-OH-WV	3	421290008	LAT/LON POINT IS TRAILER
Pennsylvania	York	HILL ST.	York-Hanover, PA	Harrisburg-York-Lebanon, PA	3	421330008	A421330008LAT/LON POINT AT CORNER OF TRAILER
Pennsylvania	York	2650 Delta Road	York-Hanover, PA	Harrisburg-York-Lebanon, PA	3	421330011	York DW
Rhode Island	Kent	W. ALTON JONES CAMPUS URI PARKERFIELD WE	Providence-Warwick, RI-MA	Boston-Worcester-Providence, MA-RI-NH-CT	1	440030002	AJ
Rhode Island	Providence	FRANCIS SCHOOL, 64 BOURNE AVE	Providence-Warwick, RI-MA	Boston-Worcester-Providence, MA-RI-NH-CT	1	440071010	FRANCIS SCHOOL East Providence
Rhode Island	Washington	TARZWELL ROAD, NARRAGANSETT	Providence-Warwick, RI-MA	Boston-Worcester-Providence, MA-RI-NH-CT	1	440090007	US-EPA Laboratory
South Carolina	Abbeville	59 JIM SCOTT LANE [Due West]	Greenwood, SC	Greenville-Spartanburg-Anderson, SC	4	450010001	DUE WEST
South Carolina	Aiken	8217 ATOMIC ROAD (INDIAN DRIVE) [Jackson]	Augusta-Richmond County, GA-SC		4	450030003	JACKSON MIDDLE SCHOOL
South Carolina	Anderson	215 McAlister Road [Big Creek]	Greenville-Anderson-Mauldin, SC	Greenville-Spartanburg-Anderson, SC	4	450070005	Big Creek
South Carolina	Berkeley	River Oak Drive (Goose Creek) [Bushy Park]	Charleston-North Charleston, SC		4	450150002	BUSHY PARK PUMP STATION
South Carolina	Charleston	390 BULLS ISLAND ROAD (AWENDAW) [Cape Romain]	Charleston-North Charleston, SC		4	450190046	CAPE ROMAIN
South Carolina	Cherokee	McGinnis Road [Cowpens]	Gaffney, SC	Greenville-Spartanburg-Anderson, SC	4	450210002	Cowpens
South Carolina	Chesterfield	RT 2 BOX 100 MCBEE (SC145) [Chesterfield]			4	450250001	CHESTERFIELD
South Carolina	Colleton	Ashton Road [Ashton]			4	450290002	ASHTON
South Carolina	Darlington	2200 POCKET ROAD [Pee Dee]	Florence, SC		4	450310003	Pee Dee Experimental Station
South Carolina	Edgefield	660 WOODYARD ROAD [Trenton]	Augusta-Richmond County, GA-SC		4	450370001	TRENTON
South Carolina	Greenville	510 Garrison Road [Hillcrest]	Greenville-Anderson-Mauldin, SC	Greenville-Spartanburg-Anderson, SC	4	450450016	Hillcrest Middle School
South Carolina	Oconee	ROUND MOUNTAIN TOWER ROAD [Long Creek]	Seneca, SC	Greenville-Spartanburg-Anderson, SC	4	450730001	LONG CREEK
South Carolina	Pickens	106 HOPEWELL ROAD [Clemson]	Greenville-Anderson-Mauldin, SC	Greenville-Spartanburg-Anderson, SC	4	450770002	CLEMSON CMS
South Carolina	Pickens	901 Allgood Bridge Road [Wolf Creek]	Greenville-Anderson-Mauldin, SC	Greenville-Spartanburg-Anderson, SC	4	450770003	Wolf Creek
South Carolina	Richland	8311 PARKLANE ROAD [Parklane]	Columbia, SC	Columbia-Orangeburg-Newberry, SC	4	450790007	PARKLANE
South Carolina	Richland	1850 SOUTH CEDAR CREEK ROAD [Congaree Bluff]	Columbia, SC	Columbia-Orangeburg-Newberry, SC	4	450790021	CONGAREE BLUFF

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South Carolina	Richland	900 CLEMSON ROAD [Sandhill]	Columbia, SC	Columbia-Orangeburg-Newberry, SC	4	450791001	SANDHILL EXPERIMENTAL STATION
South Carolina	Spartanburg	1556 JOHN DODD ROAD [North Spartanburg Fire Station #2]	Spartanburg, SC	Greenville-Spartanburg-Anderson, SC	4	450830009	NORTH SPARTANBURG FIRE STATION #2
South Carolina	York	2316 CHESTER HIGHWAY (US 321) [York]	Charlotte-Concord-Gastonia, NC-SC	Charlotte-Concord, NC-SC	4	450910006	YORK CMS
South Dakota	Brookings	3714 Western Ave, Brookings, SD 57006	Brookings, SD		8	460110003	Research Farm
South Dakota	Custer	WIND CAVE NATIONAL PARK, SOUTH DAKOTA	Rapid City, SD	Rapid City-Spearfish, SD	8	460330132	Wind Cave National Park, Visitor Center
South Dakota	Jackson	BADLANDS PO BOX 6 HEADQUARTERS			8	460710001	SOUTH OF BADLANDS NP HEADQUARTERS
South Dakota	Meade	7108 SEEAIRE ST - BLACK HAWK ELEMENTARY	Rapid City, SD	Rapid City-Spearfish, SD	8	460930001	BLACK HAWK ELEMENTARY SCHOOL GROUNDS
South Dakota	Minnehaha	2001 E 8th St	Sioux Falls, SD		8	460990008	SD School for the Deaf
South Dakota	Union	31986 475th Ave	Sioux City, IA-NE-SD	Sioux City-Vermillion, IA-SD-NE	8	461270001	Union County #1 Jensen
Tennessee	Anderson	FREELS BEND STUDY AREA MELTON LAKE Oak Ridge National Lab Reservation	Knoxville, TN	Knoxville-Morristown-Sevierville, TN	4	470010101	Free's Bend ozone and SO2 monitoring
Tennessee	Blount	GREAT SMOKY MOUNTAINS NP LOOK ROCK	Knoxville, TN	Knoxville-Morristown-Sevierville, TN	4	470090101	Great Smoky Mountains National Park, Look Rock
Tennessee	Blount	GREAT SMOKY MOUNTAINS NP - CADES COVE	Knoxville, TN	Knoxville-Morristown-Sevierville, TN	4	470090102	Great Smoky Mountains National Park, Cade's Cove
Tennessee	Claiborne	718 Russell Hill Rd, Speedwell, TN 37870			4	470259991	Speedwell
Tennessee	Davidson	1015 TRINITY LANE	Nashville-Davidson--Murfreesboro--Franklin, TN	Nashville-Davidson--Murfreesboro, TN	4	470370011	East Health
Tennessee	Davidson	3711 BELL ROAD	Nashville-Davidson--Murfreesboro--Franklin, TN	Nashville-Davidson--Murfreesboro, TN	4	470370026	Percy Priest Dam
Tennessee	DeKalb	Edgar Evans State Park, Smithville, TN 37166			4	470419991	Edgar Evans
Tennessee	Hamilton	SODDY DAISY H.S. 00618 SEQUOYAH RD	Chattanooga, TN-GA	Chattanooga-Cleveland-Dalton, TN-GA-AL	4	470651011	Soddy-Daisy High School
Tennessee	Hamilton	3018 HICKORY VALLEY RD- EASTSIDE UTILITY FILTER PLANT (RESERVOIR ROAD)	Chattanooga, TN-GA	Chattanooga-Cleveland-Dalton, TN-GA-AL	4	470654003	
Tennessee	Jefferson	2393 Forrester Road, New Market TN 37820	Morristown, TN	Knoxville-Morristown-Sevierville, TN	4	470890002	New Market ozone monitor
Tennessee	Knox	9315 RUTLEDGE PIKE MASCOT TN 37806	Knoxville, TN	Knoxville-Morristown-Sevierville, TN	4	470930021	East Knox Elementary School
Tennessee	Knox	4625 MILDRED DRIVE	Knoxville, TN	Knoxville-Morristown-Sevierville, TN	4	470931020	Spring Hill Elementary School
Tennessee	Loudon	130 WEBB DRIVE Loudon TN 37774	Knoxville, TN	Knoxville-Morristown-Sevierville, TN	4	471050108	Loudon Pope site
Tennessee	Sevier	GREAT SMOKY MOUNTAIN NP COVE MOUNTAIN	Sevierville, TN	Knoxville-Morristown-Sevierville, TN	4	471550101	

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Tennessee	Sevier	CLINGSMANS DOME, GREAT SMOKY MTNS. NP	Sevierville, TN	Knoxville-Morristown-Sevierville, TN	4	471550102	Great Smoky Mountains National Park, Clingman's Dome
Tennessee	Shelby	1330 FRAYSER BLVD	Memphis, TN-MS-AR	Memphis-Forrest City, TN-MS-AR	4	471570021	Frayser Ozone Monitor
Tennessee	Shelby	6388 Haley Rd. (Shelby Farms NCORE site)	Memphis, TN-MS-AR	Memphis-Forrest City, TN-MS-AR	4	471570075	Memphis NCORE site
Tennessee	Shelby	6855 MUDVILLE RD.	Memphis, TN-MS-AR	Memphis-Forrest City, TN-MS-AR	4	471571004	Edmund Orgill Park Ozone
Tennessee	Sullivan	Indian Springs School Shawnee Ave Blountville TN	Kingsport-Bristol-Bristol, TN-VA	Johnson City-Kingsport-Bristol, TN-VA	4	471632002	Blountville Ozone Monitor
Tennessee	Sullivan	3301 BLOOMINGDALE RD. Kingsport TN 37620	Kingsport-Bristol-Bristol, TN-VA	Johnson City-Kingsport-Bristol, TN-VA	4	471632003	Kingsport ozone monitor
Tennessee	Sumner	ROCKLAND RECREATION AREA-OLD HICKORY DAM Army Corp of Engineer Property	Nashville-Davidson--Murfreeseboro--Franklin, TN	Nashville-Davidson--Murfreeseboro, TN	4	471650007	Hendersonville Ozone Site at Old Hickory Dam
Tennessee	Williamson	FAIRVIEW MIDDLE SCHOOL CROW CUT ROAD Fairview TN 37062	Nashville-Davidson--Murfreeseboro--Franklin, TN	Nashville-Davidson--Murfreeseboro, TN	4	471870106	FAIRVIEW MIDDLE SCHOOL ozone monitor
Tennessee	Wilson	CEDARS OF LEBANON STATE PARK	Nashville-Davidson--Murfreeseboro--Franklin, TN	Nashville-Davidson--Murfreeseboro, TN	4	471890103	Cedars of Lebanon Ozone Monitor
Texas	Bell	8406 Georgia Avenue	Killeen-Temple, TX		6	480271045	Temple Georgia
Texas	Bell	1605 Stone Tree Drive	Killeen-Temple, TX		6	480271047	Killeen Skylark Field
Texas	Bexar	6655 Bluebird Lane	San Antonio-New Braunfels, TX		6	480290032	San Antonio Northwest
Texas	Bexar	F Range (1000Yd marker off Wilderness Trail)	San Antonio-New Braunfels, TX		6	480290052	Camp Bullis
Texas	Bexar	14620 Laguna Rd	San Antonio-New Braunfels, TX		6	480290059	Calaveras Lake
Texas	Brazoria	4503 Croix Pkwy	Houston-The Woodlands-Sugar Land, TX	Houston-The Woodlands, TX	6	480391004	Manvel Croix Park
Texas	Brazoria	109B Brazoria Hwy 332 West	Houston-The Woodlands-Sugar Land, TX	Houston-The Woodlands, TX	6	480391016	Lake Jackson
Texas	Brewster	BIG BEND NATIONAL PARK, TEXAS			6	480430101	Big Bend National Park, K-Bar Ranch Road
Texas	Cameron	344 Porter Drive	Brownsville-Harlingen, TX	Brownsville-Harlingen-Raymondville, TX	6	480610006	Brownsville
Texas	Cameron	1602 W Teege Avenue	Brownsville-Harlingen, TX	Brownsville-Harlingen-Raymondville, TX	6	480611023	Harlingen Teege
Texas	Collin	6590 Hillcrest Road	Dallas-Fort Worth-Arlington, TX	Dallas-Fort Worth, TX-OK	6	480850005	Frisco
Texas	Dallas	1415 Hinton Street	Dallas-Fort Worth-Arlington, TX	Dallas-Fort Worth, TX-OK	6	481130069	Dallas Hinton
Texas	Dallas	12532 1/2 Nuestra Drive	Dallas-Fort Worth-Arlington, TX	Dallas-Fort Worth, TX-OK	6	481130075	Dallas North #2
Texas	Dallas	3277 W Redbird Lane	Dallas-Fort Worth-Arlington, TX	Dallas-Fort Worth, TX-OK	6	481130087	Dallas Redbird Airport Executive
Texas	Denton	Denton Airport South	Dallas-Fort Worth-Arlington, TX	Dallas-Fort Worth, TX-OK	6	481210034	Denton Airport South

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Texas	Denton	792 E Northside Dr	Dallas-Fort Worth-Arlington, TX	Dallas-Fort Worth, TX-OK	6	481211032	Pilot Point
Texas	Ellis	2725 Old Fort Worth Road	Dallas-Fort Worth-Arlington, TX	Dallas-Fort Worth, TX-OK	6	481390016	Midlothian OFW
Texas	Ellis	900 FM 667 Ellis County	Dallas-Fort Worth-Arlington, TX	Dallas-Fort Worth, TX-OK	6	481391044	Italy
Texas	El Paso	10834 Ivanhoe (Ivanhoe Fire Station)	El Paso, TX	El Paso-Las Cruces, TX-NM	6	481410029	Ivanhoe
Texas	El Paso	250 Rim Rd	El Paso, TX	El Paso-Las Cruces, TX-NM	6	481410037	El Paso UTEP
Texas	El Paso	800 S San Marcial Street	El Paso, TX	El Paso-Las Cruces, TX-NM	6	481410044	El Paso Chamizal
Texas	El Paso	650 R E Thomason Loop	El Paso, TX	El Paso-Las Cruces, TX-NM	6	481410055	Ascarate Park SE
Texas	El Paso	320 Old Hueco Tanks Road	El Paso, TX	El Paso-Las Cruces, TX-NM	6	481410057	Socorro Hueco
Texas	El Paso	5050A Yvette Drive	El Paso, TX	El Paso-Las Cruces, TX-NM	6	481410058	Skyline Park
Texas	Galveston	9511 Avenue V 1/2	Houston-The Woodlands-Sugar Land, TX	Houston-The Woodlands, TX	6	481671034	Galveston 99th Street
Texas	Gregg	Gregg Co Airport near Longview	Longview, TX	Longview-Marshall, TX	6	481830001	Longview
Texas	Harris	4510 1/2 Aldine Mail Rd	Houston-The Woodlands-Sugar Land, TX	Houston-The Woodlands, TX	6	482010024	Houston Aldine
Texas	Harris	1405 Sheldon Road	Houston-The Woodlands-Sugar Land, TX	Houston-The Woodlands, TX	6	482010026	Channelview
Texas	Harris	16822 Kitzman	Houston-The Woodlands-Sugar Land, TX	Houston-The Woodlands, TX	6	482010029	Northwest Harris County
Texas	Harris	7330 1/2 North Wayside	Houston-The Woodlands-Sugar Land, TX	Houston-The Woodlands, TX	6	482010046	Houston North Wayside
Texas	Harris	4401 1/2 Lang Rd	Houston-The Woodlands-Sugar Land, TX	Houston-The Woodlands, TX	6	482010047	Lang
Texas	Harris	13826 1/2 Croquet	Houston-The Woodlands-Sugar Land, TX	Houston-The Woodlands, TX	6	482010051	Houston Croquet
Texas	Harris	6400 Bissonnet Street	Houston-The Woodlands-Sugar Land, TX	Houston-The Woodlands, TX	6	482010055	Houston Bayland Park
Texas	Harris	9726 1/2 Monroe	Houston-The Woodlands-Sugar Land, TX	Houston-The Woodlands, TX	6	482010062	Houston Monroe
Texas	Harris	3333 1/2 Hwy 6 South	Houston-The Woodlands-Sugar Land, TX	Houston-The Woodlands, TX	6	482010066	Houston Westhollow
Texas	Harris	7421 Park Place Blvd	Houston-The Woodlands-Sugar Land, TX	Houston-The Woodlands, TX	6	482010416	Park Place
Texas	Harris	4407 Independence Parkway South	Houston-The Woodlands-Sugar Land, TX	Houston-The Woodlands, TX	6	482011015	Lynchburg Ferry
Texas	Harris	8622 Garth Road Unit A	Houston-The Woodlands-Sugar Land, TX	Houston-The Woodlands, TX	6	482011017	Baytown Garth
Texas	Harris	1262 1/2 Mae Drive	Houston-The Woodlands-Sugar Land, TX	Houston-The Woodlands, TX	6	482011034	Houston East
Texas	Harris	9525 1/2 Clinton Dr	Houston-The Woodlands-Sugar Land, TX	Houston-The Woodlands, TX	6	482011035	Clinton
Texas	Harris	4514 1/2 Durant St	Houston-The Woodlands-Sugar Land, TX	Houston-The Woodlands, TX	6	482011039	Houston Deer Park #2

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Texas	Harris	4522 Park Rd	Houston-The Woodlands-Sugar Land, TX	Houston-The Woodlands, TX	6	482011050	Seabrook Friendship Park
Texas	Harrison	Hwy 134 & Spur 449	Marshall, TX	Longview-Marshall, TX	6	482030002	Karnack
Texas	Hidalgo	2300 North Glasscock	McAllen-Edinburg-Mission, TX	McAllen-Edinburg, TX	6	482150043	Mission
Texas	Hood	200 N Gordon Street	Dallas-Fort Worth-Arlington, TX	Dallas-Fort Worth, TX-OK	6	482210001	Granbury
Texas	Hunt	824 Sayle Street	Dallas-Fort Worth-Arlington, TX	Dallas-Fort Worth, TX-OK	6	482311006	Greenville
Texas	Jefferson	1086 Vermont Avenue	Beaumont-Port Arthur, TX		6	482450009	Beaumont Downtown
Texas	Jefferson	623 Elias Street	Beaumont-Port Arthur, TX		6	482450011	Port Arthur West
Texas	Jefferson	12552 Second St	Beaumont-Port Arthur, TX		6	482450022	Hamshire
Texas	Jefferson	5200 Mechanic	Beaumont-Port Arthur, TX		6	482450101	SETRPC 40 Sabine Pass
Texas	Jefferson	Jefferson County Airport	Beaumont-Port Arthur, TX		6	482450102	SETRPC 43 Jefferson Co Airport
Texas	Jefferson	Unavailable	Beaumont-Port Arthur, TX		6	482450628	SETRPC Port Arthur
Texas	Jefferson	1800 N. 18th Street	Beaumont-Port Arthur, TX		6	482451035	Nederland High School
Texas	Johnson	1650 Airport Drive	Dallas-Fort Worth-Arlington, TX	Dallas-Fort Worth, TX-OK	6	482510003	Cleburne Airport
Texas	Kaufman	3790 S Houston St	Dallas-Fort Worth-Arlington, TX	Dallas-Fort Worth, TX-OK	6	482570005	Kaufman
Texas	McLennan	4472 Mazanec Rd	Waco, TX		6	483091037	Waco Mazanec
Texas	Montgomery	9472A Hwy 1484	Houston-The Woodlands-Sugar Land, TX	Houston-The Woodlands, TX	6	483390078	Conroe Relocated
Texas	Navarro	Corsicana Airport	Corsicana, TX	Dallas-Fort Worth, TX-OK	6	483491051	Corsicana Airport
Texas	Nueces	Corpus Christi State School (Airport Rd)	Corpus Christi, TX	Corpus Christi-Kingsville-Alice, TX	6	483550025	Corpus Christi West
Texas	Nueces	9860 La Branch	Corpus Christi, TX	Corpus Christi-Kingsville-Alice, TX	6	483550026	Corpus Christi Tuloso
Texas	Orange	2700 Austin Ave	Beaumont-Port Arthur, TX		6	483611001	West Orange
Texas	Orange	INTERSECTION OF TX HWYS 62 AND 12	Beaumont-Port Arthur, TX		6	483611100	
Texas	Parker	3033 New Authon Rd	Dallas-Fort Worth-Arlington, TX	Dallas-Fort Worth, TX-OK	6	483670081	Parker County
Texas	Polk	361 Tombigbee Rd, Livingston, TX 77351			6	483739991	Alabama-Coushatta
Texas	Randall	Palo Duro Canyon State Park, Canyon, TX 79015	Amarillo, TX	Amarillo-Borger, TX	6	483819991	Palo Duro
Texas	Rockwall	100 E Heath St	Dallas-Fort Worth-Arlington, TX	Dallas-Fort Worth, TX-OK	6	483970001	Rockwall Heath
Texas	Smith	14790 County Road 1145	Tyler, TX	Tyler-Jacksonville, TX	6	484230007	Tyler Airport Relocated
Texas	Tarrant	14290 Morris Dido Newark Rd	Dallas-Fort Worth-Arlington, TX	Dallas-Fort Worth, TX-OK	6	484390075	Eagle Mountain Lake
Texas	Tarrant	3317 Ross Ave	Dallas-Fort Worth-Arlington, TX	Dallas-Fort Worth, TX-OK	6	484391002	Fort Worth Northwest
Texas	Tarrant	FAA Site off Alta Vista Road	Dallas-Fort Worth-Arlington, TX	Dallas-Fort Worth, TX-OK	6	484392003	Keller
Texas	Tarrant	4100 Fairway Dr	Dallas-Fort Worth-Arlington, TX	Dallas-Fort Worth, TX-OK	6	484393009	Grapevine Fairway
Texas	Tarrant	5504 South Collins Street	Dallas-Fort Worth-Arlington, TX	Dallas-Fort Worth, TX-OK	6	484393011	Arlington Municipal Airport

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Texas	Travis	3724 North Hills Dr	Austin-Round Rock, TX		6	484530014	Austin Northwest
Texas	Travis	12200 Lime Creek Rd	Austin-Round Rock, TX		6	484530020	Austin Audubon Society
Texas	Victoria	106 Mockingbird Lane	Victoria, TX	Victoria-Port Lavaca, TX	6	484690003	Victoria
Texas	Webb	2020 Vidaurri Ave	Laredo, TX		6	484790016	Laredo Vidaurri
Utah	Box Elder	140 W.FISHBURN DRIVE, BRIGHAM CITY, UT	Ogden-Clearfield, UT	Salt Lake City-Provo-Orem, UT	8	490030003	Brigham City
Utah	Box Elder	8600 WEST 24000 NORTH PORTAGE, UTAH	Ogden-Clearfield, UT	Salt Lake City-Provo-Orem, UT	8	490037001	WASHAKIE
Utah	Carbon	On Prperty of Resident at 351 W 2500 E. Price, Utah	Price, UT		8	490071003	
Utah	Davis	171 WEST 1370 NORTH, BOUNTIFUL, UTAH	Ogden-Clearfield, UT	Salt Lake City-Provo-Orem, UT	8	490110004	Bountiful Viewmont
Utah	Duchesne	290 S. 1000 W.			8	490130002	
Utah	Duchesne	6000 SOUTH AND 10000 WEST (MYTON)			8	490137011	STATION LOCATED NEAR INTERSECTION OF 6000 SO & 10000 WEST
Utah	Salt Lake	1675 SOUTH 600 EAST, SALT LAKE CITY	Salt Lake City, UT	Salt Lake City-Provo-Orem, UT	8	490353006	Hawthorne
Utah	San Juan	CANYONLANDS NATIONAL PARK, UTAH			8	490370101	Canyonlands National Park, Island in the Sky
Utah	Uintah	Dinosaur National Monument	Vernal, UT		8	490471002	
Utah	Uintah	2 Miles west of Redwash atop Deadman's Bench	Vernal, UT		8	490472002	
Utah	Uintah	2 miles south of Ouray and south of the White and Green River confluence	Vernal, UT		8	490472003	
Utah	Uintah	WHITEROCKS & COUNTY ROAD	Vernal, UT		8	490477022	STATION NEAR INTERSECTION OF WHITEROCK CANYON RD & QURAY CANAL
Utah	Utah	1355 NORTH 200 WEST PROVO UT	Provo-Orem, UT	Salt Lake City-Provo-Orem, UT	8	490490002	North Provo
Utah	Utah	312 W. 2050 NORTH, SPANISH FORK, UTAH	Provo-Orem, UT	Salt Lake City-Provo-Orem, UT	8	490495010	Spanish Fork
Utah	Washington	147 N 870 W, Hurrricane, Utah	St. George, UT		8	490530007	
Utah	Washington	ZION NATIONAL PARK, UTAH	St. George, UT		8	490530130	Zion National Park, Dalton's Wash
Utah	Weber	228 32ND STREET, OGDEN, UTAH	Ogden-Clearfield, UT	Salt Lake City-Provo-Orem, UT	8	490570002	Ogden
Utah	Weber	425 W 2550 NORTH, OGDEN, UTAH	Ogden-Clearfield, UT	Salt Lake City-Provo-Orem, UT	8	490571003	Harrisville
Vermont	Bennington	120 AIRPORT RD, BENNINGTON, VERMONT	Bennington, VT		1	500030004	Morse Airport - State of Vermont Property
Vermont	Chittenden	58 HARVEY ROAD, Underhill, Vermont	Burlington-South Burlington, VT		1	500070007	PROCTOR MAPLE RESEARCH CTR
Virginia	Albemarle	Lambs Lane, behind Albemarle High School Athletic Field	Charlottesville, VA		3	510030001	Albemarle High School
Virginia	Arlington	S 18TH AND HAYES ST	Washington-Arlington-Alexandria, DC-VA-MD-WV	Washington-Baltimore-Arlington, DC-MD-VA-WV-PA	3	510130020	Aurora Hills Visitors Center
Virginia	Caroline	U.S. GEODETIC SURVEY, OFF RT.2, CORBIN	Richmond, VA		3	510330001	USGS Geomagnetic Center, Corbin

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Virginia	Charles	SHIRLEY PLANTATION, ROUTE 5	Richmond, VA		3	510360002	Shirley Plantation
Virginia	Chesterfield	BEACH,INTERSECTION OF CO.ROADS 655 & 654	Richmond, VA		3	510410004	VDOT Chesterfield Residency Shop
Virginia	Fairfax	STA. 46-B9, LEE PARK, TELEGRAPH ROAD	Washington-Arlington-Alexandria, DC-VA-MD-WV	Washington-Baltimore-Arlington, DC-MD-VA-WV-PA	3	510590030	Lee District Park
Virginia	Fauquier	RT651 C PHELPS WILDLIFE MANAGEMENT AREA	Washington-Arlington-Alexandria, DC-VA-MD-WV	Washington-Baltimore-Arlington, DC-MD-VA-WV-PA	3	510610002	Chester Phelps Wildlife Management Area, Sumerduck
Virginia	Frederick	RTE 669, BUTLER MANUF. CO NEAR REST VA	Winchester, VA-WV	Washington-Baltimore-Arlington, DC-MD-VA-WV-PA	3	510690010	Rest
Virginia	Giles	1856 Horton Ln, Newport, VA 24128	Blacksburg-Christiansburg-Radford, VA		3	510719991	Horton Station
Virginia	Hanover	7345 MCCLELLAN RD MECHANICSVILLE VA	Richmond, VA		3	510850003	Turner Property, Old Church
Virginia	Henrico	2401 HARTMAN STREET MATH & SCIENCE CTR	Richmond, VA		3	510870014	MathScience Innovation Center
Virginia	Loudoun	38-I, BROAD RUN HIGH SCHOOL, ASHBURN	Washington-Arlington-Alexandria, DC-VA-MD-WV	Washington-Baltimore-Arlington, DC-MD-VA-WV-PA	3	511071005	Broad Run High School, Ashburn
Virginia	Madison	SHENANDOAH NP BIG MEADOWS			3	511130003	Shenandoah National Park, Big Meadows
Virginia	Prince Edward	Prince Edward-Gallion State Forest, Burkeville, VA 23922			3	511479991	Prince Edward
Virginia	Prince William	JAMES S. LONG PARK	Washington-Arlington-Alexandria, DC-VA-MD-WV	Washington-Baltimore-Arlington, DC-MD-VA-WV-PA	3	511530009	James S. Long Park
Virginia	Roanoke	HERMAN HORN ELEMENTARY SCHOOL	Roanoke, VA		3	511611004	East Vinton Elementary School
Virginia	Rockbridge	21-C.GLENWOOD-PEDLAR DISTRICT RANGER STA			3	511630003	Natural Bridge Ranger Station
Virginia	Rockingham	VDOT RESIDENCY SHOP, 3536 NORTH VALLEY PIKE, HARRISONBURG, VA	Harrisonburg, VA	Harrisonburg-Staunton-Waynesboro, VA	3	511650003	ROCKINGHAM CO. VDOT
Virginia	Stafford	WIDEWATER ELEM. SCH., DEN RICH ROAD	Washington-Arlington-Alexandria, DC-VA-MD-WV	Washington-Baltimore-Arlington, DC-MD-VA-WV-PA	3	511790001	Widewater Elementary School
Virginia	Wythe	16-B RURAL RETREAT SEWAGE DISPOSAL			3	511970002	Rural Retreat Sewage Treatment Plant
Virginia	Hampton City	Adjacent to Building 1196, Wythe Landing Loop, NASA-Langley Research Center	Virginia Beach-Norfolk-Newport News, VA-NC	Virginia Beach-Norfolk, VA-NC	3	516500008	NASA Langley Research Center
Virginia	Suffolk City	TIDEWATER COMM. COLLEGE, FREDERIC CAMPUS	Virginia Beach-Norfolk-Newport News, VA-NC	Virginia Beach-Norfolk, VA-NC	3	518000004	Tidewater Community College
Virginia	Suffolk City	TIDEWATER RESEARCH STATION, HARE ROAD	Virginia Beach-Norfolk-Newport News, VA-NC	Virginia Beach-Norfolk, VA-NC	3	518000005	VA Tech Agricultural Research Station, Holland
Washington	Clallam	LOCATED IN A TREE FARM (Cheeka Peak)	Port Angeles, WA		10	530090013	CHEEKA PEAK
Washington	Clark	1500 SE BLAIRMONT DR (Mountain View High School)	Portland-Vancouver-Hillsboro, OR-WA	Portland-Vancouver-Salem, OR-WA	10	530110011	VANCOUVER - BLAIRMONT DR

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Washington	King	2000 NW SAMMAMISH RD	Seattle-Tacoma-Bellevue, WA	Seattle-Tacoma, WA	10	530330010	ISSAQUAH - LAKE SAMMAMISH (Within Lake Sammamish State Park)
Washington	King	42404 SE NORTH BEND WAY	Seattle-Tacoma-Bellevue, WA	Seattle-Tacoma, WA	10	530330017	NORTH BEND - NORTH BEND WAY
Washington	King	30525 SE MUD MOUNTAIN ROAD	Seattle-Tacoma-Bellevue, WA	Seattle-Tacoma, WA	10	530330023	ENUMCLAW - MUD MTN (Army Corp of Engineers site)
Washington	King	4103 BEACON HILL S	Seattle-Tacoma-Bellevue, WA	Seattle-Tacoma, WA	10	530330080	SEATTLE - BEACON HILL
Washington	Pierce	JACKSON VISITOR'S CENTER MT RAINIER, WA	Seattle-Tacoma-Bellevue, WA	Seattle-Tacoma, WA	10	530530012	MT RAINIER - JACKSON VISITORS CENTER
Washington	Skagit	202 O Ave	Mount Vernon-Anacortes, WA	Seattle-Tacoma, WA	10	530570011	Anacortes-202 Ave
Washington	Skagit	Northwest Indian College, 17113 Tallawhalt Lane	Mount Vernon-Anacortes, WA	Seattle-Tacoma, WA	10	530570020	
Washington	Spokane	S 26010 Smith Road (Turnbull Slough National Wildlife Refuge)	Spokane-Spokane Valley, WA	Spokane-Spokane Valley-Coeur d'Alene, WA-ID	10	530630001	CHENEY - TURNBULL
Washington	Spokane	E 9814 GREENBLUFF RD, GREENBLUFF	Spokane-Spokane Valley, WA	Spokane-Spokane Valley-Coeur d'Alene, WA-ID	10	530630046	SPOKANE - GREENBLUFF
Washington	Thurston	931 NORTHERN PACIFIC ROAD	Olympia-Tumwater, WA	Seattle-Tacoma, WA	10	530670005	YELM - NORTHERN PACIFIC
Washington	Whatcom	1330 LOOMIS TRAIL RD	Bellingham, WA		10	530730005	CUSTER - LOOMIS
West Virginia	Berkeley	MARTINSBURG - BALL FIELD	Hagerstown-Martinsburg, MD-WV	Washington-Baltimore-Arlington, DC-MD-VA-WV-PA	3	540030003	MARTINSBURG BALL FIELD
West Virginia	Cabell	HUNTINGTON - MARSHALL UNIVERSITY	Huntington-Ashland, WV-KY-OH	Charleston-Huntington-Ashland, WV-OH-KY	3	540110006	HENDERSON CENTER/MARSHALL UNIVERSITY - MOVED FROM WATER CO. 5/98
West Virginia	Gilmer	Cedar Creek State Park, Cedarville, WV 26611			3	540219991	Cedar Creek
West Virginia	Greenbrier	SAM BLACK CHURCH - DOH GARAGE			3	540250003	SAM BLACK CHURCH - DOH GARAGE - GREENBRIER COUNTY
West Virginia	Monongalia	MORGANTOWN - AIRPORT US 119&AIRPORT BLVD.	Morgantown, WV	Morgantown-Fairmont, WV	3	540610003	
West Virginia	Ohio	WHEELING - WARWOOD WATER PLANT	Wheeling, WV-OH		3	540690010	
West Virginia	Tucker	USDA Northern Research Station, Monongahela National Forest, Parsons, WV 26287			3	540939991	Parsons
West Virginia	Wood	VIENNA - NEALE ELEM SCHOOL	Parkersburg-Vienna, WV	Parkersburg-Marietta-Vienna, WV-OH	3	541071002	Neale Elementary School
Wisconsin	Ashland	BAD RIVER TRIBAL SCHOOL - ODANAH			5	550030010	BAD RIVER TRIBAL SCHOOL - ODANAH
Wisconsin	Brown	UW-GREEN BAY, HWYS 54 & 57	Green Bay, WI	Green Bay-Shawano, WI	5	550090026	GREEN BAY - UW
Wisconsin	Columbia	N 1045 WENDT RD, COLUMBUS	Madison, WI	Madison-Janesville-Beloit, WI	5	550210015	COLUMBUS
Wisconsin	Dane	2302 HOARD ST	Madison, WI	Madison-Janesville-Beloit, WI	5	550250041	MADISON EAST

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Wisconsin	Dodge	Horicon Wildlife Area, 1210 N Palmatory St	Beaver Dam, WI	Milwaukee-Racine-Waukesha, WI	5	550270001	HORICON WILDLIFE AREA
Wisconsin	Door	475 CTH NP			5	550290004	NEWPORT PARK
Wisconsin	Eau Claire	5505 Highway 53 South Eau Claire	Eau Claire, WI	Eau Claire-Menomonie, WI	5	550350014	EAU CLAIRE - DOT SIGN SHOP
Wisconsin	Fond du Lac	N3996 KELLY RD, TWN BYRON	Fond du Lac, WI		5	550390006	FOND DU LAC
Wisconsin	Forest	FIRE TOWER RD, POTAWATOMI SITE			5	550410007	POTAWATOMI
Wisconsin	Jefferson	N4440 Laatsch Lane	Watertown-Fort Atkinson, WI	Milwaukee-Racine-Waukesha, WI	5	550550009	JEFFERSON - LAATSCH
Wisconsin	Kenosha	CHIWAUKEE PRAIRIE, 11838 FIRST COURT	Chicago-Naperville-Elgin, IL-IN-WI	Chicago-Naperville, IL-IN-WI	5	550590019	CHIWAUKEE PRAIRIE STA TELINE
Wisconsin	Kewaunee	KEWAUNEE, ROUTE 1, HWY 42	Green Bay, WI	Green Bay-Shawano, WI	5	550610002	KEWAUNEE
Wisconsin	La Crosse	3550 MORMON COULEE RD	La Crosse-Onalaska, WI-MN		5	550630012	LACROSSE - DOT BUILDING
Wisconsin	Manitowoc	2315 EAST GOODWIN RD	Manitowoc, WI		5	550710007	MANITOWOC - WDLND DUNES
Wisconsin	Marathon	LAKE DUBAY, 1780 BERGEN RD, BERGEN TNSHP	Wausau, WI	Wausau-Stevens Point-Wisconsin Rapids, WI	5	550730012	LAKE DUBAY
Wisconsin	Milwaukee	HEALTH CENTER, 1337 SO 16TH ST	Milwaukee-Waukesha-West Allis, WI	Milwaukee-Racine-Waukesha, WI	5	550790010	MILWAUKEE - SIXTEENTH ST. HEALTH CENTER
Wisconsin	Milwaukee	2300 N M. L. KING JR DR	Milwaukee-Waukesha-West Allis, WI	Milwaukee-Racine-Waukesha, WI	5	550790026	MILWAUKEE - SER DNR HDQRS
Wisconsin	Milwaukee	601 E. ELLSWORTH LANE	Milwaukee-Waukesha-West Allis, WI	Milwaukee-Racine-Waukesha, WI	5	550790085	BAYSIDE
Wisconsin	Outagamie	AAL, 4432 N MEADE ST	Appleton, WI	Appleton-Oshkosh-Neenah, WI	5	550870009	APPLETON - AAL
Wisconsin	Ozaukee	N. Port Washington Rd., East side of HWY32 AND I43, GRAFTON	Milwaukee-Waukesha-West Allis, WI	Milwaukee-Racine-Waukesha, WI	5	550890008	GRAFTON
Wisconsin	Ozaukee	531 HWY D	Milwaukee-Waukesha-West Allis, WI	Milwaukee-Racine-Waukesha, WI	5	550890009	HARRINGTON BEACH PARK
Wisconsin	Rock	1501 Ritsher St.	Janesville-Beloit, WI	Madison-Janesville-Beloit, WI	5	551050030	BELOIT - CONVERSE
Wisconsin	Sauk	DEVILS LAKE STATE PARK, E12886 TOWER RD	Baraboo, WI	Madison-Janesville-Beloit, WI	5	551110007	DEVILS LAKE PARK
Wisconsin	Sheboygan	KOHLER ANDRE PARK, 1520 Beach Park Rd.	Sheboygan, WI		5	551170006	SHEBOYGAN - KOHLER ANDRAE
Wisconsin	Taylor	W 10746 County Highway M, Medford, WI 54451			5	551199991	Perkinstown
Wisconsin	Vilas	TROUT LAKE NURSERY, 10810 COUNTY HWY M			5	551250001	TROUT LAKE
Wisconsin	Walworth	2420 ELGIN CLUB RD	Whitewater-Elkhorn, WI	Milwaukee-Racine-Waukesha, WI	5	551270005	LAKE GENEVA
Wisconsin	Waukesha	1310 CLEVELAND AVE	Milwaukee-Waukesha-West Allis, WI	Milwaukee-Racine-Waukesha, WI	5	551330027	WAUKESHA - CLEVELAND AVE
Wyoming	Albany	Roosevelt National Forest, Centennial, WY 82055	Laramie, WY		8	560019991	Centennial
Wyoming	Big Horn	Basin (WARMS station)			8	560030002	Basin
Wyoming	Campbell	THUNDER BASIN GRASSLAND SITE 35 MI N-NE GILLETTE WY	Gillette, WY		8	560050123	Thunder Basin

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Wyoming	Campbell	SOUTH CAMPBELL COUNTY APPROX 15 MILES SSW OF GILLETTE WY (SEE APPLE BUTTE QUAD MAP)	Gillette, WY		8	560050456	Campbell County
Wyoming	Carbon	ATLANTIC RIM SUNDG LOCATIONS			8	560070100	Atlantic Rim Sun Dog
Wyoming	Carbon	SINCLAIR-Intersection of CCR351 and N. 8th St. (SW corner of intersection)			8	560071000	Sinclair-Mobile #2
Wyoming	Converse	HWY 59			8	560090008	Tallgrass Energy Partners - Gaseous
Wyoming	Fremont	South Pass WyDot	Riverton, WY		8	560130099	South Pass
Wyoming	Fremont	Spring Creek Encana	Riverton, WY		8	560130232	
Wyoming	Laramie	NCore - North Cheyenne Soccer Complex	Cheyenne, WY		8	560210100	Cheyenne NCore
Wyoming	Natrona	2800 Pheasant Drive, Casper	Casper, WY		8	560250100	Casper Gaseous
Wyoming	Natrona	Sinclair, Casper	Casper, WY		8	560252601	
Wyoming	Sublette	Boulder APPROX 3 MILES WEST OF BOULDER			8	560350099	Boulder
Wyoming	Sublette	DANIEL SOUTH ~ 4 MILES SO OF DANIEL WYO OFF OF HWY 189			8	560350100	Daniel South
Wyoming	Sublette	Pinedale Gaseous Monitor on west side of City Park and Pine Creek			8	560350101	Pinedale Gaseous
Wyoming	Sublette	Big Piney Site #3			8	560350700	Big Piney Site #3
Wyoming	Sublette	Juel Spring - 20 miles NW of Farson on the west side of US Highway 191			8	560351002	Juel Spring
Wyoming	Sublette	Skyline Dr, Pinedale, WY 82941			8	560359991	Pinedale
Wyoming	Sweetwater	Hiawatha Green Site	Rock Springs, WY		8	560370077	Hiawatha
Wyoming	Sweetwater	WAMSUTTER SOUTHEAST PORTION OF SWEETWATER COUNTY	Rock Springs, WY		8	560370200	Wamsutter
Wyoming	Sweetwater	Moxa	Rock Springs, WY		8	560370300	Moxa
Wyoming	Teton	Grand Teton NP - Science School	Jackson, WY-ID		8	560390008	Grand Teton NP - Science School
Wyoming	Teton	YELLOWSTONE NATIONAL PARK	Jackson, WY-ID		8	560391011	Yellowstone National Park, Water Tank
Wyoming	Uinta	Murphy Ridge LOCATED NEAR WYOMING UTAH BORDER	Evanston, WY		8	560410101	Murphy Ridge
Wyoming	Weston	Newcastle, WARMS station			8	560450003	
Puerto Rico	Bayamon	Ave. Central Correccional	San Juan-Carolina-Caguas, PR	San Juan-Carolina, PR	2	720210010	
Puerto Rico	Catano	PR 165	San Juan-Carolina-Caguas, PR	San Juan-Carolina, PR	2	720330008	
Puerto Rico	Juncos	Rd. 183	San Juan-Carolina-Caguas, PR	San Juan-Carolina, PR	2	720770001	

