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A Regulatory Analysis of EPA's Proposed Rule to Reduce Mercury Emissions from Utility Boilers

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Executive Summary

EPA's proposed rule for mercury reductions from coal-fired utility boilers is unlikely to provide significant health benefits, both because mercury exposure at current levels is unlikely to be causing harm, and because even in a best-case scenario the mercury rule could reduce mercury in fish by no more than a few percent.

The claim that reducing mercury in fish will reduce neurological harm to fetuses of exposed pregnant women is based on the assumptions that the results of an epidemiologic study of mothers and children in the Faroe Islands represents a genuine cause-effect relationship between low-level mercury exposure and children's neurological health, and that Faroes-like effects would occur in Americans even at mercury exposures as low as $1/15^{th}$ the minimum level associated with health effects in the Faroes study.

But even accepting these assumptions at face value, the reported health effects are subtle and at current American mercury exposure levels have no implications for general neurological or cognitive health. For example, based on the Faroes results, a complete elimination of U.S. utility mercury emissions could, in a best-case scenario, move children who are at, say, the 10^{th} percentile on neurological and cognitive test scores to between the 10.3 and 10.6 percentiles. Even this small improvement is unrealistically optimistic, because it also assumes a one-to-one correspondence between mercury emission reductions and mercury levels in freshwater fish, and that people with high mercury exposures receive all of their mercury from non-commercial, freshwater fish.

Furthermore, a similar study of children in the Seychelles reported no harm from mercury exposures several times higher than even relatively highly exposed Americans. The Seychelles study may be more relevant to Americans, because people in the Seychelles are exposed to mercury through eating ocean fish, while people in the Faroe Islands are exposed through eating whale blubber.

EPA's mercury rule is thus likely to provide few or no health benefits. On the other hand, EPA estimates even modest utility mercury reductions will cost about $1.4 billion per year. These costs will in part be passed through to consumers, reducing the resources available for other health- and welfare-enhancing expenditures.

If EPA still wishes to go forward with utility regulations, rather than regulate mercury directly, EPA should scrap both its mercury rule and its companion Interstate Air Quality Rule, which would regulate utility nitrogen oxides and sulfur dioxide (SO_2), and instead require reductions only in SO_2 emissions.

SO_2 reductions will reduce mercury levels in fish by reducing sulfate levels in lakes and streams, which reduces methylmercury formation—the form of mercury that gets into fish and to which people are exposed. Furthermore, the SO_2 reductions will reduce mercury in freshwater fish regardless of where the mercury in fish is coming from and will cost less than mercury reductions. SO_2 reductions will also reduce sulfate particulate matter and regional haze, and the measures necessary to reduce SO_2 emissions will also modestly reduce mercury emissions.

While the costs of additional SO_2 reductions might still outweigh their benefits, the cost-benefit picture for utility SO_2 reductions is far superior to even the most generous best-case benefit estimates for EPA's proposed utility mercury rule.

A Regulatory Analysis of EPA's Proposed Rule to Reduce Mercury Emissions from Utility Boilers

Joel Schwartz

1. Introduction

EPA's proposed rule for mercury reductions from coal-fired utility boilers (Mercury Rule) is unlikely to provide significant health benefits, both because mercury exposure at current levels is unlikely to be causing harm, and because even in a best-case scenario the Mercury Rule could reduce mercury in fish by no more than a few percent.

The debate over the benefits of power plant mercury emission reductions hinges on (1) the extent to which such reductions will reduce fish mercury levels, (2) whether reductions in fish mercury levels will improve health, and (3) whether there are cheaper ways to achieve reductions in fish mercury levels. In other words, does the regulation maximize net benefits to Americans when compared with other potential policy choices regarding power plant mercury emissions.

The assumption that reducing mercury in fish will reduce neurological harm to fetuses of exposed women is based on several assumptions, including the following:

- The results of an epidemiologic study of mothers and children in the Faroe Islands, where people are exposed to mercury mainly from eating whale blubber, represents a genuine cause-effect relationship between low-level mercury exposure and children's neurological health.
- The Faroe Islands study is applicable to Americans, who are exposed to mercury mainly from eating fish, rather than through whale blubber.
- Mercury exposure causes neurological and cognitive harm to all people exposed to mercury even at levels as low as $1/15^{th}$ the minimum level associated with health effects in the Faroe Islands study, even though at most a minute fraction of all people would be expected to be so sensitive to mercury's effects.

But even accepting these three assumptions at face value, the reported health effects are subtle and at current American mercury exposure levels have no implications for general neurological or cognitive health. For example, under these three assumptions, a complete elimination of U.S. utility mercury emissions could, in a best-case scenario, improve average neurological and cognitive test scores of the most highly exposed American children (0.5% of all children) by about $1/35^{th}$ to $1/65^{th}$ of a standard deviation, depending on the specific test. This is the equivalent of moving from, say, the 10^{th} percentile to between the 10.3 and 10.6 percentiles in test performance. In more practical terms, this is equivalent to developmental gains of perhaps one to two weeks in cognitive and neurological performance. Improvements would be smaller for children with lower initial mercury exposures. But even this small improvement is unrealistically optimistic, because it also includes the following additional unrealistic assumptions:

- There is a one-to-one correspondence between mercury emission reductions and mercury levels in freshwater fish, when observations suggest that fish mercury levels would decline by half or less of the amount of mercury deposition reductions.
- People with high mercury exposures receive all of their mercury from non-commercial, freshwater fish, which is the only source of mercury exposure that might be reduced through utility mercury reductions.

Furthermore, a similar study of children in the Seychelles reported no harm from mercury exposures several times higher than even relatively highly exposed Americans. The Seychelles study may be more relevant to Americans, because people in the Seychelles are exposed to mercury through eating ocean fish—that is, in a similar manner to Americans.

EPA acknowledges the uncertainty of mercury benefits and instead justifies the Mercury Rule mainly on its co-benefits in reducing emissions of sulfur dioxide (SO_2) and nitrogen oxides (NOx) and touts its "integrated" approach to regulating mercury, and through the companion Interstate Air Quality Rule (IAQR), also regulating NOx and SO_2 from power plants at the same time.

The purported co-benefits are in turn mainly a result of reductions in mortality and other health effects due to reductions in particulate matter (PM) that would result from the SO2

reductions, and, to a lesser extent, from the NOx reductions. EPA attributes less than 0.1 % of the benefits of these rules to reductions in ozone. However, because toxicology studies suggest sulfate and nitrate PM are not toxic, even at levels substantially higher than current levels, these benefits may also fail to materialize.

The health effects claims for PM are instead based on epidemiological studies that reported statistical associations between PM and health outcomes. Yet critiques of these studies suggest they suffer from confounding and other statistical and methodological problems that may render their results spurious. In developing its PM co-benefit estimates, EPA also ignored epidemiologic studies that reported no association between PM and mortality. In any case, if EPA believes SO_2 reductions are what account for the vast majority of health and welfare benefits of the combined Mercury Rule and IAQR, then the agency should focus on SO_2 reductions and reject NOx and mercury reductions.

The Mercury Rule and the IAQR are thus likely to provide minimal health benefits. On the other hand, the combined rules will by EPA's estimate cost nearly $3 billion per year in 2010 and $4.7 billion in 2020. These costs will in part be passed through to consumers, reducing their disposable income. Reducing families' disposable income causes offsetting health damage of its own and should be weighed against any purported benefits of the two rules.

If EPA still wishes to go forward with utility regulations, rather than regulate mercury directly, EPA should scrap both the Mercury Rule and the Interstate Air Quality Rule and instead require reductions only in SO_2 emissions. Such reductions will reduce mercury levels in fish by reducing sulfate levels in lakes and streams, which reduces methylmercury formation—the form of mercury that gets into fish and to which people are exposed.

Reducing SO_2 emissions is likely to cause much larger fish mercury reductions than would reducing utility mercury emissions directly. Utility mercury emissions account for at most 11% of U.S. mercury deposition. Thus, even if all mercury in fish is coming from current mercury emissions, the Mercury Rule will have little effect.

To the extent that mercury in fish is coming from mercury already in the environment, the rule's effects will be that much smaller. It should also be noted that these best-case effects of reducing power plant mercury emissions apply only to freshwater fish. Ocean fish mercury levels would not be affected at all by the Mercury Rule.

On the other hand, SO_2 reductions will reduce mercury in freshwater fish regardless of where the mercury is coming from, by reducing the rate of methylmercury formation, and will cost less than mercury reductions. SO_2 reductions will also reduce sulfate PM and regional haze, and the measures necessary to reduce SO_2 emissions will also modestly reduce mercury emissions. While the costs of additional SO_2 reductions might still outweigh their benefits, the cost-benefit picture for utility SO_2 reductions is far superior to even the most generous best-case benefit estimates for the Mercury Rule.

2. Background on EPA's Proposed Power Plant Rules

On December 20, 2000 EPA issued a finding that it is "appropriate and necessary" to regulate hazardous air pollutants (HAP) from utility boilers.[1] By making this finding EPA committed to issuing a regulation under Section 112 of the Clean Air Act (CAA) requiring Maximum Achievable Control Technology (MACT) for utility boiler mercury emissions. For a given source category and pollutant, CAA Section 112 defines MACT for existing sources as the average level of pollution control achieved by the best 12% of existing sources in a given source category.

Pollution Reduction Requirements

The Mercury Rule actually includes two alternative proposals for how the agency would regulate mercury emissions from utility boilers, as follows:

Require utilities to install controls representing MACT under Section 112 of the Clean Air Act

Utilities would be regulated under Section 112 of the Clean Air Act in a similar fashion to other industrial sources of HAP. Mercury emissions would be capped at 34 tons per year in 2007. This represents a reduction of 14 tons per year or 29% below current estimated levels.[2]

EPA has also proposed a cap-and-trade alternative to traditional MACT. Under this alternative, the universe of utilities would have to meet the MACT reduction requirement overall, but individual facilities could meet their emission reduction obligation either by reducing

[1] 65 FR 79826
[2] 69 FR 4712

emissions on-site or obtaining emission permits from other facilities that have achieved reductions. Emissions would go down the required level overall, but facilities that could reduce mercury most cheaply would account for the reductions, rather than all facilities being required to meet a uniform mercury emission rate.

