Nos. 12-1182, 12-1183

IN THE

Supreme Court of the United States

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY, *ET AL.*,

Petitioners,

and

AMERICAN LUNG ASSOCIATION, *ET AL.*,

Petitioners,

v.

EME HOMER CITY GENERATION, L.P., *ET AL.*,

Respondents.

On Writs of Certiorari to the United States Court of Appeals for the District of Columbia Circuit

BRIEF OF *AMICUS CURIAE* AMERICAN THORACIC SOCIETY IN SUPPORT OF PETITIONERS

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INTEREST OF THE *AMICUS CURIAE*[1]

The American Thoracic Society (ATS) is an international educational and scientific organization founded in 1905 that represents more than 15,000 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 Petitioners' position because cross-border air pollution harms public health in downwind states. In light of this case's vital importance to the millions of citizens living in downwind states, *amicus* urges that this Court reverse the decision of the U.S. Court of Appeals for the D.C. Circuit and reinstate the U.S. Environmental Protection Agency's Cross State Air Pollution Rule, referred to below as the Transport Rule.

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[1] Pursuant to this Court's Rule 37.2(a), all parties were timely notified of *amicus*' intention to file this brief. Counsel for petitioners and respondents have consented to the filing of this brief and their written consent has been lodged with the Court. Pursuant to this Court's Rule 37.6, *amicus* states that this brief was not authored in whole or in part by counsel for any party and that no person or entity other than *amicus* or her counsel made a monetary contribution intended to fund the preparation or submission of this brief.

SUMMARY OF ARGUMENT

Amicus curiae submits this brief to assist the Court in understanding the importance of the public health impacts of the air pollution at issue in this case. Air pollution measurably and substantially shortens lives. Vacating EPA's Transport Rule prevents the U.S. Environmental Protection Agency (EPA) from enforcing protections against such pollution. Should the decision of the U.S. Court of Appeals for the D.C. Circuit stand, it would condemn millions of American citizens to suffer preventable harm in violation of the Clean Air Act (CAA).

The Transport Rule addresses air pollution emitted by various sources, chiefly by electricity-generating facilities. Exposure to the pollutants emitted by these sources can have serious impacts on human health, including premature death, asthma exacerbations, and increased hospitalizations for cardiovascular and respiratory illnesses. These pollutants are especially harmful to children, whose respiratory systems are developing, to the elderly, whose respiratory systems are compromised by age, and to those whose respiratory systems are compromised by disease or disability.

The D.C. Circuit grounded its decision to vacate the Transport Rule upon a concern for "unnecessary over-control" of air pollution. In so doing, the D.C. Circuit cast aside EPA's carefully calibrated rule, which rationally took into account the benefits to human health available from reducing interstate air pollution. Numerous scientific studies demonstrate 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 needless health risks caused by air pollution from upwind states. Ensuring that emissions from upwind states do not push air quality in downwind states out of compliance with national ambient air quality standards (NAAQS) is a crucial aspect of protecting Americans' air quality more generally. Accordingly, *amicus* ATS urges this Court to reverse the D.C. Circuit's decision.

ARGUMENT

I. AIR POLLUTION THAT CROSSES STATE LINES ENDANGERS THE LIVES AND HARMS THE HEALTH OF CITIZENS IN DOWNWIND STATES

This case presents issues of extraordinary importance because interstate air pollution threatens the lives and health of millions of Americans. Nitrogen oxide ($NO_{x)}$) and sulfur dioxide ($SO_{2)}$) emissions react in the atmosphere to form other dangerous pollutants, such as fine particulate matter (PM) and ground-level ozone. Plumes from electricity-generating facilities and other sources spread emissions great distances and affect PM and ozone levels in areas well beyond their place of origin, compromising public health in downwind regions. Exposure to these pollutants has long been

understood to have significant and severe health impacts, 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).[2]

An extensive body of scientific and medical research documents the link between these emissions and human health.[3] PM emissions are especially dangerous because they can bypass the body's defensive mechanisms and become lodged deep in the smaller airways of the human lung; the smallest "ultrafine" particles can enter the blood

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[2] *See also* 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); 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).