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Alabama	Baldwin	FAIRHOPE HIGH SCHOOL, FAIRHOPE, ALABAMA	30.498001	-87.881412	96	91	95	94	0.070	0.063	0.062	0.065
Alabama	Colbert	WILSON DAM RD AND 2ND ST.	34.758781	-87.650562	100	97	97	98	0.059	0.057	0.061	0.059
Alabama	DeKalb	Sand Mountain Alabama Agricultural Experiment Station, Crossville, AL 35962	34.289000	-85.970070	99	98	92	96	0.062	0.065	0.064	0.063
Alabama	Elmore	DEWBERRY TRAIL, ELMORE CO., WETUMPKA, ALABAMA	32.498567	-86.136587	99	93	47	80	0.060	0.061	0.057	0.059
Alabama	Etowah	1450 PARKER ANDERSON LANE, SOUTHSIDE, AL 35907	33.904039	-86.053867	99	97	98	98	0.059	0.060	0.064	0.061
Alabama	Houston	161 BUFORD LANE	31.190657	-85.423117	99	98	97	98	0.059	0.061	0.059	0.059
Alabama	Jefferson	NO. B'HAM,SOU R.R., 3009 28TH ST. NO.	33.553056	-86.815000	96	100	93	96	0.065	0.071	0.069	0.068
Alabama	Jefferson	FAIRFIELD, PFD, 5229 COURT B	33.485556	-86.915000	96	98	99	98	0.065	0.068	0.066	0.066
Alabama	Jefferson	ROUTE 8 MCADORY	33.331111	-87.003611	78	86	97	87	0.065	0.065	0.069	0.066
Alabama	Jefferson	201 ASHVILLE ROAD	33.545278	-86.549167	100	85	93	93	0.063	0.062	0.067	0.064
Alabama	Jefferson	3425 TAMASSEE LANE, HOOVER, AL 35226	33.386389	-86.816667	99	100	98	99	0.062	0.068	0.070	0.066
Alabama	Jefferson	10005 CORNER SCHOOL ROAD	33.801667	-86.942500	85	84	96	88	0.061	0.066	0.067	0.064
Alabama	Jefferson	TARRANT, ELEM. SCH., 1269 PORTLAND STREE	33.578333	-86.773889	98	93	98	96	0.063	0.073	0.070	0.068
Alabama	Madison	2201 AIRPORT ROAD	34.687670	-86.586370	100	98	99	99	0.064	0.063	0.066	0.064
Alabama	Madison	1130 Capshaw Road	34.772727	-86.756174	100	99	99	99	0.061	0.061	0.064	0.062
Alabama	Mobile	CHICKASAW, MOBILE CO., ALABAMA	30.769941	-88.087526	93	96	94	94	0.068	0.060	0.062	0.063
Alabama	Mobile	BAY RD. ,MOBILE AL.	30.474674	-88.141140	97	97	98	97	0.070	0.063	0.063	0.065
Alabama	Montgomery	1350 COLISEUM BLVD, MONTGOMERY, ALABAMA 36110	32.407120	-86.256367	98	98	100	99	0.062	0.063	0.063	0.062
Alabama	Morgan	P.O. BOX 2224 WALLACE DEVELOPMENT CENTER. DECATUR, ALABAMA	34.518735	-86.976939	98	99	99	99	0.062	0.063	0.067	0.064
Alabama	Russell	9 WOODLAND DRIVE (SCHOOL) LADONIA, AL 36869	32.467971	-85.083841	98	99	94	97	0.058	0.062	0.066	0.062
Alabama	Shelby	HELENA, BEARDEN FARM	33.317316	-86.825092	99	96	99	98	0.063	0.065	0.073	0.067
Alabama	Sumter	NNE of Ward Post Office, Sumter Co., Alabama	32.362706	-88.277954	97	89	93	93	0.058	0.057	0.058	0.057
Alabama	Tuscaloosa	11690 SOUTHFORK DR. DUNCANVILLE, AL_35456	33.089600	-87.459731	97	97	100	98	0.057	0.062	0.061	0.060
Alaska	Denali	DENALI NATIONAL PARK	63.723200	-148.967600	100	100	95	98	0.058	0.054	0.048	0.053
Alaska	Fairbanks North Star	809 Pioneer Road	64.845690	-147.727413	99	98	99	99	0.044	0.045	0.039	0.042
Arizona	Cochise	CHIRICAHUA NATIONAL MOUMENT	32.009410	-109.389060	97	99	98	98	0.068	0.065	0.064	0.065
Arizona	Coconino	755 W BONITO	35.206111	-111.652777	99	99	99	99	0.073	0.070	0.064	0.069
Arizona	Coconino	GRAND CANYON NATIONAL PARK, W RIM DRIVE	36.058640	-112.183580	96	98	98	97	0.067	0.070	0.064	0.067
Arizona	Gila	SOUTH OF SR88	33.654700	-111.107400	100	96	99	98	0.072	0.073	0.070	0.071
Arizona	La Paz	ALAMO LAKE STATE PARK	34.231900	-113.580000	95	96	82	91	0.071	0.070	0.067	0.069

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Arizona	Maricopa	3847 W EARLL DR-WEST PHOENIX STATION	33.483850	-112.142570	100	100	100	100	0.076	0.074	0.071	0.073
Arizona	Maricopa	310 S BROOKS, MESA	33.410450	-111.865070	99	100	100	100	0.078	0.077	0.075	0.076
Arizona	Maricopa	601 E BUTLER DR & N 6TH ST,PHOENIX	33.560330	-112.066260	100	100	99	100	0.078	0.074	0.075	0.075
Arizona	Maricopa	4530 E MCKELLIPS RD-FALCON FIELD STATION	33.452230	-111.733310	100	100	99	100	0.076	0.072	0.073	0.073
Arizona	Maricopa	6000 W OLIVE AVE, GLENDALE	33.574540	-112.191960	100	98	99	99	0.071	0.067	0.066	0.068
Arizona	Maricopa	25000 N WINDY WALK-PINNACLE PEAK STATION	33.706328	-111.855622	98	98	100	99	0.080	0.077	0.074	0.077
Arizona	Maricopa	1645 E ROOSEVELT ST-CENTRAL PHOENIX STN	33.457930	-112.046010	100	99	100	100	0.071	0.071	0.070	0.070
Arizona	Maricopa	2857 N MILLER RD-S SCOTTSDALE STN	33.479680	-111.917210	100	98	99	99	0.072	0.068	0.070	0.070
Arizona	Maricopa	33 W TAMARISK AVE, PHOENIX	33.403160	-112.075330	99	100	99	99	0.073	0.070	0.067	0.070
Arizona	Maricopa	275 S ELLIS, CHANDLER	33.298980	-111.884310	100	100	100	100	0.070	0.070	0.069	0.069
Arizona	Maricopa	1525 S COLLEGE AVE, TEMPE	33.412400	-111.934730	100	12	100	71	0.071	0.051	0.068	0.063
Arizona	Maricopa	37019 N. LAVA LANE. CAVE CREEK AZ	33.821690	-112.017390	100	100	100	100	0.074	0.069	0.071	0.071
Arizona	Maricopa	16825 N. DYSART	33.637130	-112.341840	100	100	100	100	0.070	0.067	0.063	0.066
Arizona	Maricopa	26453 W. MC85	33.370050	-112.620700	100	98	99	99	0.060	0.060	0.059	0.059
Arizona	Maricopa	4208 W. PECOS LAVEEN, AZ 85339 (GILA CROSSING NORTH ELEM SCH)	33.290229	-112.160597	99	95	68	87	0.069	0.067	0.065	0.067
Arizona	Maricopa	10844 EAST OSBORN ROAD SCOTTSDALE AZ 85256	33.488242	-111.855654	98	100	99	99	0.073	0.073	0.070	0.072
Arizona	Maricopa	15115 BEELINE HIGHWAY, SCOTTSDALE, AZ 86256	33.507992	-111.755334	98	100	98	99	0.080	0.074	0.074	0.076
Arizona	Maricopa	3250 NORTH STAPLEY DRIVE, MESA, AZ 85203	33.474609	-111.805769	97	100	99	99	0.076	0.076	0.072	0.074
Arizona	Maricopa	4827 NORTH COUNTRY CLUB DRIVE, SCOTTSDALE, AZ 85256	33.508125	-111.838520	96	96	98	97	0.071	0.072	0.070	0.071
Arizona	Maricopa	7 SPRINGS RD-FAA RADAR STN-TONTO NF	33.982800	-111.798700	100	97	100	99	0.074	0.073	0.072	0.073
Arizona	Maricopa	USERY PASS RD/BUSH HWY	33.545490	-111.609250	99	99	100	99	0.074	0.073	0.071	0.072
Arizona	Maricopa	16426 E PALISADES BLVD, FOUNTAIN HILLS, AZ	33.611030	-111.725290	61	100	100	87	0.068	0.069	0.068	0.068
Arizona	Maricopa	25608 N FOREST RD., RIO VERDE, AZ	33.718810	-111.671830	99	100	100	100	0.073	0.068	0.070	0.070
Arizona	Maricopa	4530 N 17TH AVENUE	33.503833	-112.095767	100	98	93	97	0.077	0.075	0.075	0.075
Arizona	Navajo	PET FOR NAT PARK, NEAR OLD SW ENTERANCE ON OLD ROUTE 180	34.822510	-109.892490	99	86	96	94	0.068	0.061	0.063	0.064
Arizona	Pima	3905 S. OLD SPANISH TRAIL, SAGUARO PARK EAST	32.174538	-110.737116	100	100	99	100	0.069	0.066	0.069	0.068
Arizona	Pima	1237 S. BEVERLY, TUCSON	32.204411	-110.878067	100	99	99	99	0.062	0.065	0.061	0.062
Arizona	Pima	12101 N CAMINO DE OESTE, TUCSON	32.425261	-111.063520	100	100	99	100	0.065	0.065	0.064	0.064
Arizona	Pima	11330 S HOUGHTON, TUCSON	32.047670	-110.774357	100	96	99	98	0.063	0.064	0.066	0.064

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Arizona	Pima	400 W RIVER ROAD	32.295150	-110.982300	100	100	99	100	0.066	0.063	0.063	0.064
Arizona	Pima	601 NORTH LA CANADA DRIVE	31.879520	-110.996440	99	98	98	98	0.065	0.059	0.066	0.063
Arizona	Pima	710 W. MICHIGAN ST.	32.173000	-110.980115	100	98	99	99	0.061	0.065	0.066	0.064
Arizona	Pima	9597 N. COACHLINE BLVD	32.380820	-111.127160	100	100	99	100	0.062	0.062	0.060	0.061
Arizona	Pinal	305 E. SUPERSTITION BLVD, APACHE JUNCTION	33.421400	-111.543600	97	97	97	97	0.066	0.073	0.072	0.070
Arizona	Pinal	660 W. AERO DR. CASA GRANDE	32.954361	-111.762250	99	100	95	98	0.065	0.066	0.066	0.065
Arizona	Pinal	WATER WELL #2 PINAL AIR PARK RD MARANA	32.508306	-111.308056	100	100	95	98	0.065	0.066	0.066	0.065
Arizona	Pinal	35 PINA STREET, SACATON	33.080087	-111.739999	99	100	68	89	0.066	0.064	0.066	0.065
Arizona	Pinal	10 S QUEEN ANN QUEEN VALLEY	33.293465	-111.285594	98	98	98	98	0.068	0.074	0.072	0.071
Arizona	Yavapai	330 Grove Avenue	34.546700	-112.476100	99	90	94	94	0.077	0.067	0.064	0.069
Arizona	Yuma	2323 S ARIZONA AVE	32.690278	-114.614440	98	92	100	97	0.078	0.077	0.067	0.074
Arkansas	Clark	Lower Lake Recreation Area, Caddo Valley, AR 71923	34.179500	-93.098800	99	90	96	95	0.059	0.060	0.055	0.058
Arkansas	Crittenden	LH POLK AND COLONIAL DRIVE	35.197288	-90.193141	100	100	99	100	0.067	0.066	0.070	0.067
Arkansas	Newton	HWY 16	35.832726	-93.208260	100	100	100	100	0.062	0.061	0.056	0.059
Arkansas	Polk	463 POLK 631	34.454406	-94.143316	97	99	100	99	0.063	0.065	0.060	0.062
Arkansas	Pulaski	PIKE AVE AT RIVER ROAD	34.756189	-92.281296	100	100	100	100	0.066	0.061	0.065	0.064
Arkansas	Pulaski	REMOUNT ROAD N. LITTLE ROCK, ARKANSAS	34.835721	-92.260581	100	100	100	100	0.065	0.065	0.063	0.064
Arkansas	Washington	600 SOUTH OLD MISSOURI ROAD	36.179700	-94.116827	100	100	100	100	0.061	0.064	0.056	0.060
Arkansas	Washington	429 Ernest Lancaster Dr.	36.011703	-94.167436	99	100	100	100	0.062	0.061	0.058	0.060
California	Alameda	793 Rincon Ave.	37.687526	-121.784217	99	99	98	99	0.076	0.074	0.072	0.074
California	Alameda	9925 International Blvd	37.743065	-122.169935	99	98	99	99	0.057	0.055	0.054	0.055
California	Alameda	1100 21st Street	37.814781	-122.282347	99	98	93	97	0.051	0.052	0.045	0.049
California	Alameda	3466 LA MESA DR.	37.654456	-122.031547	100	99	100	100	0.072	0.064	0.036	0.057
California	Amador	201 CLINTON ROAD, JACKSON, CA 95642	38.339905	-120.763515	98	100	100	99	0.074	0.074	0.071	0.073
California	Butte	4405 AIRPORT RD-PARADISE CA	39.714041	-121.618836	99	100	99	99	0.074	0.075	0.077	0.075
California	Butte	984 East Avenue, Chico	39.761538	-121.841620	97	98	99	98	0.066	0.067	0.067	0.066
California	Calaveras	501 GOLD STRIKE ROAD, SAN ANDREAS	38.201852	-120.681567	99	100	100	100	0.071	0.081	0.077	0.076
California	Colusa	100 SUNRISE BLVD., COLUSA	39.202935	-122.017728	99	94	97	97	0.061	0.064	0.065	0.063

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California	Contra Costa	2956-A TREAT BOULEVARD	37.936013	-122.026154	98	99	99	99	0.067	0.070	0.065	0.067
California	Contra Costa	5551 BETHEL ISLAND RD	38.006311	-121.641918	97	98	99	98	0.069	0.068	0.069	0.068
California	Contra Costa	1865 D RUMRILL BLVD, San Pablo	37.960400	-122.356811	99	99	98	99	0.055	0.059	0.049	0.054
California	Contra Costa	9885 Alcosta BLVD	37.743649	-121.934188	98	99	98	98	0.072	0.074	0.062	0.069
California	El Dorado	3111 GOLD NUGGET WAY, PLACERVILLE, CA	38.725282	-120.821916	99	99	100	99	0.082	0.080	0.093	0.085
California	El Dorado	1400 AMERICAN RIVER TRAIL, COOL	38.890941	-121.003373	93	97	99	96	0.083	0.080	0.084	0.082
California	Fresno	4706 E. DRUMMOND ST., FRESNO	36.705506	-119.741966	89	87	93	90	0.084	0.088	0.088	0.086
California	Fresno	3727 N FIRST ST, FRESNO	36.785322	-119.774174	98	98	97	98	0.090	0.087	0.091	0.089
California	Fresno	SIERRA SKYPARK#2-BLYTHE & CHNNLT, FRESNO	36.841331	-119.875415	87	93	97	92	0.091	0.084	0.084	0.086
California	Fresno	32650 West Adams Avenue Tranquillity CA 93668	36.634225	-120.382331	87	97	93	92	0.075	0.077	0.077	0.076
California	Fresno	9240 S. RIVERBEND, PARLIER 93648	36.597450	-119.504569	93	91	96	93	0.087	0.093	0.093	0.091
California	Fresno	908 N VILLA AVE, CLOVIS	36.819111	-119.717356	89	90	93	91	0.097	0.093	0.092	0.094
California	Glenn	720 N Colusa Street, Willows	39.533761	-122.191903	99	98	96	98	0.067	0.064	0.061	0.064
California	Humboldt	717 SOUTH AVENUE	40.776944	-124.177500	87	83	93	88	0.043	0.045	0.043	0.043
California	Humboldt	170 meters SE of Donna Dr. & Humboldt Hill Rd., Eureka, CA	40.715280	-124.201390	95	90	83	89	0.041	0.047	0.049	0.045
California	Imperial	1029 ETHEL ST, CALEXICO HIGH SCHOOL	32.676186	-115.484144	91	99	97	96	0.078	0.077	0.074	0.076
California	Imperial	150 9TH ST., EL CENTRO	32.792220	-115.563060	99	95	62	85	0.078	0.077	0.074	0.076
California	Imperial	7711 ENGLISH ROAD, NILAND	33.213611	-115.545280	93	89	92	91	0.069	0.071	0.062	0.067
California	Kern	JOHNSON FARM, EDISON, CA. 93320	35.346094	-118.852037	84	92	99	92	0.085	0.090	0.086	0.087
California	Kern	755 Stanislaus St., Maricopa CA 93352	35.054430	-119.404826	96	97	97	97	0.078	0.083	0.083	0.081
California	Kern	923 POOLE STREET, MOJAVE, CA 93501	35.050551	-118.147294	93	98	96	96	0.089	0.080	0.084	0.084
California	Kern	5558 California Ave., Bakersfield CA 93309	35.356092	-119.041209	85	98	98	94	0.084	0.088	0.082	0.084
California	Kern	3311 Manor St, Oildale CA 93308	35.438869	-119.016765	97	99	96	97	0.078	0.082	0.072	0.077
California	Kern	2000 South Union Ave. Bakersfield CA 93307	35.331302	-119.000974	82	87	97	89	0.087	0.097	0.088	0.090
California	Kern	19405 BUENA VISTA BLVD, ARVIN, CA., 93203	35.236680	-118.789424	99	96	97	97	0.088	0.087	0.087	0.087
California	Kern	548 WALKER ST., SHAFER, CA., 93263	35.503587	-119.272606	99	98	98	98	0.081	0.082	0.082	0.081
California	Kings	807 SOUTH IRWIN ST., HANFORD	36.314399	-119.644570	79	92	95	89	0.086	0.085	0.083	0.084
California	Lake	905 LAKEPORT BLVD., LAKEPORT	39.032700	-122.922290	96	98	97	97	0.060	0.058	0.057	0.058
California	Los Angeles	803 N. LOREN AVE., AZUSA	34.136500	-117.923910	95	90	97	94	0.081	0.088	0.095	0.088
California	Los Angeles	840 LAUREL, GLENDORA	34.144350	-117.850360	97	96	96	96	0.096	0.095	0.057	0.082
California	Los Angeles	VA HOSPITAL, WEST LOS ANGELES	34.051110	-118.456360	97	93	96	95	0.077	0.069	0.095	0.080
California	Los Angeles	1630 N MAIN ST, LOS ANGELES	34.066590	-118.226880	94	98	97	96	0.072	0.072	0.066	0.070

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California	Los Angeles	18330 GAULT ST., RESEDA	34.199250	-118.532760	98	97	95	97	0.083	0.087	0.071	0.080
California	Los Angeles	700 North Bullis Road	33.901389	-118.205000	91	80	98	90	0.073	0.065	0.086	0.074
California	Los Angeles	4144 SAN GABRIEL RIVER PKWY, PICO RIVERA	34.011940	-118.069950	95	89	93	92	0.079	0.075	0.064	0.072
California	Los Angeles	924 N. GAREY AVE., POMONA	34.067030	-117.751400	95	92	96	94	0.090	0.094	0.074	0.086
California	Los Angeles	752 S. WILSON AVE., PASADENA	34.132600	-118.127200	89	95	95	93	0.086	0.082	0.087	0.085
California	Los Angeles	2425 Webster St., Long Beach, CA	33.802500	-118.220000	79	81	98	86	0.061	0.056	0.055	0.057
California	Los Angeles	7201 W. WESTCHESTER PARKWAY	33.950800	-118.430430	96	95	93	95	0.075	0.069	0.067	0.070
California	Los Angeles	22224 PLACERITA CANYON RD, SANTA CLARITA	34.383440	-118.528400	96	95	98	96	0.097	0.091	0.100	0.096
California	Los Angeles	43301 DIVISION ST., LANCASTER, CA	34.671394	-118.131456	98	97	95	97	0.081	0.100	0.084	0.088
California	Madera	RD. 29 1/2 NO. OF AVE 8 MADERA COUNTY	36.866667	-120.010000	86	86	96	89	0.088	0.080	0.081	0.083
California	Madera	28261 Avenue 14 Madera CA 93638	36.953256	-120.034203	84	89	99	91	0.082	0.083	0.084	0.083
California	Marin	534 4TH ST.	37.972310	-122.520004	99	98	99	99	0.064	0.063	0.056	0.061
California	Mariposa	TURTLEBACK DOME, YOSEMITE NATL' PK 95389	37.713250	-119.706200	97	97	89	94	0.077	0.073	0.074	0.074
California	Mariposa	6440 JERSEYDALE ROAD, JERSEYDALE	37.549928	-119.844591	83	96	85	88	0.077	0.071	0.077	0.075
California	Mendocino	306 E. GOBBI STREET, UKIAH, CA	39.145660	-123.202980	98	91	93	94	0.052	0.053	0.048	0.051
California	Merced	385 S. COFFEE AVENUE, MERCED, CA 95340	37.281598	-120.434992	92	86	97	92	0.082	0.083	0.082	0.082
California	Monterey	35 Ford Road	36.495767	-121.731571	100	99	99	99	0.063	0.059	0.056	0.059
California	Monterey	415 Pearl Street	36.209286	-121.126371	99	99	100	99	0.062	0.062	0.058	0.060
California	Monterey	867 E. LAUREL Dr	36.696760	-121.637182	100	100	99	100	0.059	0.055	0.052	0.055
California	Napa	2552 JEFFERSON AVE.	38.310942	-122.296189	99	98	99	99	0.062	0.066	0.058	0.062
California	Nevada	200 LITTON DR., GRASS VALLEY, CA	39.234331	-121.056591	99	98	100	99	0.081	0.084	0.086	0.083
California	Orange	1630 W. PAMPAS LANE	33.830620	-117.938450	91	96	95	94	0.076	0.065	0.071	0.070
California	Orange	2850 MESA VERDE DR EAST, COSTA MESA	33.674640	-117.925680	92	92	93	92	0.076	0.068	0.065	0.069
California	Orange	26081 VIA PERA, MISSION VIEJO, CA 92691	33.630030	-117.675930	99	95	98	97	0.078	0.075	0.079	0.077
California	Orange	621 W. LAMBERT, LA HABRA	33.925130	-117.952640	94	96	96	95	0.075	0.073	0.075	0.074
California	Placer	11645 Atwood Street, Auburn	38.935680	-121.099590	96	98	97	97	0.081	0.085	0.072	0.079

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California	Placer	CITY HALL-33 S MAIN ST, COLFAX	39.100278	-120.952778	93	96	96	95	0.073	0.075	0.068	0.072
California	Placer	151 NO SUNRISE BLVD, ROSEVILLE, CA	38.745726	-121.266312	99	95	96	97	0.083	0.073	0.084	0.080
California	Placer	221 Fairway Drive, Tahoe City, CA 96145	39.166017	-120.148833	98	98	97	98	0.062	0.066	0.065	0.064
California	Placer	1445 1st Street, Lincoln	38.885630	-121.302080	98	95	98	97	0.070	0.071	0.069	0.070
California	Riverside	12705 Pechanga Rd., Temecula, CA 92592	33.447867	-117.088649	99	90	88	92	0.079	0.069	0.067	0.071
California	Riverside	200 S. HATHAWAY ST., BANNING CA	33.920860	-116.858410	96	95	94	95	0.094	0.091	0.094	0.093
California	Riverside	33700 Borel Road, Winchester, CA 92596	33.583333	-117.083333	93	99	96	96	0.077	0.079	0.077	0.077
California	Riverside	12160 Santiago Rd. Banning, CA 92220	33.945000	-116.830000	97	92	86	92	0.098	0.097	0.097	0.097
California	Riverside	46-990 JACKSON ST., INDIO	33.708530	-116.215370	99	76	88	88	0.084	0.079	0.081	0.081
California	Riverside	FS-590 RACQUET CLUB AVE, PALM SPRINGS	33.852750	-116.541010	97	95	95	96	0.089	0.086	0.087	0.087
California	Riverside	237 1/2 N. 'D' ST., PERRIS	33.789420	-117.227640	93	99	100	97	0.089	0.094	0.092	0.091
California	Riverside	5888 MISSION BLVD., RUBIDOUX	33.999580	-117.416010	96	95	95	95	0.091	0.096	0.097	0.094
California	Riverside	5130 POINSETTIA PLACE	33.995638	-117.493304	96	92	96	95	0.087	0.093	0.095	0.091
California	Riverside	506 W FLINT ST, LAKE ELSINORE	33.676490	-117.330980	93	93	94	93	0.079	0.093	0.087	0.086
California	Riverside	495 W MURPHY STREET, BLYTHE	33.612406	-114.602886	99	96	91	95	0.078	0.063	0.057	0.066
California	Sacramento	7823 BLACKFOOT WAY, NORTH HIGHLANDS	38.712090	-121.381090	97	91	90	93	0.075	0.075	0.081	0.077
California	Sacramento	DEL PASO-2701 AVALON DR, SACRAMENTO	38.613779	-121.368014	95	96	96	96	0.075	0.079	0.079	0.077
California	Sacramento	1309 T ST., SACRAMENTO, CA. 95814	38.558228	-121.492981	96	96	98	97	0.070	0.071	0.066	0.069
California	Sacramento	12490 BRUCEVILLE RD, ELK GROVE, CA	38.302591	-121.420838	94	95	91	93	0.069	0.069	0.067	0.068
California	Sacramento	50 NATOMA STREET, FOLSOM	38.683304	-121.164457	98	95	97	97	0.081	0.081	0.088	0.083
California	Sacramento	68 GOLDENLAND COURT, SACRAMENTO, CA 95834	38.650783	-121.506767	93	93	95	94	0.070	0.071	0.074	0.071
California	Sacramento	7520 SLOUGHHOUSE ROAD, SLOUGHHOUSE	38.494475	-121.211131	99	98	100	99	0.076	0.079	0.082	0.079
California	San Benito	1979 Fairview Rd	36.844096	-121.362121	98	99	97	98	0.068	0.063	0.058	0.063

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California	San Benito	Northeast entrance, Pinnacles National Park	36.483240	-121.156880	99	92	99	97	0.069	0.066	0.072	0.069
California	San Bernardino	200 E. BUENA VISTA, BARSTOW	34.895007	-117.024484	88	96	98	94	0.084	0.077	0.080	0.080
California	San Bernardino	LAKE GREGORY-LAKE DR, CRESTLINE	34.243100	-117.272350	100	98	98	99	0.102	0.107	0.116	0.108
California	San Bernardino	BEEKLEY & PHELAN RDS, PHELAN	34.426126	-117.563939	99	99	98	99	0.093	0.086	0.090	0.089
California	San Bernardino	14306 PARK AVE., VICTORVILLE, CA	34.510014	-117.331433	99	96	85	93	0.084	0.093	0.081	0.086
California	San Bernardino	1350 SAN BERNARDINO RD., UPLAND	34.103740	-117.629140	93	95	97	95	0.093	0.101	0.110	0.101
California	San Bernardino	Corner of Athol and Telescope, Trona	35.763873	-117.397004	92	99	97	96	0.068	0.068	0.073	0.069
California	San Bernardino	14360 ARROW BLVD., FONTANA	34.100020	-117.492010	86	92	95	91	0.093	0.100	0.098	0.097
California	San Bernardino	17288 OLIVE ST., HESPERIA	34.418073	-117.285597	99	98	99	99	0.087	0.093	0.092	0.090
California	San Bernardino	500 N. DEARBORN, REDLANDS, CA. 92373	34.059770	-117.147310	100	87	97	95	0.099	0.102	0.103	0.101
California	San Bernardino	JOSHUA TREE NATIONAL MONUMENT	34.069570	-116.388930	99	99	99	99	0.090	0.085	0.084	0.086
California	San Bernardino	24302 4TH ST., SAN BERNARDINO, CA.	34.106880	-117.274110	95	95	98	96	0.095	0.105	0.114	0.104
California	San Diego	80 E. 'J' ST., CHULA VISTA	32.631231	-117.059075	98	95	98	97	0.063	0.061	0.061	0.061
California	San Diego	225 9th Street	32.952124	-117.264088	91	98	99	96	0.073	0.064	0.064	0.067
California	San Diego	2300 VICTORIA DR., ALPINE	32.842242	-116.768225	97	97	98	97	0.080	0.079	0.084	0.081
California	San Diego	21441-W B STREET	33.217025	-117.396158	97	95	95	96	0.071	0.068	0.071	0.070
California	San Diego	1110 BEARDSLEY STREET, SAN DIEGO, CA 92112	32.701492	-117.149653	94	98	80	91	0.068	0.061	0.058	0.062
California	San Diego	RICHARD J DONOVAN CORRECTIONAL FACILITY, OTAY MESA, CA	32.579361	-116.929486	32	93	99	75	0.063	0.069	0.071	0.067
California	San Diego	6125A KEARNY VILLA RD., SAN DIEGO	32.845467	-117.123894	97	92	92	94	0.071	0.067	0.068	0.068
California	San Diego	10537 FLOYD SMITH DRIVE	32.817978	-116.968133	60	81	51	64	0.067	0.065	0.070	0.067
California	San Francisco	10 ARKANSAS ST.	37.765946	-122.399044	99	99	99	99	0.052	0.050	0.046	0.049
California	San Joaquin	HAZELTON-HD, STOCKTON	37.950741	-121.268523	98	98	95	97	0.071	0.069	0.066	0.068
California	San Joaquin	5749 S. TRACY BLVD., TRACY	37.682512	-121.443324	95	96	98	96	0.080	0.077	0.080	0.079
California	San Luis Obispo	235 SANTA FE AVENUE, PASO ROBLES, CA	35.631629	-120.690977	98	100	99	99	0.058	0.065	0.063	0.062
California	San Luis Obispo	3220 SOUTH HIGUERA STREET, SAN LUIS OBISPO	35.256577	-120.669861	99	96	100	98	0.062	0.057	0.054	0.057
California	San Luis Obispo	MORRO BAY BLVD & KERN AVE, MORRO BAY	35.366310	-120.842710	97	98	99	98	0.060	0.052	0.053	0.055

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California	San Luis Obispo	NIPOMO REGIONAL PARK, NIPOMO, CA.	35.031460	-120.501030	95	98	96	96	0.066	0.060	0.060	0.062
California	San Luis Obispo	5599 Traffic Way, Atascadero CA	35.494530	-120.666170	97	96	96	96	0.063	0.064	0.062	0.063
California	San Luis Obispo	3601 GILLIS CANYON ROAD	35.643680	-120.231350	98	95	96	96	0.073	0.072	0.074	0.073
California	San Luis Obispo	9640 CARRIZO HIGHWAY	35.354720	-120.040190	98	97	98	98	0.068	0.068	0.070	0.068
California	San Mateo	897 BARRON AVE.	37.482934	-122.203370	98	98	99	98	0.064	0.059	0.056	0.059
California	Santa Barbara	EL CAPITAN ST PRK, HWY 10	34.462450	-120.025510	100	98	99	99	0.065	0.057	0.061	0.061
California	Santa Barbara	700 E. CANON PERDIDO	34.427775	-119.691218	95	99	99	98	0.066	0.061	0.062	0.063
California	Santa Barbara	906 S BROADWAY - SANTA MARIA	34.949147	-120.437630	97	98	99	98	0.058	0.053	0.053	0.054
California	Santa Barbara	HS & P FACILITY-500 M SW, LOMPOC	34.725556	-120.427778	99	93	96	96	0.068	0.059	0.059	0.062
California	Santa Barbara	PARADISE RD-LOS PADRES National Forest	34.541660	-119.791460	98	92	95	95	0.065	0.063	0.064	0.064
California	Santa Barbara	GTC B-HWY 101 NEAR NOJOQUI PASS, GAVIOTA	34.527440	-120.196500	99	99	99	99	0.064	0.060	0.057	0.060
California	Santa Barbara	GOBERNADOR RD, CARPINTERIA	34.402778	-119.457500	96	97	95	96	0.076	0.060	0.055	0.063
California	Santa Barbara	LFC #1-LAS FLORES CANYON	34.489740	-120.046920	99	99	97	98	0.070	0.067	0.064	0.067
California	Santa Barbara	128 S 'H' ST, LOMPOC	34.637820	-120.457500	99	100	99	99	0.063	0.053	0.052	0.056
California	Santa Barbara	380 N FAIRVIEW AVENUE, GOLETA	34.445510	-119.828400	100	99	99	99	0.069	0.061	0.061	0.063
California	Santa Barbara	900 AIRPORT RD., SANTA YNEZ	34.605819	-120.075069	99	99	99	99	0.063	0.062	0.061	0.062
California	Santa Barbara	STS POWER PLANT, VANDENBERG AFB	34.596111	-120.630278	98	96	94	96	0.069	0.056	0.057	0.060
California	Santa Clara	9TH & PRINCEVILLE	36.999571	-121.574684	100	100	100	100	0.071	0.068	0.061	0.066
California	Santa Clara	158B JACKSON ST	37.348497	-121.894898	96	98	98	97	0.065	0.065	0.061	0.063
California	Santa Clara	306 UNIVERSITY AVE.	37.226862	-121.979675	99	96	99	98	0.069	0.072	0.062	0.067
California	Santa Clara	13030 MURPHY AVE.	37.079379	-121.600031	100	100	97	99	0.073	0.071	0.068	0.070
California	Santa Cruz	960 Bostwick Lane	36.983921	-121.989328	99	100	98	99	0.062	0.057	0.052	0.057
California	Shasta	HLTH CTR-2630 BRESLAUER WAY, REDDING	40.549985	-122.382011	99	99	98	99	0.072	0.066	0.072	0.070
California	Shasta	2220 NORTH STREET, ANDERSON, CA 96007	40.452913	-122.298866	99	99	98	99	0.071	0.068	0.067	0.068
California	Shasta	13791 Lake Blvd, Shasta Lake, CA	40.689253	-122.402194	86	80	98	88	0.057	0.072	0.076	0.068
California	Shasta	MANZANITA LAKE RS, LASSEN VOLCANIC NP	40.539990	-121.576460	98	98	92	96	0.065	0.066	0.064	0.065
California	Siskiyou	530 FOOTHILL DR., YREKA	41.726892	-122.633579	99	99	98	99	0.061	0.061	0.055	0.059
California	Solano	304 TUOLUMNE ST.	38.102507	-122.237976	98	99	98	98	0.064	0.064	0.061	0.063
California	Solano	1010 CHADBOURNE RD	38.227066	-122.075624	97	99	100	99	0.063	0.067	0.064	0.064

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California	Solano	2012 ULATIS DRIVE, VACAVILLE	38.358365	-121.950003	99	100	100	100	0.066	0.068	0.067	0.067
California	Sonoma	103 Morris Street,	38.403765	-122.818294	96	99	99	98	0.054	0.056	0.048	0.052
California	Sonoma	MUNICIPAL AIRPORT-200A HEIDELBERG WAY, HEALDSBURG	38.654069	-122.901857	99	97	93	96	0.062	0.059	0.055	0.058
California	Stanislaus	814 14TH ST., MODESTO	37.641583	-120.994622	99	98	98	98	0.081	0.083	0.080	0.081
California	Stanislaus	900 S MINARET STREET, TURLOCK, CA	37.487981	-120.837005	79	88	98	88	0.081	0.085	0.085	0.083
California	Sutter	773 ALMOND ST, YUBA CITY	39.138773	-121.618549	95	97	99	97	0.069	0.064	0.063	0.065
California	Sutter	TOP OF SOUTH BUTTE,SUTTER BUTTES, CA	39.205572	-121.820362	92	99	97	96	0.075	0.072	0.080	0.075
California	Tehama	OLD FIRE LOOKOUT ATOP TUSCAN BUTTE	40.262072	-122.092766	99	99	97	98	0.076	0.076	0.087	0.079
California	Tehama	1834 Walnut Street, Red Bluff, CA 96080	40.170930	-122.255560	93	93	92	93	0.068	0.063		0.065
California	Tulare	SEQUOIA & KINGS CANYON NP	36.489470	-118.829150	96	96	97	96	0.089	0.088	0.092	0.089
California	Tulare	310 N CHURCH ST, VISALIA	36.332179	-119.291228	98	97	96	97	0.078	0.087	0.077	0.080
California	Tulare	1839 South Newcomb Street Porterville, CA 93257	36.031831	-119.055018	92	87	98	92	0.073	0.086	0.090	0.083
California	Tuolumne	251 S BARRETTA, SONORA, CA 95370	37.981582	-120.379611	91	99	96	95	0.075	0.076	0.088	0.079
California	Ventura	2323 Moorpark Road, Thousand Oaks, CA 91360	34.210154	-118.870496	98	99	99	99	0.074	0.066	0.067	0.069
California	Ventura	3301 PACIFIC AVENUE, PIRU, CA 93040	34.404268	-118.809946	99	99	99	99	0.079	0.072	0.073	0.074
California	Ventura	1201 E. OJAI AVENUE, OJAI, CA 93023	34.448048	-119.231321	99	98	98	98	0.077	0.072	0.073	0.074
California	Ventura	5400 COCHRAN STREET, SIMI VALLEY, CA 93063	34.276362	-118.683757	99	100	99	99	0.081	0.074	0.077	0.077
California	Ventura	545 CENTRAL AVENUE, OXNARD, CA 93030	34.252378	-119.143176	99	99	99	99	0.067	0.060	0.063	0.063
California	Yolo	UC DAVIS-CAMPUS, CAMPBELL ROAD WEST OF HIWAY 113 & SOUTH OF HUTCHISON DRIVE.	38.534450	-121.773400	95	99	94	96	0.065	0.065	0.064	0.064
California	Yolo	41929 E. GIBSON ROAD, WOODLAND	38.661210	-121.732690	99	100	99	99	0.066	0.070	0.071	0.069
Colorado	Adams	3174 E. 78TH AVE.	39.838119	-104.949840	97	96	97	97	0.067	0.069	0.066	0.067
Colorado	Arapahoe	36001 E. Quincy Ave.	39.638522	-104.569335	100	98	99	99	0.067	0.068	0.066	0.067
Colorado	Denver	2105 BROADWAY	39.751184	-104.987625	100	100	99	100	0.061	0.067	0.070	0.066
Colorado	Denver	4545 Navajo St.	39.779490	-105.005180	99	94	98	97	0.066	0.071	0.069	0.068
Colorado	Douglas	11500 N. Roxborough Park Rd.	39.534488	-105.070358	99	99	97	98	0.074	0.081	0.078	0.077
Colorado	El Paso	ROAD 640, USAF ACADEMY	38.958341	-104.817215	100	100	99	100	0.064	0.067	0.069	0.066
Colorado	El Paso	101 BANKS PL.	38.853097	-104.901289	100	98	99	99	0.062	0.065	0.066	0.064
Colorado	Garfield	195 W. 14th St.	39.541820	-107.784125	93	91	89	91	0.061	0.068	0.060	0.063

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Colorado	Gunnison	Gunnison National Forest, Crested Butte, CO 81224	38.956400	-106.985800	99	97	98	98	0.063	0.068	0.062	0.064
Colorado	Jefferson	12400 W. HWY 285	39.638781	-105.139480	99	99	99	99	0.066	0.075	0.075	0.072
Colorado	Jefferson	16600 W COLO #128	39.912799	-105.188587	100	98	99	99	0.077	0.077	0.079	0.077
Colorado	Jefferson	2054 QUAKER ST.	39.743724	-105.177989	99	98	100	99	0.076	0.081	0.083	0.080
Colorado	Jefferson	26137 Conifer Road	39.541515	-105.298410	99	97	100	99	0.067	0.070	0.073	0.070
Colorado	La Plata	Weminuche Wilderness Area - Shamrock Station	37.303890	-107.484167	91	65	79	78	0.064	0.068	0.065	0.065
Colorado	La Plata	1 MI. NE OF IGNACIO ON COUNTY RD. 517	37.136780	-107.628630	93	98	92	94	0.067	0.068	0.071	0.068
Colorado	La Plata	7571 HWY. 5505	37.102580	-107.870219	96	100	90	95	0.065	0.066	0.072	0.067
Colorado	Larimer	ROCKY MOUNTAIN NP	40.278130	-105.545640	87	92	98	92	0.069	0.069	0.069	0.069
Colorado	Larimer	3416 LA PORTE AVE.	40.592543	-105.141122	96	100	99	98	0.074	0.075	0.076	0.075
Colorado	Larimer	708 S. Mason St.	40.577470	-105.078920	99	97	98	98	0.072	0.069	0.070	0.070
Colorado	Mesa	865 Rapid Creek Rd.	39.130575	-108.313835	99	99	98	99	0.062	0.065	0.063	0.063
Colorado	Montezuma	106 W. North Street	37.350054	-108.592334	97	99	82	93	0.062	0.061	0.064	0.062
Colorado	Montezuma	MESA VERDE NATIONAL PARK, COLORADO	37.198400	-108.490460	99	98	100	99	0.065	0.066	0.066	0.065
Colorado	Rio Blanco	Meeker, Colorado Plant Science Building	40.038889	-107.847500	98	98	92	96	0.062	0.064	0.059	0.061
Colorado	Rio Blanco	Rangely Colorado, Golf Course	40.086944	-108.761389	96	96	97	96	0.062	0.066	0.061	0.063
Colorado	Weld	3101 35TH AVE.	40.386368	-104.737440	99	97	97	98	0.070	0.073	0.067	0.070
Connecticut	Fairfield	Tods Driftway (see coordinates)	41.003611	-73.585000	98	98	53	83	0.078	0.084	0.079	0.080
Connecticut	Fairfield	White St at 8th Ave, parking garage roof	41.399167	-73.443056	95	99	97	97	0.074	0.079	0.081	0.078
Connecticut	Fairfield	USCG LIGHTHOUSE , PROSPECT STREET	41.152500	-73.103056	99	97	97	98	0.074	0.086	0.083	0.081
Connecticut	Fairfield	SHERWOOD ISLAND STATE PARK	41.118333	-73.336667	97	96	86	93	0.081	0.087	0.081	0.083
Connecticut	Hartford	Remington Road (see coordinates)	41.784722	-72.631667	99	99	96	98	0.077	0.075	0.072	0.074
Connecticut	Litchfield	MOHAWK MOUNTAIN ROAD (see coordinates)	41.821342	-73.297257	97	96	97	97	0.068	0.076	0.074	0.072
Connecticut	Middlesex	CONN. VALLEY HOSP., SHEW HALL, EASTERN D	41.552222	-72.630000	98	99	95	97	0.080	0.078	0.080	0.079

