January 15, 2016

By Electronic Submission to www.regulations.gov

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

RE: Comments on Proposed Supplemental Finding that it is Appropriate and Necessary to Regulate Hazardous Air Pollutants from Coal- and Oil-Fired Electric Utility Steam Generating Units

Elsie M. Sunderland, Charles T. Driscoll, Jr., James K. Hammitt, Philippe Grandjean, John S. Evans, Joel D. Blum, Celia Y. Chen, David C. Evers, Daniel A. Jaffe, Robert P. Mason, and the Emmett Environmental Law & Policy Clinic¹ welcome the opportunity to comment on the Environmental Protection Agency's (EPA) proposed supplemental finding. The signatories are academics with expertise in ecology, economics, chemistry, environmental science, and environmental law. Collectively, this group contains state-of-the-science expertise in the atmospheric transport, aquatic fate, bioaccumulation, human exposures, and health outcomes associated with mercury contamination of the environment and have authored many of the papers cited in this comment.

We strongly support EPA's conclusion that the consideration of cost confirms the agency's previous conclusion that it is appropriate and necessary to regulate coal- and oil-fired electric steam generating units (EGUs) under section 112 of the Clean Air Act (CAA). Based on the evidence before the agency, EPA correctly concluded that the monetized benefits for all air pollutants (both direct benefits and co-benefits) associated with the Mercury and Air Toxics Standards (MATS) range between \$37 and \$90 billion and far exceed the costs of regulation.

Moreover, both the scientific community and the EPA have repeatedly emphasized the existence of many additional, significant, unquantified benefits of this regulation that further outweigh the costs. Even preliminary efforts to monetize these benefits suggest they are substantially greater than the costs of the proposed regulation. Thus, even taking cost into account, the rule should stand.

Although EGUs release a variety of hazardous air pollutants (HAPs), to demonstrate these points our comments will focus specifically on the benefits associated with reducing emissions of mercury and exposures to its organic form, methylmercury, which is formed in aquatic systems and bioaccumulates in food webs. Based on our close scrutiny of the

¹ Each signatory's area of expertise is identified in the signature block at the end of this document.

peer-reviewed scientific literature, we find the monetized benefits for EGU mercury emissions reductions identified by EPA in the regulatory impact analysis (RIA) supporting MATS <u>vastly understate the benefits associated with reductions of those emissions</u>.

Specifically we elaborate upon three key points below:

- Recent research demonstrates that quantified societal benefits associated with declines in mercury deposition attributable to implementation of MATS are much larger than the amount estimated by the EPA in 2011.
- As-yet unquantified benefits to human health and wildlife from reductions in EGU mercury emissions are substantial.
- Contributions of EGUs to locally deposited mercury have been underestimated by EPA's regulatory assessments.

1) Quantified societal benefits associated with declines in mercury deposition attributable to implementation of MATS are much larger than the amount estimated by the EPA in 2011.

Due to data limitations and gaps in the available research, EPA's RIA only considered a small subset of the public health and environmental risks associated with mercury emissions from EGUs. Specifically, the EPA monetized the value of IQ losses for children born to a limited population of recreational fishers who consume freshwater fish during pregnancy from watersheds where EPA had fish tissue data. The monetized value of benefits for this small subpopulation was between \$4 and \$6 million annually.

If one considers instead all of the benefits of reducing EGU mercury emissions, recent research confirms that the benefits are orders of magnitude greater than those quantified by the EPA in 2011. One study found that the cumulative U.S. economy-wide benefits associated with implementation of MATS exceed \$43 billion.² This value is far greater than the EPA's estimate of the costs associated with the regulation. Other work has estimated an annual benefit of \$860 million associated with a 10% reduction in methylmercury exposure in the U.S. population.³

² A. Giang & N.E. Selin, Benefits of mercury controls for the United States, *PNAS (Early Edition*) (2015), http://www.pnas.org/cgi/doi/10.1073/pnas.1514395113. Lower bound estimates from this paper that includes only IQ deficits related to methylmercury exposure among freshwater fish consumers are similar to the values derived by the EPA.

³ G.E. Rice, et al., A probabilistic characterization of the health benefits of reducing methyl mercury intake in the United States, *Envtl. Sci. Tech.*, 44: 5216-5224 (2010).