[3] *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) [hereinafter INTEGRATED SCIENCE ASSESSMENT FOR OZONE] (reviewing and summarizing scientific literature on impacts of ozone on human health).

stream and travel throughout the body.[4] These interactions increase the risk of premature death and cause or contribute to a host of respiratory and cardiopulmonary ailments, including asthma. Children, the elderly, and patients with cardiopulmonary disease are particularly susceptible to the adverse health effects of air pollution.

a. Air Pollution Shortens Lives

Exposure to air pollution kills.[5] In 2008, EPA elicited an assessment from twelve of the world's leading experts on the health effects of air pollution, which revealed substantial agreement on the likelihood of a causal connection between exposure and premature death.[6]

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[4] Günter Oberdörster et al., *Nanotoxicology: An Emerging Discipline Evolving from Studies of Ultrafine Particles*, 113 ENVTL. HEALTH PERSPS. 823, 823 (2005).

[5] U.S. ENVTL. PROT. AGENCY, EXPANDED EXPERT JUDGMENT ASSESSMENT OF THE CONCENTRATION-RESPONSE RELATIONSHIP BETWEEN $PM_{2.5}$ AND MORTALITY: FINAL REPORT, vii, 3-20 through 3-24 (2006); C. Arden Pope III et al., *Fine Particulate Air Pollution and Life Expectancies in the United States: the Role of Influential Observations*, 63 J. AIR WASTE MGMT. ASS'N 129, 131-32 (2013); Johanna Lepeule et al., *Chronic Exposure to Fine Particles and Mortality: An Extended Follow-Up of the Harvard Six Cities Study from 1974 to 2009,* 120 ENVTL. HEALTH PERSPS. 965, 968 (2012).

[6] INTEGRATED SCIENCE ASSESSMENT FOR PM at 1-14 through 1-24; *see also* 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, 2270 (2008).

When air pollution levels are high, deaths can occur immediately, or within months, by inducing heart attacks or strokes.[7] Daily PM exposure, even at low levels, can lead to premature mortality through multiple pathways.[8] Acute PM exposure increases the risk of death from respiratory and cardiovascular causes;[9] chronic exposure increases the risk of death from lung cancer and cardiovascular disease.[10]

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

[8] 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 (2006) (presenting new evidence and noting consistent evidence found by serial prior studies).

[9] 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 PERSPS. 53, 53 (2007).

[10] 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) (finding 10μg/m3 increase in $PM_{2.5}$ increased mortality risk by 8-18%); 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).

Numerous studies also demonstrate that short-term exposure to ozone can shorten life.[11]

Successive assessments of the risk of premature mortality from air pollution have shown that risk to be greater than previously believed.[12] In 2007, a groundbreaking study of 66,000 women in thirty-six U.S. cities found that an increase in ten micrograms per cubic meter[13] of $PM_{2.5}$ (particulate matter less than 2.5 micrometers in aerodynamic diameter) raised the risk of death from cardiovascular disease by seventy-six percent.[14]

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[11] *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); Jonathan I. Levy et al., *Ozone Exposure and Mortality: An Empiric Bayes Metaregression Analysis*, 16 EPIDEMIOLOGY 458, 466 (2005); Kazuhiko Ito et al., *Associations Between Ozone and Daily Mortality: Analysis and Meta-Analysis*, 16 EPIDEMIOLOGY 446, 455 (2005).

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

[13] 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.

[14] 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).

These results reflect improved data collection and methodologies and update earlier, less thorough studies that had identified a twelve percent increase in risk for every increase of ten micrograms per cubic meter.[15] A 2009 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 generally, and concluded that the risk of mortality rose by ten percent for every ten micrograms per cubic meter.[16] 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.[17]

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[15] *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. (2007)).