Establish "standards of performance" limiting mercury emissions from new and existing utilities under Section 111 of the Clean Air Act

Utilities would be regulated under Section 111 of the Clean Air Act through a two-phase cap-and-trade program, capping total mercury emissions at 15 tons per year in 2018 when the Phase II requirement would come into effect. This represents a reduction of 33 tons per year, or 69% below current estimated emissions. The mercury cap for Phase I would come into effect in 2010 and would apparently require mercury reductions equal to the reductions achieved as a co-benefit of reductions required by the Interstate Air Quality Rule for SO_2 and NOx, which EPA estimates to be 14 tons per year, or 29% below current estimated levels.[3] New sources would have to meet an emissions rate standard in addition to obtaining emission permits for any remaining emissions.

If EPA elects to implement the second alternative, the agency would rescind its December 20, 2000 "appropriate and necessary" finding, which would remove the requirement that utility mercury emissions be regulated under the CAA's Section 112 MACT requirement.

The IAQR companion rule would require reductions in SO_2 and NOx from utility boilers in the eastern half of the U.S., where coal-fired utility boilers are a common electricity source. EPA estimates that utility boilers in the IAQR region emitted 9.4 million tons of SO_2 in 2002 and would have baseline emissions of 9 million tons in 2010.[4] The IAQR would cap emissions 57% lower, at 3.9 million tons in 2010, and 2.7 million tons in 2015.

For NOx, EPA estimates that utilities emitted 3.7 million tons in 2002 and that baseline emissions would be 3.1 million in 2010. The IAQR would cap emissions 48% below this, at 1.6 million tons in 2010, and 1.3 million tons in 2015.

[3] Ibid.
[4] 69 FR 4586

Costs and Benefits

For the IAQR, EPA projects that the annual incremental control costs would be $2.9 billion in 2010, $3.7 billion in 2015, and $4.9 billion in 2020, and that the full social costs would be the same as the direct control costs.[5] These control costs would include reductions in mercury equal to the reductions under the Section 112 MACT proposal, though in this case the reductions are assumed to be achieved as an ancillary benefit of measures taken to control NOx and SO_2.[6]

If implemented without the IAQR, EPA estimates that its Section 112 mercury MACT proposal would cost $1.36 billion per year in direct control costs and $1.6 billion in overall social costs.[7] These costs would be incurred due to measures necessary to reduce mercury emissions alone, on the assumptions that the IAQR SO_2 and NOx reductions are not implemented, and that each individual source would have to meet the MACT emission rate.

EPA does not estimate the monetary benefits of mercury reductions, arguing that the benefits are real, but too uncertain to put a number on. For SO_2 and NOx reductions, EPA estimates the IAQR will confer benefits worth $58 billion in 2010 and $84 billion in 2015.[8] Of these benefits, 98.2% are due to the presumed health and welfare benefits of reducing particulate matter, mainly due to reductions in mortality. The remaining benefits are due to improvements in visibility. Ozone reductions account for less than 0.1% of EPA's benefit estimate.

Of the benefits of PM reductions, EPA attributes 85.2% to sulfate reductions and 13.4% to nitrate reductions.[9] The remaining benefits are attributed to reductions in organic PM. Because most $PM_{2.5}$ monitoring locations already attain EPA's annual and 24-hour $PM_{2.5}$ standards, and because already-adopted requirements for motor vehicles and power plants will achieve substantial additional reductions in emissions of $PM_{2.5}$ and $PM_{2.5}$ precursors over the next several years,[10] most of the benefits EPA attributes to PM reductions are due to reductions from initial levels that already comply with EPA's $PM_{2.5}$ standards. In other words, EPA assumes that there is no threshold below which PM does not cause increased mortality.

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[5] 69 FR 4646 and Environmental Protection Agency, *Economic & Energy Analysis for the Proposed Interstate Air Quality Rulemaking* (Washington, DC: January 28, 2004), http://www.epa.gov/air/interstateairquality/tm0009.pdf.
[6] 69 FR 4712
[7] 69 FR 4706 and 69 FR 4712
[8] 69 FR 4646
[9] Environmental Protection Agency, *Benefit Analysis for the Section 112 Utility Rule* (Washington, DC: January 2004), http://www.epa.gov/ttn/atw/utility/proposalutilitymactbenefitsanalysisfinal.pdf.
[10] J. Schwartz, *No Way Back: Why Air Pollution Will Continue to Decline* (Washington, DC: American Enterprise Institute, July 2003), http://www.aei.org/docLib/20030804_4.pdf.

Given its estimates of costs and benefits, EPA concludes the IAQR will confer net annual social benefits of $55 billion in 2010 and $80 billion in 2015.

3. Mercury in the Environment

EPA's preamble and technical documentation for its Mercury Rule suffer from lack of context on current mercury emissions, trends in mercury emissions over time, and sources of mercury deposition in the United States. A key factor for policy is that the U.S. has reduced its mercury air emissions by about 70% during the last 15 years and probably by 80% to 90% since the early 1980s. To the extent that U.S. mercury reductions will reduce mercury levels in fish, almost all of the potential benefits of those reductions have therefore already been achieved.

Analysis of the sources of mercury deposition suggests that most deposition in the continental U.S. comes from a combination of anthropogenic sources outside the U.S. and from natural emissions. Results of a recent deposition modeling study lead to the conclusion that a complete elimination of mercury from U.S. coal-fired utility boilers would reduce mercury deposition in the U.S. by at most about 11%.[11]

EPA is remiss in not providing this important context. Such information should be provided as part of the preamble and technical documentation if EPA chooses to finalize the Mercury Rule. EPA should also estimate the rule's likely effects on mercury deposition. Doing so would provide key information on the maximum benefits that could be achieved from implementing the Mercury Rule, making the policy issues in the rulemaking more transparent.

Current Mercury Emission Inventory

EPA estimates that in 1999 coal-fired utility boilers emitted 47.9 tons of mercury per year, or about 41% of total U.S. anthropogenic emissions of 117 tons per year.[12] Figure 1 summarizes U.S. mercury sources. Sources emitting at least two tons per year are listed separately, with other source categories are grouped by how much they emit each year.

EPA estimates worldwide mercury emissions are about 5,000 to 5,500 tons per year, with about 40% of this coming from current anthropogenic sources, 40% from re-emission of mercury

[11] C. Seigneur et al., "Global Source Attribution for Mercury Deposition in the United States," *Environmental Science & Technology*, vol. 38, no. 2 (2003), pp. 555-569.

already in the environment due to human activities,[13] and 20% from natural sources.[14] Recent estimates in the scientific literature are somewhat higher, ranging from 6,600 to 7,000 tons per year for total emissions, with current anthropogenic emissions ranging from 2,200 to 2,600 tons per year.[15] In either case, U.S. anthropogenic emissions represent roughly 2%, and utility boilers one percent, of total world mercury emissions.

Mercury Emission Trends

Current mercury emissions are far lower than past levels. In its Mercury Study Report to Congress, EPA estimated 158 tons of mercury were emitted in 1995.[16] That inventory did not include emissions from gold ores, which are included in the 1999 inventory. Assuming gold-ore emissions were the same in previous years, the 1995 inventory would be 170 tons.

Emissions were substantially higher in 1989 and 1990, due to much higher emissions from waste incineration and roasting and milling of mercury ores. Researchers from the United States Geological Survey (USGS) estimate that compared with 1995, emissions from waste incineration were 47 tons/year higher in 1990 and 162 tons/year higher in 1989.[17] Likewise, total emissions from mercury ore milling and roasting were virtually zero in 1996, but 72 tons in 1990. The USGS was not able to determine how much of the mining emissions were air emissions vs. emissions to water or land.

Assuming emissions from other sources were the same in 1989/90 as in 1995, the 1989 and 1990 inventories would be, respectively, 374 and 259 tons/year.[18] The large decreases from the late 1980s to the early 1990s are due to drastic reductions in the amount of mercury in the waste stream coming into waste incinerators and to regulatory limits on mercury emissions from

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[12] EPA 1999 national emission inventory data available at http://www.epa.gov/ttn/chief/net/1999inventory.html.

[13] Most mercury in the environment from human activities is due to past mercury emissions. Re-emission refers to the fact that some of this mercury, as well as mercury from past natural emissions, is continually cycled between land, air, and water, allowing past mercury emissions to be transported by air and redeposited. These mercury re-emissions are not affected by current mercury policy.

[14] 69 FR 4658

[15] Sources for these estimates are cited in Seigneur et al., "Global Source Attribution for Mercury Deposition in the United States."

[16] Environmental Protection Agency, *Mercury Study Report to Congress Volume II: An Inventory of Anthropogenic Mercury Emissions in the United States* (Washington, DC: December 1997), http://www.epa.gov/ttn/oarpg/t3/reports/volume2.pdf.

[17] J. L. Sznopek and T. G. Goonan, *The Materials Flow of Mercury in the Economies of the United States and the World* (United States Geological Survey, 2000), http://pubs.usgs.gov/circ/2000/c1197/c1197.pdf, S. M. Jasinski, *The Materials Flow of Mercury in the United States* (United States Bureau of Mines, 1994), http://pubs.usgs.gov/usbmic/ic-9412/mercury.pdf.

waste incineration. In addition, mercury ore mining in the U.S. had essentially stopped by the mid-1990s.[19]

I was not able to locate national emissions estimates for years prior to 1989, however, other evidence suggests emissions were substantially higher than in 1989. Mercury consumption was about 70% higher in the late 1970s and early 1980s when compared with 1989.[20] A mercury emissions inventory trend compiled for Florida concluded that emissions were 3.7 times higher in 1980 than in 1990, with the declines coming mainly from waste incineration.[21]

Figure 2 displays the estimated inventories for 1989 through 1999, along with a rough guess at the potential range of the inventory in the early 1980s, based on overall U.S. mercury use and the Florida inventory. Mercury emissions declined 70% between 1989 and 1999, and probably by as much as 80% to 90% since the early 1980s.