2) As-yet unquantified benefits to human health and wildlife are substantial.

Part of the reason that these estimates are so much greater than the quantified benefits identified in EPA's RIA is that they take into account additional types of benefits from reducing EGU mercury emissions. For example, many of these benefits are associated with adverse impacts of methylmercury on cardiovascular health. It was reasonable for EPA not to quantify cardiovascular effects in the RIA because, at that time, there was a split in the scientific evidence regarding the significance of those impacts. On one side, an independent expert panel in 2011 asserted there is sufficient scientific evidence to incorporate these outcomes in regulatory assessments.⁴ On the other, a high-profile study of risks of cardiovascular disease associated with methylmercury exposures in two U.S. cohorts found no evidence of adverse effects.⁵

There are several reasons, however, to conclude that the cardiovascular impacts are substantial despite the latter study. First, the study included only low-to-moderate fish consumers and therefore lacked the statistical power to detect effects seen in studies that included a greater range in exposures (e.g., ⁶). Second, it can be challenging to isolate the neurodevelopmental and cardiovascular impacts of methylmercury exposure from seafood consumption because seafood also contains long-chained fatty acids (eicosapentaenoic acid) and docosahexaenoic acid) that serve to mask those deleterious impacts.⁷ These confounding effects make it difficult for some epidemiological studies to identify the negative health outcomes associated with methylmercury exposures against the background of beneficial effects of consuming long-chained fatty acids in seafood. However, this does not imply that methylmercury on its own is not harmful, or that it does not reduce the benefits of an otherwise healthy food source.⁸ In addition, imprecision in exposure biomarkers causes many epidemiological studies to be biased toward a null

⁴ H.A. Roman, et al., Evaluation of the cardiovascular effects of methylmercury exposures: Current evidence supports development of a dose-response function for regulatory benefits analysis, *Envtl. Health Persp.*, 119(5): 607-614 (2011).

⁵ D. Mozaffarian, et al., Mercury exposure and cardiovascular disease in two U.S. cohorts, *New England J. Med.* 364: 1116-1125 (2011).

⁶ A.L. Choi, et al., Methylmercury exposure and adverse cardiovascular effects in Faroese whaling men, *Envtl. Health Persp.*, 117(3): 367-372 (2009); B. Valera, et al., Impact of mercury exposure on blood pressure and cardiac autonomic activity among Cree adults (James Bay, Quebec, Canada), *Envtl. Res.* 11(8): 1265-1270 (2011); M. Wennberg, et al., Myocardial infarction in relation to mercury and fatty acids from fish: a riskbenefit analysis based on pooled Finnish and Swedish data in men, *Am. J. Clinical Nutrition*, 96: 706-713 (2012); M.O. Gribble, et al., Mercury exposure and heart rate variability: a systematic review, *Current Envtl. Health Reports*, 2: 304-314 (2015).

⁷ K.R. Mahaffey, et al., Balancing the benefits of n-3 polyunsaturated fatty acids and risks of methylmercury exposure from fish consumption, *Nutrition Revs.*, 69(9): 493-508 (2011); 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(10): 1171-1181 (2008).

⁸ P.W. Davidson, et al., Neurodevelopmental effects of maternal nutritional status and exposure to methylmercury from eating fish during pregnancy, *NeuroToxicology*, 29: 767-775 (2008); M.L. Lynch, et al., Varying coefficient function models to explore interactions between maternal nutritional status and prenatal methylmercury toxicity in the Seychelles Child Development Nutrition Study, *Envtl. Res.*, 111: 75-80 (2011).

finding rather than detection of adverse effects.⁹ We note that failure to find a statistically significant effect is not evidence that no such effect exists, though it may provide evidence that constrains the magnitude of the effect.

