ORAL ARGUMENT NOT YET SCHEDULED

No. 16-1127 and consolidated cases

UNITED STATES COURT OF APPEALS
FOR THE DISTRICT OF COLUMBIA CIRCUIT

MURRAY ENERGY CORPORATION, et al.,

Petitioners,

v.

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY,

Respondent.

On Petitions for Review of a Final Rule of the
United States Environmental Protection Agency

BRIEF OF THE AMERICAN THORACIC SOCIETY AS *AMICI CURIAE* IN SUPPORT OF RESPONDENT AND SUPPORTING AFFIRMANCE OF AGENCY RULE

Dated: January 25, 2017

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CERTIFICATE AS TO PARTIES, RULINGS, AND RELATED CASES

Pursuant to D.C. Circuit Rule 28(a)(1) and Fed. R. App. P. 26.1, counsel for *Amici Curiae*, the American Thoracic Society, certifies as follows:

A. Parties and *Amici*. Except for the proposed Amicus identified here, all parties, intervenors, and amici appearing in this Court are listed or referenced in the Brief for Respondent United States Environmental Protection Agency ("EPA") (filed January 18, 2017). EPA's brief references and supplements the list provided in the Petitioners' Opening Brief (filed November 18, 2016). The proposed amicus is American Thoracic Society.

B. Rulings Under Review. This case concerns petitions for review of EPA's Final Rule, "Supplemental Finding That It Is Appropriate and Necessary to Regulate Hazardous Air Pollutants From Coal- and Oil-Fired Electric Utility Steam Generating Units," 81 Fed. Reg. 24,420 (Apr. 25, 2016).

C. Related Cases. *White Stallion Energy Center, LLC v. EPA*, 748 F.3d 1222 (D.C. Cir. 2014), *rev'd Michigan v. EPA*, 135 S. Ct. 2699 (2015). This Court has ordered that this case be scheduled for argument on the same day and before the same panel as *ARIPPA v. EPA*, No. 15-1180.

Dated: January 25, 2017 Respectfully submitted,

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CORPORATE DISCLOSURE STATEMENT

Pursuant to D.C. Circuit Rule 26.1 and Federal Rule of Appellate Procedure 26.1, amicus curiae states the following: The American Thoracic Society has no parent companies and no publicly held company has a 10% or greater ownership in the American Thoracic Society. The American Thoracic Society is an incorporated organization of medical and health professionals with an interest in avoiding death and disease from, *inter alia*, air pollution.

Dated: January 25, 2017 Respectfully submitted,

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SEPARATE AMICUS CURIAE BRIEF

Pursuant to D.C. Circuit Rule 29(d), counsel for amicus curiae American Thoracic Society certifies that a separate brief is necessary for its presentation to this Court due to the American Thoracic Society's narrow focus and unique expertise on science and health effects.

Accordingly, amicus curiae, through counsel, certifies that filing a joint brief would not be practicable.

Dated: January 25, 2017

Respectfully submitted,

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STATEMENT OF IDENTITY OF *AMICUS CURIAE*, INTERESTS IN THE CASE, SOURCE OF AUTHORITY TO FILE, AND AUTHORSHIP AND FUNDING OF BRIEF

The American Thoracic Society is an international, nonprofit, nonpartisan organization with more than 15,000 members dedicated to improving the health and well-being of patients suffering from respiratory related diseases through research, clinical care, and advocacy for cleaner air. The Society participated as an *amicus* in cases related to this one, *i.e.*, *White Stallion Energy Ctr., LLC v. EPA.*, 748 F.3d 1222 (D.C. Cir. 2014), and *Michigan v. EPA*, 135 S. Ct. 2699 (2015). The Society's members are concerned that the medical and scientific literature strongly establishes the need to control the emissions at issue in the rule before this Court. Emissions from coal- and oil-fired power plants cause and contribute to avoidable death and disease in exposed populations and their reduction will have measurable public health benefits.

Fed. R. App. P. 29(a) and Circuit Rule 29 govern the American Thoracic Society's authority to file. Undersigned counsel e-mailed the list of counsel on the CM/ECF docket for this case and asked whether their clients opposed this amicus brief. Undersigned counsel received no objection to the American Thoracic Society's participation as amicus. Accordingly, upon information and belief, this motion is unopposed.

No party's counsel authored the brief in whole or in part and no party, party's counsel, or person—other than the amicus curiae, its members, or its counsel—contributed money intended to fund preparing or submitting this brief.

GLOSSARY

COPD	Chronic Obstructive Pulmonary Disease
EPA	Environmental Protection Agency
$\mu g/m^3$	Micrograms per cubic meter
NO_x	Nitrogen Oxides
NO_2	Nitrogen Dioxide
PM	Particulate Matter
$PM_{2.5}$	Fine PM: PM less than or equal to 2.5 microns in diameter
SO_2	Sulfur Dioxide

STATUTES AND REGULATIONS

Applicable statutory and regulatory provisions are contained in the Brief for Respondent.

SUMMARY OF ARGUMENT

Amicus curiae submits this brief to assist the Court in understanding the reach of the significant public health risks from hazardous air pollutant and associated emissions from power plants, the regulation of which is at issue in this case. Public health risks caused by hazardous air pollutant and associated emissions from power plants include premature death, disease, abnormal brain and lung development in children, increased hospitalization and medication requirements, and lost workdays. Constituents of these emissions travel globally. Power plants emit acid gas, metals including mercury, lead, arsenic, cadmium, nickel, and chromium, and particulate matter that can penetrate deep into human lungs. All humans are susceptible to adverse health effects from these emissions, but pregnant women, fetuses, infants, children, elderly people, and people with preexisting health conditions are especially vulnerable.

