American Academy of Pediatrics * American Lung Association * American Nurses Association * American Public Health Association * Chesapeake Bay Foundation * Clean Air Task Force * Counsel for Citizens for Pennsylvania's Future * Conservation Law Foundation * Environment America * Environmental Defense Fund * Natural Resources Council of Maine * Natural Resources Defense Council * Physicians for Social Responsibility * Sierra Club * The Ohio Environmental Council

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Attention: Docket ID No. EPA-HQ-OAR-2009-0234

Re: Comments of Public Health and Environmental Groups on U.S. EPA's Proposed Supplemental Finding That It Is Appropriate and Necessary to Regulate Hazardous Air Pollutants from Coal- and Oil-Fired Electric Utility Steam Generating Units, 80 Fed. Reg. 75,025 (Dec. 1, 2015)

The American Academy of Pediatrics; the American Lung Association; the American Nurses Association; the American Public Health Association; the Chesapeake Bay Foundation; Counsel for Citizens for Pennsylvania's Future; the Conservation Law Foundation; Environment America; the Environmental Defense Fund; the Natural Resources Council of Maine; the Natural Resources Defense Council; Physicians for Social responsibility; the Sierra Club; and The Ohio Environmental Council ("Public Health and Environmental Groups") respectfully submit these comments on the Environmental Protection Agency's (EPA) proposed "Supplemental Finding That It Is Appropriate and Necessary to Regulate Hazardous Air Pollutants From Coal- and Oil-Fired Electric Utility Steam Generating Units," published at 80 Fed. Reg. 75,025 (Dec. 1, 2015) ("Proposed Supplemental Finding"), and the "Legal Memorandum Accompanying the Proposed Supplemental Finding that it is Appropriate and Necessary to Regulate Hazardous Air Pollutants from Coal- and Oil-Fired Electric Utility Steam Generating Units (EGUs)" ("Legal Memorandum").

Public Health and Environmental Groups strongly support EPA's Proposed Supplemental Finding, in which EPA proposes to find that, after considering cost, regulation of emissions of hazardous air pollutants (HAPs) emitted by EGUs is and remains "appropriate" under section 112(n)(1)(A) of the Clean Air Act ("CAA"). In view of the compelling and long-overdue public

health and environmental benefits of controlling EGUs' enormous volumes of emissions of some of the most dangerous air pollutants, the Public Health and Environmental Groups urge EPA to finalize that finding promptly.

INTRODUCTION AND SUMMARY

In *Michigan v. EPA*, 135 S.Ct. 2699 (2015), the Supreme Court held that EPA must consider cost in determining whether regulation of HAP emissions from EGUs is "appropriate" within the meaning of CAA section 112(n)(1)(A). The Supreme Court did not prescribe *how* EPA must take into account cost. The Court expressly declined to decide that the statute requires "formal cost-benefit analysis," noting instead that "it will be up to the Agency to decide (as always, within the limits of reasonable interpretation) how to account for cost." 135 S.Ct. at 2711. In the present administrative proceeding, EPA is inquiring how to account for cost under section 112(n)(1)(A), as well as whether regulation of HAPs emitted by EGUs remains "appropriate" considering cost.

As EPA recognizes in its Proposed Supplemental Finding, the proper scope of this supplemental administrative proceeding is narrow. 80 Fed. Reg. at 75,027-28. The task here is to determine the "appropriateness" of regulation while considering cost. It is not to revisit the myriad other issues concerning the structure, design, or stringency of the Mercury and Air Toxics Standards (MATS) regulations, including the many challenges that were unanimously rejected in the D.C. Circuit decision upholding MATS, *White Stallion Energy Center v. EPA*, 748 F.3d 1222 (D.C. Cir. 2014), and not subsequently reviewed and ruled on by the Supreme Court. *See* 80 Fed. Reg. at 75,028 (enumerating some of those issues).[1]

While we support EPA's reliance on the record at the time MATS was finalized in 2011 to assess the cost issue, EPA should also take account of the reality that MATS has been on the books and operational for a considerable time. EPA should take note of the fact that actual experience under the Rule, including sources' ability to satisfy MATS requirements, the cost of doing so, sales impacts, ratepayer impacts, and other cost-related metrics are consistent with (or below) the EPA's previous findings about cost based on pre-implementation data. EPA should incorporate that analysis into the supplemental finding.

In these comments, the Public Health and Environmental Groups make the following points:

[1] While the Public Health and Environmental Groups agree with EPA concerning the properly limited scope of this supplemental proceeding, we note that, were MATS to be reopened more generally, there would be a compelling basis for emissions standards considerably stronger than those adopted in MATS. That is so, because the universe of sources that exists now would support considerably stronger "floor" standards under the well-understood approach to floor-setting under section 111(d), and given evidence that plunging costs of key control technologies and other factors have driven the costs of controlling HAP emissions from EGUs dramatically lower than EPA estimated in 2011. *See* Part III, below.

Part I: As the record shows, the benefits to the public of regulating HAP emissions from EGUs – by far the largest U.S. source of mercury and many other dangerous toxic air emissions – are very large. Section 112 reflects a clear and emphatic congressional judgment that reducing emissions of the hazardous air pollutants that Congress listed is an urgent priority. EPA's own determinations in the rulemaking regarding the particular HAPs emitted in great volumes by EGUs further underline the enormous benefits to the public of controlling these HAPs – and nothing in the statute requires that these values be "monetized" in order to receive great weight. Nothing in the statute or *Michigan* requires EPA to determine "appropriateness" by means of a cost-benefit analysis, nor to rely on monetization as the measure of the public benefits of reducing HAPs. And the threshold character of section 112(n)(1)(A) militates strongly against a cost standard that forecloses control of dangerous pollution based on preliminary estimates of future compliance costs – which history shows are often dramatically overestimated. EPA's proposal properly looks for guidance to the well-developed cost-consideration principles under CAA section 111 – which allow dangerous pollutant to remain uncontrolled by available methods only where costs would be "exorbitant" or "too much for the industry as a whole to bear" – and, if anything, the standard for leaving toxic pollutant entire uncontrolled should be more protective for the public than section 111 standards governing the mere stringency of controls of criteria pollutants. EPA's proposed metrics for judging whether costs to the power industry are excessive are reasonable, and all confirm the appropriateness of regulation.

Part II. The consideration of cost as part of the section 112(n)(1)(A) "appropriate' determination must take into account the extensive mechanisms in section 112(d) to ensure against excessive costs. The record reveals that the application of section 112(d) to EGUs produced standards that are, in fact, reasonable and far from exorbitant or oppressive. The Agency's floor analysis produced best performing units that are fairly representative, and which use no unusual or cost-prohibitive pollution-control methods. The record confirms that the utility sector has no unique attributes that render the statutory standard-setting mechanism unreasonable, from a cost-based view.

Part III: While regulation of EGU HAPs was and is clearly "appropriate" based upon the record and compliance costs estimates made then, actual experience indicates that, as with other air pollution control programs, actual compliance costs are dramatically lower than those estimated by EPA in 2011. A recent analysis of actual MATS compliance costs estimated that, due in part to rapidly lowering costs of control technologies, the actual costs of MATS compliance are approximately $2 billion, or less than one-quarter of EPA's initial $9.6 billion estimate. EPA should take notice of this information, which reflects realities on the ground and supplies a means of checking EPA's 2011 cost estimates, and EPA's tentative finding that costs are not excessive, against current realities.

Part IV: While the CAA does not require the use of formal cost-benefit analysis, the analysis that EPA performed in the RIA further and independently confirms the appropriateness of regulation. A proper and complete benefits analysis must consider all of the pollution reductions resulting from applying section 112(d) to control EGUs' emissions of HAPs. Indirect costs and benefits are properly included in a properly conducted cost-benefit analysis, and the text of section 112(n)(1)(A) as well as the explicit reasoning of the *Michigan* opinion strongly support considering all health benefits of regulation. Any cost-benefit analysis should recognize that the

HAP-specific benefits of regulation are very large, regardless of whether they are quantified. Similarly, all of the benefits associated with MATS implementation should be considered if the Agency includes a benefit-cost metric as a basis for making its final decision.

I. EPA'S PROPOSED FRAMEWORK FOR CONSIDERING COST IS CONSISTENT WITH THE STATUTE AND REASONABLE

A. The Public Benefits of Regulating HAP Emissions from EGUs Are Enormous

This administrative proceeding, prompted by the *Michigan* decision, is intended to address the question whether, considering cost, it is "appropriate" to regulate EGUs' emissions of HAPs under CAA section 112. While the current inquiry is properly focused on cost, the overall question of the appropriateness of regulation involves consideration of the harms that regulation will address. As EPA recognizes in the proposed supplemental finding, "the consideration of cost in the appropriate finding should be weighed against, among other things, the volume of HAP emitted by EGUs and the associated hazards to public health and the environment." 80 Fed. Reg. at 75,028. *See also id*. at 75,038 (noting EPA's prior findings that "HAP emissions from EGUs present significant hazards to public health and the environment").

EPA properly has not reopened its prior determinations, all undisturbed by *Michigan*, concerning the quantities of HAP emissions from EGUs; the serious harms caused by the HAPs emitted by EGUs; and the availability of effective means to control those emissions. However, because opponents of EGU HAP regulation have asserted that controlling EGU HAPs causes only minor or trivial public health benefits, EPA should make abundantly clear, as it addresses costs, that controlling HAPs from EGUs brings enormous benefits for public health and the environment. EPA should reiterate its prior determinations that, not only would regulation of EGUs under section 112 mean huge reductions in emissions of HAP, and also should reaffirm EPA's judgment, based upon the record and the agency's application of its technical and scientific expertise, that the health and environmental benefits that these enormous reductions in listed HAPs will achieve are extremely valuable and substantial, and well worth the cost.

We agree with EPA that the major reductions in HAPs represent an enormous gain to the public health and environmental quality, and these HAP benefits – whether quantifiable or not – suffice to justify the costs of regulation. EPA has properly refused to second-guess Congress' judgment, plainly reflected in the statute, that the hazards posed by listed air toxics are entitled to great weight. That judgment remains, if anything, even more sound today than it was in 1990, and the Agency's record contains abundant scientific and technical evidence demonstrating as much. We also agree that the reductions in emissions of particulate matter and other non-HAP – both due to the application of primary particulate controls at the stack, and also resulting from the sulfur dioxide/acid gas controls required by MATS to control hydrogen chloride, hydrogen fluoride, chlorine gas, and hydrogen cyanide – provide very significant public health benefits including reduced premature mortality, and that those benefits provide further confirmation that regulation is "appropriate." *See* Part IV below.

1. Section 112 Reflects a Clear and Emphatic Congressional Judgment that Reducing Emissions of Listed HAPs Is an Urgent National Priority and Provides Major Public Benefits

The statutory text and history makes clear that Congress's prime objective in section 112 is to reduce emissions of hazardous air pollutants and thereby eliminate or reduce risks to the public and the environment. In the Proposed Supplemental Finding, EPA aptly characterizes the purpose of section 112, namely: "to achieve prompt, permanent and ongoing reductions in the volume of HAP emissions that pose identified or inherent hazards to public health and the environment to reduce the risks posed by such emissions, including risks to the most exposed and most sensitive members of the population." 80 Fed. Reg. at 75,038.

Congress listed the HAPs to be regulated, and subjected industrial sources of the listed HAPs to a stringent, deadline-driven control regime for good reasons. *See* Legal Memorandum 8-10 (describing strict regime) Congress singled out these pollutants for their "potent" and "especially serious health risks," even in relatively small quantities, Legis. History of the Clean Air Act Amendments of 1990 (Cong. Research Serv. 1993) ("Leg. Hist.") at 2,522 (explaining difference between "hazardous air pollutants" and "criteria pollutants" addressed elsewhere in CAA) (House Debate). Sec. 112(b)(1) (list of pollutants). Those risks include "birth defects, damage to the brain or other parts of the nervous system, reproductive disorders, and genetic mutations," as well as cancer. Leg. Hist. at 2,524 (House Debate). *See* Sec. 112(b)(2) (air toxics may, *inter alia*, be "carcinogenic, mutagenic, teratogenic, neurotoxic," "cause reproductive dysfunction," or be "acutely or chronically toxic").

In order to provide more effective control of these pollutants, Congress completely revised section 112 in the 1990 CAA Amendments. Noting EPA's two-decade failure to promulgate sufficiently protective standards, Congress rejected proposals that would have entrusted EPA with the discretion to balance "health and economic considerations" against each other. Leg. Hist at 8,746-47 (EPA would "fail[] to protect public health" in such balancing) (Sen. Lautenberg). That rejection reflected the special regulatory difficulties Congress saw as particular to the nature of air toxics: "[t]he public health consequences of substances which express their toxic potential only after long periods of chronic exposure will not be given sufficient weight in [a] regulatory process when they must be balanced against the present day costs of pollution control and its other economic consequences." Leg. Hist. at 8,522 (Senate Report).

In section 112, Congress itself listed 189 compounds as hazardous air pollutants warranting Clean Air Act regulation, Sec. 112(b), and required EPA to list every other compound "known to cause or [that] may reasonably be anticipated to cause adverse effects to human health or adverse environmental effects," *id*. 112(b)(3)(B). With respect to mercury in particular, the statute instructs EPA to pay special attention to "sensitive populations." *Id*. 112(n)(1)(B)-(C). And the statute requires EPA to consider impacts on individuals "most exposed to emissions of such pollutants from the source." *Id.* 112(c)(9)(B)(i). The stringent provisions of section 112 leave no doubt that Congress regarded the control of these HAPs as a matter of urgent priority (and, correspondingly, regarded HAP reductions as a major benefit to the public).

EPA determined that power plants remained in 2011, by a wide margin, the largest emitters of numerous of these congressionally listed hazardous air pollutants, emitting 84 listed air toxics. 77 Fed. Reg. 9304, 9335 (Feb. 12, 2012); *see also* Environmental Health & Engineering, "Emissions of Hazardous Air Pollutants from Coal-fired Power Plants" at 1 (July 2011) ("EH&E Report"), Docket No. EPA-HQ-OAR-2009-0234-17648, Ex. 23; JA 01320-10367. EPA found that EGUs account for very large proportions of *total* U.S. emissions of numerous hazardous air pollutants listed in section 112(b)(1): Mercury – 50 percent; Arsenic – 62 percent; Cadmium – 39 percent; Chromium – 22 percent; Hydrochloric Acid – 82 percent; Hydrogen Fluoride – 62 percent; Nickel – 28 percent; and Selenium – 83 percent. 77 Fed. Reg. at 9,310 (percentages of total U.S. emissions in 2005 inventory). *See also id*. at 9,337 (noting that power plants remain the "predominant source" of anthropogenic U.S. mercury emissions, particularly the oxidized and particulate forms that are of "primary concern" for public health). As the Agency recognizes, each of these is "chemically identical to HAP that are emitted from other stationary sources and thus the risks posed by exposure to such HAP are the same." Legal Memorandum at 11. The public benefits offered by regulating EGU emissions under section 112 are obviously great due to the vast quantities of emissions of multiple listed HAPs that can only be reduced by such regulation.

In the proposed Rule, EPA projected that the Rule will in fact yield deep reductions in aggregate *nationwide* emissions of many of the toxics Congress listed in 42 U.S.C. 7412(b)(1), 76 Fed. Reg. at 25,013-14, including a 49 percent reduction in all anthropogenic emissions of hydrochloric acid gas and a 38 percent reduction in non-mercury metal hazardous air pollutants such as arsenic, chromium, and nickel, *id.* at 25,013-15. The Rule will reduce power plant mercury emissions by 75 percent, 77 Fed. Reg. at 9424, thereby eliminating over a third of total national anthropogenic mercury emissions. 76 Fed Reg. at 25,015. These emissions reductions dwarf those in most, if not all, of the other source categories that have been regulated under section 112. Congress mandated that EPA regulate even source categories that are much smaller emitters of HAPs than are EGUs; the statutory regime clearly places enormous value on reductions in HAP emissions on the order available by regulating EGUs. The enormous reductions in these especially dangerous pollutants that would be achieved by regulating EGU HAP emissions under section 112 must, based upon the sheer volume of avoided toxic emissions at issue, be counted as a very major public benefit. To do otherwise – to conclude, as some industry advocates have suggested, that the hazards posed by the vast quantities of EGU-produced air toxics may be disregarded as posing only modest harm – would run afoul of a clear congressional judgment to the contrary. Indeed, regulating EGUs' HAP emissions must rank among the most effective actions ever taken to address hazardous air pollutants, under section 112 or any other provision.

2. EPA's Determinations Further Confirm the Benefits to Public Health of Controlling HAP Emissions from EGUs under Section 112 Are Very Great

While the congressional judgments reflected in section 112, combined with the large volumes of HAP emissions at stake, suffice to establish the great public value of regulation, that conclusion is also supported by EPA's review of the administrative record and its exercise of its expert judgment concerning the health and environmental benefits of reducing HAPs emitted by EGUs, as well as the opinions of leading health scientists. EPA should forcefully reject suggestions – flatly contrary to the voluminous record here, and to the opinion of health

scientists who study the effects of these pollutants on human health and the environment – that the hazardous air pollutant emissions in question are of less than top priority, or only of modest value.

In its prior findings that regulation of EGU HAP emissions was "appropriate and necessary," EPA determined that emissions of mercury and other HAPs posed a "hazard" to public health – a term EPA understood to demand inquiry into "severity" and "magnitude." 76 Fed. Reg. at 24,992. EPA identified substantial public health harms from the HAPs in question, including "about 580,000 women" of child-bearing age with blood mercury levels sufficient to endanger a developing fetus. 76 Fed. Reg. at 24,995; *see id.* at 25,007-11 (finding that power plants were substantial contributors to these levels). EPA also found, based on a peer-reviewed risk assessment, that power plant emissions of mercury in 2016 would cause or significantly contribute to human exposures exceeding safe levels in nearly a quarter of modeled watersheds "with populations at-risk," 77 Fed. Reg. at 9355; and that power plants were responsible for significantly higher mercury pollution in the areas nearest to them, 76 Fed. Reg. at 25,013. EPA also found that non-mercury metals like chromium and nickel, emitted by power plants as particulates, pose cancer risks, *id*. at 24,978, 25,011; 77 Fed. Reg. at 9319, and that power plants continue to be a significant source of these and other toxic metals, such as arsenic and cadmium, which have serious health effects. *See* 76 Fed. Reg. 25,003-4, 25,006 tbl. 5. *See also* 77 Fed. Reg. at 9380 (most non-mercury metallic toxics are emitted, and best controlled, as particulates). Recognizing that power plants account for an overwhelming share of the hydrogen chloride and hydrogen fluoride emitted in the U.S. (and are significant sources of hydrogen cyanide), and that these acid gases have serious acute and chronic health effects, 76 Fed. Reg. at 25,004-5, EPA expressed its concern "about the potential for [power plant] acid gas emissions to add to already high atmospheric levels of other chronic respiratory toxicants," *id*. at 25,016. *See also* 77 Fed. Reg. at 9363, 9405-06. In addition, EPA found that the Rule would reduce harm to those currently exposed to the highest risks, *id*. at 9445-46, and produce "substantial health improvements for children," *id*. at 9441.

EPA also explained that emissions of mercury and other HAPs cause a variety of serious harms to the environment, including contamination of rivers and lakes, and poisoning of fish, birds and other wildlife. *See* 76 Fed. Reg. at 24,983, 25,012-13, 25,016; 77 Fed. Reg. at 9362, 9362-63, 9424. Other commenters in this proceeding – including the Center for Biological Diversity and Defenders of Wildlife – have submitted comments today discussing in detail the broad range of serious harms that emissions of HAPs from EGUs have for fish and wildlife and for entire ecosystems. A coalition of Native American tribes and tribal organizations has likewise submitted comments emphasizing the serious harms that such pollutions cause many Native Americans and to critically important tribal cultural practices. And a large coalition of states led by Massachusetts is submitting comments describing the serious harms that mercury pollution – especially from upwind EGUs – has caused to vast numbers of state waterbodies, and the burdens that pollution has caused for states' ability to comply with their obligations under water pollution laws. It is not an exaggeration to say that HAP pollution from EGUs has caused far-reaching adverse changes to the natural environment across much of the continental United States, harmed wildlife, and impaired recreational opportunities – as is evidenced by the fact that nearly every state has issued mercury-related fishing advisories, often for most of their major water bodies. "Monetized" or not, these are grave harms of the sort Congress enacted section

112 to address, and the vast reductions in HAP emissions that regulation under CAA section 112 will entail will provide great benefits by mitigating this ongoing environmental damage.

While the record before EPA when it made its 2000 and 2011 findings abundantly supports EPA's conclusions concerning the seriousness of the health and environmental hazards of EGU HAP emissions, that view gathers additional support from the opinions of leading health scientists and more recent scientific study.

As EPA recognized in its Proposed Supplemental Finding, its 2011 monetized estimate of the I.Q.-related costs of mercury pollution substantially underestimates the overall societal costs of power plant mercury emissions and does not account for the overall societal costs of non-mercury HAP emissions.[2] More recent scientific studies confirm that the overall societal costs – both quantified and unquantified – of mercury emissions from U.S. power plants are very substantial. *See* P. Grandjean, *Report on the Health Benefits of Reducing Mercury Emissions from U.S. Power Plants* (Jan. 15, 2016), attached as Ex. 1 ("Grandjean Report"); January 15, 2016 Comments of E. Sunderland, *et al.*, to Docket ID No. EPA-HQ-OAR-2009-0234 ("Sunderland Comments"). Indeed, even EPA's $4 to $6 million monetized estimate of the I.Q.-related costs of mercury pollution substantially understates that very narrow subset of mercury-related societal costs. *See* Grandjean Report at 7-8.[3] And recent attempts to assess a more comprehensive suite of mercury-related harms (including cardiovascular effects) show that the monetized societal costs of mercury pollution alone are much greater than EPA's prior narrow, I.Q.-related estimate, and that the unquantified health risks from mercury are also of significant concern. *See* Grandjean Report at 8-9[4]; *see also* Sunderland Comments.

Moreover, these mercury-focused assessments do not account for the very significant, unquantified harms from the non-mercury HAPs emitted by U.S. power plants. *See* Grandjean Report at 9; EPA Final Regulatory Impact Analysis at Chapters 4 and 5; *see also* Declaration of Amy B. Rosenstein, submitted in support of the Joint Motion of State, Local Government and Public Health Respondent-Intervenors for Remand Without Vacatur, in *White Stallion v. EPA*, D.C. Cir. No. 12-1100 (Sept. 24, 2015) ("Rosenstein Declaration"), attached as Ex. 6 (describing acid gas health effects); Declaration of Douglas W. Dockery, submitted in support of the Joint

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[2] 80 Fed. Reg. at 75,040 (stating that "the limited estimate for the single neurodevelopmental endpoint that could be monetized (IQ loss among certain recreational fishers) is a substantial underestimate of the total mercury impacts among affected populations.")

[3] *Citing, inter alia*, Karagas, *et al*., Evidence on the Human Health Effects of Low-Level Methylmercury Exposure, *Envtl. Health Perspectives* 120: 799 (2012), attached as Ex. 2; M. Bellanger, *et al.*, Economic benefits of methylmercury exposure control in Europe: Monetary value of neurotoxicity prevention, *Envtl. Health* 12: 3 (2013), attached as Ex. 3; Y. Zhang, *et al*., Observed decrease in atmospheric mercury explained by global decline in anthropogenic emissions, *PNAS Early Edition* (2016), attached as Ex 4.

[4] *Citing* A. Giang & N.E. Selin, Benefits of mercury controls for the United States, *PNAS (Early Edition)* at 3 (2015) (economy-wide benefits from the Mercury and Air Toxics Standards estimated at $43 billion by 2050, and lifetime benefits at $147 billion, including very substantial benefits from avoided cardiovascular effects), attached as Ex. 5.

Motion of State, Local Government and Public Health Respondent-Intervenors for Remand Without Vacatur, in *White Stallion v. EPA*, D.C. Cir. No. 12-1100 (Sept. 24, 2015) ("Dockery Declaration"), attached as Ex. 7 (same for particulate matter); Declaration of Jonathan I. Levy, submitted in support of the Joint Motion of State, Local Government and Public Health Respondent-Intervenors for Remand Without Vacatur, in *White Stallion v. EPA*, D.C. Cir. No. 12-1100 (Sept. 24, 2015) ("Levy Declaration"), attached as Ex. 8 (describing the metrics by which harms and benefits can, and currently cannot, be quantified). Overall, recent scientific findings on the quantified and unquantified benefits of reducing HAP exposure strongly reinforce EPA's determination that it is appropriate to regulate hazardous air pollution from power plants after considering the costs.

3. Neither The CAA Nor Any Other Authority Required that the Benefits of Controlling HAPs Must be "Monetized" Before Being Given Great Weight

The Agency's decision not to condition regulation on precise quantification and monetization of the grave health and environmental hazards associated with EGU HAP emissions is rooted in a sound judgment as to the current state of the scientific effort aimed at such quantification. Because air toxics are emitted in a complex mixture with other power plant air pollution, moreover it is well-nigh impossible, and in some cases essentially meaningless, to attempt to evaluate the monetary value of discrete reductions in certain pollutants as separate from others.[5] Moreover, the fact that many of the associated adverse health effects are

[5] Efforts are underway to assess the effects of distinct constituents of *particulate matter*, but these efforts have not produced specific dose-response values or monetization metrics. *See* Levy, J.I. *et al*., a meta-analysis and multi-site time-series analysis of the differential toxicity of major fine particulate matter constituents. 175 Am. J. Epidemiol. 1091 (2012), *available at* http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3491972/; Bell ML, Dominici F, Ebisu K, Zeger SL, Samet JM. 2007. Spatial and temporal variation in PM2.5 chemical composition in the United States for health effects studies. *Environmental Health Perspectives* 115(7): 989-995, *available at* http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1913582/. What is known is that metals interact with particles to create "reactive oxygen species" which limit the body's ability to repair damage to its cells and contribute to tissue inflammation. Carter JD, Ghio AJ, Samet JM, Devlin RB. 1997. Cytokine Production by Human Airway Epithelial Cells after Exposure to an Air Pollution Particle Is Metal-Dependent. *Toxicology and Applied Pharmacology*. 146(2):180-188, *abstract available at* http://www.ncbi.nlm.nih.gov/pubmed/9344885; Gurgueira SA, Lawrence J, Coull B, Krishna Murthy GG, Gonzalez-Flecha B. 2002. Rapid increases in the steady-state concentration of reactive oxygen species in the lungs and heart after particulate air pollution inhalation. *Environmental Health Perspectives* 110(8): 749-765, *available at* https://dash.harvard.edu/handle/1/4522597; Wilson MR, Lightbody JH, Donaldson K, Sles J, Stone V. 2002. Interactions between Ultrafine Particles and Transition Metals *in Vivo* and *in Vitro*. *Toxicology and Applied Pharmacology* 184(3): 172-179, *abstract available at* http://www.sciencedirect.com/science/article/pii/S0041008X02995013. Certain particulate constituents also have been associated with increased hospital emissions, heart attacks, and increased mortality. Zanobetti A, Franklin M, Koutrakis P, Schwartz J. 2009. Fine particulate air pollution and its components in association with cause-specific emergency admissions. *Environmental Health* 8:58, *available at* http://www.ehjournal.net/content/8/1/58; Franklin M, Koutrakis P, Schwartz J. 2008. The Role of Particle Composition on the Association Between PM2.5 and Mortality. *Epidemiology*. 19: 680-698, *available at* http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3755878/. And several of the toxic metals are known carcinogens. For example, *see*, Agency for Toxic Substances and Disease Registry

experienced as a result of exposures to the *mixture* of the pollutants[6] illustrates that such an evaluation would not much help with the larger question, which is how to value all of the pollution reductions associated with the rule. For example, when EPA finalized the MATS rule, coal-fired EGUs were the largest point source category of hydrochloric acid, mercury, and arsenic releases to the air. EH&E Report at 1-2, *citing* U.S. EPA National Emissions Inventory 2002: Inventory Data: Point Sector Data-ALLNEI HAP Annual 01232008, *available at* http://www.epa.gov/ttn/chief/net/2002inventory.html#inventorydata. EGU emissions contain 84 of the 187 listed HAP pollutants, in a mixture with conventional air pollutants like sulfur dioxide, nitrogen oxides, and particles. The primary particles, also called filterable particulates, contain many of the toxic metals emitted by coal and oil-fired EGUs. Additionally, chemical reactions in the emissions plume after emission to the ambient air produce additional pollution, including most importantly for public health, fine particulates (called "secondary particles").

The health effects caused by exposures to EGU air pollution are caused by the individual components of the uncontrolled plume, but also by the mixture of pollutants, and by the secondary particles formed after emission. Rosenstein Declaration at ¶ 7; *see also* Dockery Declaration at ¶¶ 7 & 8 (discussing the distinction between primary and secondary particulates, and the health effects of breathing particulate matter); Levy Declaration at ¶¶ 7-9 (discussing the mixture of toxic constituents present in particulates).

While there is a robust scientific literature analyzing and describing the public health effects of exposures to the air pollutants emitted by EGUs, for only some of the pollutants can specific health endpoints associated with levels of ambient concentrations of pollutants be quantified. Dockery Declaration at ¶¶ 8, 9, 13. For those specific endpoints, it is possible to calculate the health benefits of specific levels of particulate air pollution in terms of reduced hospitalization, reduced mortality, and other end points that can be valued, using assigned monetary figures – so benefits per ton of pollutant reductions can be quantified. Levy Declaration at ¶¶ 13-16. While there is more confidence in the quantitative relationship between particulate matter and sulfur dioxide exposures at various levels and human health effects today than when the MATS Rule was finalized in 2011, for other air pollutants emitted by EGUs this is not the case. For the specific non-mercury metal constituents of primary particulates, for example, it is not yet possible to derive quantified or monetized health benefit values. Levy Declaration at ¶ 9; Dockery Declaration at ¶ 13.

Though not quantifiable or monetizable at this time, the benefits of controlling EGU HAPS and reducing the incidence of acute and chronic exposures can be qualitatively described, as EPA documents in its RIA. EPA RIA Chs. 4 & 5. Controls limiting air toxic HAPs mean that

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(ATSDR). 1998. Medical Management Guidelines for Arsenic. Atlanta, GA: U.S. Department of Health and Human Services, *available at* http://www.atsdr.cdc.gov/mhmi/mmg168.html#bookmark02; Steenland K, Ward E. 1991. Lung cancer incidence among patients with beryllium disease: a cohort mortality study. *J Natl Cancer Inst* 83(19): 1380-1385, *available at* http://www.ncbi.nlm.nih.gov/pubmed/1920480.

[6] Franklin M, Koutrakis P, Schwartz J. 2008. The Role of Particle Composition on the Association Between PM2.5 and Mortality. *Epidemiology*. 19: 680-698, *available at* http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3755878/ (finding that the combination of metals in particles, a common occurrence, may increase their toxicity).

people – who are often those most vulnerable in our society, as EPA points out in the final MATS rule, 77 Fed. Reg. at 9444-46, will develop fewer cases of serious health problems, fewer hospitalizations for respiratory ailments, fewer cardiovascular effects, neurological responses, and premature mortality, among other adverse health effects of air toxics and exposures. Even though there may be no metric yet to calculate the dollar value of those benefits, they are real and substantial and fully support the conclusion that it is appropriate to regulate hazardous air pollution from power plants.

B. Considering Cost, Regulation of EGU HAP Emissions Is Clearly Appropriate

1. Section 112(n) Does Not Mandate that EPA Employ Cost-Benefit Analysis as the Measure of Whether Regulation of HAPs from EGUs is "Appropriate" and Does Not Require EPA to Monetize the Benefits of Regulation

We agree that section 112(n)(1) does not require that EPA consider costs by means of a cost-benefit analysis. *See*, *e.g.*, Legal Memorandum 20. First, the *Michigan* opinion expressly declined to hold that EPA must base its "appropriate" determination upon a cost-benefit analysis. 135 S.Ct. at 2711 ("We need not and do not hold that the law unambiguously required the Agency, when making this preliminary estimate, to conduct a formal cost-benefit analysis in which each advantage and disadvantage is assigned a monetary value. It will be up to the Agency to decide (as always, within the limits of reasonable interpretation) how to account for cost.").

Second, there is nothing in the text of section 112(n)(1) that mandates any particular way of considering cost. Cost is an implicit component of the general term "appropriate," and there is no textual reason EPA cannot consider cost in another manner, particularly given that approaches starkly different from cost-benefit analysis have long been held proper under statutory provisions that expressly require consideration of "cost," such as CAA Section 111(a). *See* Legal Memorandum 18-19.

Third, there are many obvious features of the statute that cut against use of cost-benefit analysis – particularly a version of such analysis that would require that regulatory benefits must be monetized in order to "count." Such an approach would have been especially ill-suited to a statutory program covering a broad range of disparate air toxics with a broad variety of health and environmental effects subject to different and changing levels of scientific understanding. *See*, *e.g.*, Grandjean Report at 5-9; Sunderland Comments. A requirement that any harms, to be cognizable, must be monetized, would either create enormous delays or preclude regulation outright, even of very dangerous toxin,s merely because adequate data about impacts is not available. That CAA imposes no such requirement; indeed, in enacting the comprehensive 1990 amendments to section 112, Congress recognized that such calculations are especially difficult for hazardous air pollutants. *See* Leg. Hist. at 8,522 (Senate Report) ("[t]he public health consequences of substances which express their toxic potential only after long periods of chronic exposure will not be given sufficient weight in [a] regulatory process when they must be balanced against the present day costs of pollution control and its other economic consequences."); *see also Portland Cement Ass'n v. Ruckelshaus*, 486 F.2d 375, 387 (D.C. Cir. 1973) ("The difficulty, if not impossibility, of quantifying the benefit to ambient air conditions,

further militates against the imposition of such an imperative on the agency."). The statutory emphasis upon sensitive populations and most-exposed individuals also, as EPA correctly observes, cuts against adopting a cost-benefit test as the measure of "appropriateness" of regulation.

EPA is also clearly correct to conclude that, under section 112(n)(1), Congress did not intend cost to be the "predominant or overriding factor" in the appropriate determination. *See* 80 Fed. Reg. at 75,031. The fact that cost is not specifically mentioned in the provision; the context of section 112 in a provision concerned with controlling pollutants designed by Congress as especially dangerous; and the express mentions of "hazards to health" as the sole specific factor enumerated in 112(n)(1)(A) all reinforce that conclusion. Indeed, the Supreme Court's discussion of the word "appropriate" in 112(n)(1) makes clear that Congress did not intend to limit EPA to cost or any other single factor: "One does not need to open up a dictionary in order to realize the capacious-ness of this phrase. In particular, "appropriate" is 'the classic broad and all-encompassing term that naturally and traditionally includes consideration of all the relevant factors.'" (quoting 748 F. 3d, at 1266 (opinion of Kavanaugh, J.)."

2. The Threshold Character of the Section 112(n)(1) Finding and the Statutory Context Favors An Approach to Cost Consideration that Does not Bar Regulation Based on Projected Compliance Costs Unless Those Costs are Clearly and Unalterably Prohibitive

The fact that 112(n) contemplates a *threshold* determination made before regulations have been developed – as the Supreme Court phrased it, a "preliminary estimate" – should inform the proper role of cost in the "appropriateness" inquiry.

First, before regulations have been promulgated, determination of compliance costs can only be a rough and tentative estimate. It is a familiar feature of CAA regulation – including in the power sector – that actual compliance costs often turn out to be far lower than initial estimates, as companies, engineers, and markets begin to focus on how to deliver emissions reductions most cheaply and efficiently. Many Clean Air Act programs have ended up being dramatically less costly than has been projected by initial estimates – as engineers develop better solutions and companies find more efficient ways to reduce pollution. As Professor Percival explains:

> Experience also has demonstrated that cost estimates are frequently overstated while benefits are understated for several reasons. First, it is in the strategic interest of regulatory targets to exaggerate prospective costs in an effort to avoid regulation. Some of the most striking evidence of exaggerated cost projections is provided by the precipitous decline in the cost of reducing sulfur dioxide emissions under Title IV of the Clean Air Act. When the 1990 Amendments were debated, industry representatives projected that allowances to emit a ton of SO2 could sell for $1,000 to $1,500 per ton based on their estimates of the cost of installing pollution control equipment to achieve the emissions reductions required by Title IV. [citing Michael E. Porter and Claas van der Linde, Toward a New Conception of the Environment-Competitiveness Relationship, 9 J Econ Persp 97, 108 (Fall 1995)]. The EPA estimated that the

> reductions would cost around $750 per ton; actual costs have proven to be substantially lower. Early allowance sales were reported to have been made at prices ranging from $250 to $400 per allowance. When auctioned by the Chicago Board of Trade, spot allowances sold for average prices of $159 in 1994, $132 in 1995, and $68 in 1996. [citing *id*.] The low prices for which emissions allowances are selling demonstrates that industry estimates of the costs of complying with Title IV were greatly exaggerated.
>
> Another reason why ex ante cost estimates are often too high is that regulation can stimulate technological innovations that dramatically reduce control costs. For example, prior to the decision to phaseout ozone-depleting chlorofluorocarbons ("CFCs"), there was little incentive for industry to search for alternative substances that did not harm the ozone layer. After regulatory policy required dramatic reductions in CFC use, alternatives were found much more quickly and at far lower cost than previously expected. In 1988, when the U.S. had agreed to reduce CFC production by 50% by 1998, EPA estimated that this would cost $3.55 per kilogram. [EPA, Protection of Stratospheric Ozone, 53 Fed Reg 30604, 30607 (1988)] Four years later, when the phasedown had been broadened to encompass a complete ban by the year 2000, compliance costs had plunged to $2.20 per kilogram. [citing EPA, Protection of Stratospheric Ozone, 57 Fed. Reg. 31,242, 31,259 (1992)]. In similar fashion, the petroleum industry estimated in 1971 that phasing lead additives out of gasoline would cost $7 billion per year. [citing William G. Rosenberg, Clean Air Act Amendments, 251 Science 1546, 1547 (1991)]. In 1990, when 99 percent of the phaseout had been completed, costs had proven to be 95 percent less than estimated. [citing *id*.]

Robert V. Percival, *Regulatory Evolution and the Future of Environmental Policy*, 1997 U. Chi. Legal F. 159, 180-81 (1997) (footnotes omitted). EPA recognized there was clear evidence that the same pattern would hold true here. *See* 80 Fed. Reg. at 75,037-38 (discussing development and widespread deployment of cheaper controls than those identified in Utility Study, and noting EPA's belief that "many EGUs will use these approaches to reduce the cost of compliance with MATS"). *See id.* (noting, similarly, that "the cost to reduce acid gas HAP using SO2 controls has declined over time with the increased use of alternative technologies such as spray drier absorber and dry sorbent injection").[7]

Second, and relatedly, if a source category is listed so that section 112(d) regulation is required, whether as a result of a section 112(n) determination or otherwise, the resulting, regulation is accomplished in a manner that both considers costs and affords numerous means to avoid excessive costs or unreasonable burdens. *See* Part III below.

For these reasons, in order to determine that cost estimates render regulation "inappropriate" even though regulation would eliminate large volumes of toxic air pollution, EPA would have to find that the costs were not only currently truly prohibitive, but that costs

[7] As discussed in Part III below, evidence demonstrates that in fact the pattern did hold true here, and real-world compliance costs under MATS are a small fraction of the $9.6 billion annual figure estimated in 2011.

were likely to remain so even despite the focus of engineering talent and market forces that has brought about dramatic cost reductions in other instances. Furthermore, EPA would need to find that the mechanisms in section 112 for avoiding undue costs in connection with regulation would be ineffective to avoid those prohibitive costs.

3. Precedent Interpreting EPA's Obligation to Consider Cost in Setting Standards Under Section 111 Provides an Appropriate Model for Cost Consideration under Section 112(n)(1).

EPA properly has taken guidance from the large body of administrative and judicial precedent under CAA section 111. *See, e.g.*, Legal Memorandum 18-19.

Section 111(a)(1) directs EPA to "take into account" the cost of achieving emissions (as well as specified other factors) when setting "new source performance standards" (NSPS) for stationary sources that cause air pollution EPA has found may reasonably be anticipated to endanger public health or welfare. CAA sections 111(a)(1), 111(b)(1). Over more than four decades, the D.C. Circuit has fleshed out the meaning of this cost-consideration directive, rejecting interpretations that would require the agency to conduct a traditional cost-benefit analysis. *See, e.g., Essex Chem. Corp. v. Ruckelhaus*, 486 F.2d 427, 437 (D.C. Cir. 1973) (cost-benefit analysis was not required for acid mist standards); *Lignite Energy Council v. EPA*, 198 F.3d 930 (D.C. Cir. 1999) (EPA did not exceed its discretion in setting boiler standards that modestly increased the overall cost of producing electricity). In *Essex*, the court held that EPA's standards must be "reasonably reliable, reasonably efficient, and . . . reasonably . . . expected to serve the interests of pollution control *without becoming exorbitantly costly in an economic or environmental way*." 486 F.2d at 433 (emphasis added).

Similarly, in *Portland Cement Association v. Train* (*Portland Cement II)*, 513 F.2d 506, 508 (D.C. Cir. 1975), the court upheld EPA's interpretation that section 111's cost inquiry functions as a safety valve to ensure that the costs an NSPS imposes are not "greater than the industry could bear and survive," but would instead allow industry to "adjust" in a "healthy economic fashion to the end sought by the Act as represented by the standards prescribed." And in *Lignite*, the court held that "EPA's choice will be sustained unless the environmental or economic costs of using the technology are exorbitant." 198 F.3d at 933.

While courts have used varying formulations in discussing EPA's authority under section 111(a) to take costs "into account," each has followed the same fundamental standard: an NSPS will be upheld unless the costs it imposes are exorbitant or too great for the industry to bear. In fact, the D.C. Circuit has never invalidated an NSPS for being too costly. 79 Fed. Reg. at 1464. For example, in *Portland Cement Ass'n v. Ruckelhaus*, 486 F.2d 375, 387-88 (D.C. Cir. 1973) (*Portland Cement I*), the court upheld an NSPS for particulate matter emissions, even though control technologies amounted to roughly 12 percent of the capital investment for an entire new plant and consumed five to seven percent of a plant's total operating costs. Likewise, in *Portland Cement Ass'n v. EPA*, 665 F. 3d 177, 191 (D.C. Cir. 2011) (*Portland Cement III*), the court upheld particulate matter ("PM") standards that were anticipated to increase the cost of cement by one to seven percent, with little projected decrease in demand. *See also* 73 Fed. Reg. 34,072, 34,077, 34,086 (June 16, 2008). With respect to the electricity generating industry, the *Lignite Energy Council* court held that a two percent increase in the cost of producing electricity was not

exorbitant, and upheld the 1997 nitrogen oxides ("NO_X") NSPS for EGUs and industrial boilers. *See* 198 F.3d at 933 (citing 62 Fed. Reg. 36, 948, 36,958 (July 9, 1997)).

The cases under section 111 establish that regulation to control harmful pollution may proceed unless a very demanding showing of exorbitant or untenable costs is made. As EPA puts it: "Essentially, the D.C. Circuit has held that CAA section 111 requires EPA to consider whether the standards are reasonable for the industry as a whole to bear. *See Portland Cement Ass'n v. Train*, 513 F.2d 506 (D.C. Cir. 1975) (considering whether industry has shown an inability to adjust in a healthy fashion)." Legal Memorandum 19.

We agree that the large body of precedent and agency practice under section 111 is a good roadmap to giving effect to the cost component of the "appropriateness" inquiry under section 112(n)(1)(A). To the extent section 112(n)(1)(A) differs from section 111, it differs in ways that suggest that EPA should be even more reluctant under section 112(n)(1)(A) to decline to require control of dangerous pollution on the basis of cost:

First, under section 111(a), EPA considers cost in deciding how stringently to regulate, and requirements that section 111 be revisited allow for relatively weak standards to be tightened later. By contrast, under section 112(n)(1)(A), the question is whether to regulate the source category at all – the consequence of a cost-based override is not just somewhat less stringent regulation, but no regulation at all. The potential health and environmental decisions are both greater and less correctable than under section 111.

Second, in contrast to section 111(a), section 112 contains a separate, elaborate set of provisions, set forth in section 112(d), governing the setting of standards – in which cost place an explicit, prominent role, and that includes, as EPA notes, numerous mechanisms that can mitigate undue cost burdens on industry. Therefore, since section 112 provides additional protections against unduly costly regulation that section 111 does not, employing standards developed under section 111 for considering costs under section 112 is *a fortiori* adequately protective of regulated entities and society's interest in avoiding unreasonably costly regulation.

Third, section 112(n) addresses hazardous pollutants that Congress has determined to be especially harmful, and which are harmful in relatively small quantities, and for which impacts on sensitive populations are a particular concern. This supplies an additional reason why an approach to cost that have been repeatedly found lawful under section 111(a) is *a fortiori* adequately protective of regulated entities interests under section 112(n).

4. The Metrics Considered in EPA's Proposed Supplemental Finding – (1) Annual Compliance Costs as a Percentage of Power Sector Sales, (2) Annual Compliance Capital Expenditures Compared to the Power Sector's Annual Capital Expenditures, (3) Impact on the Retail Price of Electricity, and (4) Impact on Power Sector Generating Capacity – are Reasonable and Well-Suited to the Source Category Addressed in Section 112(n)(1)(A)

The Agency has proposed four metrics to determine whether the direct and indirect costs of compliance with MATS are reasonable: a "sales test," comparing annual compliance costs to electricity sales; a comparison of the costs to annual expenditures in the sector; the impact of the

standards on the retail price of electricity; and, impacts on generating capacity within the sector. 80 Fed. Reg. at 75,033-35. Those metrics are well-suited to the analysis required under section 112(n)(1)(A), as well as to the utility sector, and each supports the Agency's conclusion that the costs of the Standards are appropriate.

The 'sales test' demonstrates that the costs of the standards are very small, as compared to annual revenue of the electric utility sector. Moreover those costs are likely easier to bear for electric utilities than they would be for smaller entities. *See* 80 Fed. Reg. 75,033 n.28 (observing that the "sales test is often used by the EPA when evaluating potential economic impacts of regulatory actions on small entities."). The large size of most utilities allows them to adjust more easily to changes in costs, and has consistently led the industry to achieve compliance at lower costs than initially projected (as has proven the case with MATS). And electric utilities, in many cases, possess the ability to pass costs on to customers, with a guaranteed rate of return on any necessary investments, leaving them much better able to withstand cost increases. *See, e.g.*, Petition of Otter Tail Power Co., *In the Matter of Otter Tail Power Co.'s Petition for Approval of the Annual Update to Environmental Cost Recovery Rider Charge* (S.D. Pub. Utilities Comm'n) (noting approval of rate recovery, and that costs of MATS compliance was over 30% below initial estimate).[8]

A comparison with annual capital expenditures further indicates that the RIA's projected compliance expenditures are very small, in relation to both the typical capital expenditures undertaken each year by the utility industry, as well as typical year-to-year increases (and decreases) in such expenditures. Capital expenditures undertaken to comply with MATS may well all reduce future expenditures for the affected utilities; the required controls enable compliance with other Clean Air Act programs that have been promulgated since MATS, such as the sulfur dioxide NAAQS and Regional Haze programs.[9]

Likewise, the Agency's analysis demonstrates that on a regional and national basis, the Rule would produce very small increases in the retail price of electricity. As the Agency noted during its rule-making, improved energy efficiency policies substantially reduce those projected increases in retail price – and in the long term, could even produce a net decrease in the retail price of electricity. 76 Fed. Reg. at 25,074. Many states have adopted aggressive energy

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[8] *Available at* https://puc.sd.gov/commission/dockets/electric/2015/el15-029/petition.pdf.

[9] *See, e.g.*, 80 Fed. Reg 51,052, 51,062 (Aug. 21, 2015) (implementation schedule for 2016 round of SO2 (sulfur dioxide) nonattainment designations designed to allow states to "account for SO2 reductions that will occur over the next several years as a result of implementation of [other] requirements (such as [MATS])"); 80 Fed. Reg. 15,340, 15,349-50 & n.47 (Mar. 23, 2015); State Implementation Plan Regional Haze Periodic Progress Report for the State of Florida 17 (2015) (MATS, along with other federal regulations, will provide "extra assurances" of the required "reasonable progress" toward national visibility goals); Regional Haze 5-Year Periodic Review State Implementation Plan for North Carolina Class I Areas 24(2013) (same). See also Kentucky State Implementation Plan (SIP) Revision: Regional Haze 5-Year Periodic Report 2008-2013 for Kentucky's Class I Federal Area App. C-5, 4 (2014) ("[MATS] ... is one of the federal control measures . . . that is an important part of Kentucky's Regional Haze SIP.").

efficiency policies since EPA promulgated the standards, suggesting that those lower estimates of retail-price impacts are likely more accurate than the base projection in the RIA. *See*, *e.g.*, Order No. 87082 (Pub. Serv. Comm'n of Maryland) (increasing statewide efficiency target).[10]

Finally, the proposal notes that the Standards' costs are not of a magnitude likely to materially affect the power sector's generating capacity or electric reliability. We note that the Agency's Enforcement Response Policy, announced together with the final rule, provides further assurance against any unreasonable effects on power generation.

As discussed in further detail below in Part III and in comments submitted by Calpine Corporation, *et al*., recent data confirm the conclusion that the costs of regulation are reasonable, as measured by each of these metrics.

II. THE CONSIDERATION OF COST AS PART OF THE SECTION 112(n)(1)(A) "APPROPRIATENESS" DETERMINATION PROPERLY TAKES INTO ACCOUNT THE EXTENSIVE MECHANISMS IN SECTION 112(d) FOR ENSURING AGAINST EXCESSIVE COSTS

The Proposed Supplemental Finding notes that the standard-setting process which follows EPA's appropriate and necessary finding under section 112(n)(1)(A) contains several statutory mechanisms which ensure that the costs of regulation remain reasonable, including minimum standards (section 112(d)'s floor) based on emissions achieved by currently operating plants, and the availability of sub-categories to accommodate units of different type, class and size. 80 Fed. Reg. at 70,039. Those statutory accommodations of costs support EPA's finding, by assuring that the decision to regulate imposes proportionate costs, evidenced by the real-world achievement of currently operating plants. And they further suggest a congressionally-struck balance between the harms of hazardous air pollution, and the costs appropriate to reduce them, to which EPA should properly defer.

As the Agency correctly notes in the Proposed Finding, "section 112(d) ensures that the MACT floor level of control is … presumptively cost reasonable because it is based on the level of control actually achieved by existing sources in the same category or subcategory." 80 Fed. Reg. at 75,039. Nothing in the statute or its history suggests that Congress though the electric utility sector unique in any fashion that might cause application of the HAP control provisions to instead produce inappropriate or disproportionate costs. And, in fact, the analysis developed by the Agency to support its floor-level standards confirms that the electric-utility sector has no special attributes that might disable the various mechanisms by which section 112(d) implicitly and explicitly addresses costs, or otherwise render it inappropriate to apply section 112's standard-setting regime to electric utilities. *See* Legal Memorandum 26-31 (describing elements of rule-making that ensure cost-feasibility).

[10] *Available at* http://www.psc.state.md.us/wp-content/uploads/Order-No.-87082-Case-Nos.-9153-9157-9362-EmPOWER-MD-Energy-Efficiency-Goal-Allocating-and-Cost-Effectiveness.pdf.

First, the Agency's floor analysis produced best performing units that are fairly representative; they use no unusual or cost-prohibitive pollution-control methods. EPA found that the best performing units from which the agency created its existing-unit floors were utilizing: fabric filters, carbon injection, and flue gas desulfurization. 76 Fed. Reg. at 25,046. Each of those controls is cost-effective, readily available, and in widespread use, even by units that were not among EPA's best performers. The mechanism prescribed by the statute – looking to the best-performing sources to establish a reasonable minimum level of control – did not prescribe pollution reductions requiring exotic or excessively expensive controls.

The Supreme Court's decision in *Michigan* expressed some concern that the floor might demand installation of controls imposed only as a result of "cost-blind regulation," 137 S. Ct. at 2,711. But the controls required to meet the floors reflect cost-conscious standards. Fabric filters, electro-static precipitators and flue gas desulfurization have been routinely demanded by the Act's Prevention of Significant Deterioration provisions, under a standard that requires, *inter alia*, consideration of "economic impacts and other costs." 42 U.S.C. 7479(3) (describing 'best available control technology' standard)[11]; and pursuant to the Agency's New Source Performance Standards, which similarly demand consideration of cost, 42 U.S.C. 7411(a)(1), *see* 76 Fed. Reg. at 25,060 (finding that fabric filters and flue gas desulfurization are proper basis for new source performance standards).[12] Activated carbon injection has similarly been installed as the result of cost-conscious, rather than cost-blind, regulation. The Texas Commission on Environmental Quality, for example, has confirmed that activated carbon injection is "technically practicable and economically reasonable to control mercury emissions," and required it on that basis. Texas Commission on Environmental Quality Findings of Fact and Conclusions of Law Approving Preconstruction Permit for White Stallion Energy Center (Oct. 19, 2010) at 40, attached as Ex. 9.

Second, the remainder of the sector (within each of the Agency's subcategories) comprises units of sufficiently similar design to readily achieve the floor levels established by those best performers. According to the data collected by the Agency, many plants outside the best performers had emissions below the standards promulgated by EPA. *See* NGO Comments at VIII-5 (noting that 60 to 70 percent of units submitted data indicating that their emissions were below individual floor-level standards). Furthermore, EPA identified 64 units that were meeting *all* of its standards, prior to regulation. 77 Fed. Reg. at 9,388. And the Energy Information

[11] For examples, *see* U.S. EPA Technology Transfer Network, RACT/BACT/LAER Clearinghouse (accessed January 4, 2016) (listing facilities at which technologies have been installed pursuant to PSD program)

[12] Even the new-source standards demand control technologies that are readily available, and which were routinely demanded by cost-conscious regulatory standards in recently built plants, even before the Standards became effective (activated carbon injection, a fabric filter or high efficiency electrostatic precipitator, and flue gas desulfurization, each operating at a higher level of efficiency). *See* Memorandum from Nick Hutson dated November 16, 2012 (Analysis of Control Technology Needs for Revised Proposed Emission Standards for New Source Coal-fired Electric Utility Steam Generating Units).

Agency estimated that even by the end of 2012, 64% of plants had all the controls in place necessary to comply. http://www.eia.gov/todayinenergy/detail.cfm?id=15611. Such rapid compliance hardly suggests onerous, or even substantial, costs. More broadly, the record demonstrates that the electric utility sector is not so wildly diverse as to prevent broad, cost-effective compliance with the emissions reductions achieved by the best-performing units within each subcategory.[13]

Third, the record indicates that MATS does not pose unreasonably disproportionate costs, on a per-ton (or per-pound) basis, when compared to the regulations governing other major sources of air toxics (regulations which Congress manifestly deemed appropriate). As EPA noted in its proposal, on a cost-per-ton basis, the reductions achieved by the standards are within the same range as those achieved by other section 112 rules, 76 Fed. Reg. at 25,075.

