Benefits of Regulating Hazardous Air Pollutants from Coal and Oil-Fired Utilities in the United States

Citation	Sunderland, Elsie M., Charles T. Driscoll, James K. Hammitt, Philippe Grandjean, John S. Evans, Joel D. Blum, Celia Y. Chen, et al. 2016. "Benefits of Regulating Hazardous Air Pollutants from Coal and Oil-Fired Utilities in the United States." Environmental Science & Technology 50 (5) (February 5): 2117–2120. doi:10.1021/acs.est.6b00239.
Published Version	10.1021/acs.est.6b00239
Citable link	http://nrs.harvard.edu/urn-3:HUL.InstRepos:37221734
Terms of Use	This article was downloaded from Harvard University's DASH repository, and is made available under the terms and conditions applicable to Open Access Policy Articles, as set forth at http://nrs.harvard.edu/urn-3:HUL.InstRepos:dash.current.terms-of-use#OAP

Benefits of Regulating Hazardous Air Pollutants from Coal and Oil-Fired Utilities in the United States

Elsie M. Sunderland[1,2,], Charles T. Driscoll, Jr.[3,*], James K. Hammitt[2], Philippe Grandjean[2], John S. Evans[2], Joel D. Blum[4], Celia Y. Chen[5], David C. Evers[6], Daniel A. Jaffe[7], Robert P. Mason[8], Shaun Goho[9], and Wendy Jacobs[9]*

[1] John A. Paulson School of Engineering and Applied Sciences, Harvard University, Cambridge MA, 02138 United States

[2] T.H. Chan School of Public Health, Harvard University, Cambridge MA, 02138 United States

[3] Department of Civil and Environmental Engineering, Syracuse University, Syracuse, NY 13244 United States

[4] Department of Earth and Environmental Sciences, University of Michigan, Ann Arbor, MI 48109 United States

[5] Department of Biological Sciences, Dartmouth College, Hanover, NH 03755 United States

[6] Biodiversity Research Institute, Portland, ME 04103 United States

[7] Science and Technology Program, University of Washington-Bothell, Bothell, WA 98011

[8] Department of Marine Sciences, University of Connecticut, Groton, CT 06340 United States

[9] Emmett Environmental Law & Policy Clinic, Harvard Law School, Harvard University, Cambridge MA, 02138 United States

[*] Corresponding authors: ems@seas.harvard.edu and ctdrisco@syr.edu

22 

On June 29, 2015, the U.S. Supreme Court ruled that the Environmental Protection Agency (EPA) acted unreasonably when it determined that cost was irrelevant to deciding whether it was "appropriate" to regulate emissions of Hazardous Air Pollutants (HAPs) from coal and oil-fired utilities (EGUs) (U.S. Supreme Court, *Michigan v. EPA,* 2015). According to the 1990 Clean Air Act Amendments, EPA must make a preliminary determination, known as the "appropriate and necessary" finding, before regulating EGUs. The Court ruled that EPA made a mistake at this preliminary stage and sent the regulation, known as the Mercury and Air Toxics Standards (MATS), back to the agency and ordered EPA to consider costs. The public comment period for this proposal closed on January 15, 2016 and EPA aims to issue a final cost consideration and renewed "appropriate and necessary" finding by April 15, 2016.

In its 2011 regulatory assessment[1], EPA concluded that the monetized benefits for all air pollutants (both direct benefits and co-benefits) associated with MATS range between $37 and $90 billion and far exceed the costs of regulation. However, most of these quantified benefits come from reductions in particulate emissions. Monetized benefits associated with reducing HAP emissions in EPA's regulatory assessment ranged between $4-$6 million, leading some critics to argue that the rule was unreasonable. However, both the scientific community and EPA have repeatedly emphasized the 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.

Although EGUs release a variety of HAPs, we will focus specifically on the benefits associated with reducing emissions of mercury and exposures to its organic form, methylmercury, which is formed in aquatic ecosystems and bioaccumulates in food webs. Based on recent peer-reviewed scientific literature, we find the monetized benefits for EGU mercury emissions reductions identified by EPA in the regulatory impact analysis supporting MATS vastly understate the benefits associated with reductions of those emissions.