Amicus curiae American Thoracic Society supports EPA's efforts to protect the public from health problems arising from hazardous air pollutants and associated emissions from power plants, including birth defects, disease, and premature death. EPA's regulation of these emissions is urgently needed, and necessary to protect public health. Accordingly, *amicus* American Thoracic Society urges this Court to affirm EPA's decision.

ARGUMENT

I. COAL- AND OIL-FIRED POWER PLANT EMISSIONS INCREASE RISKS OF DEATH AND DISEASE.

Coal- and oil-fired power plants ("power plants") emit pollutants that endanger the lives and health of U.S. citizens, including dioxins, formaldehyde, radium, benzene, acid gases, metals, and other hazardous pollutants. These emissions include complex mixtures of hazardous substances such as acid gases, carcinogenic toxins, mercury and other metals, and airborne particles. Power plant emissions contain at least 84 separate hazardous air pollutants.[1] Further, the emitted vapors contribute to the formation of other toxic gases in the atmosphere. These emissions have both local and long-range impacts, as pollutants are carried throughout the country. Impacts include premature death, disease, abnormal brain and lung development in children, increased hospitalization and medication requirements, and lost workdays. As shown below, the medical and scientific literature strongly establishes the need to control these emissions to protect human health and the environment.

[1] Environmental Health & Engineering, Inc., *Emissions of Hazardous Air Pollutants from Coal-Fired Power Plants*, 1 (2011) (citing EPA, *2002 National Emissions Inventory Data & Documentation*), *available at* http://www.lung.org/assets/documents/healthy-air/emissions-of-hazardous-air.pdf.

A. Acid Gases from Power Plants Damage Human Health.

Power plants are the largest anthropogenic source of acid gas emissions (hydrofluoric and hydrochloric acid).[2] Even at trace levels highly corrosive and water-soluble acid gases can cause irritation and tissue damage to eyes, skin, and lungs. Inhalation of acids can cause irritation and constriction of asthmatic airways.[3] Continued exposure may contribute to development of chronic airway diseases including bronchitis, asthma, and reactive airway dysfunction syndrome.[4]

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[2] *Id*. at 13.

[3] J.M. Fine et al., *The role of titratable acidity in acid aerosol-induced bronchoconstriction*, 135 Am. Review of Respiratory Disease 826 (1987), *available at* http://www.atsjournals.org/doi/abs/10.1164/arrd.1987.135.4.826#readcube-epdf; H.C. Francis et al., *Defining and investigating occupational asthma: a consensus approach*, 64 Occupational & Envtl. Med. 361 (2007), *available at* https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2078517/.

[4] S. Quirce & P. Barranco, *Cleaning Agents and Asthma*, 20 J. Investigational Allergology & Clinical Immunology 542 (2010), *available at* https://www.ncbi.nlm.nih.gov/pubmed/21313993; George D. Leikauf, *Hazardous Air Pollutants and Asthma*, 110 Envtl. Health Perspectives 505 (2002), *available at* https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1241200/; M. Medina-Ramon et al., *Asthma, chronic bronchitis, and exposure to irritant agents in occupational domestic cleaning: a nested case-control study*, 62 Occupational & Envtl. Med. 598 (2005), *available a*t http://oem.bmj.com/content/62/9/598; M.S. Shakeri et al., *Which agents cause reactive airways dysfunction syndrome (RADS)? A systematic review*, 58 Occupational Med. (Lond.) 205 (2008), *available at* http://occmed.oxfordjournals.org/content/58/3/205.full.pdf.

Hydrofluoric acid – one of the main acid gases in power plant emissions – is corrosive to the human respiratory tract and can cause severe disease.[5] Hydrogen chloride, another major acid gas emitted from power plants, rapidly converts to hydrochloric acid in the atmosphere and causes irritation and constriction of asthmatic airways.[6] The United Kingdom's Health Protection Agency reviewed the toxicology of hydrochloric acid/hydrogen chloride in 2007 and reported that acute exposure causes respiratory irritation, while chronic or repeated lower exposures cause lung function deficits and bronchial inflammation.[7]

Emission of nitrogen and sulfur-based gases from power plants contributes to formation of other strong acids in the atmosphere, including nitric acid and sulfuric acid. Susceptible populations include the young, the elderly, and those with preexisting diseases like chronic obstructive pulmonary disease (COPD) and

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[5] S. Skolnik, *Acute inhalation exposure to hydrogen fluoride*, 7 J. Occupational Envtl. Hygiene D31 (2010), *available at* http://www.tandfonline.com/doi/full/10.1080/15459621003741789; L. Tsonis et al., *Hydrofluoric acid inhalation injury*, 29 J. Burn Care & Research 852 (2008), *available at* http://www.ncbi.nlm.nih.gov/pubmed/18695605.

[6] Fine et al., *supra* note 3.

[7] S. Bull, *Hydrogen chloride/hydrochloric acid Toxicological Overview, Version 1*, Chem. Hazards & Poisons Division Headquarters, UK Health Prot. Agency (2007), *available at* https://www.gov.uk/government/uploads/system/uploads/attachment_data/file/337689/hpa_hydrogen_chloride_toxicological_overview_v1.pdf.

asthma.[8] Exposure of healthy young adults to NO_2 and oxides of nitrogen is associated with acute airway inflammation and reduced lung function.[9] Further reductions in emissions of sulfur dioxide and nitrogen oxides would substantially benefit both human health and the environment.[10]

B. Mercury from Power Plants Harms Human Health.

Coal- and oil-fired electric power plants are the largest source of anthropogenic mercury emissions in the United States.[11] Mercury emissions come in various forms, such as particulate-bound mercury and mercury in elemental or ionized forms. Microorganisms can convert ionized mercury into an organic form called methylmercury. While all chemical forms of mercury are extremely toxic to

[8] A. Faustini et al., *Short-term effects of air pollution in a cohort of patients with chronic obstructive pulmonary disease*, 23 Epidemiology 861 (2012), *available at* https://www.ncbi.nlm.nih.gov/pubmed/23018970.