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Connecticut	New Haven	IJAMES STREET	41.301400	-72.902871	97	95	98	97	0.072	0.081	0.075	0.076
Connecticut	New Haven	Meigs Point, Hammonasset State Park	41.260833	-72.550000	99	98	95	97	0.069	0.081	0.080	0.076
Connecticut	New London	141 Smith Street, Groton	41.353620	-72.078820	99	99	98	99	0.065	0.077	0.075	0.072
Connecticut	Tolland	ROUTE 190, SHENIPSIT STATE FOREST	41.976389	-72.388056	97	99	96	97	0.077	0.072	0.072	0.073
Connecticut	Windham	80 Ayers Rd, Abington, CT 06230	41.840200	-72.010000	99	97	99	98	0.067	0.070	0.067	0.068
Delaware	Kent	STATE ROAD 384, KILLENS POND RD	38.986672	-75.556800	100	100	99	100	0.066	0.066	0.068	0.066
Delaware	New Castle	LUMS POND STATE PARK	39.551300	-75.732000	94	100	99	98	0.071	0.065	0.069	0.068
Delaware	New Castle	BRANDYWINE CREEK STATE PARK	39.817222	-75.563889	74	91	99	88	0.074	0.071	0.078	0.074
Delaware	New Castle	BELLEVUE STATE PARK	39.773889	-75.496389	99	93	98	97	0.069	0.069	0.074	0.070
Delaware	New Castle	MLK BLVD AND JUSTISON ST.	39.739444	-75.558056	83	87	97	89	0.068	0.072	0.073	0.071
Delaware	Sussex	350 VIRGINIA AVE SEAFORD	38.653900	-75.610600	100	98	100	99	0.063	0.064	0.068	0.065
Delaware	Sussex	UNIV. OF DE COLLEGE OF MARINE STUDIES	38.779100	-75.163230	98	100	93	97	0.067	0.070	0.070	0.069
District Of Columbia	District of Columbia	2500 1ST STREET, N.W. WASHINGTON DC	38.921847	-77.013178	99	99	97	98	0.068	0.072	0.072	0.070
District Of Columbia	District of Columbia	301 Van Buren Street, N.W. Washington DC 20012	38.970092	-77.016715	100	100	97	99	0.069	0.072	0.071	0.070
Florida	Alachua	918 SE 119TH AVE	29.544722	-82.296111	96	97	96	96	0.060	0.056	0.060	0.058
Florida	Baker	HWY 90 OLUSTEE FOREST SERVICE OFFICE	30.201111	-82.441111	99	99	99	99	0.059	0.058	0.061	0.059
Florida	Bay	4607 STATE PARK LN PCB,FL	30.130433	-85.731517	99	99	99	99	0.065	0.061	0.062	0.062
Florida	Brevard	401 FLORIDA AVE	28.053611	-80.628611	99	94	100	98	0.059	0.058	0.059	0.058

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Florida	Brevard	400 SOUTH 4TH STREET	28.310556	-80.615556	85	100	100	95	0.063	0.059	0.061	0.061
Florida	Broward	3211 College Ave, Davie, FL 33314	26.073536	-80.338450	96	78	98	91	0.059	0.058	0.060	0.059
Florida	Broward	1951 NE 48TH ST	26.292025	-80.096470	99	70	98	89	0.061	0.060	0.059	0.060
Florida	Broward	7000 N. OCEAN DRIVE	26.087000	-80.111000	100	99	98	99	0.063	0.063	0.061	0.062
Florida	Collier	7800 IMMOKALEE ROAD NAPLES 34119	26.270000	-81.711000	99	100	99	99	0.058	0.057	0.058	0.057
Florida	Columbia	751 SE SYCAMORE TERRACE	30.178056	-82.619167	86	80	93	86	0.060	0.058	0.061	0.059
Florida	Duval	13333 LANIER RD.	30.477725	-81.587339	97	32	96	75	0.062	0.054	0.057	0.057
Florida	Duval	13600 William Davis Parkway.	30.261000	-81.454000	92	69	96	86	0.071	0.061	0.064	0.065
Florida	Escambia	ELLYSON INDUSTRIAL PARK-COPTER ROAD	30.525367	-87.203550	100	93	91	95	0.069	0.062	0.063	0.064
Florida	Escambia	NAS PENSACOLA	30.368050	-87.270967	93	99	91	94	0.070	0.061	0.061	0.064
Florida	Flagler	208 Sawgrass Rd	29.489083	-81.276833	99	100	99	99	0.063	0.058	0.059	0.060
Florida	Highlands	123 MAIN DRIVE VENUS FL 33960	27.188889	-81.340556	96	98	99	98	0.060	0.058	0.063	0.060
Florida	Hillsborough	2401 19th Avenue Northwest	27.740033	-82.465146	98	99	98	98	0.071	0.068	0.066	0.068
Florida	Hillsborough	COAST GUARD STATION DAVIS ISLAND	27.928056	-82.454722	98	85	99	94	0.063	0.066	0.070	0.066
Florida	Hillsborough	5121 GANDY BLVD	27.892222	-82.538611	98	98	97	98	0.066	0.066	0.067	0.066
Florida	Hillsborough	1167 NORTH DOVER ROAD	27.965650	-82.230400	97	99	97	98	0.066	0.068	0.066	0.066
Florida	Holmes	TRI CO AIRPORT RD (CO RD 162)	30.848611	-85.603889	100	100	99	100	0.060	0.060	0.060	0.060
Florida	Indian River	Sebastian Inlet State Recreation Area, Vero Beach, FL 32963	27.849200	-80.455400	98	88	91	92	0.064	0.058	0.062	0.061
Florida	Lake	1901 JOHNS LAKE RD	28.525000	-81.723333	100	100	87	96	0.067	0.059	0.064	0.063
Florida	Lee	5505 ROSE GARDEN RD CAPE COREL FL 33914	26.547861	-81.980000	98	98	99	98	0.063	0.058	0.060	0.060

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Florida	Lee	INTERSECTION OF SCHOOL ST AND BAY ST	26.448889	-81.939444	98	98	99	98	0.060	0.058	0.059	0.059
Florida	Leon	110 CENTURY PARK CIRCLE WEST	30.439722	-84.346389	99	94	94	96	0.059	0.061	0.061	0.060
Florida	Liberty	Apalachicola National Forest, Bristol, FL 32321	30.110300	-84.990300	96	95	96	96	0.057	0.056	0.056	0.056
Florida	Manatee	PORT MANATEE, REEDER RD & SOUTH DOCK ST.	27.632778	-82.546111	95	99	94	96	0.060	0.061	0.058	0.059
Florida	Manatee	5502 33RD AVE DRIVE W. (G T BRAY PARK)	27.480556	-82.618889	86	93	82	87	0.062	0.065	0.060	0.062
Florida	Manatee	5511 39TH STREET EAST	27.449444	-82.522222	95	88	96	93	0.064	0.060	0.059	0.061
Florida	Marion	SE 17TH STREET & SE 30TH AVENUE	29.170278	-82.100833	94	100	100	98	0.064	0.058	0.062	0.061
Florida	Marion	692 NW 30TH AVE	29.192500	-82.173333	99	99	97	98	0.059	0.057	0.060	0.058
Florida	Martin	950 SE MONTEREY RD	27.172458	-80.240689	97	100	98	98	0.061	0.060	0.062	0.061
Florida	Miami-Dade	ROSENSTIEL SCHOOL	25.733378	-80.161806	97	99	92	96	0.063	0.062	0.062	0.062
Florida	Miami-Dade	19590 OLD CUTLER RD-PERDUE MED. CENTER	25.586384	-80.326811	94	87	93	91	0.061	0.060	0.062	0.061
Florida	Okaloosa	720 Lovejoy Rd	30.426533	-86.666217	100	100	100	100	0.067	0.059	0.060	0.062
Florida	Orange	7055 WINEGARD RD., ORLANDO	28.454167	-81.381389	82	100	99	94	0.062	0.061	0.063	0.062
Florida	Orange	MORRIS BLVD.	28.596389	-81.362500	97	100	100	99	0.063	0.059	0.066	0.062
Florida	Osceola	8706_WEST 192, KISSIMMEE	28.347222	-81.636667	97	99	100	99	0.064	0.061	0.064	0.063
Florida	Pasco	30908 WARDER ROAD	28.331944	-82.305833	96	100	98	98	0.061	0.061	0.063	0.061
Florida	Pasco	3452 DARLINGTON RD., HOLIDAY	28.195000	-82.758056	98	99	95	97	0.064	0.060	0.063	0.062
Florida	Pinellas	2435 SHARKEY RD.CLEARWATER	27.946389	-82.731944	97	100	97	98	0.063	0.059	0.063	0.061
Florida	Pinellas	7200-22 AVENUE NORTH	27.785866	-82.739875	99	99	98	99	0.064	0.059	0.060	0.061
Florida	Pinellas	COUNTY ROAD 77 BOOKER CK PARK	28.090000	-82.700833	99	97	100	99	0.061	0.056	0.062	0.059
Florida	Polk	2727 SHEPHERD RD (JAMES W. SIKES ELM SCH	27.939444	-82.000278	96	99	99	98	0.063	0.062	0.064	0.063

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Florida	Polk	1015 SIKES BLVD., LAKELAND	28.028889	-81.972222	98	94	97	96	0.062	0.064	0.061	0.062
Florida	St. Lucie	1420 E Midway rd	27.389079	-80.311033	98	93	99	97	0.062	0.059	0.063	0.061
Florida	Santa Rosa	1500 WOODLAWN WAY	30.394133	-87.008033	100	99	100	100	0.070	0.061	0.061	0.064
Florida	Sarasota	450 MICINLEY DR.	27.306944	-82.570556	98	95	98	97	0.064	0.062	0.062	0.062
Florida	Sarasota	4570 17TH STREET	27.350278	-82.480000	99	100	98	99	0.063	0.063	0.061	0.062
Florida	Sarasota	201 SOUTH JACKSON ROAD VENICE FLORIDA	27.089194	-82.362583	100	94	97	97	0.062	0.061	0.060	0.061
Florida	Seminole	COUNTY HOMES RD. @ US 17-92	28.746111	-81.310556	100	98	96	98	0.060	0.060	0.063	0.061
Florida	Volusia	5200 SPRUCE CREEK RD.	29.108889	-80.993889	98	96	96	97	0.060	0.057	0.060	0.059
Florida	Volusia	1185-A DUNN AVE., DAYTONA BEACH	29.206667	-81.052500	93	100	97	97	0.062	0.057	0.059	0.059
Florida	Wakulla	COUNTY RD 59	30.092500	-84.161111	72	96	83	84	0.061	0.057	0.060	0.059
Georgia	Bibb	Georgia Forestry Commission, 5645 Riggins Mill Road, Dry Branch, Georgia, 31020	32.805408	-83.543521	95	100	98	98	0.065	0.062	0.070	0.065
Georgia	Chatham	AMERICAN RED CROSS, 2500 E. PRESIDENT STREET, BLDG-A, SAVANNAH, GA, 31404	32.069230	-81.048769	98	98	90	95	0.057	0.058	0.058	0.057
Georgia	Chattooga	DNR Fish Hatchery, 231 Fish Hatchery Road, Summerville, Georgia, 30747	34.474293	-85.408003	100	78	98	92	0.060	0.063	0.065	0.062
Georgia	Clarke	FIRE STATION #7, 2350 BARNETT SHOALS RD, ATHENS, GA 30603	33.918067	-83.344495	97	87	100	95	0.063	0.061	0.069	0.064
Georgia	Cobb	GA National Guard, 1901 McCollum Parkway, Kennesaw, Georgia, 30144	34.015482	-84.607407	33	96	100	76	0.063	0.066	0.070	0.066
Georgia	Columbia	Riverside Park, 4431 Hardy McManus Road, Evans, Georgia, 30809	33.582144	-82.131189	98	98	98	98	0.062	0.059	0.062	0.061
Georgia	Coweta	UNIVERSITY OF W. GA AT NEWNAN, 7 Solar Circle, Newnan, Georgia, 30265	33.404040	-84.745988	98	96	100	98	0.067	0.066	0.066	0.066
Georgia	Dawson	GA FORESTRY COMMISSION, 4500 Georgia Highway 53 East, Dawsonville, Georgia, 30534	34.376317	-84.059766	100	98	98	99	0.066	0.063	0.067	0.065
Georgia	DeKalb	2390-B Wildcat Road, Decatur GA 30034	33.687970	-84.290480	99	98	95	97	0.070	0.071	0.074	0.071
Georgia	Douglas	Douglas County Water Authority, 7725 W. Strickland St., Douglasville, Georgia 30134	33.743656	-84.779192	96	95	99	97	0.065	0.070	0.071	0.068
Georgia	Fulton	935 East Confederate Ave., Atlanta, Georgia, 30316	33.720192	-84.357056	97	100	98	98	0.073	0.077	0.075	0.075
Georgia	Glynn	RISLEY MIDDLE SCHOOL, 2900 ALBANY ST., BRUNSWICK, GA 31520	31.169735	-81.495881	99	100	91	97	0.057	0.055	0.057	0.056

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Georgia	Gwinnett	Gwinnett Technical College, 5150 Sugarloaf Parkway, Lawrenceville, Georgia, 30043	33.961270	-84.069010	100	100	99	100	0.068	0.071	0.078	0.072
Georgia	Henry	Henry County Extension Office, 86 Work Camp Road, McDonough, Georgia, 30253	33.433575	-84.161708	99	98	99	99	0.075	0.070	0.078	0.074
Georgia	Murray	FORT MOUNTAIN, STATE HWY 52, COHUTTA OVERLOOK, CHATSWORTH, GA 30705	34.785197	-84.626422	91	97	98	95	0.067	0.063	0.067	0.065
Georgia	Muscogee	Columbus Airport, 3100 Thruway Drive, Columbus, Georgia, 31909	32.521302	-84.944795	100	89	97	95	0.061	0.062	0.065	0.062
Georgia	Paulding	King Farm, 160 Ralph King Path, Rockmart, Georgia, 30153	33.928500	-85.045340	97	98	93	96	0.059	0.065	0.067	0.063
Georgia	Pike	Georgia Station Georgia Agricultural Experiment Station, Williamson, GA 30292	33.178700	-84.405200	99	100	96	98	0.066	0.068	0.071	0.068
Georgia	Richmond	Bungalow Road Elem School, 2216 BUNGALOW RD, AUGUSTA GA 30906	33.433349	-82.022217	97	98	98	98	0.061	0.060	0.065	0.062
Georgia	Rockdale	Monastery of the Holy Spirit, 2625 Georgia 212, Conyers, Georgia, 30094	33.591077	-84.065294	99	99	97	98	0.079	0.068	0.076	0.074
Georgia	Sumter	Leslie Community Center, N Bass S/E Allen St., Leslie, Georgia, 31764	31.954298	-84.081059	99	100	99	99	0.059	0.057	0.065	0.060
Hawaii	Honolulu	2052 LAUWILIWILI ST	21.323745	-158.088613	64	97	86	82	0.046	0.049	0.048	0.047
Hawaii	Honolulu	1039 SAND ISLAND PARKWAY	21.303382	-157.871171	92	95	92	93	0.054	0.048	0.047	0.049
Idaho	Ada	520 S. EAGLE ROAD, MERIDIAN	43.600699	-116.347853	97	99	99	98	0.062	0.066	0.062	0.063
Idaho	Ada	401 EAST LINDEN ST. BOISE, ID 83706	43.577603	-116.178156	78	98	99	92	0.065	0.064	0.072	0.067
Idaho	Butte	CRATERS OF THE MOON NATIONAL MON, IDAHO	43.460556	-113.562222	97	96	96	96	0.062	0.061	0.058	0.060
Illinois	Adams	1301 S. 48TH ST.	39.915409	-91.335868	98	100	100	99	0.061	0.064	0.061	0.062
Illinois	Champaign	NORTH THOMAS ST.	40.244913	-88.188519	100	98	100	99	0.062	0.062	0.066	0.063
Illinois	Champaign	TWP RD 500 E.	40.052780	-88.372510	94	94	95	94	0.068	0.065	0.066	0.066
Illinois	Clark	416 S. STATE ST. HWY 1/ WEST UNION	39.210857	-87.668297	98	97	95	97	0.063	0.064	0.066	0.064
Illinois	Cook	4500 W. 123RD ST.	41.670992	-87.732457	95	97	95	96	0.066	0.066	0.075	0.069
Illinois	Cook	3300 E. CHELTENHAM PL.	41.755832	-87.545350	98	97	100	98	0.067	0.066	0.077	0.070
Illinois	Cook	7801 LAWNSDALE	41.751400	-87.713488	92	99	98	96	0.067	0.065	0.075	0.069

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Illinois	Cook	6545 W. HURLBUT ST.	41.984332	-87.792002	93	99	97	96	0.065	0.068	0.075	0.069
Illinois	Cook	729 HOUSTON	41.668120	-87.990570	97	98	99	98	0.070	0.066	0.073	0.069
Illinois	Cook	4743 MANNHEIM RD.	41.965193	-87.876265	97	98	96	97	0.063	0.058	0.067	0.062
Illinois	Cook	1820 S. 51ST AVE.	41.855243	-87.752470	96	99	100	98	0.063	0.061	0.076	0.066
Illinois	Cook	9511 W. HARRISON ST	42.060285	-87.863225	97	99	99	98	0.069	0.068	0.076	0.071
Illinois	Cook	750 DUNDEE ROAD	42.139996	-87.799227	93	100	99	97	0.068	0.068	0.079	0.071
Illinois	Cook	531 E. LINCOLN	42.062053	-87.675254	95	96	96	96	0.072	0.070	0.076	0.072
Illinois	DuPage	RT. 53	41.813049	-88.072827	91	98	86	92	0.064	0.067	0.074	0.068
Illinois	Effingham	ROUTE 45 SOUTH	39.067159	-88.548934	98	94	100	97	0.063	0.064	0.066	0.064
Illinois	Hamilton	STATE ROUTE 14	38.082155	-88.624943	100	99	86	95	0.063	0.064	0.068	0.065
Illinois	Jersey	LIBERTY ST. & COUNTY RD.	39.110539	-90.324080	100	86	93	93	0.065	0.067	0.074	0.068
Illinois	Jo Daviess	10952 E. Parker Rd, Stockton, IL 61085	42.286900	-89.999700	98	86	94	93	0.067	0.062	0.067	0.065
Illinois	Kane	665 DUNDEE RD.	42.049148	-88.273029	100	99	95	98	0.066	0.065	0.074	0.068
Illinois	Lake	ILLINOIS BEACH STATE PARK	42.467573	-87.810047	99	99	100	99	0.073	0.070	0.077	0.073
Illinois	McHenry	FIRST ST. & THREE OAKS RD.	42.221442	-88.242207	100	94	98	97	0.067	0.064	0.073	0.068
Illinois	McLean	MAIN & GREGORY	40.518735	-88.996896	99	100	100	100	0.066	0.063	0.065	0.064
Illinois	Macon	2200 N. 22ND	39.866834	-88.925594	100	100	100	100	0.067	0.066	0.066	0.066
Illinois	Macoupin	HEATON & DUBOIS	39.396075	-89.809739	100	99	94	98	0.063	0.064	0.067	0.064
Illinois	Madison	409 MAIN ST.	38.890186	-90.148031	99	100	95	98	0.072	0.069	0.073	0.071
Illinois	Madison	200 W. DIVISION	38.726573	-89.959963	91	95	93	93	0.070	0.064	0.067	0.067
Illinois	Madison	54 N. WALCOTT	38.860669	-90.105851	98	99	88	95	0.070	0.069	0.075	0.071
Illinois	Madison	5403 State Road 160, Highland, IL 62249	38.869000	-89.622800	94	95	95	95	0.068	0.067	0.068	0.067
Illinois	Peoria	HURLBURT & MACARTHUR	40.687420	-89.606943	99	97	98	98	0.064	0.060	0.068	0.064
Illinois	Peoria	508 E. GLEN AVE.	40.745504	-89.585869	89	81	97	89	0.064	0.064	0.066	0.064
Illinois	Randolph	HICKORY GROVE & FALLVIEW	38.176278	-89.788459	98	98	94	97	0.071	0.065	0.066	0.067
Illinois	Rock Island	32 RODMAN AVE.	41.514727	-90.517350	95	100	92	96	0.062	0.060	0.064	0.062

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Illinois	Saint Clair	13TH & TUDOR	38.612034	-90.160477	98	100	96	98	0.067	0.066	0.073	0.068
Illinois	Sangamon	Illinois Building State Fairgrounds	39.831522	-89.640926	96	100	98	98	0.059	0.064	0.068	0.063
Illinois	Will	36400 S. ESSEX RD.	41.221537	-88.190967	89	98	100	96	0.064	0.064	0.064	0.064
Illinois	Winnebago	1405 MAPLE AVE.	42.334982	-89.037775	90	98	98	95	0.070	0.066	0.070	0.068
Indiana	Allen	14600 AMSTUTZ RD., LEO HS	41.221418	-85.016821	99	99	90	96	0.063	0.062	0.066	0.063
Indiana	Allen	2022 NORTH BEACON	41.094965	-85.101816	99	96	92	96	0.063	0.059	0.069	0.063
Indiana	Bartholomew	Hope-Hauser Jr-Sr High School, 9404 N. 775 E.	39.294322	-85.766816	99	99	96	98	0.067	0.066	0.071	0.068
Indiana	Boone	3900 E. 300 S, WHITESTOWN	39.997484	-86.395172	99	95	98	97	0.066	0.064	0.070	0.066
Indiana	Brown	4831 Helmsburg Rd.- Helmsburg	39.263914	-86.292269	64	97	74	78	0.058	0.062	0.063	0.061
Indiana	Carroll	481 S. 150 W. / FLORA AIRPORT	40.540455	-86.553035	93	98	98	96	0.064	0.064	0.066	0.064
Indiana	Clark	12500 St. Rd. 62-Charlestown State Park/ Indiana Armory	38.393833	-85.664167	93	96	98	96	0.066	0.074	0.072	0.070
Indiana	Delaware	700 W. STATE ST./ ALBANY ELEMENTARY	40.300015	-85.245367	99	98	86	94	0.054	0.058	0.067	0.059
Indiana	Elkhart	705 INDIANA AVE./ BRISTOL ELEMENTARY	41.718050	-85.830550	98	99	99	99	0.055	0.058	0.072	0.061
Indiana	Floyd	2230 GREEN VALLEY ROAD/GREEN VALLEY ELEMENTARY SCHOOL	38.308056	-85.834167	98	84	96	93	0.068	0.067	0.073	0.069
Indiana	Greene	2500 S. 275 W, PLUMMER	38.985578	-86.990120	99	99	99	99	0.064	0.067	0.069	0.066
Indiana	Hamilton	Noblesville- 9900 E. 191st St.	40.068297	-85.992451	99	99	99	99	0.058	0.063	0.069	0.063
Indiana	Hendricks	Avon- 7203 E. US HIGHWAY 36, Avon Transportation Cntr.- 255 S. Avon Ave.(updated 2016)	39.759003	-86.397124	99	99	99	99	0.057	0.055	0.068	0.060
Indiana	Huntington	423 WEST VINE STREET - ROANOKE	40.960710	-85.379842	95	97	98	97	0.056	0.054	0.066	0.058
Indiana	Jackson	225 W & 300 N, BROWNSTOWN	38.920844	-86.080489	97	90	99	95	0.064	0.064	0.070	0.066
Indiana	Johnson	200 W. PEARL ST., TRAFALGAR	39.417243	-86.152363	95	97	99	97	0.056	0.063	0.062	0.060
Indiana	Knox	Southwest Purdue Agricultural Center, Vincennes, IN 47591	38.740800	-87.485300	95	97	99	97	0.062	0.063	0.071	0.065

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Indiana	Lake	201 MISSISSIPPI ST., IITRI BUNKER	41.606680	-87.304729	96	93	97	95	0.067	0.064	0.070	0.067
Indiana	Lake	1300 141 ST STREET	41.639460	-87.493623	95	79	95	90	0.067	0.060	0.068	0.065
Indiana	LaPorte	2011 E. LINCOLNWAY LaPorte- E. Lincolnway	41.629098	-86.684615	98	99	99	99	0.061	0.061	0.068	0.063
Indiana	Madison	Emporia EAST ELEM. SCH., 893 E. US 36, PENDLETON	40.002547	-85.656937	93	98	99	97	0.054	0.054	0.064	0.057
Indiana	Marion	5753 Glenn Rd.- FORT HARRISON STATE PARK	39.858922	-86.021317	99	98	99	99	0.066	0.069	0.073	0.069
Indiana	Marion	1321 SOUTH HARDING	39.749019	-86.186314	99	91	98	96	0.066	0.061	0.069	0.065
Indiana	Marion	NAVAL AVIONICS CENTER, 6125 E. 16TH ST.	39.789486	-86.060850	99	99	97	98	0.065	0.062	0.068	0.065
Indiana	Marion	3120 E. 30TH ST., WASHINGTON PARK	39.811097	-86.114469	74	99	98	90	0.061	0.067	0.070	0.066
Indiana	Marion	1650 Ludlow Ave.- Indianapolis- I 70	39.787933	-86.130880	77	99	99	92	0.061	0.064	0.068	0.064
Indiana	Morgan	135 S. CHESTNUT, MONROVIA HIGH SCHOOL	39.575634	-86.477893	98	97	99	98	0.064	0.063	0.065	0.064
Indiana	Perry	19856 OLD ST RD 37/PERRY CENTRAL HS/ LEOPOLD	38.113159	-86.603594	94	99	99	97	0.067	0.067	0.069	0.067
Indiana	Porter	84 DIANA RD/ WATER TREATMENT PLANT	41.617558	-87.199248	95	99	99	98	0.071	0.066	0.070	0.069
Indiana	Porter	1000 WESLEY ST./ VALPARAISO WATER DEPT.	41.510293	-87.038546	97	95	94	95	0.067	0.060	0.071	0.066
Indiana	Posey	2027 S. ST. PHILLIPS RD., Evansville	38.005289	-87.718429	98	99	98	98	0.065	0.067	0.068	0.066
Indiana	St. Joseph	25601 STATE RD. 4, North Liberty/ POTATO CREEK STATE PARK	41.551696	-86.370589	99	98	99	99	0.059	0.058	0.069	0.062
Indiana	St. Joseph	2335 SHIELDS DR/ SOUTH BEND CAAP 2	41.696692	-86.214683	99	99	90	96	0.067	0.066	0.072	0.068
Indiana	St. Joseph	Granger- Beckley St. 12441 Beckley. St., Granger	41.754870	-86.110090	99	99	98	99	0.064	0.064	0.071	0.066
Indiana	Shelby	4740 W. 600 N, FAIRLAND	39.613423	-85.870648	99	99	99	99	0.062	0.059	0.065	0.062
Indiana	Vanderburgh	14940 OLD STATE ROAD/ SCOTT ELEMENTARY	38.113947	-87.536957	97	99	98	98	0.070	0.068	0.070	0.069
Indiana	Vanderburgh	Evansville-Buena Vista-1110 W. Buena Vista Rd.	38.013248	-87.577856	98	99	98	98	0.068	0.072	0.072	0.070
Indiana	Vigo	Terre Haute- Lafayette Ave. 961 N. LAFAYETTE AVE.	39.486150	-87.401398	98	99	99	99	0.062	0.064	0.069	0.065

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Indiana	Vigo	7597 N. STEVENSON RD/ SANDCUT	39.560556	-87.313056	99	98	96	98	0.056	0.062	0.067	0.061
Indiana	Wabash	Hamilton Rd, Lagro, IN 46941	40.816000	-85.661100	90	96	92	93	0.066	0.068	0.071	0.068
Indiana	Warrick	300 N. 1ST ST./ BOONVILLE HIGH SCHOOL	38.052003	-87.278337	98	99	99	99	0.065	0.069	0.070	0.068
Indiana	Warrick	5244 STATE RD 68/ TECUMSEH HIGH SCHOOL	38.194501	-87.341396	99	90	98	96	0.064	0.062	0.072	0.066
Indiana	Warrick	DAYVILLE- 3488 EBLE RD, Newburgh	37.954511	-87.321939	97	99	92	96	0.066	0.065	0.071	0.067
Iowa	Bremer	WAVERLY AIRPORT	42.743056	-92.513056	99	100	99	99	0.061	0.059	0.062	0.060
Iowa	Clinton	ROOSEVELT ST.	41.874999	-90.177574	99	99	99	99	0.065	0.061	0.063	0.063
Iowa	Harrison	206 Polk St, Pisgah, IA	41.832256	-95.928185	97	98	96	97	0.063	0.061	0.063	0.062
Iowa	Harrison	1575 HWY 183	41.780261	-95.948435	97	100	95	97	0.062	0.061	0.063	0.062
Iowa	Linn	6301 KIRKWOOD BLVD SW (IOWA HALL)	41.910556	-91.651944	100	95	93	96	0.059	0.063	0.063	0.061
Iowa	Linn	408 E. LINN ST. COGGON, IOWA	42.281013	-91.526879	99	99	97	98	0.061	0.060	0.063	0.061
Iowa	Linn	500 11TH ST NW	41.976770	-91.687660	99	99	100	99	0.059	0.060	0.064	0.061
Iowa	Montgomery	2780 VIKING LAKE ROAD	40.969112	-95.044951	99	95	99	98	0.059	0.060	0.062	0.060
Iowa	Palo Alto	IOWA LAKES COMMUNITY COLLEGE	43.123704	-94.693518	95	98	93	95	0.063	0.064	0.058	0.061
Iowa	Polk	1907 CARPENTER, DES MOINES IOWA	41.603159	-93.643118	100	98	99	99	0.061	0.058	0.061	0.060
Iowa	Scott	SCOTT COUNTY PARK	41.699174	-90.521944	99	99	99	99	0.064	0.063	0.064	0.063
Iowa	Scott	10TH ST. & VINE ST. DAVENPORT	41.530011	-90.587611	99	100	99	99	0.059	0.059	0.063	0.060
Iowa	Story	105 GREENE STREET-CITY HALL	41.882867	-93.687800	100	99	98	99	0.062	0.059	0.060	0.060
Iowa	Van Buren	24430 LACEY TRAIL	40.695078	-92.006318	90	99	97	95	0.063	0.057	0.061	0.060
Iowa	Warren	1650 118TH AVENUE, INDIANOLA	41.285533	-93.583983	99	100	99	99	0.060	0.059	0.057	0.058
Kansas	Johnson	13899 W 159TH	38.838575	-94.746424	100	100	100	100	0.062	0.062	0.058	0.060

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Kansas	Leavenworth	2010 METROPOLITAN	39.327391	-94.951020	99	100	98	99	0.068	0.060	0.062	0.063
Kansas	Neosho	1500 WEST SEVENTH	37.676960	-95.475940	98	99	99	99	0.062	0.061	0.061	0.061
Kansas	Sedgwick	HEALTH DEPT., 1900 EAST 9TH ST.	37.702066	-97.314847	99	100	100	100	0.069	0.064	0.062	0.065
Kansas	Sedgwick	12831 W. 117N	37.897506	-97.492083	100	99	98	99	0.068	0.063	0.062	0.064
Kansas	Shawnee	2501 RANDOLPH AVENUE	39.024265	-95.711275	100	99	99	99	0.064	0.062	0.063	0.063
Kansas	Sumner	707 E 119TH ST SOUTH, PECK COMMUNITY BLDG	37.476890	-97.366399	100	100	97	99	0.067	0.065	0.062	0.064
Kansas	Trego	CEDAR BLUFF RESERVOIR, PRONGHORN & MULEY	38.770081	-99.763424	96	95	98	96	0.068	0.063	0.058	0.063
Kansas	Wyandotte	1210 N. 10TH ST., JFK RECREATION CENTER	39.117219	-94.635605	95	98	100	98	0.063	0.063	0.064	0.063
Kentucky	Bell	1420 DORCHESTER AVENUE	36.608430	-83.736940	96	99	99	98	0.062	0.059	0.062	0.061
Kentucky	Boone	KY 338 & LOWER RIVER ROAD	38.918330	-84.852637	100	99	98	99	0.062	0.062	0.065	0.063
Kentucky	Boyd	FIVCO HEALTH DEPARTMENT, 2924 HOLT STREET	38.459340	-82.640410	100	98	98	99	0.065	0.069	0.066	0.066
Kentucky	Bullitt	SECOND & CARPENTER STREETS	37.986290	-85.711920	90	98	96	95	0.065	0.067	0.067	0.066
Kentucky	Campbell	524A JOHN'S HILL ROAD	39.021881	-84.474450	98	99	99	99	0.071	0.071	0.069	0.070
Kentucky	Carter	CAMP ROBERT WEBB, 1486 CAMP WEBB ROAD	38.238870	-82.988100	98	96	99	98	0.060	0.061	0.065	0.062
Kentucky	Christian	WILLIAMSON RESIDENCE, 10800 PILOT ROCK ROAD	36.911710	-87.323337	99	95	98	97	0.065	0.062	0.061	0.062
Kentucky	Daviess	716 PLEASANT VALLEY ROAD	37.780776	-87.075307	97	98	96	97	0.064	0.065	0.068	0.065
Kentucky	Edmonson	MAMMOTH CAVE NP - ALFRED COOK ROAD	37.131790	-86.142953	98	97	84	93	0.065	0.063	0.065	0.064
Kentucky	Fayette	FAYETTE COUNTY HEALTH DEPT, 650 NEWTOWN PIKE	38.065030	-84.497610	98	95	98	97	0.065	0.071	0.065	0.067
Kentucky	Greenup	SCOTT STREET & CENTER AVENUE	38.548136	-82.731163	99	98	100	99	0.061	0.066	0.063	0.063
Kentucky	Hancock	2ND STREET & CAROLINE STREET	37.938290	-86.897190	96	99	97	97	0.066	0.071	0.067	0.068
Kentucky	Hardin	AMERICAN LEGION PARK, 801 NORTH MILES STREET	37.705612	-85.852629	90	99	98	96	0.062	0.066	0.068	0.065
Kentucky	Henderson	BASKETT FIRE DEPARTMENT, 7492 DR. HODGE ROAD	37.871200	-87.463750	98	98	98	98	0.069	0.067	0.072	0.069
Kentucky	Jefferson	7601 BARDSTOWN RD, BATES ELEMENTARY SCH	38.137840	-85.576480	100	82	87	90	0.065	0.071	0.073	0.069
Kentucky	Jefferson	7201 WATSON LN, WATSON LN ELEMENTARY SCH	38.060910	-85.898040	97	91	96	95	0.069	0.069	0.070	0.069
Kentucky	Jefferson	2730 CANNONS LANE, BOWMAN FIELD	38.228760	-85.654520	99	100	99	99	0.070	0.076	0.076	0.074

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Kentucky	Jessamine	KYTC MAINTENANCE GARAGE, 260 WILSON DRIVE	37.891470	-84.588250	100	99	99	99	0.064	0.066	0.066	0.065
Kentucky	Livingston	LIVINGSTON CO ROAD DEPT, 706 STATE DRIVE	37.155392	-88.394024	99	99	98	99	0.065	0.063	0.068	0.065
Kentucky	McCracken	JACKSON PURCHASE RECC, 2901 POWELL STREET	37.058220	-88.572510	99	99	99	99	0.065	0.063	0.063	0.063
Kentucky	Morgan	State Highway 437, West Liberty, KY 41472	37.921400	-83.066200	90	98	100	96	0.064	0.064	0.066	0.064
Kentucky	Oldham	KYTC MAINTENANCE FACILITY, 1601 SOUTH HWY 393	38.400200	-85.444280	100	77	97	91	0.068	0.073	0.069	0.070
Kentucky	Perry	PERRY COUNTY HORSE PARK, 354 PERRY PARK ROAD	37.283290	-83.209320	99	98	93	97	0.061	0.057	0.058	0.058
Kentucky	Pike	KYTC DISTRICT OFFICE, 109 LORAIN STREET	37.482600	-82.535320	100	98	99	99	0.063	0.058	0.061	0.060
Kentucky	Pulaski	SOMERSET GAS CO WAREHOUSE, 305 CLIFTY STREET	37.097980	-84.611520	100	98	95	98	0.063	0.062	0.063	0.062
Kentucky	Simpson	KYTC MAINTENANCE FACILITY, 573 HARDING ROAD	36.708607	-86.566284	100	84	99	94	0.063	0.067	0.063	0.064
Kentucky	Trigg	5720 Old Dover Rd, Cadiz, KY 42211	36.784100	-87.849900	93	96	97	95	0.066	0.060	0.063	0.063
Kentucky	Warren	226 SUNSET STREET	37.049260	-86.214870	98	86	99	94	0.063	0.061	0.062	0.062
Kentucky	Washington	542 Wesley-Miller Rd, Harrodsburg, KY 40330	37.704600	-85.048500	99	98	95	97	0.065	0.064	0.065	0.064
Louisiana	Ascension	11153 Kling Road	30.233889	-90.968333	97	99	98	98	0.069	0.074	0.071	0.071
Louisiana	Bossier	1425 Airport Drive	32.536260	-93.748910	98	98	96	97	0.066	0.065	0.065	0.065
Louisiana	Caddo	HAGOOD ROAD	32.676389	-93.859722	98	99	98	98	0.065	0.066	0.062	0.064
Louisiana	Calcasieu	HIGHWAY 27 AND HIGHWAY 108	30.143333	-93.371944	96	99	99	98	0.067	0.072	0.065	0.068
Louisiana	Calcasieu	2284 Paul Bellow Road	30.227778	-93.578333	99	99	95	98	0.066	0.066	0.062	0.064
Louisiana	East Baton Rouge	EAST END OF ASTER LANE	30.419763	-91.181996	97	100	98	98	0.075	0.073	0.068	0.072
Louisiana	East Baton Rouge	1061-A Leesville Ave	30.461980	-91.179220	99	100	97	99	0.070	0.069	0.061	0.066
Louisiana	East Baton Rouge	11245 Port Hudson-Pride Rd. Zachary, La	30.700921	-91.056135	97	99	73	90	0.068	0.062	0.061	0.063
Louisiana	Iberville	65180 Belleview Road	30.220556	-91.316111	98	98	99	98	0.061	0.069	0.064	0.064
Louisiana	Iberville	HIGHWAY 171, CARVILLE	30.206985	-91.129948	97	87	61	82	0.068	0.075	0.069	0.070
Louisiana	Jefferson	West Temple Pl	30.043573	-90.275091	98	99	99	99	0.071	0.068	0.067	0.068
Louisiana	Lafayette	646 Cajundome	30.217500	-92.051389	100	99	99	99	0.068	0.067	0.063	0.066
Louisiana	Lafourche	Nicholls University Farm Highway 1	29.763889	-90.765183	98	96	98	97	0.067	0.064	0.065	0.065
Louisiana	Livingston	Highway 16, French Settlement	30.312500	-90.812500	99	98	94	97	0.073	0.070	0.067	0.070
Louisiana	Ouachita	5296 Southwest	32.509713	-92.046093	94	92	87	91	0.059	0.060	0.060	0.059
Louisiana	Pointe Coupee	TED DAVIS RESIDENCE. HIGHWAY 415	30.681736	-91.366172	97	100	99	99	0.071	0.069	0.065	0.068
Louisiana	St. Bernard	4101 Mistrot Dr. Meraux, LA 70075	29.939610	-89.923880	100	100	92	97	0.071	0.063	0.065	0.066
Louisiana	St. James	ST. JAMES COURTHOUSE, HWY 44 @ CANAPELLA	29.994444	-90.820000	97	98	99	98	0.067	0.066	0.063	0.065
Louisiana	St. John the Baptist	Anthony F. Monica Street	30.058333	-90.608333	96	100	99	98	0.068	0.067	0.064	0.066

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Louisiana	St. Tammany	1421 Hwy 22 W, Madison Ville, LA 70447	30.429304	-90.199683	99	99	99	99	0.074	0.067	0.064	0.068
Louisiana	West Baton Rouge	1005 Northwest Drive, Port Allen	30.500643	-91.213556	100	99	99	99	0.066	0.066	0.066	0.066
Maine	Androscoggin	ROUTE 9, DURHAM	43.974622	-70.124608	99	82	99	93	0.065	0.058	0.057	0.060
Maine	Aroostook	8 NORTHERN ROAD, PRESQUE ISLE, ME 04769	46.696431	-68.033006	95	73	87	85	0.049	0.054	0.048	0.050
Maine	Aroostook	45 Radar Rd, Ashland, ME 04732	46.604100	-68.413500	96	99	98	98	0.051	0.055	0.052	0.052
Maine	Cumberland	TWO LIGHTS STATE PARK, Near the Obsevation Tower	43.561043	-70.207324	100	99	100	100	0.066	0.064	0.065	0.065
Maine	Hancock	TOP OF CADILLAC MOUNTAIN	44.351697	-68.226980	97	86	99	94	0.065	0.069	0.066	0.066
Maine	Hancock	567 Eagle Lake Road	44.377050	-68.260900	98	100	98	99	0.062	0.065	0.060	0.062
Maine	Kennebec	14 Pray Street	44.230622	-69.785000	98	99	95	97	0.057	0.063	0.059	0.059
Maine	Knox	PORT CLYDE, MARSHALL POINT LIGHTHOUSE	43.917955	-69.260590	99	94	97	97	0.061	0.067	0.063	0.063
Maine	Penobscot	SUMMIT OF RIDER BLUFF (WLBZ TRANSMITTER)	44.735978	-68.670752	99	100	99	99	0.054	0.063	0.057	0.058
Maine	Washington	PUBLIC LANDING, Jonesport	44.531907	-67.595870	89	98	99	95	0.054	0.062	0.057	0.057
Maine	Washington	184 COUNTY ROAD	44.963633	-67.060728	98	60	99	86	0.050	0.053	0.054	0.052
Maine	York	34 Town Farm Road, HOLLIS	43.656764	-70.629138	97	98	99	98	0.059	0.058	0.058	0.058
Maine	York	Rt. 11, Shapleigh Ball Park, West Newfield Road, Shapleigh, Maine	43.588890	-70.877340	97	99	99	98	0.061	0.062	0.061	0.061
Maine	York	OCEAN AVE / PARSONS WAY, KENNEBUNKPORT	43.343167	-70.471034	91	78	100	90	0.066	0.067	0.068	0.067
Maryland	Baltimore	Padonia Elementary School, 9834 Greenside Drive	39.462029	-76.631673	91	92	95	93	0.067	0.078	0.073	0.072
Maryland	Baltimore	600 Dorsey Avenue	39.310833	-76.474444	98	97	95	97	0.068	0.072	0.077	0.072
Maryland	Calvert	350 Stafford Road	38.536722	-76.617194	99	95	99	98	0.070	0.067	0.068	0.068
Maryland	Carroll	1300 W. Old Liberty Road	39.444294	-77.042252	100	98	99	99	0.064	0.070	0.066	0.066
Maryland	Cecil	Fair Hill Natural Resource Mgmt Area, 4600 Telegraph Road	39.701444	-75.860051	95	89	98	94	0.074	0.074	0.075	0.074
Maryland	Charles	14320 Oaks Road	38.508547	-76.811864	96	98	99	98	0.070	0.068	0.073	0.070
Maryland	Dorchester	University of Maryland for Environmental and Estuarine Studies	38.587525	-76.141006	99	96	99	98	0.065	0.061	0.067	0.064