Although EPA's RIA did quantify one type of neurological effect (IQ loss) among one group of fish consumers, its consideration of neurodevelopmental benefits from the proposed rule is incomplete. For example, the assessment did not consider benefits associated with reductions in methylmercury in coastal U.S. fisheries. It therefore significantly underestimates the neurodevelopmental benefits of the rule, because marine fish account for >90% of methylmercury intake by the U.S. population.¹⁰ These benefits are difficult to quantify because such quantification requires attributing changes in methylmercury exposure from domestic, international, and natural sources of mercury. Nevertheless, many species of marine fish eaten by Americans spend a large portion of their lifecycle foraging in coastal U.S. domestic waters of the Atlantic (including the Gulf of Mexico) and Pacific Oceans. Recent research suggests the regulation of domestic U.S. mercury emissions will have a substantial effect on mercury inputs to coastal waters (see point [3] below). For example, a recent study reported marked decreases in mercury in Atlantic coastal fisheries in response to decreases in mercury emissions.¹¹

Furthermore, recent epidemiological data have revealed a suite of more sensitive neurodevelopmental effects than full-IQ, the impact valued in the EPA's 2011 RIA. Even the original National Academy of Sciences Panel on the *Toxicological Effects of Methylmercury* conceded that full-IQ was not the most sensitive indicator of neurodevelopment.¹² In addition, neurodevelopmental impacts of methylmercury have more recently been documented at exposure levels below the reference dose established by the NRC Panel in 2000.¹³ Similar to lead exposure, there is no evidence from epidemiological studies for a health effects threshold, below which neurodevelopmental effects do not occur.¹⁴ As a result, compared with the RIA, a full quantification of the neurodevelopmental impacts of EGU mercury emissions would take into account both other kinds of fish consumption and effects other than reductions in IQ.

¹³ M. Bellanger, et al., Economic benefits of methylmercury exposure control in Europe: Monetary value of neurotoxicity prevention, *Envtl. Health*, 12:3, doi: 10.1186/1476-069X-12-3 (2013).

⁹ P. Grandjean & E. Budtz-Jørgensen, Total imprecision of exposure biomarkers: Implications for calculating exposure limits, *Am. J. Indus. Med.*, 50: 712-719 (2007).

¹⁰ E.M. Sunderland, Mercury exposure from domestic and imported estuarine and marine fish in the U.S. seafood market, *Envtl. Health Persp.*, 115(2): 235-242 (2007).

¹¹ F.A. Cross, et al., Decadal declines of mercury in adult bluefish (1972-2011) from the mid-Atlantic coast of the U.S.A., *Envtl. Sci. Tech.*, 49: 9064-9072 (2015).

¹² National Research Council, *Toxicological Effects of Methylmercury*. Board on Environmental Studies and Toxicology. ISBN: DOI: 10.17226/9899, 368 pp. (2000).

¹⁴ M.R. Karagas, et al., Evidence on the human health effects of low level methylmercury exposure, *Envtl. Health Persp.*, 120(6): 799-806 (2012); P. Grandjean, et al., Calculation of mercury's effects on neurodevelopment. *Envtl. Health Persp.*, 120 (12): A 452 (2012).

Many other benefits of regulating mercury emissions from EGUs have not been monetized on a national scale due to the heterogeneity in effects across ecosystems, lack of data, and challenges associated with monetization. These additional benefits include:

- Reductions in the deleterious impacts of methylmercury exposure on endocrine function¹⁵, risk of diabetes¹⁶, and compromised immune health.¹⁷
- Benefits to fish and wildlife, including sensitive bird species (songbirds, loons), marine mammals, fish, and amphibian populations threatened by high levels of mercury contamination in many U.S. ecosystems. Emerging research on the ecological impacts of methylmercury exposures indicates that adverse effects on the reproductive and behavioral health of wildlife populations occur at low levels of environmental exposure.¹⁸

3) Contributions of EGUs to locally deposited mercury have been underestimated by EPA's regulatory assessments.

The RIA supporting MATS also underestimates the benefits of reducing EGU mercury emissions because it is based on an underestimation of the portion of those emissions that are deposited to the land and waters of U.S. ecosystems. Human and ecological health risks associated with utility-derived mercury emissions are greatest in regions that are most affected by locally deposited mercury. Some of the mercury emissions from EGUs are highly water-soluble and locally deposited while the rest are emitted into the atmosphere as a stable, long-lived species that is transported and distributed globally.

