ORAL ARGUMENT NOT YET SCHEDULED

No. 16-1406, consolidated with
Nos. 16-1410, 16-1428, 16-1429, 16-1432, 16-1435, 16-1436, 16-1437, 16-1438, 16-1439, 16-1440, 16-1441, 16-1442, 16-1443, 16-1444, 16-1445, 16-1448, 17-1066

UNITED STATES COURT OF APPEALS
FOR THE DISTRICT OF COLUMBIA CIRCUIT

State of Wisconsin, *et al.*,
Petitioners,

v.

United States Environmental Protection Agency, *et al.*,
Respondents.

On Petition for Review of Final Action by the United States Environmental Protection Agency

AMICUS CURIAE BRIEF BY THE AMERICAN THORACIC SOCIETY IN SUPPORT OF RESPONDENT-INTERVENORS AMERICAN LUNG ASSOCIATION, APPALACHIAN MOUNTAIN CLUB, ENVIRONMENTAL DEFENSE FUND, AND SIERRA CLUB

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Dated: February 20, 2018

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CERTIFICATE AS TO PARTIES, RULINGS, AND RELATED CASES

Pursuant to Circuit Rule 28(a)(1), counsel certifies as follows:

(A) **Parties and Amici**. All parties, intervenors, and amici appearing in this Court are listed in the Briefs for State Petitioners, Cedar Falls Utilities, and City of Ames, Dkt. No. 1693484; Petitioners Conservation Groups and the State of Delaware, Dkt. No. 1693488; and Industry Petitioners, Dkt. No. 1693490.

(B) **Rulings Under Review**. The final agency action under review is a rule entitled "Cross-State Air Pollution Rule Update for the 2008 Ozone NAAQS," published at 81 Fed. Reg. 74,504 (Oct. 26, 2016).

(C) **Related Cases**. *Amicus curiae* is unaware of any related cases other than these consolidated cases.

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CORPORATE DISCLOSURE STATEMENT

In compliance with Federal Rule of Appellate Procedure 26.1 and Circuit Rule 26.1, *amicus curiae* the American Thoracic Society (ATS) states that it is an educational and scientific non-profit organization that represents more than 15,000 respiratory health care professionals. ATS works to prevent and combat respiratory disease through research, education, patient care, and advocacy. ATS does not have a parent corporation, and no publicly held corporation owns 10% or more of its stock.

STATEMENT OF COUNSEL PURSUANT TO FEDERAL RULE OF APPELLATE PROCEDURE 29(a)(4)(E)

Pursuant to Federal Rule of Appellate Procedure 29(a)(4)(E), counsel for *amicus curiae* hereby states that no counsel for any party to this litigation authored this brief in whole or in part, no party or party's counsel contributed money that was intended to fund, or did fund, the preparation or submission of this brief, and no person, other than the *amicus curiae*, contributed money that was intended to fund, or did fund, the preparation or submission of this brief.

PERTINENT STATUTES AND REGULATIONS

Pertinent statutes and regulations are contained in the addenda to the Briefs for State Petitioners, Cedar Falls Utilities, and City of Ames, Dkt. No. 1693484; Petitioners Conservation Groups and the State of Delaware, Dkt. No. 1693488;

Industry Petitioners, Dkt. No. 1693490; and Respondent Environmental Protection Agency, Dkt. No. 1713512.

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GLOSSARY

ATS	American Thoracic Society
CAA	Clean Air Act
CSAPR	Cross-State Air Pollution Rule
CSAPR Update	Cross-State Air Pollution Rule Update for the 2008 Ozone NAAQS
EPA	U.S. Environmental Protection Agency
NAAQS	National Ambient Air Quality Standard(s)
NO_X	Nitrogen oxides
ppm	Parts per million
PM	Particulate matter
$PM_{2.5}$	Particulate matter less than 2.5 micrometers in aerodynamic diameter

STATEMENT OF IDENTITY AND INTEREST OF *AMICUS CURIAE*

The American Thoracic Society (ATS) is an international educational and scientific organization founded in 1905 that represents more than 15,000 respiratory health care professionals. ATS works to prevent and fight respiratory disease around the globe through research, education, patient care, and advocacy. ATS publishes three peer-reviewed scientific journals that disseminate groundbreaking research, including studies on air pollution and health.

Amicus curiae ATS supports Respondent-Intervenors American Lung Association, Appalachian Mountain Club, Environmental Defense Fund, and Sierra Club because the remedy sought by Petitioners, invalidation of a rule reducing air pollution that crosses state boundaries, will do grave harm to public health. The collective medical, scientific, and clinical expertise of *amicus* leads it to participate in this action to demonstrate the severity of the health impacts associated with the air pollution addressed by the challenged rule. In light of the vital importance of this case to the millions of people living in eastern states affected by out-of-state air pollution, *amicus* urges the Court to deny Petitioners' request to set aside the rule.

ATS filed a motion for leave to participate in this action on February 16, 2017. Dkt. No. 1661827. The Court granted ATS's motion on March 2, 2017. Dkt. No. 1663967.

BACKGROUND

Air pollution does not respect state lines. Emissions from upwind states may hinder the ability of downwind states to achieve compliance with the Clean Air Act's (CAA) National Ambient Air Quality Standards (NAAQS), which are set by the U.S. Environmental Protection Agency (EPA) at levels which, "allowing an adequate margin of safety, are requisite to protect the public health." 42 U.S.C. § 7409(b)(1). To address this problem, Congress enacted the "good neighbor" provision of the CAA, which bars a state from emitting air pollutants in amounts that "contribute significantly" to non-attainment or interference with maintenance of the NAAQS in downwind states. *Id.* § 7410(a)(2)(D)(i). Under the CAA, states have the primary duty to develop and submit to EPA state implementation plans that implement the NAAQS and the good neighbor provision, among other elements. *Id.* § 7410(a)(1)–(2). For states that fail to submit adequate state implementation plans, the CAA requires EPA to promulgate federal implementation plans. *See id.* § 7410(c)(1).

