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, *et al.*,
Respondents.

On Petitions for Review of Final Agency Action of the
United States Environmental Protection Agency
81 Fed. Reg. 24,420 (Apr. 25, 2016)

BRIEF OF ELSIE M. SUNDERLAND, JOEL D. BLUM, CELIA Y. CHEN, CHARLES T. DRISCOLL, JR., DAVID C. EVERS, PHILIPPE GRANDJEAN, DANIEL A. JAFFE, ROBERT P. MASON, AND NOELLE ECKLEY SELIN AS *AMICI CURIAE* IN SUPPORT OF RESPONDENTS

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Dated: January 25, 2017

CERTIFICATE OF COUNSEL AS TO PARTIES, RULINGS, AND RELATED CASES

Pursuant to D.C. Circuit Rule 28(a)(1) and Federal Rule of Appellate Procedure 26.1, counsel for *Amici Curiae* Elsie M. Sunderland, Joel D. Blum, Celia Y. Chen, Charles T. Driscoll, Jr., David C. Evers, Philippe Grandjean, Daniel A. Jaffe, Robert P. Mason, and Noelle Eckley Selin certifies as follows:

A. Parties and *Amici*. Except for proposed *amici* Elsie M. Sunderland, et al., 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).

B. Rulings Under Review. This case addresses petitions for review of EPA's final agency action titled, "Supplemental Finding That It Is Appropriate and Necessary to Regulate Hazardous Air Pollutants from Coal- and Oil-Fired Electric Utility Steam Generating Units," published on April 25, 2016, at 81 Fed. Reg. 24,420.

C. Related Cases. *Amici* adopt the statement of related cases set forth in the Brief for Respondent EPA.

Dated: January 25, 2017 /s/ Shaun A. Goho
Shaun A. Goho

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GLOSSARY

ADHD	Attention deficit/hyperactivity disorder
EPA	United States Environmental Protection Agency
IQ	Intelligence Quotient

CERTIFICATION REGARDING SEPARATE *AMICUS* BRIEF

Pursuant to D.C. Circuit Rule 29(d), counsel for *amici curiae* Elsie M. Sunderland, et al., certifies that a separate brief is necessary because no other brief addresses the scientific evidence supporting EPA's conclusion that there are significant benefits from regulating mercury emissions from coal-fired power plants that were not monetized in the Regulatory Impact Analysis. *Amici* provide unique expertise regarding atmospheric transport, aquatic fate, bioaccumulation, human exposures, and health outcomes associated with environmental mercury contamination. Because only *amici* submit information to the Court in their expert area, it would not be practicable to file a joint brief with experts on other issues or interests. Therefore, it is necessary to submit a separate brief.

Dated: January 25, 2017 /s/ Shaun A. Goho
Shaun A. Goho

INTERESTS OF THE AMICI CURIAE[1]

Amici are scientists who collectively represent current scientific expertise in the atmospheric transport, aquatic fate, bioaccumulation, human exposures, and health outcomes associated with environmental mercury contamination. *Amici* have a strong interest in supporting EPA's conclusion that it was appropriate and necessary to regulate power plant emissions of mercury and other air toxics under section 112 of the Clean Air Act, 42 U.S.C. § 7412, because mercury is a highly toxic, bioaccumulative, and persistent pollutant and the Rule will lead to significant reductions in the harms associated with power plant mercury emissions.

Amicus Elsie M. Sunderland is the Thomas D. Cabot Associate Professor of Environmental Science and Engineering at the John A. Paulson School of Engineering and Applied Science and the T.H. Chan School of Public Health at Harvard University. Dr. Sunderland's research focuses on the impacts of past and future releases of heavy metal and organic contaminants on human and ecological health. She has specifically researched the effects of mercury exposure from domestic and imported fish and shellfish in the United States, the global mercury cycle, the impacts of mercury discharges from rivers and sediment burial, and the levels of methylmercury in Arctic marine biota.

[1] Counsel for *amici* certifies that no counsel for a party authored this brief in whole or in part and that no person, other than *amici* or their counsel, made a monetary contribution to the preparation or submission of this brief.

Amicus Joel D. Blum is Distinguished University Professor in the Department of Earth and Environmental Sciences at the University of Michigan. His research focuses on studies of geochemical controls on the structure and function of ecosystems, and on the application of trace element and isotope geochemistry across the Earth and Environmental Sciences. Current research projects include investigations of mercury sources, transport, and fate in the atmosphere, rivers, lakes, soils, forests, aquifers, and marine and coastal ecosystems.

Amicus Celia Y. Chen is Research Professor in the Department of Biological Sciences at Dartmouth College. Dr. Chen's research has focused on the fate and effects of metal contaminants in aquatic food webs both in freshwater and estuarine ecosystems. Dr. Chen's specific research has included the bioaccumulation and transfer of mercury in marine food webs and implications for human health, factors influencing methylmercury production and bioaccumulation across multiple estuaries in the Northeast United States, and environmental and ecological factors affecting methylmercury fate in lakes and streams.

Amicus Charles T. Driscoll, Jr. is University Professor of Environmental Systems Engineering at the Department of Civil and Environmental Engineering at Syracuse University. Dr. Driscoll's research focuses on atmospheric deposition, transport, fate, bioavailability and effects of mercury. Dr. Driscoll's research is

mainly field-based and emphasizes assessing and quantifying the response of ecosystems to disturbances, including the effects of mercury deposition on forest, aquatic, and coastal ecosystems.

Amicus David C. Evers is the Executive Director and Chief Scientist at the Biodiversity Research Institute. Dr. Evers' research focuses on ecotoxicology, especially the impacts of methylmercury and oil exposure on birds, fish, and other wildlife. Dr. Evers is a conservation biologist and has testified or presented scientific results to Congressional committees, state legislative committees, and regulatory agencies. Dr. Evers's specific research includes the historic and contemporary mercury exposure and potential risk to Yellow-Billed Loons breeding in Alaska and Canada, the mercury concentrations in the Goliath Grouper of Belize, and evaluating the effectiveness of the Minamata Convention on mercury.