[9] Maciej Strak et al., *Respiratory Health Effects of Airborne Particulate Matter: The Role of Particle Size, Composition, and Oxidative Potential – The RAPTES Project*, 120 Envtl. Health Perspectives 1183 (2012), *available at* https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3440077.

[10] Lauraine G. Chestnut & David M. Mills, *A fresh look at the benefits and costs of the US acid rain program*, 77 J. Envtl. Mgmt. 252 (2005), *available at* https://pdfs.semanticscholar.org/2247/3e62ce123eec1f7148608315ede6f5590cbe.pdf.

[11] *Mercury Study Report to Congress*, EPA (Dec. 1997), *available at* https://www.epa.gov/mercury/mercury-study-report-congress.

all cells in the human body,[12] methylmercury is a potent neurotoxin.[13] Once emitted, mercury returns to the earth in rain and snow – contaminating land and water. Elemental mercury persists in the atmosphere for up to two years and transports globally.[14] Several studies from eastern Ohio have found that nearby coal-fired power plants contribute as much as a 72% increase in mercury levels in local rainfall.[15]

Methylmercury bio-accumulates through the food chain, especially in fish.[16] High to moderate doses of methylmercury can cause debilitating health effects and, because methylmercury targets the nervous system and brain, damage from even

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[12] *Global Mercury Assessment*, United Nations Env't Programme (2002), *available at* http://www.unep.org/gc/gc22/Document/UNEP-GC22-INF3.pdf.

[13] EPA, *Human Health of Exposures to Mercury* (2002), available at https://www.epa.gov/mercury/health-effects-exposures-mercury (last visited Jan. 24, 2017).

[14] Noelle E. Selin et al., *Sources of Mercury Exposure for U.S. Seafood Consumers: Implications for Policy*, 118 Envtl. Health Perspectives 137, 138 (2010), *available at* https://ehp.niehs.nih.gov/0900811/.

[15] Gerald J. Keeler et al., *Sources of Mercury Wet Deposition in Eastern Ohio, USA*, 40 Envtl. Sci. & Tech. 5874, 5879 (2006), available at http://pubs.acs.org/doi/abs/10.1021/es060377q; Emily M. White et al., *Spatial variability of mercury wet deposition in eastern Ohio: summertime meteorological case study analysis of local source influences*, 43 Envtl. Sci. & Tech. 4946 (2009), *available at* http://www.ncbi.nlm.nih.gov/pubmed/19673290.

[16] Shigeo Ekino et al., *Minamata disease revisited: An update on the acute and chronic manifestations of methyl mercury poisoning*, 262 J. Neurological Sci. 131, 131 (2007), *available at* http://www.institute-of-mental-health.jp/thesis/pdf/thesis-02/thesis-02-18.pdf.

low doses of methylmercury can persist over a lifetime.[17] Even very low-level methylmercury exposures in adults who consume contaminated fish can result in subclinical neurobehavioral abnormalities.[18] Significant decreases in psychomotor coordination have been found in consumers of fish.[19] All forms of mercury exposure damage the kidneys, liver, and immune systems in both adults and children.[20]

C. Other Metals from Power Plants Harm Human Health.

Power plants emit particles that contain metals besides mercury, including lead, arsenic, cadmium, nickel, and chromium. Lead damages the developing nervous system. Arsenic is a carcinogen and highly toxic. Nickel and chromium are associated with an increased risk of cancer.[21] Close proximity to industrial

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[17] *Id*.; Katsuyuki Murata et al., *Delayed Brainstem Auditory Evoked Potential Latencies in 14-Year-Old Children Exposed to Methylmercury*, 144 J. Pediatrics 177 (2004), *available at* https://www.ncbi.nlm.nih.gov/pubmed/14760257.

[18] Plinio Carta et al., *Sub-clinical Neurobehavioral Abnormalities Associated with Low Level of Mercury Exposure Through Fish Consumption*, 24 NeuroToxicology 617 (2003), *available at* https://www.ncbi.nlm.nih.gov/pubmed/12900074.

[19] Ekino et al., *supra* note 16, at 131.

[20] U.S. Agency for Toxic Substances and Disease Registry, *Toxicological Profile for Mercury*, U.S. Dep't of Health & Human Serv. (Mar. 1999), *available at* https://www.atsdr.cdc.gov/toxprofiles/tp.asp?id=115&tid=24.

[21] Rachelle J. Beveridge, *Lung cancer risk associated with occupational exposure to nickel, chromium VI, and cadmium in two population-based case-control studies in Montreal*, 53 Am. J. of Indep. Med. 476 (2010), *available at* http://www.collectionscanada.gc.ca/obj/thesescanada/vol1/QMU/TC-QMU-2653.pdf; *see also* Juhua Luo et al., *Association Between Six Environmental*

facilities emitting arsenic, lead and mercury is associated with increased prevalence of autism spectrum disorders.[22] While these metals are toxic on their own, their incorporation into airborne particulates increases risks – including the risk of death – posed by their inhalation.[23]

D. Particulate Matter from Power Plants Injures People.

Power plants emit small particles less than 2.5 microns in diameter ($PM_{2.5}$), which can penetrate deep into the lungs. Power plants also emit gases such as sulfur dioxide (SO_2), oxides of nitrogen (NO_x), and organic compounds that react to form additional $PM_{2.5}$ in the atmosphere. Exposure to $PM_{2.5}$ is strongly linked to

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Chemicals and Lung Cancer Incidence in the United States, 2011 J. Envtl. & Pub. Health 1, 1, *available at* https://www.ncbi.nlm.nih.gov/pubmed/21776439.