The record thereby confirms that the utility sector has no unique attributes that render the statutory standard-setting mechanism unreasonable, from a cost-based perspective. The best-performers provided an effective benchmark for what other units could achieve at reasonable cost, further supporting the Agency's conclusion that it is appropriate to subject electric generating units to the section 112(d) regulatory regime.

III. WHILE REGULATION IS CLEARLY APPROPRIATE USING THE COMPLIANCE COSTS AS ESTIMATED BY EPA IN 2011 -- AND IT IS REASONABLE TO RELY ON THE 2011 RECORD IN MAKING THE SUPPLEMENTAL FINDING -- ACTUAL EXPERIENCE HAS DEMONSTRATED THAT COMPLIANCE COSTS ARE IN FACT DRAMATICALLY LOWER THAN ESTIMATED

Under section 112(n)(1)(A), the "appropriate and necessary" finding for power plants is made before actual regulations are promulgated and well before they go into effect. Consistent with this statutory structure, it was reasonable for EPA to rely upon its previous RIA compliance cost estimates in its Proposed Supplemental Finding.[14] The RIA cost estimates incorporated the actual MATS regulations as the compliance target, so they are much more reliable than the type of pre-regulatory estimate anticipated by the statute. And as EPA's Proposed Supplemental Finding correctly concludes, the regulation of EGUs' emissions of HAPs is clearly warranted under the $9.6 billion/year estimate employed in the RIA and in EPA's analysis.

Yet now, EPA has not only promulgated actual regulations, but it also has information about how a significant segment of the regulated industry is complying with those regulations. Under these circumstances, EPA should take notice of the information about compliance costs

[13] Where EPA felt that certain units' idiosyncrasies were not reflected by the best performers, the Agency created a subcategory for those units, to ensure that the statutory mechanism functioned in a cost-reasonable fashion. *See* Legal Memorandum at 28 (noting that because no units burning low-rank virgin coal were among best performers, EPA created subcategory).

[14] 80 Fed. Reg. at 75,032.

that can be gleaned from ongoing implementation and compare that against its 2011 projection of annual MATS compliance costs of $9.6 billion – at least for the purpose of checking the validity of the cost projections upon which the RIA was based.[15] While current compliance information and associated cost estimates are necessarily provisional, EPA can and should conclude that, by each relevant metric, recent data on costs provide further support for the conclusion that the costs of MATS are reasonable. Experience with the rule shows that EPA likely took a conservative approach, significantly overestimating the costs of MATS compliance, and further reinforces the conclusion that it is appropriate to regulate hazardous air pollution from power plants after considering the costs.

Approximately 59% of power plants subject to MATS have been complying since April 2015,[16] and there is now publicly available information on the specific control technologies being deployed at those plants.[17] The approximately 41% of remaining affected power plants have submitted information to their state regulatory authorities concerning their plans to comply by April 2016, including, in many cases, information about the specific control technologies that they plan to use.[18] Therefore, it is possible to compare the actual deployment of particular control technologies and fuels – and their associated capital and operating costs – under MATS to the RIA's projections of control technology deployment, fuel use, and associated capital and operating costs. Andover Technology Partners recently performed such an analysis and estimated that the actual costs of MATS compliance is approximately $2 billion, or less than one-quarter of EPA's $9.6 billion estimate.[19] This more comprehensive analysis is further supported by public statements of some affected utilities showing that actual compliance costs have been much lower than initial projections.[20] There are, in other words, strong indications

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[15] MATS RIA at 3-31.

[16] National Association of Clean Air Agencies, *Survey on MATS Compliance Extension Requests* (Aug. 11, 2015), *available at* http://www.4cleanair.org/news/details/nacaa-collects-data-mats-extension-requests.

[17] *See* Andover Technology Partners, *Review and Analysis of the Actual Costs of Complying With MATS in Comparison to Predicted in EPA's Regulatory Impact Analysis* at 3, attached as Ex. 10 ("ATP Report") (using EPA's Air Markets Program Data to determine what control technologies are being deployed to comply with MATS).

[18] *See* M.J. Bradley & Associates LLC, *MATS Compliance Extension Status Update* (June 24, 2015) (reviewing MATS compliance extension requests to determine compliance strategies at individual units), *available at* http://www.mjbradley.com/reports/mats-compliance-extension-status-update .

[19] *See* ATP Report at 1.

[20] *Compare* FirstEnergy, November 2011 Earnings Call Transcript, Anthony Alexander, CEO (initialing projecting MATS compliance costs of $2 billion to $3 billion), *available at* http://seekingalpha.com/article/304211-firstenergys-ceo-discusses-q3-2011-results-earnings-call-transcript?page=2, *with* FirstEnergy, 2014 U.S. SEC Form 10-K at 48 (stating that "[s]everal new opportunities to lower costs were identified in 2014, and FirstEnergy's total cost for MATS compliance is expected to be approximately $370 million"), *available at* https://www.firstenergycorp.com/content/fecorp/investor/sec_filings_other_financial_reports/annual_reports_proxy_statements.html; *compare* AEP, February 2013 Earnings Call Transcript, Nicholas Akins, CEO

that MATS is another instance in which actual compliance costs are dramatically lower than pre-implementation estimates.

These actual costs establish that the Agency's decision to use the RIA represents a very conservative choice from a cost-perspective; an *ex post* assessment would only produce lower compliance costs, and further support the Agency's determination. Further, these actual costs confirm the utility industry's capacity (well demonstrated before MATS) to comply with regulatory requirements at vastly lower costs than initially predicted, additionally supporting the appropriateness of regulation here. Similarly, EPA should take notice of the fact that recent data confirm its conclusion that the costs of MATS are reasonable because they do not threaten the ability of the electric generation industry to provide reliable electric power. Recent analysis conclude that MATS is "not expected to have a significant system-wide impact" on electric reliability and is "not likely to significantly impact costs for consumers".[21] MATS is "expected to affect the economics of at most a small number of units and thus [is] not expected to have a significant system-wide impact."[22] As the North American Electric Reliability Corporation concluded in its State of Reliability report in May 2015, "reliability performance continued to remain high, sustaining the positive trends documented" in the prior year.[23]

(initialing projecting MATS compliance costs of $5 billion to $7 billion), *available at* http://seekingalpha.com/article/1188551-american-electric-power-management-discusses-q4-2012-results-earnings-call-transcript, *with* AEP, 2014 U.S. SEC Form 10-K at 10 (estimating costs to comply with federal environmental rules of between $2.8 billion and $3.3 billion), *available at* https://www.aep.com/investors/FinancialFilingsAndReports/Filings/.

[21] Electric Reliability Council of Texas (ERCOT), Impacts of Environmental Regulations in the ERCOT Region (Dec. 16, 2014) at ES-2, *available at* http://www.ferc.gov/CalendarFiles/20150327133925-Lasher,%20ERCOT.pdf.

[22] *Id.*

[23] North American Electric Reliability Corporation, State of Reliability 2015 (May 2015) at 6, *available at* http://www.nerc.com/pa/RAPA/PA/Performance%20Analysis%20DL/2015%20State%20of%20Reliability.pdf.

IV. WHILE THE STATUTE DOES NOT REQUIRE THE USE OF A FORMAL BENEFIT-COST ANALYSIS, THE BENEFIT-COST ANALYSIS THAT EPA PERFORMED IN THE RIA FURTHER AND INDEPENDENTLY CONFIRMS THE APPROPRIATENESS OF REGULATION; ALL COSTS AND BENEFITS – INCLUDING BENEFICIAL HEALTH EFFECTS OF REDUCING NON-HAP EMISSIONS – SHOULD BE CONSIDERED IF BENEFIT-COST ANALYSIS IS USED TO ASSESS WHETHER REGULATION IS "APPROPRIATE"

While, as discussed above, the Supreme Court's ruling in *Michigan* made clear that a benefit-cost analysis is not *required* of EPA when it "considers" the cost of HAP regulation, EPA's RIA analysis provides further, independent grounds demonstrating the reasonableness of regulation. The 2011 final RIA could also be considered to provide an additional, alternative basis for concluding that section 112(d) regulation is "appropriate" for coal- and oil-fired power plants.

As provided in the OMB's Circular No. A-4, *Regulatory Analysis* (Sept. 17, 2003) ("Circular A-4") and other authorities, a proper benefits-cost analysis for a regulation of this kind must consider *all* benefits and costs. See Part IV.B, below. Here, the initial decision is whether to impose a specific level of control (which in this instance is driven by the text of the statute) on multiple pollutants,. For that reason, if a benefit-cost analysis informs the cost consideration under section 112(n) demanded by the Court in *Michigan*, it is appropriate that all of the health (and environmental) benefits of regulating HAP under 112(d) must be considered, as well as all of the costs of this level of regulation. That is so, whether the total benefit analysis is framed or described as an assessment of "direct" plus "co-benefits" or whether all the benefits of the required level of control are considered simply as benefits or avoided costs of the regulation (including the health benefits of the reduced levels of secondary particulates resulting from the application of controls to meet the acid gas limitations required by section 112(d)).

A. A Proper and Complete Benefits Analysis Must Consider All of the PM Reductions Associated with Applying Section 112(d) to Control Coal- and Oil-Fired Power Plant HAP Emissions.

A decision that it is "appropriate" and necessary to regulate power plants under section 112(n)(1)(A) is a listing decision triggering regulation under section 112(d). The finding reached by EPA in 2000 placed coal- and oil-fired power plants on the list of industries for which section 112(d) standards are required. *New Jersey v. EPA*, 517 F.3d 574, 578 D.C. Cir. 2008). Section 112(d) standards in turn require emissions limits for all HAPs listed by Congress in section 112(b). *National Lime Ass'n v. EPA*, 233 F.3d 625, 634 (D.C. Cir. 2000) (*Nat'l Lime II*) (section 112(d) standards include a clear obligation to set emissions limits for each listed air toxic).

The Agency may, however, set section 112(d) emissions standards for "surrogate pollutants," rather than emissions limits for each HAP, where the identified HAP is "invariably present" in the surrogate pollutant, methods to control or capture the surrogate pollutant "indiscriminately capture the listed HAP as well," and surrogate controls are the "only means"

by which facilities can control the listed HAP. *Sierra Club v. EPA*, 353 F.3d 976, 984 (D.C. Cir. 2004) (quoting *Nat'l Lime II*, 233 F.3d at 639).[24]

In MATS, EPA established alternative final standards for the non-mercury metal HAPs emitted by coal- and oil-fired power plants. 77 Fed. Reg. at 9,380. The Agency finalized a choice between meeting standards based on total metals, filterable particulates, or metal-specific emissions levels. Tables 1 & 2 to subpart UUUU of [40 C.F.R.] Part 63—Emission Limits for New & Existing EGUs, 77 Fed. Reg. at 9,487-9,493. Filterable particulates, controlled with electrostatic precipitators or baghouses, were the surrogacy choice for metal HAPs, rather than the total particulates metric proposed by the Agency, because EPA explained, "[m]ost of the non-mercury metal HAP, for which PM is a surrogate, are filterable PM and the one that is not (Se [selenium]) is well controlled by the limit on acid gases."[25] 77 Fed. Reg. at 9,380.

EPA also established alternative final standards for the acid gas HAPs emitted by coal- and oil-fired power plants, under which coal-fired units can meet HCl or SO2 standards, as surrogates for HCl, HCn, Cl_2, and HF.[26] Again, EPA found the surrogacy relationship was met because the controls for SO_2 and HCl – wet and dry scrubbers – also control the acid gas HAP (and volatized selenium as well, as discussed above).

In order to evaluate the health benefits of controlling EGU HAPs, it is necessary to understand that all of these pollutants are emitted in a complex mixture – essentially an acid soup containing SO2, NOx, particulate matter containing metals, and other pollution. See EH&E Report at 30-31, 35-36 (listing control technologies and noting the variety of pollution including air toxics they control, and noting the health benefits likely due to the need for multiple kinds of controls, and resulting multi-pollutant emission reductions from EGUs due to MATS). EPA concluded that the relationships between particles and metals, and between sulfur dioxide and acid gases, and the fact that controls for conventional pollutants also control toxics, makes surrogacy regulation legally appropriate. 77 Fed. Reg. 9,380. EPA's findings about surrogacy have survived review.

More pollutants are created after the uncontrolled mixture is emitted. The plume conditions allow formation of secondary particulates (PM2.5) that are created by chemical reactions between the components of the emissions, particularly sulfur dioxide and acids, in the atmosphere beyond the stack. Dockery Declaration ¶ 7; Levy Declaration ¶ 7; *C.f.* EPA-HQ-OAR-2009-0234-20506, "National Acid Precipitation Assessment Program Report to Congress" (noting that the pollutants that contribute to acid deposition also lead to the formation of fine particles and gases that harm public health). These occur because of the chemistry of the emissions, even where direct particulate matter controls (ESPs or baghouses) are reducing

[24] The surrogate must also allow EPA to identify "the best achieving sources, and what they can achieve" with respect to hazardous air pollutants. *Sierra Club*, 353 F.3d 976, 985 (D.C. Cir, 2004).

[25] The final filterable PM standard therefore does not function as a surrogate for all selenium emissions, because that metal occurs in volatized form in post-stack emissions and is not captured by PM controls. Acid gas controls – scrubbers, primarily – do control volatized selenium, although the Agency has not described SO2 or HCl as a surrogate pollutant for selenium.

[26] EPA sets distinct HCl and HF standards for oil-fired units.

primary particulates. Controlling directly emitted or primary particles, alone, therefore will not result in controlling secondary particulate matter air pollution. But controlling SO2 (the surrogate for acid gas pollution) does control secondary particles by eliminating or reducing their precursors. And the health benefits of lower levels of secondary particles in the ambient air are well characterized, and monetizable. Dockery Declaration ¶ 8 (particulate matter public health effects well-understood); EPA RIA Chapter 5.

The total benefits achieved by controlling HAP under section 112(d) standards therefore include the benefits of controlling primary particles on which non-mercury metal HAP are found, but also the benefits associated with lower ambient secondary particulates due to the application of controls to meet the acid gas limits. The monetized health benefits of that lower level of secondary particulate matter are the result of the power plant standards; even if they are not the "primary objective of the rule," 77 Fed. Reg. at 9,305, they are inescapable consequences of the decision to regulate. Whether described as "ancillary" or "co-benefits" or as direct benefits of the rule, the health benefits of reducing levels of ambient secondary particulates are an unavoidable result of EPA's (legally valid) regulatory choices in pursuit of reductions in section-112 listed HAPs, and must "count" in any benefit-cost assessment.

B. Any Cost-Benefit Analysis Used to Inform the "Appropriateness" Inquiry Under 112(n)(1) Should Account for All Costs and All Benefits – Including Health Impacts Associated with Increases or Decreases in Emissions of Non-HAP Pollutants

It is clearly reasonable for EPA to consider the ancillary health and environmental benefits (and costs) of regulating EGUs' HAP emissions in determining whether such regulation is "appropriate" under section 112(n)(1)(A). Doing so is consistent with well settled principles of regulatory analysis that have been supported by multiple consecutive presidential administrations, and well as scholarly opinion. And just as EPA could not (as the *Michigan* majority noted) reasonably find regulation of EGUs "appropriate" if the real-world effect of regulating HAPs were to cause even greater health harms, it would be unreasonable for EPA to exclude from the "appropriateness" inquiry the fact that HAP regulation of EGUs will bring massive public health and environmental benefits via reductions in non-HAP pollutants caused by the use of pollution control technologies that most cost-effectively control HAPs. Even though, as noted above, the benefits of the major reductions of HAPs are alone easily sufficient to make regulation "appropriate," these ancillary or coincidental benefits strongly reinforce the conclusion that regulation is "appropriate."

1. Coincidental or Ancillary Costs and Benefits are Included in a Properly Conducted Cost- Benefit Analysis

Including indirect costs and benefits is consistent with well settled principles of policy analysis and currently applicable Executive Orders and guidance. Throughout the history of regulatory review, Presidential administrations of both parties have stressed that regulatory analysis should focus on the overall societal benefits and costs expected to come from regulatory

action.[27] This focus continued in Executive Order 12,866, issued by President Clinton in 1993, which requires assessment of "*all* costs and benefits of available regulatory alternatives." 58 Fed. Reg. 51,735 (Oct. 4, 1993).

In a guidance document circulated to agencies in 1996, the OMB told agencies that "an attempt should be made to quantify *all potential real incremental benefits to society* in monetary terms to the maximum extent possible" including any interaction effects between different federal regulations. Office of Management and Budget, "Economic Analysis of Federal Regulations under Executive Order 12866," January 11, 1996 at III (B) (emphasis added). In 2000, OMB directed agencies to consider benefits that are indirectly traded in markets (like health and safety risks and "use" values of environmental resources), and even benefits that have no tradable economic value at all, like the existence value of environmental or cultural resources. Office of Management and Budget, "Guidelines to Standardize Measures of Costs and Benefits and the Format of Accounting Statements," 10, 11 (March 22, 2000).

The George W. Bush Administration took this commitment to full accounting of societal effects a step further in the most formal – and still governing – guidelines for agency RIAs, issued in OMB Circular A-4 in 2003. Circular A-4 was intended to "standardiz[e] the way benefits and costs of Federal regulatory actions are measured." Circular A-4 at 1. The Circular instructs agencies to consider "any important" indirect benefits, which include any "favorable impact . . . secondary to the statutory purpose of the rulemaking," and recommends that agencies use the "same standards" for assessing indirect and direct benefits. *Id.* at 26. Circular A-4 instructs that, in cost-benefit analysis, agencies must:

> Identify the expected undesirable side-effects and ancillary benefits of the proposed regulatory action and the alternatives. These should be added to the direct benefits and costs as appropriate. . . A complete regulatory analysis includes a discussion of non-quantified as well as quantified benefits and costs.

Id. at 3. The Circular emphasizes that agencies are to "look beyond the direct benefits and direct costs of your rulemaking" to include "any important ancillary benefits and countervailing risks." Circular A-4 at 26. The limitations on considering or valuing co-benefits are no different than for any other kind of effect – *i.e*., data and methodological limitations that undermine certainty. *Id*. Considering them at an initial level, though, is essential to proper analysis since "in some cases the mere consideration of these secondary effects may help in the generation of a superior regulatory alternative with strong ancillary benefits and few countervailing risks." *Id*.

Currently, EPA and other federal agencies, in conducting regulatory review of proposed regulations, are required to take indirect costs and benefits into account. Executive orders governing regulatory review call for agencies accurately to measure the "actual results of regulatory requirements" and explicitly require analysis of both direct and indirect costs and

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[27] *E.g.*, George P. Shultz, Director, Office of Management and Budget, "Memorandum for the Heads of Departments and Agencies: Agency regulations, standards, and guidelines pertaining to environmental quality, consumer protection, and occupational and public health and safety," (Oct. 5, 1971); Exec. Order 12,044, 43 Fed. Reg. 12,661 (Mar. 24, 1978); Exec. Order No. 12,291, 46 Fed. Reg. 13,193 (Feb. 17, 1981).

benefits. Exec. Order No. 13,563 § 1, 76 Fed. Reg. 3821, 3821 (Jan. 21, 2011) (affirming Exec. Order No. 12,866); *accord.* Exec. Order No. 12,866 § 6(a)(3)(C), 58 Fed. Reg. 51,735, 51,741 (Oct. 4, 1993) (detailing the requirements for cost-benefit analysis). The executive orders treat indirect benefits in parity with indirect costs.

EPA's own cost-benefit guidelines, adopted after extensive peer review, likewise instruct the agency to assess "all identifiable costs and benefits," including both direct effects "as well as ancillary [indirect] benefits and costs." EPA, *Guidelines for Preparing Economic Analyses* at 11-2 (2010). The assessment of both direct and indirect effects is needed to "inform decision making" and allow meaningful comparisons between policy alternatives. *Id.* at 7-1.

Accordingly, under multiple administrations of both parties, EPA has consistently taken indirect benefits into account when evaluating Clean Air Act regulations. See, *e.g.*, 52 Fed. Reg. 25,399, 25,406 (July 7, 1987) (in proposing new NSPS for municipal waste combustors, EPA noting intent to "consider the full spectrum of the potential impacts of regulation," including "indirect benefits accruing from concomitant reductions in other regulated pollutants"); 56 Fed. Reg. 24,468, 24,469 (May 30, 1991) (in proposing performance standards for landfill gases, justifying the regulation partly on "the ancillary benefit of reducing global loadings of methane"); 63 Fed. Reg. 18,504, 18,585-86 (Apr. 15, 1998) (analyzing the indirect benefits of reducing co-pollutants like volatile organic compounds, particulate matter, and carbon monoxide from emissions standards addressing hazardous pollutants from pulp and paper producers); 72 Fed. Reg. 8428, 8430 (Feb. 26, 2007) ("Although ozone and PM2.5 are considered criteria pollutants rather than 'air toxics,' reductions in ozone and $PM_{2.5}$ are nevertheless important co-benefits of this proposal."); 75 Fed. Reg. 51,570, 51,578 (Aug. 20, 2010) (considering indirect benefits of regulating HAP from combustion engines). *See also* Albert L. Nichols, "Lead in Gasoline," Chapter 4 in *Economic Analyses at EPA* 74 (ed. Richard D. Morganstern) (Resources for the Future 1997) (discussing EPA's consideration of co-benefits in Reagan Administration rulemakings addressing lead in gasoline); Peter Caulkins and Stuart Sessions, "Water Pollution and the Organic Chemicals Industry," Chapter 5 in *Economic Analyses at EPA* 113-14 (ed. Richard D. Morganstern) (Resources for the Future 1997) (addressing collateral air pollution harms (VOC emissions) produced by water pollution control at wastewater treatment plants); James Hammitt, "Stratospheric Ozone Depletion," Chapter 6 in *Economic Analyses at EPA* 153 (ed. Richard D. Morganstern) (Resources for the Future 1997) (discussing EPA's analysis of ground-level ozone reduction co-benefits of measures to address depletion of ozone layer in upper atmosphere).[28] Ancillary effects like reducing (or increasing) emissions of other pollutants are part of any proper cost-benefit analysis.

Case law also strongly supports the conclusion that ancillary health benefits and costs, where identifiable, should be included in an agency's cost-benefit analysis. For example, in *Am.*

[28] Leading scholars and treatises similarly instruct that ancillary, coincidental, or collateral benefits and costs should be considered in a properly performed benefit-cost analysis. *E.g.*, E.J. Mishan & Euston Quah, Cost Benefit Analysis 104 (5th ed. 2007); Richard L. Revesz & Michael A. Livermore, Retaking Rationality 55–65 (2008); Christopher C. DeMuth & Douglas H. Ginsburg, Rationalism in Regulation, 108 Mich. L. Rev. 877, 888 (2010); Kenneth J. Arrow, *et al.*, Benefit-Cost Analysis in Environmental, Health, and Safety Regulation: A Statement of Principles 6-7, 15 (1996).

Trucking Ass'ns v. EPA, the court held that EPA must consider both the direct and indirect effects of pollutants, rather than only "half of a substance's health effects." 175 F.3d 1027, 1051–52 (D.C. Cir. 1999), *rev'd on other grounds sub nom. Whitman v. Am. Trucking Ass'ns, Inc.*, 531 U.S. 457 (2001); *see also Corrosion Proof Fittings v. EPA*, 947 F.2d 1201, 1225 (5th Cir. 1991) (holding that EPA must consider the indirect safety effects of substitute options for car brakes when banning asbestos-based brakes under the Toxic Substances Control Act). See also *Competitive Enterprise Inst. v. Nat'l Highway Traffic Safety Admin.*, 956 F.2d 321, 326-27 (D.C. Cir. 1992) (striking down National Highway Traffic Safety Administration rule for failing to consider whether benefits from more fuel-efficient cars outweighed the potential increased safety risks"); *Am. Dental Ass'n v. Martin*, 984 F.2d 823, 826-27 (7th Cir. 1993) (remanding in part an Occupational Safety and Health Administration regulation for failure to consider indirect costs). An artificially constrained view is especially unsuitable, given the capacious nature of the word "appropriate." *See* section IV.B.2, below.

It would be especially perverse to conclude that co-benefits of HAP regulation cannot be considered in weighing whether regulation is "appropriate," given that section 112(n)(1) itself was predicated upon the recognition that environmental regulations aimed at one set of pollutants may have significant collateral benefits by simultaneously reducing emissions of another set of pollutants. As EPA notes, a central premise of the special regime for EGUs under section 112(n)(1) was that the Acid Rain Program adopted as part of the 1990 Amendments would have the effect of reducing not just the criteria pollutants (sulfur dioxides and oxides of nitrogen) that were the target of that program and responsible for the acid rain problem, but also would serve to reduce emissions. Legal Memorandum 12, 24-25. Thus, "co-benefits" were at the core of the reasons for the provision's adoption; and Congress recognized that regulation would turn on whether toxic emissions were adequately controlled as a co-benefit of programs directed at other provisions.

Furthermore, the legislative history of the 1990 Clean Air Act Amendments indicates that Congress specifically contemplated that "[w]hen establishing technology-based standards" to regulate hazardous air pollutants under Section 112(d), EPA would "consider the benefits which result from control of air pollutants that are not listed but the emissions of which are, nevertheless, reduced by control technologies or practices necessary to meet the prescribed limitation." S. Rep. No. 101-228, at 172 (1989). Congress noted that these "other compounds, although not listed [under section 112], would be precursors of ozone pollution," and their "control, even in attainment areas, may produce substantial health and environmental benefits." *Id.*

Nor is there any basis to ignore real co-benefits of reducing pollutants merely because they happen to be the subject of regulation under state and federal plans to implement the National Ambient Air Quality Standards (NAAQS). As EPA noted in the Rule:

> It is important to emphasize that NAAQS are not set at a level of zero risk. . . . While benefits occurring below the standard may be less certain than those occurring above the standard, EPA considers them to be legitimate components of the total benefits estimate.

77 Fed. Reg. 9304, 9431 (Feb. 16, 2012). EPA's analysis in the RIA was based upon an extensive review of peer-reviewed epidemiological studies as well as expert opinion requested by EPA concerning health effects of particulate matter. MATS RIA at 5-26 to 5-27. The scientific literature and expert responses support using a no-threshold model, MATS RIA at 5-98; 77 Fed. Reg. at 9430, meaning that there is no concentration above zero (including concentrations below the NAAQS) for which health risks do not exist. These are real benefits – including real premature deaths and serious illnesses avoided as a result of MATS – and there is no legitimate basis for ignoring them as part of EPA's determination whether regulation is appropriate.

2. The Text of Section 112(n)(1)(A) and the Logic of the *Michigan* Opinion Strongly Support Considering All Health and Environmental Benefits of Regulation, Including "Ancillary" Benefits

While it is conceivable that a particular statute could mandate a narrower inquiry than that of orthodox, best-practices approach reflected in OMB Circular A-4, scholarly treatises, and other sources, that is certainly not the case with the statute here. As the Supreme Court emphasized in *Michigan*, the term "appropriate" is an encompassing, and is naturally read to require consideration of the costs of regulation. 135 S. Ct. at 2707 ("In particular, 'appropriate' is 'the classic broad and all-encompassing term that naturally and traditionally includes consideration of all the relevant factors.'") (quoting Judge Kavanaugh's partial dissent, 748 F. 3d at 1266. Nothing in this term supports the view that, if it opted to consider cost under section 112(n)(1) by means of a cost-benefit analysis, EPA should or may ignore some categories of real, well documented health benefits, or should depart from established principles governing cost-benefit analysis that are expressed in executive orders and guidance.

Indeed, the Supreme Court majority's explanation that negative health side effects of regulation are inevitably a part of the "appropriateness inquiry" strongly supports the conclusion that the correlative positive side effects of regulation must be considered. The *Michigan* decision discusses a hypothetical scenario raised at oral argument (which is not presented by the actual record in this case) in which regulation of power plants under section 112 would involve technologies that control emissions of HAPs but, at the same time, cause *harms* to human health which are greater in magnitude than the health benefits from reducing HAPs:

> EPA's interpretation precludes the Agency from considering any type of cost – including, for instance, harms that regulation might do to human health or the environment. The Government concedes that if the Agency were to find that emissions from power plants do damage to human health, but that the technologies needed to eliminate these emissions do even more damage to human health, it would still deem regulation appropriate. See Tr. of Oral Arg. 70. No regulation is "appropriate" if it does significantly more harm than good.

135 S. Ct. at 2707.

The logic of the Supreme Court's decision strongly supports considering not just indirect health costs, but also indirect health benefits (which may also be characterized as avoided health costs). *See also* 135 S. Ct. at 2707 (observing that "any disadvantage could be termed a cost"). Just as the prospect of serious adverse health impacts would bear on "appropriateness" of regulation under 112(n)(1)(A), so do does the prospect of major positive health impacts caused by, the Court's phrase, "the technologies needed to eliminate these [HAP] emissions." The breadth of the term at issue "appropriate," combined the the strong tradition in administrative judicial precedent and scholarly analysis, makes clear that all consequences of regulation – positive and negative – are properly considered in a benefit-cost analysis of the potential EGU HAP regulation.

Particularly given the breadth of the term "appropriate," and the specific indications in the text and history of the 1990 Amendments that Congress was aware of the potential of significant health co-benefits, there is no basis to conclude that Congress meant to mandate an unusual, gerrymandered kind of benefit-cost analysis – sharply at odds with cost-benefit best practices – in which indirect *costs* factor into the analysis but indirect *benefits* are excluded. No reason exists to include indirect costs but exclude indirect benefits, since the two "are simply mirror images of each other." Samuel J. Rascoff & Richard L. Revesz, *The Biases of Risk Tradeoff Analysis: Towards Parity in Environmental and Health-and-Safety Regulation*, 69 U. Chi. L. Rev. 1763, 1793 (2002). Agencies must treat costs and benefits alike, and may not "put a thumb on the scale by undervaluing the benefits and overvaluing the costs of more stringent standards." *Ctr. for Biological Diversity v. Nat'l Highway Traffic Safety Admin.*, 538 F.3d 1172, 1198 (9th Cir. 2008). Under the executive orders on regulatory analysis, Circular A-4, and EPA's own guidelines, indirect benefits must be counted "equivalently" with other costs and benefits, in order to "offer a full accounting" of a rule. Cass R. Sunstein, *The Real World of Cost-Benefit Analysis: Thirty Six Questions (and Almost as Many Answers)*, 114 Colum. L. Rev. 167, 190 (2014).

C. HAP-Specific Benefits Are Large and Not Captured in the RIA's Monetized Estimate

The HAP-specific benefits of regulation are very large and (as EPA has recognized not captured in the RIA's monetized estimate. The total health benefits associated with section 112(d) standards for coal- and oil-fired power plants are dramatically greater, using accepted monetization metrics, than the monetized partial estimate of mercury benefits included in the final RIA ($4-6 million). EPA Final RIA at ES-1. This was true in 2011 when EPA finalized the MATS rule, and it is likely even more true today, as we learn more about the health benefits of reducing exposures to power plant air pollution. In 2011, EPA's final RIA assessed total monetized benefits of $37-90 billion due to the rule, and noted that there were many additional benefits of the MATS rule – including the health benefits of reduced exposures to toxic metals and acid gases – that could not be monetized. *Id.*

These figures are still robust; if anything they are underestimates, given new information that continues to be developed about the health effects of power plant pollution specifically. *See, e.g.*, Thurston, GD, *et al.*, "Ischemic Heart Disease Mortality and Long-Term Exposure to Source-Related Components of U.S. Fine Particle Air Pollution," advance publication, Dec. 2, 2015, *available at* http://dx.doi.org/10.1289/ehp.1509777 (associating coal-combustion PM2.5

particles with a risk of ischemic heart disease mortality 5 times higher than that associated with PM2.5 mass in general).

Additionally, we have more confidence today regarding the quantitative relationship between both long term and short term population exposures to various levels of PM and SO2 air pollution and the association with adverse public health impacts, than was the case in 2011. Dockery Declaration at ¶ 9. Our understanding of the relationship between reduced exposures to each of the constituents of power plant air pollution continues to grow. However, EPA's well-understood and well-confirmed approach to calculating economic values associated with specific health endpoints and trying that back to pollution reductions necessarily requires the development and application of a dose-response function to the levels of exposure associated with the pre- and post-emissions controls pollution levels. While there is ongoing scientific research and analysis directed at assessing the health effects associated with non-mercury metal toxic constituents of PM air pollution, Levy Declaration at ¶ 9 (citing Levy, JI, Diez D, Dou Y, Barr CD, Dominici F. A meta-analysis and multi-site time-series analysis of the differential toxicity of major fine particulate matter constituents. 175 Am. J. Epidemiol. 1095 (2012)), the state of that work is that it has not currently progressed to the point at which it is possible to develop concentration-response functions for the specific non-mercury toxic metal constituents of particulates. Nor is it yet possible to monetize the benefits achieved when exposures to those pollutants are reduced or eliminated. However, the fact that a health benefit cannot be precisely quantified does not make it insignificant, nor does it mean that unquantifable benefits of the regulation are irrelevant to the determination at hand.

What EPA can do (and has done in the final RIA for the rule) is to discuss qualitatively the real and significant benefits associated with reduced exposures to these air toxics. EPA RIA Chapter 5. That is the appropriate response where health benefits due to reduced exposures are real, but methods by which they can be precisely quantified and monetized do not yet exist.

We appreciate the opportunity to comment. Please do not hesitate to contact any of the representatives listed below if you have any questions.

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EXHIBIT 1

To Comments of Public Health and Environmental Groups
on Docket ID No. EPA-HQ-OAR-2009-0234
80 Fed. Reg. 75,025 (Dec. 1, 2015)

Philippe Grandjean, M.D., Ph.D.
"Report on the Health Benefits of Reducing Mercury Emissions from U.S. Power Plants"

Report on the Health Benefits of Reducing Mercury Emissions from U.S. Power Plants

Philippe Grandjean, M.D., Ph.D.
January 15, 2016

In response to the U.S. Environmental Protections Agency's (EPA) Proposed Supplemental Finding[1] requesting comment on the consideration of cost in making the "appropriate and necessary" determination for the listing of power plants under Section 112 of the Clean Air Act, this report provides updated scientific information concerning the societal costs of exposure to mercury pollution. While the EPA aimed at incorporating the best scientific research available at the time of the publication of its 2011 Regulatory Impact Analysis (RIA), additional and more convincing evidence of the societal costs of mercury emissions from power plants is now available. Based on a more current, comprehensive analysis, previous estimates of methylmercury toxicity and associated societal costs relied on by the EPA in its 2011 RIA are substantial underestimates. Additionally, it is important to recognize—as EPA does in its Proposed Supplemental Finding[2]—that the cost estimates attempted by EPA in 2011 are only a partial estimate of the overall societal cost of power plant mercury emissions. It is inherently difficult to monetize the long-term damage to humans associated with mercury released from power plants, but that has not prevented scientists who study the impacts of such pollution from concluding with confidence that the toxic damage represents a significant public health problem that warrants effective policy intervention.

I, Philippe Grandjean, am qualified to report regarding these developments in our understanding of the cost of mercury exposure because:

- I am an Adjunct Professor of Environmental Health at the Harvard T.H. Chan School of Public Health and a Professor and Chair of Environmental Medicine at the University of Southern Denmark.
- I have previously served as the Director of the Department of Occupational Medicine at the Danish National Institute of Occupational Health
- I have served for 30 years as Consultant in Toxicology for the Danish National Board of Health of the Danish Ministry of Health.
- I have served on expert committees under the auspices of the World Health Organization, the International Agency for Research on Cancer, the European

[1] Supplemental Finding that it is Appropriate and Necessary to Regulate Hazardous Air Pollutants from Coal- and Oil- Fired Electric Utility Steam Generating Units. 80 Fed. Reg. 75,025 (Dec. 1, 2015).
[2] *Id*. at 75,040 (stating that "the limited estimate for the single neurodevelopmental endpoint that could be monetized (IQ loss among certain recreational fishers) is a substantial underestimate of the total mercury impacts among affected populations.")

Commission, the European Food Safety Authority, the U.S. Environmental Protection Agency, and other organizations.

- In 1994, I was elected Fellow of the American Association for the Advancement of Science.
- My research focuses on the health effects of exposures to environmental chemicals, including mercury and other pollutants, such as lead, arsenic, and a variety of organic chemicals. My efforts have concentrated on the effects of environmental pollutants on fetal development, and my main focus during the last 25-30 years has been on methylmercury.[3] This research has been almost entirely financed by U.S. agencies, the European Commission, and the Danish Medical Research Council.
- I have published more than 500 scientific papers, of which more than half are in international scientific journals with peer review. I have also authored or edited 20 books, including text books in environmental health and risk assessment. In the new edition of the Handbook on the Toxicology of Metals,[4] I am the lead author of the chapter on epidemiological approaches to metal toxicology, and contributed to the chapter on principles for prevention of toxic effects from metals.
- Most recently, in 2015 I edited a special issue of a major journal with review articles on vulnerability to toxic chemicals during early development, based on a conference I organized in Boston in 2014 with support from the World Health Organization and U.S. federal agencies.

In regard to methylmercury specifically:

- I chaired the Working Group that evaluated methylmercury for the World Health Organization's International Agency for Research on Cancer in 1994.
- I served on the Expert Panel on Mercury of the Agency for Toxic Substances and Disease Registry in 1998.
- I chaired the scientific committee for an international conference on mercury in 1998 and served as editor of the proceedings.
- I served as an invited expert to the Food Advisory Committee on Methylmercury of the Food and Drug Administration in 2002.
- I served as a member of the Global Mercury Assessment Working Group of the U.N. Environment Programme in 2002.
- I served on the Working Group on mercury and methylmercury in food of the European Food Safety Authority in 2003-2004.
- I served as expert on mercury toxicity for the U.S. Department of Justice in a court case regarding pollution from coal-fired power plants in 2008.

[3] In the aquatic environment, mercury is methylated, mostly by microbiologically catalyzed reactions, to form methylmercury. Methylmercury is accumulated by fish and marine mammals and attains its highest concentrations in large predatory species at the top of the aquatic and marine food chains. By this means, methylmercury enters the human diet.

[4] HANDBOOK ON THE TOXICOLOGY OF METALS, Fourth Edition (2015).

- I was an invited speaker at the World Health Organization workshop on implementation of the Minamata Convention in 2015.
- I have been invited to prepare chapters on mercury for major handbooks on public health and toxicology.
- I am frequently invited to lecture on mercury at universities, governmental agencies, and international research conferences.

The current risk of excess methylmercury exposure is substantial within the U.S. population, and it is therefore a public health priority to eliminate emissions that increase this risk. Methylmercury exposure presents a wide range of cognitive and non-cognitive health risks to both children and adults, but, due to limitations or uncertainties in the data and scientific findings, EPA's 2011 RIA attempted to monetize only one type of adverse effect in a highly limited scenario: the loss of I.Q. in children exposed *in utero* to methylmercury from freshwater fish caught by a recreational angler in the same household. In attempting to monetize that subset of methylmercury effects, EPA relied upon some key assumptions concerning: the threshold above which methylmercury exposure causes harm (this is referred to as the "reference dose"); the effects of methylmercury exposure at incremental doses above the reference dose (this is referred to as the "dose-response" curve); and the number of people exposed to methylmercury from polluted waterways. More recent scientific studies provide a basis for updating these key assumptions and, therefore, the estimates of I.Q.-related costs imposed by methylmercury pollution in the U.S. Two recent studies (one of which I co-authored) estimate that the total I.Q.-related costs of methylmercury pollution in the U.S. are up to 1,000 times greater than what EPA, in a very narrow scenario, estimated in its 2011 RIA.

Current Data Show Significant Methylmercury Exposure in the U.S. at Levels Associated with Cognitive Deficits, and Such Deficits Can Be Permanent.

In 2000, the National Research Council (NRC) recommended that EPA set a target maximum dose of 5.8 µg/L in cord blood, which reflects prenatal exposure. This conclusion was derived from results obtained by the Faroe Islands study that my colleagues and I performed.[5] Since mercury is concentrated in fetal blood cells, maternal blood concentrations tend to be lower than cord blood concentrations. Hence, this translates into a maximum reference dose of 3.5 µg/L in the mothers' blood.[6] The EPA adopted NRC's 2000 reference dose recommendation soon thereafter, and it has not been revised since then.

[5] E. Budtz-Jørgensen, *et al*., Benchmark dose calculations of methylmercury-associated neurobehavioural deficits. *Toxicol. Lett.* 112-3: 193–99 (2000).
[6] K.R. Mahaffey, Mercury Exposure: Medical and Public Health Issues, 116 *Transactions of the Am. Clinical Climatological Ass'n* 127: 144–46, (2005), *available at* http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1473138/pdf/tacca116000127.pdf.

The most recent exposure data indicate that considerable numbers of people in the U.S. have blood mercury concentrations above the level that corresponds to the EPA reference dose.[7] American women of reproductive age who eat average amounts of fish and seafood have an average blood-mercury concentration of about 1.4 µg/L, with higher concentrations at higher incomes and in certain ethnic groups.[8] Increased methylmercury exposures are seen in subjects who frequently eat fish and seafood, in particular in those who eat species with high accumulation levels. However, even less frequent intakes, *e.g.*, among anglers consuming fish from polluted waterways, can result in high-level exposures.[9]

Previous data from the National Health and Nutrition Examination Survey show that about 16% of U.S. women of childbearing age have mercury concentrations in their blood at least as high as the EPA reference dose maximum of 3.5 µg/L in the mothers' blood.[10] This prevalence is noteworthy, given that few women consume the recommended two fish dinners per week. For another example, the Project Viva study in Boston, where fish consumption is higher than average for the U.S., showed a mean maternal hair mercury concentration of 0.53 µg/g[11] that corresponds to about 1.8 µg/L in the mothers' blood. Even at these levels, the maternal hair mercury was associated with a reduction in children's cognition at 6 months of age and again at three years of age. This also suggests that cognitive impairment occurs at mercury concentrations seen in the general U.S. population, and hence constitutes a matter of serious public health concern. Thus, even a small addition to current exposures may push exposures into ranges that are known to be associated with adverse effects.

Follow-up studies since the issuance of the 2011 RIA further demonstrate that the deficits experienced by children exposed to methylmercury *in utero* tend to be permanent. Thus, in our published follow-up of these subjects at ages 14 and 22, we found that the deficits

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[7] EPA, Trends in Blood Mercury Concentrations and Fish Consumption Among U.S. Women of Childbearing Age at 21-22 & tbl.5, EPA-823-R-13-002 (July 2013), *available at* http://water.epa.gov/scitech/swguidance/fishshellfish/fishadvisories/upload/Trends-in-Blood-Mercury-Concentrations-and-Fish-Consumption-Among-U-S-Women-of-Childbearing-Age-NHANES-1999-2010.pdf.

[8] *Id*.

[9] R.A. Lincoln, *et al*., Fish Consumption and Mercury Exposure among Louisiana Recreational Anglers, Envtl. Health Perspectives 119: 245 (2011), *available at* http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3040613/pdf/ehp-119-245.pdf; L. Knobeloch, *et al*., Fish consumption, advisory awareness, and hair mercury levels among women of childbearing age, *Envtl. Research* 97: 220 (2005); Methylmercury exposure in Wisconsin: A case study series, *Envtl. Research* 101: 113 (2006).

[10] Mahaffey, *supra* n.6, at 144–46.

[11] E. Oken, *et al*., Maternal fish intake during pregnancy, blood mercury levels, and child cognition at age 3 years in a US cohort, *Am. J. Epidemiology* 167: 1171, 1174 (2008), *available at* http://aje.oxfordjournals.org/content/167/10/1171.full.pdf+html.

remained.[12] Children exposed to methylmercury prenatally exhibit decreased motor function, attention span, verbal abilities, memory, and other mental functions.[13] These effects are dose dependent: the greater the mercury exposure, the greater the effect. We found that a doubling of the prenatal mercury exposure of a child, even at relatively low levels, resulted in a developmental delay of one to two months at the age of seven years, *i.e.*, at the age when the child is expected to enter school. Each delay corresponds to about 1.5 I.Q. points.

The Reference Dose, Dose-Response, and Affected Population Estimates Used in EPA's 2011 RIA Underestimate I.Q.-Related Costs as Compared with More Current Estimates.

In the 2011 RIA analysis, EPA presented a very narrowly-defined estimate of lost earnings from slightly lower I.Q. in children exposed *in utero* to mercury from freshwater fish caught by a recreational angler in the same household.[14] Cognitive deficits expressed in terms of I.Q. decreases will result in a lower chance of completing high school and higher education, and will lead to lower lifetime earnings, and by using annual discounting factors, this amount can be translated into an estimated value today. EPA's narrow monetized estimate of lost earnings from lower I.Q. in children exposed *in utero* to mercury from freshwater fish caught by a recreational angler in the same household—$4 to $6 million—should not be mistaken for an estimate of the aggregate annual societal cost of hazardous air pollution from U.S. power plants, even from mercury pollution alone. Furthermore, EPA's 2011 calculation relied on a dose-response estimate calculated in 2007[15] from data available at that time and that has since then been found to underestimate[16] the true toxicity from methylmercury exposure. In fact, the newest research suggests that adverse effects may begin at exposures that are below the current reference dose *and* that exposure-associated losses of cognitive function are likely to be steeper than was originally thought.

Further developments since the rulemaking only strengthen the basis for EPA's conclusion that power plant mercury emissions cause very substantial costs to human health. Scientific understanding of the harms of lower methylmercury exposures has increased, and the scientific and public health community is unable to identify a level below which methylmercury

[12] F. Debes, *et al*., Impact of prenatal methylmercury exposure on neurobehavioral function at age 14 years, *Neurotoxicology & Teratology* 28: 363, 540–44 (2006); F. Debes, *et al*., Cognitive deficits at age 22 years associated with prenatal exposure to methylmercury, *Cortex* (June 2015), 5–9.
[13] P. Grandjean, *et al*., Cognitive deficit in 7-year-old children with prenatal exposure to methylmercury, *Neurotoxicology & Teratology* 19: 417 (1997).
[14] EPA, Regulatory Impact Analysis at 4-9 to 4-13, EPA-HQ-OAR-2009-0234-20131 (Dec. 2011).
[15] D.A. Axelrad, *et al*., Dose-response relationship of prenatal mercury exposure and IQ: an integrative analysis of epidemiologic data, *Envtl. Health Perspectives* 115: 609-15 (2007).
[16] P. Grandjean, *et al*., Calculation of mercury's effects on neurodevelopment (letter), *Envtl. Health Perspectives* 120: A452 (2012), *available at* http://ehp.niehs.nih.gov/wp-content/uploads/2012/11/ehp.1206033.pdf.

is truly safe, in particular to the brain during early development. Of additional concern is that, on a body weight basis, small children may receive a substantially higher exposure than adults. Researchers, including myself, are studying the effects of methylmercury exposure from dietary intakes at lower and lower levels. The results from high-levels of contamination have long been clear, but a substantial base of scientific evidence and data now exists to show that methylmercury is also neurotoxic at low doses, in particular in regard to brain development.[17]

Based upon our current understanding, not only did EPA's reference dose and dose-response assumptions in the 2011 RIA underestimate the I.Q.-related costs of mercury pollution, but EPA's assessment of affected waterways and populations also underestimates those costs. In the 2011 RIA analysis, when EPA found that certain mercury data was unavailable for a waterway frequented by recreational freshwater anglers, EPA assumed that the mercury contributed by the waterway was zero, thereby reducing the already-low and narrowly-defined[18] exposed population estimate by 44%.[19] According to the EPA's most recent National Listing of Fish Advisories technical fact sheet, dated December 2013, about three of four advisories warn anglers against consuming freshwater fish or coastal seafood because of mercury contamination that affects about 16.4 million lake acres and 1.1 million river miles.[20] Also, the December 2013 National Listing of Fish Advisories database shows that the number of fish advisories issued and the percentage of total lake acres and river miles under advisory continues to rise with additional monitoring activities.[21] Mercury contamination is the most frequent reason for freshwater fish advisories by U.S. states,[22] and power plants are "by far the largest anthropogenic [i.e., human-caused] source of [mercury] in the U.S."[23] Based on more

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[17] United Nations Envtl. Programme, Global Mercury Assessment at 9, 190 (Dec. 2002), *available at* http://www.unep.org/gc/gc22/Document/UNEP-GC22-INF3.pdf, at 38-42, 44-45, 48; European Food Safety Authority, Opinion of the Scientific Panel on Global Mercury Assessment, EFSA-Q-2003-030 (Feb. 2004), at 82-108, *available at* http://www.efsa.europa.eu/sites/default/files/scientific_output/files/main_documents/2985.pdf; M.R. Karagas, *et al*., Evidence on the Human Health Effects of Low-Level Methylmercury Exposure, *Envtl. Health Perspectives* 120: 799, 801–03 (2012), *available at* http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3385440/pdf/ehp.1104494.pdf; P. Grandjean, Mercury (Chapter 29), in: P.J. Landrigan, R.A. Etzel, eds. *Children's Environmental Health*, New York: Oxford University Press (2014) at 273–80.

[18] 80 Fed. Reg. at 75,040 (estimate did not account for, among other things, consumers of commercial (store-bought) fish or self-caught fish from oceans, estuaries or large lakes such as the Great Lakes.)

[19] RIA at 4-49.

[20] EPA, National Listing of Fish Advisories: Technical Fact Sheet 2011, EPA-820-F-13-058 (Dec. 2013), *available at* http://water.epa.gov/scitech/swguidance/fishshellfish/fishadvisories/technicalfs2011.cfm#table1.

[21] *Id*. at 2.

[22] *Id*. at 4-6.

[23] 77 Fed. Reg. 9304, 9310 (Feb. 12, 2012) ("In 2005, U.S. EGUs emitted 50 percent of total domestic anthropogenic [mercury] emissions.")

recent information about mercury contamination in waterways and ocean shores, the EPA's 44% reduction in the affected freshwater waterbodies and their exclusion of other exposure pathways clearly caused them to underestimate lost earnings substantially.

Current Estimates of I.Q.-Related Costs of Methylmercury Exposure are Much Greater than EPA's 2011 Estimate.

In 2007, we recalculated the reference dose using the methods and data endorsed by the National Research Council,[24] while applying advanced statistical modeling. We found that the EPA reference dose is twice as high as it should be.[25] Given the recent study results showing adverse effects associated with habitual exposures associated with common fish consumption,[26] an updated exposure limit would likely be even lower. Thus, in our recent assessment of costs for mercury toxicity to children in the European Union, we used a threshold of 50% of the EPA's reference dose.[27] Previous estimates of methylmercury toxicity, and associated adverse human health effects, should therefore be regarded as underestimates.

Based upon updated information concerning the reference dose, the dose-response curve, and the number of people exposed to methylmercury, more current estimates of the I.Q.-related societal cost of mercury exposure are more accurate than the estimate of lost earnings given by EPA in its 2011 RIA. Although the sources of mercury present in fish used for human consumption are only partially known, available evidence shows that coal-fired power plants constitute the major domestic source[28] and that most of the mercury released is deposited locally and regionally, thus affecting fish in U.S. waterways and coastal shores[29].

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[24] Nat'l Research Council, Toxicological effects of methylmercury (2000), *available at* https://www.nap.edu/login.php?record_id=9899&page=https://www.nap.edu/download.php?record_id=9899.

[25] P. Grandjean, *et al*., Total Imprecision of Exposure Biomarkers: Implications for Calculating Exposure Limits, *Am. J. Indus. Med*. 50: 712 (2007).

[26] Karagas, *supra* n.17, at 801–04; Oken, *supra* n.11, at 1175; S.A. Lederman, *et al*., Relation between Cord Blood Mercury Levels and Early Child Development in a World Trade Center Cohort, *Envtl. Health Perspectives* 116: 1085, 1090 (2008), *available at* http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2516590/pdf/ehp0116-001085.pdf.

[27] M. Bellanger, *et al*., Economic benefits of methylmercury exposure control in Europe: Monetary value of neurotoxicity prevention, *Envtl. Health* 12: 3 (2013).

[28] *See supra* n.23.

[29] Y. Zhang, *et al*., Observed decrease in atmospheric mercury explained by global decline in anthropogenic emissions, *PNAS Early Edition* (2016), *available at* http://www.pnas.org/cgi/doi/10.1073/pnas.1516312113.

Thus, using updated information, Trasande and Liu calculated lost earnings due to I.Q. decreases from overall mercury exposure of $5.1 billion annually.[30] The Trasande and Liu estimate of $5.1 billion in I.Q.-related costs is a more appropriate estimate than the partial societal cost of mercury exposure of $4 to $6 million annually used by the EPA in the 2011 RIA. Trasande and colleagues also stated, "[s]imilarly, great economic savings can be achieved by preventing methylmercury contamination of fish, which is the major source of human exposure to this chemical."[31] Using similarly updated assumptions regarding the reference dose, the dose-response curve, and the affected waterways and seacoasts, my calculations are very similar to Trasande's.[32] Thus, my December 2012 published estimate of the societal cost of I.Q. decreases, due to prenatal exposures of methylmercury in children, is approximately $4.7 billion.[33] The current loss of I.Q. points (estimated to be 264,000 I.Q. points per year in our 2012 calculation) associated with methylmercury exposure represents a very substantial cost to society, and does not account for the other significant cognitive and non-cognitive effects of methylmercury exposure.

I.Q.-Related Cost Estimates Do Not Account for the Other Very Significant Societal Costs—Both Quantified and Unquantified—of Hazardous Air Pollution from Power Plants.

Significantly, even these updated and more accurate I.Q.-related estimates capture only one aspect of the adverse human health effects of hazardous air pollution emitted by U.S. power plants. Other scientifically-documented, mercury-related harms include, but are not limited to: other, less tangible consequences of lowered I.Q.; other cognitive and behavioral deficits; cardiovascular risk; other serious disease; and, the negative health implications of reduced fish intake. Evidence is building that mercury exposure compromises cardiovascular health, and that these cardiovascular effects impose very significant societal costs.[34] Mercury exposure also produces a range of other toxic effects reported in human populations.[35] For example, methylmercury may spur the development of degenerative disease of the nervous system, such as Parkinson's disease.[36] Other toxic elements are also emitted from coal-fired

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[30] L. Trasande, *et al*., Reducing The Staggering Costs Of Environmental Disease In Children, Estimated At $76.6 billion In 2008, *Health Affairs* 30: 863, 865 Exh. 1 (2011), *available at* http://content.healthaffairs.org/content/30/5/863.full.pdf+html.

[31] *Id*. at 867.

[32] Grandjean, *supra n.16*.

[33] *Id*.

[34] A. Giang & N.E. Selin, Benefits of mercury controls for the United States, *PNAS (Early Edition*) at 3 (2015), *available at* http://www.pnas.org/cgi/doi/10.1073/pnas.1514395113 (economy-wide benefits from the Mercury and Air Toxics Standards estimated at $43 billion by 2050, and lifetime benefits at $147 billion, including very substantial benefits from avoided cardiovascular effects).

[35] Karagas, *supra* n.17, at 803–04.