Amicus Philippe Grandjean is Adjunct Professor of Environmental Health at the Department of Environmental Health at the T.H. Chan School of Public Health at Harvard University. Dr. Grandjean's research focuses on the health outcomes associated with methylmercury exposures. In 2016, Dr. Grandjean received the John F. Goldsmith Award from the International Society for Environmental Epidemiology for his sustained and outstanding contributions to the knowledge and practice of environmental epidemiology. Dr. Grandjean's most recent research

has focused on brain development and immune functions in regard to exposures to environmental pollutants such as perfluorinated compounds and mercury.

Amicus Daniel A. Jaffe is Professor of Atmospheric and Environmental Chemistry at the University of Washington-Bothell. Dr. Jaffe is also an Adjunct Professor in the Department of Atmospheric Sciences at the University of Washington-Seattle. Dr. Jaffe has extensive expertise in global and regional atmospheric pollution in the Arctic and Pacific regions. His research focuses on understanding the local, regional, and global sources of pollution in the western United States, with an emphasis on mercury, ozone, and aerosols.

Amicus Robert P. Mason is Professor of Oceanography at the Department of Marine Science with a joint appointment in the Chemistry Department at the University of Connecticut. Dr. Mason's research focuses on the cycling of mercury and methylmercury in marine ecosystems. His current research focuses on the fate, transport, and transformation of trace metals, especially mercury, in aquatic systems and the atmosphere. Dr. Mason has specifically researched the methylmercury concentrations in fish from tidal waters of the Chesapeake Bay and other coastal waters, mercury emissions from natural processes and their importance in the global mercury cycle, and the influence of sediment redox status on the flux of mercury, methylmercury and other constituents from estuarine sediment.

Amicus Noelle Eckley Selin is Associate Professor at the Massachusetts Institute of Technology in the Institute for Data, Systems, and Society and the Department of Earth, Atmospheric, and Planetary Sciences. Dr. Selin's research focuses on using atmospheric chemistry modeling to inform decision-making strategies on air pollution, climate change, and toxic substances including mercury and persistent organic pollutants. Dr. Selin has specifically researched topics such as the atmospheric chemistry, modeling, and biogeochemistry of mercury, strategies to reduce mercury risks, and the sources of mercury exposure for United States seafood consumers.

PERTINENT STATUTES AND REGULATIONS

Applicable statutory and regulatory provisions are contained in Respondent EPA's addendum and the Petitioners' addendum.

SUMMARY OF ARGUMENT

Mercury is a highly toxic, bioaccumulative, and persistent pollutant. It is responsible for a variety of harmful neurological, cardiovascular, and other health effects in humans and can also harm many forms of fish and wildlife. In concluding that it was appropriate and necessary to regulate power plant emissions of mercury and other air toxics under section 112 of the Clean Air Act, 42 U.S.C. § 7412, the Environmental Protection Agency ("EPA") relied on evidence of these impacts and on the fact that power plants are the largest domestic source of

mercury emissions. In the accompanying Regulatory Impact Analysis, EPA quantified only one type of benefit from the rule: IQ losses for children born to a limited population of recreational fishers who consume freshwater fish during pregnancy from watersheds where EPA had fish tissue data. Even this analysis included several conservative assumptions. The agency recognized, however, that the other benefits of limiting power plant mercury emissions were substantial, albeit difficult to quantify.

The scientific literature confirms and strengthens EPA's conclusion that there are significant benefits from regulating power plant mercury emissions. These include reducing IQ losses beyond the narrow population examined by EPA; other neurological effects such as attention deficit/hyperactivity disorder ("ADHD"); cardiovascular impacts; other health impacts such as damage to the renal, reproductive, and hematological systems; and environmental impacts such as harm to amphibians, birds, and mammals. In addition, it is now clear that reductions in mercury emissions from power plants result in localized reductions in mercury deposition, which amplifies the benefits of reducing domestic emissions.

As a result, attempts to quantify all of these benefits have produced results orders of magnitude greater than the monetized mercury-related benefits from the Regulatory Impact Analysis. For example, one study concluded that the cumulative U.S. economy-wide benefits of the Mercury and Air Toxics Rule as a

result of the reduction in mercury-related health effects is $43 billion. Another study estimated that a 10% reduction in methylmercury exposure in the United States results in an annual benefit of $860 million.

ARGUMENT

I. MERCURY IS A DANGEROUS TOXIC METAL AND THE PRIMARY SOURCE OF EXPOSURE IN THE UNITED STATES IS EMISSIONS FROM DOMESTIC COAL-FIRED POWER PLANTS.

A. Domestic coal-fired power plants are the largest source of mercury emissions in the United States.

Methylmercury is a highly toxic, bioaccumulative, and persistent pollutant. Over the last century and a half, anthropogenic mercury emissions have dramatically increased mercury levels in the environment. It is generally recognized that the atmosphere contains 3-5 times as much mercury as it did in 1840 and that only 17% of the mercury in the surface ocean is of natural origin.[2]

The largest contemporary anthropogenic source of mercury emissions in the United States is coal-fired power plants.[3] At the time of the initial rulemaking in 2011, EPA estimated U.S. anthropogenic mercury emissions to be around 100 tons

[2] Helen M. Amos et al., *Legacy Impacts of All-time Anthropogenic Emissions on the Global Mercury Cycle*, 27 Global Biogeochemical Cycles 410, 410 (2013); Daniel R. Engstrom et al., *Atmospheric Hg Emissions from Preindustrial Gold and Silver Extraction in the Americas: A Reevaluation from Lake-Sediment Archives*, 48 Envtl. Sci. & Tech. 6533 (2014).