[22] Aisha S. Dickerson et al., *Autism spectrum disorder prevalence and proximity to industrial facilities releasing arsenic, lead or mercury*. 536 Sci. of the Total Env. 245 (2015), *available at* https://www.ncbi.nlm.nih.gov/pubmed/26218563; Aisha S. Dickerson et al., *Autism spectrum disor*der prevalence and associations with air concentrations of lead, mercury, and arsenic. 188 Envtl. Monitoring and Assessment 407 (2016), *available at* https://www.ncbi.nlm.nih.gov/pubmed/27301968.

[23] Michelle L. Bell et al., *Hospital Admissions and Chemical Composition of Fine Particle Air Pollution*, 179 Am. J. Respiratory & Critical Care Med. 1115 (2009), *available at* http://www.atsjournals.org/doi/abs/10.1164/rccm.200808-1240OC; Shaowei Wu et al., *Blood Pressure Changes and Chemical Constituents of Particulate Air Pollution: Results from the Healthy Volunteer Natural Relocation (HVNR) Study*, 121 Envtl. Health Perspectives 66 (2013), *available at* https://ehp.niehs.nih.gov/1104812/; *see also* Carta et al., *supra* note 18 at 617; K. Pasanen et al., *Mortality among population with exposure to industrial air pollution containing nickel and other toxic metals*, 54 J. Occupational Envtl. Med. 583 (2012), *available at* http://www.ncbi.nlm.nih.gov/pubmed/22569477.

premature death.[24] Epidemiologic and other data associate $PM_{2.5}$ with premature mortality in infants and adults, systemic inflammation, altered vascular reactivity and cardiac rhythms, worsened asthma, chronic bronchitis, and other cardiopulmonary illnesses.[25] Chronic exposure to $PM_{2.5}$ increases the risk of dying from lung cancer and cardiovascular disease.[26] Acute exposure increases the risk of death from respiratory and cardiovascular failure.[27]

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[24] *Am. Farm Bureau Fed'n v. EPA*, 559 F.3d 512, 515-16, 527 (D.C. Cir. 2009); *Expanded Expert Judgment Assessment of the Concentration-Response Relationship Between PM2.5 Exposure and Mortality: Final Report*, Office of Air Quality Planning & Standards, EPA, 3-23, 3-24 (Sept. 21, 2006), *available at* http://www.epa.gov/ttn/ecas/regdata/Uncertainty/pm_ee_report.pdf [hereinafter Expanded Expert Judgment].

[25] *North Carolina v. Tenn. Valley Auth.*, 593 F. Supp. 2d 812, 821-22 (W.D.N.C. 2009); *see also* Expanded Expert Judgment, *supra* note 24.

[26] C. Arden Pope III et al., *Cardiovascular Mortality and Year-round Exposure to Particulate Air Pollution: Epidemiological Evidence of General Pathophysiological Pathways of Disease*, 109 Circulation 71 (2004), *available at* http://circ.ahajournals. org/content/109/1/71.full.pdf; *see also* C. Arden Pope III et al., *Lung Cancer, Cardiopulmonary Mortality, and Long-Term Exposure to Fine Particulate Air Pollution*, 297 J. Am. Med. Ass'n 1132 (2002), *available at* https://www.ncbi.nlm.nih.gov/pubmed/11879110.

[27] 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, 285 (2007), *available at* https://www.ncbi.nlm.nih.gov/pubmed/17006435; *see also* Yun-Chul Hong et al., *Effects of Air Pollutants on Acute Stroke Mortality*, 110 Envtl. Health Perspectives 187, 190 (2002), *available at* https://www.ncbi.nlm.nih.gov/pubmed/11836148; Cathryn Tonne et al., *A Case Control Analysis of Exposure to Traffic and Acute Myocardial Infarction*, 115 Envtl. Health Perspectives 53, 53 (2007), *available at* https://www.ncbi.nlm.nih.gov/pubmed/17366819.

$PM_{2.5}$ induces a number of biological processes that contribute to cardiovascular morbidity and other life-threatening diseases.[28] Systemic inflammation caused by $PM_{2.5}$ affects the vascular system[29] and can contribute to clots, heart attacks, or strokes.[30] Changes in vascular reactivity can alter the caliber

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[28] Antonella Zanobetti & Joel Schwartz, *The Effect of Particulate Air Pollution on Emergency Admissions for Myocardial Infarction: A Multicity Case-Crossover Analysis*, 113 Envtl. Health Perspectives 978 (2005), *available at* https://www.ncbi.nlm.nih.gov/pubmed/16079066; *see also* Daniela D'Ippoliti et al., *Air pollution and myocardial infarction in Rome: a case-crossover analysis*, 14 Epidemiology 528 (2003), *available at* https://www.ncbi.nlm.nih.gov/pubmed/14501267.