[36] M.S. Petersen, *et al*., Impact of dietary exposure to food contaminants on the risk of Parkinson's disease, *Neurotoxicology* 29: 584–90 (2008).

power plants in large quantities, including arsenic, lead, and cadmium, all of which are neurotoxic and likely contribute to the pandemic of developmental neurotoxicity.[37] The toxicity of each of these substances is supported by a large scientific literature. Like mercury, arsenic and lead cross the placental barrier and thereby expose the developing fetus, with impacts on neonatal and early childhood outcomes. Substantial evidence also exists that methylmercury chloride is carcinogenic to experimental animals.[38] All of these harms would also have to be considered and monetized to reach a more comprehensive estimate of the total societal cost of hazardous air pollution from U.S. power plants.

In conclusion, the analysis in this report provides additional and updated scientific support for the EPA's conclusion that exposure to mercury is harmful to human beings in numerous ways. New data and advances in scientific understanding demonstrate that EPA's estimate, in its 2011 Regulatory Impact Analysis, of the I.Q.-related costs from U.S. power plant mercury pollution was an underestimate, and only a partial indication of the full societal cost of hazardous air pollution emitted by U.S. power plants. Therefore, reducing the emissions of mercury and other hazardous air pollutants from U.S. power plants remains a well-documented and critical priority to protect public health.

[37] P. Grandjean, *et al*., Developmental neurotoxicity of industrial chemicals, *Lancet* 368: 2167 (2006).
[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.

EXHIBIT 2

To Comments of Public Health and Environmental Groups
on Docket ID No. EPA-HQ-OAR-2009-0234
80 Fed. Reg. 75,025 (Dec. 1, 2015)

Margaret R. Karagas, *et al*.
"Evidence on the Human Health Effects of Low-Level Methylmercury Exposure"
***Environmental Health Perspectives* (June 2012)**

Evidence on the Human Health Effects of Low-Level Methylmercury Exposure

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Background: Methylmercury (MeHg) is a known neurotoxicant. Emerging evidence indicates it may have adverse effects on the neurologic and other body systems at common low levels of exposure. Impacts of MeHg exposure could vary by individual susceptibility or be confounded by beneficial nutrients in fish containing MeHg. Despite its global relevance, synthesis of the available literature on low-level MeHg exposure has been limited.

Objectives: We undertook a synthesis of the current knowledge on the human health effects of low-level MeHg exposure to provide a basis for future research efforts, risk assessment, and exposure remediation policies worldwide.

Data sources and extraction: We reviewed the published literature for original human epidemiologic research articles that reported a direct biomarker of mercury exposure. To focus on high-quality studies and those specifically on low mercury exposure, we excluded case series, as well as studies of populations with unusually high fish consumption (e.g., the Seychelles), marine mammal consumption (e.g., the Faroe Islands, circumpolar, and other indigenous populations), or consumption of highly contaminated fish (e.g., gold-mining regions in the Amazon).

Data synthesis: Recent evidence raises the possibility of effects of low-level MeHg exposure on fetal growth among susceptible subgroups and on infant growth in the first 2 years of life. Low-level effects of MeHg on neurologic outcomes may differ by age, sex, and timing of exposure. No clear pattern has been observed for cardiovascular disease (CVD) risk across populations or for specific CVD end points. For the few studies evaluating immunologic effects associated with MeHg, results have been inconsistent.

Conclusions: Studies targeted at identifying potential mechanisms of low-level MeHg effects and characterizing individual susceptibility, sexual dimorphism, and nonlinearity in dose response would help guide future prevention, policy, and regulatory efforts surrounding MeHg exposure.

Key words: birth outcomes, cardiovascular disease, epidemiology, health outcomes, low-level exposure, metals, methylmercury, neurologic outcomes. *Environ Health Perspect* 120:799–806 (2012). http://dx.doi.org/10.1289/ehp.1104494 [Online 24 January 2012]

Methylmercury (MeHg) from natural or anthropogenic sources biomagnifies through the food chain and gives rise to human exposure primarily through consumption of higher trophic level fish and marine mammals [National Research Council (NRC) 2000]. MeHg crosses the placenta and readily passes through the blood–brain barrier, with even higher MeHg levels in fetal than in maternal circulation (Stern and Smith 2003). Vulnerability of the developing fetus to MeHg exposure was exemplified in Minamata, Japan, when pregnant women consumed seafood highly contaminated with MeHg. This resulted in extreme fetal abnormalities and neurotoxicity (i.e., microcephaly, blindness, severe mental and physical developmental retardation) even among infants born to mothers with minimal symptoms (Harada 1995).

More subtle neurodevelopmental effects have been observed in populations with moderate MeHg exposures from regular consumption of fish and/or marine mammals, including associations of MeHg biomarkers at birth with decrements in memory, attention, language, and visual-motor skills in childhood (NRC 2000). Most recently, a growing body of literature has explored the impact of even lower levels of MeHg on a variety of health outcomes in both adults and children. Findings include potential adverse effects on fetal growth, neorulogic function, the cardiovascular system, and immune function.

Given that fish is a key source of dietary protein in much of the world, MeHg contamination of fish has the potential to impact the health of geographically diverse populations. Furthermore, fish is an important source of beneficial nutrients such as polyunsaturated fatty acids (PUFA), iodine, selenium, and vitamin D. Development of dietary recommendations that balance nutritional benefits of fish with the contaminant risk has been a challenge for government regulatory agencies and public health professionals (Teisl et al. 2011). In this context, characterization of MeHg health risks is critical for the development of optimal fish consumption guidelines (Cohen et al. 2005a; Shimshack and Ward 2010). However, there has been limited, if any, synthesis of the available literature on the health effects of low-level MeHg exposure, despite its global relevance.

To synthesize the current state of knowledge on the human health effects of low-level MeHg exposure, we focused on the epidemiologic literature of mercury concentrations measured in biologic tissue. We examined the following questions: *a*) What are the key health effects of lower, prevalent levels of MeHg exposure in the general population, and what are the strengths and limitations of recent evidence regarding those health effects? *b*) What are potential confounders or modifiers of human health risks (synergistic or antagonistic) at low-level exposure? *c*) What important gaps exist in the current literature? The ultimate goal of this review was to provide a basis for optimizing future research efforts, as well as risk–benefit assessment and exposure remediation policies, worldwide.

Biomarkers of MeHg Exposure

Biomarkers of MeHg exposure, such as total mercury levels in hair or blood, are regarded as more accurate measures of human exposure than dietary assessment (i.e., of fish consumption) because MeHg concentrations vary both between and within fish species and because recall of specific species may be imprecise (Groth 2010). Although it is correlated with maternal hair, cord blood mercury may

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The opinions presented here are those of the authors and do not necessarily reflect the policies of the U.S. EPA.

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better reflect fetal exposure than maternal hair (Grandjean et al. 2002). Mercury is excreted in breast milk, but it is not typically used as a matrix for assessing exposure, primarily because of low concentrations and variability in the proportion present as MeHg (Björnberg et al. 2005; García-Esquinas et al. 2011; Miklavcic et al. 2011). Meconium and other tissues, such as umbilical cord, placenta, and nail tissue, although potentially useful, have not been used widely in epidemiologic studies (Gundacker et al. 2010; Rees et al. 2007). Urinary mercury reflects inorganic mercury levels and thus is not used as an indicator of MeHg exposure; however, in hair, nails, and blood, MeHg is the primary contributor to total mercury levels (Grandjean et al. 2002).

Even the best exposure biomarkers are imprecise measures of MeHg in target organs such as the fetal brain. Furthermore, the average coefficient of variation is about 25% for cord blood mercury analysis and about twice that for maternal hair mercury (Grandjean and Budtz-Jørgensen 2010). Typically, imprecision in an exposure measure will attenuate its calculated effect (Rothman and Greenland 1998); this highlights the potential for measurement errors in MeHg exposure assessment to affect comparability of findings across studies.

Low-Level Exposure

Because most of the published epidemiologic literature reports measures of total mercury rather than MeHg, we focused principally on studies with mercury exposure measures in blood or hair as matrices most reflective of MeHg. We excluded case reports or case series and reports that were not original research. We further limited our review to studies of low-dose mercury exposures, that is, we excluded analyses of the poisoning episodes in Japan and Iraq, as well as studies of populations with mean measured mercury levels above any of the following: 4 μg/g in hair; 20 μg/L in cord blood, or approximately 12 μg/L in adult blood. We based our definition of low dose on a qualitative assessment of the literature and appreciation that findings from the three major cohort studies with moderate mercury exposures (the Faroe Islands, the Seychelles, and New Zealand) are already extensively reviewed (e.g., Axelrad et al. 2007; Cohen et al. 2005b; Rice 2004). Among the major prospective cohort studies of MeHg and child development in high exposure risk populations, the Faroes had the lowest mercury levels with approximately 4 μg/g in maternal hair and 20 μg/L in cord blood, on average (Steuerwald et al. 2000). We assumed a factor of 1.7 (Stern and Smith 2003) in estimating the corresponding adult blood mercury level of 12 μg/L. By design, our definition of low dose excludes studies focused on moderately MeHg-exposed groups, such as those with particularly high fish consumption (e.g., the Seychelles Islands), marine mammal consumption (e.g., the Faroe Islands and most circumpolar and other indigenous populations), or unusually contaminated fish consumption (e.g., gold-mining regions of the Amazon).

Study Selection

Our review encompasses human epidemiologic studies that measured mercury using a biomarker. For example, in prenatal or childhood MeHg exposure assessment, studies measure primarily total mercury in whole blood (maternal, umbilical cord, or child) or hair (maternal, infant, or child). For adult exposure, MeHg levels are typically estimated using total mercury levels in whole blood, hair, or toenails. We included cohort studies irrespective of sample size and geographic location. To identify relevant studies, we performed a literature search for studies that analyzed the relation between mercury exposure and health outcomes using PubMed (National Library of Medicine 2012) and ScienceDirect (Elsevier 2012).

Birth Outcomes and Infant Growth

In searching the published literature on birth outcomes and infant growth, we used the following key words: "mercury," "infant," "fetus," "birth outcome," "biomarker," "anthropometric," "maternal," "mother," "child," birth," "pregnancy," "blood," "cord blood," "hair," "birth weight," "birth length," "infant weight," and "postnatal growth." The studies reviewed are summarized in Table 1 and Supplemental Material, Table S1 (http://dx.doi.org/10.1289/ehp.1104494).

Overall, studies on fetal mercury exposure and birth outcomes show mixed results [see Supplemental Material, Table S1 (http://dx.doi.org/10.1289/ehp.1104494)]. A small study from Poland (n = 41) published in 1986 found that infant, but not maternal, hair mercury was inversely associated with birth weight without consideration of fish or seafood consumption (Sikorski et al. 1986). In a more recent, larger cohort (n = 554) in Spain, Ramon et al. (2009) found that cord blood mercury was inversely related to birth weight. Newborns in the highest quartile for cord blood mercury weighed 143.7 g less [95% confidence interval (CI): –2251.8, –235.6] than those in the first quartile, after adjusting for fish consumption and other variables. These authors also observed a similar pattern for small-for-gestational-age newborns, although the results were not statistically significant. In a study examining cord blood mercury and maternal blood mercury both early (12–20 weeks) and late (28–42 weeks) in pregnancy in a South Korean cohort (n = 417), Lee et al. (2010) observed that birth weight was inversely related to all measures of mercury exposure, with the strongest magnitude of effect observed for cord blood. Of particular interest is that the associations were more pronounced among those with the glutathione *S*-transferase (GST) M1 (*GSTM1*) null genotype or both *GSTM1* and *GSTT1* null genotypes. MeHg excretion rates vary widely among individuals and involve glutathione conjugation by selenium-dependent GSTs (Custodio et al. 1994). Birth weight was unrelated to maternal hair mercury in a French cohort (n = 645) (Drouillet-Pinard et al. 2010); maternal or cord blood mercury in a New York City cohort (n = 329) (Lederman et al. 2008); maternal blood, hair, or cord blood mercury in a small study (n = 53) from Vienna, Austria (Gundacker et al. 2010); cord blood mercury in a cohort study from Nunvik, Canada (n = 439) (Lucas et al. 2004); and cord tissue mercury in a large study in the United Kingdom (n = 1,040) (Daniels et al. 2007). The French, New York City, Austrian, and U.K. studies considered fish or seafood consumption, and the Canadian study accounted for PUFA in their analysis.

We found little to no evidence of effects of low-level mercury exposure on other studied anthropometric measures at birth. Of the five studies that evaluated birth length, none found any association (Drouillet-Pinard et al. 2010; Gundacker et al. 2010; Lederman et al. 2008; Ramon et al. 2009; Sikorski et al. 1986). Likewise, four studies recorded measurements of infant head circumference at birth, but none found clear associations with mercury exposure (Drouillet-Pinard et al. 2010; Gundacker et al. 2010; Lederman et al. 2008; Sikorski et al. 1986).

Gestational age was evaluated in five studies that met our criteria. No association was observed with gestational age in the Canadian study with cord blood mercury (Lucas et al. 2004), the U.K. cohort with cord tissue mercury (Daniels et al. 2007), the New York City cohort with maternal or cord blood mercury (Lederman et al. 2008), or the French cohort with maternal hair (Drouillet-Pinard et al. 2010). In a study in Michigan (USA), however, Xue et al. (2007) found that women who delivered very preterm (< 35 weeks) were more likely to have had higher hair mercury levels (0.55–2.5 μg/g) than women who delivered at term (odds ratio = 3.0; 95% CI: 1.3, 6.7).

Of further interest, cord blood mercury (Grandjean et al. 2003; Kim et al. 2011) and late-pregnancy maternal blood mercury (Kim et al. 2011) have been associated with impaired infant growth within the first 2 years of life. One of these studies (Kim et al. 2011), based on a South Korean birth cohort, met our inclusion criteria [Table 1; see also Supplemental Material, Table S1 (http://dx.doi.org/10.1289/ehp.1104494)].

Neurocognitive and Behavioral Outcomes

For neurodevelopmental outcomes, we searched databases using combinations of the following terms: "mercury," "MeHg," "blood," "cord blood, "hair," "low-dose," "cognition," "cognitive function," "intelligence," "IQ" (intelligence quotient), "memory," "executive function," "sensory function," "visual evoked potentials," "auditory evoked potentials," "human behavior," "behavior," "neurobehavior," "attention," "impulsivity," "impulse control," "hyperactivity," "motor skills," and "fine motor performance." The studies reviewed are summarized in Tables 1 and 2 and Supplemental Material, Table S2 (http://dx.doi.org/10.1289/ehp.1104494).

Prospective cohort studies have demonstrated associations of prenatal mercury exposure with neonatal motor function (Suzuki et al. 2010) and behavior (Gao et al. 2007). In descriptive analyses without adjustment for potential confounders, maternal pregnancy hair mercury ≥ 1 μg/g was associated with a smaller cerebellar volume among 137 full-term Croatian newborns (Cace et al. 2011).

Among infants 6–24 months of age, prospective studies of low-level prenatal mercury exposure have had mixed results. Mercury (adjusted for pregnancy seafood intake) was associated with decrements in infant cognition including poorer visual recognition memory at 6 months of age in U.S. infants (Oken et al. 2005) and poorer performance on both Psychomotor Development Index (PDI) and Mental Development Index (MDI) components of the Bayley Scales of Infant Development at 12 months, as well as modest but nonsignificant declines at 24 months, among Polish infants in models unadjusted for fish consumption (Jedrychowski et al. 2006, 2007). In contrast, among more highly exposed urban New York City children (e.g., geometric mean cord blood mercury = 4.4 μg/L vs. 0.9 μg/L in Polish infants), no significant prenatal mercury-associated decrements in 12- and 24-month Bayley PDI and MDI scores were observed despite adjustment for multiple potential confounders including fish consumption (Lederman et al. 2008). Similarly, in a prospective U.K. study, cord tissue mercury was not associated with scores on the MacArthur Communicative

Table 1. Summary of studies of low-level mercury exposure.

Outcome	No. of studies	Sample size (range)	Age (range)	Exposure measures	References
Birth outcomes and infant growth					
Birth weight	8	41–645	—	Cord blood, cord tissue, maternal hair	Daniels et al. 2007; Drouillet-Pinard et al. 2010; Gundacker et al. 2010; Lederman et al. 2008; Lee et al. 2010; Lucas et al. 2004; Ramon et al. 2009; Sikorski et al.1986
Birth length	5	41–645	—	Cord blood, maternal hair	Drouillet-Pinard et al. 2010; Gundacker et al. 2010; Lederman et al. 2008; Ramon et al. 2009; Sikorski et al. 1986
Head circumference	4	41–645	—	Cord blood, maternal hair	Drouillet-Pinard et al. 2010; Gundacker et al. 2010; Lederman et al. 2008; Sikorski et al. 1986
Gestational age	5	329–1,024	—	Cord blood, cord tissue, maternal hair	Daniels et al. 2007; Drouillet-Pinard et al. 2010; Lederman et al. 2008; Lucas et al. 2004; Xue et al. 2007
Infant growth	1	921	24 months	Cord blood	Kim et al. 2011
Neurologic outcomes					
Birth–2 years	10	53–1,054	Birth–26 months	Cord blood, cord tissue, infant hair, maternal hair, maternal blood	Barbone et al. 2004; Cace et al. 2011; Cao et al. 2010; Daniels et al. 2004; Gao et al. 2007; Jedrychowski et al. 2006, 2007; Lederman et al. 2008; Oken et al. 2005; Suzuki et al. 2010
3–6 years	11	72–1,778	36 months–6 years	Cord blood, child hair, child blood, maternal hair, maternal blood	Cao et al. 2010; Després et al. 2005; Freire et al. 2010; Ha et al. 2009; Jedrychowski et al. 2007; Lederman et al. 2008; Oken et al. 2008; Plusquellec et al. 2010; Saint-Amour et al. 2006; Stewart et al. 2003; Surkan et al. 2009
7–14 years	6	100–1,778	7–14 years	Cord blood, child hair, child blood	Boucher et al. 2010; Cao et al. 2010; Cheuk and Wong 2006; Ha et al. 2009; Surkan et al. 2009; Torrente et al. 2005
Adults	4	106–474	17 to ≥ 81 years	Adult hair, adult blood	Johansson et al. 2002; Philibert et al. 2008; Weil et al. 2005; Yokoo et al. 2003
Cardiovascular outcomes					
	8	Prospective cohort: 1,014–1,871 Case–control: 431–3,427 cases; 464–3,427 controls	16–75 years	Hair, blood, toenail, urine mercury	Guallar et al. 2002; Mozaffarian et al. 2011; Rissanen et al. 2000; Salonen et al. 1995, 2000; Virtanen et al. 2005; Wennberg et al. 2011; Yoshizawa et al. 2002
Blood pressure	1	1,240	16–49 years	Blood mercury	Valera et al. 2009; Vupputuri et al. 2005
Immunologic outcomes[a]					
	1	Prospective cohort: 582	29–39 months	Hair (child, maternal)	Miyake et al. 2011
	3	Cross-sectional: 61–112	Newborns	Blood (cord, maternal delivery)	Belles-Isles et al. 2002; Bilrha et al. 2003; Nyland et al. 2011a
	1	Cross-sectional: 1,990	≥ 20 years	Blood	Park and Kim 2011

See also Supplemental Material, Table S1–S4 (http://dx.doi.org/10.1289/ehp.1104494).
[a]Studies published since the NRC report (NRC 2000).

Table 2. Summary of findings on MeHg and neurocognitive and behavioral outcomes.

Age at assessment			
Birth–2 years	3–6 years	7–14 years	Adults
Inconsistent effects: no effect; increased risk associated with prenatal or postnatal mercury	Adverse effects if adjusted for fish intake: multiple associations with prenatal mercury (psychomotor function, memory, verbal skills cognition, etc.); inconsistent effects with concurrent mercury	Inconsistent effects: protective; no effect; increased risk (e.g., electrophysiologic testing) with prenatal or postnatal mercury	Inconsistent effects: no effect or adverse neuropsychological test performance with current mercury

See also Supplemental Material, Table S2 (http://dx.doi.org/10.1289/ehp.1104494).

Development Inventory at 15 months of age or the Denver Developmental Screening Test at 18 months of age despite adjustment for multiple potential confounders including fish consumption (Daniels et al. 2004). However, increased measurement error of mercury in cord tissue (compared with hair or blood) may have contributed to null findings (Grandjean and Budtz-Jørgensen 2007; Grandjean and Herz 2011).

In preschool-age children, prenatal mercury exposure has been consistently associated with adverse subsequent neurodevelopment in analyses accounting for maternal pregnancy fish consumption (Freire et al. 2010; Lederman et al. 2008; Oken et al. 2008). For example, among New York City infants in whom prenatal mercury was not associated with significant Bayley decrements at 12 and 24 months of age, significant PDI (but not MDI) declines were seen at 36 months, and a 3.6-point decline in IQ (per log increase in cord blood mercury) was seen at 4 years (Lederman et al. 2008). Prenatal mercury exposures have been associated with other cognitive and psychomotor measures in this age group, such as lower scores on tests of vocabulary (Peabody Picture Vocabulary Test) and visual-motor ability (Wide Range Assessment of Visual Motor Abilities) at 3 years of age (Oken et al. 2008) and poorer general cognition, memory, and verbal skills (McCarthy Scales of Children's Abilities) at 4 years of age (Freire et al. 2010).

However, without adjustment for fish intake, prenatal mercury exposures have been associated with inconsistent (Stewart et al. 2003) or even beneficial (Jedrychowski et al. 2007) findings in this age group (Stewart et al. 2003). For example, among Polish infants in whom prenatal mercury was associated with decrements in Bayley PDI and MDI at 12 months of age (and nonsignificant decrements at 24 months), mercury was associated with nonsignificant increases in Bayley scores at 36 months in analyses unadjusted for fish intake (Jedrychowski et al. 2006, 2007). Among 3-year-old U.S. children born to mothers consuming MeHg-contaminated Great Lakes fish, prenatal mercury was associated with poorer general cognition on the McCarthy Scales only among children with high prenatal exposure to polychlorinated biphenyls (PCBs). Stewart et al. (2003) observed no mercury-related effects on the McCarthy Scales in follow up at 4.5 years of age, but their analyses were not adjusted for fish consumption.

Studies of postnatal mercury exposures in young children have produced mixed results. In a small sample (n = 53) of 26-month-old Italian children, higher 3-month postpartum infant or maternal hair mercury levels (e.g., < 1 μg/g vs. ≥ 1 μg/g) were marginally associated with increased risk of expected or delayed (vs. advanced) fine motor skill on the Denver Developmental Screening Test in analyses unadjusted for fish consumption (Barbone et al. 2004). Among 24-month-old U.S. participants in a randomized trial of chelation for lead poisoning, current mercury concentration was associated with better (nonsignificant) Bayley MDI scores (Cao et al. 2010). In follow-up of these children at 5 and 7 years of age, increased blood mercury at baseline (age 2 years) was associated with no significant differences in IQ or behavior; however, point estimates of effect were all positive (i.e., improved with increasing mercury) (Cao et al. 2010). No information about fish consumption was available in that study.

Even with adjustment for fish consumption or related nutritional measures, associations of postnatal mercury exposure with neurodevelopment have been inconsistent. A prospective study of Inuit children in Nunavik, Canada (the high end of the low-exposure range) is of interest given its simultaneous assessment of prenatal and postnatal (concurrent) mercury levels and its inclusion of measures of other neurotoxicants (PCBs, organochlorine pesticides, and lead), as well as potential nutritional confounders (selenium and omega-3 PUFA) (Després et al. 2005; Plusquellec et al. 2010; Saint-Amour et al. 2006). In a study using the Infant Behavioral Rating Scale from the Bayley Scales of Infant Development-II and scoring of video-recorded behaviors, neither prenatal nor childhood mercury were related to behavior at 4–6 years of age (Plusquellec et al. 2010). However, in tests of gross and fine motor skill, current, but not prenatal, mercury was associated with increased action tremor amplitude at 4–6 years of age (Després et al. 2005). Finally, at ages 5–6 years, prenatal mercury was associated with longer latencies in visual evoked potentials (VEPs), whereas concurrent mercury was associated with shorter VEP latencies (Saint-Amour et al. 2006), an indicator of visual information–processing efficiency. The most recent published follow-up of this population assessed performance on auditory event-related potentials (ERPs) at ages 10–13 years. Cord blood mercury was associated with both adverse and potentially beneficial effects on early auditory information processing, with increased reaction time and increased latency but fewer false alarms (i.e., false-positive errors) and greater amplitude of response on the auditory ERP task (Boucher et al. 2010). Of note, concurrent blood mercury (median, 2.8 μg/L) was not associated with auditory ERP performance.

To date, most published studies among older children of school age are cross-sectional, with mercury biomarkers indicating postnatal exposure levels. With few exceptions (Boucher et al. 2010), studies in this age group do not demonstrate adverse associations of concurrent mercury exposure with a range of cognitive and neurobehavioral measures, and most lack information about potential confounding by fish consumption. For example, among 355 participants 6–10 years of age in a randomized trial of mercury amalgam versus composite restorations for dental carries, Surkan et al. (2009) observed no significant linear relationship of baseline mercury obtained before amalgam exposure across 18 psychometric tests, including measures of IQ, achievement, memory, executive function, and visual-motor and fine motor ability. Null results remained after adjustment for multiple potential confounders, including fish consumption. However, the authors identified nonlinearities in dose response with suggestive evidence of improved math reasoning and visual-motor skills at hair mercury < 0.5 μg/g but decrements on the same tests at higher levels (0.5 μg/g ≤ hair mercury ≤ 1.0 μg/g) and insufficient data at hair mercury > 1.0 μg/g. In a school survey of 1,778 South Korean 6- to 10-year-old children, parental report of attention deficit hyperactivity disorder (ADHD) symptoms was unrelated to concurrent mercury (Ha et al. 2009); information about fish intake was not included in the study. Conversely, in a case–control study of ADHD among 111 7- to 8-year-old children in Hong Kong, China, higher blood mercury was associated with increased odds of ADHD diagnosis (Cheuk and Wong 2006). However, that study had a number of limitations, including lack of comparability of cases and controls on ADHD risk factors and lack of information on fish intake. Finally, in a cross-sectional assessment of 100 Spanish children 12–14 years of age, concurrent hair mercury level was correlated with better visuospatial skill after adjustment for age and socioeconomic status (Torrente et al. 2005). These authors postulated that findings were consequent to confounding by fish consumption, which was not assessed.

Only a few cross-sectional studies of neurologic effects of low-level mercury exposure have been carried out in adults. Although Yokoo et al. (2003) did not include fish intake in assessments, higher hair mercury levels were associated with decrements in fine motor speed and dexterity, as well as memory and response inhibition, among 129 adults (mean age, 35 years; range, 17–81 years) from Brazilian fishing communities. Conversely among a random subset (n = 474) of older adults (ages 50–70 years) in the Baltimore Memory Study (Baltimore, MD), blood mercury was associated with improved manual dexterity (finger tapping) but poorer visual memory in analyses considering fish intake (Weil et al. 2005). In a study of 106 elderly Swedish adults (mean age, 87 years) by Johansson et al. (2002), blood mercury was not associated with general

cognitive status, including memory, assessed on the Mini Mental State Examination; however, there was no assessment for confounding. Finally, among lake-fish eaters in Quebec, Canada (mean age, 47–50 years), higher hair mercury was associated with increased self-reported neuropsychiatric symptoms of depression, anxiety, and obsessive–compulsive behavior (Philibert et al. 2008). However, this association was seen only in women, and symptom reporting was not related to blood mercury levels or serum PUFA levels.

Cardiovascular Outcomes

For literature searches for MeHg and cardiovascular outcomes, we used the key words "mercury" or "methylmercury," "cardiovascular" or "coronary," or "hypertension." References cited in articles were also identified. The studies reviewed are summarized in Tables 1 and 3 and in Supplemental Material, Table S3 (http://dx.doi.org/10.1289/ehp.1104494).

Although the developing brain is considered the critical target organ of MeHg toxicity for children, the cardiovascular system may be most sensitive for adults. In the studies we reviewed, the cardiovascular outcomes included myocardial infarction, blood pressure, heart rate variability, and atherosclerosis. Among the studies that met our definition of a low-level exposure, the studies carried out in Finland were the first to assess the association between MeHg and cardiovascular disease (CVD) (e.g., Salonen et al. 1995). A > 2-fold risk of acute myocardial infarction and mortality from coronary heart disease and CVD was associated with elevated hair mercury (> 2 μg/g). Inclusion of fatty acids had no appreciable effect on the relative risk estimates. As recently reviewed by Roman et al. (2011), subsequent studies have corroborated a potential link between MeHg and acute myocardial infarction. Mercury was associated with accelerated progression of carotid atherosclerosis, as determined by intima-media thickness (Salonen et al. 2000). The association remained significant after adjusting for fatty acids and selenium. Rissanen et al. (2000) reported that fish oil–derived fatty acids reduced the risk of acute coronary events. In a later study of Finnish men, Virtanen et al. (2005) reported that increased mercury exposure was associated with increased risk of acute coronary events and cardiovascular mortality, with adjustment for selenium and fatty acids. These two studies (Rissanen et al. 2000; Virtanen et al. 2005) concluded that mercury may attenuate the protective effects of fish on cardiovascular health. A large multicenter study from Europe showed an increased risk of CVD associated with toenail mercury concentrations after controlling for DHA (docosahexaenoic acid) (Guallar et al. 2002), whereas no association was found in the U.S. Health Professionals Study, with adjustment for DHA plus eicosapentaenoic acid (Yoshizawa et al. 2002). In a nested case–control study combining the U.S. male health professionals and the female registered nurses cohorts (Nurses' Health Study), Mozaffarian et al. (2011) found no adverse effects of mercury exposure on coronary heart disease, stroke, or total CVD. Findings were similar in additional analyses adjusted for DHA, eicosapentaenoic acid, and selenium. In a Swedish nested case–control study with low exposure, Wennberg et al. (2011) found no association between the risk of myocardial infarction and mercury concentration in erythrocytes with adjustment for DHA plus eicosapentaenoic acid. Another nested case–control study reported an inverse association between myocardial infarction and erythrocyte mercury (Hallgren et al. 2001); however, that study did not meet our definition of low-level exposure.

Several studies have found an association between increased mercury and increased blood pressure in adults, although only two met our low-dose exposure definition: Valera et al. (2009) adjusted for fish nutrients (DHA, eicosapentaenoic acid, and selenium), whereas Vupputuri et al. (2005) controlled for fish intake. In cross-sectional population data from the U.S. National Health and Nutrition Examination Survey (NHANES), associations were seen only among individuals who did not consume fish (Vupputuri et al. 2005). Among children more heavily exposed than criteria used in our review, an association between prenatal mercury exposure and childhood blood pressure has been observed in some (Sorensen et al. 1999; Thurston et al. 2007) but not all (Grandjean et al. 2004) studies; however, information on nutrients and fish consumption was not available in these studies.

MeHg may induce oxidative stress, an early biological response that can produce cell damage in several organs or systems including the cardiovascular system (Grotto et al. 2009). Experimental models suggest that oxidative stress plays an important role in the toxicodynamics of mercury (Grotto et al. 2010). A few recent studies have examined associations between mercury exposure and oxidative stress or antioxidant defense in populations exposed through fish consumption, although the findings have been inconclusive (Bélanger et al. 2008; Grotto et al. 2010; Pinheiro et al. 2008). These studies, however, have reported mercury concentrations that exceeded our definition of low-level exposure. Except for Grotto et al. (2010), information on fish intake was not available.

Immunologic Outcomes

In 2000, the NRC summarized the available literature on immunotoxicity of mercury (NRC 2000). Study results showed that occupational exposure to inorganic forms of mercury was associated with alterations in B lymphocytes, T-helper cells, T-suppressor cells, and T-cell proliferative responses (Moszczynski et al. 1995; Queiroz and Dantas 1997a, 1997b). The NRC report also cited several animal studies involving exposure to MeHg and indicators of immunotoxicity (e.g., Ilbäck et al. 1991). The NRC (2000) concluded that the immune system appears to be sensitive to mercury and noted that toxicologic studies have observed effects on immune-cell ratios, cellular response, and the developing immune system. However, at the time of the NRC report, there were no published epidemiologic studies of MeHg and immune function.

For immunologic outcomes, we focused on studies published since the comprehensive NRC report (i.e., post-1999), most of which have hair or blood mercury levels well in excess of the low-level range of our review or focus on elemental mercury exposure. For example, evidence of mercury-associated immunotoxicity, including increased frequency of antinuclear autoantibodies, changes in serum cytokine levels, and risk of malaria infection, has been observed in studies of heavily exposed Amazonian fish eaters and gold-mining populations (Alves et al. 2006; Crompton et al. 2002; Gardner et al. 2010; Nyland et al. 2011b). However, urinary mercury levels reflective of elemental mercury exposure from amalgam dental restorations

Table 3. Summary of findings on MeHg and cardiovascular outcomes.

Study location or group	Outcomes
Positive associations	
Finland	Hair mercury positively related to acute myocardial infarction and CHD and CVD mortality.
Europe and Israel	Toenail mercury positively associated with myocardial infarction
No associations	
Health Professionals Study and Nurses' Health Study	Toenail mercury unrelated to incident CVD
National Health and Nutrition Examination Survey (NHANES)	Blood mercury not associated with blood pressure
Sweden	Erythrocyte mercury not associated with risk of myocardial infarction

See also Supplemental Material, Table S3 (http://dx.doi.org/10.1289/ehp.1104494).

in children has not been associated with immunotoxicity (Shenker et al. 2008).

Table 1 and Supplemental Material, Table S4 (http://dx.doi.org/10.1289/ehp.1104494) summarize the studies with low-level MeHg exposures that we reviewed. Nyland et al. (2011a) reported significant correlations of both maternal and cord blood mercury (respective geometric means of 6.9 μg/L and 9.6 μg/L) with increases in cord blood total IgG among 61 mother–infant pairs in the Brazilian Amazon. In that study, which did not adjust for fish consumption, blood mercury was not associated with either maternal or fetal levels of antinuclear autoantibodies or serum cytokines. In a recent population-based survey of Korean adults (n = 1,990), higher blood mercury (geometric mean, 4.3 μg/L) was associated with increased risk of self-reported atopic dermatitis in multivariable analyses adjusted for fish consumption (Park and Kim 2011). As part of the Osaka Maternal and Child Health Study, Miyake et al. (2011) evaluated 582 mother–child pairs in Japan for mercury exposure, using both maternal hair (median, 1.5 μg/g) and hair collected from their offspring 29–39 months of age (median, 1.4 μg/g). After adjustment for multiple potential confounders, including maternal fish consumption during pregnancy and child fish consumption, the authors detected no association between either maternal or child hair mercury and risk of childhood wheeze or eczema.

Belles-Isles et al. (2002) compared Canadian infants (n = 48) born to a population of subsistence fishers with a reference population (n = 60 infants) from coastal towns (geometric mean cord blood mercury of 1.8 and 0.9 μg/L, respectively). Cord blood mercury was inversely correlated with the proportion of naive helper T cells and plasma IgM levels in cord blood but unrelated to multiple other measures of T-, B-, and natural killer cell proportions and function. Of note, these analyses were not adjusted for potential confounding, including the substantial organochlorine exposures and greater prevalence of smoking during pregnancy among subsistence fishers compared with referent mothers. Bilrha et al. (2003) studied children born to subsistence fishers (n = 47) and town residents (n = 65) from the same Canadian region to expand on assessments in Belles-Isles et al. (2002). In correlational analyses (unadjusted for potential confounders), the authors observed no relationship between cord blood mercury and cord blood lymphocyte activation markers or cytokine secretion.

Summary

Birth outcomes and infant growth. To date, relatively few studies have evaluated the effects of MeHg on fetal growth and gestation. An advantage of studying birth outcomes is the limited time window of exposure. Lee et al. (2010) measured maternal mercury levels at multiple time points during pregnancy, with similar results. The largest number of studies assessed birth weight, but the differing matrices used to measure exposure make comparison between studies challenging. Several studies evaluated cord blood mercury concentrations but employed differing statistical approaches (e.g., analysis of categorical vs. continuous data). Of interest, two studies that provided a statistical estimate adjusted for fish intake both found evidence of reduced birth weight in relation to cord blood mercury concentrations (Lee et al. 2010; Ramon et al. 2009), whereas another study that did not adjust for fish and seafood intake (but did examine plasma PUFA) did not observe such associations (Lucas et al. 2004). Of the studies that evaluated associations of cord blood mercury levels with gestational age, one provided a statistical estimate of an association with prematurity (Xue et al. 2007); another study did not find a relation with gestational age but did not specifically evaluate preterm deliveries (Lucas et al. 2004). Size for gestational age is of interest, but only one study to date has reported on this outcome (Ramon et al. 2009); although results were suggestive of a mercury effect, they lacked statistical power. Thus, the potential impact of low-level MeHg on fetal growth is uncertain, although there is suggestive evidence of an effect. Finally, in addition to possible influences of low-level *in utero* mercury exposure on fetal growth, recent data raise the possibility of effects on postnatal growth (Kim et al. 2011).

Neurocognitive and behavioral outcomes. The literature on low-level mercury and neurodevelopment underscores the importance of exposure timing, precision of the exposure assessment, confounding, age at assessment, the specific neurobehavioral outcome, sex differences, and dose–response modeling in determining the observed results. For example, in children, prenatal exposure may be more deleterious than postnatal exposure for most, but likely not all, neurodevelopmental measures (Myers et al. 2009; Saint-Amour et al. 2006). With some exceptions (Daniels et al. 2004; Plusquellec et al. 2010), studies in which null, or potentially beneficial, associations with mercury were seen typically lacked measures of fish consumption or related nutrients, such as PUFA, which could confound findings and account for null or apparently neuroprotective mercury effects (Cao et al. 2010; Ha et al. 2009; Johansson et al. 2002; Torrente et al. 2005). An example of the complexity of this literature is the variability among associations of low-level prenatal mercury with performance on the Bayley Scales of Infant Development between 12 and 36 months of age. Prenatal mercury effects were seen only at 12 months of age in some populations (Jedrychowski et al. 2006, 2007) or only at 36 months of age in others (Lederman et al. 2008). In contrast, postnatal exposure has been associated with improved Bayley performance at 24 months of age (Cao et al. 2010) but without adjustment for fish consumption. In the low-level exposure literature, differences in exposure and confounding (including diet), as well as differences in neurodevelopmental test sensitivity, may account for some apparent inconsistencies.

Findings in children and adults are difficult to compare, at least in part, because of differences in testing protocols. In addition, among populations with chronic mercury exposure, associations with mercury measured in later life may reflect the long-term developmental consequences of early-life exposure. However, where there is general overlap in assessments, findings are surprisingly concordant. For example, concurrent blood mercury was associated with increased action tremor amplitude in 4- to 6-year-old Inuit children (Després et al. 2005) and poorer fine motor speed and dexterity in Brazilian adults (Yokoo et al. 2003). Regardless of age, certain domains of function may be more sensitive to mercury toxicities, including memory (Freire et al. 2010; Oken et al. 2005; Weil et al. 2005), verbal or language skills (Freire et al. 2010; Lederman et al. 2008; Oken et al. 2008), and visual-motor functions (Oken et al. 2008; Surkan et al. 2009). In contrast, except in a case–control analysis of ADHD (Cheuk and Wong 2006), adverse behaviors were not associated with mercury exposure in children (Cao et al. 2010; Ha et al. 2009; Plusquellec et al. 2010).

Cardiovascular outcomes. No clear pattern has been observed among the limited number of studies that assessed the association between low-level mercury and cardiovascular function. The evidence, however, suggests that adverse cardiovascular effects can occur at very low levels of mercury exposure. For example, men who had a hair mercury concentration of ≥ 2 μg/g had a 2-fold increased risk of suffering an acute myocardial infarction or dying from coronary heart disease or CVD compared with less-exposed men (Salonen et al. 1995). Although essential fatty acids from fish may reduce the risk of acute coronary events, mercury in fish could attenuate this beneficial effect (Rissanen et al. 2000). Eastern Finnish men in the lowest two tertiles of hair mercury concentration (0–2 μg/g) who also were in the highest quintile of serum fatty acid levels had a 67% reduced risk of acute coronary events compared with men in the highest tertile of hair mercury who were in the lowest quintile of serum fatty acids. In that cohort, the increased risk seemed to occur at hair mercury concentrations > 2 μg/g, that is, only 2 times the level corresponding to

daily mercury intake at the U.S. EPA reference dose (a level estimated to be without significant risk of adverse effects over a lifetime) (Salonen et al. 1995). In contrast, in a large multicenter cohort in Europe, an increased risk of myocardial infarction was found in participants with toenail mercury concentrations of 0.36 μg/g, approximating an even lower hair mercury level of < 1 μg/g (Guallar et al. 2002; Ohno et al. 2007).

Immunologic outcomes. Relatively high exposure to elemental mercury has been linked to a range of immunologic outcomes (including markers of autoimmunity) in epidemiologic studies, but evidence of immunotoxicity of low-level MeHg is inconclusive in the limited literature available to date. It is difficult, however, to make any definitive statements as to which forms of mercury are likely to affect which immune components. Much of the available epidemiologic literature is limited by small sample sizes, incomplete adjustment for potential confounders, and lack of consistency across exposure media (e.g., only hair in some populations, only urine in others), which have impeded making the comparisons across studies that are necessary for refined hypothesis generation and testing. Dialogue among mercury researchers is also needed to identify optimal measures of potential immune impairment.

Priorities for Future Studies

Despite evidence of possible differences in mercury toxicities between the sexes (Marsh 1994), most studies did not report assessing such differences. Only two of the reviewed studies on neurologic outcomes reported sex differences in mercury effects: Only male infants had mercury-associated decrements in behavior (Gao et al. 2007), and only adult women reported increased psychiatric symptoms associated with mercury (Philibert et al. 2008). In addition to sex-specific effects, other host factors could influence susceptibility to MeHg effects. Although research on these factors is scarce, a recent Korean study of fetal growth found evidence of genetic susceptibility, with genetic variation in *GSTM1* and *GSTT1* affecting risk (Lee et al. 2010). Future studies should emphasize the use of the most precise exposure indicators in sensitivity analyses to model the impact of likely imprecision (Budtz-Jørgensen et al. 2003). Similarly, future studies should use outcome measures for which there are mechanistic or other *a priori* bases for assuming mercury sensitivity. The potential for nonlinear dose–response relationships (e.g., a threshold dose response) needs to be considered consistently. Because MeHg originates from fish and seafood, which also contain nutrients that may be beneficial to health (including birth outcomes, neurodevelopment, cardiovascular health, and immune function), proper adjustment for potential negative confounding by nutrition is crucial in any future study.

Summary of Findings

- The possibility that MeHg at low exposure levels might affect fetal growth among susceptible subgroups and infant growth requires further investigation.
- There is evidence that low levels of prenatal MeHg exposure may cause early childhood neurocognitive effects. The possibility of nonlinear effects, as well as possible differential effects by sex, should be evaluated for older children and adults.
- There are no clear patterns across populations or for specific study end points for cardiovascular effects of low-level MeHg exposure. Future studies targeted at mechanisms of effects may be informative (e.g., effects on heart rate variability). In addition, sexual dimorphism and nonlinearity should be considered.
- Although there are some indications of MeHg-associated immune effects, epidemiologic studies addressing this question are scarce, have small sample sizes, and include limited assessment of important potential confounders. Thus, we cannot draw any conclusions at this time.

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EXHIBIT 3

To Comments of Public Health and Environmental Groups
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Martine Bellanger, *et al.*
"Economic benefits of methylmercury exposure control in Europe: Monetary value of neurotoxicity prevention"
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ENVIRONMENTAL HEALTH

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Economic benefits of methylmercury exposure control in Europe: Monetary value of neurotoxicity prevention

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Abstract

Background: Due to global mercury pollution and the adverse health effects of prenatal exposure to methylmercury (MeHg), an assessment of the economic benefits of prevented developmental neurotoxicity is necessary for any cost-benefit analysis.

Methods: Distributions of hair-Hg concentrations among women of reproductive age were obtained from the DEMOCOPHES project (1,875 subjects in 17 countries) and literature data (6,820 subjects from 8 countries). The exposures were assumed to comply with log-normal distributions. Neurotoxicity effects were estimated from a linear dose-response function with a slope of 0.465 Intelligence Quotient (IQ) point reduction per μg/g increase in the maternal hair-Hg concentration during pregnancy, assuming no deficits below a hair-Hg limit of 0.58 μg/g thought to be safe. A logarithmic IQ response was used in sensitivity analyses. The estimated IQ benefit cost was based on lifetime income, adjusted for purchasing power parity.

Results: The hair-mercury concentrations were the highest in Southern Europe and lowest in Eastern Europe. The results suggest that, within the EU, more than 1.8 million children are born every year with MeHg exposures above the limit of 0.58 μg/g, and about 200,000 births exceed a higher limit of 2.5 μg/g proposed by the World Health Organization (WHO). The total annual benefits of exposure prevention within the EU were estimated at more than 600,000 IQ points per year, corresponding to a total economic benefit between €8,000 million and €9,000 million per year. About four-fold higher values were obtained when using the logarithmic response function, while adjustment for productivity resulted in slightly lower total benefits. These calculations do not include the less tangible advantages of protecting brain development against neurotoxicity or any other adverse effects.

Conclusions: These estimates document that efforts to combat mercury pollution and to reduce MeHg exposures will have very substantial economic benefits in Europe, mainly in southern countries. Some data may not be entirely representative, some countries were not covered, and anticipated changes in mercury pollution all suggest a need for extended biomonitoring of human MeHg exposure.

Keywords: Economic evaluation, Methylmercury, Prenatal exposure, Neurodevelopmental deficits

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Background

Methylmercury (MeHg) is a well-documented neurotoxicant, and prenatal exposures are therefore of particular concern [1,2]. The main sources of exposure are seafood and freshwater fish [3]. Thus, MeHg exposures vary with dietary habits, contamination levels, and species availability. While the distribution of MeHg exposures has been studied in substantial detail in the United States [4], only scattered information is available on MeHg exposures in Europe.

Because the critical effect of MeHg exposure is developmental brain toxicity, exposures among women of reproductive age groups are of primary concern [5,6]. As has previously been determined in regard to lead exposure [7], developmental MeHg exposure is linked to a loss in Intelligence Quotient (IQ), with associated lower school performance and educational attainment, thereby leading to long-term impacts on societal benefits of pollution abatement [8]. These consequences may be expressed in terms of economic impacts, as has been demonstrated in United States [9,10]. However, few economic evaluations have been performed in Europe [8,11,12], primarily because of the lack of exposure data.

Based on harmonised protocols developed in COPHES [13], the DEMOCOPHES project has just completed a multi-country study of hair-mercury concentrations in women of reproductive age groups in 17 European countries. In conjunction with literature data, we now utilise the exposure data to generate estimates of economic impacts of MeHg exposures in Europe.

The economic assessment relies on several assumptions. The hair-Hg concentrations is used as the main exposure indicator in this study, and any blood-based measurements also considered are expressed in terms of hair-mercury using a conversion factor of 250 [14,15]. In regard to the dose-response function (DRF), a linear model is usually the default [14], although it may not necessarily provide the best statistical fit to the data [16]. We therefore used the linear slope as the primary DRF and then conducted a sensitivity analysis using the log function, where each doubling of exposure above the background causes the same deficit of 1.5 IQ points [10].

With regard to background exposures and the possible existence of a threshold, the U.S. EPA's Reference Dose (RfD) of 0.1 μg/kg body weight/day corresponds to a hair-Hg concentration of about 1 μg/g hair [14]. Updated calculations [17] resulted in an adjusted biological limit about 50% below the recommended level, corresponding to 0.58 μg/g hair. The validity of this lower cut-off point below the RfD is supported by recent studies of developmental neurotoxicity at exposure levels close to the background [18-21]. We assumed that, below the 0.58 μg/g cut-off point, only negligible adverse effects would exist. As additional reference point, we use a tolerable limit proposed by the World Health Organization (WHO), which corresponds to a hair-Hg concentration of approximately 2.5 μg/g [22]. This limit takes into account the possible compensation of MeHg toxicity by beneficial nutrients in seafood [22].

Methods

Exposure information

DEMOCOPHES is a cross-sectional survey of European population exposure to environmental chemicals. The human exposure biomarkers included the hair-mercury concentration and was collected in 17 European countries based on children aged 6–11 years and their mothers. A common European protocol, developed by the COPHES project, was followed in each country. The main inclusion and exclusion criteria were (1) residence in the study area for at least five years, and (2) not having metabolic disturbances. The period of sampling was September 2011 to February 2012. A total of 1,875 child-mother pairs were recruited from urban and rural communities in the participating countries, while excluding exposure hot-spots. Major efforts were carried out to achieve high quality and comparability of data. Standard operational procedures for total mercury concentrations in hair were developed and validated by the Laboratory of Environmental Toxicology in Spain, to ensure comparable measurements, which included a strict quality assurance programme, in which seventeen European laboratories participated. Each DEMOCOPHES partner contributed information to allow estimation of the underlying distribution of exposures in the population, where rural and urban results were merged. In addition, each partner provided the frequencies of results above the cut-off levels of 0.58 μg/g, 1.0 μg/g, and 2.5 μg/g. The latter corresponds to WHO's tolerable limit, which takes into account likely toxicity compensation by beneficial nutrients in seafood [22].

Additional information on MeHg exposures in Europe was obtained to complement the DEMOCOPHES data. Thus, information of similar quality was extracted from published articles (Miklavčič, unpublished data), and distribution information from comparable studies was obtained from Belgium, Denmark, France, Norway, Slovenia, and the United Kingdom. As explained below, missing information was calculated assuming a log-normal distribution of the exposures.

Exposure distributions

Using the number of births in 2008 and the observed hair-Hg concentrations, we estimated the number of births exceeding the three exposure limits for each country and obtained the sum for all of the EU. For missing EU member states, MeHg exposures were assumed to be the same as a neighbouring country. The year 2008 was chosen as the closest to the time during which the exposure data had

been collected, and it allowed complete information for the calculations envisaged. Due to the existence of sampling uncertainty, "smoothed" proportions exceeding the three limits were calculated assuming log-normal distributions. Because log-transformed concentrations would follow a normal distribution, the parameters in the log-normal distributions could be estimated by standard normal distribution methods. Each data set included probabilities (prob) for being below specific percentiles (perc). The parameters in the logarithmic distributions were therefore obtained as the intercept and slope when regressing log(perc) on Φ^{-1} (prob), where Φ is the cumulative distribution function of the standard normal distribution. Using the total numbers of births in 2008, numbers of births exceeding the three cut-off limits in each country were calculated from observed and smoothed distributions.

Calculation of IQ benefits

A linear dose-response function was applied as the default model [14]. Thus, as a 1 μg/L increase of the cord-blood mercury concentration is associated with an average adverse impact on IQ of 0.093 times the standard deviation (which is standardised to be 15), each increase in the maternal hair-mercury by 1 μg/g is associated with an average loss of 0.465 IQ points [10]. This slope is based on a range of neuropsychological tests and subtests administered in the Faroe Islands study at age 7 years [23]. As some recent studies [18-21] suggest MeHg-associated deficits close to or below the cut-off level of 0.58 μg/g hair, the calculations may represent an underestimate. In addition, the slope may be steeper at low exposure levels. Thus, a log model was used for sensitivity analyses. In this model, a doubling in prenatal MeHg exposures is associated with a delay in development of 1.5–2 months at age 7, which corresponds to about 10% of the standard deviation, i.e. 1.5 IQ points [1]. Again, we applied this slope for exposures above the 0.58 μg/g the cut-off point.

To estimate the benefits at exposures above the cut-off point, we calculated the average hair-mercury concentration in women exceeding 0.58 μg/g based on 1,000,000 simulations from the estimated log-normal distribution (as described above). After deduction of the 0.58 μg/g and multiplication by the slope factor, an average IQ benefit was obtained. This amount was then multiplied by the annual number of births exceeding the cut-off level. A similar calculation was made in the logarithmic dose-response model except that here we calculated the average log-transformed mercury concentration in women exceeding 0.58 μg/g, deducted log(0.58) and multiplied by the slope factor of the logarithmic dose-response model (1.5/log(2)).

Annual benefits of exposure reduction

The major component of the social costs incurred by an IQ reduction is loss of productivity and thus a lower earning potential [9,24]. The economic consequence of prenatal exposure to MeHg is valued as the lifetime earning loss per person. We assumed singleton births only, so that the number of women was equal to the cohort size. We also assumed that IQ deficits present at age 7 years or preschool ages are permanent [25]. The estimated individual benefits are the avoided lifetime costs using 2008 data (slightly lower benefits are obtained if referring to more recent years, and benefits are only minimally affected by subsequent membership of the Euro zone). The benefit estimates originate from the 2008 figure of €17,363 per IQ point as recently calculated for France based on data from the United States [24]. For the various European countries involved, this value is adjusted for differences in purchasing power. While simple currency exchange conversion and Gross Domestic Product (GDP) per capita do not adjust for price differences, Purchasing Power Parity (PPP) conversion rates allow for comparison based on a common set of average international prices [26,27]. We also carried out the calculations after adjustment for productivity as the ratio of PPP-adjusted real GDP/capita in each country in relation to the US as a reference. The estimated value of an IQ point then takes into account the impact of labour costs and productivity (Additional file 1).