Sources of Mercury Deposition

The potential effect of utility boiler mercury reductions depends on the extent to which utility mercury emissions contribute to mercury deposition in the United States and in other areas where they could affect fish mercury levels and the extent to which current mercury deposition affects current fish mercury levels.

Mercury deposition around the United States is relatively poorly understood. A recent study attempted to shed light on this issue by apportioning mercury deposition among natural sources, North American anthropogenic sources, and other anthropogenic sources.[22] The study concluded that, on average, North American mercury sources contribute about 25 to 32 percent mercury deposition in the continental U.S. At only three of 19 deposition measurement sites did North American emissions contribute more than half of all mercury deposition. Natural emissions contributed an average of 25 to 45 percent of total mercury deposition, while anthropogenic emissions from Asia contributed 20 to 30 percent. The ranges result from a sensitivity analysis regarding the fraction of total mercury emissions due to natural vs. anthropogenic sources. The results were based on mercury emissions estimates for 1998.

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[18] For this estimate, I assumed that two-thirds of the mining emissions were air emissions.
[19] Sznopek and Goonan, *The Materials Flow of Mercury in the Economies of the United States and the World*.
[20] Jasinski, *The Materials Flow of Mercury in the United States*.
[21] J. D. Husar and R. B. Husar, "Trend of Anthropogenic Mercury Flow in Florida, 1930-2000," Mercury in the Environment: Assessing and Managing Multimedia Risks, Orlando, Florida, American Chemical Society, April 7-11, 2002.

The authors also concluded that their results should be considered a likely upper bound on the North American anthropogenic contribution, because current mercury transport models appear to be missing some chemical transformations of mercury, causing the models to overestimate the local and regional impacts of some anthropogenic emission sources.[23]

According to the emission inventory developed for the study, electric utilities accounted for 27% of mercury emissions in 1998. Recall that EPA estimates utilities accounted for 41% of mercury emissions in 1999. The discrepancy appears to be due to the Seigneur et al. study including more emissions from incineration and mobile sources when compared with EPA. Seigneur et al. estimated U.S. mercury emissions to be 170 tons in 1998 (the same as EPA's 1995 inventory), while EPA estimated 117 tons in 1999.

Taking 32% as the North American anthropogenic contribution to U.S. mercury deposition and the Seigneur et al. inventory breakdown, coal-fired utilities contribute 9% of U.S. mercury deposition (0.27 * 0.32 = 0.086, or 8.6%).

We can get a back-of-the-envelope estimate of the utility contribution based on EPA's mercury inventory by assuming that the North American contribution to deposition scales with total North American emissions. Seigneur et al. estimate that North American sources emitted 220 tons of mercury in 1998. This drops to 169 tons given EPA's 1999 inventory, or 23% less. Assuming a linear relation with deposition, this would result in a corresponding decline in the North American contribution to U.S. deposition and therefore an 8% decline in total U.S. deposition. North American emissions would then account for 27% of all U.S. mercury deposition. If coal-fired utility boilers contribute 41% of U.S. anthropogenic mercury emissions, then coal-fired utilities would contribute 11% of all U.S. mercury deposition. Recall that this too is likely an likely upper limit, because it relied on the upper-limit estimate of the North American contribution from Seigneur et al.

It is possible that power plant mercury emissions are more likely than other North American emissions to deposit near the emission source. On the other hand, Seigneur et al. suggest that local and regional deposition is overestimated by current mercury transport models,

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[22] Seigneur et al., "Global Source Attribution for Mercury Deposition in the United States."
[23] C. Seigneur et al., "On the Effect of Spatial Resolution on Atmospheric Mercury Modeling," *The Science of the Total Environment*, vol. 304, no. 1-3 (2003), pp. 73-81.

suggesting that the local and regional effects of current mercury emissions may be overestimated.[24]

Sources of Mercury in Fish

Consumption of methylmercury (MeHg) in fish is the main route by which Americans are exposed to mercury.[25] MeHg in fish ultimately comes from current and past anthropogenic and natural mercury emissions. Mercury from these sources can deposit in water bodies, where microbes convert some of it to MeHg, which can accumulate in fish. Fish that are progressively higher on the food chain accumulate progressively higher levels of MeHg. Thus, top-level predatory fish have the highest MeHg body burdens. For example, shark and swordfish have the highest mercury levels among commonly consumed ocean fish, while walleye and bass are among the highest in mercury for freshwater fish.[26]

The degree to which current mercury emissions are the source of current mercury in fish is uncertain. In its Mercury Study Report to Congress, EPA concluded, "it is not possible to quantify the contribution of U.S. anthropogenic emissions relative to other sources of mercury, including natural sources and re-emissions from the global pool, on methylmercury levels in seafood and freshwater fish consumed by the U.S. population. Consequently, the U.S. EPA is unable to predict at this time how much, and over what time period, methylmercury concentrations in fish would decline as a result of actions to control U.S. anthropogenic emissions."[27]

Studies that have assessed the link between mercury deposition and mercury levels in fish have had mixed results. Those that looked at small, relatively well-characterized regions have found the strongest relationships. For example, a study of mercury deposition and levels in fish in northern Wisconsin found that declines in fish mercury levels tracked declines in mercury deposition. Each 10% decline in deposition was associated with a 5% decline in fish mercury

[24] Ibid.
[25] K. R. Mahaffey et al., "Blood Organic Mercury and Dietary Mercury Intake: National Health and Nutrition Examination Survey, 1999 and 2000," *Environmental Health Perspectives*, vol. 112, no. 5 (2004), pp. 562-570.
[26] Ibid.
[27] Environmental Protection Agency, *Mercury Study Report to Congress, Volume I: Executive Summary* (Washington, DC: December 1997), http://www.epa.gov/ttn/oarpg/t3/reports/volume1.pdf.

levels.[28] Thus, declines in deposition were associated with declines in fish mercury levels, but the declines were not as rapid as might be expected based on declines in deposition. A possible reason is that mercury already in sediments may have been released into the water, offsetting some of the deposition declines.[29]

Florida researchers recently searched for a link between mercury deposition and levels in Everglades' fish. In this case the relationship was also positive, but a bit weaker than found in Wisconsin. Changes in mercury deposition between 1990 and 2000 accounted for one-third of the mercury decline observed in fish. The study concluded that other factors besides recent atmospheric mercury deposition might explain much of the variability in fish mercury levels and that key uncertainties in mercury emissions, transport, and sediment chemistry must be resolved before a clear cause-and-effect relationship can be established between given mercury sources and levels found in fish.[30]

A recent national epidemiologic study of mercury deposition and fish mercury levels reported a statistically significant *inverse* relationship between mercury deposition rates and concentrations in fish.[31] While it is not plausible that fish mercury levels are inversely related to deposition, the fact that other factors in the model, such as percent of land under cultivation and water chemistry variables, explained much of the variation in mercury levels in fish suggests uncertainty in the degree to which current mercury deposition is causing current elevated mercury levels in fish. The researchers used a multivariate regression model that accounted for other factors that could affect fish mercury levels, such as proximity to point sources of mercury water emissions (for example, sewage treatment works or paper mills), percent of land under cultivation (which could affect mercury input to water through erosion and runoff), and water chemistry variables, such as pH, and levels of sulfate and dissolved organic carbon.

The authors cited data limitations that could account for the lack of the expected positive relationship between mercury deposition and fish levels. For example, the fish mercury data

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[28] T. R. Hrabik and C. J. Watras, "Recent Declines in Mercury Concentration in a Freshwater Fishery: Isolating the Effects of De-Acidification and Decreased Atmospheric Mercury Deposition in Little Rock Lake," *Science of the Total Environment.*, vol. 297, nos. 1-3 (2002), pp. 229-237.

[29] C. J. Watras et al., "Decreasing Mercury in Northern Wisconsin: Temporal Patterns in Bulk Precipitation and a Precipitation-Dominated Lake," *Environmental Science & Technology*, vol. 34 (2000), pp. 4051-4057.

[30] T. Atkeson and C. D. Pollman, "Trends of Mercury in Florida's Environment: 1989-2001," Mercury in the Environment: Assessing and Managing Multimedia Risks, Orlando, Florida, American Chemical Society, April 7-11, 2002.

[31] N. Knuffman and R. Lutter, *Does Mercury in Fish Come from the Air?* (Washington, DC: AEI-Brookings Joint Center for Regulatory Studies, September 2000).

were collected several years before the deposition data. On the other hand, the regression model and sensitivity analyses overall accounted for a great deal of the variation in fish mercury levels, with r^2 values ranging from 0.65 to 0.69. Furthermore, variables besides deposition had the expected associations with fish mercury levels.

Based on these results, it seems likely that given percentage declines in mercury deposition will result in lower percentage declines in mercury levels in fish. For example, if fish mercury levels decline at half the rate of deposition declines, we would expect a complete elimination of power plant mercury to result in an average decline of about 5% in fish mercury levels.

It is also worth noting that these declines apply only to freshwater fish. Reductions in U.S. mercury emissions will likely have no effect on mercury levels in ocean fish, because the U.S. contributes only about 1/50th of estimated annual mercury air emissions and because ocean fish mercury levels may be relatively insensitive to recent anthropogenic mercury emissions in any case. A recent study of trends in mercury levels in tuna collected in 1971 and 1998 concluded that mercury levels had not changed over the time period, despite evidence of increasing worldwide mercury emissions, mainly due to increases from Asia.[32]

Another factor to consider is worldwide mercury emission trends. While Europe and America have eliminated most mercury emissions during the last decade, mercury emissions from Asian countries are increasing. Recent estimates indicate that during the last decade mercury emissions increased 55% in China and 27% in India.[33] Emissions from Asia and natural emissions together dominate mercury deposition in most of the U.S. and the Asian contribution will only increase with time.