Pursuant to EPA's statutory obligation under the good neighbor provision, EPA published a final rule on October 26, 2016, entitled "Cross-State Air Pollution Rule Update for the 2008 Ozone NAAQS" (CSAPR Update Rule or Rule). 81 Fed. Reg. 74,504 (Oct. 26, 2016). The Rule finalizes federal implementation plans for twenty-two eastern states that failed to submit state implementation plans

adequately addressing the requirements of the good neighbor provision as to the 2008 ozone NAAQS. *Id.* at 74,506. The Rule will reduce emissions of oxides of nitrogen (NO_X) from power plants by 75,000 tons per year. *Id.* at 74,573. NO_X reacts with other chemicals in the atmosphere, forming ozone and particulate matter (PM). Ozone and PM can then be transported up to hundreds of miles downwind where they degrade air quality and harm public health. *Id.* at 74,511.

As discussed in detail below, exposure to ozone and PM has a wide array of well-documented and serious negative health effects, including lung cancer, strokes, pneumonia, and premature birth. Consequently, the Rule will have considerable public health benefits. The Rule will have estimated benefits totaling $530–880 million at a cost of $68 million per year. *Id.* at 74,573–75. Accordingly, the benefits far outweigh the costs of implementation; every dollar invested to comply with the Rule will generate approximately $7–13 in benefits for Americans.

SUMMARY OF ARGUMENT

Amicus curiae submits this brief to assist the Court in understanding the significant public health impacts of the CSAPR Update Rule. Air pollution from ozone and PM measurably and substantially shortens lives and reduces the quality of life for affected individuals. Vacating the Rule or delaying its implementation

would cause hundreds of thousands of Americans to suffer preventable harms, including premature death.

The CSAPR Update Rule requires power plants to reduce their emissions of ozone- and PM-causing NO_X that contribute to air quality violations in downwind states. Exposure to ozone and PM can have serious health impacts, including lung cancer, premature death, asthma exacerbations, increased hospitalizations for cardiovascular and respiratory illnesses, and adverse neurological impacts. These pollutants are especially harmful to children, whose respiratory systems are developing; to the elderly, whose respiratory systems can be compromised by age; and to those whose respiratory systems are compromised by disease or disability.

EPA carefully calibrated the Rule such that its public health benefits dwarf the costs of compliance. A wealth of scientific studies demonstrates that improving air quality—in this instance, by preventing upwind states from polluting the air downwind—benefits public health.

Amicus curiae ATS supports EPA's efforts to protect citizens of downwind states from the avoidable health risks caused by air pollution from upwind states. Accordingly, *amicus* ATS urges the Court to deny Petitioners' request to set aside the Rule.

ARGUMENT

This case presents issues of extraordinary importance because the interstate air pollution targeted by the Rule threatens the lives and health of millions of Americans. NO_X emissions react in the atmosphere to form dangerous pollutants, most notably ozone and PM. 81 Fed. Reg. at 74,504. Emissions from power plants travel great distances downwind, elevating ozone and PM levels and threatening public health in areas hundreds of miles away. *See id.* at 74,511.

Health professionals have long understood that ozone and PM have significant and severe health impacts.[1] An extensive body of scientific and medical research documents the link between the emissions targeted by the Rule and human health.[2] Ozone and PM are especially dangerous because they reduce life

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[1] *See* STAFF OF S. COMM. ON THE ENV'T AND PUB. WORKS, 95TH CONG., A LEGISLATIVE HISTORY OF THE CLEAN AIR ACT AMENDMENTS OF 1977, 6634–55 (1978); *see also* Morton Lippmann, *Health Effects of Airborne Particulate Matter*, 357 NEW ENG. J. MED. 2395, 2396 (2007); Edward S. Schelegle et al., *6.6-Hour Inhalation of Ozone Concentrations from 60 to 87 Parts Per Billion in Healthy Humans*, 180 AM. J. RESPIRATORY & CRITICAL CARE MED. 265, 265 (2009); Ephraim Thaller et al., *Moderate Increases in Ambient $PM_{2.5}$ and Ozone are Associated with Lung Function Decreases in Beach Lifeguards*, 50 J. OCCUPATIONAL & ENVTL. MED. 202, 202 (2008) (reporting adverse effects of air pollution even at low levels).

[2] *See generally* U.S. ENVTL. PROT. AGENCY, INTEGRATED SCIENCE ASSESSMENT FOR PARTICULATE MATTER, EPA/600/R-08/139F (2009) [hereinafter INTEGRATED SCIENCE ASSESSMENT FOR PM] (reviewing and summarizing scientific literature on impacts of PM on human health); U.S. ENVTL. PROT. AGENCY, INTEGRATED SCIENCE ASSESSMENT FOR OZONE AND RELATED PHOTOCHEMICAL OXIDANTS, EPA 600/R-10/076F (2013) (reviewing and summarizing scientific literature on impacts

expectancy and increase mortality. These pollutants can bypass the body's defensive mechanisms and enter the blood stream, then traveling throughout the body.[3] Exposure to ozone and PM can increase the risk of premature death and cause or contribute to a host of neurological, cardiopulmonary, and respiratory ailments as varied as lung cancer, asthma, diabetes, heart attacks, strokes, pneumonia, dementia, and premature birth.[4] Children, the elderly, and patients with underlying health conditions, such as cardiopulmonary disease, are particularly susceptible to the adverse health effects of air pollution. These harmful effects are thoroughly documented in the scientific literature, some of which is presented below. This scientific consensus underscores the critical need for prompt implementation of the Rule.

I. THE RULE WILL SAVE LIVES

Without the Rule, air pollution that crosses state lines will cause preventable premature deaths. Air pollution's immediate acute effects include the increased

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of ozone on human health); George D. Thurston et al., *A joint ERS/ATS policy statement: what constitutes an adverse health effect of air pollution? An analytical framework*, EUR. RESPIRATORY J., Jan. 11, 2017, at 1.

[3] Günter Oberdörster et al., *Nanotoxicology: An Emerging Discipline Evolving from Studies of Ultrafine Particles*, 113 ENVTL. HEALTH PERSP. 823, 823 (2005); *see also* Mark R. Miller et al., *Inhaled Nanoparticles Accumulate at Sites of Vascular Disease*, 11 ACS NANO 4542, 4542–43 (2017).