Benefits of MATS in terms of declines in mercury deposition to U.S. ecosystems in the RIA were based on atmospheric modeling that suggested global (non-U.S.) anthropogenic sources would be most important for regional declines in deposition. However, for the past two decades, mercury researchers have noted slow and steady declines in atmospheric mercury concentrations in North America, Europe, and over the open oceans. Initial attempts to rationalize these observations from a scientific perspective were confounded by a commonly held (but incorrect) assumption among researchers that global mercury emission trends from anthropogenic sources were steady or increasing over this same time period. Zhang et al. recently corrected an error in previous emissions inventories on the form of mercury released by EGUs over time. This correction helps enable global models to

¹⁵ S.W. Tan, et al., The endocrine effects of mercury in humans and wildlife, *Critical Revs. Toxicology*, 29: 228-269 (2009).

¹⁶ K. He, et al., Mercury exposure in young adulthood and incidence of diabetes later in life: the CARDIA trace element study, *Diabetes Care*, 36: 1584-1589 (2013).

¹⁷ J.F. Nyland, et al., Biomarkers of methylmercury exposure and immunotoxicity among fish consumers in the Amazonian Brazil, *Envtl. Health Persp.*, 119(12): 1733-1738 (2011).

¹⁸ D.C. Depew, et al., Toxicity of dietary methylmercury to fish: derivation of ecologically meaningful threshold concentrations, *Envtl. Toxicology Chemistry*, 31(7): 1536-1547 (2012); D.C. Depew et al., Derivation of screening benchmarks for dietary methylmercury exposure for the common loon (*Gavia immer*): Rationale for use in ecological risk assessment, *Envtl. Toxicology Chemistry*, 31(10): 2399-2407 (2012).

reproduce the observed declining atmospheric mercury trends.¹⁹ This analysis shows spatial and temporal trends in atmospheric mercury concentrations and deposition are much more influenced by local and regional actions than previously assumed.

Other new studies also support the premise that declining mercury emissions in the United States will substantially reduce mercury deposition and biological exposures in U.S. ecosystems and hence to U.S. populations. For example, several U.S. studies have measured substantial declines in domestic atmospheric and ecologic mercury concentrations attributable to reductions in mercury emissions from EGUs. Castro and Sherwell observed declines in atmospheric mercury concentrations at a pristine site in Maryland downwind of power plants in Ohio, Pennsylvania, and West Virginia.²⁰ Drevnick et al. observed a mean $\sim 20\%$ decline in mercury accumulation in 104 sediment cores from the Great Lakes regions attributable to domestic emissions reductions.²¹ Evers et al. identified biological mercury hotspots in the northeastern United States driven mainly by U.S. domestic emissions.²² Similarly, Hutcheson et al. noted declines in methylmercury concentrations in freshwater fish in the United States concurrent with domestic mercury emissions reduction.²³ Cross et al. report marked decreases in mercury in Atlantic coastal fisheries in response to decreases in mercury emissions.²⁴

Together, these new studies demonstrate that declines in mercury deposition to U.S. ecosystems and resulting human and ecological exposures have been underestimated by the 2011 regulatory impact assessment performed by EPA.

Thank you for your consideration of these comments. We welcome the opportunity to discuss this important matter with you at any time. Please direct follow up communications to Elsie Sunderland, 617-496-0858 (ems@seas.harvard.edu) or Shaun Goho, 617-496-5692 (sgoho@law.harvard.edu).

¹⁹ Y. Zhang, et al., Observed decrease in atmospheric mercury explained by global decline in anthropogenic emissions, *PNAS Early Edition* (2016), http://www.pnas.org/cgi/doi/10.1073/pnas.1516312113.

²⁰ M.S. Castro & J. Sherwell, Effectiveness of emission controls to reduce the atmospheric concentrations of mercury, *Envtl. Sci. Tech.*, 49(24): 14000-14007 (2015).

²¹ P.E. Drevnick et al., Spatial and temporal patterns of mercury accumulation in lacustrine sediments across the Great Lakes region. *Environmental Pollution*. 161: 252-260 (2012).

²² D.C. Evers, et al., , Biological mercury hotspots in the northeastern United States and southeastern Canada, *Bioscience*, 57(1): 29-43 (2007).

²³ M.S. Hutcheson, et al., Temporal and spatial trends in freshwater fish tissue mercury concentrations associated with mercury emissions reductions, *Envtl. Sci. Tech.*, 48: 2193-2202 (2014).

²⁴ Cross et al., *supra* note 11.

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