[3] United Nations Envt. Programme, Global Mercury Assessment at 9 (Dec. 2002), *available at* http://www.unep.org/gc/gc22/Document/UNEP-GC22-INF3.pdf.

per year, with coal-fired power plants accounting for about half of that total.[4]

Mercury emissions from power plants occur in three forms: (1) gaseous elemental mercury, (2) gaseous oxidized mercury (also called "reactive gaseous mercury"), and (3) mercury bound to particles. In general, oxidized mercury and particle-bound mercury travel shorter distances than elemental mercury before falling to the earth because they are more water-soluble and chemically reactive. A substantial portion of the mercury emitted by coal-fired power plants is in its oxidized and particle-bound forms. As a result, most mercury from power plant emissions will settle locally. For example, one study of mercury deposition in Ohio concluded that forty-two percent of the average atmospheric mercury wet deposition was traceable to a nearby coal-fired power plant.[5]

Once mercury is deposited in the aquatic environment, microbial reactions transform it into methylmercury.[6] This methylmercury is taken up by algae, which are consumed by other aquatic organisms, and then passes to predators such as piscivorous fish. As a result, methylmercury biomagnifies in fish and other aquatic organisms, increasing with each level of the food chain, and attains its highest

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[4] 76 Fed. Reg. 24,976, 25,002 (May 3, 2011).

[5] 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, 4952 (2009).

[6] Reed C. Harris et al., *Whole-Ecosystem Study Shows Rapid Fish-Mercury Response to Changes in Mercury Deposition*, 104 Proc. Nat'l Acad. Sci. U.S. 16586, 16590 (2007).

concentrations in predatory species at the top of the food chain.[7] Because methylmercury also bioaccumulates over time, species with a longer life span are at a greater risk of having elevated methylmercury levels.

Human exposure to mercury occurs primarily through consuming fish in which methylmercury has bioaccumulated.[8] In the 2012 rulemaking, EPA concluded that "up to 28 percent of watersheds were estimated to have [mercury] deposition attributable to U.S. [power plants] that contributes to potential exposures above the [level at] which there is increased risk of neurological effects in children."[9] Studies have found that people who eat large predatory fish from freshwater lakes (such as bass and pike) and the ocean (such as swordfish and tuna) can have elevated mercury levels.[10]

B. Mercury is harmful to the human body and the environment.

1. Methylmercury is a neurotoxicant.

Methylmercury is a highly toxic substance that targets the nervous system.

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[7] James G. Wiener et al., *Toxicological Significance of Mercury in Yellow Perch in the Laurentian Great Lakes Region*, 161 Envtl. Pollution 350, 354–55 (2012).

[8] United Nations Envt. Programme, *supra* n. 3, at 38.

[9] 77 Fed. Reg. 9304, 9310 (Feb. 16, 2012).

[10] Roxanne Karimi et al., *A Quantitative Synthesis of Mercury in Commercial Seafood and Implications for Exposure in the United States*, 120 Envtl. Health Persp. 1512, 1512 (2012); Mark B. Sandheinrich et al., *Ecological Risk of Methylmercury to Piscivorous Fish of the Great Lakes Region*, 20 Ecotoxicology 1577, 1577 (2011); Bruce A. Monson et al., *Spatiotemporal Trends of Mercury in Walleye and Largemouth Bass from the Laurentian Great Lakes Region*, 20 Ecotoxicology 1555, 1555 (2011).

This relationship has been understood since the identification in the Japanese city of Minamata in the 1950s of cases of "severe motor dysfunction and mental retardardation" among children whose mothers had eaten fish contaminated with very high levels of mercury.[11] Infants and fetuses are at the highest risk, both because the developing central nervous system is more sensitive to methylmercury and because methylmercury can move through the placenta and the blood-brain barrier. At the highest levels of exposure, the result might be indistinguishable from cerebral palsy and may lead to "microcephaly, hyperreflexia, and gross motor and mental impairment, sometimes associated with blindness or deafness."[12]

Even at lower levels, prenatal exposure to methyl mercury can cause neurological harm. One traditional, well-attested measure is reductions in cognitive test performance,[13] including reductions in IQ.[14] Studies cited by EPA in the preamble to the 2011 proposed rule[15] also show a connection to changes in

[11] Bruce P. Lanphear, *The Impact of Toxins on the Developing Brain*, 36 Ann. Rev. Pub. Health 211, 212 (2015).

[12] United Nations Envt. Programme, *supra* n. 3, at 38.

[13] Emily Oken et al., *Maternal Fish Intake during Pregnancy, Blood Mercury Levels, and Child Cognition at Age 3 Years in a US Cohort*, 167 Am. J. Epidemiology 1171, 1177–79 (2008).

[14] Daniel A. Axelrad et al., *Dose-Response Relationship of Prenatal Mercury Exposure and IQ: An Integrative Analysis of Epidemiologic Data*, 115 Envtl. Health Persp. 609, 613–14 (2007).

[15] 76 Fed. Reg. at 25,079.

brainstem response to auditory signals,[16] decreased performance on motor speed, attention, and language tests,[17] and impeded memory functions.[18]

Perhaps most troubling, in light of their rapidly rising rates of diagnosis, fetal methylmercury exposure is also associated with a greater likelihood of developing ADHD.[19] For example, a recent study found that in an Inuit community in northern Quebec, children with higher pre-natal mercury exposure were four times as likely to exhibit ADHD symptoms at school.[20]

The damage suffered at this early stage of development is usually much more severe than if suffered as an adult, and the effects are long-lasting. In the Faroe Islands, where inhabitants are exposed to methylmercury from the meat of pilot whales, children exposed *in utero* exhibited decreased motor function,

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[16] Katsuyuki Murata, et al., *Delayed Brainstem Auditory Evoked Potential Latencies in 14-year-old Children Exposed to Methylmercury*, 144 J. Pediatrics 177 (2004).