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Maryland	Dorchester	Blackwater National Wildlife Refuge, Cambridge, MD 21613	38.445000	-76.111400	95	100	90	95	0.065	0.065	0.068	0.066
Maryland	Frederick	Frederick County Airport ,180 E Airport Drive	39.422760	-77.375190	96	96	98	97	0.063	0.070	0.066	0.066
Maryland	Garrett	Piney Run, Frostburg Reservoir, Finzel	39.705950	-79.012000	98	95	91	95	0.063	0.067	0.066	0.065
Maryland	Harford	Edgewood Chemical Biological Center (APG), Waehli Road	39.410191	-76.296946	100	99	98	99	0.067	0.074	0.077	0.072
Maryland	Harford	3560 Aldino Road	39.563333	-76.203889	96	96	96	96	0.070	0.073	0.074	0.072
Maryland	Kent	Millington Wildlife Management Area, Massey - Maryland Line Road (Route 330)	39.305021	-75.797317	99	92	96	96	0.068	0.072	0.069	0.069
Maryland	Montgomery	Lathrop E. Smith Environmental Education Center, 5110 Meadows Lane	39.114313	-77.106876	98	99	99	99	0.064	0.072	0.065	0.067
Maryland	Prince George's	Howard University's Beltsville Laboratory, 12003 Old Baltimore Pike	39.055277	-76.878333	99	95	97	97	0.065	0.072	0.069	0.068
Maryland	Prince George's	PG County Equestrian Center, 14900 Pennsylvania Ave.	38.811940	-76.744170	97	98	100	98	0.069	0.069	0.073	0.070
Maryland	Prince George's	Powder Mill Rd, Laurel, MD 20708	39.028400	-76.817100	87	97	93	92	0.069	0.067	0.070	0.068
Maryland	Washington	MD Correctional Institution 18530 Roxbury Road	39.564178	-77.720244	97	98	98	98	0.061	0.067	0.068	0.065
Maryland	Baltimore (City)	Furley E.S. Recreational Center, 4633 Furley Avenue.	39.328807	-76.553075	95	77	98	90	0.060	0.072	0.067	0.066
Massachusetts	Barnstable	FOX BOTTOM AREA	41.975804	-70.023598	98	61	96	85	0.059	0.071	0.065	0.065
Massachusetts	Bristol	659 GLOBE ST	41.685707	-71.169235	98	99	100	99	0.060	0.070	0.076	0.068
Massachusetts	Bristol	30 SCHOOL ST - HASTINGS MIDDLE SCHOOL	41.645381	-70.897504	98	96	99	98	0.058	0.067	0.069	0.064
Massachusetts	Dukes	1 HERRING CREEK RD, AQUINNAH(WAMPANOAG TRIBAL SITE)	41.330469	-70.785225	96	45	93	78	0.059	0.068	0.066	0.064
Massachusetts	Essex	390 PARKLAND	42.474642	-70.970816	99	98	97	98	0.063	0.065	0.067	0.065
Massachusetts	Essex	HARBOR STREET	42.814412	-70.817783	97	96	95	96	0.064	0.065	0.065	0.064
Massachusetts	Essex	685 WASHINGTON STREET	42.770837	-71.102290	99	99	98	99	0.064	0.059	0.064	0.062
Massachusetts	Franklin	VETERANS FIELD	42.605816	-72.596689	98	97	99	98	0.058	0.064	0.068	0.063
Massachusetts	Hampden	ANDERSON RD AFB	42.194380	-72.555112	94	100	96	97	0.065	0.070	0.076	0.070
Massachusetts	Hampshire	QUABBIN SUMMIT	42.298493	-72.334079	99	96	94	96	0.068	0.071	0.072	0.070
Massachusetts	Middlesex	11 TECHNOLOGY DRIVE, EPA NEW ENGLAND REGIONAL LABORATORY	42.626680	-71.362068	98	99	97	98	0.064	0.061	0.066	0.063

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Massachusetts	Norfolk	695 HILLSIDE ST Blue Hill Observatory	42.211774	-71.113970	94	94	98	95	0.065	0.067	0.070	0.067
Massachusetts	Plymouth	170 Clinton Street(Buckley)	42.065106	-71.012129	99	96	98	98	0.060	0.065	0.067	0.064
Massachusetts	Suffolk	HARRISON AVE	42.329500	-71.082600	100	99	99	99	0.054	0.056	0.058	0.056
Massachusetts	Worcester	375 AIRPORT DRIVE	42.274319	-71.875511	96	99	99	98	0.065	0.063	0.066	0.064
Massachusetts	Worcester	366 E HARTFORD DR	42.099699	-71.619399	98	91	100	96	0.064	0.059	0.070	0.064
Michigan	Allegan	966 W 32ND	42.767786	-86.148577	97	99	97	98	0.077	0.072	0.076	0.075
Michigan	Benzie	1060 WEST ST., BENZONIA TWP.	44.616943	-86.109408	95	99	92	95	0.069	0.067	0.072	0.069
Michigan	Berrien	PAW PAW WWTP, 4689 DEFIELD RD.	42.197790	-86.309694	99	99	98	99	0.073	0.072	0.078	0.074
Michigan	Cass	ROSS BEATTY HIGH SCHOOL, 22721 DIAMOND COVE ST.	41.895570	-86.001629	89	98	99	95	0.066	0.068	0.077	0.070
Michigan	Chippewa	LK SUPERIOR STATE UNIV.650 W EASTERDAY A	46.493611	-84.364167	93	92	92	92	0.056	0.059	0.062	0.059
Michigan	Clinton	8562 E STOLL RD	42.798339	-84.393795	98	99	98	98	0.066	0.064	0.073	0.067
Michigan	Genesee	WHALEY PARK, 3610 IOWA	43.047224	-83.670159	99	99	98	99	0.068	0.066	0.072	0.068
Michigan	Genesee	LAKEVILLE MIDDLE SCHOOL, G11107 WASHBURN RD	43.168336	-83.461541	99	98	99	99	0.068	0.067	0.073	0.069
Michigan	Huron	1172 S.M25,SAND BEACH TWP.	43.836388	-82.642900	90	99	99	96	0.066	0.067	0.072	0.068
Michigan	Ingham	220 N PENNSYLVANIA	42.738618	-84.534633	99	99	93	97	0.065	0.064	0.073	0.067
Michigan	Kalamazoo	FAIRGROUNDS, 1400 OLMSTEAD RD	42.278067	-85.541890	99	96	98	98	0.067	0.067	0.074	0.069
Michigan	Kent	1179 MONROE NW	42.984173	-85.671339	97	100	100	99	0.066	0.067	0.075	0.069
Michigan	Kent	10300 14 MILE RD NE #B	43.176672	-85.416608	99	99	98	99	0.066	0.065	0.072	0.067
Michigan	Lenawee	6792 RAISEN CENTER HIGHWAY	41.995568	-83.946559	93	99	98	97	0.068	0.065	0.069	0.067
Michigan	Macomb	57700 GRATIOT	42.731394	-82.793463	98	99	99	99	0.071	0.072	0.075	0.072
Michigan	Macomb	WARREN FIRE STATION 29900 HOOVER AT COMMON	42.513340	-83.005971	98	97	99	98	0.068	0.064	0.071	0.067
Michigan	Manistee	3031 DOMRES RD.	44.307000	-86.242649	97	99	98	98	0.068	0.067	0.070	0.068
Michigan	Mason	525 W US10	43.953334	-86.294415	99	99	98	99	0.070	0.066	0.074	0.070
Michigan	Missaukee	1769 S JEFFS RD	44.310555	-84.891865	100	98	99	99	0.063	0.064	0.074	0.067

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Michigan	Muskegon	1340 GREEN CREEK ROAD	43.278061	-86.311083	99	96	97	97	0.075	0.074	0.076	0.075
Michigan	Oakland	13701 OAK PARK BLVD.	42.463063	-83.183199	97	94	99	97	0.067	0.066	0.075	0.069
Michigan	Ottawa	6981 28TH AVE. GEORGETOWN TWP.	42.894451	-85.852734	96	87	89	91	0.071	0.065	0.074	0.070
Michigan	St. Clair	2525 DOVE RD	42.953336	-82.456229	99	98	99	99	0.071	0.075	0.073	0.073
Michigan	Schoolcraft	SENEY NAT'L WILDLIFE REFUGE, HCR2, BOX 1	46.288877	-85.950227	98	99	99	99	0.067	0.070	0.075	0.070
Michigan	Tuscola	1821 E. Dickerson Rd, Unionville, MI 48767	43.613800	-83.359100	97	95	98	97	0.063	0.064	0.071	0.066
Michigan	Washtenaw	555 TOWNER ST	42.240565	-83.599602	98	99	98	98	0.070	0.064	0.069	0.067
Michigan	Washtenaw	10070 Strawberry Lake Rd, Dexter, MI 48130	42.416500	-83.902000	89	92	92	91	0.067	0.064	0.074	0.068
Michigan	Wayne	14700 GODDARD	42.228620	-83.208200	97	94	99	97	0.064	0.064	0.070	0.066
Michigan	Wayne	11600 EAST SEVEN MILE ROAD	42.430840	-83.000138	96	97	95	96	0.073	0.070	0.074	0.072
Michigan	Wexford	10637 S 9 Rd, Cadillac, MI 49601	44.180900	-85.739000	97	99	95	97	0.066	0.064	0.071	0.067
Minnesota	Anoka	2660 Fawn Rd.	45.401840	-93.203060	100	100	99	100	0.062	0.058	0.061	0.060
Minnesota	Anoka	9399 Lima St	45.137680	-93.207615	100	98	99	99	0.064	0.064	0.061	0.063
Minnesota	Becker	26624 NORTH TOWER ROAD	46.851811	-95.846272	100	99	99	99	0.061	0.062	0.059	0.060
Minnesota	Crow Wing	16384 AIRPORT RD	46.396740	-94.130300	100	99	98	99	0.057	0.060	0.060	0.059
Minnesota	Goodhue	1235 HIGHWAY 19	44.473754	-93.012611	100	99	100	100	0.063	0.060	0.061	0.061
Minnesota	Hennepin	1444 E 18th St	44.965242	-93.254759	99	99	100	99	0.060	0.053	0.056	0.056
Minnesota	Lake	Fernberg Road	47.948622	-91.495574	97	97	94	96	0.056	0.056	0.058	0.056
Minnesota	Lyon	WEST HIGHWAY 19	44.443800	-95.817890	100	98	100	99	0.062	0.067	0.057	0.062
Minnesota	Mille Lacs	16687 Shaw-Bosh-Kung Drive	46.205300	-93.759450	100	100	97	99	0.060	0.059	0.061	0.060
Minnesota	Olmsted	1801 9th Ave. SE	43.996908	-92.450366	100	100	98	99	0.062	0.059	0.062	0.061
Minnesota	Saint Louis	VOYAGEURS NATIONAL PARK	48.412520	-92.829225	100	99	79	93	0.057	0.056	0.052	0.055
Minnesota	Saint Louis	1202 EAST UNIVERSITY CIRCLE	46.818260	-92.089360	95	100	96	97	0.052	0.054	0.055	0.053
Minnesota	Scott	917 DAKOTA ST.	44.791437	-93.512534	99	98	99	99	0.061	0.060	0.061	0.060
Minnesota	Stearns	1321 University Dr.	45.549839	-94.133450	100	95	100	98	0.062	0.060	0.058	0.060
Minnesota	Washington	St. Croix Trail N	45.168004	-92.765136	98	97	99	98	0.061	0.058	0.060	0.059
Minnesota	Wright	101 Central Ave. W.	45.209160	-93.669210	100	96	98	98	0.064	0.059	0.061	0.061
Mississippi	Bolivar	213 N. BAYOU AVE.	33.746056	-90.723028	97	99	98	98	0.060	0.063	0.064	0.062
Mississippi	DeSoto	5 EAST SOUTH ST. (HERNANDO)	34.821660	-89.987830	100	99	88	96	0.067	0.061	0.066	0.064
Mississippi	Hancock	400 Baltic St	30.300833	-89.395916	99	96	94	96	0.069	0.061	0.061	0.063
Mississippi	Harrison	47 Maple Street	30.390369	-89.049778	89	98	98	95	0.073	0.067	0.063	0.067

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Mississippi	Hinds	232 East Woodrow Wilson Drive	32.329111	-90.182722	97	93	90	93	0.064	0.061	0.060	0.061
Mississippi	Jackson	Hospital Road at Co. Health Dept.	30.378287	-88.533930	97	98	98	98	0.075	0.065	0.062	0.067
Mississippi	Lauderdale	Hwy 19 and 53rd Ave	32.364565	-88.731491	98	98	86	94	0.058	0.056	0.058	0.057
Mississippi	Lee	West Jackson at Tupelo Airport	34.264917	-88.766222	99	96	97	97	0.059	0.058	0.061	0.059
Mississippi	Yalobusha	Jamie L. Whitten Plant Materials Center, Coffeerville, MS 38922	34.002600	-89.799000	88	87	99	91	0.060	0.056	0.057	0.057
Missouri	Andrew	SAVANAH: 11796 Highway 71, Savanah, MO 64485	39.954400	-94.849000	99	99	100	99	0.064	0.064	0.062	0.063
Missouri	Boone	FINGER LAKES: 1505 East Peaboy Road, Columbia, MO 65202	39.078070	-92.316260	100	99	100	100	0.064	0.063	0.065	0.064
Missouri	Callaway	NEW BLOOMFIELD: 2625 Medow Lane, New Bloomfield, MO, 65063	38.706080	-92.093080	99	100	100	100	0.064	0.065	0.064	0.064
Missouri	Cass	RICHARDS GEBEUR - SOUTH: 1802 E 203rd Street, Belton, MO 64012	38.759760	-94.579970	100	100	99	100	0.065	0.065	0.061	0.063
Missouri	Cedar	EL DORADO SPRINGS: Highway 97 & Barnes Road, El Dorado Springs, 64744	37.690000	-94.035000	99	99	99	99	0.065	0.060	0.060	0.061
Missouri	Clay	WATKINS MILL STATE PARK: Watkins Mill Road, Lawson, MO 64062	39.407452	-94.265373	99	100	100	100	0.067	0.064	0.066	0.065
Missouri	Clay	LIBERTY: Highway & County Home Road, Liberty, 64068	39.303090	-94.376623	100	99	98	99	0.065	0.062	0.066	0.064
Missouri	Clay	ROCKY CREEK: 13131 Highway 169 NE, Smithville, MO 64089	39.331913	-94.580841	100	100	99	100	0.066	0.068	0.069	0.067
Missouri	Clinton	TRIMBLE: 7536 SW O Highway, Trimble, MO 64492	39.530600	-94.556000	99	100	99	99	0.064	0.068	0.069	0.067
Missouri	Greene	HILLCREST HIGH SCHOOL: 3319 N Grant, Springfield, MO 65803	37.256136	-93.299886	100	96	93	96	0.060	0.061	0.058	0.059
Missouri	Greene	FELLOWS LAKE: 4208 E Farm Road 66, Springfield, MO 65803	37.319510	-93.204635	99	100	99	99	0.060	0.061	0.059	0.060
Missouri	Jasper	ALBA: 20400 Millwood Road, Alba, MO64755	37.238500	-94.424680	100	98	100	99	0.065	0.061	0.059	0.061
Missouri	Jefferson	ARNOLD WEST: 1709 Lonedell Drive, Arnold, MO 63010	38.448633	-90.398540	99	100	99	99	0.072	0.069	0.070	0.070
Missouri	Lincoln	FOLEY: #7 Wild Horse, Foley, MO 63347	39.044700	-90.864700	100	99	99	99	0.067	0.065	0.065	0.065
Missouri	Monroe	MARK TWAIN STATE PARK: 20057 State Park Office Road, Stoutville, MO 65283	39.474976	-91.788991	100	100	100	100	0.060	0.058	0.059	0.059
Missouri	Perry	FARRAR: County Road 342, Farrar, MO 63746	37.702640	-89.698640	100	99	100	100	0.067	0.067	0.069	0.067
Missouri	Saint Charles	WEST ALTON: General Electric Store, Highway 94, West Alton, MO 63386	38.872546	-90.226488	100	90	99	96	0.072	0.070	0.075	0.072
Missouri	Saint Charles	ORCHARD FARM: 2165 Highway V, St. Charles, MO 63301	38.899400	-90.449170	100	100	100	100	0.072	0.066	0.076	0.071
Missouri	Sainte Genevieve	BONNE TERRE: 15797 Highway D, Bonne Terre, MO 63628	37.900840	-90.423880	100	100	98	99	0.069	0.063	0.067	0.066

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Missouri	Saint Louis	PACIFIC: 18701 Old Highway 66, Pacific, MO 63039	38.490200	-90.705200	100	100	99	100	0.065	0.065	0.067	0.065
Missouri	Saint Louis	MARYLAND HEIGHTS: 13044 Marine Avenue, Maryland Heights, MO 63146	38.710900	-90.475900	99	100	99	99	0.072	0.069	0.073	0.071
Missouri	Taney	BRANSON: 251 S.W. Outer Rd, Branson, MO 65616	36.707727	-93.222000	100	100	99	100	0.058	0.059	0.056	0.057
Missouri	St. Louis City	BLAIR STREET: 3247 Blair Street, St. Louis, MO 63107	38.656498	-90.198646	99	99	100	99	0.066	0.063	0.068	0.065
Montana	Fergus	303 East Aztec Drive Lewistown MT 59457	47.048537	-109.455315	91	92	97	93	0.056	0.056	0.055	0.055
Montana	Flathead	GLACIER NATIONAL PARK	48.510300	-113.996810	91	98	93	94	0.052	0.057	0.050	0.053
Montana	Lewis and Clark	I-15 Exit 209, then Sperry Dr.	46.850500	-111.987164	100	92	100	97	0.058	0.058	0.055	0.057
Montana	Missoula	BOYD PARK, 3100 WASHBURN Rd.	46.842181	-114.021499	82	99	98	93	0.053	0.055	0.051	0.053
Montana	Phillips	2309 Short Oil Road, Malta, MT 59538	48.317507	-107.862471	89	98	99	95	0.052	0.061	0.053	0.055
Montana	Powder River	Big Powder River Road East	45.440295	-105.370283	73	98	96	89	0.053	0.057	0.055	0.055
Montana	Richland	Corner Cnty Roads 335 and 131	47.803392	-104.485552	95	94	92	94	0.051	0.058	0.057	0.055
Montana	Rosebud	SR 566, 3 Miles N of Birney	45.366151	-106.489820	100	82	98	93	0.055	0.056	0.057	0.056
Nebraska	Douglas	DOUGLAS COUNTY HOSP 42ND & WOOLWORTH	41.247486	-95.973142	99	100	93	97	0.063	0.062	0.062	0.062
Nebraska	Douglas	2411 O ST.	41.207958	-95.945897	98	96	97	97	0.059	0.055	0.063	0.059
Nebraska	Knox	State Spur 54D, Niobrara, NE 68760	42.829200	-97.854000	97	95	99	97	0.063	0.063	0.064	0.063
Nebraska	Lancaster	FIRST & MAPLE , DAVEY	40.984722	-96.677222	98	97	97	97	0.061	0.061	0.058	0.060
Nevada	Churchill	280 SOUTH RUSSELL STREET	39.472471	-118.783624	98	94	95	96	0.065	0.068	0.067	0.066
Nevada	Clark	12101 Hwy 91, Nevada Las Vegas, NV 89165	36.391007	-114.907406	52	74	72	66	0.076	0.072	0.068	0.072
Nevada	Clark	465 E. OLD MILL ROAD, MESQUITE, NV	36.807913	-114.060879	50	73	72	65	0.065	0.065	0.058	0.062
Nevada	Clark	4525 NEW FOREST DRIVE	36.106372	-115.253352	81	99	98	93	0.077	0.073	0.069	0.073
Nevada	Clark	7701 DUCHARME AVE	36.169751	-115.263025	81	91	99	90	0.074	0.068	0.070	0.070
Nevada	Clark	333 PAVILION CENTER DRIVE	36.173416	-115.332714	100	99	99	99	0.077	0.072	0.069	0.072
Nevada	Clark	6651 W. AZURE AVE	36.270583	-115.238256	97	99	96	97	0.079	0.071	0.073	0.074
Nevada	Clark	4250 Karen Ave	36.141895	-115.078725	99	99	98	99	0.073	0.069	0.067	0.069
Nevada	Clark	1005 INDUSTRIAL ROAD	35.978125	-114.846298	99	92	98	96	0.073	0.068	0.062	0.067
Nevada	Clark	1965 State Hwy 161, Jean, NV	35.785671	-115.357048	95	99	98	97	0.074	0.071	0.066	0.070
Nevada	Clark	1301B EAST TONOPAH	36.191260	-115.122930	98	97	98	98	0.075	0.073	0.070	0.072

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Nevada	Clark	668 Gretta Ln, Indian Springs	36.569444	-115.676667	53	75	75	68	0.070	0.070	0.068	0.069
Nevada	Lyon	320 HARDIE LANE	39.602787	-119.247741	99	100	95	98	0.067	0.071	0.068	0.068
Nevada	Washoe	301 A STATE STREET, RENO, NV 89502	39.525083	-119.807717	95	94	95	95	0.071	0.073	0.069	0.071
Nevada	Washoe	4110 DE LUCCI LANE, RENO NV 89502	39.469219	-119.775354	100	99	98	99	0.068	0.070	0.068	0.068
Nevada	Washoe	684A STATE ROUTE 341, RENO NV 89521	39.399837	-119.739606	99	99	99	99	0.067	0.069	0.065	0.067
Nevada	Washoe	750 4TH ST, SPARKS, NV 89431	39.540917	-119.746761	99	99	99	99	0.069	0.070	0.069	0.069
Nevada	Washoe	855 ALDER DRIVE, INCLINE VILLAGE, NV 89451	39.250409	-119.956738	100	99	100	100	0.063	0.062	0.063	0.062
Nevada	Washoe	325 PATRICIAN DR, LEMMON VALLEY, NV 89506	39.645264	-119.840025	100	99	100	100	0.067	0.072	0.069	0.069
Nevada	White Pine	GREAT BASIN NATIONAL PARK	39.005120	-114.215930	93	97	97	96	0.064	0.066	0.063	0.064
Nevada	Carson City	2601 SOUTH CARSON ST (OLD NATIONAL GUARD ARMORY)	39.144700	-119.766100	100	100	99	100	0.068	0.068	0.066	0.067
New Hampshire	Belknap	GREEN STREET, LACONIA	43.566111	-71.496361	99	99	99	99	0.061	0.055	0.060	0.058
New Hampshire	Cheshire	WATER STREET	42.930472	-72.272389	100	99	100	100	0.060	0.059	0.066	0.061
New Hampshire	Coos	MT. WASHINGTON	44.270167	-71.303750	99	98	99	99	0.065	0.071	0.066	0.067
New Hampshire	Coos	CAMP DODGE, ROUTE 16, GREEN'S GRANT	44.308167	-71.217694	97	99	95	97	0.055	0.057	0.059	0.057
New Hampshire	Grafton	LEBANON AIRPORT	43.629611	-72.309583	99	99	100	99	0.062	0.054	0.055	0.057
New Hampshire	Grafton	Hubbard Brook Experimental Forest, North Woodstock, NH 03262	43.945000	-71.700800	99	98	100	99	0.058	0.052	0.054	0.054
New Hampshire	Hillsborough	GILSON ROAD	42.718664	-71.522427	99	98	99	99	0.064	0.061	0.064	0.063
New Hampshire	Hillsborough	PACK MONADNOCK SUMMIT	42.861750	-71.878389	99	100	100	100	0.069	0.066	0.069	0.068
New Hampshire	Merrimack	HAZEN DRIVE	43.218500	-71.514500	98	100	100	99	0.061	0.062	0.062	0.061
New Hampshire	Rockingham	PORTSMOUTH, PEIRCE ISLAND	43.075333	-70.748000	99	100	100	100	0.068	0.065	0.062	0.065
New Hampshire	Rockingham	SEACOAST SCIENCE CENTER	43.045278	-70.713806	99	97	99	98	0.070	0.065	0.067	0.067
New Hampshire	Rockingham	150 Pillsbury Rd	42.862536	-71.380172	99	100	98	99	0.067	0.062	0.067	0.065
New Jersey	Atlantic	Edwin B. Forsythe National Wildlife Refuge Visitor Center, Great Creek Road	39.464872	-74.448736	99	96	99	98	0.061	0.068	0.063	0.064
New Jersey	Bergen	Overpeck Park, 40 Fort Lee Road	40.870436	-73.991994	97	98	99	98	0.073	0.076	0.072	0.073
New Jersey	Camden	266 Spruce Street	39.934446	-75.125291	100	100	100	100	0.068	0.079	0.076	0.074

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New Jersey	Camden	Ancora State Hospital, 202 Spring Garden Road	39.684250	-74.861491	100	97	99	99	0.068	0.072	0.064	0.068
New Jersey	Cumberland	Lincoln Avenue and Route 55, Northeast of Millville	39.422273	-75.025204	100	99	100	100	0.067	0.068	0.068	0.067
New Jersey	Essex	360 Clinton Avenue	40.720989	-74.192892	99	99	100	99	0.070	0.072	0.068	0.070
New Jersey	Gloucester	Clarksboro Shady Rest Home, Shady Lane and County House Road	39.800339	-75.212119	84	96	95	92	0.070	0.076	0.074	0.073
New Jersey	Hudson	Veterans Park on Newark Bay, 25th Street near Park Road	40.670250	-74.126081	99	100	100	100	0.072	0.077	0.067	0.072
New Jersey	Hunterdon	Raritan Township Municipal Utilities Authority, 365 Old York Road	40.515262	-74.806671	100	100	100	100	0.065	0.073	0.073	0.070
New Jersey	Mercer	Athletic Fields, Route 206 South	40.283092	-74.742644	100	99	100	100	0.071	0.073	0.071	0.071
New Jersey	Mercer	Washington Crossing State Park, Titusville, NJ 08560	40.312500	-74.872900	100	99	97	99	0.071	0.075	0.074	0.073
New Jersey	Middlesex	Horticultural Farm #3, off Ryder's Lane	40.462182	-74.429439	97	68	95	87	0.071	0.077	0.074	0.074
New Jersey	Monmouth	Edison Science Building, 400 Cedar Avenue	40.277647	-74.005100	98	98	100	99	0.064	0.077	0.068	0.069
New Jersey	Morris	Building #1, Department of Public Works (DPW) off Route 513	40.787628	-74.676301	97	99	95	97	0.068	0.070	0.068	0.068
New Jersey	Ocean	Colliers Mills Wildlife Management Area	40.064830	-74.444050	100	100	89	96	0.072	0.075	0.071	0.072
New Jersey	Passaic	Ramapo Mountain State Forest, Access Road, off Skyline Drive	41.058617	-74.255544	98	96	96	97	0.067	0.071	0.068	0.068
New Jersey	Warren	Columbia Wildlife Management Area, Delaware Road	40.924580	-75.067815	99	100	100	100	0.060	0.066	0.065	0.063
New Mexico	Bernalillo	4700A SAN MATEO NE	35.134300	-106.585200	98	97	97	97	0.064	0.065	0.067	0.065
New Mexico	Bernalillo	201 PROSPERITY SE	35.017080	-106.657390	98	98	95	97	0.064	0.068	0.063	0.065
New Mexico	Bernalillo	8901 LOWELL NE	35.185200	-106.508150	97	98	94	96	0.061	0.067	0.064	0.064
New Mexico	Dona Ana	ST LUKES EPISCOPAL CH RT 1 (LA UNION)	31.930556	-106.630556	97	98	99	98	0.065	0.070	0.063	0.066
New Mexico	Dona Ana	680 MCCOMBS, CHAPARRAL, NM	32.041111	-106.409167	92	98	99	96	0.067	0.065	0.067	0.066
New Mexico	Dona Ana	5935A VALLE VISTA, SUNLAND PARK, NM	31.796111	-106.583889	91	98	99	96	0.072	0.074	0.070	0.072

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New Mexico	Dona Ana	104-2 SANTA TERESA INTERNATIONAL BLVD,NM	31.787778	-106.682778	90	97	99	95	0.066	0.070	0.069	0.068
New Mexico	Dona Ana	750 N.SOLANO DRIVE, LAS CRUCES, NM	32.317500	-106.767778	100	99	97	99	0.066	0.066	0.064	0.065
New Mexico	Eddy	HOLLAND ST,SE OF WATER TANK,CARLSBAD,NM	32.380000	-104.262222	92	96	98	95	0.072	0.067	0.063	0.067
New Mexico	Lea	2320 N. JEFFERSON ST, HOBBS, NEW MEXICO	32.726656	-103.122917	95	98	97	97	0.068	0.067	0.065	0.066
New Mexico	Rio Arriba	21 New Mexico 96, Coyote, NM, 87012	36.187742	-106.698369	97	98	98	98	0.065	0.064	0.063	0.064
New Mexico	Sandoval	HIGHWAY DEPT. YARD NEAR BERNALILLO	35.299444	-106.548333	90	97	98	95	0.062	0.066	0.064	0.064
New Mexico	San Juan	162 HWAY 544, BLOOMFIELD NM 87413	36.742222	-107.976944	99	99	99	99	0.062	0.061	0.065	0.062
New Mexico	San Juan	423 HWAY 539, NAVAJO DAM, NM 87419	36.809730	-107.651580	92	99	94	95	0.063	0.068	0.067	0.066
New Mexico	San Juan	USBR SHIPROCK SUBSTATION (FARMINGTON)	36.796667	-108.472500	97	99	100	99	0.063	0.061	0.062	0.062
New Mexico	Santa Fe	2001 Aviation Drive, Santa Fe, New Mexico 87507	35.619750	-106.079680	99	99	99	99	0.064	0.062	0.064	0.063
New Mexico	Valencia	1000 W. MAIN ST, LOS LUNAS, NM 87031	34.814700	-106.739600	97	97	98	97	0.064	0.064	0.065	0.064
New York	Albany	LOUDONVILLE RESERVOIR 300 ALBANY SHAKER RD	42.680750	-73.757330	93	99	98	97	0.061	0.063	0.068	0.064
New York	Bronx	IS 52 681 KELLY ST	40.816180	-73.902000	97	99	99	98	0.071	0.063	0.069	0.067
New York	Bronx	200TH STREET AND SOUTHERN BOULVDARD Pfizer Lab	40.867900	-73.878090	100	100	100	100	0.070	0.072	0.070	0.070
New York	Chautauqua	STP, Wright Park Drive, DUNKIRK	42.499630	-79.318810	95	97	100	97	0.066	0.071	0.069	0.068
New York	Dutchess	Forest Road, VILLAGE OF MILLBROOK, Inst of Ecosystem Studies	41.785550	-73.741360	97	97	96	97	0.068	0.067	0.071	0.068
New York	Erie	AUDUBON GOLF COURSE, 450 MAPLE ROAD	42.993280	-78.771530	95	100	96	97	0.063	0.071	0.074	0.069
New York	Essex	Wilmington, SUMMIT, WHITEFACE MTN, WEATHER STATION	44.366080	-73.903120	90	98	98	95	0.059	0.060	0.067	0.062
New York	Essex	Wilmington, BASE WHITEFACE MTN, ASRC, SUNY	44.393080	-73.858900	95	90	93	93	0.060	0.067	0.068	0.065

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New York	Essex	Huntington Wildlife Forest, Newcomb, NY 12852	43.973100	-74.223200	94	95	95	95	0.059	0.059	0.056	0.058
New York	Franklin	55 Library Road, HOGANSBURG, NY 13655	44.980577	-74.695005	94	93	86	91	0.054	0.071	0.047	0.057
New York	Hamilton	PISECO LAKE AIRPORT	43.449570	-74.516250	97	98	95	97	0.058	0.062	0.061	0.060
New York	Herkimer	278 Bisby Road, NICKS LAKE CAMPGROUND	43.685780	-74.985380	98	99	96	98	0.057	0.061	0.071	0.063
New York	Jefferson	VAADI ROAD, PERCH RIVER	44.087470	-75.973160	99	98	88	95	0.059	0.065	0.067	0.063
New York	Monroe	2 YARMOUTH ROAD, RG&E Substation	43.146180	-77.548170	98	98	97	98	0.058	0.065	0.067	0.063
New York	New York	160 CONVENT AVENUE	40.819760	-73.948250	97	100	100	99	0.065	0.071	0.071	0.069
New York	Niagara	MIDDLEPORT STP, 3825 NORTH HARTLAND RD	43.223860	-78.478880	83	98	97	93	0.061	0.067	0.070	0.066
New York	Onondaga	5895 ENTERPRISE PARKWAY,	43.052350	-76.059210	96	96	100	97	0.063	0.063	0.067	0.064
New York	Orange	1175 ROUTE 17K, MONTGOMERY Valley Central HS	41.523750	-74.215340	99	99	96	98	0.062	0.072	0.064	0.066
New York	Oswego	820 COUNTY ROUTE 8	43.284280	-76.463240	98	99	85	94	0.058	0.063	0.061	0.060
New York	Putnam	NYSDEC FIELD HQTRS GYPSY TRAIL ROAD	41.455890	-73.709770	100	99	98	99	0.066	0.069	0.071	0.068
New York	Queens	Queens College 65-30 Kissena Blvd Parking Lot#6	40.736140	-73.821530	92	99	95	95	0.063	0.073	0.071	0.069
New York	Richmond	SUSAN WAGNER HS, 1200 MANOR RD, near Brielle Ave	40.596640	-74.125250	96	100	98	98	0.072	0.079	0.077	0.076
New York	Rockland	2 South Mountain Road	41.182080	-74.028190	100	100	98	99	0.068	0.077	0.073	0.072
New York	Saratoga	SARATOGA NATIONAL HISTORICAL PARK	43.012090	-73.648900	100	100	98	99	0.061	0.061	0.067	0.063
New York	Steuben	8301 ACKERSON ROAD, Pinnacle State Park	42.091420	-77.209780	91	85	94	90	0.058	0.059	0.062	0.059
New York	Suffolk	EAST FARMINGDALE WATER DIST., 72 GAZZA BLVD.	40.745290	-73.419190	99	98	94	97	0.066	0.078	0.073	0.072
New York	Suffolk	3059 SOUND AVENUE, RIVERHEAD	40.960780	-72.712380	95	99	95	96	0.064	0.076	0.078	0.072
New York	Suffolk	57 DIVISION STREET	40.827990	-73.057540	89	78	98	88	0.062	0.063	0.073	0.066
New York	Tompkins	Connecticut Hill Wildlife Management Area, Newfield, NY 14867	42.400600	-76.653800	95	96	98	96	0.059	0.064	0.066	0.063
New York	Wayne	4440 Ridge Road, WAYNE County Occupational CENTER, WILLIAMSON	43.230860	-77.171360	90	98	98	95	0.064	0.061	0.067	0.064
New York	Westchester	WHITE PLAINS PUMP STATION, 240 ORCHARD STREET	41.051920	-73.763660	100	100	100	100	0.074	0.073	0.075	0.074
North Carolina	Alexander	700 Liledoun Road	35.913900	-81.191000	99	97	99	98	0.064	0.065	0.066	0.065
North Carolina	Avery	7510 BLUE RIDGE PARKWAY SPUR	35.972347	-81.933072	99	97	95	97	0.061	0.062	0.063	0.062
North Carolina	Avery	Pisgah National Forest, Newland, NC 28657	36.105800	-82.045400	94	96	90	93	0.062	0.062	0.069	0.064
North Carolina	Buncombe	ROUTE 191 SOUTH BREVARD RD	35.500102	-82.599860	100	99	99	99	0.064	0.065	0.062	0.063
North Carolina	Caldwell	219 NUWAY CIRCLE	35.935833	-81.530278	98	96	100	98	0.061	0.065	0.066	0.064
North Carolina	Carteret	Open Grounds Farm, Beaufort, NC 28516	34.884800	-76.620300	93	82	91	89	0.062	0.058	0.060	0.060

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North Carolina	Caswell	7074 CHERRY GROVE RD, REIDSVILLE	36.307033	-79.467417	100	98	97	98	0.067	0.060	0.064	0.063
North Carolina	Cumberland	7112 Covington Lane	35.158686	-78.728035	99	97	99	98	0.061	0.060	0.064	0.061
North Carolina	Durham	801 STADIUM DRIVE	36.032944	-78.905417	100	96	98	98	0.062	0.061	0.063	0.062
North Carolina	Edgecombe	7589 NC Hwy 33-NW	35.988333	-77.582778	77	95	92	88	0.062	0.061	0.064	0.062
North Carolina	Forsyth	1300 BLK. HATTIE AVENUE	36.110556	-80.226667	100	99	99	99	0.065	0.070	0.066	0.067
North Carolina	Forsyth	FRATERNITY CHURCH ROAD	36.026000	-80.342000	99	99	98	99	0.067	0.068	0.070	0.068
North Carolina	Forsyth	3656 PIEDMONT MEMORIAL DRIVE	36.050833	-80.143889	99	98	98	98	0.067	0.065	0.069	0.067
North Carolina	Graham	FOREST ROAD 423 SPUR	35.257930	-83.795620	99	94	100	98	0.063	0.065	0.066	0.064
North Carolina	Granville	800 Central Avenue	36.141111	-78.768056	100	100	100	100	0.065	0.063	0.065	0.064
North Carolina	Guilford	205 WILOUGHBY BLVD	36.109167	-79.801111	100	99	99	99	0.065	0.064	0.068	0.065
North Carolina	Haywood	2236 ASHEVILLE ROAD	35.507160	-82.963370	95	95	100	97	0.060	0.064	0.062	0.062
North Carolina	Haywood	TOWER BLUE RIDGE PKWY MILE MARKER 410	35.379167	-82.792500	91	93	99	94	0.067	0.065	0.067	0.066
North Carolina	Haywood	6905 Purchase Knob Road	35.587144	-83.074156	99	99	91	96	0.064	0.066	0.065	0.065
North Carolina	Johnston	1338 JACK ROAD	35.590833	-78.461944	99	99	99	99	0.064	0.065	0.066	0.065
North Carolina	Lee	4110 Blackstone Drive	35.432500	-79.288700	99	100	98	99	0.064	0.060	0.064	0.062
North Carolina	Lenoir	HIGHWAY 70 EAST AND HIGHWAY 58 SOUTH	35.231459	-77.568792	100	97	90	96	0.065	0.062	0.063	0.063
North Carolina	Lincoln	1487 RIVERVIEW ROAD	35.438556	-81.276750	99	97	98	98	0.064	0.068	0.069	0.067
North Carolina	Macon	USDA Southern Research Station, Coweeta Hydrologic Laboratory, Otto, NC 28763	35.060800	-83.430600	95	94	94	94	0.063	0.062	0.061	0.062
North Carolina	Martin	1210 Hayes Street	35.810660	-76.906249	100	96	92	96	0.061	0.059	0.061	0.060
North Carolina	Mecklenburg	1130 EASTWAY DRIVE	35.240100	-80.785683	100	100	99	100	0.065	0.073	0.070	0.069
North Carolina	Montgomery	136 Perry Dr, Candor, NC 27229	35.263200	-79.836500	100	96	87	94	0.062	0.059	0.062	0.061
North Carolina	New Hanover	6028 HOLLY SHELTER RD	34.364167	-77.838611	99	97	97	98	0.063	0.057	0.060	0.060
North Carolina	Person	SR49	36.306965	-79.091970	100	100	100	100	0.065	0.061	0.063	0.063
North Carolina	Pitt	403 Government Circle	35.638610	-77.358050	95	97	92	95	0.063	0.060	0.065	0.062
North Carolina	Rockingham	6371 NC 65 @ BETHANY SCHOOL	36.308889	-79.859167	100	100	99	100	0.065	0.066	0.067	0.066
North Carolina	Rowan	301 WEST ST & GOLD HILL AVENUE	35.551868	-80.395039	100	98	100	99	0.064	0.066	0.066	0.065
North Carolina	Swain	30 Recreation Park Drive	35.434767	-83.442133	100	100	97	99	0.058	0.060	0.064	0.060
North Carolina	Swain	1501 Acquoni Rd.	35.498711	-83.310242	95	92	98	95	0.060	0.063	0.061	0.061
North Carolina	Union	701 CHARLES STREET	34.973889	-80.540833	100	97	98	98	0.067	0.067	0.070	0.068
North Carolina	Wake	3801 SPRING FOREST RD.	35.856111	-78.574167	100	97	97	98	0.063	0.065	0.069	0.065
North Carolina	Yancey	2388 State Highway 128	35.765413	-82.264944	92	90	95	92	0.061	0.064	0.070	0.065
North Dakota	Billings	13881 194 EAST	46.894300	-103.378530	99	99	99	99	0.055	0.064	0.056	0.058
North Dakota	Burke	8315 HIGHWAY 8, KENMARE	48.641930	-102.401800	97	97	99	98	0.058	0.065	0.055	0.059

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North Dakota	Burleigh	1810 N 16TH STREET	46.825425	-100.768210	99	95	72	89	0.059	0.063	0.050	0.057
North Dakota	Cass	4266 40TH AVE NORTH	46.933754	-96.855350	92	78	96	89	0.059	0.057	0.054	0.056
North Dakota	Dunn	9610 SEVENTH STREET SW	47.313200	-102.527300	98	100	99	99	0.057	0.063	0.056	0.058
North Dakota	McKenzie	229 SERVICE RD., WATFORD CITY	47.581200	-103.299500	95	96	100	97	0.056	0.061	0.056	0.057
North Dakota	Mercer	6024 HIGHWAY 200	47.298611	-101.766944	98	99	54	84	0.055	0.060	0.053	0.056
North Dakota	Oliver	1575 HIGHWAY 31	47.185833	-101.428056	89	100	100	96	0.060	0.064	0.054	0.059
North Dakota	Williams	10th Street West	48.152780	-103.639510	97	99	98	98	0.056	0.059	0.053	0.056
Ohio	Allen	2850 BIBLE ROAD	40.770944	-84.053900	99	99	99	99	0.066	0.064	0.068	0.066
Ohio	Ashtabula	770 LAKE RD.	41.959695	-80.572808	99	98	99	99	0.069	0.070	0.072	0.070
Ohio	Butler	SCHULER AND BENDER	39.383382	-84.544413	99	94	95	96	0.070	0.070	0.076	0.072
Ohio	Butler	1701 Runway Dr.	39.529481	-84.393364	98	99	98	98	0.069	0.070	0.074	0.071
Ohio	Butler	Ecology Research Center, Miami University, Oxford, Ohio 45056	39.532700	-84.728600	95	88	94	92	0.069	0.068	0.072	0.069
Ohio	Clark	5171 URBANA	40.001030	-83.804560	100	100	100	100	0.065	0.071	0.071	0.069
Ohio	Clark	5400 SPANGLER	39.855670	-83.997730	100	100	96	99	0.064	0.069	0.070	0.067
Ohio	Clermont	2400 CLERMONT CENTER DR.	39.082800	-84.144100	99	98	99	99	0.068	0.070	0.073	0.070
Ohio	Clinton	62 LAUREL DR.	39.430038	-83.788502	99	100	98	99	0.070	0.070	0.071	0.070
Ohio	Cuyahoga	891 E. 152 ST.	41.555230	-81.575256	99	99	94	97	0.071	0.068	0.070	0.069
Ohio	Cuyahoga	E. 14TH & ORANGE	41.492117	-81.678449	97	98	99	98	0.066	0.063	0.063	0.064
Ohio	Cuyahoga	390 FAIR ST.	41.361890	-81.864608	96	100	99	98	0.059	0.066	0.068	0.064
Ohio	Cuyahoga	6116 WILSON MILLS	41.537344	-81.458834	99	99	99	99	0.061	0.072	0.071	0.068
Ohio	Delaware	359 MAIN RD.	40.356694	-83.063971	99	100	98	99	0.066	0.068	0.067	0.067
Ohio	Fayette	Deer Creek State Park, Mt Sterling, OH 43143	39.635900	-83.260500	93	97	99	96	0.069	0.070	0.067	0.068
Ohio	Franklin	7600 FODOR RD.	40.084555	-82.815550	100	100	99	100	0.070	0.071	0.072	0.071
Ohio	Franklin	1777 E. BROAD	39.965230	-82.955490	99	98	97	98	0.069	0.064	0.067	0.066
Ohio	Franklin	5750 MAPLE CANYON	40.087700	-82.959773	100	100	100	100	0.068	0.063	0.071	0.067
Ohio	Geauga	13000 AUBURN	41.515051	-81.249906	100	100	99	100	0.065	0.073	0.077	0.071
Ohio	Greene	541 LEDBETTER RD.,	39.665750	-83.942680	100	100	100	100	0.066	0.071	0.069	0.068
Ohio	Hamilton	11590 GROOMS RD	39.278700	-84.366250	97	98	99	98	0.071	0.072	0.075	0.072
Ohio	Hamilton	6950 RIPPLE RD.	39.214870	-84.690860	99	99	99	99	0.073	0.070	0.073	0.072
Ohio	Hamilton	250 WM. HOWARD TAFT	39.128860	-84.504040	100	99	99	99	0.069	0.071	0.073	0.071
Ohio	Jefferson	618 LOGAN ST.	40.366440	-80.615580	99	100	100	100	0.067	0.066	0.062	0.065
Ohio	Knox	WATER PLT. SR. 314	40.310025	-82.691724	100	97	99	99	0.066	0.071	0.066	0.067
Ohio	Lake	36010 LAKESHORE	41.673006	-81.422455	100	98	100	99	0.075	0.074	0.076	0.075
Ohio	Lake	177 MAIN STREET	41.726811	-81.242156	99	99	99	99	0.062	0.070	0.069	0.067
Ohio	Lawrence	S.R. 141, WILGUS	38.629010	-82.458860	97	99	98	98	0.064	0.065	0.065	0.064