Results

Table 1 and Additional file 2 show summary information on MeHg exposures in the European countries covered by DEMOCOPHES or other exposure studies. There is a clear trend from north and east to southern countries, most likely due to differences in dietary habits and availability of large fish species from the Mediterranean (the sources of exposure were not considered in the present study). In Table 1, exposures in Austria were assumed to be similar to those in Germany, as suggested by available data [28]. Exposure information from the Flemish part of Belgium [29,30] do not differ much from the national data obtained in DEMOCOPHES, which were therefore used for the calculations. The Flemish data were used to represent exposures in The Netherlands. In the absence of exposure data from Estonia, Finland, Latvia, and Lithuania, the DEMOCOPHES exposure information from Sweden was applied. National data from France are available [31] and have been used in recent economic calculations [8]. Data for Croatia and Greece were obtained from a recent birth cohort study [32]. Two exposure studies had been carried out in Italy, one in the northeast [32] and one in Naples [33], and a joint distribution was therefore used to obtain national exposure distributions that would also apply to Malta. Thus, a log-normal distribution was first fitted to each Italian data subset, and then the parameters of a joint log-normal distribution were determined as the mean of the parameters for the two distributions. Recent results

Table 1 Annual numbers of births and numbers exceeding three cut-off limits, as indicated by hair-mercury analyses (in μg/g) in population samples in European countries

Country[a]	Annual number of births (2008)	Number of samples[b]	Above 0.58 μg/g		Above 1.0 μg/g		Above 2.5 μg/g	
			Proportion in sample (%)	Estimated number of births	Proportion in sample (%)	Estimated number of births	Proportion in sample (%)	Estimated number of births
Austria	77,800	NA	(6.7)	5,213	(0.8)	622	(0)	0
Belgium	127,200	129	28.7	36,506	9.3	11,830	0	0
		242[c]	23.2	29,510	7.2	9,158	0	
Bulgaria	77,700	NA	(4.2)	3,263	(1.2)	932	(0.8)	622
Croatia	43,800	234[d]	52.0	22,776	22.0	9,636	4.7	2,059
Cyprus	9,200	60	36.7	3,376	18.3	1,684	3.3	304
Czech Republic	119,600	120	5.0	5,980	0.8	957	0	0
Denmark	65,000	145	36.6	23,790	13.1	8515	0.7	455
Estonia	16,000	NA	(10.0)	1,600	(2.0)	320	(0)	0
Faroe Islands	675	505[e]	62.6	423	30.2	204	5.3	36
Finland	59,500	NA	(10.0)	5,950	(2.0)	1,190	(0)	0
France	829,300	126[f]	44.0	364,892	14.51	120,331	0.61	5,059
Germany	682,500	120	6.7	45,728	0.8	5,460	0	0
Greece	118,300	454[d]	78	92,274	57	67,431	14	16,562
Hungary	99,100	120	0.83	823	0	0	0	0
Ireland	74,000	120	10.8	7,992	2.5	1,850	0	0
Italy	576,700	891[d] + 115[g]	(65.6)	378,315	(36.8)	212,226	(5.7)	32,872
Latvia	23,834	NA	(10.0)	2,383	(2.0)	477	(0)	0
Lithuania	35,100	NA	(10.0)	3,510	(2.0)	702	(0)	0
Luxembourg	5,600	55	32.7	1,831	18.2	1,019	0	0
Malta	4,100	NA	(65.6)	2,690	(36.8)	1,509	(5.7)	234
Netherlands	184,600	NA	(23.2)	42,827	(7.2)	13,291	(0)	0
Norway	60,500	119[h]	27.7	16,759	5.9	3,570	0	0
Poland	414,500	120	1.7	7047	0	0	0	0
Portugal	104,600	120	90.8	94,977	57.5	60,145	8.3	8,682
Romania	221,900	120	4.2	9,320	1.2	2,663	0.8	1,775
Slovakia	57,400	129	5.43	3,117	0.8	459	0	0
Slovenia	21,800	156	22.0	4,796	7.7	1,679	1.9	414
Spain	519,800	120	88.5	460,023	74.2	385,692	31.7	164,777
Sweden	109,300	100	10.0	10,930	2.0	2,186	0	0
Switzerland	76,700	120	5.0	3,835	2.1	1,611	0	0
United Kingdom	794,400	4134[h]	31.0	246,264	5.1	40,200	0	0
Total EU (27)	5,400,000			1,865,416		903,169		231,754

Exposures in EU countries without recent data are estimated from neighbouring countries (modelled results not based on observed distributions are given in parenthesis).

[a] For countries without available exposure data (for number of samples, NA denotes not available), data from a neighbouring country have been applied to allow EU-wide estimates, and frequencies are given in parenthesis. This applies to Austria (data from Germany were used), Bulgaria (Romania), Netherlands (Flanders [30]), and Estonia, Finland, Latvia, and Lithuania (Sweden); [b] All data are from DEMOCOPHES, unless otherwise noted; [c] [30]; [d] [32]; [e] Pal Weihe, unpublished data; [f] [31]; [g] [33]; [h] Jean Golding, pers.comm.

from the Norwegian national birth cohort were used for this country [34]. As DEMOCOPHES data from the United Kingdom covered only a small rural sample, we relied on data on blood-mercury in pregnant women obtained from the ALSPAC birth cohort study in the 1990s [35]. Additional exposure data from Ukraine [36] supported the notion that MeHg exposures in Eastern Europe are low, with only small percentages

exceeding the cut-off level, but this study was considered too small to be used for detailed calculations. The same applied to several other sources identified (Miklavčič, unpublished data).

The estimated number of annual births in the EU that exceed the 0.58 μg/g cut-off is about 1.8 million (Table 1, Additional file 3). The EPA limit is exceeded in about 900,000 births, and the WHO limit in 200,000 births within the EU. As each study is subject to sampling uncertainty, log-normal distribution models showed similar, though sometimes slightly higher, proportions exceeding the 0.58 cut-off level (Table 2). The data from Eastern European countries and from Croatia, the Faroe Islands, Norway, and Switzerland suggest that, within Europe, the great majority of births exceeding the various limits occur in EU member states.

Table 2 presents the estimated IQ losses associated with the MeHg exposures using the linear model, along with the estimates of economic impacts. We used both the observed data and the modelled distributions, and only small differences were seen, thus supporting the notion that the log-normal exposure distribution has an appropriate fit. The greatest benefits accrue for the largest countries with the highest proportions of subjects with exposures above the cut-off level. The total benefit from control of MeHg exposure was the highest for Spain and the lowest for Hungary. On a per capita basis, the calculated benefits are the greatest in the Faroe Islands and the southern countries, Spain, Greece, Portugal, Italy, and Croatia. The total annual benefits in terms of IQ points within the EU were estimated to be in excess of 600,000 per year for the linear DRC. With an average benefit of €13,579 per IQ point, the total economic benefits are estimated to exceed €9,000 million per year. When adjustment for productivity is included, the benefits are somewhat lower for several countries, and the EU total is slightly less than €8,000 million per year (Additional file 3).

For comparison, Table 3 shows the estimated IQ losses and economic benefits using the log transformed DRF. Due to the steeper curve shape at exposures close to the cut-off point of 0.58 μg/g, the estimated benefits are about 4-fold greater, at about 2.7 million IQ points per year, which correspond to total benefits for the EU of approximately €39,000 million or, after productivity adjustment, €33,000 million.

Discussion

This study provides for the first time regional European data on economic benefits of controlling MeHg exposure in relation to prevention of developmental neurotoxicity. It relies on data from a multi-country study of hair-Hg concentrations with a high level of quality assurance and with similar population sampling criteria. In addition, available data from other studies have been taken into consideration to provide supplementary information, thereby allowing EU-wide estimates to be calculated. Given the low MeHg exposures in Eastern Europe and the relatively small contributions from Croatia, the Faroe Islands, Norway, and Switzerland, the results suggest that benefits for all of Europe will not be substantially above the benefits calculated for the EU.

Several assumptions and caveats must be acknowledged. The hair-Hg concentration is an established biomarker of human MeHg exposure and is generally considered reliable [14]. We used available data from DEMOCOPHES and other sources, with most studies including only about 120 subjects. The sampling size and strategy may have underestimated the occurrence of uncommon high-level exposures, which would weigh more in the calculation of IQ benefits. Adjustment for this bias is obtained in the modelled distributions, which tended to show slightly greater benefits. Although these calculations rely on an assumption of a log-normal distribution of the exposures, the concurrence of the two sets of estimates support the validity of this assumption.

In calculating the IQ benefits, we used a linear dose-response function for the decrease in IQ at increased prenatal MeHg exposures, and this curve shape is an approximation of unknown validity. As has been documented for lead [37], a logarithmic DRF may be plausible, and a log curve shows a slightly better fit [16]. As the results for the log curve (Table 3) are about 4-fold higher than those obtained for the linear curve, the benefits calculated in Table 2 must be considered likely underestimates. In recent calculations using French data using similar methods [8], the logarithmic curve shape also resulted in substantially higher estimates.

The cut-off level assumed to be 0.58 μg/g hair may also result in underestimated benefits. Recent data from Poland [20], Japan [21] and the United States [18,19] suggest that a lower threshold is likely. If the threshold is indeed lower than we have assumed, the benefits of controlling MeHg exposures will likely be greater, although an additional effort may be required to achieve such lower exposures. Further, given that the much higher tolerable limit of 2.5 μg/g is likely exceeded by 200,000 births in the EU per year, clear benefits will accrue already from controlling the very highest exposures.

The IQ benefits from controlling mercury pollution were translated into economic impacts based on the calculated current life-time income benefits from a higher IQ level. These benefits are mainly based on studies carried out in the United States [24,38], and it is possible that IQ-linked differences in life-time incomes may not be the same in Europe. Adjustment for differences in purchasing power has been included to take this issue into partial account. We used data from 2008 to secure complete data sources;

Table 2 Annual number of births with excess exposure, average hair-Hg concentration, IQ benefit from prevention of excess exposure, and the value of the IQ benefits

Country	Number of births above 0.58 μg/g		Average concentration above 0.58 μg/g	Benefit in IQ points		Value of 1 IQ point (Euro)	Total benefit (million Euro)	
	Modelled	Observed		Modelled	Observed		Modelled	Observed
Austria	3,812	5,213	0.917	597	817	16,044	9.6	13.1
Belgium	39,686	36,506	0.939	6,625	6,094	16,458	109.0	100.3
Bulgaria	3,186	3,263	1.455	1,296	1,328	7,529	9.8	10.0
Croatia	21,769	22,776	1.355	7,845	8,208	11,320	88.8	92.9
Cyprus	3,514	3,376	1.311	1,195	1,148	13,747	16.4	15.8
Czech Republic	5,143	5,980	0.847	639	742	10,797	6.9	8.0
Denmark	22,815	23,790	1.027	4,742	4,945	20,220	95.9	100.0
Estonia	1,840	1,600	0.846	228	198	10,339	2.4	2.0
Faroe Islands	406	423	1.323	140	146	20,220	2.8	2.9
Finland	6,843	5,950	0.846	846	736	17,288	14.6	12.7
France	405,528	364,892	0.989	70,186	69,397	17,363	1,218.6	1,204.9
Germany	33,443	45,728	0.917	5,241	7,166	15,292	80.1	109.6
Greece	94,403	92,274	1.563	50,131	49,000	13,201	661.8	646.9
Hungary	892	823	0.884	126	116	9,691	1.2	1.1
Ireland	7,104	7,992	0.946	1,209	1,360	17,927	21.7	24.4
Italy	378,315	(378,315)	1.045	81,801	(81,801)	17,062	1,395.7	(1,395.7)
Latvia	2,741	2,383	0.846	339	295	11,568	3.9	3.4
Lithuania	4,037	3,510	0.846	499	434	9,661	4.8	4.2
Luxembourg	1,870	1,831	1.212	550	538	17,062	9.4	9.2
Malta	2,690	(2,690)	1.045	582	(582)	11,111	6.5	6.5
Netherlands	45,227	42,827	0.909	6,919	6,552	15,857	109.7	103.9
Norway	16,759	16,759	0.866	2,237	2,229	20,051	44.8	44.7
Poland	6,218	7,047	0.751	494	560	9,979	4.9	5.6
Portugal	94,349	94,977	1.482	39,573	39,836	12,221	483.6	486.8
Romania	9,098	9,320	1.455	3,702	3,797	8,187	30.3	31.1
Slovakia	2,468	3,117	0.899	366	462	10,037	3.7	4.6
Slovenia	4,840	4,796	1.194	1,382	1,369	11,939	16.5	16.3
Spain	479,775	460,023	2.136	347,137	332,845	13,558	4,706.5	4,512.7
Sweden	12,570	10,930	0.846	1,555	1,352	17,167	26.7	23.2
Switzerland	6,520	3.835	0.902	976	574	18,346	17.9	10.5
United Kingdom	248,647	246,200	0.81	26,593	26,338	15,324	407.5	403.5
EU Total	1,926,652	1,865,365		654,551	639,804		9,458	9,256

Data are for European countries with information on methylmercury exposure distributions. For countries without detailed observed data available, the modelled results are given in parenthesis. Sources of underlying data are as in Table 1.

the use of more recent records would change the estimates only slightly. An alternative approach might be to calculate benefits from prevention of specific diseases, e.g. for mental retardation or autism, associated with MeHg exposure. However, the attributable risks associated with increases in MeHg exposure are unknown, and such calculations are therefore uncertain [10,39].

Some sources of imprecision in exposure estimates must be emphasized. Thus, in several cases when exposure information was not available for an EU member state, data from a neighbouring country were used as a proxy. Further, the results reported in DEMOCOPHES and in published reports may not be representative for each country. Although high fish consumers may possibly have been oversampled, it is more likely that the avoidance of known exposure hotspots resulted in lowered exposure estimates. In addition, especially for small studies, an element of uncertainty

Table 3 Annual number of births with excess exposure, the average log hair-Hg concentration, and IQ benefit and value from prevention of excess exposure (logarithmic dose-effect relationship)

Country	Number of births above 0.58 μg/g	Average log concentration above 0.58 μg/g	Benefit in IQ points	Value of 1 IQ point (Euro)	Total benefit (million Euro)
Austria	3,812	−0.157	3,199	16,044	51.3
Belgium	39,686	−0.128	35,790	16,458	589.0
Bulgaria	3,186	0.128	4,638	7,529	34.9
Croatia	21,769	0.142	32,350	11,320	366.2
Cyprus	3,514	0.109	4,972	13,747	68.3
Czech Republic	5,143	−0.216	3,658	10,797	39.5
Denmark	22,815	−0.060	23,932	20,220	483.9
Estonia	1,840	−0.214	1,317	10,339	13.6
Faroe Islands	406	0.139	600	20,220	12.1
Finland	6,843	−0.214	4,897	17,288	84.7
France	405,528	−0.053	368,742	17,363	6,402.5
Germany	33,443	−0.157	28,060	15,292	429.1
Greece	94,403	0.355	183,808	13,201	2,426.4
Hungary	892	−0.186	692	9,691	6.7
Ireland	7,104	−0.132	6,345	17,927	113.7
Italy	378,315	−0.036	416,490	17,062	7.106.2
Latvia	2,741	−0.214	1,962	11,568	22.7
Lithuania	4,037	−0.214	2,889	9,661	27.9
Luxembourg	1,870	0.053	2,419	17,062	41.3
Malta	2,690	−0.036	2,961	11,111	32.9
Netherlands	45,227	−0.155	38,144	15,857	604.8
Norway	16,759	−0.198	12,574	20,051	252.1
Poland	6,218	−0.312	3,131	9,979	31.2
Portugal	94,349	0.277	167,777	12,221	2,050.4
Romania	9,098	0.128	13,245	8,187	108.4
Slovakia	2,468	−0.173	1,986	10,037	19.9
Slovenia	4,840	0.034	6,061	11,939	72.4
Spain	479,775	0.561	1,148,026	13,558	15,564.9
Sweden	12,570	−0.214	8,996	17,167	154.4
Switzerland	6,520	−0.167	5,329	18,346	97.8
United Kingdom	248,647	−0.244	161,816	15,324	2,479.7
EU Total	1,884,563		2,645,953		39,061

Data from European countries, sources of underlying data are as in Table 1.

exists with regard to the frequencies of the highest exposures, although this problem was addressed by modelling a log-normal distribution of exposures. Temporal variation and time trends may also play a role, especially in regard to older data. We have assumed stable diets, so that any seasonal or other time trends as well as the time dependence of MeHg sensitivity during brain development would not matter for the calculation of impacts.

Our focus on the loss in life-time earnings is similar to the avoidable costs previously calculated in relation to lead exposure [24]. Other costs were ignored, such as direct medical costs linked to treatment or interventions for children with neurodevelopmental disorders. We also neglected indirect costs, such as those related to special education or additional years of schooling for children as a consequence of these disorders, as well as intangible costs. In addition, our study did not consider other avoided direct health care costs in the longer term, such as those potentially related to the treatment of cardiovascular or neurodegenerative effects of MeHg exposure, which could be important for high fish consumers [2], but would be difficult to estimate. Any compensation of the

IQ benefit due to special education and other remedies was not taken into account. Overall, the estimates presented in Table 2 are likely underestimates of the total benefits of MeHg exposure abatement.

Clear differences are apparent between European countries. Seafood and freshwater fish constitute the main source of exposure, but countries with high fish consumption levels, such as Spain and Norway, clearly show great differences in MeHg exposure that are undoubtedly related to the choice of fish species consumed as well as the contamination level. The high exposure levels observed in Spain are in accordance with other studies [40,41]. The elevated exposures in the Faroes are likely related to the occasional consumption of pilot whale meat [23].

Calculations from the United States have resulted in several greatly varying estimates, depending on the DRF assumptions. One comparable estimate put the aggregate economic benefit for each annual birth cohort in the US at $8.7 billion (range: $0.7–$13.9 billion for year 2000) [10]. We recently calculated the annual benefit for the US at about 264,000 IQ points, which would correspond to benefits of approximately $5 billion [42]. The EU benefits of over 600,000 IQ points are much higher. However, in comparing the figures for the US and the EU, note should be taken that annual number of births in the EU (5.4 million) are 27% greater than the 4.2 million births in the US per year. In addition, MeHg exposures in parts of Europe are higher than in the US [4]. On a global scale, benefit estimates can be extended on the basis of GDP values adjusted for PPP and productivity, but the validity of such calculations is limited by the lack of exposure assessments [43]. However, the present study leaves little doubt that global benefits substantially exceed $20 billion.

The present study did not aim at calculating annual costs of investments in pollution abatement due to the paucity of available data. Relevant investment costs would consider mercury emissions controls in coal-fired power plants, reduction of mercury usage in the chlorine industry, measures taken in dentistry, plus expenses for recycling and treatment of mercury releases. Some information is available and suggests that one-time expenses may be quickly balanced by the cumulated annual benefits from exposure abatement [9]. However, mercury emissions control needs to be carried out on a global level due to the regional and hemispherical dispersion of mercury releases [43]. These costs would likely have additional socioeconomic yields from better control of mercury emissions, e.g. job creation and modernization of capital equipment.

The control of inorganic mercury emissions will only result in diminished MeHg exposure in the long term, and the benefits will therefore be delayed. As MeHg exposure mainly originates from seafood and freshwater fish, public health advice on dietary choices is an important element of the intervention [6,44]. Due to the essential nutrients present in seafood [3], a reduction in MeHg exposure should not be sought through a decrease or replacement of fish in the diet. A prudent advice would be to maintain fish consumption and minimise the MeHg exposure by consumption of fish known to have lower MeHg concentrations, e.g., smaller species, younger fish, and catches from less polluted waters. Such advice should be directed toward women during pregnancy as the most cost-effective preventive action. Restricted consumption of large, piscivorous fish species may also benefit overfished populations of pelagic fish, such as tuna [45].

The successful completion of the DEMOCOPHES project and the complements from other exposure studies in Europe illustrate the feasibility and usefulness of biological monitoring approaches, in particular when relying on hair samples that may be easily obtained, stored and transported. While such studies have become a routine function in the United States through the National Health And Nutrition Examination Survey [4], and the biomonitoring reports from the Centers for Disease Control and Prevention have become key resources for research on human exposures to environmental chemicals, Europe has lagged behind. Following international policy decisions to decrease global mercury pollution, such human biomonitoring studies will be crucial to monitor the effects of the interventions.

Conclusions

Annual benefits of removing Hg exposure can be estimated to be approximately €9 billion in Europe. While our results support enhanced public policies for the prevention of MeHg exposure, the economic estimates are highly influenced by uncertainties regarding the dose-response relationship. Thus, a logarithmic response curve results in 4-fold higher benefit estimates. In addition, benefits might be underestimated because costs linked to all aspects of neurotoxicity and long-term disease risks have not been considered. These European data and the calculated economic benefits support the need for interventions to minimize exposure to this hazardous pollutant.

Additional files

Additional file 1: Conversion rates, 2008.

Additional file 2: Exposure distributions.

Additional file 3: IQ calculation spreadsheet.

Abbreviations

DRF: Dose-response Function; EPA: Environmental Protection Agency; EU: European Union; GDP: Gross Domestic Product; hair-Hg: Mercury concentration in hair; MeHg: Methylmercury; IQ: Intelligence Quotient;

perc: Percentile; PPP: Purchasing Power Parity; prob: Probability; RfD: Reference Dose; US: United States; WHO: World Health Organization.

Competing interests

PG is an editor of this journal but did not participate in the editorial handling of this manuscript. The authors declare that they have no competing interests.

Authors' contributions

MB, CP, EBJ and PG planned the economic evaluation, carried out the calculations, and drafted the manuscript. AM reviewed published data on MeHg exposure. DA coordinated the contributions of the 17 DEMOCOPHES countries. AC and ME were responsible for the development and follow-up of the Standard Operating Procedures and Quality Assurance for hair sampling and mercury analyses in support to comparability of DEMOCOPHES measurements. DA, MB2, AC, MČ, PC, FD, MEF, AEG, KH, AK, LEK, MK-G, GK, DL, AM, MFR, PR, JST, and PW contributed unpublished exposure data from European countries and act as guarantors of the data applied. All authors commented on the draft manuscript, and all authors read and approved the final version.

Authors' information

National guarantors of the DEMOCOPHES data are listed as co-authors. The DEMO/COPHES Consortium that established and tested harmonised human biomonitoring on a European scale (www.eu-hbm.info) also included Jürgen Angerer, Pierre Biot, Louis Bloemen, Ludwine Casteleyn, Milena Horvat, Anke Joas, Reinhard Joas, Greet Schoeters, and Karen Exley.

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EXHIBIT 4

To Comments of Public Health and Environmental Groups
on Docket ID No. EPA-HQ-OAR-2009-0234
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Yanxu Zhang, *et al*.
"Observed decrease in atmospheric mercury explained by global decline in anthropogenic emissions"
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Observed decrease in atmospheric mercury explained by global decline in anthropogenic emissions

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Observations of elemental mercury (Hg^0) at sites in North America and Europe show large decreases (~1–2% y^{-1}) from 1990 to present. Observations in background northern hemisphere air, including Mauna Loa Observatory (Hawaii) and CARIBIC (Civil Aircraft for the Regular Investigation of the atmosphere Based on an Instrument Container) aircraft flights, show weaker decreases (<1% y^{-1}). These decreases are inconsistent with current global emission inventories indicating flat or increasing emissions over that period. However, the inventories have three major flaws: (*i*) they do not account for the decline in atmospheric release of Hg from commercial products; (*ii*) they are biased in their estimate of artisanal and small-scale gold mining emissions; and (*iii*) they do not properly account for the change in Hg^0/Hg^{II} speciation of emissions from coal-fired utilities after implementation of emission controls targeted at SO_2 and NO_x. We construct an improved global emission inventory for the period 1990 to 2010 accounting for the above factors and find a 20% decrease in total Hg emissions and a 30% decrease in anthropogenic Hg^0 emissions, with much larger decreases in North America and Europe offsetting the effect of increasing emissions in Asia. Implementation of our inventory in a global 3D atmospheric Hg simulation [GEOS-Chem (Goddard Earth Observing System-Chemistry)] coupled to land and ocean reservoirs reproduces the observed large-scale trends in atmospheric Hg^0 concentrations and in Hg^{II} wet deposition. The large trends observed in North America and Europe reflect the phase-out of Hg from commercial products as well as the cobenefit from SO_2 and NO_x emission controls on coal-fired utilities.

mercury | trend | emission | atmosphere

Mercury (Hg) is released to the atmosphere by human activities including coal combustion, mining, and manufacturing and discard of commercial products (1, 2). Hg is transported globally as elemental Hg (Hg^0) in the atmosphere, eventually oxidizing to divalent Hg (Hg^{II}) that deposits to the surface, accumulates in ecosystems, and endangers humans and wildlife when converted to the neurotoxin methylmercury (3, 4). Surface air Hg concentrations in the northern hemisphere declined by 30–40% between 1990 and 2010 (5–7), and similar decreases have been observed in Hg wet deposition fluxes across North America and Western Europe (8, 9). By contrast, global inventories suggest flat or increasing Hg emissions over the last two decades (1, 10). Decreasing reemission of Hg from oceans and soils has been speculated (5, 6). Here we show that the declining atmospheric concentrations can be explained by the phase-out of Hg from commercial products and by shifts in the speciation of Hg emissions driven by air pollution control technologies.

Observed Atmospheric Hg Trends Since 1990

Table 1 compiles observed 1990-to-present trends in atmospheric Hg^0 concentrations and Hg^{II} wet deposition fluxes worldwide, including our own analyses. A general decline in the concentration of Hg^0 is observed at surface sites, continuing to the most recent years. Decreases in atmospheric Hg^0 concentrations range from 1.2 to 2.1% y^{-1} at northern midlatitudes. Trends are weaker and less significant at high northern latitudes above 60° N (−0.9 to +0.1% y^{-1}). Preliminary data from an urban and a remote site in China suggest an increasing trend of about +2% y^{-1} over the last decade (17). Wet deposition trends (available only for North America and Western Europe) are similar to trends in atmospheric concentrations.

Observed Hg^0 concentrations in the free troposphere above 2-km altitude show less significant declines. CARIBIC (Civil Aircraft for the Regular Investigation of the atmosphere Based on an Instrument Container) measurements in the northern hemisphere on commercial aircraft over the past decade (www.caribic-atmospheric.com) indicate a weak decline ($-0.6 \pm 0.6\%$ y^{-1}) that is not statistically significant ($P > 0.05$). Data from Mauna Loa, Hawaii (3.4 km above sea level), similarly indicate a statistically insignificant decline of $-0.9 \pm 0.6\%$ y^{-1}. We expect these free tropospheric trends to be representative of the tropospheric background, implying trends observed at surface sites are more influenced by regional sources and thus biased for global trend evaluation.

Significance

Anthropogenic mercury poses risks to humans and ecosystems when converted to methylmercury. A longstanding conundrum has been the apparent disconnect between increasing global emissions trends and measured declines in atmospheric mercury in North America and Europe. This work shows that locally deposited mercury close to coal-fired utilities has declined more rapidly than previously anticipated because of shifts in speciation from air pollution control technology targeted at SO_2 and NO_x. Reduced emissions from utilities over the past two decades and the phase-out of mercury in many commercial products has led to lower global anthropogenic emissions and associated deposition to ecosystems. This implies that prior policy assessments underestimated the regional benefits of declines in mercury emissions from coal-fired utilities.

Author contributions: Y.Z., D.J.J., D.P.K., and E.M.S. designed research; Y.Z. performed research; H.M.H., F.S., and V.L.S.L. contributed new reagents/analytic tools; Y.Z., H.M.H., L.C., H.M.A., and F.S. analyzed data; and Y.Z., D.J.J., and E.M.S. wrote the paper.

The authors declare no conflict of interest.

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Data deposition: The GEOS-Chem source code and run directory are available to download at geos-chem.org. The emission inventory and observational data are available at bgc.seas.harvard.edu.

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Table 1. Observed 1990 to present trends in atmospheric Hg^0 concentrations and Hg^{II} wet deposition fluxes since the 1990s

Period	Location (network)	Trend, % y^{-1}[a]	Source
Atmospheric Hg^0 concentrations			
1995 to 2010	Canada (CAMNet)	-1.6 ± 0.8[b,c]	Ref. 7
1996 to 2004	Cape Point, South Africa	-1.3 ± 0.3[c,d]	Ref. 11
1990 to 1996	Wank, Germany	-6.1 ± 1.1[c,d]	Ref. 12
1996 to 2013	Mace Head, Ireland	-1.3 ± 0.2[c,e]	Ref. 13
1990 to 2009	North Atlantic, cruises	-2.5 ± 0.5[c,d]	Ref. 6
	South Atlantic	Not significant	
2000 to 2009	Alert, Canada	-0.9 ± 0.5[f,g]	Ref. 28
	Zeppelin, Norway	Not significant	
2008 to 2013	United States (AMNet)	Not significant[c,e]	This study
2005 to 2013	Experimental Lakes Area, Canada	-2.2 ± 0.6[c,e]	This study
1990 to 2011	Western Europe (EMEP)	-2.1 ± 0.5[c,e]	This study
1994 to 2012	North of 60° N	Not significant[c,e]	This study
2005 to 2014	Free troposphere (CARIBIC)	Not significant[c,e,h]	This study
2002 to 2013	Mauna Loa Observatory, Hawaii	Not significant[c,d]	Ref. 14
Hg^{II} wet deposition			
1996 to 2008	North America (MDN)	Not significant[c,i]	Ref. 9
	Western Europe (EMEP)	-1.5 ± 0.5	
1998 to 2005	Northeast United States (MDN)	-1.7 ± 0.5[c,j]	Ref. 15
	Midwest United States	-3.5 ± 0.7	
	Southeast United States	Not significant	
1996 to 2005	Northeast United States (MDN)	-2.1 ± 0.9[g,k,l]	Ref. 8
	Midwest United States	-1.8 ± 0.3	
	Southeast United States	-1.3 ± 0.3	
	West United States	-1.4 ± 0.4	
2002 to 2008	Northeast United States (MDN)	Not significant[g,l]	Ref. 16
	Midwest United States	Not significant	
2004 to 2010	Northeast United States (MDN)	-4.1 ± 0.5[c,m]	Ref. 26
	Midwest United States	-2.7 ± 0.7	
	Southeast United States	Not significant	
	Western United States	Not significant	
1996 to 2013	North America, MDN	-1.6 ± 0.3[c,j,n]	This study
1990 to 2012	Western Europe, EMEP	-2.2 ± 0.6	

[a]All trends reported are statistically significant ($P < 0.05$). Data from multiple sources are not synchronous; thus we present snapshots of changes over multiple years and regions where data are available.
[b]Trends were calculated for sites with >5 y of measurements by using monthly median concentrations with the requirement that 75% of the month had valid data.
[c]Based on linear regression.
[d]Annual median concentrations.
[e]Monthly median concentrations.
[f]Daily averaged concentrations.
[g]Seasonal Kendall Test and Sen's slope method.
[h]Stratospheric data and biomass burning plumes filtered based on potential vorticity and CO and ozone concentrations (details provided in methods).
[i]Trend were calculated for monthly mean wet deposition fluxes of sites with at least 75% data coverage.
[j]Annual wet deposition fluxes are used.
[k]Trends are calculated for sites with >5 y of data and >75% valid data coverage.
[l]Weekly wet deposition fluxes were used.
[m]Trends are calculated for sites with >75% data coverage. Monthly precipitation volume-weighted mean concentrations were used.
[n]Sites with at least 10 and 7-y data coverage are selected for MDN and EMEP networks, respectively.

Revised Inventory of Hg Emissions

Standard Hg emission inventories used in atmospheric models (1, 10) indicate flat or increasing trends since 1990, seemingly inconsistent with the observed decreases. Horowitz et al. (2) developed an emission inventory that includes a very large missing source from the atmospheric release of Hg in commercial products. The authors showed that this commercial Hg source peaked in 1970 and has been declining rapidly since, driving an overall global decrease in Hg release to the atmosphere over the 1970-to-2000 period. The authors' inventory still shows an uptick in Hg emissions between 2000 and 2010 attributable to Asian coal-fired utilities and to artisanal and small-scale gold mining (ASGM) in developing countries (1, 19). Recent work suggests weaker growth in Chinese emissions than previously estimated because of improved data on new coal-fired utilities with flue gas desulfurization (FGD) (20, 21). Also, the increasing trend in ASGM emissions appears to be a spurious effect of improved reporting (9).

Here, we revise Hg emissions and speciation from coal combustion to account for FGD and other emission controls in North America, Europe, Japan, and China. In North America and Europe, coal-fired utilities are the largest remaining atmospheric Hg source (1, 10). Combustion releases both Hg^0, which has a relatively long atmospheric lifetime and is transported globally, and Hg^{II}, which is more likely to deposit regionally. US emissions from coal combustion declined by 75% over 2005 to 2015, mainly

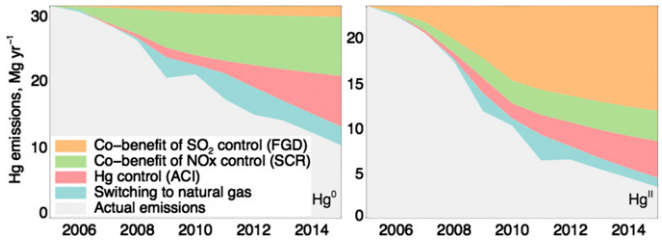

Fig. 1. Major factors driving declines in Hg emission from US coal-fired utilities between 2005 and 2015. Trends were inferred from data on the implementation of different types of emission control technologies.

because of cobenefits from controlling other atmospheric pollutants (Fig. 1). This decrease would be accompanied by a change in the Hg^0/Hg^{II} speciation of emissions that is not recognized in current inventories. Use of FGD to control sulfur dioxide (SO_2) emissions washes out Hg^{II} (22). Use of selective catalytic reduction (SCR) to control nitrogen oxide (NO_x) emissions also oxidizes Hg^0 to Hg^{II}, and application of SCR and FGD in series controls total Hg emissions (23). Activated carbon injection (ACI) to specifically target Hg emissions has also begun to penetrate the energy sector (24). By considering the installation capacity and control efficiency of these devices, we find FGD caused the fraction of total Hg released as Hg^{II} to decline disproportionately to total emissions, from 43% to 24% over the last decade (Fig. 1).

Similar changes in Hg emissions from coal combustion can be inferred for other countries that have implemented air pollution controls and fuel switches over the last two decades. The fraction of coal-fired utilities with FGD increased from 20% to 46% between 1990 to 2002 in Western Europe and from 30% to 70% between 1990 and 2005 in Japan, resulting in a total cumulative decrease of 200 Mg Hg^{II} from developed countries including the US (25, 26). In China, the fraction of coal-fired utilities with FGD capacity increased from zero in 2000 to 86% in 2010, resulting in a drop in annual Hg^{II} emissions of 30% and 250 Mg cumulatively between 1990 and 2010 (21). The growth in energy demand in China has led to a rapid increase in coal combustion (11% y^{-1}), but total Hg emissions over this period increased less (5.8% y^{-1}) because of the implementation of FGD (21). This previously unaccounted shift in speciation implies greater declines in near-field Hg deposition than previously estimated.

Table 2 summarizes our updated global inventory of anthropogenic emissions for 1990 to 2010. The inventory includes revised estimates of emissions from coal combustion as described above, ASGM emissions from Muntean et al. (9), and emissions from commercial products (incineration, volatilization) based on Horowitz et al. (2) (additional details are available in Table S1). Our results indicate a 30% global decline of anthropogenic Hg^0 emissions from 1990 to 2010 (−1.5% y^{-1}). These declines are steepest from 1990 to 2000 but continue through 2010. This contrasts the flat or increasing trends in previous inventories (1, 9, 10). Two-thirds of the decline reflects the phase out of Hg in commercial products. Horowitz et al. (2) previously found that this was offset by rising emissions from coal combustion and ASGM, but our revision to the combustion inventory, as well as the Muntean et al. (9) ASGM inventory, removes the offset.

We find a global increase of 9% in Hg^{II} emissions between 1990 and 2010. This increase is attributable to growth in coal combustion in India and China, with FGD mitigating part of the increase in China. By contrast, Streets et al. estimated a 48% global increase in Hg^{II} emission between 1990 and 2008 (1). The authors did not account for growing implementation of FGD in China because of the lack of necessary information.

Large regional differences in Hg emission trends are apparent from Table 2. The decline in emissions from commercial products has been concentrated in developed countries (2). Total Hg emissions declined by a factor of 6.3 in Western Europe, 3.8 in North America, and 2.0 for other regions of Europe over 1990 to 2010, but emissions in Asia increased by a factor of 1.5.

Consistency with Observed Atmospheric Trends

Fig. 2 shows the 1990-to-2010 trends in atmospheric Hg^0 concentrations and Hg^{II} wet deposition fluxes simulated by the GEOS-Chem (Goddard Earth Observing System-Chemistry) global model using our revised anthropogenic emission inventory and the same meteorological year (to isolate the effect of emissions). Observed trends from Table 1 are also shown. Fig. 3 shows the simulated and observed regional trends averaged across the sites of Table 1.

The model successfully reproduces the observed trends. Declines are largest in North America and Western Europe (−1.5 ± 0.18 to −2.2 ± 0.15% y^{-1}), reflecting the particularly large emission decreases in these regions. Shifts in speciation from coal-fired power plants also contribute significantly to the observed decline in wet deposition fluxes. Neglecting this change would result in an underestimate of the trend by a factor of 2 (e.g., ref. 26). The model decline in the free troposphere of the northern hemisphere (−0.6 ± 0.037% y^{-1}) reflects a global decrease in total Hg^0 emission (including anthropogenic, natural, and reemission sources) of −0.5% y^{-1}. This decreasing trend is lower than that of anthropogenic Hg^0 emissions (−1.5% y^{-1}) because natural and legacy sources are approximately twice the magnitude of anthropogenic sources and are relatively unchanged. This modeled trend is in the range of observations from CARIBIC (−0.6 ± 0.57% y^{-1}) and Mauna Loa Observatory (−0.9 ± 0.57% y^{-1}). Model increases are limited to East Asia, consistent with preliminary observations and previous modeling studies (6, 11, 27).

The model decline at northern high latitudes (including one site in North American sector and four in Western Europe) is −1.3 ± 0.11% y^{-1}, typical of the northern extratropical background (Fig. 2*A*), but observations show a much weaker and statistically insignificant decline (−0.2 ± 0.5% y^{-1}) (28). A similar discrepancy is observed for wet deposition fluxes over high-latitude regions of Western Europe (Fig. 2*C*). Fisher and coworkers (29, 30) and Zhang et al. (31) previously showed that trends in the Arctic are complicated by influences from riverine Hg discharges and sea ice cover. A GEOS-Chem simulation by Chen et al. (27), which includes long-term warming temperature and shrinking of Arctic sea ice, indicates that decreased oxidation of Hg^0 and deposition from the atmosphere as well as increased evasion of Hg^0 from the Arctic Ocean offsets the effect of the declining atmospheric background, resulting in no significant trend at high latitudes consistent with observations.

There is substantial uncertainty in current anthropogenic Hg emissions estimates (1, 9, 10, 19). Similar to Streets et al. (1), we calculated lower and upper bounds around the central estimate (Table 2 and Table S1) that correspond to an 80% confidence interval (CI) (the probability of emissions being outside this range is less than 20%). We estimate the resulting uncertainty in emissions is −33% to +60%. However, the calculated emissions trend between 1990 to 2010 is much more consistent, ranging between −1.4% to −0.53% y^{-1} and the propagated uncertainty in the simulated atmospheric trend across years is relatively small (±20%; Fig. 3).

Our results show general agreement between modeled and observed trends on the continental scale (Fig. 3), but the model does not reproduce all fine-scale variability in observations. For example, the model underestimates the atmospheric Hg^0 trend for one site in the western United States and cannot capture the observed increases in Hg^{II} wet deposition near the Four Corners Region in Colorado. These discrepancies are largely caused by

Table 2. Anthropogenic Hg emissions by world region

Region and emissions	Year of emissions		
	1990, Mg y^{-1}	2000, Mg y^{-1}	2010, Mg y^{-1}
Western Europe			
Hg^0	410	121	61
ASGM	0	0	0
Products	212	77	18
Combustion	198	44	44
Hg^{II}	73	22	15
Total	483 (382–498)	142 (106–228)	77 (57–129)
North America			
Hg^0	399	174	109
ASGM	0	0	0
Products	208	88	66
Combustion	189	85	42
Hg^{II}	70	42	15
Total	469 (361–635)	216 (167–295)	124 (93–175)
Asia and Oceania			
Hg^0	733	812	989
ASGM	81	181	243
Products	288	325	281
Combustion	363	306	465
Hg^{II}	326	358	575
Total	1,060 (774–1,590)	1,170 (806–1,860)	1,560 (1,040–2,530)
Other Europe			
Hg^0	234	126	98
ASGM	7	11	13
Products	42	29	11
Combustion	185	86	75
Hg^{II}	171	101	102
Total	405 (318–658)	227 (173–385)	200 (146–345)
Africa			
Hg^0	166	136	68
ASGM	78	83	28
Products	15	6	0
Combustion	73	46	40
Hg^{II}	67	55	55
Total	233 (151–395)	190 (114–335)	123 (82–216)
Central and South America			
Hg^0	208	174	149
ASGM	110	90	93
Products	64	51	23
Combustion	35	34	33
Hg^{II}	32	39	45
Total	240 (137–406)	214 (126–366)	194 (108–340)
Global			
Hg^0	2,150	1,540	1,480
ASGM	278	366	378
Products	829	576	398
Combustion	1040	600	699
Hg^{II}	739	617	807
Total	2,890 (2,120–4,180)	2,160 (1,490–3,470)	2,280 (1,520–3,730)

Taken from Muntean et al. (9). Products are emissions from use and disposal of commercial products from Horowitz et al. (2). Combustion indicates other and includes coal combustion, cement production, and metal smelting from Streets et al. (1). North America includes the United States and Canada. Mexico is included in Central and South America. Numbers in parenthesis are the 80% CIs.

local emission changes and meteorological effects (26), which are missed by the coarser-resolution simulations used here. More detailed, high-resolution emission inventories and models are required to fully resolve such fine-scale variability in observations.

The influence of changing climate and other environmental factors on the reemissions of Hg from soil and ocean remains unclear (32). The concentrations of major oxidants for atmospheric Hg^0, including OH, O_3, and Br, have remained relatively steady or slightly decreased since the mid-1990s and are thus not an important driver for the observed decline (5). Decreasing riverine discharges, which was previously speculated to drive the decline in North Atlantic Ocean Hg concentration and subsequent reemission flux (6), are also insufficient for forcing the global atmospheric trend (31, 33).

Our work has shown that revising anthropogenic emissions with the most up-to-date information can explain the observed

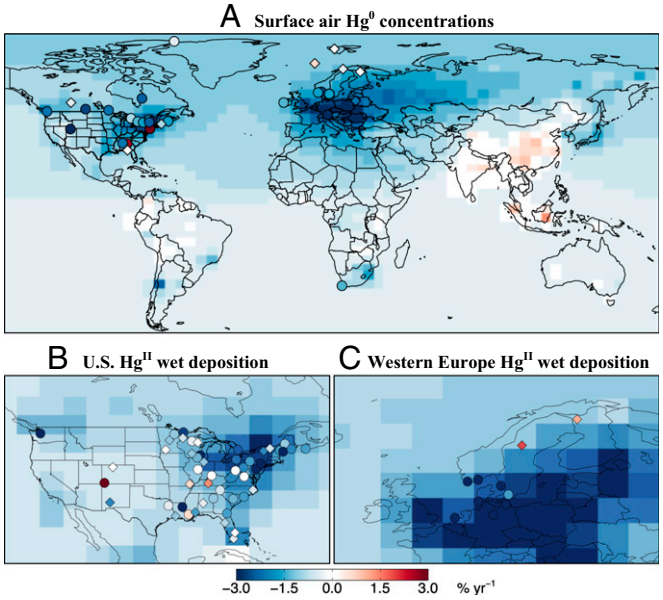

Fig. 2. Trends in atmospheric Hg^0 concentrations (*A*) and Hg^{II} wet deposition fluxes (*B* and *C*) from 1990 to present. Observations from the sites in Table 1 are shown as circles (if trends are statistically significant, $P < 0.05$) and diamonds (not significant). The background shows the trends computed in the GEOS-Chem model driven by our revised 1990 and 2010 anthropogenic emissions inventories from Table 2.

large-scale decline in atmospheric Hg over the past two decades. This finding reinforces the major benefits that have been derived from the phase-out of Hg in many products and emission controls on coal combustion.

Methods

Atmospheric Observations in 1990 to 2010. We include long-term observations (typically >5 y) for atmospheric Hg^0 concentrations and Hg^{II} wet deposition flux at sites worldwide during 1990 to 2014 (Table 1). For all of the measurements, an ordinary linear regression on the annual means is used to calculate the trend. Because gaseous phase Hg^{II} accounts for less than 1–2% of TGM concentrations in surface air, we do not differentiate between Hg^0 and TGM in ground observations (34). We do not include atmospheric Hg^{II} concentrations because long-term records are few and data quality is uncertain (35).

Atmospheric Hg concentration data are available through the Canadian Atmospheric Mercury Network (CAMNet) (eight sites; https://www.ec.gc.ca/natchem), the US Atmospheric Mercury Network (AMNet) (nine sites; nadp.sws.uiuc.edu/amn), and European Monitoring and Evaluation Program (EMEP) (six sites; www.emep.int) networks. We include an analysis of the Hg^0 data measured at a remote and forested site, Experimental Lake Area, Canada (47.9° N, 93.7° W), during 2005 to 2013. Hg^{II} wet deposition fluxes are measured by the Mercury Deposition Network (MDN) (nadp.sws.uiuc.edu/mdn) and the EMEP network over North America and Western Europe, respectively. We select 52 MDN sites with at least 10-y data coverage during 1996 to 2013 and 11 EMEP sites with at least 7-y data since 1990. Atmospheric Hg concentrations have been measured from commercial aircraft by the CARIBIC project since December 2004 (www.caribic-atmospheric.com). We exclude stratospheric data using potential vorticity (European Centre for Medium-Range Weather Forecasts: www.ecmwf.int) and O_3 concentrations (36, 37). We exclude biomass burning plumes if the measured Hg concentration is greater than 2.5 ng m^{-3}. Also included here are the Hg^0 data measured at Mauna Loa Observatory, Hawaii (19.5° N, 155.6° W), during 2002 to 2013, which also samples the free troposphere at an elevation of 3,400 m.

Updated 1990 to 2010 Anthropogenic Emissions. We track the installation of air pollution control devices (FGD, SCR, and ACI) for individual US coal-fired utilities and calculate the associated Hg emission decline and speciation change based on the measured capture efficiencies with different coal types and control device configurations. Fuel type information is obtained from air markets program data by the US Environmental Protection Agency (EPA) (ampd.epa.gov/ampd). The installation time and type of air pollution control devices during 2005 to 2011 are from Mercury and Air Toxics Standards (MATS) Information Collection Request 2011 (www3.epa.gov/ttn/atw/utility/utilitypg.html), with linear extrapolation to 2015 except for ACI, which is from the Institute of Clean Air Companies (www.icac.com). Hg capture efficiencies for different coal types and configuration of control devices are based on US EPA measurements (22). This speciation change over the US is extrapolated to all other developed countries in North America, Western Europe, and Oceania based on their similar trajectory of control technology (24). For China, we follow the change of speciation derived by Zhang et al. (20).

We develop an integrated emission inventory between 1990 and 2010 with decadal resolution, including improved estimates of Hg^{II} and Hg^0 speciation

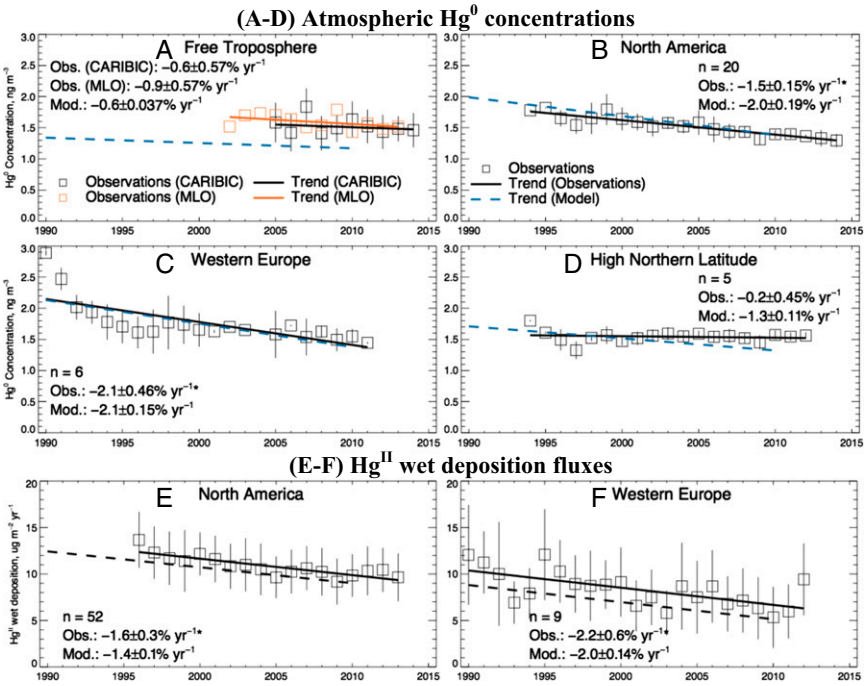

Fig. 3. Regional trends for 1990 to 2013 in atmospheric Hg^0 concentrations (*A–D*) and Hg^{II} wet deposition (*E* and *F*). Observations for individual years are shown as squares with linear regression as solid line. The dashed line is the trend from the GEOS-Chem simulation using our revised anthropogenic emissions inventory for 1990 and 2010 (Table 2). The data are averaged regionally across the sites in Table 1 for the free troposphere (*A*), North America (*B* and *E*), Western Europe (*C* and *F*), and high northern latitude regions (*D*) (vertical bars show the SDs). Regression coefficients (slope ± SE) and number of sites (*n*) are given (*Insets*). The SE of modeled trend is calculated based on the uncertainty range of the emission inventory (Table 2). North American atmospheric Hg^0 concentrations are from the CAMNet (https://www.ec.gc.ca/natchem) and AMNet (nadp.sws.uiuc.edu/amn) networks and the Experimental Lakes Area, Canada. North American Hg^{II} wet deposition is from the MDN (nadp.sws.uiuc.edu/mdn). Observations in Western Europe are from the EMEP network (www.emep.int). High-latitude sites include Alert, Canada and Zeppelin, Norway, and three sites above 60° N from the EMEP network.

from coal-fired utilities as described above. Default emissions are based on those of Streets et al. (1), who provide byproduct emissions from fossil fuel combustion, metal smelting, and waste incineration for 17 world regions. This inventory is updated using more recent country-specific estimates for China in 1995 to 2010 (20, 38), India in 2001 to 2020 (39), the US in 1990 to 2011 [National Emission Inventory (NEI) inventory: www3.epa.gov/ttnchie1/trends], and Western Europe in 1990 (40). We use global atmospheric releases from the use and disposal of commercial products from Horowitz et al. (2) and distribute to different regions based on Hg consumption (10, 18). Regional total emissions from these inventories are distributed on a $1° \times 1°$ grid following the spatial pattern of the Global Emissions InitiAtive (GEIA) inventory (41). For ASGM, we use the Emissions Database for Global Atmospheric Research gridded inventory (9).

Modeled Atmospheric Hg Concentrations and Deposition. The GEOS-Chem Hg model (version 9-01-02) is used to calculate the atmospheric Hg trends driven by Hg emission changes. The model includes a 3D global atmosphere coupled to 2D slab ocean and land models. A detailed description and evaluation of this model is available in Holmes et al. (42), Amos et al. (43), and Zhang et al. (44). The horizontal resolution is 4° latitude × 5° longitude, with 47 vertical layers extending to the mesosphere. Atmospheric transport is driven by assimilated meteorological data from the GEOS-5 of the NASA Global Modeling and Assimilation Office.

The model traces two species: elemental mercury (Hg^0) and divalent mercury (Hg^{II}). Hg^{II} is partitioned thermodynamically between the gas and particle phase on the basis of local temperatures and total aerosol concentration computed with a GEOS-Chem aerosol simulation (43). The model includes the oxidation of Hg^0 by atomic bromine and the photochemical reduction of Hg^{II} in cloud droplets. We do not include the fast reduction of Hg^{II} in coal-fired power plant plumes previously introduced by Zhang et al. (44) and Amos et al. (43) because more recent studies find that the reduction rate is slower than previously estimated (0–55% of emitted Hg^{II} is reduced with a mean value of 4.9%) (45, 46). Redox chemistry also takes place in the surface ocean and soil reservoirs, which receive atmospheric deposition and reemit to atmosphere through land–air and sea–air exchanges.

We conduct 3-y simulations with different anthropogenic emissions corresponding to years 1990 and 2010 (Table 2). The first 2 y are used for initialization, and the third year is used for analysis. The same meteorological year (2008) is used for all simulations to remove the influence of interannual meteorological variability. The soil and subsurface ocean concentrations are kept constant to isolate the impact of changing atmospheric emissions in the recent two decades.

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EXHIBIT 5

To Comments of Public Health and Environmental Groups
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Benefits of mercury controls for the United States

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Mercury pollution poses risks for both human and ecosystem health. As a consequence, controlling mercury pollution has become a policy goal on both global and national scales. We developed an assessment method linking global-scale atmospheric chemical transport modeling to regional-scale economic modeling to consistently evaluate the potential benefits to the United States of global (UN Minamata Convention on Mercury) and domestic [Mercury and Air Toxics Standards (MATS)] policies, framed as economic gains from avoiding mercury-related adverse health endpoints. This method attempts to trace the policies-to-impacts path while taking into account uncertainties and knowledge gaps with policy-appropriate bounding assumptions. We project that cumulative lifetime benefits from the Minamata Convention for individuals affected by 2050 are $339 billion (2005 USD), with a range from $1.4 billion to $575 billion in our sensitivity scenarios. Cumulative economy-wide benefits to the United States, realized by 2050, are $104 billion, with a range from $6 million to $171 billion. Projected Minamata benefits are more than twice those projected from the domestic policy. This relative benefit is robust to several uncertainties and variabilities, with the ratio of benefits (Minamata/MATS) ranging from ≈1.4 to 3. However, we find that for those consuming locally caught freshwater fish from the United States, rather than marine and estuarine fish from the global market, benefits are larger from US than global action, suggesting domestic policies are important for protecting these populations. Per megagram of prevented emissions, our domestic policy scenario results in US benefits about an order of magnitude higher than from our global scenario, further highlighting the importance of domestic action.

mercury | policy | impacts assessment | Minamata Convention | economic benefits

Toxic contamination from human activities is a global problem. Although some countries have regulated toxic substances such as heavy metals and persistent organic pollutants for several decades, chemical contamination has still been identified as a key planetary boundary at risk for exceedance in the context of global change (1). To address this challenge, existing global environmental treaties try to manage the entire life cycle of chemical contaminants (2). The newest of these is a global treaty on mercury, the Minamata Convention. In November 2013, the United States became the first country to fulfill the requirements necessary to become a party to the convention.

In the United States, analyses to support domestic environmental decision-making include socioeconomic valuations of impacts as part of the regulatory process. However, these evaluations can be both scientifically challenging and politically contentious, particularly given uncertainties and knowledge gaps (as noted in arguments in a recent case heard in the US Supreme Court, *Michigan v. Environmental Protection Agency, 2015*, addressing analysis of the costs and benefits of mercury regulation). These challenges are especially difficult for contaminants such as mercury, which cross temporal and spatial scales and have both domestic and global sources. The chain of analysis from policies, through emissions, to impacts involves a complex pathway, which for mercury includes industrial activities, atmospheric chemistry, deposition processes, bioaccumulation, and human exposure. Existing approaches have not fully combined information and knowledge from these disparate fields, and substantial gaps exist in scientific understanding of the processes that mercury undergoes through long-range transport. Thus, it has historically been difficult to quantitatively estimate prospective domestic benefits from global environmental treaty-making in ways that can be compared with socioeconomic analyses designed to support domestic environmental decision-making. Here, we use an assessment approach that enables tracing this pathway, accounting for best-available scientific understanding and addressing uncertainties and knowledge gaps with policy-appropriate assumptions.

Mercury is a naturally occurring element, but human activities such as mining and coal combustion have mobilized additional amounts, enhancing the amount of mercury circulating in the atmosphere and surface oceans by a factor of three or more (3, 4). Mercury previously deposited to land and water can revolatilize over decades to centuries. Thus, human activities have fundamentally altered the global biogeochemical cycle of mercury (5). Deposited mercury in aquatic systems can be converted to more toxic methylmercury (MeHg), which bioaccumulates. People are then exposed to MeHg by eating contaminated fish. Effects of MeHg exposure include IQ deficits in prenatally exposed children (6–8) and may include cardiovascular effects in adults (7, 9). Scientific uncertainty and variability are substantial throughout this pathway, including but not limited to atmospheric chemistry, deposition patterns, methylation processes, bioaccumulation and food web dynamics, dietary patterns of exposure, and dose–response relationships. Despite these uncertainties, scientific analyses have been conducted to support decision-making, and state-of-the-art models exist for many of these steps.