Upper-Bound Effect of Utility Mercury Reductions on Fish Mercury Levels

Overall, it appears that even large utility mercury emissions reductions could have at best a relatively small effect on mercury deposition rates and an even smaller effect on fish mercury levels. Even choosing EPA's most stringent option for the proposed Mercury Rule, that is, a 70% mercury reduction, U.S. freshwater fish mercury levels could be expected to decline on average

[32] A. M. L. Kraepiel et al., "Sources and Variations of Mercury in Tuna," *Environmental Science & Technology*, vol. 37, no. 24 (2003), pp. 5551-5558.

[33] C. Seigneur, *Global Emissions Inventory Activity Review: Mercury* (GEIA Center, May 25, 2003), www.geiacenter.org/reviews/mercury.html.

by at most 7%, while ocean fish levels would be unaffected. Given a 29% utility mercury reduction, fish mercury levels would decline by at most 3%. If fish mercury declines more slowly relative to emission reductions, as suggested by field research, then the fish mercury reductions would be substantially lower than these amounts.

EPA's claim that the benefits of the Mercury Rule can not be assessed with confidence is simply incorrect. It would be relatively easy to derive a reasonable upper-bound for the potential effect of the rule on fish mercury levels and EPA should do such an analysis before making a final decision on the rule.

While utility mercury reductions will likely do little to reduce fish mercury levels, reducing sulfur dioxide has the potential to be very effective. Water chemistry affects the rate at which microbes convert inorganic mercury to methylmercury (MeHg), the form of mercury that gets into fish and therefore of concern for human exposure. Higher levels of sulfate increase the rate of MeHg formation, indicating a link between SO_2 emissions and MeHg in fish.[34] This link has great import for mercury reduction policy, and will be discussed in more detail below.

4. Health Effects of Mercury

There is no question that high levels of mercury are neurotoxic. Tragic mercury poisonings in Japan in the 1950s and 1960s and in Iraq in the early 1970s showed the danger of high mercury exposures.[35] Highly exposed children suffered mental retardation, cerebral palsy, and seizures. But these episodes involved mercury exposures tens to hundreds of times greater than even relatively highly exposed Americans experience. Furthermore, where Americans are exposed to very small amounts of mercury over a long period of time, the poisoning incidents involved very large exposures over a short period of time. The key question for policy is whether the much lower mercury exposures experienced by Americans could be causing harm. The vast majority of mercury exposure in the United States comes from eating fish contaminated with methylmercury.[36] The main concern is whether MeHg in fish consumed by pregnant women

[34] Hrabik and Watras, "Recent Declines in Mercury Concentration in a Freshwater Fishery: Isolating the Effects of De-Acidification and Decreased Atmospheric Mercury Deposition in Little Rock Lake."
[35] G. J. Myers and P. W. Davidson, "Does Methylmercury Have A Role in Causing Developmental Disabilities in Children?" *Environmental Health Perspectives*, vol. 108, suppl. 3 (2000), pp. 413-420.
[36] K. R. Mahaffey et al., "Blood Organic Mercury and Dietary Mercury Intake: National Health and Nutrition Examination Survey, 1999 and 2000."

could be causing later cognitive and neurological harm to children exposed to MeHg in the womb.

Health Effects of Current Human Mercury Exposure Levels

Three major epidemiologic studies in the Faroe Islands, the Seychelles, and New Zealand have assessed the relationship between chronic, low-level mercury exposure in the womb and later performance on cognitive and neurological tests. The Faroe and New Zealand studies reported associations between higher MeHg levels and reductions in test scores, while the Seychelles study did not.[37] EPA set its reference dose (RfD) based on the results of the Faroe Islands study. The RfD is a daily intake of a given chemical that EPA estimates is "likely to be without an appreciable risk of deleterious effects during a lifetime."

EPA's RfD for Mercury is a blood level of 5.8 micrograms of mercury per liter of blood (ug/L), which is equivalent to 5.8 parts per billion (ppb).[38] The corresponding level in hair is 1.1 parts per million (ppm). EPA has estimated that a mercury intake of 0.1 micrograms per kilogram of body weight per day would lead to a body mercury level equal to the RfD, and has therefore set the RfD for daily mercury intake at this level. The greatest concern for low-dose mercury exposure is its effect on the developing fetus. The RfD is also intended to protect a developing fetus from harm.

Based on a random sample of more than 1,700 women aged 16 to 49, the Centers for Disease Control estimates that about 8% of women of childbearing age have body mercury levels exceeding the RfD.[39] Given these exposure levels, EPA initially estimated that about 320,000 children born each year are at risk of neurological and cognitive damage from mercury. Based on evidence from umbilical cord blood that mercury might be more concentrated in the fetus than in the mother, EPA recently raised this estimate to 630,000.[40] As a result of these estimates, EPA, environmental activists, and numerous media outlets have stated or implied that hundreds of

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[37] In the case of the New Zealand study, the association of mercury with lower neurological test performance occurred only if one child with high mercury exposure was excluded from the analysis. K. S. Crump et al., "Influence of Prenatal Mercury Exposure Upon Scholastic and Psychological Test Performance: Benchmark Analysis of a New Zealand Cohort," *Risk Analysis*, vol. 18, no. 6 (1998), pp. 701-713.

[38] D. C. Rice et al., "Methods and Rationale for Derivation of a Reference Dose for Methylmercury by the U.S. EPA," *Risk Analysis*, vol. 23, no. 1 (2003), pp. 107-115.

[39] Centers for Disease Control, *Second National Report on Human Exposure to Environmental Chemicals* (Atlanta: January 2003).

[40] J. Lowy, **"**EPA Raises Estimate of Newborns Exposed to Mercury," *Scripps-Howard News Service*, April 4, 2004.

thousands of children are suffering brain damage and mental retardation due to prenatal mercury exposure.[41]

Assuming that CDC's results are representative of the U.S. population, and there is no reason to believe otherwise, we can say with confidence that 8% of women do indeed have mercury levels exceeding the RfD. That said, EPA's large numbers and the scary claims that go with them represent a gross exaggeration of the actual harm from current mercury exposures. The exaggeration results from three errors in the portrayal of mercury exposure and risks.

- Treating the RfD as a safety level, above which harm occurs, when the evidence suggests the RfD is likely to be well below a level that could cause harm.
- Whatever the true safety level is, assuming that all people with mercury above the safety level experience harm. But by its very nature, the safety level is intended to protect the most sensitive people. Thus, the vast majority of people who exceed the nominal safety level in fact have not exceeded a substantive safety level.
- Glossing over the difference between subtle and severe health effects. The RfD is set to protect against the most subtle and mild health effects. Thus, even if the RfD were a true

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[41] See, for example:
J. Nesmith, "Senators attack mercury proposal; EPA accused of pro-industry bias," *Atlanta Journal Constitution*, April 13, 2004 ("When mercury is consumed by a pregnant woman, most often when she eats fish, it can cause her baby to be born with brain damage. Although the effect can be severe in individual cases, a report by the National Academy of Sciences warned in 2000 that mercury poisoning of unborn babies in America probably results in an overall increase in the number of children 'who have to struggle to keep up in school.' The EPA has estimated that each year 630,000 newborns in the United States, or nearly one in six, have dangerous levels of mercury in their blood.")
J. Morris, "EPA to investigate its proposed mercury rule," *Dallas Morning News*, May 13, 2004 ("Ingested in sufficient quantities, mercury _ a byproduct of coal combustion _ can harm the nervous system and cause learning disabilities, mental retardation and other problems. It's a particular threat to fetuses exposed through their mothers; the EPA estimates that 630,000 of the 4 million babies born each year could be at risk for some type of mercury-related developmental disorder.")
S. Hartsoe, "Experts: EPA emissions plan harmful to health, NC tourism," *Associated Press*, February 26, 2004 ("Mercury exposure can cause permanent brain and kidney damage, said Dr. John Pittman, and unborn and young children are particularly at risk. The EPA estimates that as many as 630,000 children may be born each year with unhealthy levels of mercury in their blood. 'The amount of mercury (in patients) is through the roof,' Pittman said.")
Natural Resources Defense Council, "Mercury Contaminated Fish: A Guide to Staying Healthy and Fighting Back," http://www.nrdc.org/health/effects/mercury/index.asp, and http://www.nrdc.org/health/effects/mercury/effects.asp, accessed August 30, 2004, ("Eating fish contaminated with mercury, a poison that interferes with the brain and nervous system, can cause serious health problems, especially for children and pregnant women." "Prenatal and infant mercury exposure can cause mental retardation, cerebral palsy, deafness and blindness.")
Friends of the Earth press release, "Hard-Hitting Ad Tells President Bush to Protect America's Children from Mercury Pollution," March 16, 2004 ("…a hard-hitting national ad in today's national edition of *USA Today* (circulation: 2.2 million) shows an image of toddlers with the headline 'They're being poisoned.'"), http://www.foe.org/new/releases/304mercpr.html.

safety limit, exceeding it would not necessarily put one anywhere near a mercury dose necessary to cause mental retardation or other serious or noticeable problems.

I discuss these issues in more detail below.

A key feature of the RfD is that it is set so as to protect the most sensitive individuals from even the most subtle health effects. In the case of mercury, this meant setting the RfD based on the Faroe Islands test results on the Boston Naming Test (BNT), a relatively specific neurological test in which children name objects based on line drawings.[42] Associations of mercury with reductions in performance on other specific tests (e.g., finger tapping, recall of names) or on more general tests of intelligence or cognitive performance required either higher mercury doses or did not occur at any dose.

The starting point for calculation of the RfD, is an estimation of the Benchmark Dose (BMD). In this case, the BMD is the dose expected to result in a doubling of the number of children performing below the 5^{th} percentile on the BNT. The BMD was calculated to be 85 ppb in blood. As a safety factor, EPA then takes the bottom of the 95% confidence interval for the BMD. This value is 58 ppb and is referred to as the benchmark dose lower limit (BMDL).