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Ohio	Lawrence	460 Commerce Drive	38.508075	-82.659241	98	97	96	97	0.062	0.069	0.070	0.067
Ohio	Licking	300 LICKING VIEW DR.	40.026037	-82.433000	100	99	99	99	0.066	0.068	0.067	0.067
Ohio	Lorain	4706 DETROIT RD.	41.420882	-82.095729	99	98	98	98	0.067	0.062	0.070	0.066
Ohio	Lucas	348 S. ERIE	41.644067	-83.546160	78	93	100	90	0.070	0.063	0.070	0.067
Ohio	Lucas	200 SOUTH RIVER RD.	41.494200	-83.718949	100	100	99	100	0.064	0.063	0.065	0.064
Ohio	Lucas	1002 N. YONDOTA	41.675231	-83.307005	93	92	83	89	0.065	0.064	0.063	0.064
Ohio	Madison	940 SR 38 SW	39.788190	-83.476060	95	100	99	98	0.069	0.069	0.068	0.068
Ohio	Mahoning	345 OAKHILL	41.096142	-80.658856	93	98	96	96	0.066	0.069	0.054	0.063
Ohio	Medina	BALLASH ROAD	41.060400	-81.923900	99	100	95	98	0.064	0.063	0.066	0.064
Ohio	Miami	3825 NORTH S. R. 589	40.085020	-84.113808	100	99	99	99	0.066	0.068	0.069	0.067
Ohio	Montgomery	1401 Harshman Road	39.785630	-84.134370	99	100	100	100	0.069	0.070	0.072	0.070
Ohio	Noble	58163 St. Johns Rd. Quaker City, OH 43773	39.942800	-81.337300	97	98	93	96	0.065	0.066	0.068	0.066
Ohio	Portage	1570 RAVENNA RD.	41.182466	-81.330486	99	100	99	99	0.061	0.064	0.059	0.061
Ohio	Preble	6940 OXFORD GETTYSBURG RD.	39.835620	-84.720490	100	100	100	100	0.065	0.067	0.069	0.067
Ohio	Stark	515 25TH. ST.	40.828052	-81.378333	94	93	98	95	0.065	0.072	0.072	0.069
Ohio	Stark	45 S. WABASH AVENUE, S.R 93	40.712778	-81.598333	96	93	97	95	0.059	0.068	0.067	0.064
Ohio	Stark	1175 WEST VINE	40.931398	-81.123519	99	94	97	97	0.061	0.067	0.071	0.066
Ohio	Summit	800 PATTERSON AVE.	41.106486	-81.503547	100	99	99	99	0.058	0.065	0.061	0.061
Ohio	Trumbull	842 YOUNGSTOWN-KINGSVILLE RD.	41.240456	-80.662559	90	100	100	97	0.065	0.070	0.071	0.068
Ohio	Warren	430 SOUTHEAST ST.	39.426890	-84.200770	100	100	97	99	0.071	0.071	0.074	0.072
Ohio	Washington	2000 4TH STREET	39.432117	-81.460443	93	100	100	98	0.063	0.068	0.064	0.065
Ohio	Wood	347 N DUNBRIDGE	41.377685	-83.611104	100	98	100	99	0.063	0.062	0.066	0.063
Oklahoma	Adair	South Highway 59, RR1, 1795 Dahlenegah Park Road, Stilwell, Oklahoma	35.750735	-94.669697	98	90	97	95	0.063	0.062	0.058	0.061
Oklahoma	Caddo	WESTERN DELAWARE TRIBAL COMPLEX	35.111944	-98.252778	87	94	49	77	0.055	0.057	0.054	0.055
Oklahoma	Canadian	12575 NW 10TH (WATER TOWER)	35.479215	-97.751503	93	96	89	93	0.068	0.066	0.062	0.065
Oklahoma	Cherokee	P.O. BOX 948 TAHLEQUAH, OK 74464	35.854080	-94.985964	100	97	95	97	0.061	0.061	0.058	0.060
Oklahoma	Cleveland	S. E. 19TH STREET	35.320105	-97.484099	99	100	99	99	0.068	0.065	0.065	0.066
Oklahoma	Comanche	2211 NW 25 Lawton, Oklahoma 73502 (LPS: Prof. Dev. Center)	34.632980	-98.428790	100	98	100	99	0.069	0.066	0.061	0.065
Oklahoma	Creek	MANNFORD WATER PLANT	36.105481	-96.361196	95	95	93	94	0.066	0.063	0.064	0.064
Oklahoma	Dewey	SEILING MUNICIPAL AIRPORT	36.158414	-98.931973	92	92	93	92	0.067	0.065	0.065	0.065
Oklahoma	Kay	NORTH OF NEWKIRK OK. ON HWY 77 - EAST HOME ROAD (E0040RD) .90 MILES	36.956222	-97.031350	96	88	97	94	0.069	0.063	0.059	0.063
Oklahoma	McClain	310 E. BURR OAK	35.159649	-97.473794	99	98	99	99	0.069	0.065	0.065	0.066
Oklahoma	Mayes	CHEROKEE HEIGHTS DRIVE	36.228408	-95.249943	98	97	100	98	0.060	0.065	0.061	0.062
Oklahoma	Oklahoma	NE 10TH & STONEWALL	35.477036	-97.494309	100	99	99	99	0.069	0.068	0.065	0.067
Oklahoma	Oklahoma	12880 A NE 10TH	35.477801	-97.303044	98	97	98	98	0.066	0.067	0.064	0.065

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Oklahoma	Oklahoma	2501 E. Memorial Rd. (OKLAHOMA CHRISTIAN UNIVERSITY)	35.614131	-97.475083	98	93	91	94	0.070	0.068	0.068	0.068
Oklahoma	Ottawa	QUAPAW TRIBE'S INDUSTRIAL PARK, MIAMI	36.922222	-94.838889	96	93	95	95	0.055	0.056	0.053	0.054
Oklahoma	Pittsburg	104 AIRPORT ROAD	34.902270	-95.784375	93	100	99	97	0.062	0.060	0.059	0.060
Oklahoma	Sequoyah	207 Cherokee Boulevard, Roland, OK 74954	35.408140	-94.524413	88	99	90	92	0.060	0.062	0.058	0.060
Oklahoma	Tulsa	1100 SOUTH OSAGE DRIVE	36.357438	-95.999247	97	98	17	71	0.065	0.066	0.060	0.063
Oklahoma	Tulsa	502 EAST 144TH. PL.	35.953708	-96.004975	99	84	83	89	0.062	0.061	0.064	0.062
Oklahoma	Tulsa	18707 E. 21ST STREET	36.133802	-95.764537	95	99	98	97	0.063	0.065	0.063	0.063
Oklahoma	Tulsa	3520 1/2 N. PEORIA	36.204902	-95.976537	100	95	96	97	0.065	0.068	0.062	0.065
Oregon	Clackamas	CARUS TRAILER/SPANGLER RD	45.259280	-122.588151	99	96	99	98	0.062	0.069	0.064	0.065
Oregon	Columbia	SAUVIE ISLAND-SSB/ROUTE 1, BOX 442	45.768531	-122.772100	99	97	99	98	0.053	0.058	0.051	0.054
Oregon	Jackson	7112 RAPP LANE, TALENT	42.229891	-122.787700	98	91	85	91	0.059	0.069	0.049	0.059
Oregon	Lane	AMAZON PARK/499 E 29TH	44.026312	-123.083737	99	99	99	99	0.058	0.068	0.057	0.061
Oregon	Lane	79980 DELIGHT VALLEY SCHOOL RD, SAGINAW	43.834500	-123.035300	99	99	99	99	0.058	0.071	0.056	0.061
Oregon	Marion	CASCADE JR HIGH 10226 MARION RD TURNER O	44.810290	-122.915085	94	79	98	90	0.066	0.065	0.065	0.065
Oregon	Multnomah	SE LAFAYETTE/5824 SE LAFAYETTE	45.496641	-122.602877	99	99	96	98	0.055	0.057	0.055	0.055
Oregon	Umatilla	HERMISTON MUNICIPAL AIRPORT, 1600 AIRPORT WAY, HERMISTON	45.828968	-119.262991	95	98	89	94	0.064	0.070	0.063	0.065
Oregon	Washington	6745 Bradbury Court, Tualatin, OR (NO2 roadway site)	45.393497	-122.747894	92	100	95	96	0.053	0.052	0.048	0.051
Oregon	Washington	17180 SW Lasich Lane, Sherwood - Lasich and Roy Rogers (SLR)	45.402446	-122.854437	98	99	98	98	0.057	0.063	0.058	0.059
Pennsylvania	Adams	747 Winding Rd, Biglerville, PA 17307	39.923100	-77.307800	94	97	97	96	0.063	0.067	0.071	0.067
Pennsylvania	Allegheny	301 39th St, Pittsburgh, PA 15201 (Lawrenceville)	40.465420	-79.960757	100	97	96	98	0.065	0.071	0.067	0.067
Pennsylvania	Allegheny	3640 Old Oakdale Rd, McDonald, PA 15057 (South Fayette)	40.375644	-80.169943	99	100	99	99	0.065	0.068	0.072	0.068
Pennsylvania	Allegheny	1500 Pacific Ave, Natrona Heights, PA 15065 (Harrison)	40.617488	-79.727664	99	93	86	93	0.071	0.074	0.067	0.070
Pennsylvania	Armstrong	GLADE DR. & NOLTE RD, KITTANNING	40.814183	-79.564750	100	100	93	98	0.068	0.070	0.068	0.068
Pennsylvania	Beaver	ROUTE 168 & TOMLINSON ROAD	40.562520	-80.503948	98	98	95	97	0.069	0.070	0.069	0.069

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Pennsylvania	Beaver	1015 SEBRING ROAD	40.684722	-80.359722	96	95	94	95	0.070	0.067	0.068	0.068
Pennsylvania	Beaver	EIGHT STREET AND RIVER ALLEY	40.747796	-80.316442	100	95	98	98	0.066	0.063	0.066	0.065
Pennsylvania	Berks	Kutztown University Campus	40.514080	-75.789721	96	90	83	90	0.063	0.066	0.064	0.064
Pennsylvania	Berks	1059 Arnold Road	40.383350	-75.968600	98	99	99	99	0.068	0.071	0.068	0.069
Pennsylvania	Blair	2ND AVE & 7TH ST	40.535278	-78.370833	98	97	77	91	0.060	0.069	0.062	0.063
Pennsylvania	Bradford	RT 4148 Main St, Monroe, PA 18832	41.705226	-76.512726	100	100	97	99	0.051	0.058	0.057	0.055
Pennsylvania	Bucks	ROCKVIEW LANE	40.107222	-74.882222	99	96	93	96	0.071	0.082	0.074	0.075
Pennsylvania	Cambria	MILLER AUTO SHOP 1 MESSENGER ST	40.309722	-78.915000	100	98	96	98	0.060	0.065	0.062	0.062
Pennsylvania	Centre	PENN STATE UNIVERSITY - ARBORETUM SITE	40.811389	-77.877028	100	90	97	96	0.063	0.064	0.063	0.063
Pennsylvania	Centre	1366 Tadpole Rd, Pennsylvania Furnace, PA 16865	40.720800	-77.931900	100	100	98	99	0.064	0.068	0.065	0.065
Pennsylvania	Chester	NEW GARDEN AIRPORT - TOUGHKENAMON	39.834461	-75.768242	94	97	96	96	0.071	0.068	0.072	0.070
Pennsylvania	Clearfield	LOCATED NEAR S.B. ELLIOTT STATE PARK	41.117500	-78.526194	91	97	93	94	0.060	0.068	0.066	0.064
Pennsylvania	Dauphin	651 Gibson Blvd., Harrisburg, PA 17104	40.246992	-76.846988	86	100	97	94	0.063	0.068	0.062	0.064
Pennsylvania	Dauphin	SIPE AVE & MAE STREET	40.272222	-76.681389	93	99	100	97	0.063	0.068	0.065	0.065
Pennsylvania	Delaware	FRONT ST & NORRIS ST	39.835556	-75.372500	98	96	97	97	0.073	0.074	0.070	0.072
Pennsylvania	Elk	Kane Experimental Forest, Allegheny National Forest, Wilcox, PA 15870	41.598000	-78.767400	100	93	91	95	0.064	0.067	0.067	0.066
Pennsylvania	Erie	10TH AND MARNE STREETS	42.141750	-80.038611	90	100	98	96	0.065	0.066	0.065	0.065
Pennsylvania	Franklin	FOREST ROAD - METHODIST HILL	39.961111	-77.475556	98	99	92	96	0.063	0.059	0.058	0.060
Pennsylvania	Greene	4.8 KM SE OF HOLBROOK	39.809330	-80.265670	99	99	91	96	0.065	0.071	0.067	0.067
Pennsylvania	Indiana	PA. DEPT. OF TRANSPORTATION - RT.403 - Strongstown	40.563330	-78.919972	100	100	98	99	0.068	0.073	0.066	0.069

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Pennsylvania	Lackawanna	WILSON FIRE CO. ERIE & PLEASANT	41.479116	-75.578186	100	99	92	97	0.061	0.069	0.068	0.066
Pennsylvania	Lackawanna	GEORGE ST TROOP AND CITY OF SCRANTON	41.442778	-75.623056	55	100	95	83	0.060	0.068	0.065	0.064
Pennsylvania	Lancaster	ABRAHAM LINCOLN JR HIGH GROFFTOWN RD	40.046667	-76.283333	100	100	93	98	0.066	0.071	0.064	0.067
Pennsylvania	Lancaster	3545 W. Newport Road	40.043833	-76.112400	100	93	96	96	0.063	0.070	0.061	0.064
Pennsylvania	Lawrence	CROTON ST & JEFFERSON ST.	40.995848	-80.346442	100	98	96	98	0.068	0.069	0.066	0.067
Pennsylvania	Lebanon	125 Birch Road	40.337328	-76.383447	100	99	99	99	0.067	0.074	0.067	0.069
Pennsylvania	Lehigh	STATE HOSPITAL REAR 1600 HANOVER AVE	40.611944	-75.432500	96	98	89	94	0.068	0.070	0.067	0.068
Pennsylvania	Luzerne	CHILWICK & WASHINGTON STS	41.265556	-75.846389	100	97	95	97	0.060	0.067	0.065	0.064
Pennsylvania	Lycoming	899 CHERRY STREET	41.250800	-76.923800	100	100	99	100	0.062	0.065	0.063	0.063
Pennsylvania	Mercer	PA518 (NEW CASTLE ROAD) & PA418	41.215014	-80.484779	98	78	99	92	0.071	0.066	0.069	0.068
Pennsylvania	Mercer	Maurice K Goddard State Park, Sandy Lake, PA 16145	41.427100	-80.145100	92	100	91	94	0.060	0.068	0.069	0.065
Pennsylvania	Monroe	HC 1, BOX 95-B NEAR RT 611 & BROOKDALE ROAD	41.083060	-75.323280	99	100	95	98	0.060	0.067	0.068	0.065
Pennsylvania	Montgomery	STATE ARMORY - 1046 BELVOIR RD	40.112222	-75.309167	99	99	93	97	0.072	0.073	0.066	0.070
Pennsylvania	Northampton	WASHINGTON & CAMBRIA STS. FREEMANSBURG	40.628056	-75.341111	98	99	93	97	0.067	0.070	0.069	0.068
Pennsylvania	Northampton	17TH AND SPRING GARDEN STREETS	40.692224	-75.237156	92	98	88	93	0.066	0.067	0.065	0.066
Pennsylvania	Philadelphia	1501 E. LYCOMING AVE.	40.008889	-75.097780	98	100	99	99	0.058	0.057	0.064	0.059
Pennsylvania	Philadelphia	GRANT-ASHTON ROADS PHILA NE AIRPORT	40.076400	-75.011549	94	100	97	97	0.072	0.079	0.074	0.075
Pennsylvania	Philadelphia	3000 LEWIS ST. (NEAR BATH ST.) Philadelphia, PA. 19137	39.991389	-75.080833	93	98	98	96	0.068	0.078	0.071	0.072
Pennsylvania	Somerset	Laurel Hill State Park, Rockwood, PA 15557	39.987800	-79.251500	98	67	97	87	0.062	0.058	0.067	0.062
Pennsylvania	Tioga	TIOGA	41.644722	-76.939167	97	96	100	98	0.058	0.065	0.062	0.061
Pennsylvania	Washington	CHARLER01 WASTE TREATMENT PLANT	40.146667	-79.902222	100	98	98	99	0.065	0.072	0.069	0.068

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Pennsylvania	Washington	MCCARRELL AND FAYETTE STS	40.170556	-80.261389	98	100	99	99	0.064	0.069	0.064	0.065
Pennsylvania	Washington	HILLMAN STATE PARK - KINGS CREEK ROAD	40.445278	-80.420833	98	95	100	98	0.064	0.071	0.068	0.067
Pennsylvania	Westmoreland	DONOHOE ROAD - PENN DOT MAINT DIST BLDG	40.304694	-79.505667	99	96	100	98	0.064	0.069	0.072	0.068
Pennsylvania	York	HILL ST.	39.965278	-76.699444	99	100	99	99	0.063	0.068	0.067	0.066
Pennsylvania	York	2650 Delta Road	39.860970	-76.462055	54	98	92	81	0.063	0.074	0.066	0.067
Rhode Island	Kent	W. ALTON JONES CAMPUS URI PARKERFIELD WE	41.615237	-71.720000	98	98	96	97	0.067	0.070	0.075	0.070
Rhode Island	Providence	FRANCIS SCHOOL, 64 BOURNE AVE	41.841573	-71.360770	98	98	97	98	0.064	0.071	0.071	0.068
Rhode Island	Washington	TARZWELL ROAD, NARRAGANSETT	41.495110	-71.423705	98	99	99	99	0.063	0.077	0.071	0.070
South Carolina	Abbeville	59 JIM SCOTT LANE [Due West]	34.325318	-82.386376	84	90	76	83	0.059	0.055	0.062	0.058
South Carolina	Aiken	8217 ATOMIC ROAD (INDIAN DRIVE) [Jackson]	33.342226	-81.788731	96	98	99	98	0.062	0.057	0.062	0.060
South Carolina	Anderson	215 McAlister Road [Big Creek]	34.623236	-82.532059	99	96	100	98	0.060	0.063	0.062	0.061
South Carolina	Berkeley	River Oak Drive (Goose Creek) [Bushy Park]	32.987252	-79.936700	97	93	85	92	0.060	0.054	0.058	0.057
South Carolina	Charleston	390 BULLS ISLAND ROAD (AWENDAW) [Cape Romain]	32.941023	-79.657187	92	99	67	86	0.059	0.054	0.060	0.057
South Carolina	Cherokee	McGinnis Road [Cowpens]	35.130396	-81.816567	98	100	0	66	0.066	0.065	0.041	0.057
South Carolina	Chesterfield	RT 2 BOX 100 MCBEE (SC145) [Chesterfield]	34.615367	-80.198787	99	96	91	95	0.059	0.059	0.063	0.060
South Carolina	Colleton	Ashton Road [Ashton]	33.007866	-80.965038	90	82	86	86	0.059	0.054	0.057	0.056
South Carolina	Darlington	2200 POCKET ROAD [Pee Dee]	34.285696	-79.744859	98	96	97	97	0.063	0.061	0.063	0.062
South Carolina	Edgefield	660 WOODYARD ROAD [Trenton]	33.739963	-81.853635	94	93	96	94	0.055	0.061	0.062	0.059
South Carolina	Greenville	510 Garrison Road [Hillcrest]	34.751848	-82.256701	98	96	95	96	0.062	0.067	0.066	0.065
South Carolina	Oconee	ROUND MOUNTAIN TOWER ROAD [Long Creek]	34.805261	-83.237700	96	98	96	97	0.064	0.060	0.065	0.063
South Carolina	Pickens	106 HOPEWELL ROAD [Clemson]	34.653606	-82.838659	98	97	96	97	0.062	0.064	0.067	0.064
South Carolina	Pickens	901 Allgood Bridge Road [Wolf Creek]	34.851537	-82.744576	96	97	97	97	0.058	0.063	0.063	0.061
South Carolina	Richland	8311 PARKLANE ROAD [Parklane]	34.093959	-80.962304	99	92	75	89	0.056	0.056	0.065	0.059
South Carolina	Richland	1850 SOUTH CEDAR CREEK ROAD [Congaree Bluff]	33.814680	-80.781135	97	90	92	93	0.056	0.055	0.056	0.055

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South Carolina	Richland	900 CLEMSON ROAD [Sandhill]	34.131262	-80.868318	98	99	99	99	0.065	0.062	0.070	0.065
South Carolina	Spartanburg	1556 JOHN DODD ROAD [North Spartanburg Fire Station #2]	34.988706	-82.075802	93	82	79	85	0.065	0.067	0.069	0.067
South Carolina	York	2316 CHESTER HIGHWAY (US 321) [York]	34.935817	-81.228409	94	94	99	96	0.056	0.061	0.061	0.059
South Dakota	Brookings	3714 Western Ave, Brookings, SD 57006	44.348604	-96.807299	99	99	93	97	0.061	0.063	0.061	0.061
South Dakota	Custer	WIND CAVE NATIONAL PARK, SOUTH DAKOTA	43.557640	-103.483860	98	99	98	98	0.057	0.059	0.060	0.058
South Dakota	Jackson	BADLANDS PO BOX 6 HEADQUARTERS	43.745610	-101.941218	99	99	100	99	0.057	0.057	0.060	0.058
South Dakota	Meade	7108 SEEAIRE ST - BLACK HAWK ELEMENTARY	44.155636	-103.315765	98	100	98	99	0.056	0.059	0.058	0.057
South Dakota	Minnehaha	2001 E 8th St	43.547920	-96.700769	96	99	98	98	0.066	0.061	0.064	0.063
South Dakota	Union	31986 475th Ave	42.751518	-96.707208	100	100	98	99	0.062	0.061	0.060	0.061
Tennessee	Anderson	FREELS BEND STUDY AREA MELTON LAKE Oak Ridge National Lab Reservation	35.965220	-84.223160	94	98	89	94	0.060	0.065	0.066	0.063
Tennessee	Blount	GREAT SMOKY MOUNTAINS NP LOOK ROCK	35.633480	-83.941606	99	98	96	98	0.064	0.068	0.070	0.067
Tennessee	Blount	GREAT SMOKY MOUNTAINS NP - CADES COVE	35.603056	-83.783611	98	95	97	97	0.058	0.060	0.063	0.060
Tennessee	Claiborne	718 Russell Hill Rd, Speedwell, TN 37870	36.470000	-83.826800	98	99	93	97	0.065	0.061	0.064	0.063
Tennessee	Davidson	1015 TRINITY LANE	36.205000	-86.744722	95	98	100	98	0.065	0.067	0.068	0.066
Tennessee	Davidson	3711 BELL ROAD	36.150742	-86.623301	98	99	100	99	0.071	0.064	0.068	0.067
Tennessee	DeKalb	Edgar Evans State Park, Smithville, TN 37166	36.038800	-85.733100	98	96	96	97	0.062	0.061	0.063	0.062
Tennessee	Hamilton	SODDY DAISY H.S. 00618 SEQUOYAH RD	35.233476	-85.181581	98	99	100	99	0.064	0.068	0.065	0.065
Tennessee	Hamilton	3018 HICKORY VALLEY RD- EASTSIDE UTILITY FILTER PLANT (RESERVOIR ROAD)	35.102638	-85.162194	99	99	99	99	0.067	0.068	0.069	0.068
Tennessee	Jefferson	2393 Forrester Road, New Market TN 37820	36.105629	-83.602077	99	100	97	99	0.067	0.070	0.068	0.068
Tennessee	Knox	9315 RUTLEDGE PIKE MASCOT TN 37806	36.085508	-83.764806	97	98	99	98	0.060	0.066	0.067	0.064
Tennessee	Knox	4625 MILDRED DRIVE	36.019186	-83.873810	98	99	100	99	0.063	0.066	0.070	0.066
Tennessee	Loudon	130 WEBB DRIVE Loudon TN 37774	35.744539	-84.317057	98	96	95	96	0.067	0.069	0.073	0.069
Tennessee	Sevier	GREAT SMOKY MOUNTAIN NP COVE MOUNTAIN	35.696667	-83.609722	97	98	96	97	0.068	0.067	0.070	0.068

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Tennessee	Sevier	CLINGSMANS DOME, GREAT SMOKY MTNS. NP	35.562778	-83.498056	72	67	73	71	0.065	0.065	0.066	0.065
Tennessee	Shelby	1330 FRAYSER BLVD	35.217501	-90.019707	99	98	98	98	0.067	0.065	0.071	0.067
Tennessee	Shelby	6388 Haley Rd. (Shelby Farms NCORE site)	35.151699	-89.850249	99	98	100	99	0.066	0.066	0.068	0.066
Tennessee	Shelby	6855 MUDVILLE RD.	35.378153	-89.834470	99	98	99	99	0.065	0.066	0.067	0.066
Tennessee	Sullivan	Indian Springs School Shawnee Ave Blountville TN	36.541452	-82.424485	99	97	98	98	0.065	0.065	0.068	0.066
Tennessee	Sullivan	3301 BLOOMINGDALE RD. Kingsport TN 37620	36.582110	-82.485742	97	99	99	98	0.061	0.066	0.067	0.064
Tennessee	Sumner	ROCKLAND RECREATION AREA-OLD HICKORY DAM Army Corp of Engineer Property	36.297560	-86.653137	99	100	99	99	0.066	0.067	0.068	0.067
Tennessee	Williamson	FAIRVIEW MIDDLE SCHOOL CROW CUT ROAD Fairview TN 37062	35.951155	-87.137041	96	98	99	98	0.063	0.061	0.061	0.061
Tennessee	Wilson	CEDARS OF LEBANON STATE PARK	36.060833	-86.286261	97	98	91	95	0.064	0.061	0.067	0.064
Texas	Bell	8406 Georgia Avenue	31.122419	-97.431052	96	100	99	98	0.067	0.072	0.064	0.067
Texas	Bell	1605 Stone Tree Drive	31.088002	-97.679734	99	99	98	99	0.069	0.067	0.066	0.067
Texas	Bexar	6655 Bluebird Lane	29.515090	-98.620166	99	97	97	98	0.069	0.079	0.071	0.073
Texas	Bexar	F Range (1000Yd marker off Wilderness Trail)	29.632058	-98.564936	95	96	96	96	0.072	0.080	0.069	0.073
Texas	Bexar	14620 Laguna Rd	29.275381	-98.311692	99	100	99	99	0.063	0.068	0.062	0.064
Texas	Brazoria	4503 Croix Pkwy	29.520443	-95.392509	99	98	98	98	0.071	0.086	0.069	0.075
Texas	Brazoria	109B Brazoria Hwy 332 West	29.043759	-95.472946	98	92	97	96	0.061	0.065	0.066	0.064
Texas	Brewster	BIG BEND NATIONAL PARK, TEXAS	29.302650	-103.177810	95	95	96	95	0.063	0.063	0.061	0.062
Texas	Cameron	344 Porter Drive	25.892518	-97.493830	98	93	92	94	0.060	0.060	0.052	0.057
Texas	Cameron	1602 W Teege Avenue	26.200335	-97.712684	93	98	98	96	0.059	0.058	0.055	0.057
Texas	Collin	6590 Hillcrest Road	33.132400	-96.786419	98	98	98	98	0.074	0.077	0.073	0.074
Texas	Dallas	1415 Hinton Street	32.820061	-96.860117	96	100	93	96	0.066	0.080	0.069	0.071
Texas	Dallas	12532 1/2 Nuestra Drive	32.919206	-96.808498	86	98	99	94	0.070	0.079	0.067	0.072
Texas	Dallas	3277 W Redbird Lane	32.676451	-96.872060	97	98	88	94	0.063	0.068	0.062	0.064
Texas	Denton	Denton Airport South	33.219069	-97.196284	100	97	98	98	0.077	0.088	0.076	0.080

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Texas	Denton	792 E Northside Dr	33.410648	-96.944590	99	98	99	99	0.075	0.079	0.075	0.076
Texas	Ellis	2725 Old Fort Worth Road	32.482083	-97.026899	98	99	100	99	0.062	0.068	0.060	0.063
Texas	Ellis	900 FM 667 Ellis County	32.175417	-96.870189	99	96	91	95	0.060	0.066	0.060	0.062
Texas	El Paso	10834 Ivanhoe (Ivanhoe Fire Station)	31.785769	-106.323578	97	98	98	98	0.062	0.065	0.061	0.062
Texas	El Paso	250 Rim Rd	31.768291	-106.501260	94	98	98	97	0.070	0.072	0.071	0.071
Texas	El Paso	800 S San Marcial Street	31.765685	-106.455227	99	99	94	97	0.066	0.070	0.065	0.067
Texas	El Paso	650 R E Thomason Loop	31.746775	-106.402806	98	98	96	97	0.062	0.064	0.066	0.064
Texas	El Paso	320 Old Hueco Tanks Road	31.667500	-106.288000	99	98	98	98	0.066	0.069	0.064	0.066
Texas	El Paso	5050A Yvette Drive	31.893913	-106.425827	98	98	99	98	0.070	0.069	0.066	0.068
Texas	Galveston	9511 Avenue V 1/2	29.254474	-94.861289	96	96	97	96	0.071	0.084	0.074	0.076
Texas	Gregg	Gregg Co Airport near Longview	32.378682	-94.711811	100	99	99	99	0.066	0.068	0.065	0.066
Texas	Harris	4510 1/2 Aldine Mail Rd	29.901036	-95.326137	96	99	98	98	0.068	0.095	0.074	0.079
Texas	Harris	1405 Sheldon Road	29.802707	-95.125495	99	98	99	99	0.064	0.081	0.061	0.068
Texas	Harris	16822 Kitzman	30.039524	-95.673951	96	99	97	97	0.063	0.078	0.067	0.069
Texas	Harris	7330 1/2 North Wayside	29.828086	-95.284096	99	97	98	98	0.062	0.078	0.062	0.067
Texas	Harris	4401 1/2 Lang Rd	29.834167	-95.489167	100	99	99	99	0.064	0.091	0.069	0.074
Texas	Harris	13826 1/2 Croquet	29.623889	-95.474167	99	99	99	99	0.067	0.079	0.067	0.071
Texas	Harris	6400 Bissonnet Street	29.695729	-95.499219	97	96	98	97	0.067	0.080	0.078	0.075
Texas	Harris	9726 1/2 Monroe	29.625556	-95.267222	98	97	99	98	0.065	0.073	0.057	0.065
Texas	Harris	3333 1/2 Hwy 6 South	29.723333	-95.635833	96	99	97	97	0.070	0.079	0.079	0.076
Texas	Harris	7421 Park Place Blvd	29.686389	-95.294722	97	100	95	97	0.066	0.087	0.065	0.072
Texas	Harris	4407 Independence Parkway South	29.761653	-95.081386	98	98	97	98	0.059	0.079	0.059	0.065
Texas	Harris	8622 Garth Road Unit A	29.823319	-94.983786	85	99	96	93	0.067	0.077	0.065	0.069
Texas	Harris	1262 1/2 Mae Drive	29.767997	-95.220582	98	93	93	95	0.066	0.088	0.067	0.073
Texas	Harris	9525 1/2 Clinton Dr	29.733726	-95.257593	98	95	97	97	0.058	0.084	0.065	0.069
Texas	Harris	4514 1/2 Durant St	29.670025	-95.128508	98	97	95	97	0.063	0.077	0.062	0.067

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Texas	Harris	4522 Park Rd	29.583047	-95.015544	97	96	97	97	0.065	0.083	0.064	0.070
Texas	Harrison	Hwy 134 & Spur 449	32.668987	-94.167457	98	99	98	98	0.066	0.062	0.059	0.062
Texas	Hidalgo	2300 North Glasscock	26.226210	-98.291069	94	93	99	95	0.056	0.058	0.052	0.055
Texas	Hood	200 N Gordon Street	32.442304	-97.803529	99	99	99	99	0.073	0.073	0.063	0.069
Texas	Hunt	824 Sayle Street	33.153088	-96.115572	97	99	99	98	0.062	0.062	0.058	0.060
Texas	Jefferson	1086 Vermont Avenue	30.036422	-94.071061	94	99	96	96	0.065	0.069	0.060	0.064
Texas	Jefferson	623 Elias Street	29.897516	-93.991084	99	99	98	99	0.063	0.075	0.064	0.067
Texas	Jefferson	12552 Second St	29.863957	-94.317802	96	98	90	95	0.067	0.068	0.069	0.068
Texas	Jefferson	5200 Mechanic	29.728000	-93.894000	94	94	97	95	0.067	0.064	0.065	0.065
Texas	Jefferson	Jefferson County Airport	29.942500	-94.000556	97	99	98	98	0.062	0.065	0.059	0.062
Texas	Jefferson	Unavailable	29.867756	-93.951163	98	99	24	74	0.057	0.070	0.049	0.058
Texas	Jefferson	1800 N. 18th Street	29.978926	-94.010872	100	98	95	98	0.067	0.074	0.063	0.068
Texas	Johnson	1650 Airport Drive	32.353595	-97.436742	99	97	97	98	0.071	0.073	0.072	0.072
Texas	Kaufman	3790 S Houston St	32.564968	-96.317687	99	99	99	99	0.062	0.064	0.057	0.061
Texas	McLennan	4472 Mazanec Rd	31.653074	-97.070698	96	99	99	98	0.063	0.068	0.060	0.063
Texas	Montgomery	9472A Hwy 1484	30.350302	-95.425128	99	99	99	99	0.072	0.073	0.071	0.072
Texas	Navarro	Corsicana Airport	32.031934	-96.399141	99	98	97	98	0.060	0.064	0.060	0.061
Texas	Nueces	Corpus Christi State School (Airport Rd)	27.765340	-97.434262	96	97	97	97	0.065	0.065	0.062	0.064
Texas	Nueces	9860 La Branch	27.832409	-97.555380	97	98	92	96	0.066	0.062	0.061	0.063
Texas	Orange	2700 Austin Ave	30.085263	-93.761341	99	95	99	98	0.063	0.062	0.058	0.061
Texas	Orange	INTERSECTION OF TX HWYS 62 AND 12	30.194167	-93.866944	97	98	23	73	0.068	0.065	0.052	0.061
Texas	Parker	3033 New Authon Rd	32.868773	-97.905931	98	100	100	99	0.072	0.079	0.068	0.073
Texas	Polk	361 Tombigbee Rd, Livingston, TX 77351	30.701700	-94.674200	90	90	93	91	0.065	0.063	0.056	0.061
Texas	Randall	Palo Duro Canyon State Park, Canyon, TX 79015	34.880300	-101.664900	96	99	92	96	0.066	0.064	0.064	0.064
Texas	Rockwall	100 E Heath St	32.936523	-96.459211	98	99	96	98	0.066	0.071	0.061	0.066
Texas	Smith	14790 County Road 1145	32.344008	-95.415752	100	99	98	99	0.066	0.066	0.063	0.065
Texas	Tarrant	14290 Morris Dido Newark Rd	32.987891	-97.477175	98	99	94	97	0.073	0.078	0.067	0.072
Texas	Tarrant	3317 Ross Ave	32.805818	-97.356568	98	98	100	99	0.079	0.079	0.066	0.074
Texas	Tarrant	FAA Site off Alta Vista Road	32.922474	-97.282088	99	92	100	97	0.074	0.076	0.070	0.073
Texas	Tarrant	4100 Fairway Dr	32.984260	-97.063721	98	98	96	97	0.073	0.079	0.075	0.075
Texas	Tarrant	5504 South Collins Street	32.656357	-97.088585	98	98	99	98	0.065	0.069	0.061	0.065

State Name	County Name	Street Address	Site Latitude	Site Longitude	2014 Percent Complete	2015 Percent Complete	2016 Percent Complete	2014-2016 Average Percent Complete	2014 4th Highest Daily Max Value	2015 4th Highest Daily Max Value	2016 4th Highest Daily Max Value	Design Value 2014-2016
Texas	Travis	3724 North Hills Dr	30.354436	-97.760255	96	100	98	98	0.062	0.073	0.064	0.066
Texas	Travis	12200 Lime Creek Rd	30.483168	-97.872301	98	96	98	97	0.063	0.073	0.062	0.066
Texas	Victoria	106 Mockingbird Lane	28.836170	-97.005530	98	96	94	96	0.062	0.070	0.064	0.065
Texas	Webb	2020 Vidaurri Ave	27.517449	-99.515219	93	96	97	95	0.057	0.056	0.050	0.054
Utah	Box Elder	140 W.FISHBURN DRIVE, BRIGHAM CITY, UT	41.492707	-112.018863	86	92	98	92	0.067	0.068	0.067	0.067
Utah	Box Elder	8600 WEST 24000 NORTH PORTAGE, UTAH	41.945950	-112.233179	98	99	92	96	0.061	0.067	0.051	0.059
Utah	Carbon	On Prperty of Resident at 351 W 2500 E. Price, Utah	39.609960	-110.800749	98	99	95	97	0.064	0.069	0.067	0.066
Utah	Davis	171 WEST 1370 NORTH, BOUNTIFUL, UTAH	40.902967	-111.884467	99	54	99	84	0.074	0.073	0.076	0.074
Utah	Duchesne	290 S. 1000 W.	40.294178	-110.009732	92	50	99	80	0.062	0.060	0.081	0.067
Utah	Duchesne	6000 SOUTH AND 10000 WEST (MYTON)	40.216779	-110.182742	39	98	98	78	0.067	0.066	0.085	0.072
Utah	Salt Lake	1675 SOUTH 600 EAST, SALT LAKE CITY	40.736389	-111.872222	99	99	84	94	0.072	0.081	0.074	0.075
Utah	San Juan	CANYONLANDS NATIONAL PARK, UTAH	38.458320	-109.821260	95	99	99	98	0.064	0.065	0.064	0.064
Utah	Uintah	Dinosaur National Monument	40.437200	-109.304700	91	96	97	95	0.064	0.067	0.075	0.068
Utah	Uintah	2 Miles west of Redwash atop Deadman's Bench	40.206291	-109.353932	20	96	95	70	0.061	0.067	0.083	0.070
Utah	Uintah	2 miles south of Ouray and south of the White and Green River confluence	40.056710	-109.688108	0	98	99	66	0.079	0.068	0.096	0.081
Utah	Uintah	WHITEROCKS & COUNTY ROAD	40.483598	-109.906796	39	97	100	79	0.064	0.068	0.081	0.071
Utah	Utah	1355 NORTH 200 WEST PROVO UT	40.253611	-111.663056	90	97	97	95	0.068	0.073	0.072	0.071
Utah	Utah	312 W. 2050 NORTH, SPANISH FORK, UTAH	40.136336	-111.660502	97	94	99	97	0.076	0.071	0.072	0.073
Utah	Washington	147 N 870 W, Hurrricane, Utah	37.179125	-113.305096	95	100	99	98	0.066	0.069	0.062	0.065
Utah	Washington	ZION NATIONAL PARK, UTAH	37.198300	-113.150600	82	84	99	88	0.065	0.066	0.064	0.065
Utah	Weber	228 32ND STREET, OGDEN, UTAH	41.206321	-111.975524	100	99	98	99	0.070	0.072	0.072	0.071
Utah	Weber	425 W 2550 NORTH, OGDEN, UTAH	41.303614	-111.987871	99	94	98	97	0.070	0.074	0.073	0.072
Vermont	Bennington	120 AIRPORT RD, BENNINGTON, VERMONT	42.887590	-73.249840	95	96	97	96	0.061	0.063	0.067	0.063
Vermont	Chittenden	58 HARVEY ROAD, Underhill, Vermont	44.528390	-72.868840	94	95	99	96	0.059	0.066	0.060	0.061
Virginia	Albemarle	Lambs Lane, behind Albemarle High School Athletic Field	38.076570	-78.503970	64	100	99	88	0.059	0.059	0.062	0.060
Virginia	Arlington	S 18TH AND HAYES ST	38.857700	-77.059220	99	99	100	99	0.071	0.073	0.072	0.072
Virginia	Caroline	U.S. GEODETIC SURVEY, OFF RT.2, CORBIN	38.200870	-77.377420	86	95	85	89	0.061	0.062	0.062	0.061