Some studies have previously traced the pathway from mercury emissions to human impacts. These studies are limited in how completely they have represented physical processes, and how they have accounted for knowledge gaps. First, many do not explicitly consider spatial transport through the environment on a global scale, and so do not explicitly link emissions to exposure changes (10–14). Timescales associated with bioaccumulation through ecosystems also are often not taken into account, making

Significance

Mercury is a globally transported pollutant with potent neurotoxic effects for both humans and wildlife. This study introduces an assessment method to estimate the potential human health-related economic benefits of global and domestic mercury control policies. It finds that for the US population as a whole, global mercury controls could lead to approximately twice the benefits of domestic action by 2050. This result is robust to several uncertainties and variabilities along the emissions-to-impacts path, although we find that those consuming locally caught freshwater fish in the United States could benefit more from domestic action.

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it difficult to evaluate how the timing of emissions changes affects benefits (15). Few studies have explicitly included more uncertain, but potentially important, health endpoints such as cardiovascular effects in their estimates (12, 16). For instance, the US Environmental Protection Agency (15) focused on only IQ-related MeHg effects in their analysis of the Mercury and Air Toxics Standards (MATS) in the United States. Finally, methods used for previous studies were not designed to highlight the relative importance of uncertainties throughout the policies-to-impacts path.

We explicitly incorporate uncertainty and sensitivity analysis for key steps along the policies-to-impacts pathway to assess the relative importance of policy-relevant uncertainties. We combine best available models to trace projected global mercury policy scenarios to their US impacts. We use atmospheric modeling to project the amount of mercury depositing to the US and global seafood source regions with and without global policy. We incorporate assessment of timescales associated with bioaccumulation through ecosystems. We then link atmospheric mercury models to economic valuation models, generating a representation of mercury impacts that takes into account environmental and human response timescales. We use this assessment approach to present what is, to our knowledge, a first assessment of potential US benefits, defined in economic terms, from the Minamata Convention. We explicitly compare benefits of global and US policies, using consistent methodology, and analyze the relative impacts of these policies on the US population. We first present results from a base case analysis of mercury policy to 2050, using our integrated model. We then present our sensitivity analyses, assessing the influence of uncertainties on our base case results.

Results and Discussion

Tracking the Policies-to-Impacts Pathway: Base Case. Globally, our emissions projections under the Minamata Convention will result in 2050 in emissions of 1,870 $Mg{\cdot}y^{-1}$, which is roughly equivalent to the present-day level, but 2,270 $Mg{\cdot}y^{-1}$ less than our no policy (NP) scenario (17). The largest sources of anthropogenic mercury emission are stationary coal combustion, artisanal and small-scale gold mining, and metals production (18). Under NP, emissions are projected to more than double, largely as a result of growth in coal use in Asia (19); thus, the main differences in policy and NP projections depend on assumptions about emission controls for coal (20). Air quality abatement technologies such as flue gas desulfurization can capture mercury as a cobenefit. For global emission projections under the Minamata Convention, which requires the application of best available technologies, taking into account technical and economic feasibility, we assume the application of flue gas desulfurization or similar technology outside of the United States (17, 19). In the United States, our policy scenario is based on MATS (currently under legal challenge), which was designed to control Hg emissions from power generation, with full implementation by 2016 (15). In the United States, emissions in 2005 were ~90 $Mg{\cdot}y^{-1}$ (15). Under our MATS projection, we extend the US Environmental Protection Agency projected trend from 2016 to 2020 (15, 21) linearly, resulting in 2050 US emissions of 46 $Mg{\cdot}y^{-1}$. Our NP case for the United States includes no further improvements in emissions control technology or policy, and thus results in an approximate doubling of 2005 emissions by 2050 (19). Benefits of the Minamata Convention to the United States are calculated as the difference between the global Minamata and NP scenarios, holding US emissions constant at the MATS scenario. Benefits of MATS to the US are calculated as the difference between the US NP and MATS scenarios, holding emissions in the rest of the world constant at the NP scenario.

Under our Minamata case, mercury deposition to the United States and to the global oceans are 19% and 57% less than under NP in 2050, respectively. Fig. 1 maps these deposition differences over the contiguous United States. We model the atmospheric transport and deposition of mercury using the global, 3D land-ocean-atmosphere mercury model GEOS-Chem v.9-02, at 4° × 5° resolution globally and 0.5° × 0.667° resolution over the United States (22–26). We use net total deposition as a measure of mercury ecosystem enrichment (27). *SI Appendix, Chemical transport modeling* gives additional details on the modeling approaches. For our MATS case, deposition to the United States is 20% less than under NP, and deposition to the global oceans is 6% less. Although the modeled avoided deposition over the entire United States is similar under MATS and Minamata, the distribution of these differences varies, as shown in Fig. 1. Avoided deposition under MATS is more highly concentrated in the Northeast, where there are significant coal-fired emission sources. In contrast, US deposition benefits under the Minamata Convention follow precipitation patterns, as policy avoids increases in the global background mercury concentration.

Because mercury is persistent in the environment, anthropogenic emissions also enrich reservoirs of mercury in the subsurface ocean and soils. Mercury from these pools can enhance reemissions, contributing further to deposition. Our GEOS-Chem simulations take into account the effect of anthropogenic emissions changes on concentrations of mercury in surface reservoirs only, and consequently underestimate the total deposition benefits attributable to policy. To roughly estimate the extent of this underestimation, we use a seven-box, biogeochemical model developed by Amos et al. (28, 29), which captures the deep ocean and soil reservoirs, but not the spatial distribution of impacts (*SI Appendix, Chemical transport modeling*). We find that globally, deposition reductions under policy are ~30% larger when taking into account enrichment of these subsurface pools.

Recent research suggests that fish concentrations in ocean (30–32) and freshwater (33–36) fish will likely respond proportionally to changes in atmospheric inputs over years to decades, although the magnitude and timing of a full response may be variable, depending on the region (see refs. 32 and 37–39 for examples). For our base case scenario, we assume that fish MeHg in both freshwater and marine ecosystems responds after 10 y to proportionally reflect changes in atmospheric inputs (we test the response to this assumption

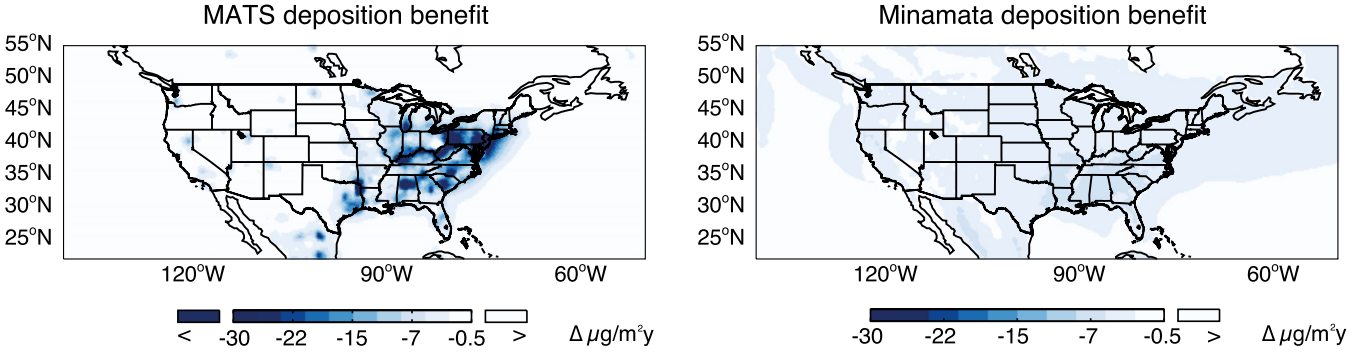

Fig. 1. Projected net deposition benefits ($\Delta\mu g/m^2{\cdot}y$) of MATS and the Minamata Convention over NP over the contiguous United States, at 0.5° × 0.667° resolution. Global results, at 4° × 5° resolution, are shown in *SI Appendix*, Fig. S4.

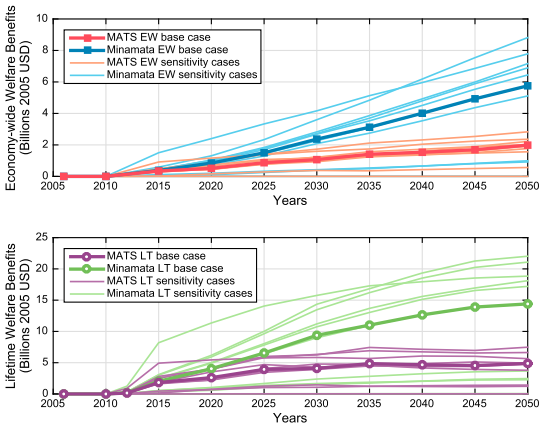

Fig. 2. Trajectories of welfare benefits under global and domestic policy until 2050, discounted at 3%. (*Top*) Modeled EW benefits realized in a given year. (*Bottom*) Projected LT benefits for that year's affected population. Base cases are indicated with markers. Unmarked lines show the range of trajectories from sensitivity cases.

in our sensitivity analysis) (30, 37). We specify base year blood MeHg, as a biomarker for MeHg exposure, by region, based on the National Health and Nutrition Examination Survey (40). We then scale blood concentrations based on the change in intake of fish MeHg (change in deposition plus time lag), taking into account consumption of domestic freshwater and imported fish species from global fisheries, using data from US seafood market studies (41) and data compiled by the US Environmental Protection Agency on noncommercial anglers (15, 42). Because of data limitations, we consider noncommercial mercury intake from local, freshwater fish only. We treat noncommercial marine anglers as average US consumers of marine and estuarine fish. This may slightly underestimate the benefits of MATS in our work; however, further data are necessary to quantify the MeHg intake of noncommercial anglers in different US coastal regions (see *SI Appendix*, *Changes in human exposure* for detailed methods).

Calculated average US mercury intake in 2050, assuming a 10-y time lag between deposition changes and fish response, as well as constant fish intake patterns, is 91% less under our Minamata scenario than under NP (*SI Appendix*, Fig. S5). Our MATS scenario reduces intake by 32% compared with the NP case. Although the deposition decreases over the United States are roughly equivalent between the MATS and Minamata scenarios, changes to modeled mercury intake are larger under the latter. More than 90% of the US commercial fish market, and the majority of US mercury intake, comes from marine and estuarine sources, particularly from Pacific and Atlantic Ocean basins (41, 43). These regions are heavily influenced by emissions from non-US sources, including East and South Asia. In addition, even locally caught freshwater fish are affected by the long-range transport of mercury emissions. Regional differences in the geographic source of dietary fish (*SI Appendix, Changes in human exposure*) and deposition lead to variations in intake change patterns across scenarios, as shown in *SI Appendix*, Fig. S4. The majority of modeled MeHg intake in the North Central region (*SI Appendix*, Fig. S5) is from self-caught, local freshwater fish, leading to a diminished intake benefit from the Minamata scenario relative to the MATS scenario. The opposite pattern holds for New York. These differences in intake lead to corresponding differences in IQ deficits and cardiovascular outcomes (see *SI Appendix*, *IQ effects; Cardiovascular impacts; and Health impacts* for health impacts methods and results, respectively).

Annual US economic benefits to 2050 (applying a 3% discount rate) from avoided health impacts under domestic and global mercury policies under our base case assumptions, relative to NP, are presented in Fig. 2. We use two economic valuation approaches: the first, a cost-of-illness and value of statistical life (VSL) approach, estimates projected lifetime (LT) benefits of avoided exposure for those born by 2050 and is consistent with US regulatory practice; the second, a human capital approach, estimates economy-wide (EW) benefits realized by 2050 from avoided labor productivity and wage losses. Given differences in methodology, results from these two approaches are not directly comparable (see *SI Appendix*, *Economic modeling of health impacts* for more details). To estimate LT benefits of avoided health effects, we apply estimates of projected lost wages and medical costs for IQ deficits and nonfatal acute myocardial infarctions (heart attacks), and VSL for premature fatalities resulting from myocardial infarctions (see ref. 12 and examples listed in ref. 44 of studies that use this approach), for each year's projected birth cohort (IQ) and affected adult population (heart attacks). The second method uses the US Regional Energy Policy model, a computable general equilibrium model of the US economy (45). Consistent with previous work valuing economic effects of air pollution through computable general equilibrium modeling (46), we take into account the effects of IQ deficits and fatal and nonfatal heart attacks on the labor force, and its cumulative effect over time. Base case cumulative EW benefits of the Minamata Convention to the United States by 2050 are $104 billion (2005 USD) (Fig. 2, *Top*, blue line), and cumulative LT benefits for those born by 2050 are $339 billion (Fig. 2, *Bottom*, green line). EW benefits from our MATS scenario (Fig. 2, *Top*, red line) are $43 billion by 2050, and LT benefits are $147 billion (Fig. 2, *Bottom*, purple line). Both EW and LT benefits are dominated (>90% for LT and >99% for EW) by avoided cardiovascular effects, consistent with previous studies, including these health endpoints (12, 16). Relative to US domestic action, estimated cumulative benefits from the Minamata Convention are more than twice as large.

Considered per unit of avoided emissions, however, the projected benefits of MATS to the United States are larger than those of the Minamata Convention: $324 million/Mg compared with $46 million/Mg for EW benefits by 2050, and $1.1 billion/Mg compared with $150 million/Mg for LT benefits for those born by 2050. Given its global scope, the Minamata Convention is likely to prevent more emissions than MATS. However, as mercury pollution has effects on both local and global scales, avoided emissions within the United States, on a per unit basis, lead to larger benefits.

Policies-to-Impacts Sensitivity Analysis. We assess uncertainty and variability along the policies-to-impacts pathway by identifying key drivers of uncertainty in our base case integrated model, and calculating how changes in assumptions affect our quantification of US benefits from the Minamata Convention, MATS, and relative benefits. Key assumptions addressed here include the effect of atmospheric chemistry, ecosystem time lags, dietary choices, dose–response parameters linking MeHg exposure and health effects, economic costs, and discount rates. We run the integrated model for realistic and policy-relevant low and high bounds for these assumptions. Fig. 2 shows the range of calculated benefits from these sensitivity scenarios, described further here. The uncertain range spanned by these cases is illustrated by the lines in Fig. 2; however, the bounds delineated by these lines for the Minamata (blue/green) and MATS (red/purple) scenarios are not independent. Some sensitivity scenarios lead to the same directional change in benefits over the base case for both the domestic and global scenario, such that the magnitude of cumulative benefits for the Minamata scenario remain larger than for MATS. This result is illustrated in Fig. 3, which shows the range in ratio of benefits between Minamata and MATS, under different sensitivity scenarios. Details of the low and high cases addressed are presented in *SI Appendix*, Table S7 and *Sensitivity analysis*.

Our low and high cases for atmospheric chemistry bound uncertainty about the form of mercury emissions and atmospheric redox reactions. Although policies address total mercury emissions, emissions of mercury occur as different chemical species with different atmospheric lifetimes. Mercury emitted in its elemental form, Hg(0), has an atmospheric lifetime of 6 mo to a year, enabling it to transport globally before its oxidation and

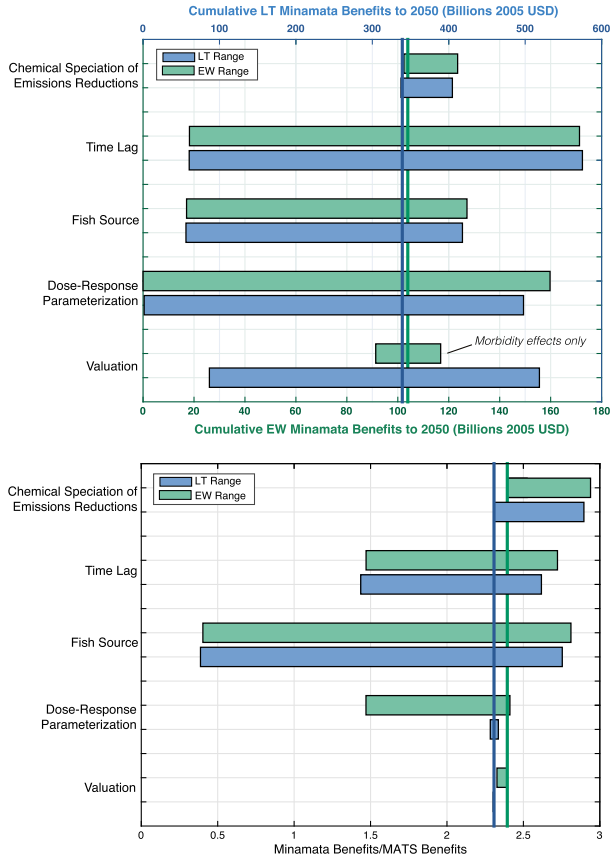

Fig. 3. (*Top*) Range in cumulative benefits of the Minamata scenario to 2050. Note the different scales for LT and EW benefits. (*Bottom*) Range in ratio of cumulative benefits to 2050 (Minamata Benefits/MATS Benefits). Blue and green lines show base case results for LT and EW benefits, respectively. Bars indicate the sensitivity of cumulative benefits to high and low case assumptions for uncertain parameters.

subsequent deposition. Mercury emitted in its oxidized form, Hg(II), in the gas phase, or Hg(P) in the particle phase, is more soluble and can deposit closer to its source. In addition, the speciation of present-day mercury emissions is uncertain. Reduction reactions may convert Hg(II) to Hg(0), lengthening its lifetime; this process may occur in the atmosphere in the aqueous phase (47), or in power plant plumes (48, 49). However, the mechanism of potential reduction is unknown. To bound this uncertainty, we assume for our low case that 90% of global Hg reductions over NP occur as Hg(II) or Hg(P), and for the high case, that 90% of reductions occur as Hg(0). This results in a range of cumulative EW benefits for Minamata between $102 billion (low) and $123 billion (high) in 2005 USD, and a range of LT benefits of $338 billion to $405 billion. That the low case results in only a small difference from the base case reflects the emphasis on control technologies that capture oxidized mercury in the base case assumptions (19). The relative benefits of Minamata versus our MATS case vary to a factor of 2.9 from the base case. If policy prevents primarily Hg(0) emissions, or there is a high rate of in-plume reduction, there is greater long-range benefit to the United States and global oceans from avoided emissions occurring elsewhere.

If fish MeHg responds rapidly and quantitatively to changes in deposition, cumulative EW and LT benefits to 2050 from Minamata are projected to be $171 billion and $575 billion (2005 USD), whereas a slower response reduces projected EW and LT benefits to $18 billion and $60 billion. Although reductions in mercury deposition, all else equal, will eventually result in decreased environmental and fish concentrations, benefits within a given time horizon, which in this case is 2050, will depend on how long ecosystems take to respond. Estimated economic benefits are therefore highly sensitive to the temporal scope of analysis. For instance, EW benefits from IQ effects are primarily accrued when those in birth cohorts with reduced exposure are of working age (see *SI Appendix*, *Economic modeling of health impacts*), and consequently are not fully captured by our 2050 time horizon. Population growth and discounting assumptions (we use a 3% discount rate; see *SI Appendix*, *Economic valuation* for others) also influence our cumulative benefit assessment. Timing effects are further discussed in *SI Appendix*, *Economic valuation*. Our lower bound incorporates an instantaneous response, which is the assumption commonly used in regulatory analyses (15, 42), and that may be roughly consistent with the behavior of certain classes of freshwater bodies (37). Our upper bound is 50 y, consistent with the high range of estimated response times for surface open ocean waters (30), where MeHg production and biomagnification are hypothesized to occur (31), and midrange estimates for watershed-fed coastal ecosystems and some lake systems, which may be the slowest to respond to changes in atmospheric deposition (32, 36).

Population dietary choice between local freshwater and global market fish alters our Minamata base case cumulative EW benefits from $17 billion (2005 USD) to $127 billion, and cumulative LT benefits from $56 billion to $418 billion. Our base case assumes that population dietary choices between local fish and global market fish remain constant over time. For low and high bounds, respectively, we assume that people's diets are 100% from either local freshwater or global sources. Where US seafood consumers eat a larger fraction of market marine and estuarine fish, benefits from Minamata are higher. Under the 100% local freshwater diet assumption, benefits from MATS exceed those of Minamata (Minamata/MATS ratio of 0.4 in Fig. 3).

With different assumptions about pharmacokinetics and dose–response functions between mercury intake and human health effects, our results for the Minamata scenario vary from $6 million to $160 billion (2005 USD) in EW benefits, and from $1.4 billion to $498 billion in LT benefits. Although convincing evidence is present to associate MeHg with adverse human effects at low to medium doses, particularly for IQ deficits (7, 50), there may be variability in the magnitude of this effect; for instance, because of genetic variability (51). As a result, we use 95% confidence interval bounds for high and low cases for biomarker and dose–response parameters (*SI Appendix*, Table S3). Associations between mercury exposure and cardiovascular impacts are less certain than IQ effects (9). Previous studies have expressed this uncertainty, using an expected value approach taking into account both the plausibility of a relationship between MeHg and cardiovascular impacts and uncertainties in the parameters of the relationship (12). Our lower bound does not include cardiovascular impacts, whereas our base case and upper bound do, with the 97.5 percentile estimate of the relationship between hair mercury and heart attack risk used in the high case (*SI Appendix*, *Sensitivity Analysis*) (52). A more detailed review of the epidemiological evidence contributing to these parameterizations is given in *SI Appendix*, *IQ effects* and *Cardiovascular impacts*. Although using different exposure–response functions leads to the largest absolute range in cumulative benefits among the sensitivity cases considered (Fig. 3), the relative benefits between Minamata and MATS do not change as substantially.

High and low assumptions for the economic valuation of mercury-related health effects lead to a range of $58 billion to $121 billion (2005 USD) in EW benefits from the Minamata scenario by 2050, and a range of $87 billion to $518 billion in LT benefits. Our sensitivity scenarios for EW benefits address only morbidity, and not mortality, effects: medical costs associated with heart attacks, and the relationship between IQ deficits and lost earnings. We use the 95% confidence interval for the IQ to income relationship and the range of estimates for medical costs from the literature as bounding cases (*SI Appendix*, Table S7). For LT valuations, we use central and range estimates for VSL and LT lost income from regulatory literature (15, 53). The valuation uncertainties considered have the smallest effect on the ratio of benefits between global and domestic scenarios (Fig. 3).

Implications for Policy Evaluation. We developed and applied an assessment method to examine the complex pathways from policies to environmental effects for global toxic pollution from mercury that accounts for uncertainties and knowledge gaps in a structured way. We showed, using this method, that by 2050, the Minamata Convention could have approximately twice the benefit of our scenario simulating domestic actions ($104 billion compared with $43 billion in cumulative EW benefits, and $339 billion compared with $147 billion in cumulative LT benefits). The relative benefit is robust to several uncertainties assessed along the policies-to-impacts pathway, including atmospheric chemical processes, ecosystem time lags, and exposure–response relationships; however, we find that domestic action has a larger benefit when dietary fish is sourced from local freshwater bodies. Per megagram of avoided emissions, the benefits to the United States of domestic action are nearly an order of magnitude larger than global action, highlighting that although mercury is a global pollutant, local policies contribute strongly to local benefits. As shown in *SI Appendix*, Fig. S4, avoided emissions associated with the Minamata Convention outside of the United States may lead to large benefits in Asia and Southern Europe. Abatement costs will also vary by region.

Although we have conducted what is, to our knowledge, the first global-scale attempt to link future emissions trajectories to domestic impacts, our ability to incorporate detailed models of the entire pathway is limited by existing scientific knowledge. In addition to these knowledge gaps, there are also variabilities in mercury's behavior across ecosystems and regions, as well as in human responses (physical and social). Our approach uses bounding assumptions along the policies-to-impacts pathway as a proxy to assess the relative influence of various uncertainties, from a range of disciplines. In a number of previous analyses, range in the benefits of mercury reduction has been specified by the range in exposure–response functions (12, 13). Although our analysis underlines the importance of these uncertainties, particularly those related to cardiovascular effects, it also suggests that previous approaches miss other potentially large contributors to uncertainty in economic effects (particularly within a given time horizon), such as marine and freshwater ecosystem dynamics and dietary intake variabilities.

Although, all else being equal, mercury emissions reductions will ultimately result in exposure reductions, our analysis indicates that uncertainties in ecosystem dynamics affecting the timescale of these reductions will strongly influence benefits within a given time horizon. Many of the processes affecting the conversion of inorganic mercury to MeHg and subsequent uptake in biota are poorly understood, particularly in marine ecosystems (54, 55). In addition, there is variability among ecosystem types, both freshwater (37) and marine and estuarine (32), in how quickly these systems and biota within them respond to changes in deposition. As described previously, our analysis focuses on changes to mercury in surface reservoirs, and accounting for these effects could increase benefits estimates by ~30%. Future research should more fully address the timescales of reemissions from subsurface reservoirs, both land and ocean, and their effects on benefits estimates. Better understanding of mercury cycling, methylation and bioaccumulation processes, their variability, and the potential effects of global changes to climate, land use, and other environmental contaminants will be critical for improving policy evaluation (56), particularly for better understanding the distribution of benefits between current and future generations.

Our analysis also reveals the importance of social factors in estimating the absolute and relative benefits of different policies. Dietary choices, including fish selection and consumption rate, can have a potentially larger influence on the ratio of benefits from global compared with domestic action than substantial scientific uncertainties about mercury's environmental behavior. This sensitivity result suggests that domestic actions may be particularly important for reducing exposure for communities that consume mostly fish sourced from the contiguous United States, such as certain Indigenous peoples and immigrant groups, subsistence fishers, and recreational anglers. In addition, it highlights the policy need for analysis and data collection on the evolving patterns in fisheries production and fish consumption (43). It has been noted that dietary guidance on fish selection and consumption frequency could be part of an adaptation strategy to minimize mercury exposure (57), and our results point toward their potentially large effect as a policy lever. However, dietary advice is highly complex. Fish consumption, and specific fish selection, can have substantial benefits, both nutritional (58, 59) and sociocultural (60). Balancing the risks and benefits of fish consumption therefore requires careful consideration of contextual factors. Even with such adaptive approaches, there is continued need to mitigate future emissions.

Although uncertainties related to chemical speciation of emissions reductions led to the smallest range in cumulative benefits for the Minamata scenario, interactions between these uncertainties and variabilities in dietary fish source could affect the relative benefits of global versus domestic action. At this time, our ability to constrain these speciation uncertainties is partially limited by measurement challenges (61). Improved measurement techniques could provide insight into distributional aspects of control policies.

Differences in valuation methods for health endpoints could lead to substantial variation in benefits estimates. Our two valuation approaches highlight some of these potential variations: Our EW approach emphasizes compounding economy-wide gains over time, but considers only effects to the economy (not individuals) realized within the 2050 time horizon; in contrast, our LT approach more closely resembles regulatory studies, taking into account projected lifetime and nonmarket effects to individuals (e.g., pain and suffering). As highlighted previously, economic benefits estimates are very sensitive to choices of temporal scope of analysis and discounting. Estimates are also sensitive to the endpoints considered: In addition to the health effects considered here, there may be other human and wildlife health endpoints not included in this study that, although not well characterized at this time (7), may also have economic effects. No less important, there may be dimensions of individual and community health and well-being that are not quantifiable within this economic framework, which should be considered in a holistic assessment of policy benefits (62).

Our assessment of US benefits from global and domestic policy is designed to be illustrative, drawing attention to uncertainties in estimating economic benefits and methods to take these uncertainties into account. As a consequence, our estimates should not be taken as a comprehensive projection of impacts. However, as scientific knowledge evolves, many uncertainties can be addressed using similar methodology. Policies-to-impacts analyses similar to the one presented here can be valuable for synthesizing available information, identifying its limitations, and when combined with sensitivity analysis, suggesting areas where scientific data collection to narrow uncertainty would lead to uncertainty reduction of importance to policy-making.

Materials and Methods

Brief explanations of methods have been included throughout *Results and Discussion*. In the *SI Appendix, Supplementary methods*, we provide a detailed description of methodology and data sources for emissions projections, chemical transport modeling, translating changes in deposition to changes in human exposure, IQ and cardiovascular impacts modeling, economic modeling of health impacts, and sensitivity analysis. Institutional review and informed consent were not necessary for this modeling study, as all human health and ecosystem input data were drawn from published sources.

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EXHIBIT 6

To Comments of Public Health and Environmental Groups on Docket ID No. EPA-HQ-OAR-2009-0234 80 Fed. Reg. 75,025 (Dec. 1, 2015)

Declaration of Amy B. Rosenstein Submitted in support of the Joint Motion of State, Local Government and Public Health Respondent-Intervenors for Remand Without Vacatur, in *White Stallion v. EPA*, D.C. Cir. No. 12-1100 (Sept. 24, 2015)

UNITED STATES COURT OF APPEALS
FOR THE DISTRICT OF COLUMBIA CIRCUIT

WHITE STALLION ENERGY LLC, *et al.,*	)	No. 12-1100 (and consolidated cases)
Petitioners.	)	
v.	)	
U.S. ENVIRONMENTAL PROTECTION AGENCY,	)	
Respondent.	)	

Middlesex County)
)
Commonwealth of Massachusetts)

DECLARATION OF AMY B. ROSENSTEIN, MPH

I, Amy B. Rosenstein, state and declare as follows:

I. Purpose of this Declaration

1. I provide this declaration in support of the Joint Motion of the American Academy of Pediatrics, American Lung Association, American Nurses Association, American Public Health Association, Chesapeake Bay Foundation, Citizens for Pennsylvania's Future, Clean Air Council, Conservation Law Foundation, Environment America, Environmental Defense Fund, Izaak Walton League of

America, National Association for the Advancement of Colored People, Natural Resources Council of Maine, Natural Resources Defense Council, Ohio Environmental Council, Physicians for Social Responsibility, Sierra Club, and Waterkeeper Alliance; and the states of California, Connecticut, Delaware, Illinois, Iowa, New York, North Carolina, Oregon, Maine, Maryland, Minnesota, New Hampshire, New Mexico, Rhode Island and Vermont, the Commonwealth of Massachusetts; the Cities of Baltimore, Chicago, New York, the District of Columbia, and Erie County, New York. The Motion requests that the Court retain in place the effectiveness of the emissions limits contained in the Mercury and Air Toxics Standards ("Air Toxics Rule"), published at 77 Fed. Reg. 9304 (February 12, 2012), during the period when a portion of the rule is remanded to the Agency, because doing so will preserve the significant public health benefits associated with EPA's regulations.

II. Qualifications.

2. I provide this declaration based on my 25 years of professional experience in human health risk assessment, exposure assessment, toxicity evaluation, and risk communication. I hold a Masters in Public Health ("MPH") degree in Environmental Health from Yale University, and a Bachelor of Arts ("B.A.") degree in Biology and Environmental Studies from Brandeis University. A current copy of my resume is attached to my declaration as Appendix A.

3. I have specific experience in air quality health impact and benefit analysis, as a co-author of the Sub-Saharan Africa Refinery Study (July 2009), for which I evaluated current health impacts of the fuels used in Sub-Saharan African countries and predicted the beneficial impacts of implementing the refining of reduced sulfur gasoline and other petroleum products. For this World Bank study, I estimated the reduction in refinery emissions and air concentrations to which populations near the refineries would be exposed, and estimated the potential for associated human health and monetary benefits in three regions of Sub-Saharan Africa.

4. I was a key contributor to the U.S. EPA's Air Toxics Risk Assessment Reference Library, the risk assessment guidance for EPA's Air Toxics Program, explaining the goals and methods of air quality risk assessments, toxicity evaluations, and risk communication.

5. I have also provided critical reviews of toxicity and epidemiologic data, along with the inhalation risks for ecological receptors following oil spills, for federal and state agencies, including for setting regulatory standards for EPA's Office of Water, and for private clients. Among my private clients were a number of the environmental organizations for whom I am providing this declaration, and for whom I completed an assessment of the literature on the toxicity of acid gases and available regulatory levels to support the development of comments on EPA's regulatory limits

on acid gas emissions from coal- and oil-fired industrial boilers. My work for other clients focuses on human health and ecological risk assessments for contaminated sites and for facility siting, related to air, water, soil, sediment, fish, and product exposures.

6. In preparing to make this declaration I reviewed the Air Toxics Rule's required emissions limitations to address the acid gas emissions from coal- and oil-fired power plants, specifically those sections of the Rule setting and discussing EPA's reasoning for setting, in the alternative, sulfur dioxide (SO_2) or hydrochloric acid gas (HCl) limits as a surrogates for the acid gases emitted by such power plants, including HCl, hydrofluoric acid (HFl), chlorine gas (Cl_2), and hydrogen cyanide (HCN). I also reviewed the sections of EPA's Regulatory Impact Analysis (RIA) accompanying the final Rule's publication and discussing the Agency's methods for assessing the health benefits associated with controlling the power plant pollution regulated by the Air Toxics Rule.

III. Human Health Effects of the Acid Gases Emitted by Coal- and Oil-fired Power Plants

7. I understand that acid gases which may include hydrogen chloride (HCl), chlorine (Cl_2), hydrogen fluoride (HF), and hydrogen cyanide (HCN) are emitted by coal- and oil-fired power plants. It is important to understand that they are emitted in a mixture with the other stack emissions from a power plant, for example, HCl, HCN, and Cl_2 are emitted together with sulfur dioxide as part of the flue gases

emitted by power plants, not as separate pollutants. These gaseous pollutants are emitted as mixtures, and exposures are therefore exposures to the mixture of pollutants in the flue gas, which includes the individual components listed above.

8. There are documented health effects associated with inhalation exposures to the acid gases emitted by coal- and oil-fired power plants, which were taken into consideration by the U.S. Environmental Protection Agency (EPA) in the Air Toxics Rule. EPA summarized available information on both the acute and chronic health impacts of acid gases. I have reviewed EPA's analysis of the acute and chronic health impacts of acid gases, and I note that their conclusions are based on an analysis of the published research that was available at the time of the Final Rule.

9. My work requires me to remain up to date on the details of the literature and research findings about the human health effects of acid gases. Since the publication of EPA's Final Air Toxics Rule, additional publications have documented the health effects of exposures to acid gases. These more recent publications do not contradict EPA's analysis in the Air Toxics Rule, and in fact further support the need for controls on acid gas emissions.

10. Acid gas exposures can cause acute or chronic human health effects, or both. Acute effects occur in the short-term, immediately following an exposure. Acute toxicity assessments are based on short-term animal tests and/or human studies

such as case reports from accidental poisonings or industrial accidents. Chronic effects occur only after some time has gone by, and are evaluated based on longer-term animal studies that usually range from 90 days to 2 years in duration. Human studies investigating chronic health effects may include studies of a population exposed to ambient air pollutants or workers exposed over time to a particular chemical, and may range from exposures of a few years to a lifetime. Evidence has shown that an acute exposure or a series of acute exposures can also result in chronic health effects.

11. EPA's Regulatory Impact Analysis (RIA)[1] for the Air Toxics Rule summarizes the acute and chronic health effects of the acid gases emitted by coal- and oil-fired power plants. These adverse health effects include severe respiratory problems, particularly in the most sensitive populations (for example, children or those suffering from asthma). I have reviewed the EPA's Air Toxics Rule and RIA summary of the adverse health effects of exposure to the acid gases, as well as more recent publications, and conclude that the following paragraphs describe important health effects of concern that are associated with inhalation of these gases.

[1] EPA, Regulatory Impact Analysis for the Final Mercury and Air Toxics Standards (Dec. 2011), EPA-HQ-OAR-2009-0234-20131 ("RIA").

a. Chlorine Gas.

12. Exposure to chlorine gas (Cl_2) causes acute effects that, even at relatively low levels, include tissue damage to the eyes, skin, throat, and respiratory tract, respiratory irritation,[2] and, at higher levels, include respiratory distress with airway constriction and pulmonary edema. Delayed pulmonary edema may also develop up to 24 hours following acute exposure.[3] These data are supported by acute exposure experiments in laboratory animals.[4] In Jonasson, *et al.* (2013), mice were exposed once to Cl_2, and, although there was a marked acute response that subsided after 48 hours, a sustained airway hyperresponsiveness was observed for at least 28 days. Other observed effects of Cl_2 inhalation in laboratory animals include cardiac

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[2] *Id.*; California Office of Environmental Health Hazard Assessment (CA OEHHA), Appendix D.2: *Acute RELs and toxicity summaries using the previous version of the Hot Spots Risk Assessment guidelines* (available at http://www.oehha.ca.gov/air/hot_spots/2008/AppendixD2_final.pdf); Appendix D.3: *Chronic RELs and toxicity summaries using the previous version of the Hot Spots Risk Assessment guidelines* (available at http://www.oehha.ca.gov/air/hot_spots/2008/AppendixD3_final.pdf) (CA OEHHA).

[3] CA OEHHA, Appendices D.2 and D.3 *supra* n.2.

[4] *Id*; Martin JG, Campbell HR, Iijima H, Gautrin D, Malo JL, Eidelman DH, Hamid Q, Maghni K, Chlorine-induced injury to the airways in mice, 168(5) Am. J. Respiratory & Critical Care Med. 568 (2003) (available at http://citeseerx.ist.psu.edu/viewdoc/download?doi=10.1.1.312.1091&rep=rep1&type=pdf). Jonasson S, Koch B, Bucht A, Inhalation of chlorine causes long-standing lung inflammation and airway hyperresponsiveness in a murine model of chemical-induced lung injury, 303 Toxicology 34 (2013).

pathology.[5] Reactive airways dysfunction syndrome, a chemically induced asthma, has been reported following acute exposure to Cl_2,[6] and reactive airways dysfunction syndrome has been reported to persist in exposed individuals.[7]

13. Chronic inhalation exposure to low concentrations of Cl_2 can cause eye and nasal irritation, sore throat, and cough, as well as corrosion of the teeth,[8] and, at higher levels, can cause respiratory distress with airway constriction, pulmonary edema, and lung collapse.[9] Breathing capacity impacts were more severe among individuals with pre-existing airway hyperresponsiveness (a characteristic feature of asthma) and reactive airways dysfunction syndrome developed among workers exposed to Cl_2.[10] These effects are supported by chronic laboratory animal studies,

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[5] Zaky A, Bradley WE, Lazrak A, Zafar I, Doran S, Ahmad A, White CW, Louis J Dell'Italia, Matalon S, Ahmad S, Chlorine inhalation-induced myocardial depression and failure, 3 Physiology Rep. e12439 (2015) (available at http://physreports.physiology.org/content/3/6/e12439.full-text.pdf+html).

[6] RIA at 4-75 to 4-76.

[7] Brooks SM, Weiss MA, Bernstein IL, Reactive airways dysfunction syndrome (RADS). Persistent asthma syndrome after high level irritant exposures. 88(3) CHEST J. 376 (1985). (available at http://journal.publications.chestnet.org/data/Journals/CHEST/21486/376.pdf).

[8] CA OEHHA, Appendices D.2 and D.3 *supra* n.2.

[9] RIA at 4-75 to 4-76.

[10] CA OEHHA, Appendices D.2 and D.3 *supra* n.2.

one of which resulted in upper respiratory epithelial lesions.[11] White and Martin (2010)[12] state that while the respiratory and lung effects of acute severe Cl_2 inhalation have been shown in some cases to be reversible, certain vulnerable populations such as smokers and atopic individuals (those with a predisposition toward developing certain allergic hypersensitivity reactions) have longer-term chronic respiratory disorders resulting from longer-term low-level exposures.

b. Hydrogen Chloride.

14. Acute inhalation exposure to hydrogen chloride gas (HCl) causes irritation of the nose, throat, and respiratory tract, with the greatest impact on the upper respiratory tract. In addition, exposure to HCl can lead to reactive airways dysfunction syndrome, with children being more vulnerable to these effects.[13] These

[11] Wolf DC, Morgan KT, Gross EA, Barrow C, Moss OR, James RA, Popp JA, Two-year inhalation exposure of female and male B6C3F1 mice and F344 rats to chlorine gas induces lesions confined to the nose, 24 Fundamentals of Appl. Toxicology 111 (1995) (as cited in CA OEHHA).

[12] White CW, Martin JG, Chlorine gas inhalation: human clinical evidence of toxicity and experience in animal models. *In:* 7 Proc. Am. Thoracic Soc. 257 (2010) (available at http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3136961/pdf/PROCATS74257.pdf).

[13] RIA at 4-77.

effects have also been observed in laboratory animal experiments, with the addition of ocular effects.[14]

15. Chronic exposure to HCl can cause changes in pulmonary function, chronic bronchitis, skin inflammation, dental enamel erosion, and effects on the mucous membranes of the nose, mouth, and eyes. For some effects, symptoms may be delayed 1-2 days.[15] Animal studies show impacts on the upper respiratory tract due to chronic HCl exposures.[16]

c. Hydrogen Flouride.

16. Acute inhalation exposure to hydrogen fluoride (HF) causes severe respiratory symptoms and damage, including severe irritation and pulmonary edema.[17] Animal data support the acute toxicity of HF.[18] While injury due to

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[14] CA OEHHA, Appendix D.2, *supra* n.2, *Individual Acute Toxicity Summaries: Hydrogen Chloride.*

[15] CA OEHHA, Appendix D.3, *supra* n.2, *Individual Chronic Toxicity Summaries: Hydrogen Chloride*.

[16] EPA, Integrated Risk Information System On-Line (IRIS) (available at http://www.epa.gov/iris/) (last accessed 9/18/15).

[17] RIA at 4-77 to 4-78.

[18] CA OEHHA, Appendix D.3, *supra* n.2, *Individual Chronic Toxicity Summaries: Hydrogen Fluoride*.

inhalation of HF is thought to be unlikely at concentrations less than 60%, there are a few reported cases[19] of pulmonary injury occurring at much lower concentrations.[20]

17. Chronic inhalation exposures to fluorides have been studied in the workplace. A statistically significant increase in the incidence of acute respiratory disease was reported, as well as statistically significant relationships between air fluoride and bone density increases. Several studies of the inhalation of HF in animals show chronic effects.[21]

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[19] Bennion JR, Franzblau A, Chemical pneumonitis following household exposure to hydrofluoric acid, 31 Am. J. Indus. Med. 474 (2003) (available at http://deepblue.lib.umich.edu/bitstream/handle/2027.42/34814/15_ftp.pdf); Franzblau A, Sahakian N, Asthma following household exposure to hydrofluoric acid, 44 Am. J. Indus. Med. 321 (2003) (available at http://deepblue.lib.umich.edu/bitstream/handle/2027.42/34824/10274_ftp.pdf?sequence=1).

[20] Miller SN, Acute Toxicity of Respiratory Irritant Exposures. *In*: The Toxicant Induction of Irritant Asthma, Rhinitis, and Related Conditions, 83 (WJ Meggs ed., 2014) (available at https://books.google.com/books?id=MOK5BAAAQBAJ&pg=PA244&dq=meggs+rhinitis&hl=en&sa=X&ved=0CDAQ6AEwAGoVChMIl9XItJyIyAIVQc-ACh2J0AO-#v=onepage&q=meggs%20rhinitis&f=false).

[21] CA OEHHA, Appendix D.3, *supra* n.2, *Individual Chronic Toxicity Summaries: Fluorides Including Hydrogen Fluoride*.

d. Hydrogen Cyanide.

18. Acute inhalation exposure to hydrogen cyanide (HCN) results primarily in central nervous system effects, ranging from headache to unconsciousness.[22] Additionally, acute exposures result in respiratory and cardiovascular health effects. These reported acute health effects are similar among animals and humans,[23] and have been reported in one recent animal study.[24]

19. The chronic effects of HCN include central nervous system, thyroid, and hematological (blood) impacts. Although occupational studies are complicated by mixed chemical exposures, several reports indicate that chronic low exposure to HCN can cause neurological, respiratory, cardiovascular, and thyroid effects.[25]

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[22] CA OEHHA, Appendix D.2, *supra* n.2, *Individual Acute Toxicity Summaries: Hydrogen Cyanide*.

[23] *Id.*

[24] Sweeney LM, Sharits B, Gargas NM, Doyle T, Wong BA, James RA, Acute Lethality of Inhaled Hydrogen Cyanide in the Laboratory Rat: Impact of Concentration x Time Profile and Evaluation of the Predictivity of Toxic Load Models (No. NAMRU-D-13-35), Naval Medical Research Unit Dayton Wright-Patterson AFB OH (2014) (available at http://www.dtic.mil/cgi-bin/GetTRDoc?AD=ADA579551).

[25] CA OEHHA, Appendix D.3, *supra* n.2, *Individual Chronic Toxicity Summaries: Hydrogen Cyanide*; EPA IRIS, *supra* n.16 (last accessed Sept. 18, 2015).

IV. The Derivation and Use of Inhalation Threshold Levels for the Acid Gases

20. After evaluation of the toxicity literature, inhalation "threshold" levels (concentrations of chemicals in air) for the general population (including sensitive sub-populations) can be established. Safety factors are often applied to animal or human study results to account for species differences and sensitive populations, resulting in a lower (that is, a more protective) threshold level. Depending on the exposure durations, safety factors, and interpretations of the data, threshold levels established by various entities (for example, government agencies) may be different. Threshold levels may be set for short-term exposures, such as 1-hour peak concentrations, or may be set in terms of exposure to average air concentrations over time. These threshold levels describe the concentrations in the air that are generally considered to be safe for the general population or for the general population of workers in specific industries. They do not indicate the absence of risk of health effects for air concentrations at or below the threshold.

21. Chronic acid gas exposure threshold levels have been established for the general public by both the California Office of Environmental Health Hazard Assessment as chronic recommended exposure limits (RELs) for all four acid gases[26]

[26] CA OEHHA, Appendix D.3, *supra* n.2, *Individual Chronic Toxicity Summaries: Chlorine; Hydrogen Cyanide; Fluorides Including Hydrogen Fluoride; Hydrogen Chloride*.

and by the EPA as chronic reference concentrations (RfC) for HCl and HCN.[27] These chronic threshold levels for inhalation of acid gases are designed to assess exposures and health risks, and to protect the general population against adverse health effects over time, but they do not take into account repeated short-term peaks in air concentrations. In addition, although sensitive populations are taken into account in some air quality standards, threshold levels are not always set at levels which will protect the most sensitive individuals in the population, such as children, elderly, or those with respiratory diseases. Each agency has based the derived threshold level on comprehensive reviews of the literature and has selected appropriate toxicity studies to support their setting of these chronic threshold levels. I note that all four of the acid gases under consideration have established threshold levels for both acute and chronic effects, and, thus, it is clear that there is solid evidence of adverse health effects associated with the inhalation of these gases.

22. Whether or not acid gas emissions from a particular power plant result in exposures above established threshold levels, adverse health effects might still occur, in particular, in sensitive individuals (for example, the elderly, children, and persons with respiratory conditions such as asthma) living near the source of the emissions, especially if these lower exposure levels occur repeatedly over time. For example, for HCl, researchers have noted that recurring exposures at low-to-moderate

[27] EPA IRIS, *supra* n.16 (last accessed Sept. 18, 2015).

levels may result in increased bronchial responsiveness and asthma-like symptoms.[28] Importantly, certain hazardous air pollutants may interact with criteria pollutants in ambient air to exacerbate asthma, and these "adverse responses after ambient exposures to complex mixtures often occur at concentrations below those producing effects in controlled human exposures to a single compound."[29]

V. Localized Acid Gas Emissions and EPA's Air Toxics Rule

23. As part of the reviews accompanying the final Air Toxics Standards, I understand that EPA assessed the demographics of the areas surrounding the existing regulated power plants, and found that individuals living within three miles of a coal-fired power plant were 48 percent more likely to be members of a racial minority, and 31 percent more likely to be living below the poverty line, than the national average. 77 Fed. Reg. 9304, 9445 (Feb. 12, 2015).

24. As acknowledged by EPA in the Air Toxics Rule, evidence points to the increased susceptibility of minority and lower-income communities to environmental

[28] Leroyer C, Malo J-L, Girard D, Dufour J-G, Gautrin D, Chronic rhinitis in workers at risk of reactive airways dysfunction syndrome due to exposure to chlorine, 56 Occupational Envtl. Med. 334 (1999) (available at http://oem.bmj.com/content/56/5/334.full.pdf).

[29] Leikauf GD, Hazardous air pollutants and asthma, 110(4) Envtl. Health Persps. 505 (2002) (available at http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1241200/pdf/ehp110s-000505.pdf).

exposures, including ambient air pollution and industrial emissions,[30] including complex mixtures of environmental air pollutants.[31] Minority and low-income communities incur disproportionate exposures to environmental contaminants, as well as being more susceptible than the general population to the effects of such exposures "because of limited understanding of environmental hazards, disenfranchisement from the political process, and socioeconomic factors such as poor nutrition, stress, and lack of adequate health care…, and … substandard housing and resource-poor communities…."[32] Although the specific components of these

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[30] Bell ML, Zanobetti A, Dominici F, Evidence on vulnerability and susceptibility to Health Risks associated with short-term exposure to particulate matter: A systematic review and meta-analysis, 178 Am. J. Epidemiology 865 (2013) (available at http://aje.oxfordjournals.org/content/early/2013/07/24/aje.kwt090.full.pdf+html); Jerrett M, Burnett R, Brook J, Kanaroglou P, Giovis C, Finkelstein N, *et al*., Do socioeconomic characteristics modify the short term association between air pollution and mortality? Evidence from a zonal time series in Hamilton. Canada. 58 J. Epidemiol. Community Health 31 (2004) (available at http://jech.bmj.com/content/58/1/31.full.pdf+html); Krewski D, Jerrett M, Burnett RT, Ma R, Hughes E, Shi Y *et al*., Extended Follow-Up and Spatial Analysis of the American Cancer Society Study Linking Particulate Air Pollution and Mortality, 140 Respiratory Rep. Health Effects Inst. 114 (2009) (available through: http://pubs.healtheffects.org/).

[31] Carter-Pokras O, Zambrana RE, Poppell CF, Logie LA, Guerrero-Preston R, The environmental health of Latino children, 21 J. Pediatric Health Care 307 (2007) (available at http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2967224/pdf/nihms-244430.pdf).

[32] *Id.* (citing Institute of Medicine, *Toward environmental justice: Research, education, and health policy needs,* Washington, D.C. (1999) (available at http://www.nap.edu/read/6034/chapter/1).

mixed air pollution exposures that cause disease are not completely characterized,[33] it is well known that some components of air pollution, including particulate matter and acid gases, can cause disease in experimental animals and in occupationally exposed humans.

VI. Power Plants and U.S. EPA's Air Toxics Rule

25. I am aware that coal-and oil-fired power plants greater than 25 MW in size are regulated by the Air Toxics Rule. I am also aware that these are the largest industrial sources of HCl and HF, emitting the majority of these acid gases nationally.

26. I am aware that U.S. EPA's Air Toxics Rule sets emissions limits for the acid gases emitted by coal- and oil-fired power plants. The Rule sets either sulfur dioxide emissions limits or HCl emissions limits as a surrogate for total toxic acid gas emissions, for each coal-fired power plant unit, and for oil-fired units, HCl and HF limits are set as surrogates for all the acid gases those power plants emit. EPA set the emissions limits based on the performance of the best performing similar source (for new sources), or the top twelve percent of sources (for existing sources) at the time the standards were set, and providing for variability of the input fuel constituents. EPA did not set health threshold-based emissions standards. EPA's emissions

[33] Delfino RJ, Epidemiologic evidence for asthma and exposure to air toxics: linkages between occupational, indoor, and community air pollution research, 110(4) Envtl. Health Persps. 573 (2002) (available at http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1241209/pdf/ehp110s-000573.pdf).

standards for coal-fired power plants regulate surrogates because the specific acid gases are invariably present in the sulfur dioxide plumes emitted by coal-fired power plants, and can be controlled by sulfur dioxide controls. The Agency found that the acid gases emitted by oil-fired power plant units are invariably present in the plume emissions from oil-fired power plants and that both HF and HCl can be measured and monitored. 76 Fed. Reg. 24976, 25023 (May 3, 2011).

27. I understand that U.S. EPA estimates that the Air Toxics Rule will decrease emissions of sulfur dioxide from coal-fired power plants (greater than 25 MW) by 1.4 million tons per year, and will reduce emissions of HCl by about 40,000 tons per year. 77 Fed. Reg. 9304, 9424, Table 7 (Feb. 12, 2012). EPA assesses the reductions in sulfur dioxide emissions because sulfur dioxide is regulated as a surrogate for the acid gas emissions from power plants. It is readily monitored and measured, and the health benefits of reducing sulfur dioxide levels are well understood.

VII. The Potential Effects of Staying or Otherwise Failing to Implement the Air Toxics Rule.

28. I understand that the Air Toxics Rule was to be implemented at existing power plants in April 2015, but that some power plants have been granted one year extensions to put on controls or shut down, to April 2016.

29. I understand that certain parties may seek to stay the effectiveness of the emissions limits under the Air Toxics Rule, including the HCl, HF and sulfur dioxide

emissions limits included under the Rule, or to strip those protections completely, during the period of time when EPA fixes a problem with the initial decision whether to regulate air toxics emissions from the power sector.

30. I understand that if the Rule is stayed, power plants that have received extensions may not be required to comply by April 2016. Additionally those plants that have put on controls to comply with the Rule's emissions limits by the initial April 2015 deadline may not be required to comply with the Rule's emissions limits during the period when the Rule is stayed or otherwise not in place.

31. Based on my understanding of acid gas health impacts, both chronic and acute, it is clear to me that if emissions remain uncontrolled, so that tonnage reductions are not achieved during any period in which the Air Toxics Rule is not in effect, there could be direct health impacts experienced by the population most exposed to the uncontrolled emissions (that is, those living near the power plants) that would otherwise not occur.

32. Those adverse health effects, which include acute effects such as severe respiratory symptoms, respiratory damage, severe irritation, nervous system effects, and pulmonary edema, and chronic effects such as chronic respiratory disorders, exacerbation of allergic diseases, changes in pulmonary function, chronic bronchitis, and effects on the mucous membranes of the nose, mouth, and eyes, will persist for as long as acid gas emissions (whether measured in terms of the total tons of the four

major acid gases HCl, HF, Cl_2 and HCN, or as sulfur dioxide levels) remain uncontrolled. That is, they will continue to occur so long as the pollution controls are not in place and operating at the power plants to meet the Air Toxics Standards, and will be reduced when the emissions of acid gases and sulfur dioxide are curtailed.

I declare under the penalty of perjury under the laws of the United States, that to the best of my knowledge, the foregoing is true and correct.

Executed on September 22, 2015, at Boston, Massachusetts.