[4] Thurston et al., *supra* note 2, at 7, 13 (referring to heart attacks as "myocardial infarctions").

risk of deadly heart attacks and strokes.[5] Further, chronic exposure to air pollution increases the risk of death from cardiovascular disease and lung cancer.[6] By reducing concentrations of ozone and PM in the air of downwind states, the Rule will prevent up to eighty-three premature deaths every year, according to EPA's estimate.[7]

Numerous studies demonstrate that exposure to ozone can shorten life.[8] Several multi-city studies illustrate the life-shortening effects of ozone,[9] including

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[5] Yun-Chul Hong et al., *Effects of Air Pollutants on Acute Stroke Mortality*, 110 ENVTL. HEALTH PERSP. 187, 188 (2002); Thurston et al., *supra* note 2, at 7, 9; Shang-Shyue Tsai et al., *Evidence for an Association Between Air Pollution and Daily Stroke Admissions in Kaohsiung, Taiwan*, 34 STROKE 2612, 2615 (2003).

[6] C. Arden Pope III et al., *Lung Cancer, Cardiopulmonary Mortality, and Long-Term Exposure to Fine Particulate Air Pollution*, 287 J. AM. MED. ASS'N 1132, 1136–37 (2002) [hereinafter Pope et al. (2002)]; C. Arden Pope III et al., *Cardiovascular Mortality and Long-Term Exposure to Particulate Air Pollution: Epidemiological Evidence of General Pathophysiological Pathways of Disease*, 109 CIRCULATION 71, 74–76 (2004) [hereinafter Pope et al. (2004)] (finding that 10 $\mu g/m^3$ increase in $PM_{2.5}$ increased mortality risk by 8–18%).

[7] U.S. ENVTL. PROT. AGENCY, REGULATORY IMPACT ANALYSIS OF THE CROSS-STATE AIR POLLUTION RULE (CSAPR) UPDATE FOR THE 2008 NATIONAL AMBIENT AIR QUALITY STANDARDS FOR GROUND-LEVEL OZONE 5-21 (2016) [hereinafter REGULATORY IMPACT ANALYSIS].

[8] *See, e.g.*, Michelle L. Bell et al., *A Meta-Analysis of Time-Series Studies of Ozone and Mortality with Comparison to the National Morbidity, Mortality, and Air Pollution Study*, 16 EPIDEMIOLOGY 436, 442 (2005); Kazuhiko Ito et al., *Associations Between Ozone and Daily Mortality: Analysis and Meta-Analysis*, 16 EPIDEMIOLOGY 446, 455 (2005); Jonathan I. Levy et al., *Ozone Exposure and Mortality: An Empiric Bayes Metaregression Analysis*, 16 EPIDEMIOLOGY 458, 466 (2005).

[9] Bell et al., *supra* note 8, at 442; KLEA KATSOUYANNI ET AL., HEALTH EFFECTS INST. RESEARCH REP. NO. 142, AIR POLLUTION AND HEALTH: A EUROPEAN AND

two that identified elevated risk of premature death in the northeastern U.S.[10]—the region most directly served by the Rule. The National Research Council has confirmed this threat from ozone and explained that premature death caused by ozone is not restricted to people who are already in poor health.[11]

Numerous studies also demonstrate that PM exposure, even at low levels, can lead to premature mortality through multiple pathways.[12] Acute PM exposure increases the risk of death from respiratory and cardiovascular causes;[13] chronic

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NORTH AMERICAN APPROACH (2009); Mercedes Medina-Ramón & Joel Schwartz, *Who Is More Vulnerable to Die from Ozone Air Pollution?*, 19 EPIDEMIOLOGY 672, 672 (2008).

[10] Michelle L. Bell & Francesca Dominici, *Effect Modification by Community Characteristics on the Short-Term Effects of Ozone Exposure and Mortality in 98 US Communities*, 167 AM. J. EPIDEMIOLOGY 986, 986 (2008); Richard L. Smith et al., *Reassessing the Relationship Between Ozone and Short-Term Mortality in US Urban Communities*, 21 INHALATION TOXICOLOGY 37, 37 (2009).

[11] NAT'L RES. COUNCIL, NAT'L ACAD. OF SCI., ESTIMATING MORTALITY RISK REDUCTION AND ECONOMIC BENEFITS FROM CONTROLLING OZONE AIR POLLUTION 8 (2008).

[12] *See, e.g.*, Robert D. Brook et al., *Particulate Matter Air Pollution and Cardiovascular Disease: An Update to the Scientific Statement From the American Heart Association*, 121 CIRCULATION 2331 (2010); C. Arden Pope III & Douglas W. Dockery, *Health Effects of Fine Particulate Air Pollution: Lines that Connect*, 56 J. AIR WASTE MGMT. ASS'N 709, 709 (2006) (presenting new evidence and noting consistent evidence found by serial prior studies).

[13] Meredith Franklin et al., *Association Between $PM_{2.5}$ and All-Cause and Specific-Cause Mortality in 27 US Communities*, 17 J. EXPOSURE SCI. & ENVTL. EPIDEMIOLOGY 279, 279, 285 (2007); Cathryn Tonne et al., *A Case-Control Analysis of Exposure to Traffic and Acute Myocardial Infarction*, 115 ENVTL. HEALTH PERSP. 53, 53 (2007); *see also* Annette Peters et al., *Exposure to Traffic and the Onset of Myocardial Infarction*, 351 NEW ENG. J. OF MED. 1721 (2004).

exposure increases the risk of death from lung cancer and cardiovascular disease.[14] Research has also found a strong and consistent correlation between adult diabetes and PM, suggesting that PM is a risk factor for life-shortening diabetes.[15]

Conversely, decreases in PM exposure improve human health and increase average life expectancy.[16] Decreases in long-term exposure reduce mortality rates

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[14] Michael Jerrett et al., *Spatial Analysis of Air Pollution and Mortality in California*, 188 AM. J. OF RESPIRATORY AND CRITICAL CARE MED. 593 (2013); Pope et al. (2002), *supra* note 6, at 1136–37; Pope et al. (2004), *supra* note 6, at 74–76; Michelle C. Turner et al., *Long-term Ambient Fine Particulate Matter Air Pollution and Lung Cancer in a Large Cohort of Never-Smokers*, 184 AM. J. OF RESPIRATORY AND CRITICAL CARE MED. 1374 (2011).