The BMDL is then divided by two safety factors of 3.16 ($10^{0.5}$), or a total of a factor of 10, to arrive at the RfD. The first factor is for variation and uncertainty in the way different individuals absorb, distribute, and excrete mercury (referred to as toxicokinetics). The second factor is for variation and uncertainty in the way different individuals respond to mercury at the organ or cellular level (referred to as toxicodynamics). The idea is to ensure that the RfD protects even the most sensitive individuals.

Population Exposure Relative to the Reference Dose and the Benchmark Dose

Figure 3 displays the cumulative percent of women of childbearing age with blood mercury below a given level. The graph on the lower right gives the full vertical scale, while the small graph in the upper left expands the scale for the highest exposure levels. The smaller graph was created by fitting a lognormal function to the mercury exposure distribution reported by

[42] Rice et al., "Methods and Rationale for Derivation of a Reference Dose for Methylmercury by the U.S. EPA," P. Grandjean et al., "Methylmercury Exposure Biomarkers as Indicators of Neurotoxicity in Children Aged 7 Years," *American Journal of Epidemiology*, vol. 150, no. 3 (1999), pp. 301-305, P. Grandjean et al., "Cognitive Deficit in 7-Year-Old Children with Prenatal Exposure to Methylmercury," *Neurotoxicology and Teratology*, vol. 19, no. 6 (1997), pp. 417-428.

CDC for women aged 16 to 49.[43] The solid line is the fit of the central exposure estimate, while the dashed line is the fit for the top of the 95% confidence interval, which represents a conservative estimate of the fraction of women with mercury above a given level. The graphs also mark the RfD, BMDL, and BMD.

Note that few women have mercury levels anywhere near the lowest level associated with health effects in the Faroe Islands study. For example, only one in 270 women have mercury levels greater than half the BMDL and only one in 1,700 are above the BMDL.[44]

Figure 4 shows the percent of women with mercury greater than any given level. The inset graph expands the vertical scale to show the small percentage of women at higher mercury exposure levels. Note that although 8% of women are above the RfD, almost all of them are much closer to the RfD than to the BMDL. For example, of the 8% of women with mercury greater than the RfD, two out of three are below 1/5th the BMDL, and more than 95% are below half the BMDL.

Health Effects Based on the Faroe Islands, New Zealand, and Seychelles Studies

The size of the associations between mercury and health outcomes in the Faroe Islands and New Zealand studies is small. Lutter and Mader (2001) estimated the change in scores on various tests used in the Faroe Islands and New Zealand studies based on the regression coefficients reported in the studies.[45] To be conservative, they assumed that reduced test scores occur on all tests[46] in all people at any mercury level above the RfD.

For example, reducing mercury levels from twice the RfD down to the RfD—a 50% reduction—would improve test scores by about 1/12th to 1/30th of a standard deviation.[47]

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[43] Centers for Disease Control, *Second National Report on Human Exposure to Environmental Chemicals*.

[44] None of the American women tested by CDC had mercury levels equal to or greater than the BMDL. However, such high mercury levels are extremely rare—estimated here to be one in 1,700—and the CDC tested 1,709 women. Thus, one wouldn't necessarily expect the CDC sample to include any of the handful of highly exposed women that might exist in the U.S. population. Centers for Disease Control, "Blood and Hair Mercury Levels in Young Children and Women of Childbearing Age—United States, 1999," *Morbidity and Mortality Weekly Report*, vol. 50, no. 8 (2001), pp. 140-143.

[45] The regression coefficient represents the size of the association between mercury exposure and test score. It takes the form of a number equal to the expected change in a test score for a given change in mercury exposure. R. Lutter and E. Mader, *Health Effects of Mercury-Contaminated Fish: A Reassessment* (Washington, DC: AEI-Brookings Joint Center for Regulatory Studies, March 2001).

[46] That is, all tests for which a statistically significant association was reported between mercury exposure and test score.

[47] Calculated by linear interpolation of Lutter and Mader's estimates for the "low" and "medium" exposure categories in Table 2 of their paper.

Reducing mercury levels from half the BMDL down to the RfD—an 80% reduction—would improve test scores by about 1/5th to 1/8th of a standard deviation.[48] Even the largest of these effect sizes is relatively small, given that plus or minus two standard deviations from the population average is considered "normal." In addition, effect sizes were smallest for the most general tests of cognitive and neurological performance (e.g., the McCarthy Perceptual Performance Scales (MPPS)) and largest for the most specific tests (e.g., a test of reaction time or the BNT). In practical terms, these mercury exposure reductions would amount to children achieving developmental gains equivalent to about one to three months in age.[49] Fewer than one in 200 children would be at the upper end of this benefit range.

Thus, even assuming that everyone experiences harm even at mercury levels as low as one-half or even 1/10th the BMDL, the implications for neurological and cognitive health are relatively minor. But this hypothetical analysis is much more pessimistic than the real-world situation, even if we continue to take the Faroe Islands and New Zealand results at face value.

First, the RfD is based on the Boston Naming Test. RfDs for other neurological tests were higher, meaning that higher mercury exposures would be necessary to cause harm.[50] For many tests, mercury exposure was not at all associated with lower scores. For example, in the New Zealand study, there was no association of mercury exposure with lower scores on any test unless the child with the highest mercury exposure was removed from the analysis. Even then, there was no relationship between higher mercury exposure and lower test scores on 20 of the 26 tests administered, including the IQ test.[51] In the Faroe Islands study, mercury was associated with lower scores on eight of 20 tests. These included the immediate recall and delayed recall portions of the California Verbal Learning Test, a test of short-term memory, but not on the

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[48] Calculated by linear interpolation of Lutter and Mader's estimates for the "medium" and "high" exposure categories in Table 2 of their paper.

[49] Estimated based on results in P. Grandjean et al., "Cognitive Deficit in 7-Year-Old Children with Prenatal Exposure to Methylmercury."

[50] A fine point here is that the BMDL for the McCarthy Perceptual Performance Scales, a test used in the New Zealand study, was similar to that for the Faroe Islands' BNT when the entire sample of children was included in the New Zealand analysis. When the child with the highest mercury exposure was removed from the analysis, the BMDL on the McCarthy test dropped to less than half the level calculated using the entire sample of children. Although this child had a mercury exposure four times greater than the next most exposed child, he was not an outlier by the conventional criterion of having a value at least three standard deviations greater than the mean. Crump et al., "Influence of Prenatal Mercury Exposure Upon Scholastic and Psychological Test Performance: Benchmark Analysis of a New Zealand Cohort."

[51] Another fine point is that even with removal of the child with the highest exposure level, the fact that six tests resulted in a statistically significant mercury association should be treated with caution. The significance levels were

learning and recognition portions of the test.[52] Mercury exposure was associated with a decreased score on the IQ subtest assessing attention, but not on the other two subtests, which assessed verbal and visuo-spatial reasoning and cognitive flexibility.

Second, the Lutter and Mader analysis made the intentionally conservative, but improbable assumption that everyone in the U.S. is several times more sensitive to mercury than the people in the Faroe Islands or New Zealand—that is, they assumed harm from mercury even at mercury exposures much lower than the levels associated with harm in the Faroe Islands or New Zealand studies. Indeed, the rationale for the RfD is based on just the opposite logic: a small percentage of people might be "outliers," which in this case means they absorb MeHg more readily, excrete it more slowly, detoxify it less effectively, and react to it more strongly than the vast majority of other human beings, including the children in the Faroe Islands and New Zealand studies. Such people are expected to be uncommon, because the odds are small that all of these "negative" traits would occur simultaneously in the same person.

Putting this all together, we can draw the following conclusions:

- The vast majority of women who exceed the RfD are still well below the BMDL. For example, of women who exceed the RfD, 95% are below 1/2 the BMDL and 65% are below 1/5th the BMDL.
- Most people with mercury exposures above the RfD, but below the BMDL would not be expected to suffer ill effects.
- Even if health effects occur below the BMDL, the effects are small and have at worst subtle and unnoticeable implications for intelligence or neurological health.

Thus, even taking the Faroe Islands and New Zealand studies at face value, we can see that the EPA's claim that 320,000 or 630,000 children are at risk of neurological harm is at best a great exaggeration. Activist and media claims and implications that hundreds of thousands of children each year are rendered brain damaged or learning disabled due to mercury exposure is

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not adjusted for multiple comparisons and are therefore biased toward unrealistically high statistical significance (i.e., unrealistically low p values). Ibid.

[52] P. Grandjean et al., "Methylmercury Exposure Biomarkers as Indicators of Neurotoxicity in Children Aged 7 Years," *American Journal of Epidemiology*, vol. 150, no. 3 (1999), pp. 301-305. P. Grandjean et al., "Cognitive Deficit in 7-Year-Old Children with Prenatal Exposure to Methylmercury," *Neurotoxicology and Teratology*, vol. 19, no. 6 (1997), pp. 417-428.

in the realm of hysterical fantasy. Indeed, the very reason for the controversy over the health effects of low-level mercury exposure is that the hypothesized effects are so small and subtle as to be difficult to detect even with large samples of children and a battery of specialized neurological tests.

Even these conclusions still depended on the assumption that the Faroe Islands and New Zealand results represent a genuine cause-effect relationship between mercury exposure and neurological development that is applicable to people in the United States. But as noted earlier, the New Zealand study suffered from unstable results. Mercury appeared to have no health effects unless the child with the highest mercury exposure was removed from the dataset. The New Zealand study also included a much smaller sample of children than the Faroe Islands study. As a result, regulators have focused on the Faroe Islands study to support derivation of the RfD and regulatory limits on mercury emissions.

In contrast to the results of the Faroe Islands and New Zealand studies, a study of mothers and children in the Seychelles suggests that current mercury exposure levels in the U.S., as well as the much higher mercury exposures in the Seychelles do not have any effect on neurological or cognitive health or development.[53] The Seychelles study followed nearly 800 children through age nine and was comparable to the Faroe Islands study in its statistical power to detect any effects of mercury exposure.[54] Furthermore, several lines of evidence suggest that the Seychelles study is more relevant than the Faroe Islands study for understanding the potential effects of mercury on children in the United States.