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Amy B. Rosenstein

EXHIBIT 7

To Comments of Public Health and Environmental Groups on Docket ID No. EPA-HQ-OAR-2009-0234 80 Fed. Reg. 75,025 (Dec. 1, 2015)

Declaration of Douglas W. Dockery Submitted in support of the Joint Motion of State, Local Government and Public Health Respondent-Intervenors for Remand Without Vacatur, in *White Stallion v. EPA*, D.C. Cir. No. 12-1100 (Sept. 24, 2015)

UNITED STATES COURT OF APPEALS
FOR THE DISTRICT OF COLUMBIA CIRCUIT

WHITE STALLION ENERGY LLC, *et al.*,	)	No. 12-1100 (and consolidated cases)
Petitioners.	)	
v.	)	
U.S. ENVIRONMENTAL PROTECTION AGENCY,	)	
Respondent.	)	

Suffolk County	)
	)
Commonwealth of Massachusetts	)

DECLARATION OF DOUGLAS W. DOCKERY, M.S. Sc.D.
HARVARD UNIVERSITY SCHOOL OF PUBLIC HEALTH

I, Douglas W. Dockery, state and declare as follows:

I. Purpose of this Declaration

1. I provide this declaration in support of the Joint Motion of the American Academy of Pediatrics, American Lung Association, American Nurses Association, American Public Health Association, Chesapeake Bay Foundation, Citizens for Pennsylvania's Future, Clean Air Council, Conservation Law

Foundation, Environment America, Environmental Defense Fund, Izaak Walton League of America, National Association for the Advancement of Colored People, Natural Resources Council of Maine, Natural Resources Defense Council, Ohio Environmental Council, Physicians for Social Responsibility, Sierra Club, and Waterkeeper Alliance; and the states of California, Connecticut, Delaware, Illinois, Iowa, New York, North Carolina, Oregon, Maine, Maryland, Minnesota, New Hampshire, New Mexico, Rhode Island and Vermont, the Commonwealth of Massachusetts; the Cities of Baltimore, Chicago, New York, the District of Columbia, and Erie County, New York. The Motion requests that the Court retain in place the effectiveness of the emissions limits contained in the Mercury and Air Toxics Standards ("Air Toxics Rule"), published at 77 Fed. Reg. 9304 (February 12, 2012), during the period when a portion of the rule is remanded to the Agency, because doing so will preserve the significant public health benefits associated with EPA's regulations.

2. I provide this declaration based on my professional experience, as outlined herein and in my curriculum vitae, attached as Appendix A to this declaration. In preparing this declaration I reviewed the Air Toxics Rule's required emissions limitations, specifically those sections of the Rule discussing the alternative particulate matter limits imposed by the Rule, and the Agency's use of particulate matter as a surrogate for the non-mercury metallic hazardous air

pollutants. I also reviewed sections of EPA's Regulatory Impacts Analysis accompanying the final Rule's publication and discussing the Agency's methods for assessing the health benefits associated with controlling the power plant pollution regulated by the Air Toxics Rule.

II. Experience and Qualifications

3. I am currently the John L. Loeb and Frances Lehman Loeb Professor of Environmental Epidemiology, and the Chair of the Department of Environmental Health at Harvard University's T.H. Chan School of Public Health. I also serve as the Director of the Harvard-National Institute of Environmental Health Studies Center for Environmental Health, and as an Associate Professor of Medicine in Epidemiology at the Harvard Medical School's Channing Laboratory. I have held appointments at the Harvard School of Public Health since 1987. I hold a Master of Science (M.S.) and a Doctorate in Science (Sc.D.) in environmental health from the Harvard School of Public Health, an M.S. in meteorology from the Massachusetts Institute of Technology, and a Bachelor of Science (B.S.) in physics from the University of Maryland.

4. I have for 40 years studied and published extensively on the human health effects of exposure to fine particulate air pollution. I was the Principal Investigator of "Respiratory Health Effects of Respirable Particles and Sulfur

Oxides," commonly known as the Harvard Six Cities Study,[1] which examined the health effects of air pollution exposures in populations who have been followed for over 35 years. The results of both that study and the subsequent work affirming those results are relied on by U.S. EPA in modelling the health benefits of the particulate matter reductions resulting from the Air Toxics Rule.

5. My work also examines the respiratory effects associated with particulate and acid aerosol air pollution,[2] the growth of lung function in children,[3] and decline in adults, the environmental risk factors affecting these trajectories, and the relationship between particulate air pollution and adverse cardiovascular

[1] Dockery DW, Pope CA, Xu X, Spengler JD, Ware JH, Fay ME, Ferris BG, Speizer FE, An association between air pollution and mortality in six United States cities, 329 New Eng. J. Med. 1753 (1993); Laden F, Schwartz J, Speizer FE, Dockery DW, Reduction in Fine Particulate and Mortality: Extended follow-up of the Harvard Six Cities Study, 173 Am. J. Respiratory & Critical Care Med. 667 (2006); Lepeule J, Laden F, Dockery D, Schwartz J. Chronic Exposure to Fine Particles and Mortality: An Extended Follow-up of the Harvard Six Cities Study from 1974 to 2009, 120(7) Envtl. Health Persp. 965 (2012).

[2] Dockery DW, Speizer FE, *et al*., Effects of inhalable particles on respiratory health of children, 139 Am. Rev. Respiratory Disease 587 (1989); Dockery DW, Cunningham J, Damokosh AI, Neas LM, Spengler JD, Koutrakis P, Ware JH, Raizenne M, and Speizer FE, Health Effects of Acid Aerosols on North American Children-Respiratory Symptoms. 104 Envtl. Health Persp. 500 (1996).

[3] Wang X, Dockery DW, Wypij D, Gold DR, Speizer FE, Ware JH, Ferris BJ, Jr., Pulmonary function growth velocity in children 6 to 18 years, 148 Am. Rev. Respiratory Disease 1460 (1993).

effects.[4] My research team in 1993 demonstrated that life expectancy is strongly associated with community particulate air pollution levels.[5] I also research the effectiveness of environmental controls in improving health, including studies of improved life expectancy in the Harvard Six Cities Study subjects following lower fine particle concentrations,[6] the health effects of coal bans on mortality in

[4] Dockery DW, Epidemiologic evidence of cardiovascular effects of particulate air pollution, 109 Envtl. Health Persp. (Supp 4), 483 (2001); Rich DQ, Schwartz J, Mittleman MA, Link M, Luttmann-Gibson H, Catalano PJ, Speizer FE, Dockery DW, Association of short-term ambient air pollution concentrations and ventricular arrhythmias, 161 Am J. Epidemiology 1123 (2005); Rich DQ, Mittleman MA, Link MS, Schwartz J, Luttman-Gibson H, Catalano PJ, Speizer FE, Gold DR, Dockery DW, Increased risk of paroxsysmal atrial fibrillation episodes associated with acute increases in ambient air pollution, 114 Envtl. Health Persp. 120 (2006).

[5] Dockery DW, Pope CA III, Xu X, Spengler JD, Ware JH, Fay ME, Ferris BG Jr, Speizer FE, An association between air pollution and mortality in six US cities. 329 New Eng. J. Med. 1753-1759 (1993).

[6] Laden L, Schwartz J, Speizer F, Dockery DW, Reduction in fine particulate air pollution and mortality: Extended follow-up of the Harvard Six Cities study. 173(6) Am. J. Respiratory & Critical Care Med. 667 (2006); Lepeule J, Laden F, Dockery D, Schwartz J, Chronic Exposure to Fine Particles and Mortality: An Extended Follow-up of the Harvard Six Cities Study from 1974 to 2009, 120(7) Envtl. Health Persp. 965 (2012).

Ireland,[7] and on the effects of reduced fine particle concentrations on life expectancy in the United States.[8]

6. Among my professional service appointments, I have provided expert advice to the U.S. Environmental Protection Agency as a Review Panel member of the Clean Air Science Advisory Committee. Of particular relevance to this declaration, I reviewed U.S. EPA's assessment of the concentration-response function for fine particulate (PM 2.5)-related mortality and the mortality impact of changes in fine particulate matter concentrations in the U.S. in 2006 and 2008. I also provided comments to the Agency in 2005 on the Staff Paper related to updating the National Ambient Air Quality Standards for Particulate Matter.

III. The Health Effects of Exposure to Particulate Matter

7. Particulate matter is produced both by direct emissions of fuel combustion (these are the primary particles) and by chemical reactions in the

[7] Clancy L, Goodman P, Sinclair H, Dockery DW, Effect of air-pollution control on death rates in Dublin, Ireland: an intervention study, 360 The Lancet 1210 (2002).

[8] Pope A, Ezzati M, Dockery DW, Fine-Particulate Air Pollution and Life Expectancy in the United States, 360(4) New England Journal of Medicine 376 (2009); Correia AW, Pope CA 3rd, Dockery DW, Wang Y, Ezzati M, Dominici F, Effect of air pollution control on life expectancy in the United States: an analysis of 545 U.S. Counties for the period from 2000 to 2007, 24(1) Epidemiology 23 (2013).

atmosphere after sulfur dioxide is emitted (the secondary particles). Both primary and secondary particles cause adverse health effects in humans.

8. There is a robust scientific literature analyzing and describing the public health effects of breathing various concentrations of particulate matter in the ambient air, including effects on mortality, as well as adverse respiratory and cardiovascular effects. This work has been ongoing since the late 1970s, and at this point over a dozen prospective cohort epidemiological studies show significant associations between various measures of long-term exposure to particulate matter and elevated rates of annual mortality.[9] These prospective cohort designs control at the individual subject level for variables other than particulate matter exposure. These studies, including my own, show consistent relationships between fine particle indicators and premature mortality over multiple locations in the United States, Canada, and similar developed countries in Europe. Additional work has examined the correlation between reductions in particulate matter exposures and improvements in health endpoints in the United States.[10]

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[9] Hoek G, Krishnan RM, Beelen R, Peters A, Ostro B, Brunekreef B, Kaufman JD, Long-term air pollution exposure and cardio- respiratory mortality: a review, 12 (1) Envtl. Health 43 (2013).

[10] Pope A, Ezzati M, Dockery DW, Fine-Particulate Air Pollution and Life Expectancy in the United States, 360(4) New Eng. J. Med. 376 (2009); Correia AW, Pope CA III, Dockery DW, Wang Y, Ezzati M, Dominici F, Effect of air pollution control on life expectancy in the United States: an analysis of 545 U.S. Counties for the period from 2000 to 2007, 24(1) Epidemiology 23 (2013).

9. The richness and consistency of this published research means we have more confidence today regarding the quantitative relationship between adverse health effects and both the long term and short term populations exposures to various levels of particulate matter and sulfur dioxide air pollution than when EPA's Air Toxics Rules were set in 2011 and in 2012.

10. Reducing exposure to particulate matter reduces premature mortality in adults. Both prospective cohort and cross-sectional comparisons between communities have demonstrated that populations living in communities with higher particulate air pollution concentrations have higher mortality rates and shorter life expectancy. Examination of changes over time in these same communities has shown that as particulate air pollution improves, mortality rates and life expectancy improve. In the United States, communities with the greatest reductions in fine particulate air pollution between 1980 and 2000 had on average the largest improvement in life expectancy. Improved life expectancy was even observed in communities with fine particle concentrations already in compliance with the National Ambient Air Quality Standards.

11. In addition to reduced mortality, the direct health benefits of reducing exposure to particulate matter emissions include reduced incidence of non-fatal heart attacks, avoided respiratory hospital admissions, avoided cardiovascular hospital admissions, reduced emergency room visits for asthma in children under

18, reduced incidence of acute bronchitis and reduced incidence of chronic bronchitis in adults, reduced asthma exacerbation and upper respiratory symptoms in asthmatic children, reduced incidence of acute bronchitis and lower respiratory symptoms in children, reduced incidence of other cardiovascular and respiratory effects, fewer lost work days and fewer restricted activity days.

12. I understand that power plant particulates include non-mercury metals which are adsorbed on to both primary and secondary fine particles. I understand that these toxic metals include, among other constituents, arsenic, beryllium, cadmium, chromium, cobalt, manganese, nickel and lead. Each of these metals has demonstrated toxic effects.

13. I am aware of current scientific research and analysis directed at assessing the health effects associated with the non-mercury metal toxic constituents of particulate matter air pollution. My understanding of the state of that scientific work is that it not possible to quantify precisely the health effects attributable to the specific non-mercury toxic metal constituents of particulates, separately for the health effects of ambient exposures to fine particles.

IV. U.S. EPA's Air Toxics Rule

14. I am aware that EPA's Air Toxics Rule sets emissions limits for the non-mercury toxic metals emitted by power plants. The Rule sets either non-mercury metal toxic-specific emissions limits or filterable particulate matter emissions limits as a surrogate for total toxic non-mercury metal emissions, for each power plant unit.

15. I understand that U.S. EPA set standards for particulate matter as an alternative to non-mercury toxic metal specific standards because the non-mercury toxic metal constituents are invariably present in the particulate matter emissions from power plants. The Agency logically concluded that control of the particulate matter emissions would also limit emissions of these non-mercury toxic metal constituents. 76 Fed. Reg. 24976, 25038 (May 3, 2011).

16. I understand that when U.S. EPA modelled the health benefits of the Air Toxics Rule, the Agency assumed that all forms of the fine particulates controlled by the Rule are equally potent in causing premature mortality and adverse health effects. In part EPA makes this assumption because the state of the science does not yet support separate assessments of the health risks of individual constituents of particulate matter. For example, the recently completed National

Particle Toxicity Component (NPACT)[11] studies did not find evidence that any specific source, component, or size class of particulate matter could be excluded as a possible contributor to PM toxicity, and concluded that regulations targeting specific sources or components of fine particulate mass would not be more effective than controlling fine particulate mass as a whole.

V. EPA's Assessment of the Health Benefits of the Particulate Matter Limits Set by the Mercury and Air Toxics Standards

17. U.S. EPA evaluates the health benefits of the Air Toxics Rule in part by evaluating the health benefits of the reductions in particulate matter to be achieved by the Air Toxics Rule. EPA estimates the annualized health benefits of the particulate matter reductions based on the published, peer-reviewed work done by Fann, *et al*. in 2009,[12] on benefit-per-ton of pollution factors. These estimates use well established and commonly used risk assessment approaches.

18. EPA also estimates the health benefits of the Air Toxics Rule based on the sulfur dioxide emissions reductions expected as a result of the rule, and the health effects associated with the secondary particulate matter formed in the atmosphere after emissions, but avoided due to the sulfur dioxide emissions limits imposed by the Rule.

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[11] Health Effects Institute, HEI NPACT Review Panel, HEI's National Particle Component Toxicity (NPACT) Initiative, Executive Summary, Boston, MA (2013), *available a*t: http://www.healtheffects.org/Pubs/NPACT-ExecutiveSummary.pdf (last visited Sept. 21, 2015).

19. EPA's calculation of the value of the health benefits associated with the Air Toxics Rule follows the established, commonly used risk assessment approach. Under that methodology, EPA translated the changes in particulate matter emissions associated with the rule into estimated population exposures. Health impact are then calculated based on population, baseline disease and mortality rates, estimated changes in air pollution exposures, and exposure-response functions from the peer-reviewed literature. This health impacts assessment quantified changes in the incidence of adverse health impacts resulting from changes in human exposures to specific pollutants, such as fine particulates. EPA's health impact assessment for the Air Toxics Rule was based on the health effects directly linked to ambient particulate matter concentrations. The health effects assessment is based on the best available methods of benefits transfer -- a means of adapting primary research from similar contexts to obtain the most accurate measure of benefits for the environmental quality change under analysis.

20. Based on my experience, this methodology for assessing the health benefits of the particulate matter standards set by the Agency is a well-established approach to estimating the retrospective or prospective change in adverse health impacts expected to result from population-level changes in exposure to pollutants.

VI. The Potential Effects of Staying or Otherwise Failing to Implement the Air Toxics Rule.

21. I understand that the Air Toxics Rule was to be implemented at existing coal- and oil-fired power plants by April 2015, but that some power plants have been granted one year extensions to put on controls or shut down, to April 2016.

22. I understand that certain parties seek to stay the effectiveness of the emissions limits under the Air Toxics Rule, including the particulate matter and sulfur dioxide emissions limits included under the Rule, or to strip those protections completely, during the period of time when EPA fixes a problem with the initial decision whether to regulate air toxics emissions from the power sector.

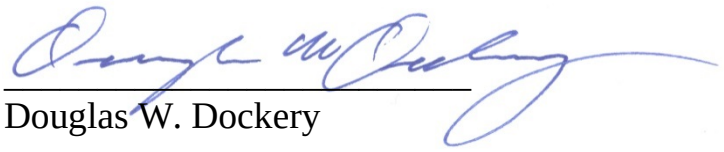
23. I understand that if the Rule is stayed, power plants that have received extensions will not be required to comply by April 2016. Additionally those plants that have put on controls to comply with the Rule's emissions limits by the initial April 2015 deadline will not be required to run those controls in order to comply with the Rule's emissions limits during the period when the Rule is stayed or otherwise not in place.

24. It is clear to me that if particulate matter and sulfur dioxide emissions remain uncontrolled, so that tonnage reductions are not achieved during any period in which the Air Toxics Rule is not in effect, there will be direct health impacts that would otherwise not be experienced, had the sulfur dioxide and particulates been controlled during the same time period. Those adverse health effects will

persist for as long as particulate matter and sulfur dioxide pollution controls are not in place and operating at the power plants, and will be reduced when the emissions of particles and sulfur dioxide are curtailed.

I declare under the penalty of perjury under the laws of the United States, that to the best of my knowledge, the foregoing is true and correct.

Executed on September 22, 2015, at Boston, Massachusetts.



Douglas W. Dockery

EXHIBIT 8

To Comments of Public Health and Environmental Groups on Docket ID No. EPA-HQ-OAR-2009-0234 80 Fed. Reg. 75,025 (Dec. 1, 2015)

Declaration of Jonathan I. Levy Submitted in support of the Joint Motion of State, Local Government and Public Health Respondent-Intervenors for Remand Without Vacatur, in *White Stallion v. EPA*, D.C. Cir. No. 12-1100 (Sept. 24, 2015)

UNITED STATES COURT OF APPEALS
FOR THE DISTRICT OF COLUMBIA CIRCUIT

WHITE STALLION ENERGY LLC, *et al.*,	)	No. 12-1100 (and consolidated cases)
Petitioners.	)	
v.	)	
U.S. ENVIRONMENTAL PROTECTION AGENCY,	)	
Respondent.	)	

Suffolk County	)
	)
Commonwealth of Massachusetts	)

DECLARATION OF JONATHAN I. LEVY, SC.D.
BOSTON UNIVERSITY SCHOOL OF PUBLIC HEALTH

I, Jonathan I. Levy, state and declare as follows:

I. Purpose of this Declaration

1. I provide this declaration in support of the Joint Motion of the American Academy of Pediatrics, American Lung Association, American Nurses Association, American Public Health Association, Chesapeake Bay Foundation, Citizens for Pennsylvania's Future, Clean Air Council, Conservation Law Foundation, Environment America, Environmental Defense Fund, Izaak Walton

League of America, National Association for the Advancement of Colored People, Natural Resources Council of Maine, Natural Resources Defense Council, Ohio Environmental Council, Physicians for Social Responsibility, Sierra Club, and Waterkeeper Alliance; and the states of California, Connecticut, Delaware, Illinois, Iowa, New York, North Carolina, Oregon, Maine, Maryland, Minnesota, New Hampshire, New Mexico, Rhode Island and Vermont; the Commonwealth of Massachusetts; the Cities of Baltimore, Chicago, New York, the District of Columbia, and Erie County, New York. The Motion requests that the Court retain in place the effectiveness of the emissions limits contained in the Mercury and Air Toxics Standards ("Air Toxics Rule"), published at 77 Fed. Reg. 9304 (February 12, 2012), during the period when a portion of the rule is remanded to the Agency, because doing so will preserve the significant public health benefits associated with EPA's regulations.

2. I provide this declaration based on my professional experience, as outlined in Section II, which included my review of EPA's methodology for assessing and quantifying health benefits from air pollution controls as a member of U.S. EPA's Advisory Council on Clean Air Compliance Analysis. Furthermore, in preparing this declaration I reviewed the Air Toxics Rule's required emissions limitations, specifically those sections of the Rule discussing the alternative particulate matter limits imposed by the Rule, and the Agency's use of particulate

matter as a surrogate for the non-mercury metallic hazardous air pollutants, which I understand include arsenic, beryllium, cadmium, chromium, cobalt, manganese, nickel and lead among other metals. I also reviewed sections of EPA's Regulatory Impact Analysis (RIA) accompanying the final Rule's publication and discussing the Agency's methods for assessing the health benefits associated with controlling the power plant pollution regulated by the Air Toxics Rule.

II. Experience and Qualifications

3. I am currently a Professor and Associate Chair in the Department of Environmental Health at the Boston University School of Public Health, where I have been a Professor of Environmental Health since 2010. I am also an Adjunct Professor at the Harvard T.H. Chan School of Public Health in the Department of Environmental Health, having served as an Assistant Professor from 2001-2006 and an Associate Professor from 2006-2010. I hold a Doctor of Science (Sc.D.) degree from the Harvard T.H. Chan School of Public Health, where my dissertation was on "Environmental Health Effects of Energy Use: A Damage Function Approach," and a Bachelor of Arts (B.A.) from Harvard College in Applied Mathematics, Decision and Control.

4. I have researched and published extensively on the relationship between exposure to air pollutants and human health effects, including developing models of exposures from power plants and other sources using atmospheric

dispersion models, quantifying the public health impacts associated with these exposures, and assessing the public health benefits of limiting emissions of particulate matter and other power plant air pollution. Among my publications relevant to this declaration are studies in which I quantified the health damages associated with particulate matter ($PM_{2.5}$), sulfur dioxide (SO_2), and nitrogen oxide (NOx) emissions from power plants in different parts of the country. [1,2,3,4,5,6] I have also published multiple articles evaluating the association between criteria air

[1] Levy JI, Spengler JD. Modeling the benefits of power plant emission controls in Massachusetts. J Air Waste Manage Assoc 52: 5-18 (2002).

[2] Levy JI, Spengler JD, Hlinka D, Sullivan D, Moon D. Using CALPUFF to evaluate the impacts of power plant emissions in Illinois: Model sensitivity and implications. Atmos Environ 36: 1063-1075 (2002).

[3] Levy JI, Greco SL, Spengler JD. The importance of population susceptibility for air pollution risk assessment: A case study of power plants near Washington, DC. Environ Health Perspect 110: 1253-1260 (2002).

[4] Levy JI, Wilson AM, Zwack LM. Quantifying the efficiency and equity implications of power plant air pollution control strategies in the United States. Environ Health Perspect 115: 740-750 (2007).

[5] Levy JI, Baxter LK, Schwartz J. Uncertainty and variability in environmental externalities from coal-fired power plants in the United States. Risk Anal 29: 1000-1014 (2009).

[6] Buonocore JJ, Dong X, Spengler JD, Fu JS, Levy JI. Using the Community Multiscale Air Quality (CMAQ) model to estimate public health impacts of $PM_{2.5}$ from individual power plants. Environ Int 68: 200-208 (2014).

pollutants and health outcomes, [7,8] including a study on the differential toxicity of major fine particulate matter constituents. [9] I also investigate and have published articles on the cumulative impact of various hazardous air pollutants on health endpoints. [10,11]

5. Among my professional service appointments, I was a member of U.S. EPA's Advisory Council on Clean Air Compliance Analysis from 2009-2014, a member of the National Research Council/Institute of Medicine Committee to Develop a Framework and Guidance for Health Impact Assessment from 2009-2011, and a member of the National Research Council Committee on Improving Risk Analysis Methods Used by U.S. EPA from 2006-2008. As part of my

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[7] Levy JI, Chemerynski SM, Sarnat JA. Ozone exposure and mortality: An empiric Bayes metaregression analysis. Epidemiology 16: 458-468 (2005).

[8] Levy JI, Hammitt JK, Spengler JD. Estimating the mortality impacts of particulate matter: What can be learned from between-study variability? Environ Health Perspect 108: 109-117 (2000).

[9] Levy JI, Diez D, Dou Y, Barr CD, Dominici F. A meta-analysis and multi-site time-series analysis of the differential toxicity of major fine particulate matter constituents. Am J Epidemiol 175: 1091-1099 (2012).

[10] Peters JL, Fabian MP, Levy JI. Combined impact of lead, cadmium, polychlorinated biphenyls and non-chemical risk factors on blood pressure in NHANES. Environ Res 132: 93-99 (2014).

[11] Loh MM, Levy JI, Spengler JD, Houseman EA, Bennett DH. Ranking cancer risks of organic hazardous air pollutants in the United States. Environ Health Perspect 115: 1160-1168 (2007).

membership on the Advisory Council on Clean Air Compliance Analysis, I was part of the Health Effects Subcommittee (HES), which reviewed EPA's approach for modeling the health effects associated with reductions in $PM_{2.5}$ concentrations. In general, I have served as a peer reviewer and scientific advisor of various health benefits modeling studies by U.S. EPA and other organizations since 2000.

6. A current copy of my curriculum vitae is attached to my declaration as Appendix A.

III. Primary and Secondary Particulate Matter Formation

7. When evaluating the health benefits of emissions control strategies for power plants, it is important to incorporate both primary and secondary particulate matter. Primary particulate matter consists of particles directly emitted from a source, often subdivided into filterable and condensable particles. Filterable particles are emitted in particle form and can typically be captured on a filter, whereas condensable particles are emitted in the gas phase but quickly convert to particle form when cooled. Primary particulate matter therefore consists of a number of chemicals, including but not limited to metals, organics, and acids. In contrast, secondary particulate matter is formed through chemical reactions in the atmosphere. For example, gaseous SO_2 and NOx emissions are converted to particulate matter through reactions with ambient ammonium, in a process influenced by temperature, atmospheric ozone, and other factors. Ambient fine

particulate matter concentrations are therefore a blend of primarily-emitted and secondarily-formed constituents.

8. Based on my experience and research, I understand that primary particulate matter emitted by power plants includes multiple toxic metals, such as arsenic, beryllium, cadmium, chromium, cobalt, manganese, nickel and lead. Secondary particulate matter consists primarily of sulfate, nitrate, ammonium, and secondary organic aerosols.

9. I am aware of current scientific research and analysis directed at assessing the health effects associated with individual constituents of particulate matter air pollution, including my own 2012 publication on the topic cited above in note 9. While individual studies have analyzed the health effects associated with various particle constituents, my understanding of the state of that scientific work is that it has not currently progressed to the point at which it is possible to synthesize the literature and develop concentration-response functions for the specific non-mercury toxic metal constituents of particulates, as opposed to the health effects of the mixture of constituents found in ambient fine particulate matter.

IV. U.S. EPA's Air Toxics Rule

10. I am aware that U.S. EPA's Air Toxics Rule sets emissions limits for the non-mercury toxic metals emitted by power plants. The Rule sets either non-

mercury metal toxic-specific emissions limits or filterable particulate matter emissions limits as a surrogate for total toxic non-mercury metal emissions, for each power plant unit. EPA set the emissions limits based on the performance of the best performing similar source (for new sources), or the top twelve percent of sources (for existing sources) at the time the standards were set, and providing for the variability of the input fuel constituents.

11. I understand that U.S. EPA chose to set standards for particulate matter as an alternative to non-mercury toxic metal specific standards because the non-mercury toxic metal constituents are invariably present in the particulate matter emissions from power plants, and because the Agency found that these pollutants can be controlled using particulate matter controls. 76 Fed. Reg. 24976, 25038 (May 3, 2011).

12. I understand that U.S. EPA estimates that the Air Toxics Rule will decrease emissions from coal-fired power plants (greater than 25 MW) of fine particulate matter by 52,000 tons per year, and will decrease emissions of SO_2 by 1.4 million tons per year. 77 Fed. Reg. 9304, 9424 (Feb. 12, 2012).

V. EPA's Assessment of the Health Benefits of the Particulate Matter Limits Set by the Mercury and Air Toxics Standards

13. In its RIA, U.S. EPA estimates the annual health benefits of the particulate matter concentration reductions associated with the Air Toxics Rule

following the well-understood health damage function approach. As described by U.S. EPA and throughout the peer-reviewed literature, [12,13] health benefits are calculated as a function of the baseline incidence rate for the health outcome in question, the number of exposed individuals, the change in air pollution levels to which the population is exposed, and a concentration-response function linking changes in air pollution with health outcomes. The underlying equations are widely accepted, and the fidelity of the calculations therefore depends on the fidelity of the input variables. As the number of exposed individuals is readily determined from Census data and baseline incidence rates are characterized from multiple well-regarded surveillance databases, the focus of any evaluation of health damage function modeling is generally on the air pollution modeling and concentration-response functions applied.

14. U.S. EPA evaluated the health benefits of the Air Toxics Rule by applying adjusted versions of the health damage functions (benefit-per-ton values)

[12] Chestnut LG, Mills DM, Cohan DS. Cost-benefit analysis in the selection of efficient multipollutant strategies. J Air Waste Manag Assoc 56: 530-536 (2006).

[13] Fann N, Lamson AD, Anenberg SC, Wesson K, Risley D, Hubbell BJ. Estimating the national public health burden associated with exposure to ambient $PM_{2.5}$ and ozone. Risk Anal 32: 81-95 (2012).

derived in Fann *et al*. 2009. [14] I have read this scientific publication and am familiar with the approach utilized within the study. For air pollution modeling, Fann *et al.* used a response surface model derived from the Community Multiscale Air Quality (CMAQ) model. CMAQ is a state-of-the-science model with the capacity to model both primary particulate matter and secondary particulate matter, and is the most appropriate atmospheric chemistry-transport model for this application. To estimate health damages, Fann *et al.* relied on a synthesis of the epidemiological literature linking $PM_{2.5}$ concentrations with both mortality and morbidity effects. The epidemiological studies utilized are consistent with the studies that U.S. EPA used when I was a member of the Advisory Council on Clean Air Compliance Analysis, and Fann *et al.* applied these studies appropriately. Based on my experience, this methodology for assessing the health benefits of the Air Toxics Rule is a well-established approach that is consistent with best practice in the scientific literature.

15. U.S. EPA evaluated the health benefits of the Air Toxics Rule with inclusion of both primarily emitted particulate matter and precursors for secondarily formed particulate matter (principally SO_2). Again, EPA's Rule regulates particulate matter as a surrogate for the non-mercury metal toxics emitted

[14] Fann N, Fulcher CM, Hubbell BJ, The influence of location, source, and emission type in estimates of the human health benefits of reducing a ton of air pollution, Air Qual Atmos Health 2: 169-176 (2009).

with and on the particulate matter. Inclusion of both forms of particulate matter is appropriate and represents standard practice for health benefits analysis.

16. U.S. EPA's estimates were that the Air Toxics Rule will annually result in between 4200-11,000 reduced incidences of premature mortality; 2800 fewer cases of chronic bronchitis; 4700 fewer non-fatal heart attacks; 830 fewer hospital admissions for respiratory symptoms; 1800 fewer hospital admissions for cardiovascular symptoms; 3100 fewer emergency room visits by children under age 18 for asthma symptoms; 6300 fewer cases of acute bronchitis in children between the ages of 8 and 12; 80,000 fewer cases of lower respiratory symptoms in children between the ages of 7 and 14; 60,000 fewer cases of upper respiratory symptoms in asthmatic children between the ages of 9 and 18; 130,000 fewer cases of exacerbated asthma in children between the ages of 6 and 18; 540,000 fewer lost work days; and 3,200,000 fewer minor restricted activity days in adults. U.S. EPA also reported that 95% of these health benefits would be associated with secondary sulfate formation, related to SO_2 emissions. These estimates by U.S. EPA are consistent with values in previous RIAs and within the peer-reviewed literature.

VI. The Potential Effects of Staying or Otherwise Failing to Implement the Air Toxics Rule.

17. I understand that the Air Toxics Rule was to be implemented at existing power plants in April 2015, but that some power plants have been granted one year extensions to put on controls or shut down, to April 2016.

18. I understand that certain parties may seek to stay the effectiveness of the emissions limits under the Air Toxics Rule, including the particulate matter and SO_2 emissions limits included under the Rule, or to strip those protections completely, during the period of time when EPA fixes a problem with the initial decision whether to regulate air toxics emissions from the power sector.

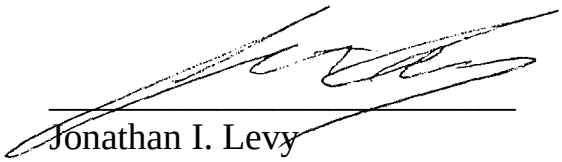
19. I understand that if the Rule is stayed, power plants that have received extensions might not be required to comply by April 2016. Additionally those plants that have put on controls to comply with the Rule's emissions limits by the initial April 2015 deadline might not be required to comply with the Rule's emissions limits during the period if the Rule were stayed or otherwise blocked.

20. Based on my understanding of power plant health impact assessment science and modelling, it is clear to me that if emissions remain uncontrolled, so that tonnage reductions are not achieved during any period in which the Air Toxics Rule is not in effect, there will be direct health impacts experienced by the population exposed to particulates that would otherwise not be emitted to the ambient air, or formed as secondary particulates after the emission of SO_2. Most of

the health outcomes quantified in U.S. EPA's RIA of the Air Toxics Rule are based on short-term exposure changes, so that health effects would be exhibited within a matter of days after air pollution levels increased (or failed to decrease). For the premature mortality estimates provided by U.S. EPA, which are based on long-term exposures, the scientific literature shows that health effects are exhibited within 1-2 years of a change in concentrations. [15] Those adverse health effects will persist for as long as particulate matter and SO_2 pollution controls are not in place and operating at the power plants, and will be reduced when the emissions of particles and SO_2 are curtailed.

I declare under the penalty of perjury under the laws of the United States, that to the best of my knowledge, the foregoing is true and correct.

Executed on September 21, 2015, at Boston, Massachusetts.



Jonathan I. Levy

[15] Schwartz J, Coull B, Laden F, Ryan L. The effect of dose and timing of dose on the association between airborne particles and survival. Environ Health Perspect 116:64–69 (2008).

EXHIBIT 9

To Comments of Public Health and Environmental Groups
on Docket ID No. EPA-HQ-OAR-2009-0234
80 Fed. Reg. 75,025 (Dec. 1, 2015)

Texas Commission on Environmental Quality
"Findings of Fact and Conclusions of Law Approving
Preconstruction Permit for White Stallion Energy Center"
(Oct. 19, 2010)

TEXAS COMMISSION ON ENVIRONMENTAL QUALITY

On September 29, 2010, the Texas Commission on Environmental Quality (TCEQ or Commission) considered the application of White Stallion Energy Center LLC for Air Quality Permit Nos. 86088, HAP28, PAL26, and PSD-TX-1160. A Proposal for Decision (PFD) was presented by Paul Keeper and Kerrie Jo Qualtrough, Administrative Law Judges (ALJs) with the State Office of Administrative Hearings (SOAH), who conducted a contested case hearing in this case from February 10 through 18, 2010, in Austin, Texas.

After considering the ALJs' PFD, the Commission adopts the following Findings of Fact and Conclusions of Law:

I. FINDINGS OF FACT

Proposed Facility

1. On September 5, 2008, White Stallion Energy Center, LLC (WSEC) filed an application with the Texas Commission on Environmental Quality (TCEQ or Commission) for a permit (Permit) to construct and operate a new 1,200 net megawatt (MW) electric generation plant in Matagorda County, Texas.
2. There are no schools located within 3,000 feet of the proposed WSEC site.
3. WSEC proposes to construct and operate a new steam-electric utility generating facility using four circulating fluidized bed (CFB) boilers. A CFB boiler relies on high pressure air to improve combustion as the fuel moves across a surface of limestone.

4. WSEC proposes four water-cooled cooling towers, each with a cooling water circulation design of 161,000 gallons per minute.

5. The fuel and the limestone for the CFB beds will be delivered by barge, rail, or truck. The materials will be transported from the delivery site by partially enclosed conveyors to large stockpiles for storage. Activated carbon for mercury control, lime for sulfur dioxide (SO_2) control, and sand for CFB bed stabilization will be delivered by railcar or truck. The fly ash and boiler bottom ash solid wastes will be stored in silos near the boilers, loaded into trucks, and sent to an on-site landfill.

6. Emission control technologies will include selective non-catalytic reduction (SNCR), dry flue gas desulfurization (FGD), fabric filter baghouse, activated carbon injection, and good combustion practices.

7. The project will include seven liquid fuel storage tanks, a tank for the storage of acid for water treatment, and pressurized storage tanks for ammonia to be used for the control of nitrogen oxides (NO_x).

8. Combustion-type support facilities include two 2,800 kilowatt (kW) emergency electric generation engines and one 250 horsepower fire water-pump engine. Each engine will be limited to operate no more than 500 hours per year.

9. WSEC evaluated several alternative technologies, including integrated gasification combined cycle (IGCC) technology and pulverized coal (PC) boilers, before selecting CFB boilers fired by petroleum coke (pet coke) and coal as the appropriate means to meet its business objectives.

10. For the proposed WSEC facility, each CFB boiler will have a design maximum heat input of 3,300 million British thermal units per hour (MMBtu/hr). The gross electric output of the four generators will be about 1,320 MW. The net output—about 1,200 MW—is the difference between the power generated and the power required to operate the facility.

11. In addition to the CFB boilers, WSEC's ancillary equipment includes two diesel-fired emergency generators to provide electricity to WSEC in case of power failure; a diesel-fired pump engine to provide water in the event of a fire; various tanks to store ammonia for the SNCR system, acid for water-conditioning and pH control, No. 2 fuel oil for CFB boiler startup, and fuel for motor vehicles associated with the plant; cooling towers; and equipment associated with the receipt, handling, storage and processing of pet coke, coal, limestone, lime, activated carbon, sand and combustion by-products.

12. The proposed fuels are bituminous coal from the Illinois Basin and pet coke, a carbonaceous, high-ash byproduct of oil refining with a high heat content.

13. Low-sulfur distillate fuel oil or coal are proposed as the CFB startup fuels.

14. On September 11, 2008, the Executive Director (ED) declared the application administratively complete.

15. On October 1, 2008, WSEC published a Notice of Receipt of Application and Intent to Obtain Air Permit.

16. Between December 22, 2008, and February 16, 2009, WSEC supplemented the application.

17. On March 9, 2009, the Commission referred the matter to the State Office of Administrative Hearings (SOAH) to conduct a contested case hearing and to issue a proposal for decision (PFD).

18. On March 13, 2009, the ED concluded that the application was technically complete, issued a draft permit, and recommended that the application be approved.

19. On March 15, 2009, WSEC published a combined Notice of Application and Preliminary Decision, Notice of Public Meeting, and Notice of Hearing. On March 30, 2009, a public meeting was held in Bay City, Texas.

20. By letter dated April 14, 2009, the U.S. Environmental Protection Agency (EPA) Region 6 submitted to TCEQ comments on the Draft Permit in which it: (1) recommended that TCEQ consider requiring continuous emission monitoring systems (CEMS) for particulate matter emissions; (2) asked TCEQ to reconcile a permit condition stating that compliance with the Plant-wide Applicability Limits (PAL) will be demonstrated with CEMS and the fact that PM CEMS were not required by the draft permit; (3) notified TCEQ that EPA was currently reviewing TCEQ's PAL rules and had not yet taken action to approve or disapprove them as part of Texas's State Implementation Plan (SIP); (4) asked TCEQ to request that WSEC forward to EPA Region 6 a final copy of the Startup/Shutdown written plan, when prepared; and (5) expressed concern about TCEQ's guidance for evaluating ozone impacts, and asked TCEQ to provide to EPA Region 6 photochemical modeling demonstrating what the effect of WSEC's emissions would be on specific ozone monitors in the Houston area.

21. On April 20, 2009, Administrative Law Judge (ALJ) Paul Keeper convened a preliminary hearing in Bay City, Texas. No party contested either notice or jurisdiction and jurisdiction was established.

22. At the preliminary hearing, the ALJ granted party status to the Environmental Defense Fund (EDF), the Sierra Club (SC), and the No Coal Coalition (NCC). SC and NCC shared counsel, and the ALJ treated SC and NCC as a single party for administrative purposes.

23. On October 2, 2009, the ED issued responses to public comments and a revised draft permit.

24. On February 10, 2010, ALJs Keeper and Kerrie Jo Qualtrough convened the hearing on the merits. On February 18, 2010, the ALJs adjourned the hearing.

25. Representatives of the parties at the hearing were:

Party	Status	Counsel
WSEC	Applicant	Eric Groten and Patrick Lee
EDF	Protestant	Tom Weber and Paul Tough
SC/NCC	Protestant	Layla Mansuri and Christina Mann
OPIC	Statutory	Scott Humphrey
ED	Statutory	Booker Harrison and Ben Rhem

26. The parties filed written closing arguments and briefs, responses, and proposed findings of fact and conclusions of law. On May 5, 2010, the ALJs closed the administrative record.

Completeness of the Application

27. WSEC's Application is for an air quality permit that would also satisfy the permitting requirements for PSD, case-by-case Maximum Achievable Control Technology (MACT), and PAL permitting requirements.

28. TCEQ assigned the Draft Permit State Air Quality Permit No. 86088, HAP Permit No. 28, PAL Permit No. 26, and PSD Permit No. PSD-TX-1160.

29. WSEC's application includes a complete Form PI-1 General Application signed by Randy Bird, an authorized WSEC representative. The application was also signed and sealed by Shanon DiSorbo, a Texas registered professional engineer.

30. WSEC paid the $75,000 permit fee.

31. WSEC provided all supplemental information required by TCEQ's PI-1 Form.

32. WSEC's Application addresses all sources of air emissions from WSEC that are subject to permitting under TCEQ's rules.

33. WSEC's Application includes a list of all facilities to be included in the PAL and their potential to emit and expected maximum capacity. The calculation procedures to be used to determine monthly and 12-month rolling emissions, and the monitoring and recordkeeping to be used to meet the requirements of 30 TEX. ADMIN. CODE (TAC) § 116.186, are also included in the Application.

34. The ED reviewed WSEC's Application to determine whether it complied with all applicable rules and policies and documented the conclusions of that review in an internal report called the "Construction Permit Source Analysis & Technical Review."

Emissions

35. WSEC's facility may emit NO_X, carbon monoxide (CO), SO_2, particulate matter (PM), including PM_{10} and $PM_{2.5}$, volatile organic compounds (VOC), lead, sulfuric acid (H_2SO_4), hydrogen fluoride (HF), ammonia (NH_3), hydrogen chloride (HCl), and mercury (Hg).

Location

36. The proposed facility will be located in Matagorda County, Texas. Matagorda County currently attains all national ambient air quality standards (NAAQS).

37. Matagorda County lies immediately to the southwest of Brazoria County, the southernmost county included in the Houston-Galveston-Brazoria nonattainment area, which is designated as severe nonattainment for ozone.

30 TAC § 116.111(a)(2)(A): Protection of public health and welfare

38. WSEC performed atmospheric dispersion modeling to demonstrate that emissions from WSEC will be protective of public health and welfare.

39. Atmospheric dispersion modeling is the use of the scientific principles of atmospheric dispersion, embodied in a computerized mathematical model, to predict the maximum concentrations of emissions released from a source in the downwind ambient air.

40. WSEC used the American Meteorological Society/Environmental Protection Agency Regulatory Model, or "AERMOD," Version 07026. AERMOD is the latest generation of atmospheric dispersion models suitable for industrial sources, and is the model recommended by TCEQ.

41. TCEQ Staff performed an audit of the modeling report submitted by WSEC and determined that the modeling performed was acceptable for all types of regulatory review and for all pollutants.

42. WSEC modeled all emission sources associated with WSEC, but did not model road dust emissions.

43. TCEQ's modeling guidance explains the difficulties of accurately modeling road dust emissions, noting that "[c]ombined with worst-case operating scenarios, the modeling tool will overpredict concentrations, particularly in the vicinity of the source, and may incorrectly identify road emissions as the major cause of air pollution at a site."

44. WSEC's application contemplates the use of barges for fuel and limestone delivery and the associated barge unloading activity must be included as an emission source. The barge unloading area is an area about 80 meters long and 30 meters wide.

45. The Commission's Air Quality Modeling Guidelines require the placement of receptors along property lines where possible and appropriate.

46. Barges would arrive at WSEC's proposed barge unloading facility on the Colorado River. Their cargo would be lifted and placed on a hopper and then moved by conveyors to storage sites. For each of these unloading events, WSEC modeled the associated emissions.

47. WSEC measured emissions from a 25-meter buffer zone surrounding the barges as they are unloading.

48. The TCEQ's Dockside Guidance Document's "set distance" approach for analyzing off-site receptors over water requires the placement of receptors beginning at a distance of 25 meters from the edge of the source instead of on the actual property line.

49. WSEC made proper use of the Dockside Guidance Document in relying on a 25-meter buffer zone.

50. Modeling of road dust emissions for averaging periods less than annual is not necessary.

51. Modeling of road dust emissions for an annual averaging period is not necessary if the emissions will not be generated in association with the transport, storage, or transfer of road-base aggregate materials, and the applicant plans to use best management practices to control any road dust emissions.

52. WSEC will be transporting no aggregate materials at WSEC site and will be required to use best management practices for minimizing dust, such as paving and cleaning all permanent plant roads.

53. WSEC assumed that the worst-case meteorological conditions for dispersion would occur simultaneously with the worst-case emissions scenario.

54. WSEC's modeling assumed that all emissions sources at WSEC would be operating simultaneously.

NAAQS Analysis

55. NAAQS are set by EPA and represent ambient concentrations at which no adverse health or welfare impacts are expected to occur.

56. EPA has set both primary and secondary NAAQS.

57. Primary or "health-based" NAAQS are set to protect the health of even the most sensitive individuals with an adequate margin of safety. Sensitive individuals include children, the elderly, and people with a pre-existing medical condition.

58. Secondary or "welfare-based" NAAQS are set to protect against welfare effects such as decreased visibility, effects on climate, effects on crops and other vegetation, effects on wildlife, and effects on the economy.

59. EPA has established primary and secondary NAAQS for six pollutants, referred to as the "criteria" pollutants: SO_2, two different size categories of particulate matter (PM_{10}, consisting of particles with diameters less than 10 microns, and $PM_{2.5}$, consisting of particles with diameters less than 2.5 microns), ozone, nitrogen dioxide (NO_2), carbon monoxide (CO), and lead.

60. WSEC directly modeled its emissions of SO_2, NO_2, CO, lead and PM_{10} for the purpose of demonstrating compliance with the NAAQS.

61. An applicant is not required to evaluate background concentrations of a particular criteria pollutant if the maximum modeled concentration of that pollutant is below the corresponding NAAQS de minimis level, in which case it is appropriate to conclude that

the source's emissions of that pollutant will not cause any adverse health or welfare effects.

62. WSEC's modeling showed maximum concentrations exceeding the NAAQS *de minimis* levels for SO_2, NO_2 and PM_{10}. For these pollutants, WSEC considered the influence of other sources in the area by modeling non-WSEC emissions along with WSEC emissions, and also adding a conservative ambient background concentration to the modeled results.

SO_2 NAAQS

63. SO_2 NAAQS exist for three averaging periods: 3-hour (1,300 $\mu g/m^3$), 24-hour (365 $\mu g/m^3$), and annual (80 $\mu g/m^3$).

3-hour SO_2 NAAQS

64. The maximum modeled 3-hour average SO_2 concentration resulting from WSEC's emissions and emissions from other sources in the area was 504.9 $\mu g/m^3$.

65. The maximum modeled 3-hour average SO_2 concentration resulting from WSEC's emissions and emissions from other sources in the area, and incorporation of a conservative background concentration, was 566 $\mu g/m^3$.

66. WSEC's emissions will not cause or contribute to an exceedance of the 3-hour SO_2 NAAQS of 1,300 $\mu g/m^3$.

24-hour SO_2 NAAQS

67. The maximum modeled 24-hour average SO_2 concentration resulting from WSEC's emissions and emissions from other sources in the area was 79.3 $\mu g/m^3$.

68. The maximum modeled 24-hour average SO_2 concentration resulting from WSEC's emissions and emissions from other sources in the area, and incorporation of a conservative background concentration, was 109 $\mu g/m^3$.

69. WSEC's emissions will not cause or contribute to an exceedance of the 24-hour SO_2 NAAQS of 365 $\mu g/m^3$.

Annual SO_2 NAAQS

70. The maximum modeled annual average SO_2 concentration resulting from WSEC's emissions and emissions from other sources in the area was 6.7 $\mu g/m^3$.

71. The maximum modeled annual average SO_2 concentration resulting from WSEC's emissions and emissions from other sources in the area, and incorporation of a conservative background concentration, was 12 $\mu g/m^3$.

72. WSEC's emissions will not cause or contribute to an exceedance of the 24-hour SO_2 NAAQS of 80 $\mu g/m^3$.

NO_2 NAAQS

73. NO_2 NAAQS exist for two averaging periods: 1-hour (100 parts per billion) and annual (100 $\mu g/m^3$).

1-hour NO_2 NAAQS

74. EPA published the 1-hour NO_2 NAAQS in the Federal Register on February 9, 2010. It became effective on April 12, 2010.

75. Rules setting forth how the 1-hour NO_2 NAAQS should be implemented, including what significant impact level should be used in evaluating 1-hour NO_2 concentrations, have not yet been established.

76. The places that are most likely to have elevated short-term NO_2 levels are near heavily travelled roadways in urbanized areas, not in rural areas such as WSEC site in Matagorda County.

77. WSEC will be located on a large tract of land, in a rural setting with no nearby heavily traveled highways.

78. WSEC's emissions will not cause or contribute to an exceedance of the 1-hour NO_2 NAAQS of 100 parts per billion.

Annual NO_2 NAAQS

79. The maximum modeled annual average NO_2 concentration resulting from WSEC's emissions and emissions from other sources in the area was 49.9 $\mu g/m^3$.

80. The maximum modeled annual average NO_2 concentration resulting from WSEC's emissions and emissions from other sources in the area, and incorporation of a conservative background concentration, was 60 $\mu g/m^3$.

81. WSEC's emissions will not cause or contribute to an exceedance of the annual average NO_2 NAAQS of 100 $\mu g/m^3$.

CO NAAQS

82. CO NAAQS exist for two averaging periods: 1-hour (40,000 $\mu g/m^3$) and 8-hour (10,000 $\mu g/m^3$).

1-hour CO NAAQS

83. The maximum modeled 1-hour average CO concentration resulting from WSEC's emissions is 326.4 $\mu g/m^3$, which was less than the modeling de minimis level of 2,000 $\mu g/m3$.

84. WSEC's emissions will not cause or contribute to an exceedance of the 1-hour CO NAAQS of 40,000 $\mu g/m^3$.

8-hour CO NAAQS

85. The maximum modeled 8-hour average CO concentration resulting from WSEC's emissions is 177.5 $\mu g/m^3$, which was less than the modeling de minimis level of 500 $\mu g/m^3$.

86. WSEC's emissions will not cause or contribute to an exceedance of the 8-hour CO NAAQS of 10,000 $\mu g/m^3$.

Lead NAAQS

87. Lead NAAQS exist for one averaging period, 3-month (0.15 $\mu g/m^3$).

88. The maximum modeled 3-month average lead concentration resulting from WSEC's emissions was 0.00049 $\mu g/m^3$.

89. The maximum modeled 3-month average lead concentration resulting from WSEC's emissions and incorporation of a conservative background concentration was 0.10049 $\mu g/m^3$.

90. WSEC's emissions will not cause or contribute to an exceedance of the 3-month lead NAAQS of 0.15 $\mu g/m^3$.

PM_{10} NAAQS

91. PM_{10} NAAQS exist for two averaging periods: 24-hour (150 $\mu g/m^3$) and annual (50 $\mu g/m^3$).

24-hour PM_{10} NAAQS

92. The maximum modeled 24-hour average PM_{10} concentration resulting from WSEC's emissions and emissions from other sources in the area was 28.2 $\mu g/m^3$.

93. The maximum modeled 24-hour average PM_{10} concentration resulting from WSEC's emissions and emissions from other sources in the area, and incorporation of a conservative background concentration, was 79 $\mu g/m^3$.

94. WSEC's emissions will not cause or contribute to an exceedance of the 24-hour PM_{10} NAAQS of 150 $\mu g/m^3$.

Annual PM_{10} NAAQS

95. The maximum modeled annual average PM_{10} concentration resulting from WSEC's emissions and emissions from other sources in the area was 6.2 $\mu g/m^3$.

96. The maximum modeled annual average PM_{10} concentration resulting from WSEC's emissions and emissions from other sources in the area, and incorporation of a conservative background concentration, was 30 $\mu g/m^3$.

97. WSEC's emissions will not cause or contribute to an exceedance of the annual average PM_{10} NAAQS of 50 $\mu g/m^3$.

$PM_{2.5}$ NAAQS

98. Demonstration of compliance with the PM_{10} NAAQS is sufficient to demonstrate compliance with the $PM_{2.5}$ NAAQS.

99. Although it was not required, WSEC modeled anticipated $PM_{2.5}$ emissions.

100. WSEC's analysis independently demonstrated that the $PM_{2.5}$ emissions were only about 10% of the PM_{10} emissions.

101. WSEC's emissions will not cause or contribute to an exceedance of the $PM_{2.5}$ NAAQS.

Ozone NAAQS

102. Ozone is one of the criteria pollutants for which EPA has set a NAAQS.

103. TCEQ does not require an applicant to numerically quantify the amount of ozone that its emissions will produce.

104. To evaluate a source's potential ozone impacts, TCEQ requires applicants to perform an evaluation technique set forth in written guidance.

105. The written guidance requires the applicant to determine whether the ozone NAAQS is already being exceeded in the area of the plant.

106. If the ozone NAAQS is not being exceeded, then the project's potential to cause a significant change to the ozone levels in the area is evaluated based on the methane-normalized VOC-to-NO_x ratio of its emissions.

107. White Stallion relied on TCEQ's ozone evaluation technique set forth in written guidance to evaluate potential ozone impacts.

108. WSEC relied on ozone monitor data from a monitor site at Aransas Pass in San Patricio County because no ambient ozone monitoring data was available for Matagorda County.

109. Using the Aransas Pass monitoring data, WSEC determined that Matagorda County would have an average ozone concentration no higher than 74.7 ppb, less than the EPA-adopted 8-hour ozone standard of 75 ppb.

110. The Aransas Pass monitor data was taken from a Commission-maintained web site. The Aransas Pass monitor data was subject to a footnote explaining that the data "does not meet EPA quality assurance criteria and cannot be used for regulatory purposes." The purpose of this notation is to denote that the data from that monitor should not be used to make area-wide attainment designations.

111. The Aransas Pass monitor data may be used for completing Step 1 of TCEQ's ozone evaluation technique.

111.a White Stallion also satisfied Step 1 of TCEQ's ozone evaluation technique with ozone data from the Corpus Christi West and Corpus Christi Tuloso monitors, both of which are located in a more populated and heavily industrialized area than the area surrounding the WSEC site, and neither of which is marked with any limits on use.

112. WSEC demonstrated compliance with TCEQ's ozone evaluation technique, and established that its emissions of ozone precursors will have no significant impact on ambient ozone concentrations.

112.a The WSEC's emissions will not cause or contribute to an exceedance of the ozone NAAQS.