[15] Ikenna C. Eze et al., *Association Between Ambient Air Pollution and Diabetes Mellitus in Europe and North America: Systematic Review and Meta-Analysis*, 123 ENVTL. HEALTH PERSP. 381 (2015); John F. Pearson et al., *Association Between Fine Particulate Matter and Diabetes Prevalence in the U.S.*, 33 DIABETES CARE 2196, 2196 (2010). A diagnosis of diabetes reduces life expectancy by an average of eight years. Oscar H. Franco et al., *Associations of* Diabetes Mellitus *With Total Life Expectancy and Life Expectancy With and Without Cardiovascular Disease*, 167 ARCHIVES INTERNAL MED. 1145, 1145 (2007).

[16] *See, e.g.*, Andrew W. Correia et al., *Effects of Air Pollution Control on Life Expectancy in the United States: An Analysis of 545 U.S. Counties for the Period from 2000 to 2007*, 24 EPIDEMIOLOGY 23, 23 (2013); Wei Huang et al., *Inflammatory and Oxidative Stress Responses of Healthy Young Adults to Changes in Air Quality during the Beijing Olympics*, 186 AM. J. OF RESPIRATORY AND CRITICAL CARE MED. 1150 (2012); David Q. Rich, *Accountability studies of air pollution and health effects: lessons learned and recommendations for future natural experiment opportunities*, ENV'T INT'L, March 2017, at 62; David Q. Rich et al., *Association Between Changes in Air Pollution Levels During the Beijing Olympics and Biomarkers of Inflammation and Thrombosis in Healthy Young Adults*, 307 J. OF THE AM. MED. ASS'N 2068 (2012); Joel Schwartz et al., *The Effect of Dose and Timing of Dose on the Association Between Airborne Particles and Survival*, 116 ENVTL. HEALTH PERSP. 64, 68 (2008) (finding no evidence of a threshold in the association between $PM_{2.5}$ exposure and risk of death, suggesting

to a greater extent than previously believed.[17] Reduced exposure is associated with reduced mortality from cardiovascular disease and lung cancer.[18] Studies also show that limiting PM pollution can produce substantial improvements in public health in a short period of time.[19]

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efforts to reduce PM as much as feasible are most effective way to improve public health).

[17] *See* Qian Di et al., *Air Pollution and Mortality in the Medicare Population*, 376 NEW ENG. J. OF MED. 2513 (2017); Henry A. Roman et al., *Expert Judgment Assessment of the Mortality Impact of Changes in Ambient Fine Particulate Matter in the U.S.*, 42 ENVTL. SCI. TECH. 2268, 2268 (2008). EPA has tightened several different NAAQS in recent years, but there is still a positive correlation between better health and reduced air pollution at levels below the NAAQS. EPA even recognized this point in its most recent decision to tighten the standard for 24-hour $PM_{2.5}$. *See* National Ambient Air Quality Standards for Particulate Matter, 78 Fed. Reg. 3,086, 3,098 (Jan. 15, 2013) ("[E]vidence- and risk-based approaches using information from epidemiological studies to inform decisions on $PM_{2.5}$ standards are complicated by the *recognition that no population threshold, below which it can be concluded with confidence that $PM_{2.5}$-related effects do not occur, can be discerned from the available evidence*.") (emphasis added).

[18] Francine Laden et al., *Reduction in Fine Particulate Air Pollution and Mortality: Extended Follow-Up for the Harvard Six Cities Study*, 173 AM. J. RESPIRATORY & CRITICAL CARE MED. 667, 668–69 (2006).

[19] Sara H. Downs et al., *Reduced Exposure to PM_{10} and Attenuated Age-Related Decline in Lung Function*, 357 NEW ENG. J. MED. 2338, 2346 (2007); Guojun He et al., *The Effect of Air Pollution on Mortality in China: Evidence from the 2008 Beijing Olympic Games*, J. OF ENVTL. ECON. AND MGMT., Sept. 2016, at 18; Robin C. Puett et al., *Chronic Particulate Exposure, Mortality and Coronary Heart Disease in the Nurses' Health Study*, 168 AM. J. EPIDEMIOLOGY 1161, 1167 (2008); Chang Su et al., *Assessing responses of cardiovascular mortality to particulate matter air pollution for pre-, during- and post-2008 Olympics periods*, ENVTL. RES., Oct. 2015, at 112; Antonella Zanobetti et al., *Particulate Air Pollution and Survival in a COPD Cohort*, 7 ENVTL. HEALTH 48, 55–56 (2008); Antonella Zanobetti & Joel Schwartz, *The Effect of Fine and Coarse Particulate*

For instance, in 2009, researchers compared data on PM pollution and life expectancy in fifty-one U.S. cities between 1980 and 2000.[20] After controlling for socioeconomic, demographic, and lifestyle factors like smoking, the study revealed that decreasing exposure to $PM_{2.5}$ (particulate matter less than 2.5 micrometers in aerodynamic diameter) could increase life expectancy.[21] The study also demonstrated that reduced pollution accounted for as much as fifteen percent of the overall increase in life expectancy seen in those cities.[22]

Even relatively small reductions of PM pollution at lower concentrations can save lives.[23] A review of epidemiological studies by the California Environmental Protection Agency's Air Resources Board found a strong relationship between $PM_{2.5}$ exposure and premature death.[24]

Air Pollution on Mortality: A National Analysis, 117 ENVTL. HEALTH PERSP. 898, 902 (2009).

[20] C. Arden Pope III et al., *Fine-Particulate Air Pollution and Life Expectancy in the United States*, 360 NEW ENG. J. MED. 371, 384–85 (2009).

[21] Concentrations of chemicals in air are typically measured in units of the mass of chemical (milligrams, micrograms, nanograms, or picograms) per cubic meter or cubic foot of air.

[22] *Id.* at 376, 384.

[23] Laden et al., *supra* note 18, at 668–69.