[17] Frodi Debes et al., *Impact of Prenatal Methylmercury Exposure on Neurobehavioral Function at Age 14 Years*, 28 Neurotoxicology & Teratology 536, 544–46 (2006).

[18] National Research Council, National Academy of Sciences, *Toxicological Effects of Methylmercury* 4 (2000), *available at* https://www.nap.edu/read/9899/chapter/1.

[19] D. K. L. Cheuk & Virginia Wong, *Attention-Deficit Hyperactivity Disorder and Blood Mercury Level: A Case-Control Study in Chinese Children*, 37 Neuropediatrics 234, 236–39 (2006).

[20] Olivier Boucher et al., *Prenatal Methylmercury, Postnatal Lead Exposure, and Evidence of Attention Deficit/Hyperactivity Disorder among Inuit Children in Arctic Québec*, 120 Envtl. Health Persp. 1456, 1459–60 (2012).

attention span, verbal abilities, memory, and other mental functions at age 7.[21] The effects were dose-dependent—the greater the exposure the greater the effect. In a study conducted in Boston, maternal hair mercury levels even lower than those measured in the Faroe Islands study were still associated with a reduction in children's cognition.[22] Recent research has extended downwards the exposure levels that may cause damage to brain development.[23]

A follow-up study of the Faroese children when they were 14 years of age showed that the mercury-associated neurological deficits had not changed.[24] A more recent study of 22-year-olds found that prenatal mercury exposure reduced the positive benefits of aerobic fitness on short-term memory and cognitive processing.[25] Cumulatively, these studies demonstrate that the effects of prenatal mercury exposure may be permanent.

Methylmercury also causes neurological harm to older children and adults.

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[21] Philippe Grandjean et al., *Cognitive Deficit in 7-Year-Old Children with Prenatal Exposure to Methylmercury*, 19 Neurotoxicology & Teratology 417, 417 (1997).

[22] Oken et al., *supra* n. 13, at 1177–79.

[23] Jordi Julvez & Philippe Grandjean, *Genetic Susceptibility to Methylmercury Developmental Neurotoxicity Matters*, 4 Frontiers Genetics 1, 1–3 (2013).

[24] Debes et al., *supra* n. 17, at 536.

[25] Youssef Oulhote et al., Advance Publication, *Aerobic Fitness and Neurocognitive Function Scores in Young Faroese Adults and Potential Modification by Prenatal Methylmercury Exposure*, Envtl. Health Persp. 1, 3 (2016), *available at* https://ehp.niehs.nih.gov/wp-content/uploads/advpub/2016/9/EHP274.acco.pdf.

The effects include symptoms such as "paresthesia, malaise, and blurred vision," and higher levels can lead to "concentric constriction of the visual field, deafness, dysarthria, ataxia, and ultimately coma and death."[26] Methylmercury's negative effects on adult cognitive functions are significant enough to outweigh the benefits of n-3 fatty acids intake from fish consumption.[27] In the rulemaking, EPA cited studies[28] which show that methylmercury can decrease several visual and motor functions, such as visual contrast sensitivity, restricted visual fields, hand-eye coordination, manual dexterity, and muscular fatigue.[29]

Methylmercury's neurological impacts might be even greater than revealed by these studies. New research demonstrates that some people are more genetically predisposed to the neurotoxic effects of methylmercury,[30] which means that studies with null findings might mask significant impacts among genetically susceptible subpopulations of the study group.[31]

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[26] United Nations Envt. Programme, *supra* n. 3, at 38.

[27] Steven C. Masley, et al., *Effect of Mercury Levels and Seafood Intake on Cognitive Function in Middle-aged Adults*, 11 Integrative Med. 32, 32 (2012).

[28] 76 Fed. Reg. at 25,079.

[29] Jean Lebel et al., *Neurotoxic Effects of Low-Level Methylmercury Contamination in the Amazonian Basin*, 79 Envtl. Res. 20, 28 (1998).

[30] Jordi Julvez et al., *Prenatal Methylmercury Exposure and Genetic Predisposition to Cognitive Deficit at Age 8 Years*, 24 Epidemiology 643, 643 (2013).

[31] Julvez & Grandjean, *supra* n. 23, at 2.

2. Methylmercury compromises cardiovascular health.

High levels of mercury in blood and tissue samples have been strongly associated with acute coronary events, coronary heart disease, and cardiovascular disease.[32] The 2000 NAS study on the health effects of methylmercury, directed by Congress in a conference report,[33] stated that even though more study was needed, it was reasonable to conclude that mercury accumulates in the heart and leads to blood pressure alterations and abnormal cardiac functions.[34] Subsequent research strengthened these findings. Looking into two longitudinal studies (the Kuopio Ischemic Heart Disease Risk Factor study and the European Community Multicenter Study on Antioxidants, Myocardial Infarction and Breast Cancer), EPA concluded that methylmercury exposure increases the risk of heart attacks.[35]

An expert panel convened in 2011 to study the health effects of methylmercury concluded that sufficient scientific evidence existed to allow incorporating cardiovascular health benefits in EPA's regulatory assessments.[36]

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[32] *See* Jyrki K. Virtanen et al., *Mercury, Fish Oils, and Risk of Acute Coronary Events and Cardiovascular Disease, Coronary Heart Disease, and All-Cause Mortality in Men in Eastern Finland*, 25 Arteriosclerosis, Thrombosis, & Vascular Biology 228, 232 (2004).

[33] 77 Fed. Reg. at 9307 (citing H.R. Conf. Rep. No 105–769, at 281–82 (1998)).

[34] National Research Council, *supra* n. 18, at 168–69.

[35] 76 Fed. Reg. at 25,080.