Specifically we elaborate upon three key points below:

1. 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 EPA in 2011.
2. As-yet-unquantified benefits to human health and wildlife from reductions in EGU mercury emissions are substantial.
3. 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.[1]

Due to data limitations and gaps in the available research, EPA's regulatory assessment only considered a small subset of the public health and environmental risks associated with mercury emissions from EGUs. Specifically, 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 estimated between $4 and $6 million annually.[1]

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 EPA in 2011. One study found that the cumulative U.S. economy-wide benefits associated with implementation of MATS exceeded $43 billion.[2] This value is far greater than 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. As-yet unquantified benefits to human health and wildlife are substantial.

In part these estimates are so much greater than the quantified benefits identified in EPA's regulatory assessment because they consider 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. EPA did not quantify cardiovascular effects in the regulatory assessment. 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 is 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, 8] 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 exposures to methylmercury on its own are not harmful, or that it does not reduce the benefits of an otherwise healthy food source.[9, 10] In addition, imprecision in exposure biomarkers biases many epidemiological studies toward a null finding rather than detection of adverse effects.[11] 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 regulatory assessment 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.[12] These benefits are difficult to quantify because they require 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 (Gulf of Mexico, Atlantic and Pacific coastal waters). 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.[13]

Furthermore, recent epidemiological data have revealed a suite of more sensitive neurodevelopmental effects than full-IQ, the impact valued in EPA's 2011 regulatory assessment. 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.[14] 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.[15] Similar to lead exposure, there is no evidence from epidemiological studies for a health effects threshold, below which neurodevelopmental effects do not occur.[16, 17] As a result, compared with EPA's regulatory assessment, a full quantification of the neurodevelopmental impacts of EGU mercury emissions would need to take into account both other kinds of fish consumption and effects other than reductions in IQ.

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 function,[18] risk of diabetes,[19] and compromised immune health.[20]

- 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.[21, 22]

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

The regulatory assessment supporting MATS[1] also underestimates the benefits of reducing EGU mercury emissions because it underestimated 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 to the atmosphere as a stable, long-lived species that is transported and distributed globally.

Benefits of MATS associated with declines in mercury deposition to U.S. ecosystems in the regulatory assessment 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.[23] 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 reproduce the observed declining atmospheric mercury trends and shows that local and regional mercury deposition to U.S. ecosystems is much more influenced by domestic 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[24] observed declines in atmospheric mercury concentrations at a pristine site in Maryland downwind of power plants in Ohio, Pennsylvania, and West Virginia. Drevnick et al.[25] observed a mean ~20% decline in mercury accumulation in 104 sediment cores from the Great Lakes regions attributable to domestic emissions reductions. Evers et al.[26] identified biological mercury hotspots in the northeastern United States driven mainly by U.S. domestic emissions. Similarly, Hutcheson et al.[27] noted declines in methylmercury concentrations in freshwater fish in the United States concurrent with domestic mercury emissions reduction. Cross et al.[13] report marked decreases in mercury in Atlantic coastal fisheries in response to decreases in mercury emissions.

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.

References

(1) U.S. Environmental Protection Agency (2011), Regulatory Impact Analysis for the Final Mercury and Air Toxics Standards (US Environmental Protection Agency, Office of Air Quality Planning and Standards, Research Triangle Park, NC). EPA-452/R-11-011. Available: http://www3.epa.gov/mats/pdfs/20111221MATSfinalRIA.pdf.

(2) Giang, A., Selin, N. E. Benefits of mercury controls for the United States. *Proc Natl Acad Sci USA Early Edition* **2015;** http://www.pnas.org/cgi/doi/10.1073/pnas.1514395113.

(3) Rice, G., Hammitt, J., Evans, J. A probabilistic characterization of the health benefits of reducing methyl mercury intake in the United States *Environ. Sci. Technol.* **2010,** *44*, 5216-5224.