[24] AIR RES. BD., CAL. ENVTL. PROT. AGENCY, METHODOLOGY FOR ESTIMATING PREMATURE DEATHS ASSOCIATED WITH LONG-TERM EXPOSURES TO FINE AIRBORNE PARTICULATE MATTER IN CALIFORNIA: DRAFT STAFF REPORT 1 (2009) (attributing 18,000 deaths annually to $PM_{2.5}$ in California alone). In addition, several studies have undermined the suggestion that increases in mortality arising from air pollution exposure merely "displace" the demise of the sick or frail by just a few days. Francesca Dominici et al., *Airborne Particulate Matter and Mortality:*

Successive assessments of the risk of premature mortality from PM pollution have shown that the risk is greater than previously believed.[25] In 2007, a groundbreaking study of 66,000 women in thirty-six U.S. cities used improved data collection and methodologies and found that increased $PM_{2.5}$ exposure raised the risk of death from cardiovascular disease by more than six times the amount calculated by earlier, less thorough studies.[26] The aforementioned studies provide only a sample from the mountain of evidence supporting the Rule's lifesaving potential.

II. THE RULE WILL REDUCE CARDIOVASCULAR, RESPIRATORY, AND NEUROLOGICAL ILLNESSES

Exposure to the air pollutants targeted by the Rule can also cause serious illness. EPA estimates the Rule will prevent over 300 hospital and emergency

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Timescale Effects in Four US Cities, 157 AM. J. EPIDEMIOLOGY 1055, 1062 (2003); Antonella Zanobetti et al., *The Temporal Pattern of Respiratory and Heart Disease Mortality in Response to Air Pollution*, 111 ENVTL. HEALTH PERSP. 1188, 1192 (2003).

[25] Michael Jerrett et al., *Spatial Analysis of Air Pollution and Mortality in Los Angeles*, 16 EPIDEMIOLOGY 727, 732 (2005); C. Arden Pope III, *Mortality Effects of Longer Term Exposures to Fine Particulate Air Pollution: Review of Recent Epidemiological Evidence*, 19 INHALATION TOXICOLOGY (SUPP. 1) 33 (2007) (concluding short-term exposure studies capture only small amount of overall health effects of long-term repeated PM exposure).

[26] Kristen A. Miller et al., *Long-Term Exposure to Air Pollution and Incidence of Cardiovascular Events in Women*, 356 NEW ENG. J. MED. 447, 456–57 (2007); *see* Douglas W. Dockery & Peter H. Stone, *Cardiovascular Risks from Fine Particulate Air Pollution*, 356 NEW ENG. J. MED. 511, 511 (2007) (noting approvingly updated results and methodological improvements in Miller et al.).

room visits per year: 246 emergency room visits for asthma, 61 hospital admissions for respiratory causes, and several hospital admissions for cardiovascular causes.[27] Additionally, according to EPA, the Rule will prevent 1,300 lost work days and 56,000 missed school days per year.[28]

Researchers have found a significant association between air pollution and the risk of heart attacks.[29] Numerous studies link both ozone and PM air pollution to increased hospitalization for cardiovascular disease, strokes, and congestive heart failure.[30] Exposure to PM also increases the risk of blood clots[31] and affects

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[27] REGULATORY IMPACT ANALYSIS, *supra* note 7, at 5-21.

[28] *Id.*

[29] Daniela D'Ippoliti et al., *Air Pollution and Myocardial Infarction in Rome: A Case-Crossover Analysis*, 14 EPIDEMIOLOGY 528, 528 (2003); David Q. Rich et al., *Triggering of Transmural Infarctions, But Not Nontransmural Infarctions, by Ambient Fine Particles*, 118 ENVTL. HEALTH PERSP. 1229 (2010); Antonella Zanobetti & Joel Schwartz, *The Effect of Particulate Air Pollution on Emergency Admissions for Myocardial Infarction: A Multicity Case-Crossover Analysis*, 113 ENVTL. HEALTH PERSP. 978, 980 (2005).

[30] *See, e.g.*, Francesca Dominici et al., *Fine Particulate Air Pollution and Hospital Admission for Cardiovascular and Respiratory Diseases*, 295 J. AM. MED. ASS'N 1127, 1133 (2006); William S. Linn et al., *Air Pollution and Daily Hospital Admissions in Metropolitan Los Angeles*, 108 ENVTL. HEALTH PERSP. 427, 427 (2000); Lynda D. Lisabeth et al., *Ambient Air Pollution and Risk for Ischemic Stroke and Transient Ischemic Attack*, 64 ANNALS NEUROLOGY 53, 53–59 (2008); Kristi B. Metzger et al., *Ambient Air Pollution and Cardiovascular Emergency Department Visits*, 15 EPIDEMIOLOGY 46, 55 (2004); Tsai et al., *supra* note 5, at 26; Bruce Urch et al., *Relative Contributions of $PM_{2.5}$ Chemical Constituents to Acute Arterial Vasoconstriction in Humans*, 16 INHALATION TOXICOLOGY 345, 345 (2004); Gregory A. Wellenius et al., *Particulate Air Pollution and Hospital Admissions for Congestive Heart Failure in Seven United States Cities*, 97 AM. J. OF CARDIOLOGY 404 (2006).

blood vessel reactivity,[32] reducing the amount of blood that reaches the heart and the brain.[33] Further, PM may inhibit the body's ability to vary its heart rate in response to environmental or situational changes.[34]

Exposure to air pollution can inflict significant damage on the lungs[35] and reduce lung function.[36] PM can induce inflammation of lung tissue in healthy adults.[37] Ambient concentrations of ozone and PM are associated with increased

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[31] Andrea Baccarelli et al., *Exposure to Particulate Air Pollution and Risk of Deep Vein Thrombosis*, 168 ARCHIVES INTERNAL MED. 920, 926 (2008); Andrew J. Ghio et al., *Exposure to Concentrated Ambient Air Particles Alters Hematologic Indices in Humans*, 15 INHALATION TOXICOLOGY 1465, 1476 (2003).

[32] Urch et al., *supra* note 30, at 350–52.

[33] Robert D. Brook et al., *Inhalation of Fine Particulate Air Pollution and Ozone Causes Acute Arterial Vasoconstriction in Healthy Adults*, 105 CIRCULATION 1534, 1535 (2002).

[34] Yuh-Chin T. Huang et al., *The Role of Soluble Components in Ambient Fine Particles-Induced Changes in Human Lungs and Blood*, 15 INHALATION TOXICOLOGY 327, 327 (2003).