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Virginia	Charles	SHIRLEY PLANTATION, ROUTE 5	37.344380	-77.259250	93	94	98	95	0.066	0.059	0.065	0.063
Virginia	Chesterfield	BEACH, INTERSECTION OF CO. ROADS 655 & 654	37.357480	-77.593550	86	88	99	91	0.061	0.063	0.063	0.062
Virginia	Fairfax	STA. 46-B9, LEE PARK, TELEGRAPH ROAD	38.773350	-77.104680	92	97	98	96	0.065	0.072	0.073	0.070
Virginia	Fauquier	RT651 C PHELPS WILDLIFE MANAGEMENT AREA	38.473670	-77.767720	98	97	99	98	0.059	0.056	0.063	0.059
Virginia	Frederick	RTE 669, BUTLER MANUF. CO NEAR REST VA	39.281020	-78.081570	99	96	99	98	0.059	0.061	0.065	0.061
Virginia	Giles	1856 Horton Ln, Newport, VA 24128	37.329700	-80.557800	98	99	93	97	0.062	0.063	0.062	0.062
Virginia	Hanover	7345 MCCLELLAN RD MECHANICSVILLE VA	37.606130	-77.218800	93	100	96	96	0.062	0.061	0.065	0.062
Virginia	Henrico	2401 HARTMAN STREET MATH & SCIENCE CTR	37.556520	-77.400270	73	100	96	90	0.062	0.064	0.066	0.064
Virginia	Loudoun	38-I, BROAD RUN HIGH SCHOOL, ASHBURN	39.024730	-77.489250	99	93	98	97	0.063	0.071	0.068	0.067
Virginia	Madison	SHENANDOAH NP BIG MEADOWS	38.523100	-78.434710	98	98	99	98	0.060	0.063	0.066	0.063
Virginia	Prince Edward	Prince Edward-Gallion State Forest, Burkeville, VA 23922	37.165500	-78.306900	91	99	95	95	0.063	0.057	0.060	0.060
Virginia	Prince William	JAMES S. LONG PARK	38.852870	-77.634620	99	99	99	99	0.062	0.067	0.067	0.065
Virginia	Roanoke	HERMAN HORN ELEMENTARY SCHOOL	37.283420	-79.884520	93	99	93	95	0.060	0.062	0.064	0.062
Virginia	Rockbridge	21-C, GLENWOOD-PEDLAR DISTRICT RANGER STA	37.626680	-79.512570	99	99	99	99	0.058	0.056	0.060	0.058
Virginia	Rockingham	VDOT RESIDENCY SHOP, 3536 NORTH VALLEY PIKE, HARRISONBURG, VA	38.477530	-78.819520	97	100	98	98	0.058	0.060	0.062	0.060
Virginia	Stafford	WIDEWATER ELEM. SCH., DEN RICH ROAD	38.481230	-77.370400	100	100	90	97	0.062	0.063	0.066	0.063
Virginia	Wythe	16-B RURAL RETREAT SEWAGE DISPOSAL	36.891170	-81.254230	99	98	97	98	0.064	0.058	0.062	0.061
Virginia	Hampton City	Adjacent to Building 1196, Wythe Landing Loop, NASA-Langley Research Center	37.103733	-76.387017	99	99	97	98	0.061	0.065	0.068	0.064
Virginia	Suffolk City	TIDEWATER COMM. COLLEGE, FREDERIC CAMPUS	36.901180	-76.438080	88	90	97	92	0.058	0.061	0.063	0.060
Virginia	Suffolk City	TIDEWATER RESEARCH STATION, HARE ROAD	36.665250	-76.730780	96	99	97	97	0.063	0.060	0.061	0.061
Washington	Clallam	LOCATED IN A TREE FARM (Cheeka Peak)	48.297860	-124.624910	75	97	96	89	0.055	0.051	0.051	0.052
Washington	Clark	1500 SE BLAIRMONT DR (Mountain View High School)	45.616667	-122.516667	99	86	92	92	0.057	0.065	0.055	0.059

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Washington	King	2000 NW SAMMAMISH RD	47.552500	-122.064722	89	94	99	94	0.052	0.059	0.054	0.055
Washington	King	42404 SE NORTH BEND WAY	47.490220	-121.772780	97	98	91	95	0.060	0.061	0.054	0.058
Washington	King	30525 SE MUD MOUNTAIN ROAD	47.141100	-121.937900	97	99	90	95	0.067	0.074	0.061	0.067
Washington	King	4103 BEACON HILL S	47.568236	-122.308628	100	90	95	95	0.044	0.048	0.046	0.046
Washington	Pierce	JACKSON VISITOR'S CENTER MT RAINIER, WA	46.784100	-121.740367	84	88	90	87	0.058	0.062	0.058	0.059
Washington	Skagit	202 O Ave	48.520590	-122.614280	92	93	98	94	0.041	0.045	0.054	0.046
Washington	Skagit	Northwest Indian College, 17113 Tallawhalt Lane	48.397787	-122.504971	93	99	99	97	0.048	0.050	0.052	0.050
Washington	Spokane	S 26010 Smith Road (Turnbull Slough National Wildlife Refuge)	47.416450	-117.529970	83	83	88	85	0.060	0.066	0.053	0.059
Washington	Spokane	E 9814 GREENBLUFF RD, GREENBLUFF	47.827280	-117.274220	92	98	99	96	0.060	0.063	0.056	0.059
Washington	Thurston	931 NORTHERN PACIFIC ROAD	46.952562	-122.595270	97	98	97	97	0.056	0.058	0.058	0.057
Washington	Whatcom	1330 LOOMIS TRAIL RD	48.950740	-122.554410	85	99	99	94	0.047	0.048	0.045	0.046
West Virginia	Berkeley	MARTINSBURG - BALL FIELD	39.448006	-77.964125	92	98	96	95	0.060	0.066	0.064	0.063
West Virginia	Cabell	HUNTINGTON - MARSHALL UNIVERSITY	38.424133	-82.425900	99	98	99	99	0.063	0.066	0.065	0.064
West Virginia	Gilmer	Cedar Creek State Park, Cedarville, WV 26611	38.879500	-80.847700	97	96	93	95	0.058	0.057	0.062	0.059
West Virginia	Greenbrier	SAM BLACK CHURCH - DOH GARAGE	37.908533	-80.632633	97	98	97	97	0.060	0.057	0.062	0.059
West Virginia	Monongalia	MORGANTOWN - AIRPORT US 119&AIRPORT BLVD.	39.649367	-79.920867	97	97	96	97	0.064	0.069	0.059	0.064
West Virginia	Ohio	WHEELING - WARWOOD WATER PLANT	40.114876	-80.700972	100	98	100	99	0.066	0.069	0.069	0.068
West Virginia	Tucker	USDA Northern Research Station, Monongahela National Forest, Parsons, WV 26287	39.090500	-79.661700	99	98	95	97	0.059	0.062	0.065	0.062
West Virginia	Wood	VIENNA - NEALE ELEM SCHOOL	39.323533	-81.552367	96	99	98	98	0.067	0.071	0.067	0.068
Wisconsin	Ashland	BAD RIVER TRIBAL SCHOOL - ODANAH	46.602000	-90.656000	95	97	96	96	0.056	0.057	0.062	0.058
Wisconsin	Brown	UW-GREEN BAY, HWYS 54 & 57	44.530980	-87.907990	97	99	98	98	0.066	0.065	0.068	0.066
Wisconsin	Columbia	N 1045 WENDT RD, COLUMBUS	43.315600	-89.108900	99	99	98	99	0.069	0.064	0.070	0.067
Wisconsin	Dane	2302 HOARD ST	43.100838	-89.357298	99	100	100	100	0.066	0.064	0.066	0.065

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Wisconsin	Dodge	Horicon Wildlife Area, 1210 N Palmatory St	43.466111	-88.621111	99	98	98	98	0.071	0.066	0.068	0.068
Wisconsin	Door	475 CTH NP	45.237000	-86.993000	98	97	99	98	0.065	0.074	0.077	0.072
Wisconsin	Eau Claire	5505 Highway 53 South Eau Claire	44.761000	-91.143000	99	95	98	97	0.061	0.059	0.064	0.061
Wisconsin	Fond du Lac	N3996 KELLY RD, TWN BYRON	43.687400	-88.422045	99	99	99	99	0.067	0.065	0.066	0.066
Wisconsin	Forest	FIRE TOWER RD, POTAWATOMI SITE	45.563000	-88.808800	93	98	93	95	0.061	0.063	0.065	0.063
Wisconsin	Jefferson	N4440 Laatsch Lane	43.003400	-88.828300	99	99	100	99	0.071	0.065	0.071	0.069
Wisconsin	Kenosha	CHIWAUKEE PRAIRIE, 11838 FIRST COURT	42.504722	-87.809300	100	99	100	100	0.076	0.075	0.080	0.077
Wisconsin	Kewaunee	KEWAUNEE, ROUTE 1, HWY 42	44.443120	-87.505240	100	100	99	100	0.065	0.070	0.072	0.069
Wisconsin	La Crosse	3550 MORMON COULEE RD	43.777500	-91.226900	96	98	99	98	0.063	0.061	0.063	0.062
Wisconsin	Manitowoc	2315 EAST GOODWIN RD	44.138619	-87.616100	98	99	99	99	0.066	0.077	0.074	0.072
Wisconsin	Marathon	LAKE DUBAY, 1780 BERGEN RD, BERGEN TNSHP	44.707350	-89.771830	90	95	98	94	0.064	0.063	0.068	0.065
Wisconsin	Milwaukee	HEALTH CENTER, 1337 SO 16TH ST	43.016667	-87.933333	100	99	96	98	0.062	0.063	0.068	0.064
Wisconsin	Milwaukee	2300 N M. L. KING JR DR	43.060975	-87.913504	98	97	99	98	0.068	0.066	0.070	0.068
Wisconsin	Milwaukee	601 E. ELLSWORTH LANE	43.181000	-87.900000	99	100	100	100	0.069	0.068	0.077	0.071
Wisconsin	Outagamie	AAL, 4432 N MEADE ST	44.307380	-88.395178	99	99	100	99	0.070	0.066	0.066	0.067
Wisconsin	Ozaukee	N. Port Washington Rd., East side of HWY32 AND I43, GRAFTON	43.343000	-87.920000	99	99	99	99	0.074	0.070	0.071	0.071
Wisconsin	Ozaukee	531 HWY D	43.498060	-87.810000	98	99	100	99	0.070	0.071	0.079	0.073
Wisconsin	Rock	1501 Ritsher St.	42.518310	-89.063470	99	100	100	100	0.072	0.064	0.072	0.069
Wisconsin	Sauk	DEVILS LAKE STATE PARK, E12886 TOWER RD	43.435100	-89.679700	99	93	99	97	0.064	0.063	0.066	0.064
Wisconsin	Sheboygan	KOHLER ANDRE PARK, 1520 Beach Park Rd.	43.679000	-87.716000	98	99	98	98	0.072	0.081	0.085	0.079
Wisconsin	Taylor	W 10746 County Highway M, Medford, WI 54451	45.206600	-90.596900	98	94	95	96	0.062	0.057	0.064	0.061
Wisconsin	Vilas	TROUT LAKE NURSERY, 10810 COUNTY HWY M	46.052000	-89.653000	91	93	96	93	0.061	0.060	0.063	0.061
Wisconsin	Walworth	2420 ELGIN CLUB RD	42.580009	-88.499046	98	100	100	99	0.073	0.067	0.072	0.070
Wisconsin	Waukesha	1310 CLEVELAND AVE	43.020075	-88.215070	98	97	100	98	0.067	0.066	0.067	0.066
Wyoming	Albany	Roosevelt National Forest, Centennial, WY 82055	41.364200	-106.239900	93	96	91	93	0.065	0.064	0.064	0.064
Wyoming	Big Horn	Basin (WARMS station)	44.280000	-108.041111	94	96	68	86	0.056	0.059	0.055	0.056
Wyoming	Campbell	THUNDER BASIN GRASSLAND SITE 35 MI N-NE GILLETTE WY	44.652200	-105.290300	98	97	99	98	0.058	0.059	0.057	0.058

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Wyoming	Campbell	SOUTH CAMPBELL COUNTY APPROX 15 MILES SSW OF GILLETTE WY (SEE APPLE BUTTE QUAD MAP)	44.146964	-105.529994	93	89	97	93	0.059	0.062	0.060	0.060
Wyoming	Carbon	ATLANTIC RIM SUNDGOG LOCATION	41.386944	-107.616667	97	81	98	92	0.062	0.059	0.060	0.060
Wyoming	Carbon	SINCLAIR-Intersection of CCR351 and N. 8th St. (SW corner of intersection)	41.783389	-107.119184	92	99	0	64	0.060	0.061	0.047	0.056
Wyoming	Converse	HWY 59	42.796372	-105.361822	98	96	98	97	0.058	0.060	0.060	0.059
Wyoming	Fremont	South Pass WyDot	42.529999	-108.720003	98	98	98	98	0.065	0.062	0.062	0.063
Wyoming	Fremont	Spring Creek Encana	43.081667	-107.549444	97	91	96	95	0.058	0.063	0.062	0.061
Wyoming	Laramie	NCore - North Cheyenne Soccer Complex	41.182227	-104.778334	89	95	94	93	0.065	0.063	0.061	0.063
Wyoming	Natrona	2800 Pheasant Drive, Casper	42.822310	-106.365010	98	99	95	97	0.061	0.060	0.061	0.060
Wyoming	Natrona	Sinclair, Casper	42.860800	-106.235860	85	96	98	93	0.058	0.058	0.056	0.057
Wyoming	Sublette	Boulder APPROX 3 MILES WEST OF BOULDER	42.719000	-109.753000	93	89	95	92	0.060	0.055	0.060	0.058
Wyoming	Sublette	DANIEL SOUTH ~ 4 MILES SO OF DANIEL WYO OFF OF HWY 189	42.790700	-110.055100	97	96	96	96	0.062	0.062	0.063	0.062
Wyoming	Sublette	Pinedale Gaseous Monitor on west side of City Park and Pine Creek	42.869824	-109.870760	94	90	96	93	0.057	0.059	0.059	0.058
Wyoming	Sublette	Big Piney Site #3	42.486361	-110.098861	96	93	99	96	0.060	0.059	0.065	0.061
Wyoming	Sublette	Juel Spring - 20 miles NW of Farson on the west side of US Highway 191	42.373499	-109.560498	98	100	97	98	0.062	0.061	0.059	0.060
Wyoming	Sublette	Skyline Dr, Pinedale, WY 82941	42.928800	-109.788000	91	87	98	92	0.062	0.065	0.064	0.063
Wyoming	Sweetwater	Hiawatha Green Site	41.158000	-108.619000	78	88	87	84	0.062	0.062	0.061	0.061
Wyoming	Sweetwater	WAMSUTTER SOUTHEAST PORTION OF SWEETWATER COUNTY	41.677453	-108.024575	99	96	95	97	0.060	0.060	0.045	0.055
Wyoming	Sweetwater	Moxa	41.750556	-109.788333	97	87	97	94	0.063	0.071	0.064	0.066
Wyoming	Teton	Grand Teton NP - Science School	43.670833	-110.599472	96	96	99	97	0.060	0.059	0.060	0.059
Wyoming	Teton	YELLOWSTONE NATIONAL PARK	44.565360	-110.400340	96	88	97	94	0.060	0.062	0.060	0.060
Wyoming	Uinta	Murphy Ridge LOCATED NEAR WYOMING UTAH BORDER	41.373097	-111.042376	96	98	98	97	0.059	0.066	0.060	0.061
Wyoming	Weston	Newcastle, WARMS station	43.873056	-104.191944	98	100	95	98	0.059	0.061	0.060	0.060
Puerto Rico	Bayamon	Ave. Central Correccional	18.420089	-66.150615	31	45	52	43	0.033	0.029	0.033	0.031
Puerto Rico	Catano	PR 165	18.440774	-66.126531	92	95	94	94	0.020	0.029	0.033	0.027
Puerto Rico	Juncos	Rd. 183	18.177939	-65.915482	94	87	83	88	0.037	0.041	0.064	0.047

Attachment 3



You are here: EPA Home > Green Book > 8-Hour Ozone (2008) Clean Data Determination/Attainment Determinations For Areas with Current Nonattainment Status

8-Hour Ozone (2008) Clean Data Determination/Attainment Determinations For Areas with Current Nonattainment Status

Data is current as of June 20, 2017
Ordered by Area Name

Click on the 'FR Page' link to view the Federal Register Notice

MODERATE

Baltimore, MD

Clean Data Determination

Proposed Approval	03/18/2015	80 FR Page 14041	
Final Approval	06/01/2015	80 FR Page 30941	Effective: 07/01/2015

Mariposa County, CA

Clean Data Determination

Proposed Approval	12/21/2016	81 FR Page 93653	
Direct Final	12/21/2016	81 FR Page 93624	Effective: 03/21/2017
Effective Date Delay to March 21, 2017	01/26/2017	82 FR Page 8499	

MARGINAL

Allentown-Bethlehem-Easton, PA

Determination of Attainment by Attainment Date

Proposed Approval	08/27/2015	80 FR Page 51992	
Final Approval	05/04/2016	81 FR Page 26697	Effective: 06/03/2016

Calaveras County, CA

Determination of Attainment by Attainment Date

Proposed Approval	08/27/2015	80 FR Page 51992	
Final Approval	05/04/2016	81 FR Page 26697	Effective: 06/03/2016

Chico (Butte County), CA

Determination of Attainment by Attainment Date

Proposed Approval	08/27/2015	80 FR Page 51992	
Final Approval	05/04/2016	81 FR Page 26697	Effective: 06/03/2016

Cincinnati, OH-KY-IN

Determination of Attainment by Attainment Date

Proposed Approval	08/27/2015	80 FR Page 51992	
Final Approval	05/04/2016	81 FR Page 26697	Effective: 06/03/2016

Dukes County, MA

Determination of Attainment by Attainment Date

Proposed Approval	08/27/2015	80 FR Page 51992	
Final Approval	05/04/2016	81 FR Page 26697	Effective: 06/03/2016

Jamestown, NY

Determination of Attainment by Attainment Date

Proposed Approval	08/27/2015	80 FR Page 51992	
Final Approval	05/04/2016	81 FR Page 26697	Effective: 06/03/2016

Lancaster, PA

Determination of Attainment by Attainment Date

Proposed Approval	08/27/2015	80 FR Page 51992	
Final Approval	05/04/2016	81 FR Page 26697	Effective: 06/03/2016

Philadelphia-Wilmington-Atlantic City, PA-NJ-MD-DE

Both Clean Data Determination and Determination of Attainment by Attainment Date

Proposed Approval	04/18/2017	82 FR Page 18268	
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Pittsburgh-Beaver Valley, PA

Determination of Attainment by Attainment Date

Proposed Approval	08/25/2016	81 FR Page 58435	
Final Approval	12/06/2016	81 FR Page 87819	Effective: 01/05/2017

Reading, PA

Determination of Attainment by Attainment Date

Proposed Approval	08/27/2015	80 FR Page 51992	
Final Approval	05/04/2016	81 FR Page 26697	Effective: 06/03/2016

San Francisco Bay Area, CA

Determination of Attainment by Attainment Date

Proposed Approval	08/27/2015	80 FR Page 51992	
Final Approval	05/04/2016	81 FR Page 26697	Effective: 06/03/2016

San Luis Obispo (Eastern San Luis Obispo), CA

Determination of Attainment by Attainment Date

Proposed Approval	10/12/2016	81 FR Page 70382	
Final Approval	12/21/2016	81 FR Page 93620	Effective: 01/20/2017

Seaford, DE

Determination of Attainment by Attainment Date

Proposed Approval	08/27/2015	80 FR Page 51992	
Final Approval	05/04/2016	81 FR Page 26697	Effective: 06/03/2016

St. Louis-St. Charles-Farmington, MO-IL

Determination of Attainment by Attainment Date

Proposed Approval	06/27/2016	81 FR Page 41497	
Direct Final	06/27/2016	81 FR Page 41444	Effective: 08/26/2016

Tuscan Buttes, CA

Determination of Attainment by Attainment Date

Proposed Approval	08/27/2015	80 FR Page 51992	
Final Approval	05/04/2016	81 FR Page 26697	Effective: 06/03/2016

Upper Green River Basin Area, WY

Determination of Attainment by Attainment Date

Proposed Approval	08/27/2015	80 FR Page 51992	
Final Approval	05/04/2016	81 FR Page 26697	Effective: 06/03/2016

Washington, DC-MD-VA

Both Clean Data Determination and Determination of Attainment by Attainment Date

Proposed Approval	04/25/2017	82 FR Page 19011	
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Attachment 4



You are here: EPA Home > Green Book > 8-Hour Ozone (2008) Designated Area Area/State/County Report

8-Hour Ozone (2008) Designated Area Area/State/County Report

Data is current as of June 20, 2017

Allentown-Bethlehem-Easton, PA (Marginal - Nonattainment)

PENNSYLVANIA (Region III)

Carbon Co

Lehigh Co

Northampton Co

Atlanta, GA (Moderate - Maintenance)

GEORGIA (Region IV)

Bartow Co

Cherokee Co

Clayton Co

Cobb Co

Coweta Co

De Kalb Co

Douglas Co

Fayette Co

Forsyth Co

Fulton Co

Gwinnett Co

Henry Co

Newton Co

Paulding Co

Rockdale Co

Baltimore, MD (Moderate - Nonattainment)

MARYLAND (Region III)

Anne Arundel Co

Baltimore (City)

Baltimore Co

Carroll Co

Harford Co

Howard Co

Baton Rouge, LA (Marginal - Maintenance)

LOUISIANA (Region VI)

Ascension Par

East Baton Rouge Par

Iberville Par

Livingston Par

West Baton Rouge Par

Calaveras County, CA (Marginal - Nonattainment)

CALIFORNIA (Region IX)

Calaveras Co

Charlotte-Rock Hill, NC-SC (Marginal - Maintenance)

Cabarrus Co (P)
 Gaston Co (P)
 Iredell Co (P)
 Lincoln Co (P)
 Mecklenburg Co
 Rowan Co (P)
 Union Co (P)
 SOUTH CAROLINA (Region IV)
 York Co (P)

Chicago-Naperville, IL-IN-WI (Moderate - Nonattainment)

ILLINOIS (Region V)
 Cook Co
 Du Page Co
 Grundy Co (P)
 Kane Co
 Kendall Co (P)
 Lake Co
 Mc Henry Co
 Will Co
 INDIANA (Region V)
 Lake Co
 Porter Co
 WISCONSIN (Region V)
 Kenosha Co (P)

Chico (Butte County), CA (Marginal - Nonattainment)

CALIFORNIA (Region IX)
 Butte Co

Cincinnati-Hamilton, OH-KY-IN (KY portion) (Marginal - Nonattainment)

KENTUCKY (Region IV)
 Boone Co (P)
 Campbell Co (P)
 Kenton Co (P)

Cincinnati-Hamilton, OH-KY-IN (OH and IN portions) (Marginal - Maintenance)

INDIANA (Region V)
 Dearborn Co (P)

Lawrenceburg Township

OHIO (Region V)
 Butler Co
 Clermont Co
 Clinton Co
 Hamilton Co
 Warren Co

Cleveland-Akron-Lorain, OH (Marginal - Maintenance)

OHIO (Region V)
 Ashtabula Co
 Cuyahoga Co
 Geauga Co
 Lake Co
 Lorain Co
 Medina Co
 Portage Co
 Summit Co

Columbus, OH (Marginal - Maintenance)

OHIO (Region V)

Delaware Co
Fairfield Co
Franklin Co
Knox Co
Licking Co
Madison Co

Dallas-Fort Worth, TX (Moderate - Nonattainment)

TEXAS (Region VI)

Collin Co
Dallas Co
Denton Co
Ellis Co
Johnson Co
Kaufman Co
Parker Co
Rockwall Co
Tarrant Co
Wise Co

Denver-Boulder-Greeley-Ft. Collins-Loveland, CO (Moderate - Nonattainment)

COLORADO (Region VIII)

Adams Co
Arapahoe Co
Boulder Co
Broomfield Co
Denver Co
Douglas Co
Jefferson Co
Larimer Co (P)
Weld Co (P)

Dukes County, MA (Marginal - Nonattainment)

MASSACHUSETTS (Region I)

Dukes Co

Greater Connecticut, CT (Moderate - Nonattainment)

CONNECTICUT (Region I)

Hartford Co
Litchfield Co
New London Co
Tolland Co
Windham Co

Houston-Galveston-Brazoria, TX (Moderate - Nonattainment)

TEXAS (Region VI)

Brazoria Co
Chambers Co
Fort Bend Co
Galveston Co
Harris Co
Liberty Co
Montgomery Co
Waller Co

Imperial County, CA (Moderate - Nonattainment)

CALIFORNIA (Region IX)

Jamestown, NY (Marginal - Nonattainment)
NEW YORK (Region II)
Chautauqua Co

Kern Co (Eastern Kern), CA (Moderate - Nonattainment)
CALIFORNIA (Region IX)
Kern Co (P)

Knoxville, TN (Marginal - Maintenance)
TENNESSEE (Region IV)
Anderson Co (P)
Blount Co
Knox Co

Lancaster, PA (Marginal - Nonattainment)
PENNSYLVANIA (Region III)
Lancaster Co

Los Angeles-San Bernardino Counties (West Mojave Desert), CA (Severe 15
- Nonattainment)
CALIFORNIA (Region IX)
Los Angeles Co (P)
San Bernardino Co (P)

Los Angeles-South Coast Air Basin, CA (Extreme - Nonattainment)
CALIFORNIA (Region IX)
Los Angeles Co (P)
Orange Co
Riverside Co (P)
San Bernardino Co (P)

Mariposa County, CA (Moderate - Nonattainment)
CALIFORNIA (Region IX)
Mariposa Co

Memphis, TN-MS-AR (Marginal - Maintenance)
ARKANSAS (Region VI)
Crittenden Co
MISSISSIPPI (Region IV)
De Soto Co (P)

Portion along MPO Lines

TENNESSEE (Region IV)
Shelby Co

Morongo Band of Mission Indians (Serious - Nonattainment)
CALIFORNIA (Region IX)
Riverside Co (P)

Nevada Co. (Western part), CA (Moderate - Nonattainment)
CALIFORNIA (Region IX)
Nevada Co (P)

New York-N. New Jersey-Long Island, NY-NJ-CT (Moderate -
Nonattainment)
CONNECTICUT (Region I)
Fairfield Co
Middlesex Co

New Haven Co
NEW JERSEY (Region II)

Bergen Co
Essex Co
Hudson Co
Hunterdon Co
Middlesex Co
Monmouth Co
Morris Co
Passaic Co
Somerset Co
Sussex Co
Union Co
Warren Co

NEW YORK (Region II)

Bronx Co
Kings Co
Nassau Co
New York Co
Queens Co
Richmond Co
Rockland Co
Suffolk Co
Westchester Co

Pechanga Band of Luiseno Mission Indians of the Pechanga Reservation
(Moderate - Nonattainment)

CALIFORNIA (Region IX)

Riverside Co (P)
San Diego Co (P)

Philadelphia-Wilmington-Atlantic City, PA-NJ-MD-DE (Marginal - Nonattainment)

DELAWARE (Region III)

New Castle Co

MARYLAND (Region III)

Cecil Co

NEW JERSEY (Region II)

Atlantic Co
Burlington Co
Camden Co
Cape May Co
Cumberland Co
Gloucester Co
Mercer Co
Ocean Co
Salem Co

PENNSYLVANIA (Region III)

Bucks Co
Chester Co
Delaware Co
Montgomery Co
Philadelphia Co

Phoenix-Mesa, AZ (Moderate - Nonattainment)

ARIZONA (Region IX)

Maricopa Co (P)
Pinal Co (P)

Pittsburgh-Beaver Valley, PA (Marginal - Nonattainment)

PENNSYLVANIA (Region III)

Allegheny Co
Armstrong Co
Beaver Co
Butler Co
Fayette Co
Washington Co
Westmoreland Co

Reading, PA (Marginal - Nonattainment)
PENNSYLVANIA (Region III)
Berks Co

Riverside Co, (Coachella Valley), CA (Severe 15 - Nonattainment)
CALIFORNIA (Region IX)
Riverside Co (P)

Sacramento Metro, CA (Severe 15 - Nonattainment)
CALIFORNIA (Region IX)
El Dorado Co (P)
Placer Co (P)
Sacramento Co
Solano Co (P)
Sutter Co (P)
Yolo Co

San Diego County, CA (Moderate - Nonattainment)
CALIFORNIA (Region IX)
San Diego Co (P)

San Francisco Bay Area, CA (Marginal - Nonattainment)
CALIFORNIA (Region IX)
Alameda Co
Contra Costa Co
Marin Co
Napa Co
San Francisco Co
San Mateo Co
Santa Clara Co
Solano Co (P)
Sonoma Co (P)

San Joaquin Valley, CA (Extreme - Nonattainment)
CALIFORNIA (Region IX)
Fresno Co
Kern Co (P)
Kings Co
Madera Co
Merced Co
San Joaquin Co
Stanislaus Co
Tulare Co

San Luis Obispo (Eastern San Luis Obispo), CA (Marginal - Nonattainment)
CALIFORNIA (Region IX)
San Luis Obispo Co (P)

Seaford, DE (Marginal - Nonattainment)
DELAWARE (Region III)
Sussex Co

Sheboygan County, WI (Moderate - Nonattainment)

St. Louis-St. Charles-Farmington, MO-IL (Marginal - Nonattainment)

ILLINOIS (Region V)

Madison Co

Monroe Co

St Clair Co

MISSOURI (Region VII)

Franklin Co

Jefferson Co

St Charles Co

St Louis

St Louis Co

Tuscan Buttes, CA (Marginal - Nonattainment)

CALIFORNIA (Region IX)

Tehama Co (P)

Upper Green River Basin Area, WY (Marginal - Nonattainment)

WYOMING (Region VIII)

Lincoln Co (P)

Sublette Co

Sweetwater Co (P)

Ventura County, CA (Serious - Nonattainment)

CALIFORNIA (Region IX)

Ventura Co (P)

Washington, DC-MD-VA (Marginal - Nonattainment)

DISTRICT OF COLUMBIA (Region III)

Entire District

MARYLAND (Region III)

Calvert Co

Charles Co

Frederick Co

Montgomery Co

Prince George's Co

VIRGINIA (Region III)

Alexandria

Arlington Co

Fairfax

Fairfax Co

Falls Church

Loudoun Co

Manassas

Manassas Park

Prince William Co

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Attachment 5



You are here: EPA Home > Green Book > 8-Hour Ozone (2008) Nonattainment Area Area/State/County Report

8-Hour Ozone (2008) Nonattainment Area Area/State/County Report

Data is current as of June 20, 2017

Allentown-Bethlehem-Easton, PA (Marginal - Nonattainment)

PENNSYLVANIA (Region III)

Carbon Co

Lehigh Co

Northampton Co

Baltimore, MD (Moderate - Nonattainment)

MARYLAND (Region III)

Anne Arundel Co

Baltimore (City)

Baltimore Co

Carroll Co

Harford Co

Howard Co

Calaveras County, CA (Marginal - Nonattainment)

CALIFORNIA (Region IX)

Calaveras Co

Chicago-Naperville, IL-IN-WI (Moderate - Nonattainment)

ILLINOIS (Region V)

Cook Co

Du Page Co

Grundy Co (P)

Kane Co

Kendall Co (P)

Lake Co

Mc Henry Co

Will Co

INDIANA (Region V)

Lake Co

Porter Co

WISCONSIN (Region V)

Kenosha Co (P)

Chico (Butte County), CA (Marginal - Nonattainment)

CALIFORNIA (Region IX)

Butte Co

Cincinnati-Hamilton, OH-KY-IN (KY portion) (Marginal - Nonattainment)

KENTUCKY (Region IV)

Boone Co (P)

Campbell Co (P)

Kenton Co (P)

Dallas-Fort Worth, TX (Moderate - Nonattainment)

Collin Co
Dallas Co
Denton Co
Ellis Co
Johnson Co
Kaufman Co
Parker Co
Rockwall Co
Tarrant Co
Wise Co

Denver-Boulder-Greeley-Ft. Collins-Loveland, CO (Moderate - Nonattainment)

COLORADO (Region VIII)

Adams Co
Arapahoe Co
Boulder Co
Broomfield Co
Denver Co
Douglas Co
Jefferson Co
Larimer Co (P)
Weld Co (P)

Dukes County, MA (Marginal - Nonattainment)

MASSACHUSETTS (Region I)

Dukes Co

Greater Connecticut, CT (Moderate - Nonattainment)

CONNECTICUT (Region I)

Hartford Co
Litchfield Co
New London Co
Tolland Co
Windham Co

Houston-Galveston-Brazoria, TX (Moderate - Nonattainment)

TEXAS (Region VI)

Brazoria Co
Chambers Co
Fort Bend Co
Galveston Co
Harris Co
Liberty Co
Montgomery Co
Waller Co

Imperial County, CA (Moderate - Nonattainment)

CALIFORNIA (Region IX)

Imperial Co

Jamestown, NY (Marginal - Nonattainment)

NEW YORK (Region II)

Chautauqua Co

Kern Co (Eastern Kern), CA (Moderate - Nonattainment)

CALIFORNIA (Region IX)

Kern Co (P)

Lancaster, PA (Marginal - Nonattainment)

Los Angeles-San Bernardino Counties (West Mojave Desert), CA (Severe 15 - Nonattainment)

CALIFORNIA (Region IX)
 Los Angeles Co (P)
 San Bernardino Co (P)

Los Angeles-South Coast Air Basin, CA (Extreme - Nonattainment)

CALIFORNIA (Region IX)
 Los Angeles Co (P)
 Orange Co
 Riverside Co (P)
 San Bernardino Co (P)

Mariposa County, CA (Moderate - Nonattainment)

CALIFORNIA (Region IX)
 Mariposa Co

Morongo Band of Mission Indians (Serious - Nonattainment)

CALIFORNIA (Region IX)
 Riverside Co (P)

Nevada Co. (Western part), CA (Moderate - Nonattainment)

CALIFORNIA (Region IX)
 Nevada Co (P)

New York-N. New Jersey-Long Island, NY-NJ-CT (Moderate - Nonattainment)

CONNECTICUT (Region I)
 Fairfield Co
 Middlesex Co
 New Haven Co

NEW JERSEY (Region II)
 Bergen Co
 Essex Co
 Hudson Co
 Hunterdon Co
 Middlesex Co
 Monmouth Co
 Morris Co
 Passaic Co
 Somerset Co
 Sussex Co
 Union Co
 Warren Co

NEW YORK (Region II)
 Bronx Co
 Kings Co
 Nassau Co
 New York Co
 Queens Co
 Richmond Co
 Rockland Co
 Suffolk Co
 Westchester Co

Pechanga Band of Luiseno Mission Indians of the Pechanga Reservation (Moderate - Nonattainment)

CALIFORNIA (Region IX)

Philadelphia-Wilmington-Atlantic City, PA-NJ-MD-DE (Marginal - Nonattainment)

DELAWARE (Region III)

New Castle Co

MARYLAND (Region III)

Cecil Co

NEW JERSEY (Region II)

Atlantic Co

Burlington Co

Camden Co

Cape May Co

Cumberland Co

Gloucester Co

Mercer Co

Ocean Co

Salem Co

PENNSYLVANIA (Region III)

Bucks Co

Chester Co

Delaware Co

Montgomery Co

Philadelphia Co

Phoenix-Mesa, AZ (Moderate - Nonattainment)

ARIZONA (Region IX)

Maricopa Co (P)

Pinal Co (P)

Pittsburgh-Beaver Valley, PA (Marginal - Nonattainment)

PENNSYLVANIA (Region III)

Allegheny Co

Armstrong Co

Beaver Co

Butler Co

Fayette Co

Washington Co

Westmoreland Co

Reading, PA (Marginal - Nonattainment)

PENNSYLVANIA (Region III)

Berks Co

Riverside Co, (Coachella Valley), CA (Severe 15 - Nonattainment)

CALIFORNIA (Region IX)

Riverside Co (P)

Sacramento Metro, CA (Severe 15 - Nonattainment)

CALIFORNIA (Region IX)

El Dorado Co (P)

Placer Co (P)

Sacramento Co

Solano Co (P)

Sutter Co (P)

Yolo Co

San Diego County, CA (Moderate - Nonattainment)

CALIFORNIA (Region IX)

San Diego Co (P)

San Francisco Bay Area, CA (Marginal - Nonattainment)

CALIFORNIA (Region IX)

Alameda Co
Contra Costa Co
Marin Co
Napa Co
San Francisco Co
San Mateo Co
Santa Clara Co
Solano Co (P)
Sonoma Co (P)

San Joaquin Valley, CA (Extreme - Nonattainment)

CALIFORNIA (Region IX)

Fresno Co
Kern Co (P)
Kings Co
Madera Co
Merced Co
San Joaquin Co
Stanislaus Co
Tulare Co

San Luis Obispo (Eastern San Luis Obispo), CA (Marginal - Nonattainment)

CALIFORNIA (Region IX)

San Luis Obispo Co (P)

Seaford, DE (Marginal - Nonattainment)

DELAWARE (Region III)

Sussex Co

Sheboygan County, WI (Moderate - Nonattainment)

WISCONSIN (Region V)

Sheboygan Co

St. Louis-St. Charles-Farmington, MO-IL (Marginal - Nonattainment)

ILLINOIS (Region V)

Madison Co
Monroe Co
St Clair Co

MISSOURI (Region VII)

Franklin Co
Jefferson Co
St Charles Co
St Louis
St Louis Co

Tuscan Buttes, CA (Marginal - Nonattainment)

CALIFORNIA (Region IX)

Tehama Co (P)

Upper Green River Basin Area, WY (Marginal - Nonattainment)

WYOMING (Region VIII)

Lincoln Co (P)
Sublette Co
Sweetwater Co (P)

Ventura County, CA (Serious - Nonattainment)

CALIFORNIA (Region IX)

Ventura Co (P)

Washington, DC-MD-VA (Marginal - Nonattainment)

DISTRICT OF COLUMBIA (Region III)

Entire District

MARYLAND (Region III)

Calvert Co

Charles Co

Frederick Co

Montgomery Co

Prince George's Co

VIRGINIA (Region III)

Alexandria

Arlington Co

Fairfax

Fairfax Co

Falls Church

Loudoun Co

Manassas

Manassas Park

Prince William Co

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Exhibit 24

DECLARATION OF WILLIAM JOSEPH (JOEL) BROCK, JR.

1. My name is Joel Brock. I have continuously been a Sierra Club member for the last 6 months and have been a member on and off for the last three years. I do video work for the Georgia Chapter of Sierra Club; for example, I did a video shoot of a gathering with Sierra Club members where the mayor of Atlanta spoke about climate change.

2. I am 28 years old and live with my sister and her four year old daughter in Winston, Georgia, which is in Douglas County, in the greater Atlanta area.

3. When I was in the fourth grade, my family moved from a more rural area to the more polluted metropolitan Atlanta area, and by high school I had developed asthma.

4. I like to go for walks at least five days a week; I enjoy going to local parks or walking around the neighborhood. I especially enjoy Fairplay Park, which is in the next town over, in the same county. I also like to play basketball at Fairplay Park a couple times a month. I often like to go to the Beltline in Fulton County, which is a bike/walk path that used to be an old railroad line. I attend outdoor sporting events, like Atlanta Braves games, about once every couple months. I intend to continue these activities in the future.

5. Every day I take preventative medications regardless of the weather, but on bad air quality days I have to carry a "rescue inhaler" that I take as needed. On these poor air quality days, I have to limit my outdoor exposure completely, and I do not engage in outdoor activities that I would otherwise like to do. On such days, when I breathe it feels like trying to breathe with a towel or a shirt over my mouth, a feeling that feeling lasts all day long. On these days, I use a breathing treatment machine to avoid a more severe wheezing attack where it feels like I can't catch my breath, as if I just sprinted the fastest sprint I've ever done.

6. I have a Weather Channel app on my phone that tells me the air quality for the day. I check it on a daily basis or have a family member warn me when pollution levels are high so I know when I need to remain inside. I notice that we get more air quality alerts in the summer, and also on particularly hot days.

7. My niece who lives with me has asthma, too.

8. I am aware that ozone is the main component of smog, and that we get more of it on hot, sunny days. From information EPA publishes, I am aware that ozone can harm people of all ages, including those who are active outdoors, is especially dangerous for people with asthma, like me, and can cause health problems including chest pain, coughing, inflammation, and even reduced lung function. See:

www.epa.gov/ozone-pollution/ozone-basics.

9. I am concerned about my exposure to ozone pollution, especially because of my asthma. I strongly feel that the greater Atlanta area would be more appealing without ozone smog pollution.

10. I support the Sierra Club's efforts to ensure that EPA does its job to designate attainment and non-attainment areas so that states must clean up areas that are most polluted. Any delay in cleaning up ozone pollution puts my health at risk and keeps me from doing outdoor activities that I enjoy.

I declare under penalty of perjury that the foregoing is true and correct.

Executed on: July 6th, 2017


William Joseph (Joel) Brock, Jr.

Exhibit 25

DECLARATION OF JEFF CARTER

1. My name is Jeff Carter. I am the Executive Director of Physicians for Social Responsibility (PSR). I am an attorney by training and have worked in the nonprofit field for many years; I have held my current position at PSR since January of this year. I am responsible for the overall management and operation of the organization. In that capacity, I am familiar with the organization's structure, function, purpose, and membership.
2. Physicians for Social Responsibility is a nonprofit organization based in Washington, DC and with chapters in multiple states across the country. Our mission is to protect human life from the gravest threats to health and survival; we number environmental pollution among those threats.
3. As part of our efforts to protect against environmental pollution, we have repeatedly pressed for strong, timely implemented protections against ozone air pollution. When EPA released its proposed rule to strengthen ground-level ozone regulations in November 2014, we filed detailed written comments, presented in-person testimony at EPA hearings, and mobilized our members to submit individual comments pressing EPA to adopt the scientific and health-based standard of 60 parts per billion ("ppb"). After the ozone standards were finalized in 2015, we intervened in legal challenges that sought to weaken them, *Murray Energy Corp. v. EPA*, No. 15-1385 (D.C. Cir. motion to intervene filed Nov. 24, 2015), to help defend the public health improvements that will result from their implementation. We later filed comments with EPA on its proposed rule to implement the 2015 ozone standard. EPA-HQ-OAR-2016-0202-0118. Those comments urge EPA to implement the 2015 standards to ensure swift, timely attainment of them.
4. PSR has approximately 36,900 members nationwide. Many of these members are medical, health care, and public health professionals. Our membership includes the following numbers of members in the following cities:


Louisville, KY	42
Detroit, MI	24
Las Vegas, NV	79
San Antonio, TX	91
El Paso, TX	24
Salt Lake City, UT	71
Milwaukee, WI	65
Atlanta, GA	103
Cincinnati, OH	67
Columbus, OH	73

Oakland, CA	159
Baltimore, MD	137
St. Louis, MO	108
Cleveland, OH	69
Los Angeles, CA	394
Philadelphia, PA	249
New York, NY	917
Chicago, IL	325
Washington, DC	216

5. PSR members living in the above-named and other areas have a strong interest in full, timely, and effective implementation of the Clean Air Act requirements designed to protect them from unhealthy levels of pollution in the air they breathe. By delaying initial air designations under the 2015 ozone standards, EPA delays implementation of those standards and denies these and other PSR members the protections guaranteed under the Clean Air Act. Such members are harmed by the denial of these important health and welfare protections.

I declare under penalty of perjury that the foregoing is true and correct.

Executed on June 30, 2017.



Jeff Carter

Exhibit 26

DECLARATION OF CECIL CORBIN-MARK

I, Cecil Corbin-Mark, declare as follows:

1. I am Deputy Director and Director of Policy Initiatives at West Harlem Environmental Action, Inc., which commonly goes by the name WE ACT for Environmental Justice. I have served in that role for about 11 years, and have worked at WE ACT since 1994. In my current capacity, I am responsible for and participate in management and operation of the organization, as well as for strategies and efforts to achieve our organizational mission. In that capacity, I am required to be familiar with the organization's structure, function, purpose, and membership.