[16] 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).

[17] Antonella Zanobetti et al., *The Temporal Pattern of Respiratory and Heart Disease Mortality in Response to Air Pollution*, 111 ENVTL. HEALTH PERSPS. 1188, 1192 (2003); Francesca Dominici et al., *Airborne Particulate Matter and Mortality: Timescale Effects in Four US Cities*, 157 AM. J. EPIDEMIOLOGY 1055, 1062 (2003).

Ozone also shortens lives, as demonstrated by several multi-city studies,[18] including two that identified elevated risk of premature death in the northeastern U.S.[19]—the states most directly served by the Transport Rule. The National Research Council confirmed this threat from ozone in a 2008 report, in which the Council also explained that premature death caused by ozone is not restricted to people who are already in poor health.[20]

b. Air Pollution Impairs Cardiovascular and Respiratory Health

Exposure to air pollution can also cause serious illness and disease. Researchers have found a significant association between air pollution and risk

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[18] Michelle. L. Bell et al., *supra* note 10, at 442; Mercedes Medina-Ramón & Joel Schwartz, *Who Is More Vulnerable to Die from Ozone Air Pollution?*, 19 EPIDEMIOLOGY 672 (2008); K. KATSOUYANNI ET AL., HEALTH EFFECTS INST. RESEARCH REP. NO. 142, AIR POLLUTION AND HEALTH: A EUROPEAN AND NORTH AMERICAN APPROACH (APHENA) (2009).

[19] 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 (2008); Richard L. Smith et al., *Reassessing the Relationship Between Ozone and Short-Term Mortality in US Urban Communities*. 21 INHALATION TOXICOLOGY 37 (2009).

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

of heart attacks.[21] Numerous studies link both ozone and PM air pollution to increased hospitalization for cardiovascular disease, strokes, and congestive heart failure.[22] Exposure to PM also increases the risk of blood clots[23] and affects blood vessel reactivity,[24] reducing the amount of blood that reaches the heart

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[21] Antonella Zanobetti & Joel Schwartz, *The Effect of Particulate Air Pollution on Emergency Admissions for Myocardial Infarction: A Multicity Case-Crossover Analysis*, 113 ENVTL. HEALTH PERSPS. 978, 980 (2005); Daniela D'Ippoliti et al., *Air Pollution and Myocardial Infarction in Rome: A Case-Crossover Analysis*, 14 EPIDEMIOLOGY 528, 528 (2003).

[22] *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); Kristi B. Metzger et al., *Ambient Air Pollution and Cardiovascular Emergency Department Visits,* 15 EPIDEMIOLOGY 46, 55 (2004); William S. Linn et al., *Air Pollution and Daily Hospital Admissions in Metropolitan Los Angeles*, 108 ENVTL. HEALTH PERSPS. 427, 427 (2000); Tsai et al., *supra* note 6, at 26; Bruce Urch et al., *Relative Contributions of $PM_{2.5}$ Chemical Constituents to Acute Arterial Vasoconstriction in Humans*, 16 INHALATION TOXICOLOGY 345 (2004); Lynda D. Lisabeth, et al., *Ambient Air Pollution and Risk for Ischemic Stroke and Transient Ischemic Attack*, 64 ANNALS NEUROLOGY 53, 53-59 (2008).

[23] 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).

[24] Urch et al., *supra* note 21, at 350-52.

and the brain.[25] Further, PM may inhibit the body's ability to vary its heart rate in response to environmental or situational changes.[26]

Long-term exposure to air pollution can inflict significant damage on the lungs[27] and reduce lung function.[28] Ambient concentrations of ozone and PM are associated with increased hospital admissions for pneumonia and chronic obstructive pulmonary disease.[29] Exposure to PM also increases emergency

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[25] 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).

[26] 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).