- The Seychellois get their MeHg from a diet rich in the same types of ocean fish eaten by Americans, while the Faroese get their MeHg from eating whale blubber.[55] Thus, the Seychellois are exposed to mercury in a fashion similar to people in the U.S.
- Whale blubber is also high in PCBs, inorganic mercury, and other contaminants, while the fish eaten by the Seychellois and by Americans are not.[56]

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[53] G. J. Myers et al., "Prenatal Methylmercury Exposure from Ocean Fish Consumption in the Seychelles Child Development Study," *Lancet*, vol. 361, no. 9370 (2003), pp. 1686-1692.
[54] Ibid.
[55] Ibid., P. Grandjean et al., "Cognitive Performance of Children Prenatally Exposed to 'Safe' Levels of Methylmercury," *Environmental Research*, vol. 77, no. 2 (1998), pp. 165-172.
[56] G. J. Myers and P. W. Davidson, "Does Methylmercury Have a Role in Causing Developmental Disabilities in Children?"

- Although the mercury levels in the Seychellois and the Faroese are similar, the whale blubber eaten by the Faroese has about five times the mercury per unit mass as the fish eaten by the Seychellois.[57] Thus, to the extent mercury is actually causing neurological deficits in the Faroese, it could be due to higher acute exposures to mercury than occur in the Seychelles.
- The Seychelles study used mercury in maternal hair as the exposure measure, while the Faroe Islands study used mercury in umbilical cord blood.[58] The Faroe Islands study also measured mercury in maternal hair, but the association of mercury with neurological outcomes was generally smaller and less statistically insignificant based on this measure.[59] There is some controversy over whether one method is better than the other.[60] The Faroe Islands researchers argued that umbilical cord blood is a better marker of mercury exposure than hair, because they found a stronger association between mercury in cord blood and lower test scores. But this appears to be circular reasoning. What matters is which mercury measurement provides a better marker of exposure to the brain, which is where mercury actually has its effects. Maternal hair mercury levels have been calibrated to fetal brain levels, while cord-blood mercury has not.[61] Another factor to consider is that cord blood provides a measure of mercury exposure only at birth, while maternal hair can be used to assess mercury exposure to a fetus throughout pregnancy.[62]
- The Faroe Islands population is descended from Scandinavians, while the Seychellois are ethnically European and African.[63] Neither population is as ethnically diverse as the U.S., but the Seychellois appear to be more diverse and therefore may be more representative of the range of variation in response to MeHg.

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[57] The shark eaten by New Zealanders has about seven times the mercury concentration as the fish eaten by the Seychellois. Myers et al., "Prenatal Methylmercury Exposure from Ocean Fish Consumption in the Seychelles Child Development Study."

[58] N. Keiding et al., "Prenatal Methylmercury Exposure in the Seychelles," *Lancet*, vol. 362, no. 9384 (2003), pp. 664-665; author reply 665.

[59] P. Grandjean et al., "Cognitive Deficit in 7-Year-Old Children with Prenatal Exposure to Methylmercury," Myers and Davidson, "Does Methylmercury Have a Role in Causing Developmental Disabilities in Children?"

[60] Keiding et al., "Prenatal Methylmercury Exposure in the Seychelles."

[61] E. Cernichiari et al., "Monitoring Methylmercury During Pregnancy: Maternal Hair Predicts Fetal Brain Exposure," *Neurotoxicology*, vol. 16, no. 4 (1995), pp. 705-710, Keiding et al., "Prenatal Methylmercury Exposure in the Seychelles."

[62] Keiding et al., "Prenatal Methylmercury Exposure in the Seychelles."

[63] Rice et al., "Methods and Rationale for Derivation of a Reference Dose for Methylmercury by the U.S. EPA."

The opposite results of the Faroe Islands and Seychelles studies still needs to be explained. However, given the results available, the Seychelles study appears to be more relevant to the U.S. population and the way in which it is exposed to methylmercury. If so, then no one is being harmed by current mercury emissions and further reductions in mercury would not provide any health benefits, even if such reductions do reduce mercury levels in fish.

Potential Health Benefits of EPA's Mercury Rule

EPA rightly notes that the benefits of mercury reductions can not be precisely estimated because of uncertainties in the degree to which utility mercury reductions will reduce deposition and mercury levels in fish, and uncertainties in the health benefits of reducing mercury levels in fish.

Despite these uncertainties, EPA should have done a "best-case" analysis by assuming (1) that utility mercury reductions translate directly into one-to-one mercury reductions in freshwater fish, (2) that the freshwater fish mercury reductions translate directly into one-to-one reductions in Americans' mercury exposure , and (3) that the mercury exposure reductions translate directly into health benefits for all people with blood mercury levels greater than the RfD, with the benefits determined by the effect sizes estimated in the Faroe Islands and New Zealand studies.

As shown above, these maximum benefits are small even if we make the improbable assumption that everyone experiences health effects at any exposure above the RfD and that we reduce everyone's exposure down to the RfD. But to get even these small and unlikely-to-materialize benefits, we would need to eliminate virtually all worldwide mercury emissions, including natural emissions and re-emission of mercury already in the environment. Eliminating all U.S. mercury emissions would reduce mercury deposition by at most one-third, and eliminating all utility mercury emissions would reduce mercury deposition by at most 11%. To estimate the Mercury Rule's maximum benefit, I make the following assumptions:

- Given that power plants account for 11% of U.S. mercury deposition (see above), a 70% reduction in power plant mercury emissions, EPA's most stringent alternative, would reduce U.S. mercury deposition by about 7%.

- Assuming that freshwater fish mercury levels are proportional to deposition, freshwater fish mercury levels would be reduced by an average of 7%. Ocean fish mercury levels would likely remain unchanged.[64]
- If all mercury exposure is due to eating freshwater fish, the utility mercury reductions would, on average, reduce women's mercury exposure by 7%. Women with blood mercury above the RfD have an average mercury level of about 12 ppb, or about twice the RfD, so the reduction in average blood mercury levels would be 0.84 ppb under these conservative assumptions. For women at five times the RfD, or 29 ppb (about 0.37% of all women are at or above this level), the exposure reduction would be 2.2 ppb.

Taking Lutter and Mader's estimates of the benefits of mercury reductions based on the Faroe Islands and New Zealand studies, these mercury exposure reductions would improve average cognitive and neurological test scores of the children of women who are above the RfD by about $1/90^{th}$ to $1/200^{th}$ of a standard deviation for the average exposure, and $1/55^{th}$ to $1/90^{th}$ of a standard deviation for those at high exposure levels (five times the RfD or half the BMDL). A complete elimination of utility mercury emissions would result in a $1/35^{th}$ to $1/65^{th}$ of a standard deviation improvement for those at high exposures. In terms of practical developmental improvements as measured by test scores, this would be equivalent to speeding a child's development by perhaps one to two weeks. In terms of test scores, this is the equivalent of moving from, say, the 10^{th} percentile to between the 10.3 to 10.6 percentiles.

To be even more conservative in estimating the potential benefits of mercury reductions, we can look at those areas of the U.S. that may be more affected by regional sources of mercury emissions than the average U.S. location. Seigneur et al. estimated that for three of 19 deposition monitoring sites, more than half of U.S. mercury deposition comes from North American sources. Assuming 75% of U.S. mercury deposition comes from North American emissions, and that coal-fired power plants represent 41% of those emissions, a 70% reduction in power plant emissions would reduce deposition by 21% (0.75 * 0.41 * 0.7 = 0.21). Once again assuming this translates directly into exposure reductions, test scores would improve by about $1/20^{th}$ to $1/65^{th}$ of a standard deviation, depending on the test and the initial mercury exposure level. Complete

[64] This is consistent with trends in ocean tuna mercury levels reported in Kraepiel et al., "Sources and Variations of Mercury in Tuna."

elimination of utility mercury emissions would improve scores by 1/12th to 1/45th of a standard deviation.

In practical terms, this is the equivalent of moving development ahead by a few to several weeks for the types of cognitive and neurological tasks measured in the various epidemiological studies. Or in terms of test scores, it is the equivalent of moving from the 10th percentile to between the 10.4 and 11.5 percentiles in relative performance. In either case, the vast majority of children would be toward the lower end of these benefit ranges, because so few people are exposed mercury at levels substantially above the RfD.

These estimates represent an extreme best case for the benefits of completely eliminating utility mercury emissions, and are far more optimistic than a genuine best case. First, because only a fraction of the population would experience any benefits from reductions in mercury exposure from levels already below the BMDL. Second, because any given reduction in mercury emissions would result in substantially less than a one-to-one reduction in mercury exposure. This is because few people receive 100% of their mercury exposure from eating non-commercial freshwater fish, the main source of mercury exposure that could be significantly affected by reductions in U.S. mercury emissions,[65] and because reductions in freshwater fish mercury levels would not track one-to-one with mercury deposition reductions. Of course, if mercury has no effects at current exposure levels, as suggested by the Seychelles study, then reducing mercury would have no effect at all on Americans' health.

Thus, despite the uncertainties, it is clearly possible to estimate the potential health benefits of a best-case scenario for the Mercury Rule. EPA should do so and then determine whether the benefits are worth having given the costs, and whether the best-case benefits are likely to materialize. This would provide far more transparent information on the worth of the Mercury Rule than is currently provided by the Mercury Rule's preamble or Regulatory Impact Analysis (RIA).