[35] Andrew Churg et al., *Chronic Exposure to High Levels of Particulate Air Pollution and Small Airway Remodeling*, 111 ENVTL. HEALTH PERSP. 714, 717–718 (2003); Patrick L. Kinney & Morton Lippmann, *Respiratory Effects of Seasonal Exposures to Ozone and Particles*, 55 ARCHIVES ENVTL. HEALTH 210, 215 (2000); Ira B. Tager et al., *Chronic Exposure to Ambient Ozone and Lung Function in Young Adults*, 16 EPIDEMIOLOGY 751, 751 (2005).

[36] Zhanghua Chen et al., *Chronic effects of air pollution on respiratory health in Southern California children: findings from the Southern California Children's Health Study*, 7 J. OF THORACIC DISEASE 46 (2015); John M. Peters et al., *A Study of Twelve Southern California Communities with Differing Levels and Types of Air Pollution: II. Effects on Pulmonary Function*, 159 AM. J. RESPIRATORY & CRITICAL CARE MED. 759, 765–66 (1999).

[37] Andrew J. Ghio et al., *Concentrated Ambient Air Particles Induce Mild Pulmonary Inflammation in Healthy Human Volunteers*, 162 AM. J. RESPIRATORY

hospital admissions for pneumonia and chronic obstructive pulmonary disease.[38] Exposure to PM also increases emergency room visits for patients suffering from acute and chronic respiratory ailments.[39]

Scientists observe that the relationship between ozone and respiratory illness is "so well established that emergency admissions have been suggested as a surrogate measure of ozone."[40] Even in healthy adults, short-term exposure can inflame the lungs.[41] A study of hikers in New Hampshire indicated that healthy individuals were more likely to experience significant declines in lung function on days with higher ambient ozone. The study observed adverse health effects in

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& CRITICAL CARE MED. 981, 986 (2000); Huang et al., *supra* note 16; Rich et al., *supra* note 16.

[38] Mercedes Medina-Ramón et al., *The Effect of Ozone and PM_{10} on Hospital Admissions for Pneumonia and Chronic Obstructive Pulmonary Disease: A National Multicity Study*, 163 AM. J. EPIDEMIOLOGY 579, 583–84 (2006); *see also* Dominici et al., *supra* note 30, at 1133.

[39] STEPHEN VAN DEN EEDEN ET AL., PARTICULATE AIR POLLUTION AND MORBIDITY IN THE CALIFORNIA CENTRAL VALLEY: A HIGH PARTICULATE POLLUTION REGION 3–4 (2002).

[40] David V. Bates, *Ambient Ozone and Mortality*, 16 EPIDEMIOLOGY 427, 428 (2005).

[41] Ian S. Mudway & Frank J. Kelly, *An Investigation of Inhaled Ozone Dose and the Magnitude of Airway Inflammation in Healthy Adults*, 169 AM. J. RESPIRATORY & CRITICAL CARE MED. 1089, 1093 (2004); W.F. McDonnell et al., *Ozone-Induced Respiratory Symptoms: Exposure-Response Models and Association with Lung Function*, 14 EUROPEAN RESPIRATORY J. 845, 852 (1999).

healthy individuals even on days when ozone levels were well below the most recent regulatory standard for ozone.[42]

The Rule will also reduce the suffering of individuals with asthma. Asthma is a chronic respiratory disease affecting 25.7 million Americans—8.4 percent of the nation's population.[43] Asthma inflames and narrows the airways of the lungs, making it difficult for an individual to breathe.[44] EPA estimates the Rule will prevent over 67,000 asthma attacks per year.[45]

Exposure to ozone and PM is especially harmful for people with asthma.[46] People with asthma have heightened sensitivity to airway irritants, such as ozone and PM, and airway irritation leads to recurring symptoms, such as wheezing, chest tightness, shortness of breath, and coughing.[47] An asthma attack can be both

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[42] Susan Korrick et al., *Effects of Ozone and Other Pollutants on the Pulmonary Function of Adult Hikers*, 106 ENVTL. HEALTH PERSP. 93, 97–99 (1998) (reporting adverse effects from exposure to average ozone levels ranging from 0.021–0.074 ppm, below 0.075 ppm level mandated by 8-hour ozone NAAQS in 2008).

[43] LARA. J. AKINBAMI ET AL., CTRS. FOR DISEASE CONTROL AND PREVENTION, NHCS DATA BRIEF NO. 94, TRENDS IN ASTHMA PREVALENCE, HEALTH CARE USE, AND MORTALITY IN THE UNITED STATES, 2001–2010, at 1 (2012).

[44] NAT'L HEART, LUNG, AND BLOOD INST., NAT'L INSTS. OF HEALTH, PUB. NO. 09-7429, AT A GLANCE: ASTHMA 1 (2009).

[45] REGULATORY IMPACT ANALYSIS, *supra* note 7, at 5-21.

[46] Helene Desqueyroux et al., *Short-Term Effects of Low-Level Air Pollution on Respiratory Health of Adults Suffering from Moderate to Severe Asthma*, 89 ENVTL. RES. 29, 29 (2002); Janneane F. Gent et al., *Association of Low-Level Ozone and Fine Particles with Respiratory Symptoms in Children with Asthma*, 290 J. AM. MED. ASS'N 1859, 1859 (2003).

[47] NAT'L HEART, LUNG, AND BLOOD INST., *supra* note 44, at 1.

painful and frightening, as its onset is often sudden. Left untreated, asthma can lead to permanent lung damage or fatalities.[48] Recurrent asthma exacerbations can cause permanent airway damage, and they are dangerous and often expensive.[49] Indeed, health experts maintain that air pollution is "one of the most under-appreciated contributors to asthma exacerbation."[50]

Finally, the Rule will reduce the risk of neurological illness. The smallest air pollution particles can pass through natural barriers in the lungs and nervous system and spread throughout the central nervous system. This exposure to ultra-fine PM can adversely impact the central nervous system.[51] Exposure to PM is associated with a worsening of cognitive function in older adults[52] and children.[53]

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[48] Diane E. McLean et al., *Asthma Among Homeless Children: Undercounting and Undertreating the Underserved*, 158 ARCHIVES PEDIATRICS & ADOLESCENT MED. 244, 247 (2004).

[49] *See* Susan M. Pollart et al., *Management of Acute Asthma Exacerbations*, 84 AM. FAMILY PHYSICIAN 40, 40–47 (2011) (describing symptoms and treatment strategies).