[29] *See, e.g.*, Robert D. Brook et al., *Air Pollution and Cardiovascular Disease: A Statement for Healthcare Professionals from the Expert Panel on Population and Prevention Science of the American Heart Association*, 109 Circulation 2655, 26632665 (2004), *available at* https://www.ncbi.nlm.nih.gov/pubmed/15173049 (describing physiological responses to pollution leading to mortality and morbidity); Jiu-Chiuan Chen & Joel Schwartz, *Metabolic Syndrome and Inflammatory Responses to Long Term Particulate Air Pollutants*, 116 Envtl. Health Perspectives 612, 616 (2008), *available at* https://www.ncbi.nlm.nih.gov/pubmed/18470293 (finding inflammatory response associated with long-term exposure to particulate matter); 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 (2000), *available at* https://www.ncbi.nlm.nih.gov/pubmed/10988117 (finding airway inflammation induced in healthy volunteers after short-term exposure to $PM_{2.5}$ indicated risk for vascular events).

[30] Andrea Baccarelli et al., *Exposure to Particulate Air Pollution and Risk of Deep Vein Thrombosis*, 168 Archives of Internal Med. 920, 926 (2008), *available at* https://www.ncbi.nlm.nih.gov/pubmed/18474755 (finding association between blood clots in legs and exposure to PM10); Andrew J. Ghio et al., *Exposure to Concentrated Ambient Air Particles Alters Hematologic Indices in Humans*, 15 Inhalation Toxicology 1465 (2003), *available at* https://www.ncbi.nlm.nih.gov/pubmed/14648359 (finding blood changes in healthy volunteers after PM exposure).

of blood vessels and affect the amount of blood that flows to organs like the heart or brain.[31] $PM_{2.5}$ can also inhibit the body's ability to vary the heart rate in response to environmental or situational changes,[32] which can result in arrhythmia, the immediate cause of death for most heart attacks.[33] In fact, studies have linked short-term increases in PM to increased hospitalization for cardiovascular

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[31] 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), *available at* https://www.ncbi.nlm.nih.gov/pubmed/11927516 (finding vasoconstriction caused by short-term inhalation of $PM_{2.5}$ reflects risk for myocardial infarction, stroke, or other cardiovascular events); Miriam Lemos et al., *Chronic Exposure to Urban Air Pollution Induces Structural Alterations in Murine Pulmonary Coronary Arteries*, 18 Inhalation Toxicology 247 (2006), *available at* https://www.ncbi.nlm.nih.gov/pubmed/22397321 (finding mice exposed to PM and other traffic pollutants developed significant thickening of arterial wall).

[32] R.B. Devlin et al., *Elderly humans exposed to concentrated air pollution particles have decreased heart rate variability*, 21 European Respiratory J. 76s (Supp. 2003), *available at* https://www.ncbi.nlm.nih.gov/pubmed/12762579; Yuh-Chin Huang et al., *The Role of Soluble Components in Ambient Fine Particles-Induced Changes in Human Lungs and Blood*, 15 Inhalation Toxicology 327 (2003), *available at* https://www.ncbi.nlm.nih.gov/pubmed/12635002.

[33] Sverre Vedal et al., *Air Pollution and Cardiac Arrhythmias in Patients with Implantable Cardioverter Defibrilators*, 16 Inhalation Toxicology 252 (2004), *available at* https://www.ncbi.nlm.nih.gov/pubmed/15204751 (finding link between SO_2 exposure and implanted defibrillator activation).

diseases.[34] $PM_{2.5}$ can also affect blood vessel reactivity,[35] and increases diastolic blood pressure.[36] Year-round exposure to PM can significantly damage the small airways of the lungs.[37] Recent studies have strengthened the conclusion that exposure to PM causes decreased lung function, even at levels below National Ambient Air Quality Standards,[38] and that improvement in air quality can improve lung function.[39]

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[34] Francesca Dominici et al., *Fine Particulate Air Pollution and Hospital Admission for Cardiovascular and Respiratory Diseases*, 295 J. Am. Med. Assoc. 1127 (2006), *available at* https://www.ncbi.nlm.nih.gov/pubmed/16522832 (finding increase in hospital admissions associated with $PM_{2.5}$); *see also* Shang-Shyue Tsai et al., *Evidence for an Association Between Air Pollution and Daily Stroke Admissions in Kaohsiung, Taiwan*, 34 Stroke 2612 (2003), *available at* https://www.ncbi.nlm.nih.gov/pubmed/14551399.

[35] Bruce Urch et al., *Relative Contributions of $PM_{2.5}$ Chemical Constituents to Acute Arterial Vasoconstriction in Humans*, 16 Inhalation Toxicology 345 (2004), *available at* https://www.ncbi.nlm.nih.gov/pubmed/15204750 (finding exposure to $PM_{2.5}$ and ozone increased blood pressure).

[36] Bruce Urch et al., *Acute Blood Pressure Responses in Healthy Adults During Controlled Air Pollution Exposures*, 113 Envtl. Health Perspectives 1052, 1052 (2005), *available at* https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1280348/.

[37] Andrew Churg et al., *Chronic Exposure to High Levels of Particulate Air Pollution and Small Airway Remodeling*, 111 Envtl. Health Perspectives 714 (2003), *available at* https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1241480/.

[38] Sara H. Downs et al., *Reduced Exposure to PM10 and Attenuated Age-Related Decline in Lung Function*, 357 New Eng. J. Med. 2338, 2346 (2007), *available at* https://www.ncbi.nlm.nih.gov/pubmed/18057336; James McCreanor et al., *Respiratory Effects of Exposure to Diesel Traffic in Persons with Asthma*, 357 New Eng. J. Med. 2348 (2007), *available at* https://www.ncbi.nlm.nih.gov/pubmed/18057337.

[39] Downs et al., *supra* note 38, at 2346 (concluding that relatively small reductions in particulate pollution could have measurable benefits for lung function).