State property line analysis

113. State property line standards, also called "Chapter 112 standards" or "NGLC standards," are maximum allowable concentrations resulting from emissions originating within a source's property line.

114. State property line standards are enforced only through actual measurement, but it is TCEQ's policy to require a preconstruction modeling demonstration that they are not likely to be exceeded.

115. WSEC will emit two substances for which state property line standards exist, SO_2 and sulfuric acid mist (H_2SO_4).

SO_2 state property line standard

116. An SO_2 state property line standard exists for one averaging period, 30 minutes (1,021 $\mu g/m^3$).

117. The maximum modeled 30-minute average SO_2 concentration resulting from WSEC's emissions was 351.9 $\mu g/m^3$.

118. WSEC's emissions will not cause an exceedance of the 30-minute average SO_2 state property line standard of 1,021 $\mu g/m^3$.

H_2SO_4 state property line standards

119. H_2SO_4 state property line standards exist for two averaging periods, 1-hour (50 $\mu g/m^3$) and 24-hour (15 $\mu g/m^3$).

1-hour H_2SO_4 state property line standard

120. The maximum modeled 1-hour average H_2SO_4 concentration resulting from WSEC's emissions was 27.4 $\mu g/m^3$.

121. WSEC's emissions will not cause an exceedance of the 1-hour average H_2SO_4 state property line standard of 50 $\mu g/m^3$.

24-hour H_2SO_4 state property line standard

122. The maximum modeled 24-hour average H_2SO_4 concentration resulting from WSEC's emissions was 6.2 $\mu g/m^3$.

123. WSEC's emissions will not cause an exceedance of the 24-hour average H_2SO_4 state property line standard of 15 $\mu g/m^3$.

State property line analysis summary

124. WSEC's emissions will not cause an exceedance of any state property line standard.

ESL analysis

125. To assist in evaluating the potential for adverse health or welfare effects from exposure to air contaminants for which no ambient standards exist, TCEQ has developed approximately 4,700 guideline levels called Effects Screening Levels (ESLs).

126. Some ESLs are based on health effects, while others are based on welfare effects including odor, nuisance, vegetation damage, or materials damage such as corrosion.

127. Health-based ESLs are set by TCEQ at levels lower than levels reported to produce adverse health effects, and are set to protect the general public, including sensitive subgroups such as children, the elderly, or people with existing respiratory conditions.

128. ESLs incorporate margins of safety to take into account even the most sensitive individual, typically using 1/100th of occupational health exposure limits for short-term ESLs and 1/1000th for long-term ESLs.

129. ESLs are typically lower, or more restrictive, than comparable guidelines established by the Environmental Protection Agency and other state air pollution control agencies.

130. If a modeled air concentration of a constituent is below the ESL, adverse effects are not expected. If an air concentration of a constituent is above the ESL, it is not indicative that an adverse effect will occur, but rather that further evaluation by a toxicologist is warranted.

131. Although there exist ESLs for certain substances such as hydrogen and carbon dioxide, no modeling of them is required because they are simple asphyxiants.

132. WSEC modeled expected emissions of the following substances for which no ambient standards exist: ammonia, aluminum, arsenic, beryllium, cadmium, calcium oxide, hydrogen chloride, chromium, copper, hydrogen fluoride, iron oxide, magnesium, manganese, mercury, nickel, potassium, selenium, silicon dioxide (silica), sodium, titanium, vanadium, gasoline, diesel, coal dust, pet coke, limestone, and calcium sulfate (gypsum).

133. WSEC did not model emissions of pollutants that would be present only in trace amounts, or pollutants for which modeling was not needed to conclude that the ESL will not be exceeded. ESLs are established for long-term (annual) and short-term (one-hour average) analysis. Exceedances of short-term ESLs are evaluated using various factors, including the number of hours and the frequency with which a predicted concentration exceeds the respective guideline.

134. A health effects review begins with a Tier I analysis. If no modeled concentration at any receptor exceeds the ESL for that pollutant, then the analysis ends.

135. If a modeled concentration at a receptor exceeds the ESL, then a Tier II examination is performed. In Tier II, if the modeled ESL exceedance occurs at an industrial receptor and the modeled concentration of the pollutant is more than twice the ESL, then a Tier III

analysis is performed. In Tier II, if the modeled ESL exceedance occurs at a non-industrial receptor, then a Tier III analysis is performed.

136. A Tier III analysis is a case-by-case review, taking into account various factors, including surrounding land use, type of toxic effects, magnitude of concentration, frequency of exceedance, and margins of safety.

Ammonia

137. The maximum modeled 1-hour average ammonia concentration resulting from WSEC's emissions was 47.5 $\mu g/m^3$, which is below the 1-hour ESL for ammonia of 170 $\mu g/m^3$.

138. The maximum modeled annual average ammonia concentration resulting from WSEC's emissions was 0.3 $\mu g/m^3$, which is below the annual average ESL for ammonia of 17 $\mu g/m^3$.

139. No adverse health or welfare effects will result from any emissions of ammonia from WSEC.

Aluminum

140. The maximum modeled 1-hour average aluminum concentration resulting from WSEC's emissions was 3.3 $\mu g/m^3$, which is below the 1-hour ESL for aluminum of 50 $\mu g/m^3$.

141. The maximum modeled annual average aluminum concentration resulting from WSEC's emissions was 0.098 $\mu g/m^3$, which is below the annual average ESL for aluminum of 5 $\mu g/m^3$.

142. No adverse health or welfare effects will result from any emissions of aluminum from WSEC.

Arsenic

143. The maximum modeled 1-hour average arsenic concentration resulting from WSEC's emissions was 0.018 $\mu g/m^3$, which is below the 1-hour ESL for arsenic of 0.1 $\mu g/m^3$.

144. The maximum modeled annual average arsenic concentration resulting from WSEC's emissions was 0.0001 $\mu g/m^3$, which is below the annual average ESL for arsenic of 0.01 $\mu g/m^3$.

145. No adverse health or welfare effects will result from any emissions of arsenic from WSEC.

Beryllium

146. The maximum modeled 1-hour average beryllium concentration resulting from WSEC's emissions was 0.003 $\mu g/m^3$, which is below the 1-hour ESL for beryllium of 0.02 $\mu g/m^3$.

147. The maximum modeled annual average beryllium concentration resulting from WSEC's emissions was 0.00003 $\mu g/m^3$, which is below the annual average ESL for beryllium of 0.002 $\mu g/m^3$.

148. No adverse health or welfare effects will result from any emissions of beryllium from WSEC.

Cadmium

149. The maximum modeled 1-hour average cadmium concentration resulting from WSEC's emissions was 0.001 $\mu g/m^3$, which is below the 1-hour ESL for cadmium of 0.1 $\mu g/m^3$.

150. The maximum modeled annual average cadmium concentration resulting from WSEC's emissions was 0.00001 $\mu g/m^3$, which is below the annual average ESL for cadmium of 0.01 $\mu g/m^3$.

151. No adverse health or welfare effects will result from any emissions of cadmium from WSEC.

Calcium oxide

152. The maximum modeled 1-hour average calcium oxide concentration resulting from WSEC's emissions was 2.85 $\mu g/m^3$, which is below the 1-hour ESL for calcium oxide of 20 $\mu g/m^3$.

153. The maximum modeled annual average calcium oxide concentration resulting from WSEC's emissions was 0.18 $\mu g/m^3$, which is below the annual average ESL for calcium oxide of 2 $\mu g/m^3$.

154. No adverse health or welfare effects will result from any emissions of calcium oxide from WSEC.

Hydrogen chloride

155. The maximum modeled 1-hour average hydrogen chloride concentration resulting from WSEC's emissions was 82 $\mu g/m^3$, which is below the 1-hour ESL for hydrogen chloride of 190 $\mu g/m^3$.

156. The maximum modeled annual average hydrogen chloride concentration resulting from WSEC's emissions was 0.15 $\mu g/m^3$, which is below the annual average ESL for hydrogen chloride of 7.5 $\mu g/m^3$.

157. No adverse health or welfare effects will result from any emissions of hydrogen chloride from WSEC.

Chromium

158. The maximum modeled 1-hour average chromium concentration resulting from WSEC's emissions was 0.026 $\mu g/m^3$, which is below the 1-hour ESL for chromium of 1 $\mu g/m^3$.

159. The maximum modeled annual average chromium concentration resulting from WSEC's emissions was 0.0003 $\mu g/m^3$, which is below the annual average ESL for chromium of 0.1 $\mu g/m^3$.

160. No adverse health or welfare effects will result from any emissions of chromium from WSEC.

Copper

161. The maximum modeled 1-hour average copper concentration resulting from WSEC's emissions was 0.005 $\mu g/m^3$, which is below the 1-hour ESL for copper of 10 $\mu g/m^3$.

162. The maximum modeled annual average copper concentration resulting from WSEC's emissions was 0.0002 $\mu g/m^3$, which is below the annual average ESL for copper of 1 $\mu g/m^3$.

163. No adverse health or welfare effects will result from any emissions of copper from WSEC.

Hydrogen fluoride

164. The maximum modeled 1-hour average hydrogen fluoride concentration resulting from WSEC's emissions was 1.9 $\mu g/m^3$, which is below the 1-hour ESL for hydrogen fluoride of 5 $\mu g/m^3$.

165. The maximum modeled annual average hydrogen fluoride concentration resulting from WSEC's emissions was 0.0122 $\mu g/m^3$, which is below the annual average ESL for hydrogen fluoride of 0.5 $\mu g/m^3$.

166. No adverse health or welfare effects will result from any emissions of hydrogen fluoride from WSEC.

Iron oxide

167. The maximum modeled 1-hour average iron oxide concentration resulting from WSEC's emissions was 8.59 $\mu g/m^3$, which is below the 1-hour ESL for iron oxide of 50 $\mu g/m^3$.

168. The maximum modeled annual average iron oxide concentration resulting from WSEC's emissions was 0.2081 $\mu g/m^3$, which is below the annual average ESL for iron oxide of 5 $\mu g/m^3$.

169. No adverse health or welfare effects will result from any emissions of iron oxide from WSEC.

Magnesium

170. The maximum modeled 1-hour average magnesium concentration resulting from WSEC's emissions was 0.16 $\mu g/m^3$, which is below the 1-hour ESL for magnesium of 50 $\mu g/m^3$.

171. The maximum modeled annual average magnesium concentration resulting from WSEC's emissions was 0.0042 $\mu g/m^3$, which is below the annual average ESL for magnesium of 5 $\mu g/m^3$.

172. No adverse health or welfare effects will result from any emissions of magnesium from WSEC.

Manganese

173. The maximum modeled 1-hour average manganese concentration resulting from WSEC's emissions was 0.250 $\mu g/m^3$, which is below the 1-hour ESL for manganese of 2 $\mu g/m^3$.

174. The maximum modeled annual average manganese concentration resulting from WSEC's emissions was 0.0005 $\mu g/m^3$, which is below the annual average ESL for manganese of 0.2 $\mu g/m^3$.

175. No adverse health or welfare effects will result from any emissions of manganese from WSEC.

Mercury

176. The maximum modeled 1-hour average mercury concentration resulting from WSEC's emissions was 0.003 $\mu g/m^3$, which is below the 1-hour ESL for mercury of 0.25 $\mu g/m^3$.

177. The maximum modeled annual average mercury concentration resulting from WSEC's emissions was 0.00004 $\mu g/m^3$, which is below the annual average ESL for mercury of 0.025 $\mu g/m^3$.

178. No adverse health or welfare effects will result from any emissions of mercury from WSEC.

Nickel

179. The maximum modeled 1-hour average nickel concentration resulting from WSEC's emissions was 0.16 $\mu g/m^3$, which is above the 1-hour ESL for nickel of 0.15 $\mu g/m^3$.

180. The maximum modeled 1-hour average nickel concentration at a sensitive receptor resulting from WSEC's emissions was less than the 1-hour ESL for nickel.

181. The maximum modeled annual average nickel concentration resulting from WSEC's emissions was 0.0100 μg/m3, which is below the annual average ESL for nickel of 0.015 $\mu g/m^3$.

182. No adverse health or welfare effects will result from any emissions of nickel from WSEC.

Potassium

183. The maximum modeled 1-hour average potassium concentration resulting from WSEC's emissions was 0.610 $\mu g/m^3$, which is below the 1-hour ESL for potassium of 50 $\mu g/m^3$.

184. The maximum modeled annual average potassium concentration resulting from WSEC's emissions was 0.0163 $\mu g/m^3$, which is below the annual average ESL for potassium of 5 $\mu g/m^3$.

185. No adverse health or welfare effects will result from any emissions of potassium from WSEC.

Selenium

186. The maximum modeled 1-hour average selenium concentration resulting from WSEC's emissions was 0.003 $\mu g/m^3$, which is below the 1-hour ESL for selenium of 2 $\mu g/m^3$.

187. The maximum modeled annual average selenium concentration resulting from WSEC's emissions was 0.00005 $\mu g/m^3$, which is below the annual average ESL for selenium of 0.2 $\mu g/m^3$.

188. No adverse health or welfare effects will result from any emissions of selenium from WSEC.

Silicon dioxide (silica)

189. The maximum modeled 1-hour average silica concentration resulting from WSEC's emissions was 29.7 $\mu g/m^3$,which is above the 1-hour ESL for silica of 14 $\mu g/m^3$.

190. Modeled 1-hour average silica concentrations resulting from WSEC's emissions exceeded the 1-hour ESL for silica of 14 $\mu g/m^3$ two hours per year.

191. The maximum modeled annual average silica concentration resulting from WSEC's emissions was 0.24 $\mu g/m^3$, which is below the annual average ESL for silica of 0.33 $\mu g/m^3$.

192. No adverse health or welfare effects will result from any emissions of silica from WSEC.

Sodium

193. The maximum modeled 1-hour average sodium concentration resulting from WSEC's emissions was 0.285 $\mu g/m^3$, which is below the 1-hour ESL for sodium of 20 $\mu g/m^3$.

194. The maximum modeled annual average sodium concentration resulting from WSEC's emissions was 0.006 $\mu g/m^3$, which is below the annual average ESL for sodium of 2 $\mu g/m^3$.

195. No adverse health or welfare effects will result from any emissions of sodium from WSEC.

Titanium

196. The maximum modeled 1-hour average titanium concentration resulting from WSEC's emissions was 0.191 $\mu g/m^3$, which is below the 1-hour ESL for titanium of 50 $\mu g/m^3$.

197. The maximum modeled annual average titanium concentration resulting from WSEC's emissions was 0.006 $\mu g/m^3$, which is below the annual average ESL for titanium of 5 $\mu g/m^3$.

198. No adverse health or welfare effects will result from any emissions of titanium from WSEC.

Vanadium

199. The maximum modeled 1-hour average vanadium concentration resulting from WSEC's emissions was 0.7 $\mu g/m^3$, which is above the 1-hour ESL for vanadium of 0.5 $\mu g/m^3$.

200. The maximum modeled 1-hour average vanadium concentration at a sensitive receptor resulting from WSEC's emissions was 0.51 $\mu g/m^3$, which is approximately 2 percent higher than the 1-hour ESL of 0.5 $\mu g/m^3$, with a frequency of occurrence of one hour per year.

201. The maximum modeled annual average vanadium concentration resulting from WSEC's emissions was 0.048 $\mu g/m^3$, which is less than the annual average ESL for vanadium of 0.05 $\mu g/m^3$.

202. No adverse health or welfare effects will result from any emissions of vanadium from WSEC.

Gasoline vapor

203. The maximum modeled 1-hour average gasoline vapor concentration resulting from WSEC's emissions was 1039 $\mu g/m^3$, which is below the 1-hour ESL for gasoline vapor of 3500 $\mu g/m^3$.

204. The maximum modeled annual average gasoline vapor concentration resulting from WSEC's emissions was 3.05 $\mu g/m^3$, which is below the annual average ESL for gasoline vapor of 350 $\mu g/m^3$.

205. No adverse health or welfare effects will result from any emissions of gasoline vapor from WSEC.

Diesel vapor

206. The maximum modeled 1-hour average diesel vapor concentration resulting from WSEC's emissions was 149 $\mu g/m^3$, which is below the 1-hour ESL for diesel vapor of 1000 $\mu g/m^3$.

207. The maximum modeled annual average diesel vapor concentration resulting from WSEC's emissions was 0.43 $\mu g/m^3$, which is below the annual average ESL for diesel vapor of 100 $\mu g/m^3$.

208. No adverse health or welfare effects will result from any emissions of diesel vapor from WSEC.

Coal Dust

209. Coal dust is a component of PM emissions.

210. The occupational standard for coal dust is an 8-hour average of 900 $\mu g/m^3$.

211. It is the respirable portion of coal dust emissions that is of concern from a toxicological perspective.

212. WSEC adjusted the PM emissions by 50 percent to model the respirable coal dust particles.

213. WSEC's modeled coal dust exceedances extend into and across the Colorado River onto the opposite bank.

213.a The maximum modeled 1-hour average coal dust concentration resulting from the WSEC's emissions was 55 $\mu g/m^3$, which is above the 1-hour average ESL for coal dust of 9 $\mu g/m^3$.

213.b The maximum 1-hour average coal dust concentration at any residence according to the modeling was 24 $\mu g/m^3$, and the frequency of exceedance at that receptor is 6 hours per year.

213.c The maximum modeled annual average coal dust concentration resulting from the WSEC's emissions was 1.69 $\mu g/m^3$, which is above the annual average ESL for coal dust of 0.9 $\mu g/m^3$.

213.d The area within which the annual average coal dust ESL was exceeded according to the modeling does not include any residences, schools, or other sensitive receptors.

213.e No adverse health or welfare effects will result from any emissions of coal dust from the WSEC.

Pet coke

214. Pet coke is a component of particulate matter emissions.

215. It is the respirable portion of pet coke emissions that is of a concern from a toxicological perspective.

216. WSEC applied an adjustment factor of 50 percent to the PM emissions to model the respirable pet coke particles.

217. The maximum modeled 1-hour average pet coke concentration resulting from WSEC's emissions was 52 $\mu g/m^3$, which is above the 1-hour average ESL for pet coke of 50 $\mu g/m^3$.

218. The maximum 1-hour average pet coke concentration at any residence according to the modeling was 23 $\mu g/m^3$, which is below the 1-hour average ESL for pet coke of 50 $\mu g/m^3$.

219. The maximum modeled annual average pet coke concentration resulting from WSEC's emissions was 1.14 $\mu g/m^3$, which is below the annual average ESL for pet coke of 5 $\mu g/m^3$.

220. No adverse health or welfare effects will result from any emissions of pet coke from WSEC.

Limestone

221. Limestone is a component of particulate matter emissions.

222. The maximum modeled 1-hour average limestone concentration resulting from WSEC's emissions was 102 $\mu g/m^3$, which is above the 1-hour average ESL for limestone of 50 $\mu g/m^3$.

223. The maximum 1-hour average limestone concentration at any residence according to the modeling was 44 $\mu g/m^3$, which is below the 1-hour average ESL for limestone of 50 $\mu g/m^3$.

224. The maximum modeled annual average limestone concentration resulting from WSEC's emissions was 1.74 $\mu g/m^3$, which is below the annual average ESL for limestone of 5 $\mu g/m^3$.

225. No adverse health or welfare effects will result from any emissions of limestone from WSEC.

Calcium sulfate (gypsum)

226. Gypsum is a component of particulate matter emissions.

227. The maximum modeled 1-hour average gypsum concentration resulting from WSEC's emissions was 53 $\mu g/m^3$, which is above the 1-hour average ESL for gypsum of 50 $\mu g/m^3$.

228. The maximum 1-hour average gypsum concentration at any residence according to the modeling was 19 $\mu g/m^3$, which is below the 1-hour average ESL for gypsum of 50 $\mu g/m^3$.

229. The maximum modeled annual average gypsum concentration resulting from WSEC's emissions was 0.27 $\mu g/m^3$, which is below the annual average ESL for gypsum of 5 $\mu g/m^3$.

230. No adverse health or welfare effects will result from any emissions of gypsum from WSEC.

Additional findings concerning air emissions

231. Emissions of particulate matter from the CFB boilers at WSEC will not be greater than the limit established under 30 TAC § 151.153(b) of 0.3 lb/MMBtu on a two-hour average basis.

232. Emissions of particulate matter from the stationary vents at WSEC will not exceed the opacity limit of 20 percent over a six-minute period established at 30 TAC § 111.111(a)(1)(B).

233. WSEC will comply with the limits on particulate matter emissions established under 30 TAC § 111.151.

234. WSEC's diesel fuel tanks, which will supply fuel to fire the emergency engines, will only store diesel fuels that meet the Chapter 114 specifications.

235. The unloading of diesel fuel from trucks into storage tanks at WSEC will be subject to and will comply with the control, inspection, and recordkeeping requirements of Chapter 115, Subchapter C, Division 1.

236. Emissions of SO_2 from the CFB boilers at WSEC will not exceed the limit established under 30 TAC § 112.8(a) of 3.0 lb/MMBtu on a 3-hour average basis.

237. The requirement to prepare a disaster review for WSEC was triggered by the on-site storage of anhydrous ammonia, which will be used as a reagent in the SNCR NO_x emission control equipment.

238. WSEC prepared a disaster review demonstrating that the disaster potential associated with the storage of anhydrous ammonia will be minimized and that the public health and welfare will be protected.

248.a Emissions from the WSEC will not cause adverse effects on human health or welfare, animal life, vegetation, or property, or interfere with the normal use and enjoyment of animal life, vegetation, or property.

Best Available Control Technology (BACT): 30 TAC § 116.111(a)(2)(C)

239. TCEQ defines BACT as "best available control technology with consideration given to the technical practicability and the economic reasonableness of reducing or eliminating emissions from the facility." 30 TAC § 116.10(3).

240. To implement the BACT requirement, the TCEQ developed a regulatory guidance document entitled "Evaluating Best Available Control Technology (BACT) in Air Permit Applications," also known as RG-383.

241. RG-383 describes the process to conduct and evaluate BACT proposals submitted in an NSR air permit application.

242. The TCEQ BACT evaluation is conducted using a "tiered" analysis approach, involving three different tiers.

243. A Tier I evaluation involves a comparison of an applicant's BACT proposal to the emission reduction performance levels that have been accepted as BACT in recent permit reviews involving the same process or industry.

244. Evaluation of new technical developments may also be necessary under Tier I.

245. A Tier II evaluation involves consideration of controls that have been accepted as BACT in recent permits for similar air emission streams in a different process or industry.

246. A Tier III evaluation is a detailed technical and quantitative economic analysis of all emission reduction options available for the process under review.

247. Technical practicability is established through demonstrated success of an emission reduction option based on previous use, and/or engineering evaluation of a new technology.

248. In its permitting process, TCEQ refers to portions of EPA's draft "October 1990 New Source Review Workshop Manual Prevention of Significant Deterioration and Nonattainment Area Permitting."

249. EPA defines BACT as an emissions limitation (including a visible emissions standard) based on the maximum degree of reduction for each regulated NSR pollutant that would be emitted from any proposed major stationary source or major modification, which the reviewing authority, on a case-by-case basis, taking into account energy, environmental, and economic impacts and other costs, determines is achievable for such source or modification through application of production processes or available methods, systems,

and techniques, including fuel cleaning or treatment or innovative fuel combustion techniques for control of such pollutant.

250. EPA uses a "top-down" approach for BACT analysis and requires the following steps: (1) identify all potential control technologies; (2) eliminate technically infeasible options; (3) rank remaining control technologies by control effectiveness; (4) evaluate the most effective controls and document the results; and (5) select the BACT by choosing the best technology not eliminated in step four (based upon concerns regarding collateral energy, environmental, or economic impacts).

251. In its approval of the Texas SIP and PSD program, EPA determined that Texas was not required to use its top-down approach.

252. WSEC reviewed the permit requirements in two recently TCEQ-issued permits for CFB boilers: Formosa Plastics (Formosa), TCEQ Permit No. 76044/PSD-TX-1053; and Calhoun County Navigation District (CCND), TCEQ Permit No. 45586/PSD-TX-1055.

253. WSEC's BACT analysis was done in accordance with RG-383 and included a review of the RACT/BACT/LAER Clearinghouse (RBLC) database to collect information on control technologies required of CFBs by other states.

254. WSEC considered information from vendors and engineering experts on the most realistic emissions limits available with BACT, as well as other permit applications and state websites.

255. WSEC will utilize the most stringent emissions control technology.

256. There are no new technical developments that are both technically practicable and economically reasonable that offer the potential for WSEC to further reduce its emissions.

257. Each of WSEC's CFB boilers will be equipped with all of the control technologies accepted by TCEQ as BACT for Formosa's and CCND's CFB boilers, with the addition of activated carbon injection for mercury control, which was not required by TCEQ of either Formosa or CCND, and a post-combustion scrubber, which was not required by TCEQ of CCND.

258. WSEC performed its BACT analysis under Tier I in accordance with TCEQ guidance.

259. WSEC did not consider integrated gasification combined cycle (IGCC) technology as part of its BACT analysis because IGCC would constitute redefinition of WSEC's proposed CFB power plant design.

260. WSEC's decision to use bituminous coal from the Illinois Basin and pet coke as fuel for the proposed facility was a fundamental business decision that affected the design and location of the facility.

261. Pet coke is generated in the Gulf Coast region of Texas, providing a fuel source close to WSEC's facility.

262. The local availability of pet coke was an important factor in selecting the site and design of the facility.

263. The use of Illinois Basin coal would diversify the overall mix of fuels used for power generation in Texas since other solid-fuel generation in Texas is fueled by either Texas lignite or western sub-bituminous coals from the Power River Basin.

264. A compulsion to use other fuels as a result of the BACT analysis would result in a redefinition of the source.

265. The operational and design differences between a CFB boiler and a PC boiler are substantial and the emissions streams are different.

266. In addition to the Formosa and CCND BACT determinations and the information in the RBLC (a nationwide inventory of BACT determinations), WSEC also reviewed actual permits and permit applications, as well as data from state websites and information obtained through contacts with state regulators and utility representatives.

267. WSEC sought input from engineering firms and potential equipment providers in conducting its BACT analysis.

268. The ED performed his own BACT review of WSEC's project, which included consideration of the RBLC as well as the air permits recently issued for CFBs in Texas (CCND, Formosa Plastics, and Sandow 5) and the draft permit developed for Las Brisas Energy Center.

269. The ED concluded that WSEC's control technologies and emission limits constituted BACT, and documented his reasons for approving slightly different emission rates than those listed in the RBLC for a few of the plants.

270. The specific fuel used by a combustion device affects its emission rates, and must be taken into account when setting permit limits.

271. There are several kinds of coals available, including bituminous, sub-bituminous, lignite and others. Within the bituminous category, the chemical makeup of the coal varies from basin to basin, and, to a lesser degree, from seam to seam.

272. The results of isolated stack tests conducted on emissions at other plants do not establish the emission levels achieved during all operating scenarios, and therefore should not be used to set BACT-based limits at WSEC.

NO_x

273. In conducting its BACT analysis, WSEC limited its consideration of other BACT determinations to those made for other CFB projects, and did not include consideration of PC boilers.

274. WSEC will use selective non-catalytic reduction (SNCR) for additional NO_x removal.

275. An SNCR system injects ammonia into the gases leaving the boiler, where it reacts with NO_x to form nitrogen and water.

276. Using SNCR technology, the BACT emission limits for NO_x are 0.10 lb/MMBtu (hourly limit) and 0.070 lb/MMBtu (30-day average).

277. WSEC's 30-day rolling average NO_x limit of 0.070 pound per million British thermal units (lb/MMBtu) is the most stringent limit for any pet coke or coal-fired CFB plant in the U.S. It also is the lowest emission rate that Alstom Power and Foster Wheeler, the two vendors that manufacture CFBs in the 300 MW range, would guarantee.

278. In addition to SNCR, WSEC and the ED investigated use of another technology to control NO_x emissions, selective catalytic reduction (SCR), which uses a catalyst bed to promote the ammonia-NO_x reactions.

279. SCR is not a new technical development—it has been in existence for decades.

280. No permitting authority has ever determined that SCR in any configuration represents BACT for a CFB.

281. There are no coal or pet coke-fired CFBs anywhere in the world that use SCR in any configuration.

282. The high-dust configuration for SCR is not technically feasible because of catalyst poisoning or deactivation by calcium oxide in the flue gas stream from the limestone introduced during combustion for sulfur emissions capture.

283. High-dust SCR on a CFB is not commercially available.

284. The only application of a tail-end SCR on a PC boiler in the U.S., the retrofitting of tail-end SCR at the Mercer power plant located within a nonattainment area in New Jersey, was the result not of a BACT determination, but of an EPA consent decree imposed to resolve violations of the Clean Air Act.

285. For the reactions to occur in an SCR, the flue gas temperature must be between 580°F and 750°F, whereas the flue gas temperature exiting a baghouse from a CFB unit is typically 140°F. This means that the tail gas must be re-heated before treatment, with a substantial energy and emissions penalty.

286. Tail-end SCR is not technically feasible or economically reasonable.

287. A side effect of having to re-heat flue gases for treatment in a tail-end SCR would be increased emissions due to additional fuel combustion.

288. In a tail-end SCR configuration, some amount of calcium oxide still would remain in the flue gas, which would not be removed by the baghouse and would pose a risk of catalyst poisoning.

289. SNCR is an effective NO_x control for CFBs, and does not have the same problems associated with SCR.

SO_2

290. WSEC will use two systems for SO_2 control: the CFB combustion process; and a dry flue gas desulfurization (FGD). These two systems and their combined control efficiency of 99 percent are BACT for the control of SO_2.

291. The following emission limits are BACT for SO_2: 0.114 lb/MMBtu (30-day rolling average) and 0.086 lb/MMBtu (12-month average) while burning pet coke; and 0.063 lb/MMBtu (30-day rolling average) and 0.063 lb/MMBtu (12-month average) while burning coal.

292. Wet FGD is not a new technological development. Wet FGD on a CFB is not technologically practicable or economically reasonable for the control of SO_2.

293. The RBLC indicates that there are CFBs with lower permitted SO_2 emission rates, but this is a function not of superior control technology performance—they all use limestone bed and a dry FGD with a combined control efficiency of less than 99%—but of lower sulfur concentrations in the fuel.

294. Fuel selection is based on availability, reliability, performance, cost, and other factors, and changing the fuel source in the course of the BACT analysis would likely throw other design considerations into question, including the economics of the project.

295. Wet FGD requires more energy and water to operate, and produces a scrubber sludge waste stream.

PM

296. To control PM/PM_{10} emissions, WSEC will equip its CFB boilers with fabric filter baghouses. The injection of limestone into the boilers and use of a dry FGD will further reduce PM emissions.

297. Total PM is the sum of filterable PM and condensable PM.

298. Texas is one of a few states that sets emissions limits for total PM.

299. EPA's reference test method for condensable PM has an erratic and positive bias, and EPA proposed a new test method on March 25, 2009. EPA's proposed test method is expected to be more accurate. As of the date of the evidentiary hearing, EPA had not adopted this new test method.

300. There is scientific uncertainty in the measurement of condensable PM, a constituent of total PM.

301. There is variation in the total PM emission limits of other CFBs in the RBLC. The emission limits range from 0.012 to 0.050 lb/MMBtu, with WSEC's limits falling within that range.

302. The following emission limit is BACT for the control of filterable PM/PM_{10}: 0.010 lb/MMBtu based on a 3-hour average.

303. The following emission limit is BACT for the control of total PM: 0.025 lb/MMBtu for pet coke and coal based on a 3-hour average.

304. The following emission limits are BACT for the control of total $PM_{2.5}$: 0.025 lb/MMBtu based on a 3-hour average for pet coke and 0.018 lb/MMBtu based on a 3-hour average for coal.

305. The use of a wet electrostatic precipitator (ESP) is not a new technological development for use on a CFB. Wet ESP is not technologically practicable or economically reasonable. A wet ESP is not BACT for the control of total PM on a CFB.

306. The technology that controls PM_{10} emissions is the same technology that controls $PM_{2.5}$. Because $PM_{2.5}$ is a percentage of PM_{10}, WSEC's control technologies will also control emissions of $PM_{2.5}$.

307. WSEC's BACT analysis properly addressed $PM_{2.5}$ emissions as a subset of PM/PM_{10}.

Lead

308. Any lead emissions from each of the CFB boilers would be in the form of particulate matter, and would be controlled by the fabric filter baghouse.

Mercury

309. WSEC will use a combination of limestone injection, a fabric filter baghouse, and activated carbon injection for the control of mercury emissions.

310. Recent Texas permits for CFBs have not required the use of activated carbon injection to control mercury. Activated carbon injection is a new technology that is technically practicable and economically reasonable to control mercury emissions.

311. WSEC's control systems are BACT for the control of mercury.

312. The following emission limit is BACT for the control of mercury: 0.86×10^{-6} lb/MMBtu based on a 12-month average.

313. The mercury emission rate is directly influenced by the amount of mercury in the coal.

Ammonia

314. Emissions of ammonia from the CFB boilers will be controlled through the use of operational instrumentation systems to limit the ammonia injection rate such that the annual average ammonia slip from the SNCR system will be less than 5 parts per million by volume (dry, corrected to 3% oxygen).

CO

315. WSEC will use good combustion practices to control CO emissions. There are no other existing control measures to reduce emissions of CO.

316. The following emission limit is BACT for the control of CO: 0.10 lb/MMBtu based on a 12-month rolling average.

317. An oxidation catalyst cannot be used at a CFB plant such as WSEC because of the problem of catalyst fouling.

VOCs

318. WSEC will use good combustion practices to control VOCs.

319. The following emission limit is BACT for the control of VOCs: 0.005 lb/MMBtu based on a 3-hour average.

320. The use of an oxidation catalyst to control VOCs is not a new technology for use on a CFB.

321. Since an oxidation catalyst must be used in conjunction with an SCR, it is not technically practicable or economically reasonable to use on a CFB.

322. An oxidation catalyst is not BACT for the control of VOCs on a CFB.

H_2SO_4

323. H_2SO_4 is Sulfuric acid mist (SAM) and is an acid gas that is a component of condensable PM emissions.

324. WSEC will control H_2SO_4 through the use of a limestone bed CFB and a dry FGD, which will provide a 95 percent removal efficiency.

325. WSEC's control technology and 95 percent removal efficiency is BACT for the control of H_2SO_4 on a CFB.

326. H_2SO_4 is a constituent of condensable PM and there is uncertainty in the test methods and inaccuracies in the results.

327. The following emission limits are BACT for H_2SO_4: 0.016 lb/MMBtu based on a 3-hour average for pet coke and 0.012 lb/MMBtu based on a 3-hour average for coal.

Hydrogen Chloride (HCl) and Hydrogen Fluoride (HF)

328. WSEC will control the emission of HCl and HF through the injection of limestone into the boilers and the use of polishing scrubbers.

329. These control methods will provide a 98 percent removal efficiency for the removal of HCl and 95 percent removal efficiency for the removal of HF. These removal efficiencies are BACT for the control of HCl and HF.

330. The following limits are BACT for the control of HCl: 0.0013 lb/MMBtu on a 3-hour average when firing pet coke and 0.005 lb/MMBtu on a 3-hour average when firing coal.

331. The following limits are BACT for the control of HF: 0.0004 lb/MMBtu on a 3-hour average when firing pet coke and 0.0003 lb/MMBtu on a 3-hour average when burning coal.

Control of emissions from the CFB boilers during startup

332. WSEC will be required to prepare and submit to TCEQ a written Startup, Shutdown, and Malfunction Plan, which will detail procedures for minimizing emissions during startup, including starting-up with No. 2 fuel oil and minimizing the length of time to achieve steady-state operations.

Material handling facilities

333. WSEC will minimize emissions from material handling facilities through a combination of partial or total enclosure of conveyors; use of water and/or dust suppression, where technically practical, at transfer points, conveyors, and stockpiles; and use of fabric filter baghouses where technically practical. These control methods are consistent with those approved by TCEQ for material handling emissions at recently permitted coal-fired power plants.

Diesel-fired emergency generators and fire water pump

334. WSEC will minimize emissions from the diesel-fired emergency generators and fire water pump through proper engine operation and limiting the number of annual operating hours to less than 500.

Storage tanks

335. WSEC will minimize emissions from storage tanks by using fixed roof tanks that are submerged-filled, and, with the exception of the gasoline vehicle tank, storing materials with a vapor pressure less than 0.5 psia. The tank storing gasoline for vehicles will be less than 25,000 gallons.

336. The anhydrous ammonia storage tanks are pressure tanks and will not have any emissions during normal operations.

Fugitive emissions from process equipment in ammonia service

337. WSEC will minimize fugitive emissions from process equipment in ammonia service by using an audio/visual/olfactory leak detection and repair program.

Cooling towers

338. WSEC will control PM emissions from cooling towers by minimizing the drift rate through cooling tower design and by using mist eliminators. No additional technologies are available for drift control.

339. The use of air-cooled condensers, or dry cooling, is not a technology to control emissions, but a different method of cooling the plant processes.

340. Dry cooling results in less electrical generation, higher capital costs, more noise, higher auxiliary power requirements, larger footprint requirements, and higher maintenance costs due to the large number of fans.

341. Because dry cooling creates a parasitic load, more fuel input is required to produce the same amount of electricity, and so more plant emissions are produced.

342. By replacing the wet cooling system with a dry cooling system, WSEC would be more than doubling the associated PM_{10} emissions, and increasing emissions of other pollutants as well.

30 TAC § 116.111(a)(2)(G): Performance demonstration

343. WSEC provided information sufficient to demonstrate that WSEC has been planned to operate, and can and will be operated in a manner such that the performance specified in the Application and the Draft Permit will be achieved.

344. WSEC will be required by the terms of the Draft Permit to demonstrate achievement of the performance specified in the Application once WSEC is operating.

345. WSEC will be required by the terms of the Draft Permit to perform testing of emissions from the CFB boilers and various other emission sources, and operate CEMS and COMS on the CFB boiler stacks to demonstrate continuous compliance with certain emissions and opacity limits.

346. WSEC will be required by the terms of the Draft Permit to maintain, report, and make available a variety of records related to the fuels it uses and its ongoing operations under the permit – records that will be available to TCEQ to confirm that the facilities achieve the performance represented in the Application and specified in the Draft Permit.

30 TAC § 116.111(a)(2)(H): Nonattainment review

347. WSEC will be located in Matagorda County, Texas, which is not a designated nonattainment area for any air contaminant; therefore, it is not subject to nonattainment new source review requirements.

30 TAC § 116.111(a)(2)(I): Prevention of Significant Deterioration

347.a The WSEC has the potential to emit NO_x, SO_2, CO, PM, PM_{10}, $PM_{2.5}$, VOC and H_2SO_4 in quantities greater than their published significant emissions levels, and therefore triggers PSD review for those pollutants.

347.b White Stallion conducted an appropriate source impact analysis showing that the WSEC's allowable emissions will not cause or contribute to air pollution in violation of any NAAQS or PSD increment.

347.c White Stallion conducted an appropriate additional impacts analysis to assess the impairment to visibility, soils, and vegetation as a result of the WSEC and associated

commercial, residential, and industrial growth, and to assess air quality impacts as a result of such growth.

347.d Although the WSEC project is expected to create approximately 200 additional permanent jobs, because these jobs are expected to be filled by workers in the local area, this does not trigger the requirement for an in-depth growth analysis and is not expected to significantly increase the emissions of air contaminants from secondary sources.

347.e White Stallion's air dispersion modeling results demonstrated that the WSEC project will not result in adverse effects to soils and vegetation.

347.f The Class I area nearest to the site of the WSEC is the Caney Creek Wilderness Area, located 400 miles (645 kilometers) north-northeast of the site in southwestern Arkansas.

347.g The WSEC will not have adverse impacts on visibility because its emissions will comply with the visibility and opacity requirements in 30 TEX. ADMIN. CODE Chapter 111, and the WSEC will be located greater than 100 kilometers from the nearest Class I area.

347.h A Class I area visibility analysis is not required because the nearest Class I area is more than 100 kilometers from the site of the WSEC.

PSD Increment Analysis

347.i PSD increments are allowable incremental changes in off-property concentrations of certain pollutants for which PSD review has been triggered.

347.j White Stallion performed a PSD increment analysis for NO_2, PM_{10}, and SO_2.

PSD Increment Analysis: NO_2

347.k An NO_2 increment exists for one averaging period, annual (25 $\mu g/m^3$).

347.l The maximum modeled annual average NO_2 concentration resulting from the WSEC's emissions and emissions from other increment-consuming sources in the area constructed or modified after the increment baseline date was 4.8 $\mu g/m^3$.

347.m The WSEC's emissions will not cause or contribute to an exceedance of the annual average NO_2 increment of 25 $\mu g/m^3$.

PSD Increment Analysis: PM_{10}

347.n PM10 increments exist for two averaging periods: 24-hour (30 $\mu g/m^3$) and annual (17 $\mu g/m^3$).

347.o For its PM_{10} increment analysis, White Stallion conservatively compared its NAAQS modeling results, which included emissions from any non-increment-consuming sources constructed or modified prior to the increment baseline date, to the applicable increment.

24-hour PM_{10} Increment

347.p The maximum modeled 24-hour average PM_{10} concentration resulting from the WSEC's emissions and emissions from other sources in the area was 28.2 $\mu g/m^3$.

347.q The WSEC's emissions will not cause or contribute to an exceedance of the 24-hour PM_{10} increment of 30 $\mu g/m^3$.

Annual PM_{10} Increment

347.r The maximum modeled annual average PM_{10} concentration resulting from the WSEC's emissions and emissions from other sources in the area was 6.2 $\mu g/m^3$.

347.s The WSEC's emissions will not cause or contribute to an exceedance of the annual PM_{10} increment of 17 $\mu g/m^3$.

PSD Increment Analysis: SO_2

347.t SO_2 increments exist for three averaging periods: 3-hour (512 $\mu g/m^3$), 24-hour (79.3 $\mu g/m^3$), and annual (20 $\mu g/m^3$).

347.u For its SO_2 increment analysis, White Stallion conservatively compared its NAAQS modeling results, which included emissions from any non-increment-consuming sources constructed or modified prior to the increment baseline date, to the applicable increment.

3-hour SO_2 Increment

347.v The maximum modeled 3-hour average SO_2 concentration resulting from WSEC's emissions and emissions from other sources in the area was 505 $\mu g/m^3$.

347.w The WSEC's emissions will not cause or contribute to an exceedance of the 3-hour SO_2 increment of 512 $\mu g/m^3$.

24-hour SO_2 Increment

347.x The maximum modeled 24-hour average SO_2 concentration resulting from WSEC's emissions and emissions from other sources in the area was 79.3 $\mu g/m^3$.

347.y The WSEC's emissions will not cause or contribute to an exceedance of the 24-hour SO_2 increment of 91 $\mu g/m^3$.

Annual SO_2 Increment

347.z The maximum modeled annual average SO_2 concentration resulting from the WSEC's emissions and emissions from other sources in the area was 6.7 $\mu g/m^3$.

347.aa The WSEC's emissions will not cause or contribute to an exceedance of the 24-hour SO_2 increment of 20 $\mu g/m^3$.

PSD Increment Analysis Summary

347.bb Emissions from the WSEC will not cause or contribute to an exceedance of any PSD increment.

PSD Preconstruction Monitoring Analysis

347.cc White Stallion modeled the WSEC's emissions of NO_2, CO, SO_2, and PM_{10} and compared the maximum predicted concentrations with the PSD monitoring significance levels.

347.dd No preconstruction monitoring data for NO_2 or CO were required because the maximum predicted concentrations were less than the PSD monitoring significance levels.

347.ee Preconstruction monitoring data for SO_2 and PM_{10} were required because the predicted concentrations of these pollutants were greater than the PSD monitoring significance levels.

347.ff The monitoring data for SO_2 and PM_{10} that were used to determine background concentrations were used to satisfy the TCEQ's PSD preconstruction monitoring data requirements.

Federal Standards of Review for Constructed or Reconstructed Major Sources of Hazardous Air Pollutants (HAPs): 30 TAC § 116.111(a)(2)(K) (Case-By-Case MACT).

348. EPA's definition states:

> Maximum achievable control technology (MACT) emission limitation for new sources means the emission limitation which is not less stringent than the emission limitation achieved in practice by the best controlled similar source, and which reflects the maximum degree of reduction in emissions that the permitting authority, taking into consideration the cost of achieving such emission reduction, and any non-air quality health and environmental impacts and energy requirements, determines is achievable by the constructed or reconstructed major source. 40 CFR § 63.41.

349. The TCEQ's definition mirrors the EPA's definition. 30 TAC § 116.15.

350. WSEC prepared an FCAA § 112(g) case-by-case MACT analysis as part of the application and applied for a HAP Major Source Permit to establish case-by-case MACT requirements.

351. The case-by-case MACT analysis was complete and included all information necessary for the ED to render a case-by-case MACT determination for WSEC facility.

352. ED staff reviewed the case-by-case MACT analysis and determined it to be complete and in compliance with all applicable rules and policies as documented in the Administrative Record.

353. Based on the case-by-case MACT analysis contained in the Application and other information available to the ED, the ED followed the proper procedure for case-by-case MACT determination for WSEC facility as described in the Preliminary Determination Summary.

354. WSEC performed the case-by-case MACT analysis in two primary steps. In the first step, WSEC established the "MACT floor" or the most stringent limitation achieved in practice by the best controlled similar source. In the second step, WSEC performed a "beyond-the-floor" analysis of the other methods for potentially reducing emissions to a greater degree, considering such factors as the cost of achieving such emissions reductions and any non-air quality health and environmental impacts and energy requirements to establish whether further reductions are achievable.

355. WSEC's facility may emit four categories of HAPs: non-mercury HAP metals, which are emitted as PM; mercury; organic HAPs; and acid gases, which include HCl and HF.

356. WSEC developed emission limits for five pollutants, with two of these pollutants serving as surrogates for two categories of HAPs. The five specific emissions limits proposed in the MACT application are: CO, for organic HAPs; filterable PM, for non-mercury HAP metals; mercury; HCl; and HF.

357. All necessary HAPs were evaluated as part of WSEC's MACT analysis.

358. There are no technologies available for controlling emissions of any specific non-mercury metals from WSEC beyond a fabric filter baghouse.

359. WSEC will use CO as a surrogate for organic HAPs. Organic HAPs are a subset of the VOC emissions and both VOCs and CO are products of incomplete combustion. The use of CO as a surrogate for ensuring the required MACT level of control for organic HAPs is appropriate because low levels of CO in the flue gas are indicators of good combustion,

and thus good indicators of the destruction of the organic HAPs. The CO emission limit represents the MACT emission limit for organic HAPs.

360. WSEC will use filterable PM as a surrogate for non-mercury HAP metals. The use of filterable PM is an appropriate surrogate for ensuring the required MACT level of control for non-mercury HAP metals because filterable PM and non-mercury HAP metals have common formation mechanisms and control techniques. The filterable PM limit will set a MACT emission limit for non-mercury HAP metals.

361. EPA and the TCEQ define the term "similar source" as "a stationary source or process that has comparable emissions and is structurally similar in design and capacity to a constructed or reconstructed major source such that the source could be controlled using the same control technology." 40 CFR § 63.41; 30 TAC § 116.15(10).

362. A MACT analysis should review facilities with similar combustion technology. Flue gases from CFBs and PCs have different concentrations of pollutants and different physical properties. The type of fuel burned has a major impact on the amount and type of pollutants emitted from the facility.

363. Evaluating facilities with similar combustion processes is sufficient to determine the best controlled similar source in a MACT analysis. WSEC properly evaluated the best controlled similar sources in its MACT analysis.

364. A removal efficiency of 90 percent and limit of 0.86×10^{-6} lb/MMBtu is MACT for the control of mercury at WSEC facility.

365. A filterable PM limit of 0.010 lb/MMBtu is MACT for the control of non-mercury HAP metals.

366. A 98 percent removal efficiency for HCl is MACT for the control of this pollutant. The following limits are MACT for the control of HCl: 0.0013 lb/MMBtu on a 3-hour average when firing pet coke and 0.005 lb/MMBtu on a 3-hour average when firing coal.

367. A 95 percent removal efficiency for HF is MACT for the control of this pollutant. The following limits are MACT for the control of HF: 0.0004 lb/MMBtu on a 3-hour average when firing pet coke and 0.0003 lb/MMBtu on a 3-hour average when burning coal.

368. A CO limit of 0.010 lb/MMBtu based on a 12-month average is MACT for the control of organic HAPs.

369. Utilization of good pollution control practices to meet the hourly emission limits set forth in the Maximum Allowable Emission Rate Table (MAERT) of the Draft Permit is MACT for start-up and shut down emissions from WSEC facility.

370. WSEC is required to comply with the lb/hr emission rates listed in the draft MAERT. The lb/hr emission rates in the MAERT are calculated directly from the BACT/MACT-based lb/MMBtu standards in Special Condition No. 10 using a MMBtu/hr conversion factor.

371. The ED performed a review of WSEC's case-by-case MACT analysis, and determined that WSEC will apply MACT to control HAP emissions. The results of that determination are incorporated into the terms of the Draft Permit.

Special Condition 45

372. Special Condition No. 45 is not a substitute for determining BACT and MACT. Special Condition No. 45 allows for permit limits to be adjusted downward if actual emission levels prove to be less than the permitted limit.

Monitoring Provisions

373. To monitor compliance with applicable standards for PM, VOC, H_2SO_4, HCl, and HF, WSEC will conduct periodic stack sampling, install bag break detectors, and monitor the pressure drop across the baghouse to ensure that it is operating according to manufacturers' guidelines. WSEC will use a continuous opacity monitoring system (COMS) to aid compliance with PM emission limits.

374. Neither Texas nor federal law requires WSEC to monitor PM emissions with a CEMS.

375. Bag break detectors, which alert the operator to any problems with the functioning of the fabric filter baghouse, have an advantage over PM CEMS in that they provide immediate feedback to the operator regarding the location of a bag break, which allows for faster, more directed corrective action to shut down a particular compartment right away if necessary to minimize PM emissions.

PAL Permit

376. EPA has not determined whether the TCEQ's rules regarding plantwide area permit limits comply with the SIP. EPA Region 6 has expressed no legal challenge to the Commission's authority to grant WSEC's application under the current PAL rules.

30 TAC § 116.111(a)(2)(L): Mass cap and trade allowances

377. WSEC will not be located in the Houston-Galveston-Brazoria nonattainment area.

Compliance history

378. WSEC's compliance history is classified as "average by default" because it is a new entity.

Draft permit

379. The special conditions contained in the Draft Permit are comparable to those contained in other permits issued by the TCEQ.

380. The WSEC, as designed, is expected to comply with the terms of the Draft Permit.

381. The Draft Permit prescribes requirements for demonstrating initial and ongoing compliance with all applicable requirements of the permit and of the Texas Clean Air Act (TCAA).

382. Special Condition No. 45 states that, if the first annual compliance sampling after startup indicates measured emission rates below 50 percent of the limits for certain pollutants, WSEC must, within 60 days, submit a request to adjust those limits to reflect the results of the testing.

383. Inclusion of Special Condition No. 45 in the Draft Permit did not affect the BACT determination for this project.

Transcript Costs

384. The ALJs required the court reporter to prepare the transcript, and no specific party actually requested it.

385. All of the parties participated in the hearing. Although WSEC presented the most number of direct witnesses and the only rebuttal witnesses, the parties actively cross-examined each others' witnesses. All parties relied on the transcript in their closing arguments and replies. Each party benefitted from a hearing transcript.

386. The transcript costs are allocated equally among the three non-statutory parties: 1/3 to WSEC; 1/3 to EDF; and 1/3 to SC/NCC.

387. WSEC paid $7,529.75 for court reporting services. One-third of the fee for the court reporting services is $2,509.91.

Other remaining issues

388. With respect to all other contested issues and all unrefuted issues, the Application and the remainder of the evidentiary record contain sufficient factual information to satisfy all applicable statutory and regulatory requirements.

II. CONCLUSIONS OF LAW

Jurisdiction

1. The Commission has jurisdiction over WSEC's Application pursuant to TEX. HEALTH & SAFETY CODE Chapter 382 and TEX. WATER CODE Chapter 5.

2. WSEC's Application was directly referred to SOAH pursuant to TEX. WATER CODE § 5.557.

3. Pursuant to TEX. GOV'T CODE § 2003.047, SOAH has jurisdiction to conduct a hearing and to prepare a PFD in this matter.

4. Proper notice of WSEC's Application was provided pursuant to TEX. HEALTH & SAFETY CODE §§ 382.0516, 382.0517, and 382.056, 30 TAC § 39.601, *et seq.*, and TEX. GOV'T CODE §§ 2001.051 and 2001.052.

5. WSEC properly submitted a complete Application pursuant to TEX. HEALTH & SAFETY CODE §§ 382.0515 and 382.0518 and 30 TAC §§ 116.110, 116.111, 116.140, and 116.404.

Burden of Proof

6. Pursuant to 30 TAC §§ 55.210 and 80.17(a), in a contested case hearing involving an air quality permit application that has been directly referred, the burden of proof is on the applicant to prove by a preponderance of the evidence that the application satisfies all statutory and regulatory requirements.

7. WSEC met its burden of proof that the Application satisfies all statutory and regulatory requirements.

TCAA Standards

8. Under Texas law, WSEC may not construct its proposed facility until it has obtained a permit from the Commission. TEX. HEALTH AND SAFETY CODE § 382.0518(a).

9. TEX. HEALTH AND SAFETY CODE § 382.0518(b) states:

> The commission shall grant within a reasonable time a permit or permit amendment to construct or modify a facility if, from the information available to the commission, including information presented at any hearing held under Section 382.056(k), the commission finds:
>
> (1) the proposed facility for which a permit, permit amendment, or a special permit is sought will use at least the best available control technology, considering the technical practicability and economic reasonableness of reducing or eliminating the emissions resulting from the facility; and
>
> (2) no indication that the emissions from the facility will contravene the intent of [the TCAA], including protection of the public's health and physical property.

10. Under the FCAA, new major sources of HAPs are prohibited from commencing construction unless the source demonstrates it will achieve an emission standard equivalent to the "maximum achievable control technology emission limitation" for each HAP emitted. 42 U.S.C. § 7412(g).

10.a TEX. HEALTH AND SAFETY CODE § 382.0541(a) authorizes the Commission to require certain sources to use BACT, or MACT, if it is more stringent, and to establish MACT requirements.

30 TAC § 116.111(a)(2)(A): Protection of Public Health and Welfare

11. A demonstration of compliance with the PM_{10} NAAQS suffices to demonstrate compliance with the $PM_{2.5}$ NAAQS.

12. There is no legal requirement that WSEC consult with EPA on the ozone analysis for this project.

13. Low levels of ozone precursors may be allowed to flow into an ozone nonattainment area without that contribution legally violating the "cause or contribute to" standard set forth at 40 CFR 52.21(k), as incorporated into TCEQ's rules at 30 TAC § 116.160(c)(2)(B).

14. WSEC met its burden or proving that in accordance with 40 CFR 52.21(k), as incorporated into TCEQ's rules at 30 TAC § 116.160(c)(2)(B), WSEC's emissions will not cause or contribute to air pollution in violation of any NAAQS in any air quality control region, or any applicable maximum allowable increase over the baseline concentration in any area.