2. WE ACT for Environmental Justice is incorporated in New York, with its headquarters in New York, NY. It is recognized as a not-for-profit corporation under section 501(c)(3) of the United States Internal Revenue Code. WE ACT for Environmental Justice is a Northern Manhattan community-based membership organization whose mission is to build healthy communities by assuring that people of color and/or those with low-income participate meaningfully in the creation of sound and fair environmental health and protection policies and practices. As a result of its ongoing work to educate and mobilize the more than 630,000 residents of Northern Manhattan on environmental issues affecting their quality of life, WE ACT for Environmental Justice has become a leader in the nationwide movement for environmental justice, influencing the creation of federal, state and local policies affecting the environment. It also has an office in Washington, DC, to assist with its work on federal environmental policy.

3. Air pollution is an important issue for the health and wellbeing of WE ACT's members and the Northern Manhattan community, which is overburdened by health and pollution challenges, like asthma. WE ACT participated in the comment process for the 2015

ozone standard, urging EPA to set a protective health standard, explaining that “it is extremely important that we use all of the regulatory and non-regulatory mechanisms to protect the health of some of our most overburdened, sensitive populations.” *See* Comment of WE ACT for Environmental Justice at 1, EPA-HQ-OAR-2008-0699-2252. It also organized a mass comment campaign in the comment process for the 2015 ozone standard, where over a hundred individuals sent postcards to EPA calling for EPA to fulfill its promise to “keep environmental justice a priority.” EPA-HQ-OAR-2008-0699-2578. WE ACT also participated in the comment process for EPA’s proposed rule to implement the 2015 ozone standards, urging EPA to have control measures with the greatest degree of public health protection be prioritized in implementation and to ensure public involvement in ozone control planning.

4. WE ACT has over 400 members, most of whom live, work, and recreate in Upper Manhattan. Timely and full implementation of the 2015 ozone standard will help drive greater pollution reductions that will benefit WE ACT’s members. Delayed implementation allows harmful pollution to remain for more time than it otherwise would, and thus harms WE ACT’s members.

I declare under penalty of perjury that the foregoing is true and correct.

Executed on June 30, 2017.



Cecil Corbin-Mark

Exhibit 27

DECLARATION OF DR. ELENA CRAFT

I, Dr. Elena Craft, declare:

1. I am a Senior Scientist at the Environmental Defense Fund (“EDF”), a non-profit organization focused on protecting human health and the environment from airborne contaminants by using sound science. I received a Ph.D. in toxicology from Duke University’s Nicholas School of the Environment and Earth and Ocean Sciences. I also have a Master of Science degree in toxicology from North Carolina State University. This declaration is submitted in support of EDF’s petition for review of Environmental Protection Agency’s (“EPA”) decision to extend by one year the initial area designation deadline for the implementation of the 2015 national ambient air quality standards for ozone (“extension”), and in support of EDF’s motion for vacatur or a stay of this action.

2. As a Senior Scientist in Texas, I conduct outreach and help research, formulate, and implement measures to reduce air pollution that leads to ozone formation, particularly emissions from the oil and gas industry, fossil fuel-fired power plants, and other industrial air pollution emissions. I also work with government, civic, business, and other groups at the local level, and EDF staff across the nation to design solutions to air quality challenges. I have provided expert testimony at two House Congressional hearings related to issues of air quality, and ozone specifically. I have served on various advisory committees to

the EPA, including the Mobile Source Technical Review Subcommittee (“MSTRS”) under the Clean Air Act Advisory Committee (“CAAAC”); the Air, Climate, and Energy Subcommittee of the Board of Scientific Counselors; an Environmental Justice Technical Review Subcommittee; and a ports work group.

The 2015 Ozone Standards

3. In 2015, EPA strengthened the national health-based standard for ozone, lowering the standard’s level from 75 parts per billion (“ppb”) to 70 ppb. *National Ambient Air Quality Standards for Ozone*, 80 Fed. Reg. 65,292 (Oct. 26, 2015) (“2015 ozone standards”). The revision of the ozone standards was based on EPA’s recognition that exposure to ground-level ozone has significant health impacts at lower concentrations than EPA’s previous standard and that a revised standard was necessary to protect the public health with an adequate margin of safety. *Id.* at 65,294 (“[T]he Administrator concludes that the current primary [ozone] standard is not requisite to protect public health with an adequate margin of safety, as required by the [Clean Air Act], and that revision of the level to 0.070 ppm is warranted to provide the appropriate degree of increased public health protection for at-risk populations against an array of adverse health effects.”)

4. The record for the 2015 rulemaking, however, along with subsequent scientific studies, demonstrates that health effects can occur at much lower levels, especially in sensitive populations. For that reason, EPA’s independent scientific

advisors recommended that the agency establish the standard's level between 60-70 ppb, with the caution that a level of 70 ppb would provide little margin of safety for the protection of public health, particularly for sensitive subpopulations.

Similarly, health and medical associations called on EPA to set a more protective standard than the 70 ppb one EPA selected. 80 Fed. Reg. at 65,321-23; 65,355.

5. In its Regulatory Impact Analysis ("RIA") for the 2015 standards, EPA estimated that the standards would result in 96 to 160 ozone-related premature deaths avoided, 220 to 550 fine particulate matter ("PM_{2.5}") related premature deaths avoided, 630 asthma emergency department visits avoided, and 160,000 missed school days avoided among children age 5-17 in 2025, a year EPA selected based on the fact that it anticipated all of the country besides California to attain the standards by then. U.S. EPA, *Regulatory Impact Analysis for the final Revisions to the National Ambient Air Quality Standards for Ground-Level Ozone* ("RIA"), Table ES 6, pp. ES-16, ES-2 to -3 (Sept. 2015),

<https://www3.epa.gov/ttn/naaqs/standards/ozone/data/20151001ria.pdf>. Other expected health benefits include 230,000 cases of asthma exacerbation avoided among children age 6-18, 340 cases of acute bronchitis avoided among children age 8-12, and 11,000 upper and lower respiratory symptoms avoided among children age 7-14. *Id.* at Table ES-6, p. ES-16. These expected health benefits are

likely understated because they do not include the health benefits that would be realized in California. *Id.* at ES-3 to -4.

6. In estimating these benefits, EPA assumed that all areas would be designated by late 2017, as required by the Clean Air Act. *See* RIA at ES-3. A postponement of designation will result in continued ground-level ozone pollution and delays in developing and implementing abatement measures to reduce ozone. Such postponement will reduce, or delay many of the benefits of reduced ozone pollution expected to result from implementation of the 2015 ozone standards.

7. As demonstrated by daily ozone air quality monitoring readings, many areas of the country experience unhealthy levels of air quality on a frequent basis. These unhealthy levels of ozone air quality can result in acute respiratory illness and other damaging health outcomes so severe that people are hospitalized, as described below.

Exposure to Ground-Level Ozone Harms Human Health

8. A longstanding body of scientific research, including numerous EPA assessments, demonstrates that exposure to ozone harms human health. EPA concluded that “a very large amount of evidence spanning several decades supports a relationship between exposure to [ozone] and a broad range of respiratory effects.” *2013 Final Report: Integrated Science Assessment of Ozone and Related Photochemical Oxidants* (“ISA”) (EPA/600/R-10/076F) at 1-6. These effects

range from decreases in lung function and increases in respiratory-related hospital admissions and emergency room visits, to premature death from ozone exposure.

Id. at 6-131 to 6-158, 6-162 to -163.

9. EPA's most recent Integrated Science Assessment for Ozone concluded that there is a causal relationship or likely causal relationship between both short-term ozone exposure, and long-term ozone exposure and a broad range of harmful respiratory and cardiovascular effects in humans. ISA at 1-5-1-8, Table 1-1. Short-term exposure is defined as hours, days, or weeks, and long-term exposure is measured in months to years. *Id.* at 1-4.

10. Short-term exposure to ozone can have critical health implications. For instance, there is strong evidence of an association between out-of-hospital cardiac arrests and short-term exposure to ozone, as reported in Ensor, et al., 2013. Katherine B. Ensor, *et al.*, *A Case-Crossover Analysis of Out-of-Hospital Cardiac Arrest and Air Pollution*, 127 CIRCULATION 1192 (2013), <https://www.ncbi.nlm.nih.gov/pubmed/23406673>. Time scales of exposure up to three hours in duration and also at the daily level on the day of the event were significant. This evidence augments the long-standing body of literature demonstrating the serious impacts from short-term exposure to ozone pollution, including the increased risk of premature death. ISA at 1-14 (concluding that there is "likely to be a causal relationship between short-term exposures to [ozone] and

total mortality.”). EPA recognized that positive associations have been reported between “short-term [ozone] exposures and respiratory mortality, particularly during the summer months.” 80 Fed. Reg. 65,307; *See* ISA 6-220 to 6-221. In addition, there is likely to be a causal relationship between short-term ozone exposure and non-accidental and cardiopulmonary-related mortality. *ISA* at 6-222.

11. EPA has also found that the scientific evidence for respiratory effects from long-term ozone exposure had strengthened since the last review. 80 Fed. Reg. 65,307. EPA now concludes that there is “likely to be a causal relationship between long-term exposure to [ozone] and respiratory effects.” *ISA* at 1-8. Similarly, EPA notes that “recent evidence is suggestive of a causal relationship between long-term [ozone] exposures and total mortality.” *Id.* Some longitudinal studies have further demonstrated that “long-term [ozone] exposure influences the risk of asthma development in children.” *ISA* at 7-2.

12. Ozone pollution is particularly harmful for vulnerable populations, such as children, people with respiratory diseases or asthma, older adults, and people who are active outdoors, especially outdoor workers. *ISA* at 1-8. Children with asthma also face heightened risks from ozone exposure. Many studies have demonstrated that children with asthma experience decrements in lung function and increases in respiratory symptoms when exposed to ozone pollution. K.

Mortimer et al., *The Effect of Air Pollution on Inner-City Children with Asthma*, 19 EUR. RESPIRATORY J. 699 (2002), *ISA*, 6-120–21, 6-160.

13. A recent study of almost 61 million Medicare patients conducted nationwide indicates a significant association between ozone exposure and all-cause mortality, with effects strongest in minorities and those of low socioeconomic status. These effects were seen at ozone concentrations well below the current standard of 70 ppb. Di et al., *Air Pollution and Mortality in the Medicare Population*, NEW ENGLAND J. OF MEDICINE (June 29, 2017).

A Delay in Initial Area Designations Will Harm People's Health

14. EPA's delay in designating nonattainment areas will allow additional emissions of smog-forming pollutants in these areas already burdened with unhealthy levels of ozone pollution. Delaying designation will cause many areas to remain legally not classified as nonattainment areas, despite having ambient ground-level ozone levels that violate EPA's 2015 ozone standard. This delay will lead to a longer period of inaction before measures to abate health-harming ozone are undertaken in these heavily impacted areas. The delay in abatement measures will ultimately result in delayed attainment and more exposure to ground-level ozone. Both the delay in implementing pollution control measures and in ultimately attaining the standards expand the risk of near-term harm to all populations, and especially harm to children, older adults, those suffering from

respiratory diseases such as asthma, low income populations, outdoor workers, and others recreating outdoors. The result will be more asthma attacks, hospitalizations, emergency room visits, and premature deaths in those areas. *See* RIA at ES-16 Table ES-6.

Conclusion

15. Administrator Pruitt's decision to delay designation of areas under the 2015 ozone standards will result in additional harm from exposure to unhealthy levels of ozone. Individuals exposed to this pollution face a higher risk of adverse health effects, including acute and immediate respiratory ailments like asthma and enhanced risk of longer term, deleterious health effects associated with ground-level ozone pollution exposure.

I declare that the foregoing is true and correct.



Elena Craft, PhD

Dated July 10, 2017

Exhibit 28

DECLARATION OF STEPHEN C. CRANE, Ph.D., MPH

MARC MOSS, MD
President

POLLY E. PARSONS, MD
President-Elect

DAVID GOZAL, MD, MBA
Immediate Past President

JAMES M. BECK, MD
Vice President

JUAN C. CELEDON, MD, DrPH
Secretary-Treasurer

STEPHEN C. CRANE, PhD, MPH
Executive Director

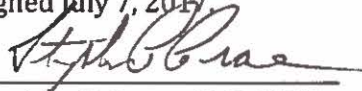
1. I am Executive Director of the American Thoracic Society (ATS). I am responsible for management and strategic direction for the organization and as such, I have direct knowledge of and am directly involved in the organization's structure, function, mission and membership.
2. The American Thoracic Society is a medical professional organization with over 16,000 members dedicated to the prevention, detection, treatment, cure and research of respiratory disease, critical care illness and sleep disordered breathing. The ATS seeks to achieve this mission through research, education, and advocacy. The ATS is a 501(c)(3) organization chartered and headquartered in New York State. The ATS has members in all 50 U.S. states and over 100 countries around the globe.
3. As part of our mission to support research, the ATS publishes 3 peer-reviewed academic journals – the American Journal of Respiratory and Critical Care Medicine, the American Journal of Respiratory Cell and Molecular Biology, and the Annals of the American Thoracic Society – that features cutting edge research articles on all topics impacting pulmonary, critical care and sleep medicine. These journals frequently include publications that document the adverse health effects associated with exposure to air pollution.
4. In addition to our journals, that ATS publishes clinical practice guidelines and policy statements regarding a wide range of topics impacting respiratory health, including the health effects of air pollution. The ATS published the statement: **What Constitutes an Adverse Health Effect of Air Pollution (1999)**. This document has been frequently relied on by EPA as part of its justification for rule-making on standards for various criteria air pollutants.
5. Many members of the ATS are thought leaders in research that documents the adverse health effects of air pollution. Their scholarship has added to the scientific community's understanding of the adverse health effects associated with exposure to air pollution, including adverse health effects that occur at levels below current EPA standards.



6. Multiple lines of research, including animal models, chamber studies, epidemiological studies and observational studies have documented the adverse health effects associated with exposure to ozone. Adverse health effects associated with ozone exposure include reduced lung function, missed school and work days, increased asthma exacerbations, increase asthma medication use, increased asthma hospitalizations, increased COPD exacerbations, increased hospitalizations and premature mortality. These adverse health effects have been consistently documented in both U.S and international studies.
7. To protect the American public, including vulnerable populations from the harms of ozone air pollution, the ATS has consistently urged EPA to establish a National Ambient Air Quality Standard for ozone at 60 ppb/8-hours.
8. Many ATS members live in areas of the U.S. that meet the 2008 EPA ozone standard of 75 ppb/8-hours, but fail to meet the 2015 revised standard of 70 ppb/8-hours. Further, many ATS members treat patients with lung disease who work and/or reside in U.S. communities that meet the 2008 standard but fail to meet the 2015 standards.
9. Both the ATS members who reside in these areas and the patients that they treat who work and/or reside in these areas would materially benefit from swift implementation of the 2015 EPA National Ambient Air Quality Standard for ozone. Conversely, delayed implementation of the 2015 EPA ozone standard of 70 ppb/8-hours means ATS members and the patients whom they serve will face additional exposure to ozone pollution that causes significant adverse health effects including asthma attacks, missed school days, missed work days, days of restricted outdoor activity, increased medication use, increased emergency room visits, increased hospitalization and increased mortality.
10. Failure of the EPA to promptly implement the 2015 National Ambient Air Quality Standard for ozone denies our ATS members and the patients they serve well documented health benefits and exposes them to significant and severe adverse health effects.

I declare under penalty of perjury that the foregoing is true and correct.

Signed July 7, 2017.



Stephen C. Crane, PhD, MPH
Executive Director
American Thoracic Society



Exhibit 29

DECLARATION OF TRENT A. DOUGHERTY

I, Trent A. Dougherty, declare and state as follows:

1. This declaration is based on my personal knowledge. I am over the age of eighteen (18) and suffer no legal incapacity. I submit this declaration in support of the petition of The Ohio Environmental Council (“The OEC”) challenging U.S. Environmental Protection Agency (“EPA”) Administrator E. Scott Pruitt’s decision to postpone the implementation of the 2015 National Ambient Air Quality Standards (“NAAQS”) for ozone.

2. I am The OEC’s General Counsel. I have served in this position since 2015; before that I was Managing Director of Legal Affairs. I have worked as an attorney for The OEC for over 12 years. My current position at The OEC requires me to be aware of The OEC’s goals and mission, and up-to-date on the organization’s program activities, including regulatory and litigation efforts. In my capacity as General Counsel, I have access to current information about The OEC’s membership.

3. The OEC is a nonprofit corporation organized and existing under the laws of the State of Ohio. Our mission is to secure healthy air, land, and water for all who call Ohio home. Working to reduce Ohioans’ exposure to harmful air pollution, including ground-level ozone, is a core part of that mission. Using legislative initiatives, legal

action, scientific principles, and statewide partnerships, we secure a healthier environment for Ohio's families and communities.

4. As a result of my work at The OEC, I am familiar with EPA's efforts to regularly evaluate, update and implement the ozone NAAQS. In particular, I am aware that in 2015 EPA strengthened the ozone NAAQS by tightening the maximum level of ozone allowed in the ambient air to 70 parts per billion ("ppb"), down from the 75 ppb concentration allowed under the previous standards which were issued in 2008. EPA's decision was based in part on studies examining health outcomes for real people, linking ozone levels allowed by the 2008 standards to hospital visits and increased risk of death. EPA's independent science advisors unanimously recommended strengthening the ozone standards in 2015 because the 2008 standards were not protective enough of public health.

5. Based on my review of The OEC's membership, I can declare that the organization has over 2,866 individual members. Of that number, 925 live in the Ohio counties of Butler (47), Franklin (568), Geauga (62), Hamilton (187), Lake (36), or Warren (25) counties. These counties are currently maintaining compliance with the 2008 ozone NAAQS, but I understand have ozone measurements that would likely violate the 2015 ozone NAAQS.

6. As a result of my work at The OEC, I know that ground-level ozone smog irritates the respiratory system, reduces lung function, inflames and damages the cell that

line the lungs, making them more susceptible to infections, asthma and aggravated asthma, and aggravates chronic lung disease. Exposure to ozone smog can cause permanent lung damage.

7. In addition to my professional role, I have been a member of The OEC for 13 years. I am 39 years old. My family and I are residents of Columbus, Franklin County, Ohio, and have been for over 10 years.

8. I have asthma. I enjoy outdoor activities, including jogging, hiking, and kayaking, but I sometimes have to curtail these activities in order to avoid shortness of breath, wheezing, and sometimes even chest pain. This is particularly the case when ozone levels are high, but sometimes also occurs on days when the 2008 standard is met. At times, these symptoms are so severe that I must take inhaler medication, and at least once in the past few years I had to visit an urgent care facility for asthma related breathing problems. Lower ambient ozone levels, as will be the case where I live when the 2015 ozone standards are fully implemented, will benefit me directly. For the same reason, failure to implement the 2015 standard in a timely way will prolong my exposure to unhealthy levels of ozone, and cause me physical hardship.

9. My 10-year old daughter lives with me in Franklin County. She also has mild asthma, and must curtail her outdoor activities when ozone levels are at or above the concentrations permitted by the 2008 ozone standard. On bad air days it is especially difficult for her to breathe when we are out on our family hikes or simply walking our

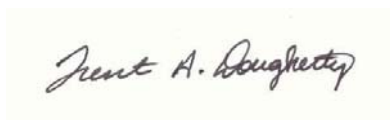
dogs around the neighborhood. It causes her discomfort and sadness to have to stop playing outdoors when the air is bad, and in turn it pains me to know that she is uncomfortable. I am anxious and concerned that she will have difficulty breathing on bad air days.

10. As a result of my work with The OEC, I am aware that EPA Administrator E. Scott Pruitt has announced his decision to delay for a year the implementation of the more health-protective 2015 ozone standards. That action, if left in place, will delay for a year or more the achievement of cleaner air in urban areas like Franklin County.

11. The OEC supports timely implementation of the 2015 ozone standards, and opposes any attempt by EPA or Administrator Pruitt to postpone their full implementation, or otherwise delay or weaken them. I also support full and timely implementation of the 2015 ozone standards as a member of The OEC, because delay means prolonged exposure to unhealthy levels of ozone for myself and my daughter.

I declare under the penalty of perjury that the foregoing is true and correct to the best of my knowledge and belief.

Executed this 7th day of July, 2017

A handwritten signature in black ink that reads "Trent A. Dougherty". The signature is written in a cursive style and is centered within a light yellow rectangular box.

Trent A. Dougherty, Esq.

Exhibit 30

DECLARATION OF BARBARA EINZIG

1. My name is Barbara Einzig. I am a member of the Sierra Club, having joined in November of 2016.

2. I am 66 years old and have lived in northern Baltimore, Maryland in the county of Baltimore City, since 2005. I work as a writer and grant specialist, consulting with healthcare programs and organizations.

3. As a result of living through the September 11, 2001 terrorist attacks in New York (I lived in the building closest to the World Trade Center), I suffer from asthma and reactive airway dysfunction which affects both my upper and lower respiratory tracts and makes me particularly sensitive to air pollution. I came to Baltimore after my doctor recommended that I move out of New York because the building I had lived in was still contaminated with World Trade Center dust, and the high ozone levels downtown were exacerbating my respiratory problems.

4. I'd hoped that my move to Baltimore would allow me to spend more time outside, but I didn't realize until living here how bad the air quality actually is. When ozone levels are high and I try to go outside, I get sinus pain, chest pain, phlegm, and have coughing fits so bad that people always ask me if I'm ok. It feels like trying to suck in air through a straw, but you can't quite get all the air in; it's exhausting and makes me feel extremely worn out at the end of the day. And sometimes a bad pollution reaction episode can trigger the onset of a virus or bacteria-based illness, like the flu, which would not occur if my bronchial tract were not already inflamed. I have to visit the doctor regularly for respiratory-related health care.

5. I can always tell when ozone pollution is high, because I have an increased sensitivity to smell, which is actually a common trait of people who have asthma and who suffered acute chemical exposures from events like 9/11. It smells to me like an oil tanker.

However, I don't have to rely on my own senses; air quality alerts are publicly available and I live my life by them. I cannot go out at all on days classified as "red" or "orange," and I limit my outdoor activities on "yellow" days. Ozone pollution is worse in the summer, which often makes me sick, so I primarily limit my outdoor activities to the spring and fall. I have noted that even during the winter months, we sometimes have suddenly high ozone levels. Following prolonged bouts of chronic bronchitis, my doctor suggested if possible I leave Baltimore at the times when there are the highest levels of ozone and particulate matter, so I try to travel to visit friends in more rural areas of the country, particularly during the summer months. If I can't control where I'm going to be, I use a rescue inhaler, which opens up my airways.

6. I really wish ozone pollution levels did not keep me from doing outdoor activities in the summer, because I love bicycling, hiking, and swimming. I would do rowing if I could; there's an excellent rowing club in Baltimore. However, with current ozone pollution, I cannot.

7. Even simple social activities become difficult for me because of the damper ozone pollution puts on my life. I can't make long term plans; for example, I don't buy season tickets to outdoor events. If my friends want to share a meal outside at a café or have a picnic, I have to wait and see what the air pollution alerts say. The Baltimore Museum of Art has a wonderful "Jazz in the Sculpture Garden" series – but I can only go if it's a good ozone day. There are many other great outdoor festivals in Baltimore, but I can't usually take part in them because they're given during the summer. If ozone pollution were not a constant problem for me, I could plan social events with more stability and certainty and could attend many events that I cannot now attend.

8. The interaction of my medical condition and ozone pollution has also limited my earning power by necessitating, in many instances, the ability to work from home.

9. I was really excited when I found out that EPA changed the ozone standard in 2015, because I think it might make a big difference for me and improve my health and quality of life. Environmental regulations affect people far more than they realize. I really feel that this is something that's very important.

10. I am aware that, as someone above the age of 65 with persistent respiratory conditions, I am especially vulnerable to ozone pollution. I hate the idea of going to the emergency room, so I am very proactive about my health. But any delay in cleaning up ozone pollution puts my health at risk, and denies me the ability to spend time outdoors without having serious health consequences. And I am not the only one; others affected by 9/11 or similar disasters, the elderly, young children, and people with asthma are all particularly sensitive to ozone pollution and need EPA to do its job. This is particularly important for the many people in Baltimore who live in older buildings in disrepair, where the indoor air pollution they must put up with adds to their vulnerability.

11. I am providing this statement to support Sierra Club's work to compel EPA to carry out its duties under the Clean Air Act to protect all Americans from ozone pollution, particularly those of us for whom ozone has enormous quality-of-life impacts.

I declare under penalty of perjury that the foregoing is true and correct.

Executed on: June 29, 2017



Exhibit 31

DECLARATION OF PAT ELKA

I, Pat Elka, hereby declare as follows:

1. I am currently a member of the Natural Resources Defense Council (“NRDC”). I have been a member for eight years.
2. I live in Mariposa, California. I have lived at my current address for almost twelve years, and in Mariposa County for the same length of time.
3. I have been diagnosed with COPD – chronic obstructive pulmonary disease. I have an underlying disease, myopathy, which causes my muscles to become weak – which impedes me from breathing properly, and causes restriction of my airways.
4. I like to enjoy the outdoors – I garden, take short hikes and walks, and try to see outdoor music events and dine outside in my area.
5. In the wintertime, because where I live is the county seat for Yosemite National Park, people are allowed to burn brush all winter long. From the first rain to the last part of May when they aren’t allowed to anymore, people burn their tree trimmings. During this time of year, the air quality gets really bad, to the point where I don’t want to go outside about nine months of the year.
6. I keep track of the air mostly by just looking out the window – it often looks murky for most of the autumn and winter months, particularly just ahead of rainstorms. It seems as if there are almost no restrictions on controlled burns. Due to my location in a valley, it takes any kind of air pollution a long time to clear out.

7. I am also concerned about smog pollution. Since smog is something you can't even see, it is hard for me to protect myself. I remember from when I was a teen, hearing that Mariposa County had worse smog than the San Joaquin valley because of all the engines idling waiting to get into Yosemite National Park.

8. I'm aware of the risks to the heart, lungs, and the inflammatory response that air pollution can have on the body. I'm aware that inhaling elevated concentrations of particulate matter and ozone pollution can cause immediate injury to a person, as well as increase that person's risk of developing serious and even life-threatening illnesses. Since I already have respiratory problems, I am particularly concerned about these impacts. For these reasons, I am very concerned about the health threat posed to my health and the health of my community by particulate pollution and ozone pollution.

9. I believe that regulating particulate pollution and ozone pollution will help reduce the amount of air pollution to which I am exposed. I am concerned that if the U.S. EPA weakens air quality standards or rules enforcing these standards, that there will be a negative impact on my health and the health of my community. I support litigation by NRDC to ensure that requirements of the Clean Air Act for controlling air pollution like particulate pollution and ozone pollution are fully and expeditiously implemented so as to protect my health.

I declare that the foregoing is true and correct to the best of my knowledge, information, and belief. Executed in Mariposa, California on July 6, 2017.

 7/6/17
Pat Elka

Exhibit 32

DECLARATION OF HUDA FASHHO

1. I am the Associate Director of Member Care for the Sierra Club, a non-profit corporation organized under the laws of the State of California. I work in Sierra Club's national office in Oakland, California. I became Member Services Manager in 2011, and became Associate Director of Member Care in 2016. In that capacity, I am responsible for planning, developing, and directing the programs and Club staff responsible for: providing information services to members, the operational and user aspects of the Club's member/donor database, the delivery of member/donor acknowledgments and membership renewals. My work requires that I be familiar with the Club's purpose, organization, structure and activities, and with environmental interests and concerns of Club members. My work also requires me to be familiar with the nature and scope of the Club's membership programs, its membership records, and the manner in which information on members can be retrieved.

2. The Sierra Club is a nonprofit corporation existing under the laws of California, with its principal place of business in San Francisco, California. The Club is a membership organization dedicated to exploring, enjoying, and protecting the wild places of the earth, and to protecting and restoring the quality of the natural and human environment. The Club's actions to protect and enhance the environment include advocacy and litigation to strengthen and enforce environmental laws and regulations. Club members are greatly concerned about air quality, and the Club has a long history of activities at both the local and national levels to protect air quality, often working closely with our members to provide them with services and information that are helpful to them locally.

3. The Club regularly maintains membership records that include the address of each member. These records are regularly updated each business day to add new members, reflect address changes, and change membership status for those who are no longer active members. The records are maintained on a computer database, from which I obtained the information provided below.

4. Sierra Club currently has 814,097 individual members, residing in all 50 states and Puerto Rico.

5. As of June 28, 2017, the Club has the following number of members living in the following counties:

- a. Pinal, AZ: 483
- b. Yuma, AZ: 98
- c. Alameda, CA: 11,828
- d. Amador, CA: 238

- e. Los Angeles, CA: 35,640
- f. Tuolumne, CA: 498
- g. Washington, DC: 3,156
- h. DeKalb, GA: 2,257
- i. Douglas, GA: 65
- j. Gwinnett, GA: 755
- k. Fulton, GA: 2,058
- l. Jefferson, KY: 1,799
- m. East Baton Rouge, LA: 444
- n. Baltimore, MD: 2,336
- o. Macomb, MI: 1,175
- p. Wayne, MI: 2,420
- q. St. Louis, MO: 3,657
- r. New York, NY: 8,264
- s. Clark, NV: 2,439
- t. Franklin, OH: 3,129
- u. Hamilton, OH: 2,620
- v. Lake, OH: 490
- w. Warren, OH: 353
- x. Bexar, TX: 1,918
- y. El Paso, TX: 550
- z. Davis, UT: 205
- aa. Salt Lake, UT: 2,889
- bb. Weber, UT: 375
- cc. Milwaukee, WI: 2,432

I declare under penalty of perjury that the foregoing is true and correct to the best of my knowledge and belief.

Executed this 29th day of June, 2017.


Huda Fashho

Exhibit 33

DECLARATION OF THOM GLICK

I, Thom Glick, state that:

1. I live in Columbus, Ohio. I have lived at my current address for nine years, and in Franklin County for seventeen years.
2. I have been a member of the Natural Resources Defense Council since 2016.
3. I am a freelance illustrator and adjunct college faculty member. I enjoy working outside when I can. I live in a pedestrian-friendly part of Columbus, so I often walk or bicycle to the grocery, to meet with clients, and run other errands. For my health and fitness, I bicycle, climb, hike, and garden, and typically spend more than eight hours per week on these activities. I spend about five hours or more a week walking our dogs. I spend time outside working on home-improvement projects, as well, which can keep me outside for several hours at a time, for days at a time.
4. My partner and I have seasonal allergies and sensitive respiratory systems. Because of this and because we spend a lot of time outside, we pay close attention to air quality advisories. We follow the advisories whenever possible. For example, I have had to alter my schedule to exercise and work on outdoor projects early in the morning to avoid being outside when air quality is at its worst, which has resulted in disrupting other activities, compromising productivity, and spending less time outside. When I cannot alter my schedule or shorten my time outside, during these poor air quality days, the exposure has left me feeling unwell (headaches, scratchy throat, sore lungs). Having to adjust my schedule and behavior to follow these air advisories negatively impacts my work productivity and fitness goals.
5. I am concerned about environmental regulations not being enforced fully. I am afraid that declining air quality will force my partner and me to stay indoors more often and limit or

TG

eliminate our ability to work and enjoy being outside. I worry that declining air quality will make it more difficult for us to garden our own food, require us to spend more money on indoor air quality and require us to spend more time and money on indoor fitness options. I worry that these effects will negatively impact our finances and health. Additionally, I worry that declining air quality will negatively impact the future of my community and the value of my property; if people are spending less time outside, the value of living in a pedestrian-friendly community will decrease.

6. I support litigation by NRDC to ensure that requirements of the Clean Air Act for controlling air pollution like particulate pollution and ozone pollution are fully and expeditiously implemented so as to protect my health.

I declare that the foregoing is true and correct to the best of my knowledge, information, and belief. Executed in Columbus, Ohio on June 27, 2017.


Thom Glick

Exhibit 34

DECLARATION OF KRYSTAL HENAGAN

I, Krystal Henagan, declare as follows:

1. I am an active member of Environmental Defense Fund (EDF). I am also a field organizer and consultant for Moms Clean Air Force, a special project of EDF that works to unite moms and dads to protect our children's health from the harmful effects of air pollution. I am also a member of Sierra Club.

2. I currently reside in Helotes, Texas with my partner, Treg Addison, and our son, [REDACTED]. We have lived in our current home since May of 2015, and have lived in various other locations in Bexar County, outside San Antonio, for the last 4 years.

3. I joined EDF and began working with Moms Clean Air Force to help restore the promise of clean air for my son and future generations of Texans. As a seventh-generation Texan, I am deeply concerned about the worsening air quality in the San Antonio area and in the state of Texas more broadly. I have watched as industrial facilities, including facilities like the Alamo cement plant that was permitted to expand in June and growing natural gas operations in the nearby Eagle Ford Shale, have contributed to deteriorating air quality in my region. I helped launch Moms Clean Air Force Texas in September 2015 because of my deep concerns about the poor air quality in San Antonio and other parts of my home state. Moms Clean Air Force Texas has now grown to nearly 40,000 members.

My work with Moms Clean Air Force has been especially focused on collaborating with local, regional, and federal agencies and representatives to help create a plan to address the severe air pollution problems we face here in San Antonio.

4. As part of my work as a consultant for Moms Clean Air Force, I am aware that the U.S. Environmental Protection Agency (EPA) recently revised the primary health-based ozone standard to 70 parts per billion (ppb). I am also aware that the San Antonio metro region experiences unhealthy levels of ozone pollution, including days with air quality far worse than the EPA national standard. For example, on June 8, 2017, San Antonio recorded an 8-hour ozone average of 89 ppb, which represents the highest 8-hour average recorded in the state of Texas this year. *See* Tex. Comm'n on Env'tl. Quality, *High Ozone in Your Metro Area*, https://www.tceq.texas.gov/cgi-bin/compliance/monops/ozone_summary.pl.

Already, the Texas state government says on its website that San Antonio would violate the national standard for the 2015-2017 period, even though we are just six months into 2017. *See* Tex. Comm'n on Env'tl. Quality, *Compliance with Eight-Hour Ozone Standard*, https://www.tceq.texas.gov/cgi-bin/compliance/monops/8hr_attainment.pl (giving "Current Three-Year Average" at two monitoring sites in Bexar County as above 70 ppb).

5. I experience the consequences of our poor air quality and high ozone levels firsthand. When my family first moved to the San Antonio region, my son [REDACTED]

was 4 years old. Due in part to the air quality here, ██████'s asthma became life threatening after we arrived. ██████ developed severe respiratory problems and, at just four years old, ██████ was on seven different medications to manage his asthma and other respiratory conditions.

6. While his health has improved with treatment, ██████ still suffers from severe asthma, and our local air quality and ozone levels have serious impacts on his daily quality of life. ██████ enjoys spending time outside, from riding his bike, jumping on his trampoline, playing basketball, swinging, looking for fossils, to hunting and fishing. However, we have to limit the amount of, or often restrict altogether, time he gets to enjoy such activities outdoors when the air quality is poor. When the air quality is very poor, his activities are completely restricted to indoors. When he has to stay inside our home during poor air quality days, he often gets very frustrated. I don't blame him; he is an active child and gets much more joy spending time outdoors where he is free and can move around than being confined to the house. As a family, our favorite thing to do together is bass fish on our boat. There have been times we have had to cancel family fishing outings to Medina Lake, a 17-mile drive from our home, because poor air quality in the San Antonio region could trigger ██████'s asthma and respiratory sensitivities.

7. Now eight years old and in elementary school, [REDACTED] has missed countless school days due to poor air quality exacerbating his asthma. In just the last school year, I would estimate that he missed more than 10 days of school due to breathing difficulties. When he is unable to attend school or leave the house because of the air quality, I stay at home and care for him.

8. As the air quality problems in San Antonio continue to worsen, I constantly worry that [REDACTED] could experience an asthma attack or that I would need to rush him to the hospital due to his inability to breathe.

9. As a parent with a child with asthma, I have had many sleepless nights managing his breathing difficulties and have had to call his doctor's office for an emergency appointment as soon as they open their office phone lines in the morning. One recent example is when [REDACTED] woke up coughing in the early morning hours of Monday April 10th, 2017. I've learned from his doctor that when he coughs and grunts, it's because his asthma is flaring. I quickly began administering a breathing treatment in the middle of the night; the effects from albuterol usually end up keeping him awake, and this time was no different. As soon as his pediatrician's office opened at 8 a.m., I called and scheduled an asthma appointment for him. The doctor evaluated [REDACTED] and heard he was still wheezing from asthma and administered an albuterol breathing treatment. He also had developed a respiratory infection and was given antibiotics.

10. ██████ missed school during this particular episode, and I missed a City of San Antonio workshop I was selected to attend because of the work that I and Moms Clean Air Force contributes to the San Antonio community.

11. In addition to my concerns for his health on these poor air quality days, I worry about the effects these frequent disruptions will have on his education.

12. I also worry that ██████ may experience long-term and life-long health impacts from breathing polluted air at a time when his lungs were still developing. He developed 12-14 mucus plugs in his lungs shortly after moving to San Antonio in 2013. Because this happened at such young age, when his lungs were still developing, I worry that his lungs may never fully recover from this damage at such a critical time in his development and that breathing polluted air has hindered his normal lung function more permanently.

13. ██████ currently takes four medications, and the costs associated with his ongoing treatments are significant for my family. For instance, thus far in 2017, he has visited his pediatrician and specialists 12 times.

14. In spite of the severe air quality problems we currently experience in San Antonio, I am aware that San Antonio is not designated as a nonattainment area under the 2008 ozone standards. However, I am aware that Texas submitted a recommendation to EPA that Bexar County be designated as in nonattainment with the 2015 ozone standards. I am also aware that EPA has recently decided to delay

by one year the initial area designations that would begin the process of implementation for the 2015 ozone standards. If the San Antonio area were designated as an area in nonattainment with the ozone standards, as the state recommended, I understand from information I learned through my work with Moms Clean Air Force that the state would be required to develop and implement a plan to reduce ozone-causing emissions, which would provide vital safeguards to public health in our region. Similarly, I understand that large new sources of ozone-forming air pollution would also be required to better control their emissions of those chemicals.

15. I am also aware that Governor Abbott recently vetoed a 1.46 million dollar funding package that would have helped to address San Antonio's ozone problems on the grounds that state funding "should be prioritized to directly address problems in our *non-attainment* areas of the state." Michael Locklear, *Governor Cuts Nearly \$1.5 Million for Air Quality Planning in San Antonio*, NEWS 4 SAN ANTONIO (June 13, 2017) available at <http://news4sanantonio.com/news/local/governor-cuts-nearly-15-million-for-air-quality-planning-in-san-antonio> (emphasis added). By delaying designations, EPA deprives us of protections we need because state officials have not acted to clean up our air quality.

16. EPA's extension of its deadline to designate areas under the 2015 health-based ozone standards delays the implementation of much-needed pollution reductions in the San Antonio region, which is currently exceeding those standards. The air pollution here has already severely affected us. EPA's delay allows the ongoing harms to my son's health to continue longer and to worsen. He is growing up now, and I don't want him to grow up with more lung damage.

I declare that the foregoing is true and correct.



Krystal Henagan

Dated July 11, 2017

Exhibit 35

DECLARATION OF MARVIN KODISH

1. I have been a member of National Parks Conservation Association since February 26, 2009.
2. I live in Sagamore Hills, Ohio, about two miles from the Ohio Canal Tow Path of the Cuyahoga Valley National Park. My wife Marjorie and I purchased our home here in 1996 in part due to its proximity to the Cuyahoga Valley National Park.
3. I spend a good deal of time outdoors near our home. We frequently have lunch and dinner outside on our deck during the warmer months. We have a yard with a variety of plants and trees which we regularly water and tend. We also put out feed for birds, since my wife and I like to watch the birds in our area. I recognize that the health and diversity of plant and animal life as a unique attribute of the region.
4. I have been coming to the Cuyahoga Valley National Park and other national parks for over fifty years. I regularly hike and bike in the Cuyahoga Valley National Park in the warmer months, and I intend to continue hiking, biking and enjoying the scenery of Cuyahoga Valley National Park regularly. I appreciate the aesthetic and recreational opportunities the park affords me as well as the healthy lifestyle it supports for me, my family and community.
5. I recognize and value Cuyahoga Valley National Park for protecting our unique regional ecosystem, including air, water, plant and wildlife resources.
6. I am very concerned about the impact of air pollution on the Cuyahoga Valley National Park and the plants and trees in our yard at home. I am also concerned about the impact of air pollution on my health as well as that of my wife.
7. I have a persistent cough and my wife is asthmatic. I am concerned that the prevalence of air pollution in our area, and ozone pollution in particular, may contribute to our respiratory ailments.
8. I am aware that in 2015 the Environmental Protection Agency lowered the amount of ozone pollution allowed nationwide. I believe that enforcing this standard would have a particular benefit on my area, which has persistently had a problem with elevated ozone levels.
9. I understand that power plants, vehicles and other industrial sources release pollution that contributes to ozone pollution in our area. I also understand that ozone pollution can harm human health and also have a negative impact on plant life.
10. I am concerned that ozone pollution is one of the factors doing harm to plant life. Nature is very important to me. I care a lot about biodiversity and like to see a variety of plants

and animals thriving in the Cuyahoga Valley National Park and other parks around the country. I also like to see the plants in my yard thriving. I am concerned that harm to trees and plants will also harm the wildlife in these ecosystems, especially the birds and smaller mammals, by damaging their habitat. When I visit the Cuyahoga Valley National Park and other national parks, the whole panorama of the experience is important to me – the air quality, the animals and vegetation that I see, and the beautiful scenery. Noticing poor air quality, sometimes appearing as smoggy skies, or seeing sick vegetation diminishes the enjoyment of my visits to the Cuyahoga Valley National Park and to other parks that are suffering as well from ozone pollution.

11. I am definitely concerned that my children and grandchildren will not be able to fully enjoy the Cuyahoga Valley National Park and other national parks and have the same wonderful experiences I've had if we do not solve our pollution problems, in particular ozone.
12. I am aware, however, that EPA has delayed making air quality designations for the new standard. I am concerned that delays in implementing the new ozone standard threaten my health, my family's health, and the health of the outdoor environment we enjoy. When ozone pollution is reduced, I believe the air quality in my area would improve and the related negative effects to which it contributes as mentioned above would be minimized. I believe that reducing ozone pollution in my area would improve air quality and be beneficial for the health of my community and the environment. The longer it takes for that to happen, the longer the harms ozone pollution causes me and my family will persist.
13. I fully support National Parks Conservation Association in their efforts to see the Clean Air Act implemented with a national ozone standard that fully and timely protects the health and ecosystem of the national parks I visit.
14. For the good of my health and that of family as well as the area in which we live and recreate, including Cuyahoga Valley National Park, it is important to me that ozone pollution be reduced as quickly as possible.

I declare under penalty of perjury that the foregoing is true and correct.

Executed on June 27, 2017.



Marvin Kodish

Exhibit 36

DECLARATION OF SARAH M. LYON, MD

1. My name is Sarah M. Lyon and I am a physician specializing in pulmonary and critical care medicine in Philadelphia, Pennsylvania. I received my medical degree from Boston University Medical School in 2004, and I completed my residency in internal medicine in 2007 and my fellowship training in pulmonary and critical care medicine in 2011 at the University of Pennsylvania. I care for patients with respiratory illnesses and those with medical critical illness.