[36] *Id.* at 25,080–81 (citing Henry A. Roman et al., *Evaluation of the Cardiovascular Effects of Methylmercury Exposures: Current Evidence Supports Development of a Dose–Response Function for Regulatory Benefits Analysis*, 119

According to the panel, methylmercury is both directly linked to acute myocardial infarction and intermediary impacts that contribute to myocardial infarction risk.[37] These intermediary impacts include oxidative stress, atherosclerosis, heart rate variability, and to a certain degree, blood pressure and hypertension.

3. Methylmercury has additional impacts on human health.

Methylmercury also causes a variety of other adverse health impacts. Both animal studies and human epidemiological observations establish methylmercury as a possible carcinogen,[38] especially with regard to leukemia and liver cancer.[39] Methylmercury can have toxic effects on the renal, reproductive, and hematological systems.[40] There are also potential risks of chromosomal damage[41] and weakening of the immune system.[42] Finally, some studies indicate that

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Envtl. Health Persp. 607, 607 (2011)).

[37] Roman et al., *supra* n. 36, at 607.

[38] Int'l Agency for Research on Cancer, IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, Volume 58 Beryllium, Cadmium, Mercury, and Exposures in the Glass Manufacturing Industry at 277–83 (1993), *available at* http://monographs.iarc.fr/ENG/Monographs/vol58/mono58.pdf.

[39] National Research Council, *supra* n. 18, at 150–51.

[40] *Id.* at 153–54, 161–63, 173–74.

[41] Marúcia I. M. Amorim et al., *Cytogenetic Damage Related to Low Levels of Methyl Mercury Contamination in the Brazilian Amazon*, 72 Anais da Academia Brasileira de Ciências 497, 497 (2000), *available at* http://www.scielo.br/pdf/aabc/v72n4/0048.pdf.

[42] National Research Council, *supra* n. 18, at 156–61; Jennifer F. Nyland et al., *Biomarkers of Methylmercury Exposure Immunotoxicity among Fish Consumers in Amazonian Brazil*, 119 Envtl. Health Persp. 1733, 1736–38 (2011).

mercury can cause endocrine disruption[43] and diabetes.[44]

The consumption of mercury in fish also counteracts the health benefits that people would otherwise obtain from consuming seafood.[45] While fatty acids in fish oil are recommended for cardiovascular health, the mercury accumulated in the fish can offset the health effects,[46] a finding confirmed by studies conducted in Boston[47] and New York City.[48] In fact, it is difficult to consume the amount of fish recommended by the American Heart Association while simultaneously remaining below EPA's mercury reference dose because of the high levels of mercury present in most fish.[49]

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[43] Shirlee W. Tan et al., *The Endocrine Effects of Mercury in Humans and Wildlife*, 39 Critical Rev. Toxicology 228, 228 (2009).

[44] Ka He et al., *Mercury Exposure in Young Adulthood and Incidence of Diabetes Later in Life: The CARDIA Trace Element Study*, 36 Diabetes Care 1584, 1587–89 (2013).

[45] Eliseo Guallar et al., *Mercury, Fish Oils, and the Risk of Myocardial Infarction*, 347 New England J. Med. 1747, 1753 (2002).

[46] Anna L. Choi et al., *Negative Confounding in the Evaluation of Toxicity: The Case of Methylmercury in Fish and Seafood*, 38 Critical Reviews in Toxicology, 877, 877 (2008).

[47] Oken et al., *supra* n. 13, at 1177–79.

[48] Sally Ann Lederman et al., *Relation between Cord Blood Mercury Levels and Early Child Development in a World Trade Center Cohort*, 116 Envtl. Health Persp. 1085, 1090 (2008).

[49] *See* Rune Dietz et al., *Anthropogenic Contributions to Mercury Levels in Present-Day Arctic Animals—A Review*, 407 Sci. Total Env't 6120, 6125–26 (2009); Helen M. Amos et al., *Observational and Modeling Constraints on Global Anthropogenic Enrichment of Mercury*, 49 Envtl. Sci. & Tech. 4036, 4040–42 (2015).

The inverse is also true: past studies analyzing the effects of mercury in the human body have underestimated the dangers because nutrients in fish mask the true adverse effects of methylmercury.[50] Although the mercury-related damage may be masked, the result is that the benefits that consumers would otherwise obtain from a healthy diet are removed, thus counteracting the purpose of including fish in the diet.

4. Methylmercury causes a variety of environmental harms.

Even at low levels, methylmercury threatens numerous aquatic and terrestrial species of amphibians, birds, and mammals.[51] For example, studies report that mercury has severe reproductive effects on fish such as trout, bass, and salmon.[52] Predatory species that consume fish, such as birds and marine mammals, suffer more severe impacts. In the rulemaking, EPA recognized the well-documented effects of mercury on loons,[53] in which the substance causes

[50] Esben Budtz-Jorgensen et al., *Separation of Risks and Benefits of Seafood Intake*, 115 Envtl. Health Persp. 323, 325–26 (2007); Anna L. Choi et al., *Selenium as a Potential Protective Factor Against Mercury Developmental Neurotoxicity*, 107 Envtl. Res. 45, 51 (2008).

[51] David C. Depew et al., *Toxicity of Dietary Methylmercury to Fish: Derivation of Ecologically Meaningful Threshold Concentrations*, 31 Envtl. Toxicology & Chemistry 1536, 1538–45 (2012).

[52] Kate L. Crump et al., *Mercury-Induced Reproductive Impairment in Fish*, Envtl. Toxicology & Chemistry 895, 902–04 (2009).

[53] 77 Fed. Reg. at 9427.

behavioral, physiological, and reproductive impairments.[54] Other piscivorous birds have exhibited decreased foraging efficiency,[55] decreased reproductive success,[56] and liver and kidney damage.[57] Insectivorous birds have likewise shown reduced reproductive capacity,[58] survival rate,[59] immune function,[60] and singing behavior.[61] Mammals that also heavily depend on fish as a food source, such as river otters, suffer from reduced mobility, abnormal reflexes, and impaired escape behavior.[62]

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[54] David C. Evers et al., *Adverse Effects from Environmental Mercury Loads on Breeding Common Loons*, 17 Ecotoxicology 69, 69 (2008); Matthew G. Mitro et al., *Common Loon Survival Rates and Mercury in New England and Wisconsin*, 72 J. Wildlife Mgmt. 665, 665–66 (2008).