Exposure to $PM_{2.5}$ also has non-morbid effects that are expensive, harmful, and disrupt patients' lives. It can aggravate asthma.[40] Asthma is a medical condition in which the smooth muscles of the bronchial wall tighten in response to stimulants, like allergens or pollutants, and it affects more than 8% of the U.S. population.[41] The onset is often sudden.[42] Affected children describe feeling as if they are suffocating or "smothering" and can require immediate medical

[40] Verena Morgenstern et al., *Atopic Diseases, Allergic Sensitization, and Exposure to Traffic-Related Air Pollution in Children*, 177 Am. J. Respiratory & Critical Care Med. 1331 (2008), *available at* https://www.ncbi.nlm.nih.gov/pubmed/18337595 (finding link between allergic sensitivity, including asthma, and $PM_{2.5}$ exposure).

[41] Peter B. Noble et al., *Airway smooth muscle in asthma: Linking contraction and mechanotransduction to disease pathogenesis and remodeling*, 29 Pulmonary Pharmacology & Therapeutics 96, 98 (2014), *available at* https://www.ncbi.nlm.nih.gov/pubmed/25062835; *Asthma Facts, CDC's National Asthma Control Program Grantees*, Department of Health and Human Services, Center for Disease Control, i (July 2013), *available at* https://www.cdc.gov/asthma/pdfs/asthma_facts_program_grantees.pdf.

[42] Janelle Yorke et al., *Assessment of Dyspnea in Asthma: Validation of the Dyspnea-12*, 48 J. Asthma 602 (2011), *available at* https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3149863/.

treatment.[43] Besides this painful suffering, untreated asthma can scar the lungs and bronchial tubes,[44] reducing lung function by as much as sixty percent.[45]

Reductions in $PM_{2.5}$ yield public health benefits.[46] A 2009 study explored the effect of improved air quality by comparing data on $PM_{2.5}$ pollution and life expectancy in fifty-one cities throughout the United States for two periods of time – from the late 1970s and early 1980s, and from the late 1990s to the early 2000s.[47] After controlling for socioeconomic, demographic, and social factors (like smoking), the study revealed that a decrease of 10 $\mu g/m^3$ of fine particulate matter was associated with an increase in life expectancy of six months to two years, and reductions in air pollution accounted for as much as 15% of the overall increase in

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[43] Andrew Harver et al., *Descriptors of Breathlessness in Children with Persistent Asthma*, 139 Chest J. 832, 834-35 (2011), *available at* https://www.ncbi.nlm.nih.gov/pubmed/21183610.

[44] A.M. Vignola et al., *Structural consequences of airway inflammation in asthma*, 105 J. Allergy & Clinical Immunology S514 (Supp. 2000), *available at* https://www.ncbi.nlm.nih.gov/pubmed/10669534.

[45] Kian Fan Chung et al., *International ERS/ATS guidelines on definition, evaluation and treatment of severe asthma*, 43 Eur. Respiratory J. 343 (2014), *available at* https://www.ncbi.nlm.nih.gov/pubmed/24337046.

[46] Joel Schwartz et al., *The Effect of Dose and Timing of Dose on the Association between Airborne Particles and Survival*, 116 Envtl. Health Perspectives 64, 67-68 (2008), *available at* https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2199297/ (finding no evidence of a threshold in the association between exposure to PM2.5 and the risk of death, suggesting that efforts to reduce particle concentrations as low as feasible is the most effective way to improve public health).

[47] C. Arden Pope III et al., *Fine Particulate Air Pollution and Life Expectancy in the United States*, 360 New Eng. J. Med. 371 (2009), *available at* https://www.ncbi.nlm.nih.gov/pubmed/19164188.

life expectancy seen in the study areas.[48] Other studies also show that reductions in air pollution could be expected to produce substantial improvements in public health.[49]

E. The Danger is Greater to Susceptible Populations, Including Pregnant Women and Fetuses.

Power plant emissions are especially dangerous to susceptible populations.[50] These susceptible populations include pregnant women, fetuses, infants, children, the elderly, and people with pre-existing health conditions.[51] Constituents of power

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[48] *Id.*

[49] 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), *available at* https://www.ncbi.nlm.nih.gov/pubmed/18835862 ("[O]ur findings add to a growing coherence of the literature across multiple time scales indicating that the public health benefits of reducing particle concentrations will be realized within years, not decades, of the reduction."); Antonella Zanobetti & Joel Schwartz, *The Effect of Fine and Coarse Particulate Air Pollution on Mortality: a National Analysis*, 117 Envtl. Health Perspectives 898, 902 (2009), *available at* https://www.ncbi.nlm.nih.gov/pubmed/19590680 (concluding that the strong association between particle pollution and deaths suggests that tens of thousands of early deaths per year could be avoided by reducing particle concentrations and recommending controls on power plants); Antonella Zanobetti et al., *Particulate air pollution and survival in a COPD cohort*, 7 Envtl. Health 48 (2008), *available at* https://www.ncbi.nlm.nih.gov/pubmed/18847462 (concluding that results of study heightens urgency for pollution control measures because "reductions in air pollution should be followed quickly by improvements in public health").

[50] Parinaz Poursafa & Roya Kelishadi, *What health professionals should know about the health effects of air pollution and climate change on children and pregnant mothers*, 16 Iranian J. Nursing & Midwifery Research 257 (2011), *available at* https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3249808/.

[51] *Id.*

plant emissions can travel and impose harmful effects across large distances, and can have even greater effects locally.[52] Individuals who live near emission sources bear the brunt of the most concentrated emissions and those with special susceptibilities compose a large proportion of the overall population; thus, they represent a large proportion of those exposed to these emissions. Power plant emissions disproportionately cause morbidity and mortality in susceptible populations but also change the quality of life for susceptible individuals in ways that are difficult to quantify or monetize.[53] Poor air quality changes lifestyles.[54]

Asthma is the most common chronic childhood lung disease, and affects more than 9% of all U.S. children.[55] Emissions from power plants worsen asthma.[56] Exposure of children to SO_2 is associated with active asthma and poor

[52] *Global Mercury Assessment*, *supra* note 12.