2. I have been a member of the American Thoracic Society (“ATS”) since 2009. I have been a member of ATS’s Health Policy Committee for three years, the last two of which as Vice Chair. I was previously a member of the Health Equality and Diversity Committee for two years, and was the inaugural recipient of the ATS Health Equality Fellowship Award in 2015.

3. I live with my husband and three children, ages 7 months, 3 years, and 5 years, in Philadelphia. I have lived in Philadelphia since 2004. We are a family of outdoor enthusiasts, and we spend time outside frequently in the warmer months. For example, approximately three days a week we enjoy biking or running along the Schuylkill River trail in the city, hike in Fairmount Park approximately once a month and we tend our community garden plot about twice a week. Exercising outdoors is both a recreational activity for our family as well as an important commuting modality. My husband regularly walks about 1 mile to work. I bike or run to work (a 2 – 2.5 mile trip) a couple of days a week. Our oldest daughter recently started to ride her bike to school. My family plans to continue our outdoor activities well into the future, because when the air quality is good, such activities provide substantial benefits to physical as well as emotional and intellectual health.

4. As a pulmonary and critical care physician, I am aware that ozone harms the human respiratory tract and that ozone exacerbates conditions such as asthma and chronic obstructive pulmonary disease (“COPD”).

5. I am very concerned about the ways that ozone pollution harms my patients. Many of my patients suffer from asthma and COPD. My patients also include those with interstitial lung diseases such as sarcoidosis and pulmonary fibrosis. Many of my patients say that they have difficulty breathing during bad air quality days, and I advise them to stay indoors and to use air conditioners to reduce exposure to outside air and help control their symptoms. However many of my patients have limited financial resources, and the higher energy costs associated with air conditioning are financially burdensome to them. In some cases my patients forego air conditioning due to high energy cost, resorting to opening their windows, thereby increasing their exposure to poor air quality outside, which worsens their respiratory conditions. On bad air quality days, I also find that some of my patients miss their appointments because they cannot leave the house without their symptoms flaring up.

6. I also have concerns for my own health and that of my family due to exposure to unhealthy levels of ozone pollution. I have had asthma since childhood, and have to use my rescue inhaler more often on high ozone days. On such days, I refrain from outdoor activities when possible, and I also limit my children’s outdoor activities because of my concerns that exposure to ozone pollution will harm them, since I know from the medical literature that ozone exposure may induce asthma development in children, as well as increase susceptibility to respiratory infections, and may also impair children’s lung growth.

7. From my activities with ATS, I am aware that the United States Environmental Protection Agency (“EPA”) has a responsibility to designate attainment and non-attainment areas under the new, more protective 2015 National Ambient Air Quality Standards for ozone under the Clean Air Act. I am aware that EPA is supposed to do this within two

years of the revision of the standards, but that EPA Administrator Scott Pruitt has decided to delay the designations.

8. Any delay in cleaning up ozone pollution puts the health of my patients, myself, and my family at risk, and means that my I, my patients, and my family must modify our daily lives to avoid exposure to air pollution on high ozone days.

9. I am providing this statement to support the American Thoracic Society's work to ensure EPA fulfills its legal responsibilities under the Clean Air Act.

I declare under penalty of perjury that the foregoing is true and correct.

Executed on: July 10, 2017

Janet M Lyon MD

Exhibit 37

DECLARATION OF JOSEPH O. MINOTT

I, Joseph O. Minott, declare and state as follows:

1. This declaration is based on my personal knowledge. I am over the age of eighteen (18) and suffer no legal incapacity. I submit this declaration in support of the petition of the Clean Air Council (“CAC”) challenging U.S. Environmental Protection Agency (“EPA”) Administrator E. Scott Pruitt’s decision to postpone the implementation of the 2015 National Ambient Air Quality Standards (“NAAQS”) for ozone.

2. I am CAC’s Executive Director and Chief Counsel. I have served in this position for thirty (30) years. Before serving as Executive Director, I was a staff attorney at CAC for four years. My position at CAC makes me responsible for achieving CAC's goals and mission, and requires that I be up to date on CAC’s program activities and membership.

3. The Delaware Valley Citizen’s Council for Clean Air, now known as Clean Air Council, or CAC, was established in 1967. CAC is a 501(c)(3) non-profit, membership organization incorporated in Pennsylvania and headquartered at 135 South 19th Street, Suite 300, Philadelphia, Pennsylvania 19103.

4. CAC works to achieve its mission, which includes protecting everyone's right to breathe clean air, through advocacy and legal action. Among CAC's programmatic activities is its Public Health initiative, which includes advocating for clean air in urban areas. CAC's work includes a focus on ensuring that national standards for health damaging air pollutants like ground-level ozone are met.

5. I understand from my reading and work with CAC that ground level ozone smog irritates the respiratory system, reduces lung function, inflames and damages the cells that line your lungs, making them more susceptible to infections, asthma and aggravated asthma, and aggravating chronic lung disease. Ozone smog also can cause permanent lung damage.

6. CAC currently has nearly 9,000 members in the Mid-Atlantic region. Of those members, most live in the Philadelphia area. In Pennsylvania, Philadelphia is currently designated nonattainment for the 2008 ozone standard, and would also be designated nonattainment for the 2015 standard.

7. Because of my work with CAC, I am familiar with EPA's efforts to regularly evaluate, update and implement the ozone NAAQS. In particular, I am aware that in 2015 EPA strengthened the ozone NAAQS by tightening the maximum level of ozone allowed in the ambient air to 70 parts per billion ("ppb"), down from the 75 ppb allowed under the previous standards which were issued in

2008. EPA's decision was based in part on studies examining health outcomes on real people, linking ozone levels allowed by the 2008 standards to hospital visits and deaths. EPA's independent science advisors unanimously recommended strengthening the ozone standards in 2015 because the 2008 standards were not protective enough of public health and welfare. EPA found, based on studies of 15 urban areas (including the Philadelphia area), that the 2008 standard leaves substantial numbers of children facing dangerous exposure to unsafe levels of ozone smog. Together with other environmental organizations, CAC submitted comments on the need for tighter ozone standards during the rulemaking leading to the final 2015 ozone standards.

8. I am further aware that EPA's Administrator E. Scott Pruitt has announced his decision to delay for a year the implementation of the new, more health-protective ozone standards. That action, if left in place, will delay for a year the achievement of cleaner air in urban areas like Philadelphia, which would see tighter rules governing the emissions of ozone precursor pollutants than is the case under the 2008 rule.

9. In addition to my professional role at CAC, I also have been a CAC member for over twenty (20) years. I am sixty-two (62) years old. I have been a resident of Philadelphia or its suburbs for forty-three (43) years.

10. I suffer from a chronic medical condition called sarcoidosis, which causes shortness of breath, wheezing, and chest pain. The symptoms of sarcoidosis are aggravated by ground-level ozone and ozone smog. I am therefore directly impacted by any EPA decision to delay or postpone the implementation of more health protective ozone standards, as it will prolong my exposure to higher concentrations of ozone that aggravate my condition.

11. I do not own a car, so I walk around Philadelphia on a daily basis. I also enjoy running when the air is clean enough, but I must curtail my running during periods of high concentrations of ground level ozone. I also enjoy simply being outdoors, particularly in the summer months, but I must curtail that activity as well when the ozone is at unhealthy levels.

12. I have children and two small grandchildren who visit me in Philadelphia on a regular basis from their homes in New York and Philadelphia. I am concerned about their health on days when the ozone exceeds the 70 ppb level set by the 2015 ozone standards.

13. CAC supports timely implementation of the 2015 ozone standards, and opposes any attempt by the EPA or Administrator Pruitt to postpone or otherwise delay the steps towards their full implementation. I also support full and timely implementation as a member of CAC, because further delay means my own

and my children's and grandchildren's continued exposure to unhealthful levels of ground-level ozone.

I declare under the penalty of perjury that the foregoing is true and correct to the best of my knowledge and belief.

Executed this 10th day of July, 2017.

A handwritten signature in blue ink, appearing to read "Joseph O. Minott", written over a horizontal line.

Joseph O. Minott, Esq.

Exhibit 38

DECLARATION OF DR. BRIAN MOENCH

1. My name is Brian Moench. I am 66 years old, and I am a doctor with a private practice as an anesthesiologist in Salt Lake City, where I have worked since 1981. I have been Board Certified in anesthesiology since 1983.
2. I have been a member of Physicians for Social Responsibility for about 6 years. In 2007, I founded the Utah Physicians for Health Environment (“UPHE”). I am UPHE’s President and Board chairman, as well as a member of UPHE. UPHE is dedicated to protecting the health and well-being of the citizens of Utah by promoting science-based health education and interventions that result in progressive, measurable improvements to the environment.
3. I currently reside on the east side of Salt Lake County, just south of Salt Lake City, with my wife. I grew up in Salt Lake County, went away to college, and returned in 1981. I have lived at my current residence since 1982 and intend to continue to live here for the foreseeable future. My children and grandchildren visit me at my home frequently.
4. I engage in outdoor activities on a regular basis. I commute to work by bike whenever I can; this is a trip of roughly 26 miles round trip. I also spend many hours a week gardening and doing landscaping maintenance at my home. I plan on continuing these activities for as long as I am able.
5. I am familiar with the harmful effects on human health associated with ozone pollution. As President and founder of UPHE, I give lectures, publish articles, and submit comments to the U.S. Environmental Protection Agency (EPA) and Utah Division of Air Quality (UDAQ) compiling scientific information concerning the health effects of air pollution. Additionally, I coordinated and did the majority of the research for the document *The Health Consequences of Air Pollution* that is featured on UPHE’s website.
6. I have reviewed an important new study that just came out in the esteemed publication The New England Journal of Medicine on June 29, 2017 in which Harvard University scientists found a correlation with mortality at ozone levels *well* below the 70 ppm level of the 2015 ozone NAAQS. This means there is a significant benefit to be derived from reducing air pollution even in areas that meet the NAAQS—and highlights the importance of reducing air pollution in area that do not meet the NAAQS. Other studies have shown similar results; but the size and duration of this study make it particularly noteworthy. The bottom line is

that this study shows quite strikingly that delaying or rolling back any of these standards will cause significant spikes in mortality. The LA Times did a story on this study: www.latimes.com/science/sciencenow/la-sci-sn-air-pollution-death-20170628-story.html. Other studies show synergistic effects between PM2.5 and ozone. That is all the more important for the urban areas of Utah because we are in serious nonattainment for the 24 hour PM2.5 standard.

7. I am aware from my advocacy work that the Salt Lake City area has elevated levels of ozone air pollution. The Wasatch Front is already subject to acute spikes in air pollution. There are several oil refineries near me including the Holly, Tesoro, Chevron, Big West, and Silver Eagle refineries, which I am concerned add to the ozone pollution in my community. As a physician, I am personally aware of the adverse health effects of pollution, including on vulnerable members of the population.
8. I have had a type of cancer that some studies have found to be correlated with air pollution. I also have immediate family members in the Salt Lake Valley who have suffered from diseases that can be attributed in whole in or part to higher rates of air pollution we have in our area. For example, daughter has had breast cancer, which is known to occur more frequently where there are higher rates of air pollution. In total, seven of my twelve immediate family members have had cancer, including two of my children.
9. Other (non-cancer) serious medical conditions related to air pollution have afflicted my family members. My mother died of pneumonia during one of the severe wintertime pollution episodes that are common in Salt Lake County. Poor air quality increases the likelihood that common illnesses, like pneumonia, will be fatal. My mother-in-law died of a pulmonary embolus; the incidence of this disease is dramatically increased by higher levels of air pollution. For the last 12 years of his life, my father was debilitated by a stroke. Medical studies have shown that air pollution can trigger strokes, along with heart attacks and irregular heart rhythms, especially for people already at risk for these conditions.
10. I also know, based on medical studies that I have read and that mainstream medical societies have embraced, that poor air quality compromises my granddaughter's health in multiple ways, even if the effects are currently subclinical and may not become clinical for decades. Some of the well-documented subclinical consequences of air pollution exposure are an accelerated aging process, shortened life span, higher blood pressure, and more rapid decline in cognition.

11. For all of these reasons, I have serious concerns about the way air pollution is affecting and could affect my health and my family's health.
12. As an anesthesiologist, I spend approximately 40 hours per week practicing at the LDS Hospital in Salt Lake City. Because I ride my bicycle to work frequently, the quality of the air that I breathe is a particular concern to me. I track publicly available air quality alerts, and I avoid biking on most high ozone days. If I do bike on high air pollution days, I feel a shortness of breath that I do not feel when biking on clearer days.
13. I regularly see air pollution releases coming from the oil refineries when looking at the northern and western horizon from my home and as I ride to work. From my home, I can see the drift of pollution coming from the "refinery row" as it spreads over the rest of the valley. This haze provides a visual reminder of the safety threats we face from these refineries, as well as the air pollution that I am breathing.
14. Both the Holly and Tesoro Refineries will be expanding operations in the near future (see: www.deseretnews.com/article/865669286/Utah-Supreme-Court-hears-second-refinery-expansion-challenge.html), and the Utah Department of Transportation is planning several major transportation projects over the next two years that will impact air quality (see: www.udot.utah.gov/main/f?p=100:pg:0:::1:T,V:4845). I want those refinery expansions and roadwork projects to be subject to the strongest, most effective pollution control requirements for ozone-forming chemicals.
15. I am aware from published reports that the ozone levels in the Salt Lake City area exceed the levels allowed under the 2015 revised air quality standards for ozone. Issuing attainment and nonattainment designations means that the protections from the revised ozone standards will finally kick in here. This will improve the air that my family and I breathe, will make it less dangerous for us to live and work where we do, and will give me more enjoyment of daily activities like biking or just going downtown.
16. I am submitting this statement in support of Physicians for Social Responsibility's efforts to prevent EPA from delaying making area designations under the 2015 NAAQS. We should not have to delay receiving the protections that the Clean Air Act requires for an additional year.

I declare, under penalty of perjury, that the foregoing is true and correct.

Dated: July 6, 2017

Brian Moench, MD.
Brian Moench, MD

Exhibit 39

DECLARATION OF JANE Z. REARDON

I, Jane Z. Reardon, declare as follows:

1. I live in Granby, Connecticut, approximately 15 miles north of Hartford. I'm 72 years old. I have sat on the National Board of Directors of the American Lung Association ("ALA") since July 2011, and have been involved in supporting ALA's activities since 1975. I am a member of a public policy committee addressing issues in the Northeast, carry out advocacy, and support ALA's research agenda. I have previously served as Chair of the American Lung Association of Connecticut and, most recently, of the ALA of New England's Chartered Association.

2. In addition to my work with the ALA, I have served on the board of the American Association of Cardiovascular and Pulmonary Rehabilitation ("AACVPR"), the AACVPR's Racial and Cultural Diversity Task Force, and the AACVPR's National Strategic Planning Committee.

3. Throughout my career in medical nursing, I have focused on issues of respiratory health. After graduating from nursing school in 1966, I worked for nine years as a nurse in the respiratory intensive care unit of Hartford's St. Francis Hospital. It was there that I first observed the impact the region's dirty air was having on those with chronic respiratory conditions. During Hartford's hot and humid summer months, air pollution forced many with respiratory ailments to make frequent trips to the emergency room, often then requiring the intensive care unit. The struggle of these patients—who often came, as they still do, from Hartford's most impoverished populations—provided me with the impetus to become involved in the work of the American Lung Association. It also inspired my work with several other Hartford nurses on an informational book, "Living with Lung Disease," that was published by the ALA in the 1970s.

4. After nearly three decades at St. Francis Hospital, I joined Hartford Hospital's Pulmonary Care Unit, then a ventilator-weaning unit. There, I developed further interest and expertise in matters of pulmonary health.

5. In 1988, I graduated from Yale University School of Nursing with a Master of Science in Nursing degree. I have worked for many years as a Pulmonary Clinical Nurse Specialist and as a nurse practitioner hospitalist at the Hartford Hospital in the city of Hartford, Connecticut. In my current position at that hospital, I continue to assist patients who are hospitalized as a result of respiratory ailments, among other conditions. A large part of my work involves educating patients about environmental risk factors—like ozone pollution—for their respiratory ailments.

6. In my decades of providing hospital care, I have observed a definite correlation between poor air quality—including elevated levels of ozone pollution—and an increased incidence of respiratory-related hospital admissions. I am also aware from published research that ozone is a severe lung irritant that threatens the well-being of both healthy adults and the more vulnerable, including children, the elderly, and especially those with asthma and Chronic Obstructive Pulmonary Disease. In fact, many of my patients are older than 65, like me.

7. I spend significant time outside in my community. I regularly walk outside in my neighborhood for exercise. I also have a fairly large garden, where I mostly grow flowers, as well as some vegetables. I regularly do yard work tending it.

8. I understand that ozone levels where I live and work sometimes exceed the national ambient air quality standard established in 2015 to protect human health and welfare. Delays in implementing the ozone standard harm me and my patients by allowing dangerous levels of ozone to remain longer. The longer and the more ozone pollution exceeds safe levels,

the more respiratory-related hospital admissions, and the more my health and my patients' health are in danger.

9. I strongly support the American Lung Association's efforts to require full and timely compliance with all requirements of the Clean Air Act to limit ozone pollution.

I declare under penalty of perjury that the foregoing is true and correct.

Executed on June 29, 2017.



Jane Z. Reardon

Exhibit 40

DECLARATION OF NICHOLAS ROGERS

I, Nicholas Rogers, hereby declare and state:

1. This declaration is based on my personal knowledge. I am over the age of eighteen (18) and suffer no legal incapacity. I am a member of Clean Air Council (“CAC”) and have been since January 2012. I submit this declaration in support of the CAC’s petition challenging U.S. Environmental Protection Agency (“EPA”) Administrator E. Scott Pruitt’s decision to postpone the implementation of the 2015 National Ambient Air Quality Standard (“NAAQS”) for ozone.

2. I currently reside at 1839 South Hicks Street, Philadelphia, Pennsylvania 19145. I have lived in Philadelphia for ten years.

3. I run, bike, and walk throughout east, central, and northeast Pennsylvania. I spend approximately ten hours a week outdoors recreating. I am also a competitive cyclist. I regularly compete in bike races (approximately five times a year) throughout southeastern Pennsylvania. Accordingly, I breathe whatever air pollution is present in the ambient air in these areas.

4. I am also an asthmatic. I have had asthma since I was a child. I use my inhaler most weekends as a precautionary measure and suffer from aggravated asthma occasionally throughout the year. I have acute asthma attacks that are triggered by poor outdoor air quality, particularly on bad ozone days, especially while I am exercising.

5. As a member of CAC, I am aware that the United States Environmental

Protection Agency (“EPA”) updated and strengthened the National Ambient Air Quality Standards for ozone pollution in 2015, and that the new standards will improve the air quality where I live, work, and recreate, once they are fully implemented. I understand that EPA’s decision was made based on the fact that the previous (currently effective) standards were not sufficiently health protective, particularly for individuals like me.

6. As a member of CAC, I am further aware that EPA’s Administrator Scott Pruitt has announced his decision to delay for a year the implementation of the new, more health protective ozone standards.

7. I understand that areas where I bike in Pennsylvania will need to take steps to comply with the new standards once the new standards are implemented. That will mean less exposure to unhealthy ozone levels for me when I am biking in those areas.


8. By the same token, I understand that the yearlong delay that the EPA and Administrator Pruitt have announced will prolong my exposure to unhealthy levels of ozone, exacerbating my asthma and putting my health in danger particularly while I engage in cycling and other outdoor recreation.

9. I understand further that the CAC is bringing a lawsuit challenging Administrator Pruitt’s decision to postpone implementing the new more health-protective ozone standard. Vacating or otherwise stopping EPA’s attempt to postpone the full implementation of the 2015 ozone standard will benefit my interest in breathing

cleaner air and avoiding ozone-induced asthma attacks when I am cycling and otherwise recreating outdoors in Pennsylvania. I therefore support CAC bringing this action.

I declare under penalty of perjury that the foregoing is true and correct to the best of my knowledge and belief.

Executed on this 10th day of July, 2017.



Nicholas Rogers

Exhibit 41

DECLARATION OF RUSSELL SEAL

1. My name is Russell Seal and I am of legal age and competent to give this declaration. All information herein is based on my own personal knowledge.
2. I have been a member of the Sierra Club for almost 30 years, since 1988. I am part of the Alamo Group of the Sierra Club's Lone Star Chapter, and have been an active volunteer for years. For example, for 6-7 years I was the Alamo Group Political Chair. Currently I am a member of the Alamo Group's political committee.
3. I also serve as the Sierra Club representative for the Alamo Area Council of Governments ("AACOG") Advisory Committee. AACOG's Natural Resources Department addresses air quality issues in the region by bringing together stakeholders from all interests - government, industry, business, and residents - to develop air pollution reduction plans that benefit our quality of life.
4. I am 58 years old and live with my wife, Karen Dalglish Seal, in LaCoste, Texas, which is in Medina County, just across the county line from Bexar County, which contains the city of San Antonio. I am a retired pharmacist.
5. I have reviewed EPA's continuing education for health professionals on ozone entitled "Ozone and Your Patients' Health Training Course." See: www.epa.gov/ozone-pollution-and-your-patients-health/ozone-and-your-patients-health-about-course. From my work as a pharmacist, my work with AACOG, my own experience with family members, and from information EPA publishes, I am aware that ozone can harm people of all ages, including those who are active outdoors, and can cause health problems including chest pain, coughing,

- inflammation, and even reduced lung function. Ozone predisposes those exposed to have lung irritation and exacerbates effects of other pollutants.
6. I am aware that ozone is the main component of smog. Due to local air flow patterns, ozone smog is worst in the spring and fall in San Antonio and the surrounding counties, particularly from late August through early October. Ozone also harms sensitive vegetation, especially during the growing season. EPA's "Air Now" website (www.airnow.gov) shows that our area has some of the highest readings in the nation in August and September.
 7. From my work as a pharmacist, I understand what ozone means for people's health and wellbeing. I spent 10 years on the south side of San Antonio, then 20 years on the west side of San Antonio; both of these are impoverished areas. During the two primary ozone seasons, our pharmacy would see an enormous spike in asthma-related problems and purchases of asthma products. In particular, during the second ozone season starting in the second week of August, asthma products were the number one product we were selling, constituting about 20% of the pharmacy's total business. We would have to refill our large rack of inhalers 20 deep every two days.
 8. I am aware that Bexar County, which contains San Antonio, is a county that does not comply with the more protective 2015 ozone standards, even though it always has been considered an attainment area under earlier standards. See: <http://setexasrecord.com/stories/510644751-18-texas-counties-deemed-nonattainment-areas-by-epa-due-to-increased-ozone-regulations> and www.tceq.texas.gov/assets/public/implementation/air/sip/ozone/2015Designations/

TXRecommendation/2015Ozone_DesignationRecommendation_Submittal_to_EPA.pdf.

9. My wife and I go to Bexar County every single day, and we routinely spend time outside there. Everything that we need is in San Antonio, so we are there all the time to do things like grocery shop, eat at restaurants, and go to the post office. San Antonio has a river walk area which we visit regularly that has 15 miles of sidewalks and paths as well as restaurants with outdoor seating. We also frequently spend time outdoors in San Antonio's city parks, such as the Medina River Park, to walk our dogs. We plan to continue all of these activities into the foreseeable future and would enjoy them more if ozone conditions were better.
10. Because I am aware that the harm from ozone is a product of the rate of inspiration and length of exposure, I stay inside on high ozone alert days and have to avoid outside activities that I would otherwise enjoy doing like walking in downtown San Antonio or hiking in Texas's national parks. An additional reason for staying indoors on high ozone days is that I have a heart arrhythmia, which could have dangerous consequences if I engage in taxing physical activities outdoors while breathing ozone pollution. When ozone levels are high, I can tell. My throat gets scratchy and I feel a burning sensation and irritation in my chest and lungs.
11. My wife suffers from asthma, and thus is particularly susceptible to ozone, and high ozone alert days very much limit what she can do. When she can, she stays inside, but since she works full time in San Antonio she has little choice but to face some exposure in her commute. She cannot take part in activities that she and I like to do together such as walking around San Antonio or hiking in Texas'

Big Bend National Park, which also has ozone problems, according to EPA's "Air Now" website.


12. Also, my eight year old granddaughter, who lives in San Antonio, is severely asthmatic and requires lots of care and treatment for this condition. On high ozone days, she must remain inside. When ozone is bad, my wife and granddaughter have difficulty breathing, have more asthma attacks, and use their inhalers more often. These health effects happen to them not just on the day of the ozone event, but often within 2-3 days after the initial exposure because of the way that ozone works to harm the human body.
13. I feel strongly that my home, community, and environment are less appealing because of smog in our area. In fact, I attended a City Council meeting last week and this very topic came up. San Antonio's air quality is negatively impacting life in the city.
14. Besides the health harms it causes, ozone also harms trees. This is particularly bad for San Antonio, which is considered a "heat island" because of the local topography combined with the high temperatures. A healthy tree canopy is crucial to mitigating heat island effect, and healthy trees also filter air pollutants and storm water runoff.
15. I understand that the United States Environmental Protection Agency (EPA) has a responsibility to designate attainment and non-attainment areas under the new, more protective 2015 National Ambient Air Quality Standards for ozone under the Clean Air Act. I am aware that EPA is supposed to do this within two years of the revision of the standards (so by October of 2017), but EPA

Administrator Scott Pruitt has decided to give EPA an additional year to make the designations.

16. As mentioned above, I am the Sierra Club representative for AACOG. Among other crucial functions, AACOG provides air quality technical projects including air pollution monitoring and data analysis. However, it is not clear whether AACOG will be able to continue performing these important data-gathering functions because Texas's governor recently cut its funding. This blow to AACOG means it is more critical than ever for EPA to make the nonattainment designation (which will lead to health protections for the San Antonio area) now while AACOG's scientific data is still up to date.
17. Any delay in cleaning up ozone pollution puts the health of myself and my family at risk, and causes me and my family to refrain from or curtail our outdoor activities, diminishes our enjoyment of those activities, and harms vegetation in our community.
18. I support Sierra Club's work to ensure EPA fulfills its obligation to make timely attainment and non-attainment designations, and to provide me and my community protections from ozone pollution.

I declare under penalty of perjury that the foregoing is true and correct.

Executed on: June 27, 2017



Russell Seal

Exhibit 42

DECLARATION OF JOHN STITH

I, John Stith, declare as follows:

1. I am Director of Database Marketing and Analytics at the Environmental Defense Fund (“EDF”). I have had this position for more than ten years.

2. My duties include maintaining an accurate list of members. My colleagues and I provide information to members, acknowledge gifts and volunteer actions, and manage the organization’s member databases. My work requires me to be familiar with EDF’s purposes, staffing, activities, and membership.

3. EDF is a membership organization incorporated under the laws of the State of New York. It is recognized as a not-for-profit corporation under section 501(c)(3) of the United States Internal Revenue Code.

4. EDF relies on science, economics and law to protect and restore the quality of our air, water and other natural resources. EDF employs more than 150 scientists, economists, engineers, business school graduates, and lawyers to help solve challenging environmental problems in a scientifically sound and cost-effective way.

5. It is my understanding that EPA recently announced it was extending the deadline for it to promulgate initial area air quality designations for the 2015 ozone national ambient air quality standards. I am aware that this extension

creates a nationwide delay in the implementation of the 2015 national ambient air quality standards for ozone (80 Fed. Reg. 65,292 (Oct. 26, 2017)). I am aware that the ozone standards delayed by this action were projected to result in significant public health and air quality benefits. I also understand exposure to ozone pollution is harmful to our members' health, and aggravates respiratory diseases such as asthma.

6. EDF has a strong organizational interest, and a strong interest that is based in its members' public health, recreational, aesthetic, professional, educational, environmental, and economic interests, in reducing harmful air pollution.

7. Through its programs aimed at protecting human health, EDF has long pursued initiatives at the state and national levels designed to reduce emissions of harmful air pollutants, including criteria pollutants for which national ambient air quality standards are set under the Clean Air Act. EDF has been involved in previous efforts to address criteria pollutants including efforts to address ozone, sulfur dioxide, and particulate matter.

8. When an individual becomes a member of EDF, his or her current residential address is recorded in our membership database. The database entry reflecting the member's residential address is verified or updated as needed. The database is maintained in the regular course of business and each entry reflecting a


member's residential address and membership status is promptly updated to reflect changes. I obtained the information about our membership discussed below from our membership database.

9. EDF currently has over 410,000 members in the United States, and we have members in all 50 states and the District of Columbia.

10. EDF has 12,517 members that live in the following counties: Gila, AZ; Yuma, AZ; Calaveras, CA; Amador, CA; Tuolumne, CA; Alameda, CA; Butte, CA; Mariposa, CA; San Luis Obispo, CA; Tehama, CA; DeKalb, GA; Fulton, GA; Gwinnett, GA; Henry, GA; Rockdale, GA; Madison, IL; Jefferson, KY; Ascension, LA; East Baton Rouge, LA; Baltimore, MD; Harford, MD; Allegan, MI; Berrien, MI; Macomb, MI; Muskegon, MI; St. Clair, MI; Wayne, MI; Saint Charles, MO; Saint Louis, MO; Clark, NV; Washoe, NV; Dona Ana, NM; Butler, OH; Franklin, OH; Geauga, OH; Hamilton, OH; Lake, OH; Warren, OH; Bexar, TX; El Paso, TX; Davis, UT; Duchesne, UT; Salt Lake, UT; Uintah, UT; Utah, UT; Weber, UT; Door, WI; Manitowoc, WI; Milwaukee, WI; Ozaukee, WI.

11. EDF members living in such counties and in other counties have a strong interest in protecting their health, their family's health, and the environment from ground-level ozone pollution and ensuring that the 2015 health-based ozone standards are properly implemented.

I declare that the foregoing is true and correct.


John Stith

Dated: July 10, 2017

Exhibit 43

DECLARATION OF DREW TOHER

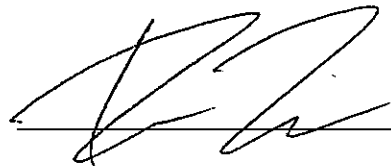
1. I have been a member of National Parks Conservation Association for at least the past six years.
2. I live in Arlington, VA. I've lived here for about three years, and have lived in the Washington, DC, area for about 10 years. I work at a nonprofit organization called Beyond Pesticides. My office is in Washington, DC.
3. I spend a lot of time in national parks around where I live and work. I go to nearby parks on the weekends and after work. I especially like going to Theodore Roosevelt Island, in the District of Columbia, which is a great place to go for a weekend hike. I go there at least once a month. I also regularly go hiking on the Potomac Heritage Trail and at Great Falls National Park, in Virginia. I enjoy the scenery and bird-watching. I see redwing blackbirds at Theodore Roosevelt Island, as well as cardinals, jays, finches, warblers, hawks, and ospreys. I see bald eagles along the Potomac River, too. I also enjoy bird-watching at the Occoquan Bay National Wildlife Refuge and Prince William Forest Park, both located in Prince William County, Virginia. I go to Occoquan Bay National Wildlife Refuge about once a month, and Prince William Forest Park a few times a year.
4. I often walk around the Tidal Basin, in Washington, and visit Arlington Cemetery from time to time, too. I also go walk around the monuments around the National Mall in Washington, DC, a few times a year, when I have visitors from out-of-town.
5. I go hiking or camping in Shenandoah National Park, and to parks farther west in Virginia about every other month. Annually, I also go visit national parks in other parts of the country.
6. The health and wellbeing of the trees and vegetation in the places where I enjoy outdoor activities is very important to me. When they look like they're in poor health or even dying,

that diminishes the aesthetic appeal of the place. The plants and trees are part of the same ecosystem as the wildlife I like watching, so when the plants and trees are doing poorly, it doesn't bode well for the wildlife that depend on them for food and shelter.

7. I am very concerned about the impact of air pollution on the parks I visit. I am aware that air pollution harms plants and trees as they grow and that the Washington, DC, area where I live, work, and spend a lot of time outdoors in parks has levels of ozone pollution above what EPA says is safe for people and the environment.
8. Delays in implementing the ozone standard harm me by allowing the air to remain dirty for longer, and thus harming the plants, trees, and entire ecosystems of the national parks I visit. I fully support National Parks Conservation Association in its efforts to ensure the national ozone standard is timely and fully implemented.

I declare under penalty of perjury that the foregoing is true and correct.

Executed on July 6 2017.



Drew Toher

Exhibit 44

DECLARATION OF GINA TRUJILLO

I, Gina Trujillo, declare as follows:

1. I am the director of membership at the Natural Resources Defense Council, Inc. ("NRDC"). I have been the director of membership since January 2015.
2. My duties include supervising the preparation of materials that NRDC distributes to members and prospective members. Those materials describe NRDC and identify its mission.
3. NRDC is a membership organization incorporated under the laws of the State of New York. It is recognized as a not-for-profit corporation under section 501(c)(3) of the United States Internal Revenue Code.
4. NRDC's mission statement declares that "The Natural Resources Defense Council's purpose is to safeguard the Earth; its people, its plants and animals, and the natural systems on which all life depends." The mission statement goes on to declare that NRDC works "to restore the integrity of the elements that sustain life—air, land, and water—and to defend endangered natural places."
5. When an individual becomes a member of NRDC, his or her current residential address is recorded in NRDC's membership database. When a member renews his or her membership or otherwise makes a contribution to NRDC, the database entry reflecting the member's residential address is verified or updated.
6. Since its inception in 1970, NRDC has worked on issues relating to clear air and climate change. In particular, protecting its members and the public from the substantial adverse health effects and harm caused by ozone pollution and exposure to polluted air is central to NRDC's purpose. NRDC brings cases to protect its members from harm caused by air pollution. NRDC also carries out education and advocacy to inform its members and the public about the harms to both human health and the environment from air pollution.
7. NRDC currently has more than 346,000 members. There are NRDC members residing in each of the 50 United States and in the District of Columbia.

I declare under penalty of perjury that the foregoing is true and correct to the best of my knowledge and belief.

Executed on June 21, 2017.

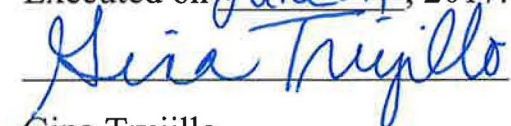

Gina Trujillo

Exhibit 45

DECLARATION OF HAROLD WIMMER

I, Harold Wimmer, declare as follows:

1. I am the President and Chief Executive Officer for the American Lung Association (“ALA”). I am responsible for the overall management and operation of the organization. In that capacity, I am required to be familiar with the organization’s structure, function, purpose, and membership.

2. ALA is incorporated in Maine with headquarters in Chicago, IL. It operates with paid staff and dedicated volunteers in all fifty states and the District of Columbia. ALA is recognized as a not-for-profit corporation under section 501(c)(3) of the United States Internal Revenue Code.

3. ALA has a vision of a world free of lung disease. Its mission statement is “to save lives by improving lung health and preventing lung disease.” As scientific research has shown that air pollution is a major contributor to the worsening of lung disease, the ALA has conducted advocacy and litigation to promote full and timely implementation of the Clean Air Act for many years. ALA is also committed to improving lung health and preventing lung disease through education, including informing the public about health threats from air pollution.

4. ALA has members and supporters throughout the country residing in counties with ozone levels that are below the 2008 national ambient air quality standard (“NAAQS”) for ozone averaged over eight hours of 0.075 parts per million (“ppm”) and above 0.070 ppm, the NAAQS EPA adopted on October 1, 2015, in the last review because of the scientific evidence of harm to human health above that level. The harm to human health includes increased risk of asthma attacks, hospitalization and premature death. The individuals residing in these areas will benefit from those areas’ meeting the tightened, 2015 ozone standards as soon as possible.

5. ALA also has members and supporters throughout the country that reside in areas exceeding both the 2015 and the 2008 standards for ozone. These individuals would also benefit from those areas' meeting the tightened, 2015 ozone standards as soon as possible.

6. ALA members and supporters living in the above-named and other areas have a strong interest in full, timely, and effective implementation of the Clean Air Act requirements designed to protect them from unhealthy levels of pollution in the air they breathe. EPA's failure to implement the 2015 NAAQS for ozone by extending by a full year its deadline for making determinations of attainment and nonattainment ALA's members and supporters the protections guaranteed under the Clean Air Act. The extension means that the ozone pollution levels will be allowed to exceed the level EPA has determined is required to protect public health for a year longer than they otherwise would be, and clean up efforts will be allowed to be delayed by a year. For the ALA members and supporters living, working, and enjoying outdoor recreation in the above-named and other areas, this delay in pollution control puts their health and welfare on the line, as the pollution at issue causes asthma attacks, hospitalization, and, likely, premature death.

I declare under penalty of perjury that the foregoing is true and correct.

Executed on June 30, 2017.


Harold Wimmer

Exhibit 46

DECLARATION OF NSEDU OBOT WITHERSPOON

1. My name is Nsedu Obot Witherspoon. Since 1998, I have been a member of the American Public Health Association (“APHA”) and its Environmental Health and Maternal and Child Health sections. I am currently on the board of governing counselors, a group that sets policies and standards which then go to executive board for approval. I served three years on the executive board between 2008 and 2011, when I was Chair of the Intersection Council. Also, from 2003 to 2005, I was the Chair of APHA’s Environment Section.

2. I have a B.S. in Biology Pre Med from Siena College and an M.P.H. in Maternal and Child Health from The George Washington University, School of Public Health and Health Services. I am a public health professional and leader in children’s environmental health and I present, lecture, and publish on the topic of protecting all children from environmental harm. I previously served as a member of the Institute of Medicine’s Environmental Health Sciences Roundtable, and Co-Chair of the Leadership Council for the National Conversation on Public Health and Chemical Exposures. I am a member of the Friends of the Columbia Center for Children’s Environmental Health and a former Strategy Advisor for the California Breast Cancer Prevention Initiatives (CBCPI) Project. I am also a Board member for the Pesticide Action Network of North America and I serve on the Maryland Children’s Environmental Health Advisory Council. I have held past positions with the Children’s Health Protection Advisory Committee for the Environmental Protection Agency, the National Association of Environmental Health Sciences Council, and the National Institute for Environmental Health Sciences Public Interest Partners.

3. I am 42 years old and live in Waldorf, Maryland (in Charles County, within the Washington, DC metro area) with my husband and four children ages 14, 11, 7, and 15 months. We have been here for 14 years.

4. In the summer months, my kids are outside in our yard almost every day playing basketball or soccer, swimming in our pool, or riding their bikes. Our family loves to hike and take walks about 3-4 times a month during the summer on rail trails in our county, and we enjoy outdoor time together and with friends on our deck. We also have a garden that we enjoy tending, and this year we've been growing some of our own vegetables.

5. Our seven year old has asthma. He had his first bad asthma attack at just three years old. We took him to the doctor not realizing how bad things were; when we got there, they said his oxygen levels were so low that we had to leave our car and go immediately to the ER in an ambulance. This was one of the scariest moments of my life. He was hospitalized for two nights. He was again hospitalized at the age of four. These visits to the ER were long, strenuous, and frightening occasions.

6. Now our son has a rigorous asthma management plan with frequent check-ups at the pediatrician's office. He has to use a nebulizer during the change of seasons, he sometimes has to take medications that make him hyperactive, and we have to have a rescue inhaler on hand. When his asthma acts up, as frequently occurs when air quality is bad, we have to bring him to the doctor on a weekly basis for check-ins. A basic chest cold gets magnified by his asthma; it's a ripple effect where he can get to the point where he can't sleep well, gets dark circles under his eyes, and can't concentrate. He comes into our room crying at 2 or 3am because his chest hurts and neither he nor my husband or I can sleep. He has to miss school because of asthma about three to four times per year. We always monitor air quality alerts and take extra precautions when ozone levels are high.

7. Of course, our son's asthma, which is exacerbated by exposure to air pollution, impacts his quality of life. When he's sick, our 7-year old can't always fully participate in the outdoor activities he usually does, and he often has to slow down or sit out on high air pollution days. We try to manage his asthma regime so he can do as much as possible, but we cannot control what pollutants are in the air. His asthma impacts quality of life of the

whole family, because on bad air quality days, we often need to cancel plans for outdoor activities.

8. My husband has been asthmatic all his life, and has to use an inhaler just about every day, and with increased frequency in high ozone summer months, including early in the morning or late at night. This year for the first time in over 20 years, he had to use a nebulizer. Because of his asthma, any respiratory tract infection can hit him hard. For example, he was diagnosed with bronchitis in the late fall of last year and had a terrible cough that kept re-emerging over the course of many months. Especially when air pollution levels are high, my husband cannot participate in outdoor family activities because his chest feels tight and he does not want to exacerbate his asthma.

9. My husband's and son's asthma also present a significant economic strain for our family. The costs of inhalers, albuterol (the liquid that goes into nebulizers), and rescue inhaler are insane. One inhaler was over \$100 – and that was *with* health insurance; I can't imagine what families manage who don't have health insurance do. Each visit to the doctor entails a \$30 copay, and when my son's asthma is bad, these visits are weekly. Economic and time costs are huge every time he's home from school because we have to take time off from work to care for him.

10. I am aware that ozone is the main component of smog, which is a problem in the DC area. I am aware that ozone can harm people of all ages, but especially children with asthma, like my son, by causing a host of respiratory health problems. Smog is a problem in Charles County because of the recent population growth, and the traffic pollution in my city has become *intense*. We also have other pollution sources nearby that concern me, such as the Panda Cedarville natural gas and oil power plant in Brandywine and the huge Morgantown Generating Coal Power plant where we see smoke coming out of the stacks every time we drive past.

11. Knowing the health and environmental effects of ozone pollution from my work, I am very concerned about my family's exposure to it. I especially worry about the impacts to my asthmatic husband and son, and we are hoping that our 15 month old little girl will not develop asthma. I'm concerned that pollution levels could lead to another hospitalization event for our son. I never want that to happen again.

12. I am a member and President of the Parent Teacher Association at my children's elementary school and a soccer coach for the county. As a resident, I expect more. I expect to be protected. There are enough challenges in the course of the day just maintaining our homes and families. We need decent air, water, and a food supply in order to live. I see this as a basic right for me, my family, and my community.

13. EPA's delay in designations for the 2015 ozone standards means that my family will have to wait an extra year for Clean Air Act protections against ozone levels that harm us. Any delay in cleaning up ozone pollution means more pollution that harms our health, leading to preventable expenditures on medications, preventable asthma attacks and exacerbated respiratory infections, preventable missed days of school and work, and potentially even a preventable frightening hospitalization event. The additional pollution during the delay will also negatively impact our quality of life, especially that of my son and husband who have to refrain from outdoor activities they enjoy and experience diminished enjoyment of those outdoor activities they do participate in. This is unacceptable.

14. I am providing this statement to support the APHA's work to ensure EPA fulfills its obligation to make attainment and non-attainment designations within two years of the revision of the ozone national ambient air quality standards, as the Clean Air Act requires, and to provide me and my community protections from ozone pollution.

I declare under penalty of perjury that the foregoing is true and correct.

Executed on: July 6, 2017

A handwritten signature in blue ink, appearing to read "Nadeu U. Thompson", is written over a horizontal line.