[50] George D. Thurston & David V. Bates, *Air Pollution as an Underappreciated Cause of Asthma Symptoms*, 290 J. AM. MED. ASS'N 1915, 1915 (2003); *see also* Ariel Spira-Cohen et al., *Personal Exposures to Traffic-Related Air Pollution and Acute Respiratory Health among Bronx Schoolchildren with Asthma*, 119 ENVTL. HEALTH PERSP. 559, 559, 564 (2011) (collecting studies linking PM emissions to asthma exacerbation and identifying key causal factors in relationship).

[51] Michelle L. Block & Lilian Calderón-Garcidueñas, *Air Pollution: Mechanisms of Neuroinflammation and CNS Disease*, 32 TRENDS NEUROSCIENCE 506, 506–07 (2009).

[52] *See, e.g.*, Jennifer A. Ailshire & Eileen M. Crimmins, *Fine Particulate Matter Air Pollution and Cognitive Function Among Older US Adults*, 180 AM. J. EPIDEMIOLOGY 359, 359 (2014); Melinda C. Power et al., *Traffic-Related Air*

It increases hospital admissions and the risk of developing dementia, Alzheimer's disease, and Parkinson's disease.[54] Exposure to PM has also been linked to worsening of symptoms of depression and anxiety.[55]

III. THE RULE WILL PROTECT CHILDREN, THE ELDERLY, AND OTHER VULNERABLE GROUPS

The Rule is also vitally necessary to protect vulnerable subpopulations. The adverse health effects of ozone and PM pose greater risks for certain groups, including children (eighteen years and younger), the elderly (sixty-five years and older), people with chronic cardiovascular and lung disease, and people with

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Pollution and Cognitive Function in a Cohort of Older Men, 119 ENVTL. HEALTH PERSP. 682, 682 (2011); Ulrich Ranft et al., *Long-term exposure to traffic-related particulate matter impairs cognitive function in the elderly*, 109 ENVTL. RES. 1004, 1004–05 (2009); Jennifer Weuve et al., *Exposure to Particulate Air Pollution and Cognitive Decline in Older Women*, 172 ARCHIVES INTERNAL MED. 219, 219 (2012).

[53] S. Franco Suglia et al., *Association of Black Carbon with Cognition among Children in a Prospective Birth Cohort Study*, 167 AM. J. EPIDEMIOLOGY 280, 280 (2008).

[54] *See, e.g.*, Marianthi-Anna Kiomourtzoglou et al., *Long-term $PM_{2.5}$ Exposure and Neurological Hospital Admissions in the Northeastern United States*, 124 ENVTL. HEALTH PERSP. 23, 23 (2016); Beate Ritz et al., *Traffic-Related Air Pollution and Parkinson's Disease in Denmark: A Case-Control Study*, 124 ENVTL. HEALTH PERSP. 351, 351–52 (2016); Antonella Zanobetti et al., *A national case-crossover analysis of the short-term effect of $PM_{2.5}$ on hospitalizations and mortality in subjects with diabetes and neurological conditions*, 13 ENVTL. HEALTH 38, 38 (2014).

[55] *See, e.g.*, Melinda C. Power et al., *The relation between past exposure to fine particulate air pollution and prevalent anxiety: observational cohort study*, 350 BMJ 1, 1 (2015); Yi Wang et al., *Ambient Air Pollution and Depressive Symptoms in Older Adults: Results from the MOBILIZE Boston Study*, 122 ENVTL. HEALTH PERSP. 553, 553–54 (2014).

diabetes.[56] Children are especially susceptible because their lungs are still developing[57] and because they breathe more air per pound of body weight than adults, which increases the dose of inhaled pollutants.[58] Children also spend more time outdoors and have higher activity levels than adults, which means they generally inhale greater volumes of polluted air.[59]

Ozone and PM can affect health even before birth. PM exposure during pregnancy is connected to increased risk of premature birth,[60] and PM and ozone are both connected to increased risk of lower birth weight.[61] One study found that prenatal exposure to emissions from a Pennsylvania power plant in a large region of New Jersey increased the risk of low birth weight by 6.5% and the risk of very

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[56] INTEGRATED SCIENCE ASSESSMENT FOR PM, *supra* note 2, at Ch. 8 ("Populations Susceptible to PM-Related Health Effects").

[57] Comm. on Envtl. Health, Am. Acad. of Pediatrics, *Ambient Air Pollution: Health Hazards to Children*, 114 PEDIATRICS 1699, 1699 (2004) (observing that eighty percent of alveolar function develops postnatally).

[58] *See* Kent E. Pinkerton et al., *Ozone, a Malady for All Ages*, 176 AM. J. RESPIRATORY & CRITICAL CARE MED. 107, 107 (2007) (collecting and summarizing studies that illustrate nature of and reasons for ozone's adverse impact on lungs of children).

[59] *Id.*; *see also* Comm. on Envtl. Health, Am. Acad. of Pediatrics, *supra* note 57, at 1699.

[60] Sharon K. Sagiv et al., *A Time Series Analysis of Air Pollution and Preterm Birth in Pennsylvania, 1997–2001*, 113 ENVTL. HEALTH PERSP. 602, 605 (2005).

[61] Michelle L. Bell, *Prenatal Exposure to Fine Particulate Matter and Birth Weight: Variations by Particulate Constituents and Sources*, 21 EPIDEMIOLOGY 884, 884 (2010); Muhammad T. Salam et al., *Birth Outcomes and Prenatal Exposure to Ozone, Carbon Monoxide, and Particulate Matter: Results from the Children's Health Study*, 113 ENVTL. HEALTH PERSP. 1638, 1638 (2005).

low birth weight by 17% for children born to mothers living up to twenty to thirty miles downwind from the plant.[62] Another study found that infants exposed to higher levels of ambient $PM_{2.5}$ had a higher risk of hospitalization for bronchiolitis.[63] In Canada's largest cities, ozone is associated with increased hospitalization for respiratory problems in babies under one month old.[64]

The adverse effects of high ozone exposure can stay with children for life. A five-year study tracking 3,500 students in Southern California found that children who played team sports in areas with high daytime ozone concentrations had a greater risk of developing asthma.[65] Asthmatic children who are exposed to air pollution also have increased hospitalization rates, more severe asthma attacks, and decreased pulmonary function.[66] A study of 255 college freshmen similarly found

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[62] Muzhe Yang et al., *The Impact of Prenatal Exposure to Power Plant Emissions on Birth Weight: Evidence from a Pennsylvania Power Plant Located Upwind of New Jersey*, 36 J. POL'Y ANALYSIS & MGMT. 493, 557–83 (2017).