[55] Evan M. Adams & Peter C. Frederick, *Effects of Methylmercury and Spatial Complexity on Foraging Behavior and Foraging Efficiency in juvenile White Ibises (Eudocimus Albus)*, 27 Envtl. Toxicology & Chemistry 1708, 1708 (2008).

[56] Peter Frederick & Nilmini Jayasena, *Altered Pairing Behaviour and Reproductive Success in White Ibises Exposed to Environmentally Relevant Concentrations of Methylmercury*, Proc. Royal Soc'y B 1, 4–5 (2010).

[57] David J. Hoffman et al., *Mercury and Drought Along the Lower Carson River, Nevada: III. Effects on Blood and Organ Biochemistry and Histopathology of Snowy Egrets and Black-Crowned Night-Herons on Lahontan Reservoir, 2002-2006*, 72 J. Toxicology & Envtl. Health, Part A 1223, 1223 (2009).

[58] Rebecka L. Brasso & Daniel A. Cristol, *Effects of Mercury Exposure on the Reproductive Success of Tree Swallows (Tachycineta Bicolor)*, 17 Ecotoxicology 133, 133 (2008).

[59] Kelly K. Hallinger et al., *Mercury Exposure and Survival in Free-Living Tree Swallows (Tachycineta Bicolor)*, 20 Ecotoxicology 39, 39 (2011).

[60] Dana M. Hawley et al., *Compromised Immune Competence in Free-Living Tree Swallows Exposed to Mercury*, 18 Ecotoxicology 499, 499 (2009).

[61] Leen Gorissen et al., *Heavy Metal Pollution Affects Dawn Singing Behaviour in a Small Passerine Bird*, 145 Oecologia 504, 504 (2005).

[62] Anton M. Scheuhammer et al., *Effects of Environmental Methylmercury on the*

EPA thus correctly concluded that mercury "emissions pose a hazard to the environment and wildlife, adversely impacting species of fish-eating birds and mammals."[63]

II. THE BENEFITS OF REGULATING POWER PLANT MERCURY EMISSIONS ARE SIGNIFICANT.

A. Reducing emissions quickly decreases mercury deposition and biological exposure to mercury.

Reducing mercury emissions has immediate local and regional benefits. This is because, as described above, a substantial fraction of mercury emissions from power plants deposits into local ecosystems.[64] For example, Evers et al. identified biological mercury hotspots in the northeastern United States driven mainly by domestic emissions.[65] A study of sites in Massachusetts found that mercury concentrations in largemouth bass and yellow perch declined by 44% and 43%, respectively, in lakes in a mercury hotspot area from 1999 to 2011, tracking emissions reductions from major point sources in the region.[66] Studies in Florida[67]

Health of Wild Birds, Mammals, and Fish, 36 Ambio 12, 12 (2007).

[63] 77 Fed. Reg. at 9310.

[64] *See supra* Section I.A.

[65] David C. Evers et al., *Biological Mercury Hotspots in the Northeastern United States and Southeastern Canada*, 57 BioScience 29, 41 (2007).

[66] Michael S. Hutcheson et al., *Temporal and Spatial Trends in Freshwater Fish Tissue Mercury Concentrations Associated with Mercury Emissions Reductions*, 48 Envtl. Sci. & Tech. 2193, 2196 (2014).

[67] Peter C. Frederick et al., *Wading Birds as Bioindicators of Mercury Contamination in Florida, USA: Annual and Geographic Variation*, 21 Envtl.

and Wisconsin[68] have produced similar findings. Other researchers observed close to a 20% decline in mercury accumulation in 104 sediment cores from the Great Lakes regions attributable to domestic emissions reductions.[69]

One recent study found that declining U.S. mercury emissions has led to a decrease in mercury levels detected in ocean fish.[70] The study examined bluefish in the Mid-Atlantic bight, ranging from the continental shelf waters of Cape Cod, Massachusetts, to Cape Hatteras, North Carolina.[71] Another study detected large declines in atmospheric mercury concentrations at a site in Maryland downwind of power plants in Ohio, Pennsylvania, and West Virginia.[72]

New studies bolster EPA's findings on the benefits of reducing emissions,

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Toxicology & Chemistry 163, 163 (2001).

[68] Thomas R. Hrabik & Carl J. Watras, *Recent Declines in Mercury Concentration in a Freshwater Fishery: Isolating the Effects of De-Acidification and Decreased Atmospheric Mercury Deposition in Little Rock Lake*, 297 Sci. Total Env't 229, 236 (2002); Brick M. Fevold et al., *Bioaccumulation Patterns and Temporal Trends of Mercury Exposure in Wisconsin Common Loons*, 12 Ecotoxicology 83, 83 (2003).

[69] Paul E. Drevnick et al., *Spatial and Temporal Patterns of Mercury Accumulation in Lacustrine Sediments Across the Great Lakes Region*, 161 Envtl. Pollution 252, 252 (2012).

[70] Ford A. Cross et al., *Decadal Declines of Mercury in Adult Bluefish (1972–2011) from the Mid-Atlantic Coast of the U.S.A.*, 49 Envtl. Sci. & Tech. 9064, 9064 (2015).

[71] *Id.* at 9065–66.