[27] Ira B. Tager et al., *Chronic Exposure to Ambient Ozone and Lung Function in Young Adults*, 16 EPIDEMIOLOGY 751, 751 (2005); Andrew Churg et al., *Chronic Exposure to High Levels of Particulate Air Pollution and Small Airway Remodeling*, 111 ENVTL. HEALTH PERSPS. 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).

[28] 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).

[29] 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 21, at 1133.

room visits for patients suffering from acute and chronic respiratory ailments.[30]

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."[31] Even in healthy adults, short-term exposure can inflame the lungs and cause immediate discomfort.[32] 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 even on days when ozone levels were well below the most recent regulatory standard for ozone.[33] PM can also induce inflammation of lung

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

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

[32] 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).

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

tissue in healthy adults.[34] Recent research has also found a strong and consistent correlation between adult diabetes and air pollution, suggesting that PM is a risk factor for diabetes.[35]

c. Air Pollution Exacerbates Asthma

Asthma is a chronic respiratory disease affecting 25.7 million Americans—8.4 percent of the nation.[36] Asthma inflames and narrows the airways of the lungs, making it difficult for an individual to breathe.[37] People with asthma have heightened sensitivity to airway irritants, such as PM and ozone, and airway irritation leads to recurring symptoms, such as wheezing, chest tightness, shortness of breath, and coughing.[38] An asthma attack can be both painful and frightening, as its onset is often sudden. Left untreated, asthma can lead to

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[34] Andrew J. Ghio et al., *Concentrated Ambient Air Particles Induce Mild Pulmonary Inflammation in Healthy Human Volunteers*, 162 AM. J. RESPIRATORY & CRITICAL CARE MED. 981, 986 (2000).

[35] John F. Pearson et al., *Association Between Fine Particulate Matter and Diabetes Prevalence in the U.S.*, 33 DIABETES CARE 2196 (2010).

[36] 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).

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

[38] *Id.*

permanent lung damage or fatalities.[39] Exposure to PM and ozone is especially harmful for people with asthma.[40] Indeed, health experts maintain that air pollution is "one of the most under-appreciated contributors to asthma exacerbation."[41] Recurrent asthma exacerbations can cause permanent airway damage, and, as well as being inconvenient, they are dangerous and often expensive.[42]

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

[40] 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); 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).

[41] 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 PERSPS. 559, 559, 564 (2011) (collecting studies linking PM emissions to asthma exacerbation and identifying key causal factors in relationship).

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

d. Air Pollution Increases Health Risks for Vulnerable Subpopulations Such as Children

The adverse health effects of air pollution pose greater risks for certain populations, including children (18 years and younger), the elderly (65 years and older), people with chronic cardiovascular and lung disease, and people with diabetes.[43] Children are especially susceptible because their lungs are still developing,[44] and because they breathe more air per pound of body weight than adults, which increases the dose of inhaled pollutants.[45] Children also spend more time outdoors and have higher activity levels than adults, which means they generally inhale greater volumes of polluted air.[46]

Air pollution can affect health even before birth, as PM exposure during pregnancy is linked to

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

[44] 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 post-natally).

[45] *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).

[46] *Id.*; *see also* Comm. on Envtl. Health, Am. Acad. of Pediatrics, *supra* note 43 at 1699.

increased risk of premature birth[47] and PM and ozone are linked to increased risk of lower birth weight.[48] One study found that infants faced an increased risk of bronchiolitis for every increase of ten micrograms per cubic meter in $PM_{2.5}$ in the ambient air.[49] In Canada's largest cities, ozone is associated with increased hospitalization for respiratory problems in babies under one month old.[50]

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

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

[48] Michelle L. Bell, *Prenatal Exposure to Fine Particulate Matter and Birth Weight: Variations by Particulate Constituents and Sources*, 21 EPIDEMIOLOGY 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 PERSPS. 1638 (2005).