[53] Maureen R. Gwinn et al., *Meeting Report: Estimating the Benefits of Reducing Hazardous Air Pollutants – Summary of 2009 Workshop and Future Considerations*, 119 Envtl. Health Perspectives 125, 126-27 (2010), *available at* https://www.ncbi.nlm.nih.gov/pubmed/20920952.

[54] *Asthma Facts*, *supra* note 41, at i.

[55] *Asthma Facts*, *supra* note 41, at i.

[56] Committee of the Envtl. & Occupational Health Assembly of the Am. Thoracic Society, *Health effects of outdoor air pollution*, 153 Am. J. Respiratory & Critical Care Med. 3 (1996), *available at* https://www.ncbi.nlm.nih.gov/pubmed/8542133.

control of existing asthma.[57] Exposure to $PM_{2.5}$ can aggravate asthma.[58] Short-term increases in PM are linked to a rise in hospitalizations for children with asthma attacks.[59] Asthma is not the only lung disease exacerbated by power plant emissions. Individuals with other lung diseases such as cystic fibrosis[60] or COPD[61]

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[57] Leylâ Deger et al., *Active and uncontrolled asthma among children exposed to air stack emissions of sulphur dioxide from petroleum refineries in Montreal, Quebec: A cross-sectional study*, 19 Can. Respiratory J. 97 (2012), *available at* https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3373279/.

[58] Morgenstern et al., *supra* note 40.

[59] James C. Slaughter et al., *Effects of Ambient Air Pollution on Symptom Severity and Medication Use in Children with Asthma*, 91 Annals of Allergy, Asthma & Immunology 346 (Supp. 2003), *available at* https://www.ncbi.nlm.nih.gov/pubmed/14582813 ($PM_{2.5}$ associated with aggravated asthma attacks); *see also* Shao Lin et al., *Childhood asthma hospitalization and residential exposure to state route traffic*, 88 Envtl. Research 73 (2002), *available at* https://www.ncbi.nlm.nih.gov/pubmed/11908931; Gary Norris et al., *An Association Between Fine Particles and Asthma Emergency Department Visits for Children in Seattle*, 107 Envtl. Health Perspectives 489 (1999), *available at* https://www.ncbi.nlm.nih.gov/pubmed/10339450; Paige E. Tolbert et al., *Air Quality and Pediatric Emergency Room Visits for Asthma in Atlanta, Georgia*, 151 Am. J. Epidemiology 798 (2000), *available at* https://www.ncbi.nlm.nih.gov/pubmed/10965977.

[60] 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 (2004), *available at* https://www.ncbi.nlm.nih.gov/pubmed/14718248.

[61] Faustini et al., *supra* note 8.

are also negatively affected by air pollutants. Infants face a 9% greater risk of bronchiolitis for each 10 μg/m^3 increase in $PM_{2.5}$.[62]

Power plant emissions can be particularly dangerous for normal growing fetuses as well as infants and children because emissions alter both overall and organ-specific growth and development: Pollutant effects are especially prominent in the brain and nervous system and the respiratory system.[63] Exposure to polycyclic aromatic hydrocarbons (PAHs), which are emitted from power plants, is associated with smaller head size at birth and decreased height as a child grows.[64] Furthermore, exposure to power plant emissions that include SO_2 during the first trimester of pregnancy results in lower body mass in newborns.[65] Reducing or

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[62] Catherine Karr et al., *Effects of Subchronic Exposure to Ambient Air Pollutants on Infant Bronchiolitis*, 165 Am. J. Epidemiology 553, 557 (2007), *available at* https://www.ncbi.nlm.nih.gov/pubmed/17158471.

[63] Sermin Genc et al., *The Adverse Effects of Air Pollution on the Nervous System*, 2012 J. Toxicology 1 (2012), https://www.hindawi.com/journals/jt/2012/782462/.

[64] Kinga Polanska et al., *Effect of Prenatal Polycyclic Aromatic Hydrocarbons Exposure on Birth Outcomes: The Polish Mother and Child Cohort Study*, 2014 BioMed Research Int'l 408939 (2014), available at https://www.ncbi.nlm.nih.gov/pubmed/25140312.

[65] Lucijan Mohorovic, *First two months of pregnancy – critical time for preterm delivery and low birthweight caused by adverse effects of coal combustion toxics*, 80 Early Human Dev. 115 (2004), *available at* https://www.ncbi.nlm.nih.gov/pubmed/15500992.

eliminating exposure to power plant emissions results in positive effects on neurocognitive development in children.[66]

Mercury is particularly hazardous to infants and children, causing abnormal neurological development including brain damage, birth defects, diminished intelligence, and developmental delays.[67] Methylmercury can accumulate in a fetus's blood to a concentration higher than that in the mother.[68] Each year, 300,000 to 600,000 U.S. children are born with blood methylmercury levels that exceed the EPA reference dose (the acceptable oral dose of a toxic substance).[69] Those children have more mercury in their blood than will permit healthy brain development as they grow.[70] Thus, those children's capacity to see, hear, move,

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[66] Deliang Tang et al., *Molecular and Neurodevelopmental Benefits to Children of Closure of a Coal Burning Power Plant in China*, 9 PLoS One e91966 (2014), available at https://www.ncbi.nlm.nih.gov/pubmed/24647528.