[63] Catherine Karr et al., *Effects of Subchronic Exposure to Ambient Air Pollutants on Infant Bronchiolitis*, 165 AM. J. EPIDEMIOLOGY 553, 557 (2007) (finding a relationship between subchronic and chronic exposure to $PM_{2.5}$ and an increased risk of bronchiolitis in infants).

[64] Richard T. Burnett et al., *Association Between Ozone and Hospitalization for Acute Respiratory Diseases in Children Less than 2 Years of Age*, 153 AM. J. EPIDEMIOLOGY 444, 449 (2001); Robert E. Dales et al., *Gaseous Air Pollutants and Hospitalization for Respiratory Disease in the Neonatal Period*, 114 ENVTL. HEALTH PERSP. 1751, 1754 (2006).

[65] Rob McConnell et al., *Asthma in Exercising Children Exposed to Ozone: A Cohort Study*, 359 LANCET 386, 389–91 (2002).

[66] Toby C. Lewis et al., *Air Pollution-Associated Changes in Lung Function Among Asthmatic Children in Detroit*, 113 ENVTL. HEALTH PERSP. 1068, 1073

that students who grew up in areas with more ambient ozone had decreased lung function, a risk factor for lung disease later in life.[67]

Older adults are more susceptible to the adverse health effects of air pollution because they have a higher prevalence of pre-existing illness and the aging process has contributed to their sensitivity.[68] Healthy elderly adults can experience significant decreases in heart rate variability following PM exposure, which may induce adverse cardiovascular events.[69] PM can trigger hospitalization for congestive heart failure among the elderly,[70] and low-level ozone exposure increases emergency room visits for respiratory illnesses among older populations.[71]

(2005); George D. Thurston et al., *Summertime Haze Air Pollution and Children with Asthma*, 155 AM. J. RESPIRATORY & CRITICAL CARE MED. 654, 659–60 (1997); Leonardo Trasande & George D. Thurston, *The Role of Air Pollution in Asthma and Other Pediatric Morbidities*, 115 J. ALLERGY & CLINICAL IMMUNOLOGY 689, 691–96 (2005).

[67] Tager et al., *supra* note 3535, at 756–58.

[68] INTEGRATED SCIENCE ASSESSMENT FOR PM, *supra* note 2, at 8-3.

[69] R.B. Devlin et al., *Elderly Humans Exposed to Concentrated Air Pollution Particles Have Decreased Heart Rate Variability*, 21 EUROPEAN RESPIRATORY J. 76s, 79s (2003).

[70] Gregory Wellenius et al., *Particulate Air Pollution and the Rate of Hospitalization for Congestive Heart Failure Among Medicare Beneficiaries in Pittsburgh, Pennsylvania*, 161 AM. J. EPIDEMIOLOGY 1030, 1030 (2005).

[71] Ralph J. Delfino et al., *Emergency Room Visits for Respiratory Illnesses Among the Elderly in Montreal: Association with Low Level Ozone Exposure*, 76 ENVTL. RES. 67, 75 (1998).

Also vulnerable are individuals with pre-existing medical conditions. Adults with chronic obstructive pulmonary disease are particularly sensitive to ozone exposure,[72] and patients with cystic fibrosis are at greater risk of pulmonary exacerbations and significant loss in lung function when exposed to air pollution.[73] Individuals with diabetes may experience greater adverse health burdens from exposure to air pollution due to their increased baseline cardiovascular health risks.[74] The Rule takes a critical step toward addressing the myriad adverse health effects from ozone and PM pollution that disproportionately impact vulnerable subpopulations.

CONCLUSION

An overwhelming body of science demonstrates both the harms caused by ozone and PM and the urgent need for the CSAPR Update Rule. *Amicus* ATS urges the Court to deny Petitioners' challenge and to protect the health of millions

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[72] Helene Desqueyroux et al., *Effects of Air Pollution on Adults with Chronic Obstructive Pulmonary Disease*, 6 ARCHIVES ENVTL. HEALTH 554, 554 (2002).

[73] Christopher H. Goss et al., *Effect of Ambient Air Pollution on Pulmonary Exacerbations and Lung Function in Cystic Fibrosis*, 169 AM. J. RESPIRATORY & CRITICAL CARE MED. 816, 816 (2004).

[74] Marie S. O'Neill et al., *Diabetes Enhances Vulnerability to Particulate Air Pollution-Associated Impairment in Vascular Reactivity and Endothelial Function*, 111 CIRCULATION 2913, 2918 (2005); Marie S. O'Neill et al., *Air Pollution and Inflammation in Type 2 Diabetes: A Mechanism for Susceptibility*, 64 OCCUPATIONAL & ENVTL. MED. 373, 376 (2007); Antonella Zanobetti & Joel Schwartz, *Are Diabetics More Susceptible to the Health Effects of Airborne Particles?*, 164 AM. J. RESPIRATORY & CRITICAL CARE MED. 831, 832–33 (2001).

of Americans by allowing prompt implementation of the scientifically sound and vitally necessary CSAPR Update Rule.

Dated: February 20, 2018

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CERTIFICATE OF COMPLIANCE

I hereby certify the following:

1. This brief complies with the type-volume limit of Federal Rule of Appellate Procedure 29(a)(5) because, excluding the parts of the document exempted by Federal Rule of Appellate Procedure 32(f) and Circuit Rule 32(e)(1), this document contains 6,300 words.
2. This brief also complies with the typeface and type-style requirements of Federal Rules of Appellate Procedure 32(a)(5) and 32(a)(6) because it has been prepared in a proportionally spaced typeface using Microsoft Word 2013 in 14-point Times New Roman.

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CERTIFICATE OF SERVICE

I hereby certify that on February 20, 2018, I electronically filed the foregoing brief with the Clerk of the Court for the United States Court of Appeals for the District of Columbia Circuit using the appellate CM/ECF system. I further certify that all participants in the case are registered CM/ECF users and that service will be accomplished by the appellate CM/ECF system.

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