[49] Catherine Karr et al., *Effects of Subchronic Exposure to Ambient Air Pollutants on Infant Bronchiolitis*, 165 AM. J. EPIDEMIOLOGY 553, 557 (2007).

[50] Robert E. Dales et al., *Gaseous Air Pollutants and Hospitalization for Respiratory Disease in the Neonatal Period*, 114 ENVTL. HEALTH PERSPS. 1751, 1754 (2006); 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).

developing asthma.[51] Asthmatic children also have increased hospitalization rates, more severe asthma attacks, and decreased pulmonary function when exposed to air pollution.[52] A study of 255 college freshmen similarly found that students who grew up in areas with more ambient ozone had decreased lung function, a risk factor for lung disease later in life.[53]

Older adults are 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.[54] Healthy elderly adults can experience significant decreases in heart rate variability following PM exposure, which may induce adverse cardiovascular events.[55] PM can trigger hospitalization for

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

[52] 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); Toby C. Lewis et al., *Air Pollution-Associated Changes in Lung Function Among Asthmatic Children in Detroit*, 113 ENVTL. HEALTH PERSPS. 1068, 1073 (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).

[53] Tager et al., *supra* note 26 at 756-58.

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

[55] 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).

congestive heart failure among the elderly,[56] and low-level ozone exposure increases emergency room visits for respiratory illnesses among older populations.[57]

Also vulnerable are non-elderly individuals with pre-existing medical conditions. Adults with chronic obstructive pulmonary disease are particularly sensitive to ozone exposure,[58] and patients with cystic fibrosis are at greater risk of pulmonary exacerbations and significant loss in lung function when exposed to air pollution.[59] Individuals with diabetes are especially sensitive to air pollution as well, in particular because of increased risks from pollution-associated cardiovascular events.[60]

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[56] 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).

[57] 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).

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

[59] 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).

[60] 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); 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);

II. SIGNIFICANT PUBLIC HEALTH BENEFITS WILL RESULT FROM EPA'S IMPLEMENTATION OF THE TRANSPORT RULE

Imposing the air pollution restrictions embodied in the Transport Rule would reduce precursor emissions substantially, thereby removing a significant amount of PM and ozone from the ambient air downwind. Numerous studies demonstrate that decreases in air pollution, like PM and ozone, improve human health and increase average life expectancy.[61] Reduced exposure is associated with reduced mortality from various causes, including cardiovascular disease and lung cancer.[62] Even incremental reductions at lower

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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).

[61] *See, e.g.*, Joel Schwartz et al., *The Effect of Dose and Timing of Dose on the Association between Airborne Particles and Survival*, 116 ENVTL. HEALTH PERSPS. 64, 68 (2008) (finding no evidence of a threshold in the association between $PM_{2.5}$ exposure and risk of death, suggesting efforts to reduce PM as low as feasible are most effective way to improve public health); 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).

[62] Francine Laden et al., *Reduction in Fine Particulate Air Pollution and Mortality: Extended Follow-Up for the Harvard*

concentrations can save lives.[63] In 2009, researchers compared data on PM pollution and life expectancy in fifty-one U.S. cities between 1980 and 2000.[64] After controlling for socioeconomic, demographic, and lifestyle factors like smoking, the study revealed that decreasing $PM_{2.5}$ by ten micrograms per cubic meter could increase life expectancy by between six months and two years. 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. Other studies also show that limiting air pollution can produce substantial improvements in public health in a short period of time.[65] Decreases in long-term exposure reduce mortality rates to a

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Six Cities Study, 173 AM. J. RESPIRATORY & CRITICAL CARE MED. 667, 668-69 (2006).

[63] *Id.*

[64] 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).