5. "Co-Benefits" of EPA's Mercury Rule

[65] Addressing mercury levels in freshwater, non-commercial fish is key for reducing mercury exposure. First, as noted earlier, ocean fish mercury levels are unlikely to be affected by changes in U.S. emissions. Second, non-commercial freshwater fish, that is, fish caught by anglers for consumption by themselves and their family and

EPA does not attempt to quantify the benefits of the mercury reductions in the Mercury Rule, and instead promotes the rule based on ostensible "co-benefits" due to SO_2 and NOx reductions.[66] Even these co-benefits are questionable, as will be discussed below. More importantly, if EPA believes that the benefits of mercury reductions are due mainly to other pollutants reduced as a consequence of mercury reductions, then the agency should directly regulate those other pollutants, rather than using "co-benefits" as a marketing tactic to justify an otherwise foolish regulation.

EPA claims in its RIA for the Mercury Rule that "the health benefits of addressing mercury, SO_2, and NOx in an integrated fashion are dramatic." In fact, the claim of "integrated" benefits merely masks EPA's own conclusion that almost all of the purported benefits of the reductions are due to reductions in PM, which in turn are due mainly to SO_2 reductions. The RIA attributes 85.2% of the reduction in PM levels to SO_2 reductions and 13.4% to NOx reductions.[67] Providing separate estimates of the net benefits of SO_2 and NOx reductions would provide more insight and transparency regarding the net benefits of reducing each individual pollutant.

The likely lack of benefits for mercury emissions reductions has already been discussed. NOx emission reductions will also have small health benefits, because NOx makes only a small contribution to PM in the eastern U.S., while even EPA has previously concluded that ozone reductions to attain the 8-hour standard will impost net social costs on Americans. Indeed, in its RIA for the 8-hour ozone standard, EPA estimated that the social costs of attaining the standard would be roughly twice as large as the benefits.[68] Independent analysts have shown that EPA substantially underestimated the likely costs of attaining the standard, making the situation much worse than EPA predicted.[69] In any case, even EPA attributes less than 0.1% of the benefits of the IAQR to ozone reductions.[70]

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friends, is believed to be the greatest source of mercury exposure since most commercial fish have relatively low mercury levels. Environmental Protection Agency, *Mercury Study Report to Congress*.

[66] 69 FR 4707

[67] Environmental Protection Agency, *Benefit Analysis for the Section 112 Utility Rule*.

[68] Environmental Protection Agency, *Regulatory Impact Analyses for the Particulate Matter and Ozone National Ambient Air Quality Standards and Proposed Regional Haze Rule* (Washington, DC: July 17, 1997), www.epa.gov/ttn/oarpg/naaqsfin/ria.html.

[69] See, for example, Susan E. Dudley, *Comments on the U.S. Environmental Protection Agency's Proposed National Ambient Air Quality Standard for Ozone* (Arlington, Virginia: Mercatus Center, March 1997), www.mercatus.org/research/RSP19972.htm, Randall Lutter, *Is EPA's Ozone Standard Feasible?* (Washington, DC: AEI-Brookings Joint Center for Regulatory Studies, December 1999), www.aei.brookings.org/publications/reganalyses/reg_analysis_99_06.pdf.

[70] 69 FR 4646

EPA's NOx SIP Call regulation was implemented in May 2004 and requires a 60% reduction in ozone-season NOx emissions from coal-fired power plants and industrial boilers.[71] EPA's regulations for on- and off-road diesel and gasoline vehicles will progressively reduce total NOx emissions from these sources by more than 80% during the next 20 years or so as the fleet turns over to 21st Century vehicles.[72] Thus, EPA has already taken actions that will eliminate the vast majority of remaining NOx emissions. The marginal benefits of additional NOx reductions are therefore likely to be zero.

The ostensible benefits of additional PM reductions are questionable. Toxicology studies suggest that sulfate and nitrate are not toxic even at levels substantially higher than those currently found in ambient air. For example, ammonium sulfate is used as an inert control in studies of the health effects of acid aerosols, while inhaled magnesium sulfate is used therapeutically to reduce airway constriction in asthmatics.[73] Studies with healthy and asthmatic volunteers have failed to find respiratory effects of inhaled nitrate PM, even at levels many times higher than ever experienced today.[74] Likewise, a number of researchers have raised concerns over the validity of the epidemiologic results.[75] EPA also ignored contrary evidence from studies that did not find a link between PM and mortality.[76]

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[71] Environmental Protection Agency, *Addendum to the Regulatory Impact Analysis for the NOx SIP Call, FIP, and Section 126 Petitions* (Washington, DC: September 1998).

[72] J. Schwartz, *Public Interest Comment on EPA's Proposed Nonroad Engine and Fuel Standards* (Arlington, Virginia: Mercatus Center, August 2003), http://www.mercatus.org/pdf/materials/381.pdf, Schwartz, *No Way Back: Why Air Pollution Will Continue to Decline*, Environmental Protection Agency, "Control of Air Pollution from New Motor Vehicles: Tier 2 Motor Vehicle Emissions Standards and Gasoline Sulfur Control Requirements; Final Rule," *Federal Register*, February 10, 2000, pp. 6698-6870, www.epa.gov/otaq/tr2home.htm#preamble, Environmental Protection Agency, *Regulatory Impact Analysis: Heavy-Duty Engine and Vehicle Standards and Highway Diesel Fuel Sulfur Control Requirements* (Washington, DC: December 2000), www.epa.gov/otaq/diesel.htm, Environmental Protection Agency, *Final Regulatory Analysis: Control of Emissions from Nonroad Diesel Engines* (Washington, DC: May 2004), http://www.epa.gov/nonroad-diesel/2004fr/420r04007a.pdf.

[73] J. Q. Koenig et al., "Respiratory Effects of Inhaled Sulfuric Acid on Senior Asthmatics and Nonasthmatics," *Archives of Environmental Health*, vol. 48, no. 3 (1993), pp. 171-175, L. J. Nannini, Jr. and D. Hofer, "Effect of Inhaled Magnesium Sulfate on Sodium Metabisulfite-Induced Bronchoconstriction in Asthma," *Chest*, vol. 111, no. 4 (1997), pp. 858-861, E. L. Avol et al., "Controlled Exposures of Human Volunteers to Sulfate Aerosols. Health Effects and Aerosol Characterization," *American Review of Respiratory Disease*, vol. 120, no. 2 (1979), pp. 319-327.

[74] M. T. Kleinman et al., "Effect of Ammonium Nitrate Aerosol on Human Respiratory Function and Symptoms," *Environmental Research*, vol. 21, no. 2 (1980), pp. 317-326.

[75] See, for example, F. W. Lipfert, "Commentary on the HEI Reanalysis of the Harvard Six Cities Study and the American Cancer Society Study of Particulate Air Pollution and Mortality," *Journal of Toxicology and Environmental Health, Part A*, vol. 66, no. 16-19 (2003), pp. 1705-1714, F. W. Lipfert, *Unresolved Questions in Air Pollution Epidemiology, Review Comments on the U.S. Environmental Protection Agency's Air Quality Criteria for Particulate Matter, Third External Review Draft* (Annapolis, MD: Annapolis Center for Science-Based Public Policy, July 2002), F. W. Lipfert and R. E. Wyzga, "Statistical Considerations in Determining the Health Significance of Constituents of Airborne Particulate Matter," *Journal of the Air and Waste Management*

Also worth noting is that almost all of the PM reductions for which EPA takes credit will occur in areas that already comply with federal $PM_{2.5}$ standards. As of the end of 2003, national $PM_{2.5}$ data show that only about 18% of monitoring locations violate the annual $PM_{2.5}$ standard and only 0.6% violate the 24-hour standard.[77] Furthermore, about 58% of violating locations would reach attainment with $PM_{2.5}$ reductions of less than 10% and another 24% of locations would reach attainment with $PM_{2.5}$ reductions of 10% to 20%. Existing motor vehicle and power plant requirements will achieve these reductions and more.[78] To the extent that some locations will still be out of attainment after upcoming "baseline" reductions in $PM_{2.5}$, almost all are in California, an area where the Mercury Rule and the IAQR will have no effect in any case. Thus, from the perspective of $PM_{2.5}$ attainment, the marginal benefits of the IAQR are likely to be close to zero.

EPA claims in the Mercury Rule preamble and RIA that there will also be direct health benefits from reduced ambient NO_2 and SO_2 levels, in addition to the effect of these reductions on PM and ozone levels. But neither the Mercury Rule nor the associated RIA mention that virtually the entire U.S. has been in attainment of federal SO_2 and NO_2 standards for years, that ambient levels of both pollutants have steadily declined and continue to decline, and that NO_2 and SO_2 levels at nearly all monitoring sites are below the respective standards by a substantial margin.[79] Since there are likely no additional direct health benefits to be had by further reductions in these pollutants, EPA should not imply that current low levels of NO_2 and SO_2 are causing harm.

Association, vol. 49, no. 9 (1999), pp. 182-191, S. H. Moolgavkar, *Review of Chapter 8 of the Criteria Document for Particulate Matter (Comments Submitted to EPA)*, 2002, S. H. Moolgavkar and E. G. Luebeck, "A Critical Review of the Evidence on Particulate Air Pollution and Mortality," *Epidemiology*, vol. 7, no. 4 (1996), pp. 420-428, G. Koop and L. Tole, "Measuring the Health Effects of Air Pollution: To What Extent Can We Really Say That People Are Dying from Bad Air?" *Journal of Environmental Economics and Management*, vol. 47 (2004), pp. 30-54. For a review with numerous additional references to the literature, see J. Schwartz, *Particulate Air Pollution: Weighing the Risks* (Washington, DC: Competitive Enterprise Institute, April 2003), http://www.cei.org/pdf/3452.pdf.

[76] F. W. Lipfert and S. C. Morris, "Temporal and Spatial Relations between Age Specific Mortality and Ambient Air Quality in the United States: Regression Results for Counties, 1960-97," *Occupational and Environmental Medicine*, vol. 59, no. 3 (2002), pp. 156-174, F. W. Lipfert et al., "The Washington University-EPRI Veterans' Cohort Mortality Study," *Inhalation Toxicology*, vol. 12 (suppl. 4) (2000), pp. 41-73.

[77] Calculated from national $PM_{2.5}$ monitoring data for 2001-2003 downloaded from EPA at http://www.epa.gov/air/data/montrnd.html?us~usa~United%20States.