(4) Roman, H. A., Walsh, T. L., Coull, B. A., Dewailly, É., Guallar, E., Hattis, D., Mariën, K., Schwartz, J., Stern, A. H., Virtanen, J. K., Rice, G. Evaluation of the cardiovascular effects of methylmercury exposures: Current evidence supports development of a dose–response function for regulatory benefits analysis. *Environ Health Perspect* **2011,** *119*, (5), 607-614.

(5) Mozaffarian, D., Shi, P., Morris, S. J., Spiegelman, D., Grandjean, P., Siscovick, D. S., Williett, W. C., Rimm, E. B. Mercury exposure and risk of cadivascular disease in two U.S. cohorts. *New England Journal of Medicine* **2011,** *364*, 1116-1125.

(6) Choi, A. L.; Weihe, P.; Budtz-Jorgensen, E.; Jorgensen, P. J.; Salonen, J. T.; Tuomainen, T. P.; Murata, K.; Nielsen, H. P.; Petersen, M. S.; Askham, J.; Grandjean, P. Methylmercury exposure and adverse cardiovascular effects in faroese whaling men. *Environ Health Perspect* **2009,** *117*, (3), 367-372.

(7) Mahaffey, K. R.; Sunderland, E. M.; Chan, H. M.; Choi, A. L.; Grandjean, P.; Marien, K.; Oken, E.; Sakamoto, M.; Schoeny, R.; Weihe, P.; Yan, C. H.; Yasutake, A. Balancing the benefits of n-3 polyunsaturated fatty acids and the risks of methylmercury exposure from fish consumption. *Nutr. Rev.* **2011,** *69*, (9), 493-508.

(8) Oken, E.; Radesky, J. S.; Wright, R. O.; Bellinger, D. C.; Amarasiriwardena, C. J.; Kleinman, K. P.; Hu, H.; Gillman, M. W. Maternal fish intake during pregnancy, blood mercury levels, and child cognition at age 3 years in a US cohort. *Am. J. Epidemiol.* **2008,** *167*, (10), 1171-1181.

(9) Davidson, P. W.; Strain, J. J.; Myers, G. J.; Thurston, S. W.; Bonham, M. P.; Shamlaye, C. F.; Stokes-Riner, A.; Wallace, J. M. W.; Robson, P. J.; Duffy, E. M.; Georger, L. A.; Sloane-Reeves, J.; Cernichiari, E.; Canfield, R. L.; Cox, C.; Huang, L. S.; Janciuras, J.; Clarkson, T. W.

Neurodevelopmental effects of maternal nutritional status and exposure to methylmercury from eating fish during pregnancy. *Neurotoxicology* **2008,** *29*, (5), 767-775.

(10) Lynch, M. L.; Huang, L.-S.; Cox, C.; Strain, J. J.; Myers, G. J.; Bonham, M. P.; Shamlaye, C. F.; Stokes-Riner, A.; Wallace, J. M. W.; Duffy, E. M.; Clarkson, T. W.; Davidson, P. W., Varying coefficient models to explore interactions between maternal nutritional status and prenatal methylmercury exposure in the Seychelles Child Development Nutrition Study. *Environmental Research* **2011,** *111*, (1), 75-80.

(11) Grandjean, P.; Budtz-Jorgensen, E., An ignored risk factor in toxicology: The total imprecision of exposure assessment. *Pure Appl. Chem.* **2010,** *82*(2), 383-391.

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

(13) Cross, F. A.; Evans, D. W.; Barber, R. T., Decadal declines of mercury in adult bluefish (1972-2011) from the mid-Atlantic coast of the U.S.A. *Environ. Sci. Technol.* **2015,** *49*, 9064-9072.

(14) NRC, *Toxicological Effects of Methylmercury*. National Academy Press: Washington, DC, 2000; p 368.