[67] *Id.*; *see also* Margaret R. Karagas et al., *Evidence on the Human Health Effects of Low-Level Methylmercury Exposure*, 120 Envtl. Health Perspectives 799 (2012), *available at* http:// https://www.ncbi.nlm.nih.gov/pubmed/22275730; Leonardo Trasande et al., *Public Health and Economic Consequences of Methyl Mercury Toxicity to the Developing Brain*, 113 Envtl. Health Perspectives 590 (2005), *available at* https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1257552/.

[68] *Toxicological Profile for Mercury*, *supra* note 20, at 179.

[69] Kathryn R. Mahaffey et al., *Blood Organic Mercury and Dietary Mercury Intake: National Health and Nutrition Examination Survey, 1999 and 2000*, 112 Envtl. Health Perspectives 562, 562 (2004), *available at* https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1241922/; Trasande et al., *supra* note 67, at 590.

[70] Trasande et al., *supra* note 67.

feel, learn, and respond is compromised.[71] Reduced cognitive development can result in significant costs to society.[72] Accumulation of mercury in fish, coupled with the known developmental hazards of mercury exposure on fetal, infant, and child development, prompted both the Federal Drug Administration and EPA to advise women of childbearing age to limit consumption of fish and to check local advisories.[73]

$PM_{2.5}$ adversely impacts lung growth and development.[74] Lung function increases during childhood until twenty to twenty-five years of age, when lung

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[71] Phillip W. Davidson et al., *Neurodevelopmental Effects of Maternal Nutritional Status and Exposure to Methylmercury from Eating Fish During Pregnancy*, 29 Neurotoxicology 767 (2008), *available at* https://www.ncbi.nlm.nih.gov/pubmed/18590763; Kathryn R. Mahaffey et al., *Adult Women's Blood Mercury Concentrations Vary Regionally in the United States: Association with Patterns of Fish Consumption (NHANES 1999-2004)*, 117 Envtl. Health Perspectives 47 (2009), *available at* https://www.ncbi.nlm.nih.gov/pubmed/19165386; Mahaffey et al., *supra* note 69; Trasande et al., *supra* note 67; *see also* Brooks B. Gump et al., *Fish Consumption, Low-Level Mercury, Lipids, and Inflammatory Markers in Children*, 112 Envtl. Research 204 (2012), *available at* https://www.ncbi.nlm.nih.gov/pubmed/22030286.

[72] Trasande et al., *supra* note 67.

[73] *What You Need to Know about Mercury in Fish and Shellfish*, EPA & FDA (2004), *available at* http://www.foodprotect.org/issues/packets/2010Packet/attachments/I_006__all.pdf.

[74] *See, e.g.*, Thais Mauad et al., *Chronic Exposure to Ambient Levels of Urban Particles Affects Mouse Lung Development*, 178 Am. J. Respiratory & Critical Care Med. 721, 727 (2008), *available at* https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2556454/ (suggesting exposure to ambient levels of $PM_{2.5}$ and other urban pollutants may adversely impact lung growth and development).

function peaks at the maximal capacity to breathe.[75] A pernicious effect of exposure to damaging air pollution is reduction in this peak lung function, which represents a loss of functional reserve.[76] Lower peak lung function as an adult predisposes the subject to a greater potential effect of lung disease later in life as lung function decreases with age.[77] Children exposed to NO_2 acids and $PM_{2.5}$ suffer diminished lung function growth.[78] Children who grow up in regions with high levels of NO_2 and PM air pollution have decreased lung function as adults.[79]

These widespread, serious adverse effects that power plant emissions impose on people, and the urgent public need for reduction in hazardous air pollutants and associated emissions form a backdrop against which EPA reasonably determined that it is "appropriate and necessary" to include power plants among the categories of sources required to control emissions of hazardous air pollutants.

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[75] W. James Gauderman et al., *Association Between Air Pollution and Lung Function Growth in Southern California Children*, 162 Am. J. Respiratory & Critical Care Med. 1383, 1389 (2000), *available at* https://www.ncbi.nlm.nih.gov/pubmed/11029349; *see also* J. Dubnov et al., *Estimating the effect of air pollution from a coal-fired power station on the development of children's pulmonary function*, 103 Envtl. Research 87 (2007), *available at* https://www.ncbi.nlm.nih.gov/pubmed/16618483; B.F. Hwang et al., *Relationship between exposure to fine particulates and ozone and reduced lung function in children*, 137 Envtl. Research 382 (2015), *available at* https://www.ncbi.nlm.nih.gov/pubmed/25614339.

[76] Gauderman et al., *supra* note 75.

[77] *Id.*

[78] *Id.*

[79] *Id.*

CONCLUSION

For all the foregoing reasons, EPA's decision should be AFFIRMED and the Petitions for Review should be DISMISSED.

Dated: January 25, 2017 Respectfully submitted,

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CERTIFICATE OF COMPLIANCE WITH RULE 32(a)(7)

Pursuant to Federal Rule of Appellate Procedure 32(a)(7)(C), I certify that the foregoing brief is in 14-point, proportionately spaced, Times New Roman typeface and contains 5265 words, excluding the parts of the brief exempted by Fed. R. App. P. 32(a)(7)(B)(iii). The word processing software used to prepare this brief was Microsoft Word 2016.

Dated: January 25, 2017 /s/ Adam Babich
Adam Babich

CERTIFICATE OF SERVICE

I hereby certify that on January 25, 2017, I electronically filed the foregoing brief with the Clerk of the Court by using the appellate CM/ECF system, which will send a notice of electronic filing to all registered counsel.

Dated: January 25, 2017

/s/ Adam Babich
Adam Babich