[65] Antonella Zanobetti & Joel Schwartz, *The Effect of Fine and Coarse Particulate Air Pollution on Mortality: A National Analysis*, 117 ENVTL. HEALTH PERSPS. 898, 902 (2009); 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); Antonella Zanobetti et al., *Particulate Air Pollution and Survival in a COPD Cohort*, 7 ENVTL. HEALTH 48, 55-56 (2008); 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).

greater extent than previously believed.[66]

EPA has estimated that the Transport Rule would prevent between 13,000 and 34,000 premature deaths annually, measured against a state of affairs in which the Clean Air Interstate Rule (CAIR) does not govern interstate air pollution. Federal Implementation Plans: Interstate Transport of Fine Particulate Matter and Ozone and Correction of SIP Approvals, 76 Fed. Reg. 48,208, 48,309 (Aug. 8, 2011). Measured against the framework established by CAIR, it is estimated that the Transport Rule—a tougher approach than CAIR—would prevent an additional 2,550 to 6,560 premature deaths annually. Response of Intervenors American Lung Ass'n, Clean Air Council, Environmental Defense Fund, Natural Resources Defense Council, & The Sierra Club in Opposition to the Motion of Southwestern Public Service Co. for a Partial Stay of the Transport Rule Ex. 3, at 12-13, *EME Homer City Generation, L.P. v.*

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[66] *See* Roman et al., *supra* note 5, at 2268. 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) ("evidence- 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).

EPA, 696 F.3d 7 (D.C. Cir. 2011) (No. 11-1302) (Declaration of David Schoengold) [hereinafter Schoengold Declaration].

III. THE TRANSPORT RULE IS NECESSARY TO IMPROVE AMERICANS' AIR QUALITY AND HEALTH WITHOUT DELAY

Respondents make several spurious points in support of their contention that the Transport Rule is not necessary for achieving reductions in harmful air pollution. Respondents suggest—wrongly—that the CAIR framework currently in place is sufficient to achieve attainment in downwind states as required under the Clean Air Act. Br. in Opp'n of Indust. & Labor Resp'ts at 30-31. Respondents also point out—wrongly again—that EPA "design value" data show that air pollution concentrations are declining even without implementation of the Transport Rule. *Id.* at 31.

a. Without the Transport Rule, EPA Will Have No Workable Regulatory Framework for Implementing the Clean Air Act's Good Neighbor Provision

CAIR was to be a comprehensive regulatory framework for implementing the CAA's requirement that upwind states act as "good neighbors" to downwind states by limiting cross border air pollution. Clean Air Interstate Rule, 70 Fed. Reg. 25,162, 25,170 (May 12, 2005). Its requirements

reflect 1997 NAAQS for both PM and ozone, *id.* at 25,168, both of which have since been superseded by more protective standards. *See* National Air Quality Standards for Ozone, 73 Fed. Reg. 16,436, 16,471 (Mar. 27, 2008) ("the current [ozone] standard . . . does not provide sufficient protection"); National Air Quality Standards for Particulate Matter, 71 Fed. Reg. 61,144, 61,155 (Oct. 17, 2006) ("the available information clearly calls into question the adequacy of the current suite of $PM_{2.5}$ standards and provides strong support for revising the current suite of $PM_{2.5}$ standards to provide increased public health protection."). In 2008, the D.C. Circuit found "more than several fatal flaws in [CAIR]." *North Carolina v. EPA*, 531 F.3d 896, 901 (D.C. Cir. 2008). Rather than vacate CAIR, however, the D.C. Circuit limited its life, making it a stopgap that will cease to operate once EPA issues a regulatory replacement. *North Carolina v. EPA*, 550 F.3d 1176, 1178 (D.C. Cir. 2008). EPA has sought to replace CAIR with the Transport Rule, which would reduce air pollution and improve public health to a greater degree than CAIR. Schoengold Declaration at 12-13; 76 Fed. Reg. at 48, 209.