(15) Bellanger, M.; Pichery, C.; Aerts, D.; Berglund, M.; Castano, A.; Cejchanova, M.; Crettaz, P.; Davidson, F.; Esteban, M.; Fischer, M. E.; Gurzau, A. E.; Halzlova, K.; Katsonouri, A.; Knudsen, L. E.; Kolossa-Gehring, M.; Koppen, G.; Ligocka, D.; Miklavcic, A.; Reis, M. F.; Rudnai, P.; Tratnik, J. S.; Weihe, P.; Budtz-Jorgensen, E.; Grandjean, P., Economic benefits of methylmercury control in Europe: Monetary value of neurotoxicity prevention. *Environmental Health* **2013,** *12*, (3), doi: 10.1186/1476-069X-12-3.

(16) Karagas, M. R.; Choi, A. L.; Oken, E.; Horvat, M.; Schoeny, R.; Kamai, E.; Cowell, W.; Grandjean, P.; Korrick, S., Evidence on the human health effects of low-level methylmercury exposure. *Environ Health Perspect* **2012,** *120*, (6), 799-806.

(17) Grandjean, P.; Pichery, C.; Bellanger, M.; Budtz-Jorgensen, E., Calculation of mercury's effect on neurodevelopment. *Environ Health Perspect* **2012,** *120*, (12), A452.

(18) Tan, S. W.; Meiller, J. C.; Mahaffey, K. R., The endocrine effects of mercury in humans and wildlife. *Critical Reviews in Toxicology* **2009,** *39*, (3), 228-269.

(19) He, K.; Xun, P., Liu, K., Morris, S., Reis, J., Guallar, E. Mercury exposure in young adulthood and incidence of diabetes later in life: the CARDIA trace element study, *Diabetes Care*, 36: 1584-1589 (2013).

(20) J.F. Nyland, Fillon, M, Barbosa, R., Jr., Shirley, D.L., Chine, C., Lemire, M., Mergler, D., Sibergeld, E.K. Biomarkers of methylmercury exposure and immunotoxicity among fish consumers in the Amazonian Brazil, *Envtl. Health Persp.*, 119(12): 1733-1738 (2011).

(21) Depew, D.C.; Basu, N.; Burgess, N.M.; Campbell, L.M.; Devin, E.W.; Drevnick, P.E.; Hammerschmidt, C.R.; Murphy, C.A.; Sandheinrich, M.B.; Wiener, J.G. Toxicity of dietary methylmercury to fish: derivation of ecologically meaningful threshold concentrations. *Envtl. Toxicology Chemistry* **2012**, *31*(7): 1536-1547.

(22) Depew, D.C.; Basu, N.; Burgess, N.M.; Campbell, L.M.; Evers, D.C.; Grasman, K.A.; Scheuhammer, A.M. Derivation of screening benchmarks for dietary methylmercury exposure for the common loon (*Gavia immer*): Rationale for use in ecological risk assessment. *Envtl. Toxicology Chemistry* **2012** *31*(10): 2399-2407.

(23) Zhang, Y.; Jacob, D.J.; Horowitz, H.M.; Chen, L.; Amos, H.M.; Krabbenhoft, D.P.; Slemr, F.; St. Louis, V.; Sunderland, E.M. 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.

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

(25) Drevnick, P.E.; Engstrom, D.R.; Driscoll, C.T.; Swain, E.B.; Balogh, S.J.; Kamman, N.C.; Long, D.T.; Parsons, M.J.; Rolfhus, K.R.; Rossmann, R. Spatial and temporal patterns of mercury accumulation in lacustrine sediments across the Great Lakes region. *Environ. Poll.* **2012** *161*: 252-260.

(26) Evers, D.C.; Han, Y-J.; Driscoll, C.T.; Kamman, N.C.; Goodale, W.; Fallon Lambert, K.; Holsen, T.; Chen, C.Y.; Clair, T.A.; Butler, T. Biological mercury hotspots in the northeastern United States and southeastern Canada. *Bioscience* **2007**, *57*(1): 29-43.

(27) Hutcheson, M.S.; Smith, M.C.; Rose, J.; Batdorf, C.; Pancorbo, O.; West, C.R.; Strube, J.; Francis, C. Temporal and spatial trends in freshwater fish tissue mercury concentrations associated with mercury emissions reductions. *Envtl. Sci. Tech.* **2014**, *48*: 2193-2202.