[72] Mark S. Castro & John Sherwell, *Effectiveness of Emission Controls to Reduce the Atmospheric Concentrations of Mercury*, 49 Envtl. Sci. & Tech. 14000, 14000 (2015).

because they reveal that the local effects of mercury emissions are even greater than previously understood. As mentioned above, for the past two decades, mercury researchers have noted slow and steady declines in atmospheric mercury concentrations in North America, Europe, and over the open oceans. Initial attempts to rationalize these observations were confounded by a commonly held (but incorrect) assumption among researchers that global mercury emission trends from anthropogenic sources were steady or increasing over the same time period. Zhang et al. recently corrected an error in previous emissions inventories of the form of mercury released by power plants over time. This correction helps enable global models to reproduce the observed declining atmospheric mercury trends.[73] This analysis shows that spatial and temporal trends in atmospheric mercury concentrations and deposition are much more influenced by local and regional actions than previously assumed.

Therefore, when EPA issued the Mercury and Air Toxics Rule, it overestimated the disruptive impacts that foreign emissions would have on the Rule's effectiveness. If domestic emissions have a more immediate benefit than what EPA initially expected, then the benefits of the Rule are also greater.

[73] Yanxu Zhang et al., *Observed Decrease in Atmospheric Mercury Explained by Global Decline in Anthropogenic Emissions*, 113 Proc. Nat'l Acad. Sci. U.S. 526, 526 (2016).

B. EPA recognized that the mercury emissions reductions of the Rule would produce significant benefits.

1. EPA's monetization of IQ-related benefits incorporated several conservative assumptions.

In the Regulatory Impact Analysis, the mercury-related benefits that EPA monetized consisted of only a small subset of the total benefits. EPA monetized only neurological benefits and even within these considered only impacts on IQ.[74] The analysis therefore left out other neurological impacts such as ADHD and non-neurological impacts such as cardiovascular harms. In addition, when translating IQ's benefits to society, EPA looked only at the relationship between lost IQ and an individual's earning potential.[75] This approach omits lost IQ's other societal impacts, such as costs of requiring medical care or additional special education programs. And lastly, EPA did not quantify *every* individual's earning potential. Instead, EPA only accounted for children born to a limited population of recreational fishers who consume freshwater fish during pregnancy from watersheds where EPA had fish tissue data.[76] This demographic choice significantly underestimates the neurodevelopmental benefits of the rule. Marine

[74] 77 Fed. Reg. at 9428.

[75] *Id.*

[76] Regulatory Impact Analysis for Final Mercury and Air Toxics Standards at 4-49 [EPA-HQ-OAR-2009-0234-20131], JA __.

fish account for more than 90% of methylmercury intake by the U.S. population,[77] whereas EPA considered only freshwater fish.

Even within its analysis of the impacts to this one small group, EPA adopted several conservative assumptions. For example, in deciding which watersheds to include in the analysis, EPA included only those in which either (1) the total potential exposures exceeded the reference dose, *and* in which power plants contributed more than 5% of the mercury deposition, or (2) the power plant emissions alone would result in an exposure in excess of the reference dose.[78] As EPA explained, "[r]equiring at least a 5 percent EGU contribution is a conservative approach given the increasing risks associated with incremental exposures above the" reference dose.[79] EPA also excluded watersheds near coastal areas and the Great Lakes due to modeling uncertainty.[80] EPA omitted these areas even though they "may have elevated U.S. [power plant] deposition relative to the average

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[77] Elsie M. Sunderland, *Mercury Exposure from Domestic and Imported Estuarine and Marine Fish in the U.S. Seafood Market*, 115 Envtl. Health Persp. 235, 235 (2007).

[78] 77 Fed. Reg. at 9311.

[79] *Id.* at 9311 n.15.

[80] EPA, Revised Technical Support Document: National-Scale Assessment of Mercury Risk to Populations with High Consumption of Self-caught Freshwater Fish at 7 n.10, EPA-452/R-11-009 (2011) [EPA-HQ-OAR-2009-0234-19913], JA __.

levels in the continental U.S."[81]

In addition, EPA's final quantification of the benefits is undervalued for two reasons. First, when EPA estimated the sensitivity of IQ to cord blood methylmercury, it relied on dose-response information from a 2007 study by Axelrad et al.[82] This study is outdated and results in a severe underestimation of the actual harms. Estimates that rely on more recent information present a much greater magnitude of harm.[83]

Second, when translating the relationship between mercury and IQ, EPA applied a linear model that underestimated the true effect of the exposure.[84] In reality, the relationship between the daily intake and brain mercury is a power function with a coefficient greater than 1.0. Therefore, "a decrease in [mercury] intake will produce a greater-than-linear decrease in brain concentration."[85] However, EPA decided on a linear model because the calculation would be much simpler. It recognized that this would underestimate the benefits of mercury

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[81] *Id.*

[82] Regulatory Impact Analysis for Final Mercury and Air Toxics Standards at 4-31 [EPA-HQ-OAR-2009-0234-20131], JA __.

[83] Philippe Grandjean et al., *Calculation of Mercury's Effects on Neurodevelopment*, 120 Envtl. Health Persp. a452, a452 (2012).

[84] Revised Technical Support Document at 20 [EPA-HQ-OAR-2009-0234-19913], JA __.

[85] *Id.*

reduction.[86]

5. EPA recognized that there were many other benefits from reducing power plant mercury emissions, even if it did not quantify them.

EPA recognized that it did not monetize most of the benefits of mercury emission reductions in its analysis.[87] Immediately after EPA stated that the monetized benefits of mercury's effect on IQ for the small subpopulation were $4 million to $6 million, it acknowledged that the other benefits are much greater: "EPA recognizes that these calculated benefits are a small subset of the benefits of reducing [mercury] emissions."[88] As indicated above, these other benefits include reductions in non-IQ neurological harms such as ADHD, in cardiovascular and other human health effects, and in environmental impacts. The decision not to quantify these other benefits did not reflect uncertainty about their *existence*, but had to do with the uncertainty in translating the benefits to a monetary value. However, "unquantifiable benefits . . . are just as real as the targeted benefits that can be monetized."[89] The Utility Study, the Mercury Study, and the NAS Study all support the conclusion that regulating mercury emissions in power plants would be

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[86] *Id.*

[87] 77 Fed. Reg. at 9306.