The D.C. Circuit's decision to vacate the Transport Rule effectively guarantees that millions of Americans will spend years needlessly breathing more heavily polluted air and suffering the predictable health consequences. This guarantee takes two forms. First, as EPA has explained, the decision effectively delays those emission reductions that upwind states must undertake in order for downwind states to attain the NAAQS—particularly

for ozone and PM. EPA Pet. at 29-30. And second, by instructing EPA to craft yet another novel replacement for CAIR, the decision ensures that the weaker and flawed CAIR will remain in place for the months and years EPA will require to comply with that instruction. Each year that CAIR remains in place will see an estimated 2,550 to 6,560 more premature deaths than would occur under the Transport Rule. Schoengold Declaration at 12-13.

b. Air Pollution Concentrations Are Rising Once Again

EPA regularly publishes air pollution "design values"—data reflecting official concentrations of particular pollutants, such as PM and ozone. These data report concentrations of pollution based on community monitors, and those concentrations, in turn, reflect a host of factors, including emissions from anthropogenic and natural sources and the impact of weather patterns and temperature over each three-year period studied.[67] Design values for most U.S. regions fell from the 2007-2009 period to the 2009-2011 period, but, as evident from EPA's 2010-2012 design values, ozone levels have risen

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[67] *See generally* OFFICE OF AIR QUALITY PLANNING & STANDARDS EMISSIONS, MONITORING, AND ANALYSIS DIVISION, U.S. ENVTL. PROT. AGENCY, GUIDANCE ON THE USE OF MODELS AND OTHER ANALYSES IN ATTAINMENT DEMONSTRATIONS FOR THE 8-HOUR OZONE NAAQS, EPA-454/R-05-002 (2005) (describing EPA's approach to air quality monitoring).

sharply.[68] For example, in Washington, D.C. (a nonattainment area for ozone), concentrations of ozone have increased nine percent since 2009.[69]

These recent EPA data show that air pollution concentrations can rise as well as fall under CAIR and so belie Respondents' contention that EPA has failed to demonstrate why the Transport Rule is necessary. *See* Br. in Opp'n of Indust. & Labor Resp'ts 30-31. Respondents ask this Court to look closely at the EPA data showing an earlier dip in pollution concentrations, but Respondents ignore EPA's more recent data, which show a significant

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[68] U.S. Envtl. Prot. Agency, Design Values—Period Ending 2012, Ozone Detailed Information, tbl. 3a, *available at* http://www.epa.gov/airtrends/values.html (scroll to row "2012" under "Design Value period ending" then click on "Ozone Detailed Information" under "Design Value Reports"). Notably, the higher ambient temperatures that likely contributed to this rise in 2012 are expected to persist—and continue increasing—in future years. Noah S. Diffenbaugh & Martin Scherer, *Likelihood of July 2012 U.S. Temperatures in Preindustrial and Current Forcing Regimes*, at s6, s8-s9, *and* Thomas R. Knutson et al., *The Extreme March-May 2012 Warm Anomaly over the Eastern United States: Global Context and Multimodel Trend Analysis*, at s13, s16, *in Explaining Extreme Events of 2012 from a Climate Perspective*, 94 BULL. AM. METEOROLOGICAL SOC'Y (SPECIAL SUPP.) (Thomas C. Peterson et al. eds., 2013), *available at* http://www.ametsoc.org/2012extremeeventsclimate.pdf. This warming trend, with its promise of higher rates of ozone, makes it all the more pressing to reduce ozone precursors below levels currently set by CAIR.

[69] U.S. Envtl. Prot. Agency, Design Values—Period Ending 2012, Ozone Detailed Information, tbl. 3a.

rise in ozone concentrations.[70] Contrary to Respondents' characterization of air pollution trends, EPA is right to push for implementation of the Transport Rule, because the problem of air pollution that harms and sometimes kills Americans urgently needs the stronger solution the Transport Rule provides.

CONCLUSION

For the foregoing reasons, *amicus* ATS urges this Court to reverse the D.C. Circuit's decision in order to protect the health of millions of Americans.

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[70] U.S. Envtl. Prot. Agency, Design Values—Period Ending 2012, Ozone Detailed Information, tbl. 3a.