14.a In accordance with 30 TAC § 101.4, the WSEC's emissions will not cause any nuisance conditions.

15. WSEC's emissions will comply with the opacity limits and particulate matter emission rates set forth in 30 TAC Chapter 111 concerning control of air pollution from visible emissions and particulate matter.

16. WSEC's emissions will comply with the sulfur compound emission requirements set forth in 30 TAC Chapter 112 concerning control of air pollution from sulfur compounds.

17. WSEC will comply with all applicable standards adopted by reference in 30 TAC Chapter 113.

18. WSEC's diesel fuel tanks will only store diesel that meets the specifications set forth in 30 TAC Chapter 114.

19. The unloading of diesel fuel from trucks into storage tanks at WSEC will comply with applicable control, inspection, and recordkeeping requirements set forth in 30 TAC Chapter 115.

20. WSEC is not subject to the rules set forth in 30 TAC Chapter 117 regarding the control of NO_X because it will not be located in an ozone nonattainment area.

21. WSEC is required to operate in compliance with any orders of the Commission relating to generalized and localized air pollution episodes under 30 TAC Chapter 118.

22. WSEC is not subject to the emission reduction plan requirements of 30 TAC Chapter 118.

23. In accordance with 30 TAC § 116.111(a)(2)(A)(i), emissions from WSEC will comply with all Commission rules and regulations and the intent of the TCAA, including protection of the health and property of the public.

24. WSEC is not required to evaluate any impacts from WSEC's emissions of substances that are not regulated under the TCAA, such as water vapor, nitrogen, methane, ethane, and carbon dioxide.

25. It was appropriate for WSEC to not model road emissions even for an annual averaging period.

26. The following standards or guidelines are appropriate to determine whether a source's emissions are likely to cause adverse health or welfare effects: National Ambient Air Quality Standards (NAAQS), Net Ground Level Concentration (NGLC) or "state property line" standards, and Effects Screening Levels (ESLs).

27. Special Condition No. 20 of the Draft Permit should be revised to read as follows: Permanent plant roads shall be paved with a cohesive hard surface which can be and cleaned by sweeping and washing as necessary to maintain compliance with all TCEQ rules and regulations. Other roads shall be sprinkled with water and/or surface crusting agents as necessary to maintain compliance with all TCEQ rules and regulations.

30 TAC § 116.111(a)(2)(B): Measurement of emissions

28. In accordance with 30 TAC §116.111(a)(2)(B), WSEC will have provisions for measuring the emission of air contaminants as determined by the Commission's ED.

29. WSEC will be required by the Draft Permit to properly install, operate, and maintain continuous emissions monitoring systems (CEMS) to provide a continuous demonstration of compliance with limits on emissions of NO_x, SO_2, CO, mercury and ammonia from the CFB boilers.

30. To monitor compliance with applicable standards for PM, VOC, H_2SO_4, HCl and HF, WSEC will be required by the Draft Permit to conduct periodic stack sampling and use other pollutant-specific techniques.

31. Other monitoring requirements in the Draft Permit include periodic sampling of fuel for sulfur and metals content.

32. For PM, in addition to periodic stack sampling, WSEC will install bag break detectors to monitor the pressure drop across the baghouse to ensure that it is meeting manufacturer guidelines for proper operation, and install and operate a continuous opacity monitoring system (COMS).

33. The ED considered and rejected requiring WSEC to use PM CEMS.

34. PM CEMS are an evolving technology that has not yet been required in permits in Texas.

35. PM CEMS have not been in existence long enough, or installed on enough sources, to provide a sufficient record of measurement from specific source categories to determine what limits are achievable on a continuous basis.

36. The Draft Permit's provisions for measuring emissions from WSEC are comparable to those required of similar facilities permitted by TCEQ.

37. The Draft Permit contains appropriate emissions-measuring provisions for each type of emission from each emission point, with consideration given to the relative significance of each, as well as to the measurement methods and data that were used to determine the

limits, and any emissions-measurement requirements of federal programs such as the NSPS and Acid Rain Rules.

38. The methods for measuring emissions from WSEC required by the Draft Permit are adequate to assure compliance with the permit conditions and emissions limitations.

30 TAC § 116.111(a)(2)(D): New Source Performance Standards

39. In accordance with 30 TAC § 116.111(a)(2)(D), the emissions from WSEC will meet the requirements of any applicable NSPS as listed under 40 CFR Part 60, promulgated by the EPA under authority granted under Section 111 of the FCAA, as amended.

30 TAC § 116.111(a)(2)(E): National Emissions Standards for Hazardous Air Pollutants (NESHAP)

40. There are no national emissions standards for hazardous air pollutants (NESHAPs) applicable to facilities of a type including WSEC.

30 TAC § 116.111(a)(2)(F): NESHAP for Source Categories

41. The only NESHAP for source categories applicable to facilities of a type including WSEC are those set forth at 40 CFR Part 63, Subpart ZZZZ (National Emission Standards for Hazardous Air Pollutants for Reciprocating Internal Combustion Engines), which generally apply to the diesel-fired emergency generators at WSEC. However, only the initial notification requirements of those rules apply.

30 TAC § 116.111(a)(2)(G): Performance demonstration

42. In accordance with 30 TAC § 116.111(a)(2)(G), WSEC facilities will achieve the performance specified in the permit application.

30 TAC § 116.111(a)(2)(I): Prevention of Significant Deterioration

43. In accordance with 30 TAC § 116.111(a)(2)(I), WSEC complies with all applicable requirements of Chapter 116 regarding PSD review.

30 TAC § 116.111(a)(2(J): Air Dispersion Modeling

44. In accordance with 30 TAC § 116.111(a)(2)(J), computerized air dispersion modeling was performed as required to determine the air impacts from WSEC.

30 TAC § 116.111(a)(2)(C): Best Available Control Technology

45. TCEQ defines BACT as, "[Best Available Control Technology] with consideration given to the technical practicability and the economic reasonableness of reducing or eliminating emissions from the facility." 30 TAC § 116.10(3).

46. The application of BACT, as defined at 40 CFR § 52.21(b)(12) or in EPA's top down methodology, would not result in more stringent emissions limits for WSEC's proposed facility.

47. In accordance with TEX. HEALTH & SAFETY CODE § 382.0518 and 30 TAC § 116.111(a)(2)(C), WSEC's facility will utilize BACT, with consideration given to the technical practicability and economic reasonableness of reducing or eliminating emissions from its facilities.

48. There is no statutory or regulatory requirement to evaluate BACT for carbon dioxide emissions.

49. An applicant that is proposing to construct a circulating fluidized bed power plant is not required to include other electric generation technologies, such as integrated gasification/combined cycle (IGCC) technology, in its BACT analysis.

50. In the context of a Tier I review, "new technical developments" encompass only those occurring since the most recent permitting decisions.

30 TAC § 116.111(a)(2)(K): Hazardous Air Pollutants & Maximum Achievable Control Technology (MACT)

51. In accordance with 30 TAC § 116.111(a)(2)(K) and Chapter 116, Subchapter C, WSEC will utilize MACT to control emissions from the CFB boilers.

52. In accordance with 30 TAC § 116.111(a)(2)(K), WSEC has complied with all applicable requirements of Chapter 116 regarding case-by-case MACT review.

53. TCEQ rules found at 30 TAC §§ 116.400-406 implement 40 CFR Part 63, Subpart B, which govern Hazardous Air Pollutant from Constructed or Reconstructed Major Sources.

54. Under 30 TAC § 116.15(5), a hazardous air pollutant is "any air pollutant listed under the FCAA, § 112(b)."

55. A "[s]ource" is "[a] point of origin of air contaminants, whether privately or publicly owned or operated. 30 TAC § 116.10(17).

56. An "affected source" is a "stationary source or group of stationary sources which, when fabricated (on-site), erected, or installed meets the criteria in §116.180(a)(1) and (2) of this title (relating to Applicability) and for which no MACT standard has been promulgated under 40 CFR Part 63." 30 TAC § 116.15(1). The cross-reference is § 116.15(1) to § 116.180, pertaining to plant-wide applicability limits, is incorrect. It should be a cross-reference to § 116.400, pertaining to case-by-case MACT review as required under FCAA § 112(g).

57. Major source is defined by 40 CFR § 63.2 as:

> any stationary source or group of stationary sources located within a contiguous area and under common control that emits or has the potential to emit considering controls, in the aggregate, 10 tons per year or more of any hazardous air pollutant or 25 tons per year or more of any combination of hazardous air pollutants, unless the Administrator establishes a lesser quantity, or in the case of radionuclides, different criteria from those specified in this sentence.

58. WSEC's facility would be a new major source of HAPs and an affected source as defined at 30 TAC § 116.15(1).

59. An affected source of HAPs is required to submit a permit application. 30 TAC § 116.404 states:

> Consistent with the requirements of 40 Code of Federal Regulations § 63.43 (concerning maximum achievable control technology determinations for constructed and reconstructed major sources), the owner or operator of a proposed affected source (as defined in §116.15(1) of this title (relating to Section 112(g) Definitions)) shall submit a permit application as described in §116.110 of this title (relating to Applicability).

60. MACT is defined by 30 TAC § 116.15(7) as:

> The emission limitation which is not less stringent than the emission limitation achieved in practice by the best controlled similar source, and which reflects the maximum degree of reduction in emissions that the executive director, taking into consideration the cost of achieving such emission reduction, and any non-air quality health and environmental impacts and energy requirements, determines is achievable by the constructed or reconstructed major source.

61. Similarly, 40 CFR § 63.41 provides:

> *Maximum achievable control technology (MACT) emission limitation* for new sources means the emission limitation which is not less stringent than the emission limitation achieved in practice by the best controlled similar source, and which reflects the maximum degree of reduction in emissions that the permitting authority, taking into consideration the cost of achieving such emission reduction, and any non-air quality health and environmental impacts and energy requirements, determines is achievable by the constructed or reconstructed major source.

62. WSEC's facility is an affected source of HAPs for which no MACT standard is in place.

63. Under 30 TAC § 116.110, before any actual work is begun on the facility, any person who plans to construct any new facility or to engage in the modification of any existing facility which may emit air contaminants into the air of this state shall either obtain a permit under 30 TAC §116.111, or comply with an alternative requirement.

64. Based on the above Findings of Fact and Conclusions of Law, WSEC has made all demonstrations required under applicable federal and state laws and regulations, including 30 TAC § 116.404 regarding hazardous air pollutant major source permit applications, to be issued a hazardous air pollutant major source air quality permit with case-by-case MACT review.

65. In accordance with 30 TAC §§ 116.111(a)(2)(K) and 116.404, an application for a case-by-case MACT determination was properly conducted and submitted by WSEC to establish federally enforceable MACT emission limits.

66. The case-by-case MACT application for WSEC facility is complete and complies with all applicable requirements for a HAP major source permit found in 30 TAC Chapter 116 and 40 CFR Part 63 regarding MACT review.

67. WSEC met its burden of proof regarding MACT.

68. WSEC's removal efficiencies for HCl and HF satisfy the requirements for MACT for the facility.

30 TAC § 116.111(a)(2)(B): Measurement of Emissions

69. In accordance with 30 TAC § 116.111(a)(2)(B), WSEC will have provisions for measuring the emission of air contaminants as determined by the ED.

30 TAC § 116.111(a)(2)(L): Mass cap and trade allowances

70. WSEC is not subject to the Mass Emissions Cap and Trade program.

WSEC's Permit

71. The special conditions in the permit are appropriately added under 30 TAC §§ 116.115(c)(1) and 116.186(c) and are consistent with the TCAA.

72. The PAL provisions of the permit are severable, meaning that their removal from the permit would have no effect on the rest of the permit terms and conditions.

73. Based on the above Findings of Fact and Conclusions of Law, WSEC has made all demonstrations required under applicable statutes and regulations, including 30 TAC § 116.111 regarding air permit applications, to be issued an air quality permit with PSD review.

74. Based on the above Findings of Fact and Conclusions of Law, WSEC has made all demonstrations required under applicable statutes and regulations, including 30 TAC § 116.182 regarding PAL permit applications, to be issued a PAL permit.

75. Based on the above Findings of Fact and Conclusions of Law, WSEC has made all demonstrations required under applicable statutes and regulations, including 30 TAC §§ 116.400 – 116.406 regarding HAP permit applications, to be issued a HAP permit.

76. In accordance with TEX. HEALTH & SAFETY CODE § 382.0518(b)(1), WSEC will use at least the BACT, considering the technical practicability and economic reasonableness of reducing or eliminating its emissions.

77. In accordance with TEX. HEALTH & SAFETY CODE § 382.0518(b)(2), emissions from WSEC will not contravene the intent of the TCAA and will be protective of the public's health and physical property, consistent with the long-standing interpretation of the Commission's rules, regulations, and guidance.

78. In accordance with Tex. Health & Safety Code §382.0518(b), the application for Air Quality Permit Nos. 86088, HAP28, PAL26 and PSD-TX-1160 should be approved and Air Quality Permit Nos. 86088, HAP28, PAL26 and PSD-TX-1160 should be issued with the following changes to Special Condition No. 20:

> Permanent plant roads shall be paved with a cohesive hard surface which can be and cleaned by sweeping and washing as necessary to maintain compliance with

all TCEQ rules and regulations. Other roads shall be sprinkled with water and/or surface crusting agents as necessary to maintain compliance with all TCEQ rules and regulations.

Transcript Costs

79. Based on the above Findings of Fact, EDF and SC/NCC should each be required to reimburse WSEC for one-third of the total invoice, or $2,509.91 each.

III. EXPLANATION OF CHANGES

At its September 29, 2010 agenda meeting, the Commission adopted the ALJs' Proposed Order granting White Stallion Energy Center's applications for Air Quality Permit No. 86088, PSD Air Quality Permit No. PSD-TX-1160; Major Source Permit HAP-28; and Plantwide Applicability Limit Permit PAL-26, as modified by the ALJs' reply, dated August 11, 2010, with the following exceptions and additional changes:

a. Deleted Finding of Fact No. 312;
b. Modified Finding of Fact No. 314 to set the BACT limit for Total PM at 0.025 lb/MMBtu for pet coke and coal, based on a 3-hour average;
c. Modified Finding of Fact No. 315 to set the BACT limit for Total $PM_{2.5}$ at 0.025 lb/MMBtu for pet coke and 0.018 lb/MMBtu coal, based on a 3-hour average;
d. Modified Finding of Fact No. 338 to set the BACT limit for H_2SO_4 at 0.016 lb/MMBtu for pet coke and 0.012 lb/MMBtu for coal based on a 3-hour average;
e. Adopted the following changes, as recommended by Applicant in the red-lined, revised proposed order attached to its Exceptions:
   - Added Applicant's proposed Finding of Fact Nos. 222(e) and 248(a);
   - Deleted the ALJs' Finding of Fact No. 223 and Conclusions of Law Nos. 8, 28, 46, and 78;
   - Accepted Applicant's proposed changes to the ALJs' Conclusions of Law Nos. 7, 24, 45;
   - Deleted the ALJs' Ordering Provisions Nos. 1 and 2; and
   - Accepted the Applicant's proposed Ordering Provision No. 1.

The Commission directed that the above changes to the ALJs' Proposed Order be made in light of the Commission's determination that the record establishes that no adverse health or welfare effects will result from emissions of coal dust from WSEC and that WSEC met its burden of proof regarding the validity of the ozone monitor data and based on the Commission's determination that the record establishes that: 1) the BACT emission limit for Total PM is 0.025 lb/MMBtu for pet coke and coal, based on a 3-hour average; 2) the BACT emission limits for Total $PM_{2.5}$ are 0.025 lb/MMBtu for pet coke and 0.018 lb/MMBtu coal, based on a 3-hour average; 3) the BACT emission limits for H_2SO_4 are 0.016 lb/MMBtu for pet coke and 0.012 lb/MMBtu for coal based on a 3-hour average; and 4) the ALJs correctly determined in their August 11, 2010 reply that the record establishes that the MACT emission limits for the control of HCl are: 0.0013 lb/MMBtu on a 3-hour average when firing pet coke and 0.005 lb/MMBtu on a 3-hour average when firing coal; and the MACT emission limits for the control of HF are: 0.0004 lb/MMBtu on a 3-hour average when firing pet coke and 0.0003 lb/MMBtu on a 3-hour average when burning coal.

In addition, the General Counsel renumbered the ALJs' Proposed Order, as directed by the Commission at its September 29, 2010 agenda.

NOW, THEREFORE, BE IT ORDERED BY THE TEXAS COMMISSION ON ENVIRONMENTAL QUALITY, IN ACCORDANCE WITH THESE FINDINGS OF FACT AND CONCLUSIONS OF LAW, THAT:

1. The application of White Stallion Energy Center, LLC for Air Quality Permit Nos. 86088, HAP28, PAL26, and PSD-TX-1160 is approved and the attached draft permit is issued.

2. The ED's Response to Comments concerning WSEC's Air Permit Nos. 86088, HAP28, PAL26, and PSD-TX-1160 is adopted and approved. If there is any conflict between the Commission's Order and the ED's Response to Comments, the Commission's Order prevails.

3. EDF and SC/NCC are each required to reimburse WSEC for one-third of the total invoice, or $2,509.91 each.

4. The effective date of this Order is the date the Order is final, as provided by 30 TAC § 80.273 and TEX. GOV'T CODE § 2001.144.

5. The Chief Clerk of the Commission shall forward a copy of this Order to all parties and issue the attached permit as changed to conform to this Order.

6 All other motions, requests for specific Findings of Fact or Conclusions of Law, and other requests for general and specific relief, if not expressly granted, are denied for want of merit.

7. If any provision, sentence, clause, or phrase of this Order is for any reason held to be invalid, the invalidity of any portion shall not affect the validity of the remaining portions of this Order.

ISSUED: OCT 19 2010

TEXAS COMMISSION ON ENVIRONMENTAL QUALITY

Bryan W. Shaw

Bryan W. Shaw, Ph.D., Chairman
For the Commission

EXHIBIT 10

To Comments of Public Health and Environmental Groups
on Docket ID No. EPA-HQ-OAR-2009-0234
80 Fed. Reg. 75,025 (Dec. 1, 2015)

Andover Technology Partners
"Review and Analysis of the Actual Costs of Complying With MATS in Comparison to Predicted in EPA's Regulatory Impact Analysis"

REVIEW AND ANALYSIS OF THE ACTUAL COSTS OF COMPLYING WITH MATS IN COMPARISON TO PREDICTED IN EPA'S REGULATORY IMPACT ANALYSIS

At this point we are in a position to make a post-hoc assessment of what the cost has been to comply with US EPA's Mercury and Air Toxics Standards (MATS) for power plants. In its Regulatory Impact Analysis (RIA) for the final rule.[1] EPA estimated a cost for the rule of $9.6 billion (2007 dollars) versus quantified benefits of between $33 billion to $81 billion, depending upon discount rate (plus other unquantified benefits). The $9.6 billion annual cost is primarily the cost to control coal-fired units, at an estimated $9.4 billion. This $9.4 billion includes the following components:

- Amortized capital
- Costs associated with change in fuel
- Variable operating and maintenance (VOM)
- Fixed operating and maintenance (FOM)

These costs are estimated using the Integrated Planning Model (IPM), which is described later. The fuel costs are associated with the costs of switching to natural gas or to lower chlorine coal.

Experience with technologies deployed for MATS compliance has shown them to be less expensive and more effective than originally assumed in EPA's analysis. Technological improvements and a lower price of natural gas than originally projected have further reduced costs. As a result, the true cost of complying with the MATS rule is approximately $7 billion per year per year less than estimated by EPA, making the true cost of the rule approximately $2 billion, or less than one-quarter of what EPA originally estimated the Rule to cost.

Except for the fuel charge, EPA's forecast of the cost impact of the MATS rule is determined in large part by the forecast of installed air pollution control equipment, which is shown in Figure 1. This figure shows the forecast installations (expressed as GW of installed capacity) in the Base Case and forecast installations in the case of the MATS rule. As shown, EPA forecast a reduction in wet FGD systems (fewer FGD retrofits in the policy case than in the Base Case) and increases in dry FGD systems, FGD upgrades, increase in Dry Sorbent Injection (DSI), an increase in Activated Carbon Injection (ACI), and increases in Fabric Filters (FF) and ESP upgrades. These forecasts are determined using ICF International's Integrated Planning Model (IPM), which is described briefly in the insert on the following

[1] Regulatory Impact Analysis for the Final Mercury and Air Toxics Standards, EPA-452/R-11-011, December 2011

page, and the methodology and assumptions for IPM are described in detail in the documentation found on EPA's web site.

Methods to comply with the regulation may include addition of control technology, changing fuels, or even retirement. For every technology considered EPA makes assumptions about the capital and operating cost of the technology and the performance of the technology with regard to emissions control performance. Costs for fuels are considered as well, and this is particularly important when an option is to change to different fuels. IPM selects the approach that provides the lowest cost to comply, or, alternatively, the highest future value for operation of the facility. IPM estimates the future dispatch of the facility based upon the economics of that facility relative to other facilities in the region. In cases where the facility is determined to be uneconomical to operate in the future, IPM will determine that the facility will be retired and electricity supplied from other sources.

According to the RIA issued with the final rule: *"This analysis projects that by 2015, the final rule will drive the installation of an additional 20 GW of dry FGD (dry scrubbers), 44 GW of DSI, 99 GW of additional ACI, 102 GW of additional fabric filters, 63 GW of scrubber upgrades, and 34 GW of ESP upgrades. . . .With respect to the increase in operating ACI, some of this increase represents existing ACI capacity on units built before 2008. EPA's modeling does not reflect the presence of state mercury rules, and EPA assumes that ACI controls on units built before 2008 do not operate in the absence of these rules. In the policy case, these controls are projected to operate and the projected compliance cost thus reflects the operating cost of these controls. Since these controls are in existence, EPA does not count their capacity toward new retrofit construction, nor does EPA's compliance costs projection reflect the capital cost of these controls (new retrofit capacity is reported in the previous paragraph)."*

EPA uses the Integrated Planning Model (IPM) to analyze the projected impact of environmental policies on the electric power sector in the 48 contiguous states and the District of Columbia. Developed by ICF Consulting, Inc. and used to support public and private sector clients, IPM is a multi-regional, dynamic, deterministic linear programming model of the U.S. electric power sector. It provides forecasts of least-cost capacity expansion, electricity dispatch, and emission control strategies for meeting energy demand and environmental, transmission, dispatch, and reliability constraints. IPM can be used to evaluate the cost and emissions impacts of proposed policies to limit emissions of sulfur dioxide (SO_2), nitrogen oxides (NO_x), carbon dioxide (CO_2), and mercury (Hg) from the electric power sector. The IPM was a key analytical tool in developing the Clean Air Interstate Rule (CAIR).

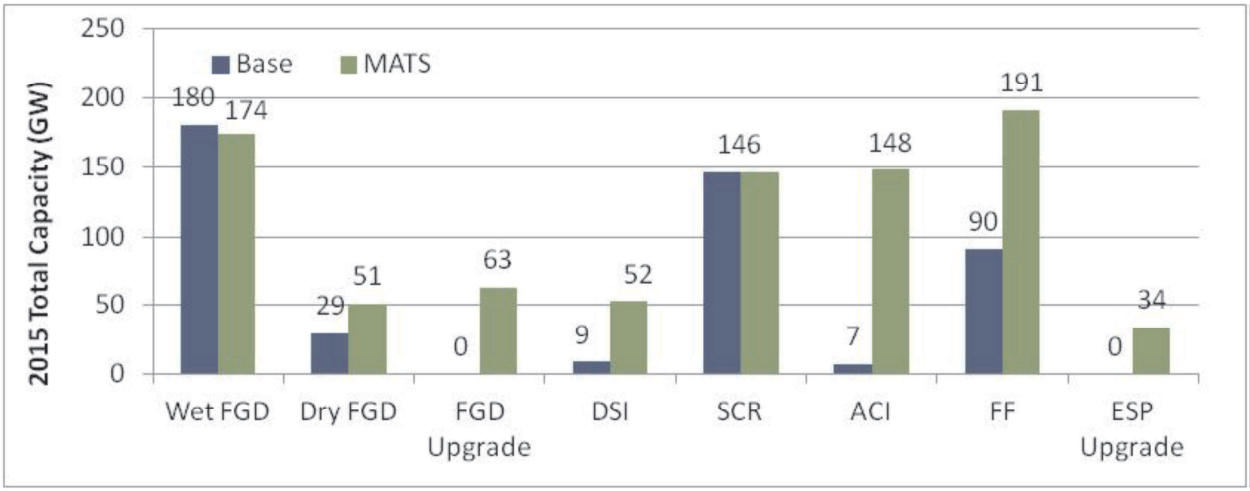
Among the factors that make IPM particularly well suited to model multi-emissions control programs are (1) its ability to capture complex interactions among the electric power, fuel, and environmental markets; (2) its detail-rich representation of emission control options encompassing a broad array of retrofit technologies along with emission reductions through fuel switching, changes in capacity mix and electricity dispatch strategies; and (3) its capability to model a variety of environmental market mechanisms, such as emissions caps, allowances, trading, and banking. IPM's ability to capture the dynamics of the allowance market and its provision of a wide range of emissions reduction options are particularly important for assessing the impact of multi-emissions environmental policies like CAIR.

http://www.epa.gov/airmarkets/progsregs/epa-ipm/

Now that we know what companies have done to comply with the MATS rule, we are in a position to determine how accurate this forecast was. There are a few things that stand out about the methods that were projected by EPA for industry to comply with the rule:

- The very high level of projected fabric filter systems
- The level of projected dry FGD systems
- The level of scrubber upgrades
- The high cost of dry sorbent injection ("DSI") and activated carbon injection ("ACI") systems that did not take account of technological advances reducing those costs
- The limited amount of fuel switching compared to actual levels driven by low shale gas prices

Figure 1. Operating Pollution Control Capacity on Coal-fired Capacity (by Technology) under the Base Case and with MATS, 2015 (GW)[2]



Fabric Filter - EPA's Air Markets Program Data shows only about 82 GW of Electric Utility or Small Power Producer Generation equipped with baghouses for particulate matter control at the end of second quarter 2015. Another 8.7 GW of fabric filter projects – not part of dry FGD projects - are underway with extensions for a total of perhaps 91 GW.[3] In other words, IPM overestimated the baghouse installations by about 100 GW (191 GW of total FF projected to be installed versus 91 GW) as shown in Figure 2. This is related to assumptions about DSI, dry FGD and the need for PM upgrades.

Dry FGD - IPM forecast 51 GW of dry FGD to be installed in the MATS policy case versus 29 GW in the Base Case when, in fact, AMPD data shows that at the end of second quarter 2015 there were only about 33 GW of dry FGD installed – or an overestimate of 18 GW as shown in Figure 2. Although there are an estimated 22 GW of dry FGD projects underway to be completed in the coming years and MATS extensions have been permitted associated with these projects,[3] these

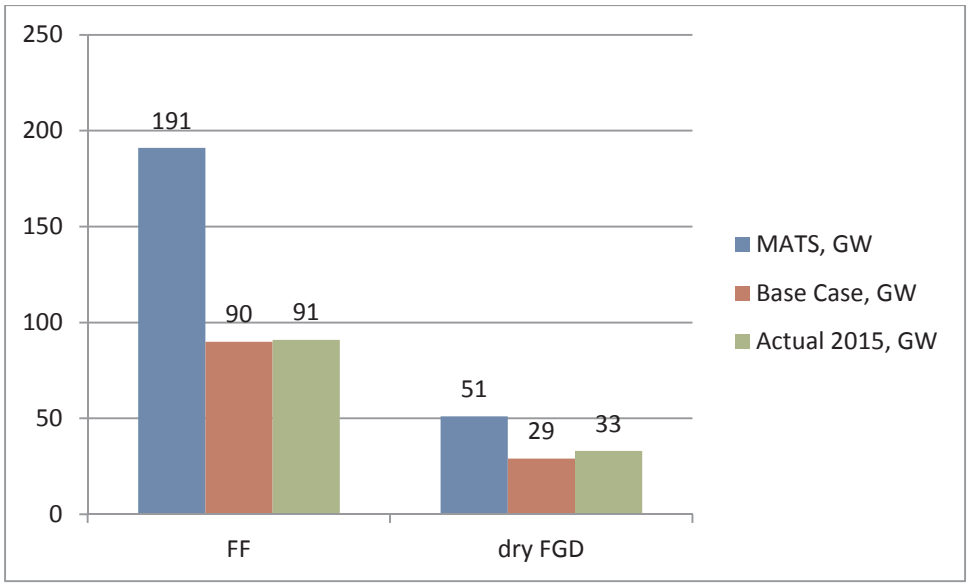
[2] Note: The difference between controlled capacity in the base case and under the MATS may not necessarily equal new retrofit construction, since controlled capacity above reflects incremental operation of dispatchable controls in 2015. Additionally, existing ACI installed on those units online before 2008 are not included in the base case to reflect removal of state mercury rules from IPM modeling. For these reasons, and due to rounding, numbers in the text below may not reflect the increments displayed in this figure. See IPM Documentation for more information on dispatchable controls.

[3] Michael J. Bradley and Associates, "MATS Compliance Extension Status Update", MJB&A Issue Brief, June 24, 2015. Examination of the data showed that of the 17 GW of FF with extensions, 8.3 GW were associated with dry FGD systems, leaving 8.7 GW of FF not associated with dry FGD.

dry FGD systems are primarily part of plans for compliance with the Regional Haze Rule or other SO_2 control requirements.

Scrubber upgrades – EPA's forecast of 63 GW in wet FGD upgrades is higher than actual. In 2015 there was about 170 GW of wet FGD installed on coal fired electric utility units or small power plants. On the other hand, a review of the Information Collection Request (ICR) data shows only about 7,600 MW of the roughly 52,000 MW of capacity with wet FGD installed that reported HCl emissions to the ICR, or about 15%, had HCl emissions in excess of the MATS limit. This would suggest only about 30 GW of FGD upgrades to be expected. About 16 GW of scrubber upgrades have been identified in applications for MATS extensions.[3] While there is no official data showing the level of wet FGD upgrades, it is reasonable to assume that at least 16 GW and no more than 30 GW of scrubber upgrades were performed. To that point, most of the FGD system upgrades were justified on the basis of improved SO_2 control for CAIR or CSAPR rather than MATS.

Figure 2. MATS and Base Case projections, and 2015 actual or planned installations of FF and dry FGD, expected to be directly a result of MATS, GW



The projected fixed and variable operating costs are also impacted by the type of equipment projected to be used and the assumed reagent usage rates for this equipment. Of particular concern with regard to variable operating cost are reagent usage assumptions relating to dry sorbent injection (DSI).
This Report will review each of the following as they relate to EPA's projection of cost to the MATS rule.

- Capital and operating cost projections relating to EPA forecasts for DSI
- Capital and operating cost projections relating to EPA forecasts for dry FGD
- Forecasts for PM control retrofits to fabric filters
- Forecasts for ACI variable operating and maintenance costs

- Fuel cost projections

Projections for the capital and operating costs for Dry Sorbent Injection (DSI)

In practice, DSI may be deployed for control of SO_3, HCl or SO_2. For SO_3 control the DSI system may be deployed in combination with an ACI system to enhance the Hg capture of the ACI system. On the other hand, IPM only forecasts DSI systems for MATS compliance as a means for controlling HCl. Therefore, many of the DSI systems installed to enhance Hg control in response to the MATS rule were not installed to control the pollutant EPA targeted DSI for. By and large, DSI systems for SO_3 control, however, are quite inexpensive to own and operate compared to those used for SO_2 or HCl control as a result of the comparatively very low reagent demand necessary to control SO_3. Therefore, the costs of the DSI systems associated with SO_3 capture can be ignored when compared against these other costs.

DSI capital cost

EPA's assumptions regarding use of a fabric filter in combination with DSI and EPA's assumptions about DSI treatment rates for controlling HCl introduce a number of issues. As described in Section 5.5.3 of the IPM documentation, EPA assumes that facilities that select DSI for reduction of HCl emissions always install a fabric filter. Treatment rate is assumed by EPA to be at a Normalized Stoichiometric Ratio of 1.55 using milled Trona per Appendix 5-4 of the IPM v4.10 documentation.[4] Experience has shown that lower treatment rates are possible without the need to retrofit a fabric filter.

Sodium based sorbents, such as Trona actually improve ESP capture efficiency due to the beneficial impact on fly ash resistivity making a fabric filter retrofit unnecessary. In fact, very few DSI systems that have been installed in response to the MATS rule entailed installation of a fabric filter. EPA's overestimation of fabric filters is due in part to the assumption that use of DSI for HCl control requires a baghouse. Assuming that the 9 GW of DSI forecast in the Base Case does not have FF, this means that IPM forecast at least an additional 43 GW of DSI that was equipped with FF (52 GW projected in the policy case versus 9 GW in the Base Case). Fabric filters increase the installed cost of a DSI system by a substantial amount – costing on the order of $150-$250/kW, depending upon the size of the facility and other factors.

Although EPA assumed that a fabric filter would be necessary for control of HCl, it is also worth examining the capital costs EPA uses for use of DSI upstream of an ESP, because this is by far the most common application of DSI. Appendix 5-4 of the IPM documentation describes the cost estimating approach developed by Sargent & Lundy for use in the IPM.[4] This methodology predicts capital costs of $40/kW for a 500 MW plant and costs well in excess of $100/kW for plants of about 100 MW in size. Discussions of these costs with both utilities and technology providers indicates pretty clearly that these capital cost estimates are well above what has been experienced in practice. This may be the result of the overestimation of Trona demand – that would necessitate more equipment than in fact is necessary.

[4] Sargent & Lundy, "IPM Model – Updates to Cost and Performance for APC Technologies Dry Sorbent Injection for SO2 Control Cost Development Methodology Final", August 2010 Project 12301-007

DSI operating costs

DSI operating costs are also lower than estimated. EPA assumed that DSI would provide 90% HCl removal and would require a normalized stoichiometric ratio (NSR) of 1.55 when using DSI in combination with a baghouse for capturing HCl. Studies by Solvay[5] showed DSI achieving over 98% HCl removal at much lower treatment rates. They examined several sorbents at different milling levels.

- Trona (S200) - d50 : 30 µm
- Milled Trona (S250) - d50 : 15 µm, d90 : 60 µm
- Milled Sodium Bicarbonate (S350) - d50 : 12 µm, d90 : 40 µm
- Finely Milled Sodium Bicarbonate (S450) - d50 : 7 µm ,d90 : 17 µm
- Hydrated Lime - d90 : 45 µm, purity: 96.8%

Figures 3a and 3b show the results of pilot tests performed with injection upstream of an ESP and Figures 4a and 4b show the results of pilot tests performed with injection upstream of a baghouse. As demonstrated by Figure 3a, 90% HCl capture was achieved with milled Trona (D250) with an NSR or roughly 0.3 and 99% capture was achieved with an NSR of roughly 0.6. This compares to an assumed forecast of 1.55 for 90% capture. EPA's assumed treatment rate at 90% removal was therefore almost five times what is shown in this data. As demonstrated in Figure 3a, with an ESP milled trona produced 90% capture at an NSR of about 0.35 and 99% capture with an NSR of about 0.70. However, in this case much better performance was provided by the more reactive sodium bicarbonate (S350 and S450). While any given facility may experience slightly different results than shown in these pilot tests, it is clear that whether using trona or sodium bicarbonate it is possible to achieve well in excess of 90% without a fabric filter at treatment rates well below those assumed by EPA.

SO_2 capture is normally well below that of HCl because SO_2 is slower to react, and Figures 3b and 4b confirm that. At treatment rates where milled trona is expected to achieve 90% HCl capture, roughly 20% SO_2 capture is expected, and at treatment rates where 99% HCl capture is achieved, roughly 40% SO_2 capture is expected. These significant levels of SO_2 capture are nonetheless lower than the 70% assumed by EPA.

Another aspect of operating costs is waste disposal. EPA assumes that the by-product must be disposed of at a much higher cost than normally used for landfill of coal combustion products. This is an unnecessary cost because sodium by product can be blended or neutralized and disposed of as a non-hazardous waste at a much lower cost. Moreover, if this were a sufficiently large concern, the facility owner could use calcium-based reagent, such as hydrated lime, which produces a highly stable product.

Other factors that caused the IPM forecast of fabric filters to be too high was the result of overestimation of dry FGD, overestimation of waste disposal costs associated with ACI, and underestimation of the ability of existing ESPs to achieve the MATS PM emission standard with simple upgrades.

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[5]. Yougen Kong, Mike Wood, Solvay Chemicals Inc.," HCl Removal in the Presence of SO2 Using Dry Sodium Sorbent Injection", Houston, Texas, available at www.solvay.com

Figure 3a. HCl removal with injection upstream of an ESP

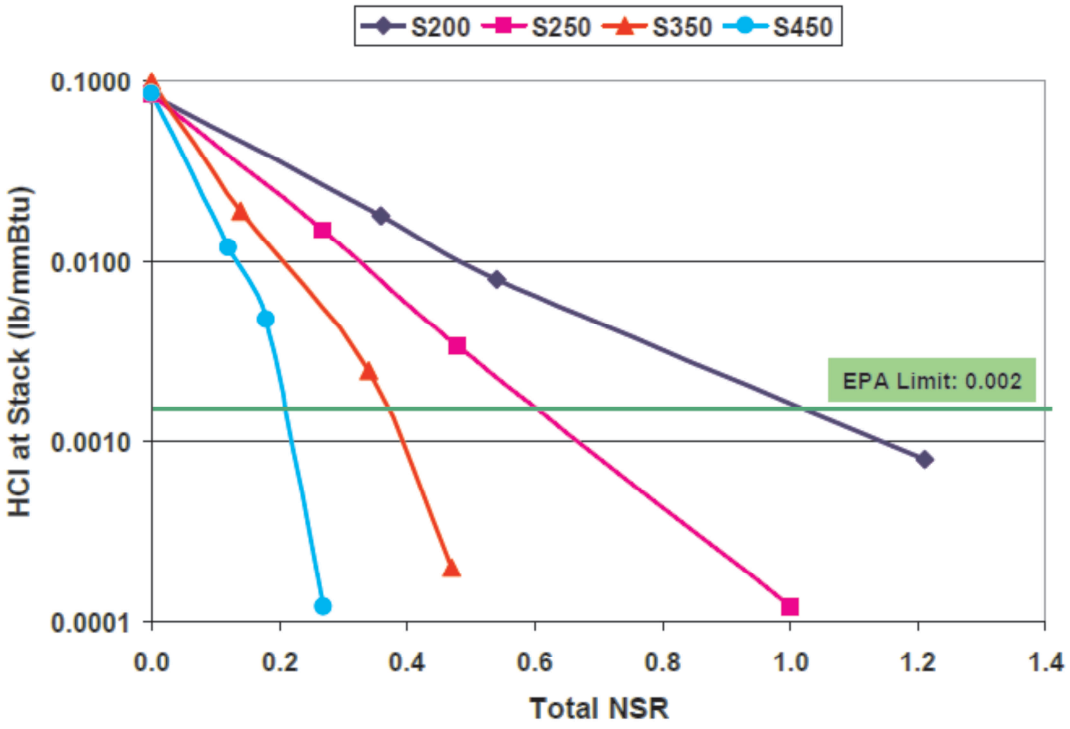


Figure 3b. SO_2 reduction with injection upstream of an ESP

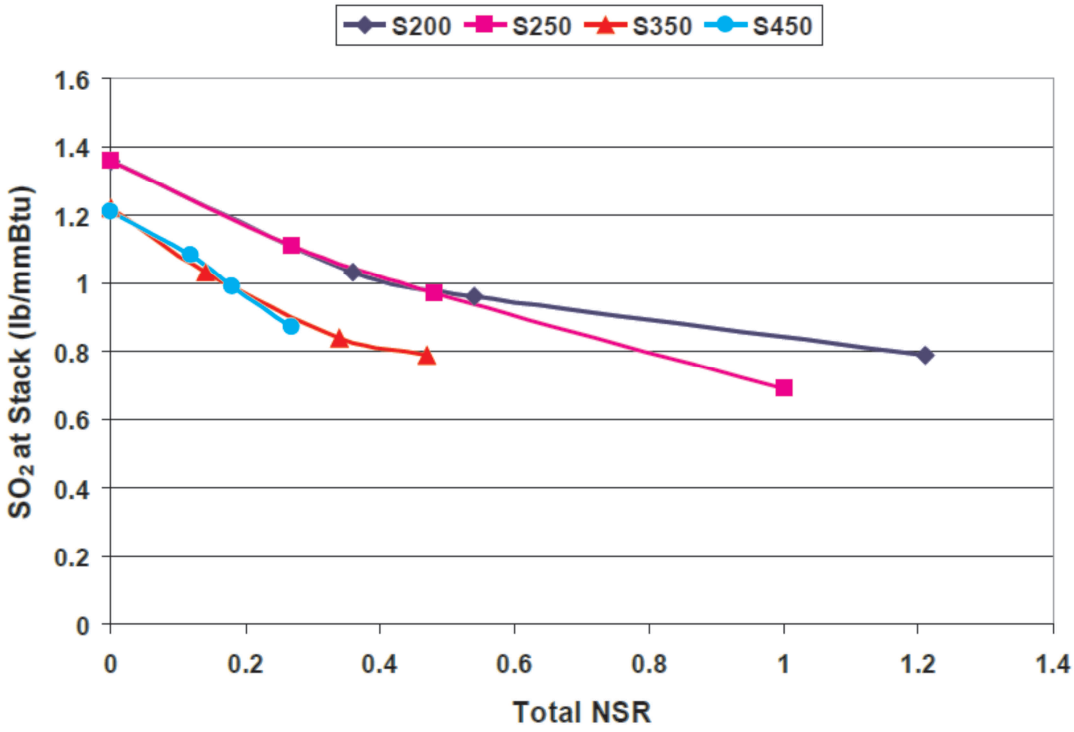


Figure 4a. HCl removal with injection upstream of baghouse

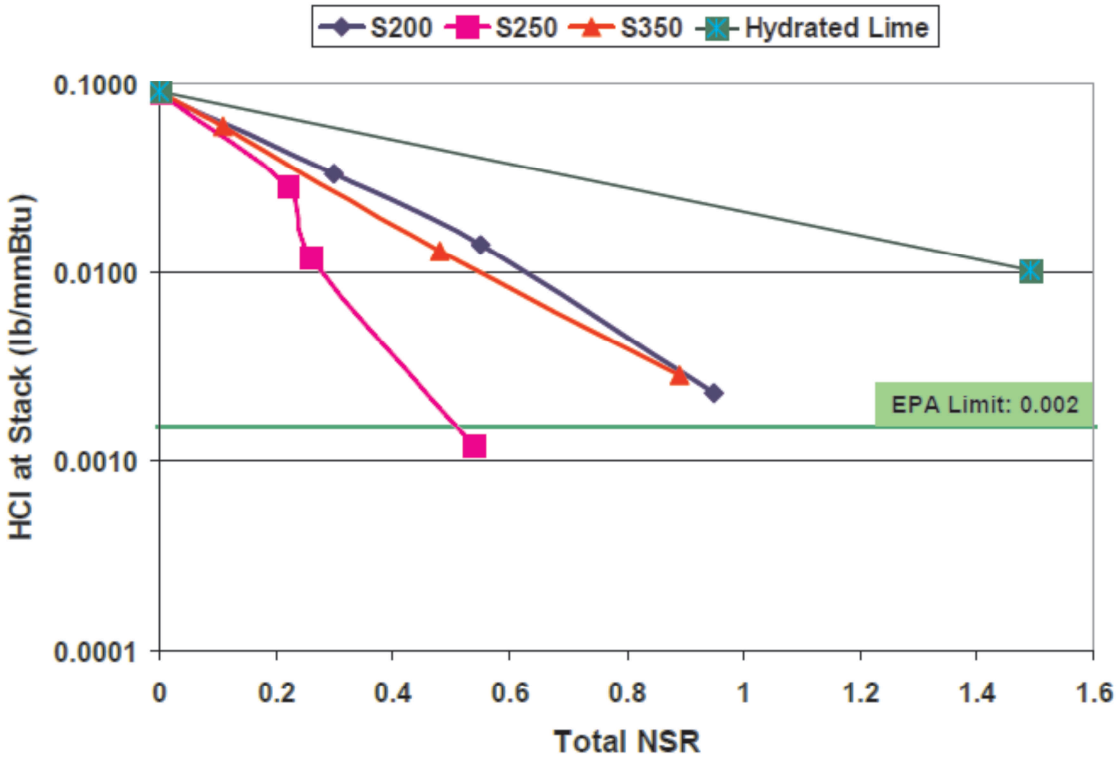
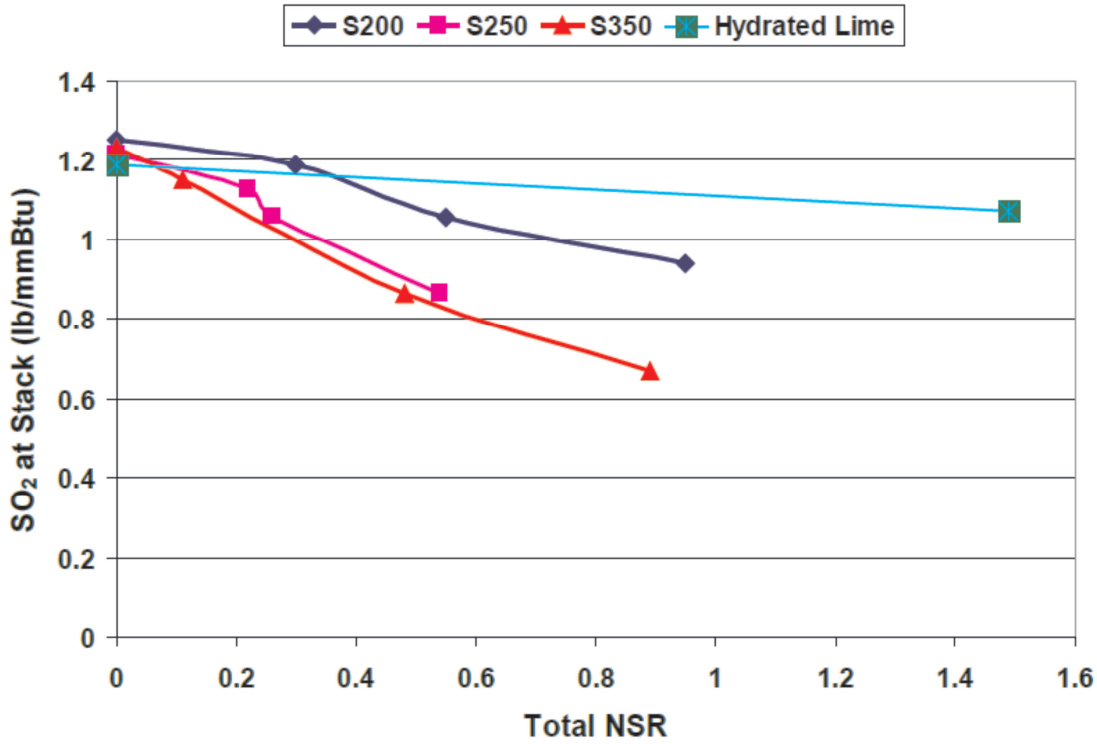


Figure 4b. SO_2 reduction with injection upstream of a baghouse



Projections for dry FGD

Dry FGD systems are commonly installed with fabric filters. As a result, an overestimation of dry FGD installations will result in an overestimation of fabric filter installations. The reason for the high forecast for dry FGD is likely the result of forecasts for DSI costs with a fabric filter (that may have made the incremental cost for dry FGD more acceptable) or the assumption by EPA that DSI is limited to only 90% HCl capture (that would force dry FGD to be selected by the IPM if greater than 90% HCl reduction was necessary). These assumptions would cause IPM to project that companies would select dry FGD for acid gas control rather than DSI in situations where DSI is, in fact, capable of providing adequate acid gas control. But, the effects of DSI and dry FGD can explain about 65 GW[6] of the roughly 100 GW of FF that were forecast but are not actually installed.

Projections for PM control

EPA's assumptions regarding DSI and dry FGD do not adequately explain the overestimation of fabric filters in their MATS cost estimate. EPA also made assumptions about the need to retrofit fabric filters for PM control to meet the MATS PM standard or for use in ACI systems. The assumptions for PM were used in a spreadsheet to identify facilities projected to need upgrade of their ESP or retrofit of a fabric filter. The projection developed with the spreadsheet was exogenously input to the IPM model to determine if improvement in PM collection efficiency was needed and, if so, what kind of improvement would be performed and what it would cost. In this manner that spreadsheet determined if a PM retrofit with a baghouse was necessary or if ESP upgrade was adequate. The approach used apparently underestimated the ability of the existing ESP to achieve the MATS PM emission standard. In fact, most ESPs were capable of achieving the emission standard without any modifications or with relatively modest changes – at most changes to the transformer rectifier sets and perhaps electrodes. In many cases rebalancing of flows was adequate at minimal cost.

The result is that EPA projected more fabric filter retrofits than were, in fact, built. EPA's modeling attributes 101 GW of FF to MATS versus the Base Case, some of which are attributed to dry scrubbers. Moreover, EPA also likely overestimated the cost of modifying existing ESPs to comply with the regulation. ATP's estimate of the market size for ESP upgrades in 2014 was only in the range of about $50 million based upon interviews with discussions with suppliers of these services and equipment.

ACI variable operating and maintenance costs

According to Appendix 5-3 to Chapter 5 of the IPM documentation,[7] EPA assumes that when activated carbon and fly ash are collected in the same PM control device that the cost of disposal for all solids – fly ash and activated carbon – are increased. The effect is that the projected cost of waste disposal exceeds that of the carbon sorbent – more than doubling the VOM. This is based upon the presumption that addition of activated carbon renders beneficial reuse of fly ash impossible. In practice, this does not

[6] 22 GW of additional dry FGD for MATS versus the Base Case plus 43 GW of additional FF on DSI for MATS versus the Base Case

[7] Sargent & Lundy, "IPM Model – Revisions to Cost and Performance for APC Technologies Mercury Control Cost Development Methodology, Final", March 2011, Project 12301-009

happen. First, despite the desirability of beneficially reusing fly ash as a concrete additive, in practice most fly ash is not used for this purpose because of local market conditions or other reasons. Furthermore, activated carbon suppliers have developed "cement friendly" carbons that do not have the adverse impact of conventional carbons. The assumption that waste disposal costs increase so much may also partially account for the overestimate of fabric filters, as installation of an additional fabric filter would facilitate segregation of fly ash from activated carbon.

EPA also overestimated the ACI that is attributable to MATS – 148 GW of ACI forecast for MATS versus 7 GW in the Base Case. According to ATP's estimates, *at least* 20 GW of ACI was in operation in 2014, clearly well over the 7 GW attributed by EPA to the Base Case. Furthermore, EPA's estimate of 148 GW of ACI exceeds somewhat ATP's estimates of total ACI systems, which is about 120 GW once MATS is fully implemented. ATP estimates that with the rule fully implemented, about 100 GW of ACI is attributable to MATS.

Fuel Costs

Facility owners will convert to natural gas or switch to higher cost coal if in their estimation this is a less costly approach to complying with the MATS rule. EPA's forecast Policy Case projected a cost of natural gas in 2015 of $5.66/MMBtu versus $5.40/MMBtu in its Base Case. Data from the Energy Information Administration indicates that in 2015 natural gas to utility customers has ranged from a high of $4.99/thousand cubic feet down to $3.24/thousand cubic feet, or about $4.99/MMBtu to about $3.24/MMBtu because a cubic foot of gas has very close to 1,000 Btu's of energy. Therefore, much lower natural gas prices than forecast by EPA have made gas a much more attractive fuel and has resulted in the cost of compliance with the rule to be much lower.

Impact on cost

A rough estimate of the impact on cost of the various assumptions addressed in this memo is shown in Table 1. This shows the estimated excess costs associated with:

- the fabric filter overestimate that is not associated with dry FGD,
- the overestimate of dry FGD
- the overestimate of reagent consumption associated with DSI
- the overestimate of capital cost associated with wet FGD upgrades,
- the overestimate associated with waste disposal assumptions for ACI,
- an adjustment to account for the underestimate of carbon use if the facilities that are assumed to install TOXECON systems do not,
- the overestimate of the ACI systems attributable to the MATS rule

Section 8 of the IPM documentation states that a capital charge rate of 11.3% is used for environmental retrofits, which is what is used to determine amortized capital charges. the assumed capacity factor is 65%. Cost estimates are developed using capital costs ($/kW), VOM ($/MWh) and FOM ($/kW-yr) rates taken from the IPM v4.10 documentation used to develop the MATS rule. The fabric filter overestimate

is clearly the most significant, followed by the overestimate of dry FGD and the overestimate associated with DSI.

The overestimate of FF that is not explained by dry FGD is 82 GW. 43 GW of this is explained by DSI attributed to MATS, leaving 40 GW unexplained by DSI or dry FGD. This results in an additional 40 GW that can be ACI systems in TOXECON arrangements. As a result, there are roughly 101 GW (141 GW – 40 GW) that are ACI systems without TOXECON that where waste-disposal costs are overestimated. This is offset in part by the underestimate of sorbent costs if the 40 GW of forecast TOXECON systems are made to be conventional ACI systems upstream of an ESP.

Table 1. Approximate overestimate of costs

	FF [1]	dry FGD [2]	DSI [3]	wet FGD upgrade [4]	Wet FGD [5]	ACI Waste [6]	ACI carbon [7]	ACI excess [8]	Total
million $	$16,072	$8,838	$0	$4,700	$992	$0	$0	$414	$31,016
Annualized, capital, million $	$1,816	$999	$0	$531	$112	$0	$0	$47	$3,505
Operating costs, million $	$102	$391	$1,400	$0	$37	$1,196	-$207	$798	$3,718
Million $	$1,918	$1,390	$1,400	$531	$149	$1,196	-$207	$845	$7,223

Notes:

1. *The overestimate of FF is the amount over actual installations that is not explained by dry FGD*
2. *Dry FGD estimate for excess dry FGD over actual installed*
3. *DSI estimate assumes that actual reagent is roughly one third of EPA assumption.*
4. *Wet FGD upgrade assumes 30 GW of actual upgrade versus 63 GW predicted. No formal data is available.*
5. *The actual reduction in wet FGD versus the Base Case was greater than forecast by EPA*
6. *Accounts for EPA assumption about fly ash waste for facilities where fly ash is collected with carbon*
7. *Accounts for higher carbon demand from units with ESP versus TOXECON. EPA assumed more TOXECON installations, which include new baghouses.*
8. *Accounts for overestimate of ACI installations after rule is fully implemented. Only includes carbon for VOM as waste already addressed.*

Conclusion

Experience with technologies deployed for MATS compliance has shown them to be less expensive and more effective than originally assumed in EPA's analysis. As a result, the true cost of complying with the MATS rule is more than $7 billion per year less than estimated by EPA, making the true cost of the rule about one quarter of what EPA originally estimated the rule to cost.