[88] *Id.* at 9428.

[89] Legal Memorandum Accompanying the Proposed Supplemental Finding 6 [EPA-OAR-2009-0234-20519], JA__.

beneficial for public health.[90]

EPA also considered the environmental justice implications of domestic mercury emissions. First, it focused its quantitative analysis on subsistence fishing populations.[91] Second, EPA noted that coal-fired power plants are frequently located in areas where there are more minority populations and people below the poverty line than the national average.[92] Finally, EPA outlined how mercury deposition hurts indigenous communities in the United States because it impairs subsistence fishing, the quality of water used for ceremonies, and tourism.[93] EPA's recognition of the distributional effects demonstrates that it considered benefits that are not translatable to an aggregate monetary value.

6. It was reasonable for EPA not to quantify these benefits, given the information before it at the time.

It was reasonable for EPA not to quantify the other benefits because, although there was ample evidence of their significance, the precise quantification of them presented considerable difficulties. For example, EPA's decision not to quantify cardiovascular effects was reasonable because it was faced with, on one side, an independent expert panel which asserted that there was sufficient scientific

[90] 77 Fed. Reg. at 9335.

[91] *Id.* at 9313.

[92] *Id.* at 9445.

[93] *Id.* at 9440–41.

evidence to incorporate these outcomes in regulatory assessments,[94] and on the other, a high-profile study of risks of cardiovascular disease associated with methylmercury exposures in two U.S. cohorts that found no evidence of adverse effects.[95]

There are several reasons, however, to conclude that the cardiovascular impacts are substantial despite the latter study. First, the study included only low-to-moderate fish consumers and therefore lacked the statistical power to detect effects seen in studies that included a greater range in exposures.[96] Second, it can be challenging to isolate the neurodevelopmental and cardiovascular impacts of methylmercury exposure from seafood consumption because, as discussed above, seafood also contains long-chained fatty acids that mask those deleterious impacts.[97] In addition, imprecision in exposure biomarkers causes many epidemiological studies to be biased toward a null finding rather than detection of

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[94] Roman et al., *supra* n. 36, at 607.

[95] Dariush Mozaffarian et al., *Mercury Exposure and Risk of Cardiovascular Disease in Two U.S. Cohorts*, 364 New Eng. J. Med. 1116, 1116 (2011).

[96] *See, e.g.*, Anna L. Choi et al., *Methylmercury Exposure and Adverse Cardiovascular Effects in Faroese Whaling Men*, 117 Envtl. Health Persp. 367, 367 (2009); Matthew O. Gribble et al., *Mercury Exposure and Heart Rate Variability: A Systematic Review*, 2 Current Envtl. Health Rep. 304, 304 (2015).

[97] Kathryn R. Mahaffey et al., *Balancing the Benefits of n-3 Polyunsaturated Fatty Acids and the Risks of Methylmercury Exposure from Fish Consumption*, 69 Nutrition Rev. 493, 493 (2011); Oken et al., *supra* n. 13, at 1171.

adverse effects.[98] Finally, failure to find a statistically significant effect is not evidence that no such effect exists, though it may provide evidence that constrains the magnitude of the effect.

C. Multiple studies bolster EPA's assessment that the Mercury and Air Toxics Rule produces substantial economic benefits.

Despite these challenges in monetizing the full range of benefits, several studies have concluded that the economic benefits of the mercury emissions reductions are substantial. A 2016 study indicated that when policymakers account for lost wages, medical costs from IQ deficits and nonfatal heart attacks, and premature fatalities quantified into a value of statistical life (VSL) model,[99] the benefits of the Rule exceed $43 billion.[100] Another study noted that the expected monetary value of a 10% reduction of methylmercury would be $860 million a year.[101] Even if one were to limit the discussion to the regulation's effects on IQ, the benefits remain large. Trasande et al. determined that taking into account the effects of lower IQ on schooling, probability of workforce participation, and

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[98] Philippe Grandjean & Esben Budtz-Jorgensen, *Total Imprecision of Exposure Biomarkers: Implications for Calculating Exposure Limits*, 50 Am. J. Indus. Med. 712, 712 (2007).

[99] Amanda Giang & Noelle E. Selin, *Benefits of Mercury Controls for the United States*, 113 Proc. Nat'l Acad. Sci. U.S. 286, 288 (2016).

[100] *Id.* at 286.

[101] Glenn E. Rice et al., *A Probabilistic Characterization of the Health Benefits of Reducing Methyl Mercury Intake in the United States*, 44 Envtl. Sci. & Tech. 5216, 5216 (2010).

lifetime earnings showed that mercury emissions from U.S. power plants cost an estimated $1.3 billion annually to the economy.[102]

CONCLUSION

For the foregoing reasons, the Petitions for Review should be DENIED.

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Respectfully submitted,

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[102] Leonardo Trasande, *Public Health and Economic Consequences of Methyl Mercury Toxicity to the Developing Brain*, 113 Envtl. Health Persp. 590, 592 (2005).

[103] *Amici* would like to acknowledge the contributions to this brief by Joshua Lee, a student in the Emmett Environmental Law & Policy Clinic at Harvard Law School.

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

1. This brief complies with the type-volume limitations of Fed. R. App. P. 32(a)(7)(B) and 29(d) because this brief contains 6,496 words, excluding the parts of the brief exempted by Fed. R. App. P. 32(f).

2. This brief complies with the typeface requirements of Fed. R. App. P. 32(a)(5) and the type style requirements of Fed. R. App. P. 32(a)(6) because it has been prepared in a proportionally spaced typeface using Microsoft Word 2010 in Times New Roman 14-point font type.

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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

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