[78] Schwartz, *No Way Back: Why Air Pollution Will Continue to Decline*, Schwartz, *Public Interest Comment on EPA's Proposed Nonroad Engine and Fuel Standards*.

[79] J. Schwartz and S. F. Hayward, *Air Quality in America: A Dose of Reality on Air Pollution Levels, Trends and Health Risks* (Washington, DC: American Enterprise Institute, April 28, 2004), http://www.aei.org/docLib/200404301_schwartzhayward.pdf.

6. SO_2 Reductions and Mercury in Fish

Ironically, if EPA wants to reduce mercury in fish, reducing SO_2 emissions is likely to be far more effective and far less expensive than reducing power plant mercury emissions. In order to get into fish, mercury must be converted to MeHg by microbes. Water chemistry affects the rate at which microbes convert inorganic mercury to MeHg. For example, higher levels of sulfate increase the rate of MeHg formation, indicating a link between SO_2 emissions and MeHg in fish.[80] A recent study showed that reducing sulfate levels in a lake caused large reductions in MeHg levels in fish.[81]

This research is particularly robust, because the effect was measured through a controlled experiment. The lake under study is naturally separated into two basins with little exchange between them. Small amounts of sulfate were added to the "treatment" basin in the 1980s, while nothing was done to the "reference" basin. During the 1990s, the treatment basin naturally deacidified at a faster rate than the reference basin. In the treatment basin, fish mercury levels were slightly higher to begin with, but declined 60 percent more than in the reference lake. About half of the decline was due to de-acidification and about half to declines in mercury deposition.

As detailed earlier, there is great uncertainty about where current mercury in fish is coming from and substantial evidence that current power plant emissions are a small factor. Thus, reducing power plant emissions is likely to do little to reduce mercury levels in fish and may even be ineffective. On the other hand, reducing SO_2 emissions is likely to cause substantial reductions in fish mercury levels, and will do so *regardless of where the mercury in fish is coming from*. Thus, SO_2 reduction is a less risky mercury-control policy, because it is less subject to uncertainties in where mercury comes from or how it is getting into fish.

Reducing SO_2 emissions will also have co-benefits in reducing people's PM exposure and reducing regional haze over eastern national parks and other scenic areas, and will likely also cause modest reductions in power plant mercury emissions. Furthermore, large reductions in

[80] T. R. Hrabik and C. J. Watras, "Recent Declines in Mercury Concentration in A Freshwater Fishery: Isolating the Effects of De-Acidification and Decreased Atmospheric Mercury Deposition in Little Rock Lake."
[81] Ibid.

sulfur dioxide emissions are likely to be less expensive than large reductions in mercury emissions.[82]

If sulfate PM is not toxic, SO_2 reductions alone will still not confer net benefits. However, reducing utility SO_2 and scrapping plans for additional mercury or NOx reductions promises the best combination of comparatively low costs and comparatively high benefits, as well as the greatest certainty that at least some of the hoped-for benefits will actually materialize. EPA should therefore consider SO_2 reductions alone as a replacement policy for the Mercury Rule and the Interstate Air Quality Rule.

7. Health-Health Tradeoffs in EPA's Proposed Regulation

As shown above, the harm from current U.S. mercury emissions appears to be somewhere between small and non-existent. This means that additional reductions will provide at best small benefits and possibly no benefits. On the other hand, the costs of additional pollution reductions can cause harm of their own.

The policy problem is that pollution reduction measures involve "health-health" tradeoffs for the public.[83] Reducing pollution may improve health. But regulations to reduce pollution make most goods and services more expensive, reducing families' disposable income. Because people on average use their income to make their lives safer and healthier—for example, by buying better and safer products, more nutritious food, better medical care, more leisure time, etc.—reducing peoples' disposable income reduces their health.

A number of researchers have attempted to estimate the health effects of regulatory costs. These estimates suggest that every $15 million in additional regulatory costs results in one additional induced fatality.[84] Expected health benefits of a regulation must be weighed against

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[82] See, for example, Energy Information Administration, *Analysis of Strategies for Reducing Multiple Emissions from Electric Power Plants: Sulfur Dioxide, Nitrogen Oxides, Carbon Dioxide, and Mercury and a Renewable Portfolio Standard* (Washington, DC: July 2001), www.eia.doe.gov/oiaf/servicerpt/epp/pdf/sroiaf(2001)03.pdf.

[83] Randall Lutter and John Morrall appear to be the first to use this term. See R. Lutter and J. F. Morrall, "Health-Health Analysis: A New Way to Evaluate Health and Safety Regulation," *Journal of Risk and Uncertainty*, vol. 8 (1994), pp. 43-66.

[84] R. Lutter et al., "The Cost-Per-Life-Saved Cutoff for Safety-Enhancing Regulations," *Economic Inquiry*, vol. 37, no. 4 (1999), pp. 599-608. Fifteen million dollars was their "best estimate," with a range of $10 million to $50 million. Health-health analysis is only a partial analysis of the net welfare effects of a regulation, because such analyses currently include only mortality. Benefit-cost analyses attempt to include all costs and benefits of a regulation—not only mortality, but morbidity (that is, disease and disability), and all the other social-welfare effects

these health costs in order to increase the likelihood that a given regulation will provide net health benefits to the public.

EPA did not include the negative health effects of regulatory costs when considering the worth of the Mercury Rule. Given the likely lack of benefits from additional U.S. mercury reductions and EPA's estimate of $1.6 billion per year for the cost of the MACT rule, the Mercury Rule is virtually certain to do net harm to public health.

By considering the net welfare effects of pollution-control regulations, policymakers can explicitly address the tradeoffs between the health benefits of lower pollution levels, and the health costs of reducing people's disposable income through imposition of regulatory costs. EPA should perform such an assessment for the Mercury Rule.

8. Conclusion

EPA's Mercury Rule and Interstate Air Quality Rule will provide few benefits and are likely to impose billions of dollars per year in net costs on Americans. Based on the results of the Seychelles study, which is the one most relevant for Americans, mercury at current levels in fish is unlikely to be causing harm to American children. Indeed, even if we assume the results of the Faroe Islands study apply in the U.S. and that people in the U.S. experience harm from mercury exposures even $1/15^{th}$ as large as the minimum effect level in the Faroe Islands, the effects of current mercury exposures are still small, subtle, and unnoticeable. Sulfate and nitrate PM are also unlikely to be causing harm at current levels.

To the extent EPA nevertheless wishes to go forward with additional utility pollution reductions, SO_2 reductions appear to be the best approach. Reducing utility SO_2 emissions has a much greater potential to reduce fish mercury levels and a much greater certainty that the hoped-for mercury reductions will actually occur, when compared with reductions in utility mercury emissions. Furthermore, SO_2 reductions will also reduce $PM_{2.5}$ haze and increase visibility, ensuring that SO_2 reductions will provide some tangible welfare benefits.

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of a regulation. In this sense, health-health analysis is a weaker test of the value of a regulation than benefit-cost analysis. However, because it is a weaker test, if a regulation can't be shown to have net health benefits in a health-health analysis, than it is likely that the regulation in question will cause net harm to the public. Health-health analysis also has the virtue of making the net health effects of a regulation explicit to the public, while benefit-cost analysis is often perceived, inaccurately, as divorced from concerns over human welfare.

Figure 1. EPA 1999 U.S. Mercury Emission Inventory, in Tons per Year

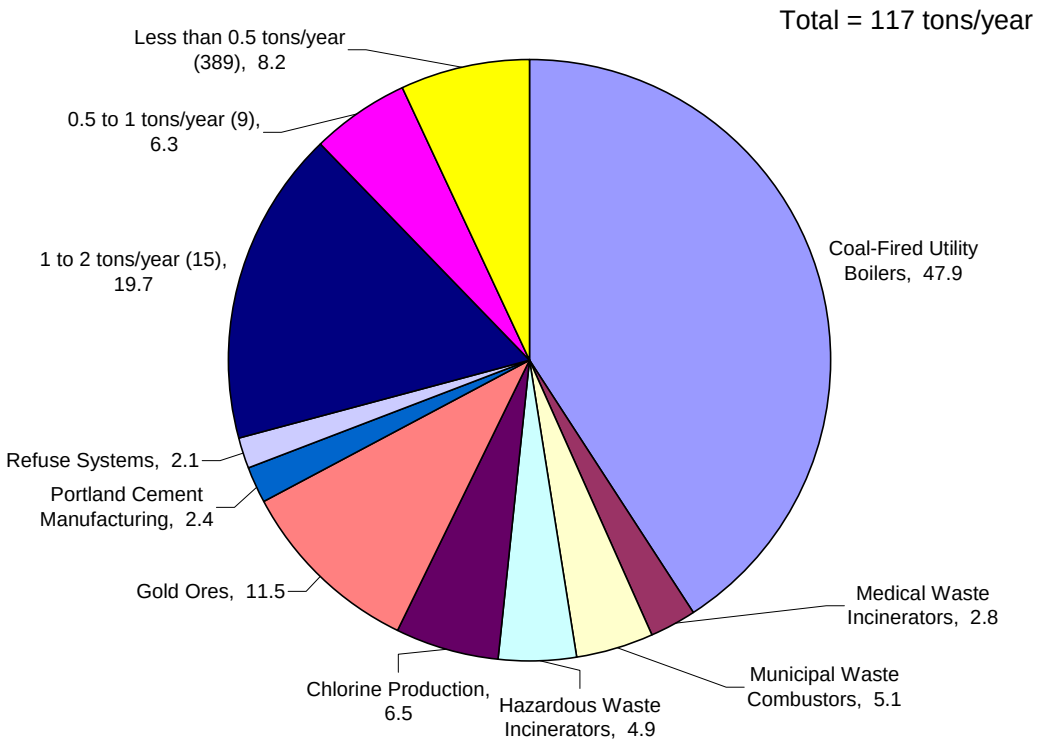


Figure 2. Trend in U.S. Mercury Emissions

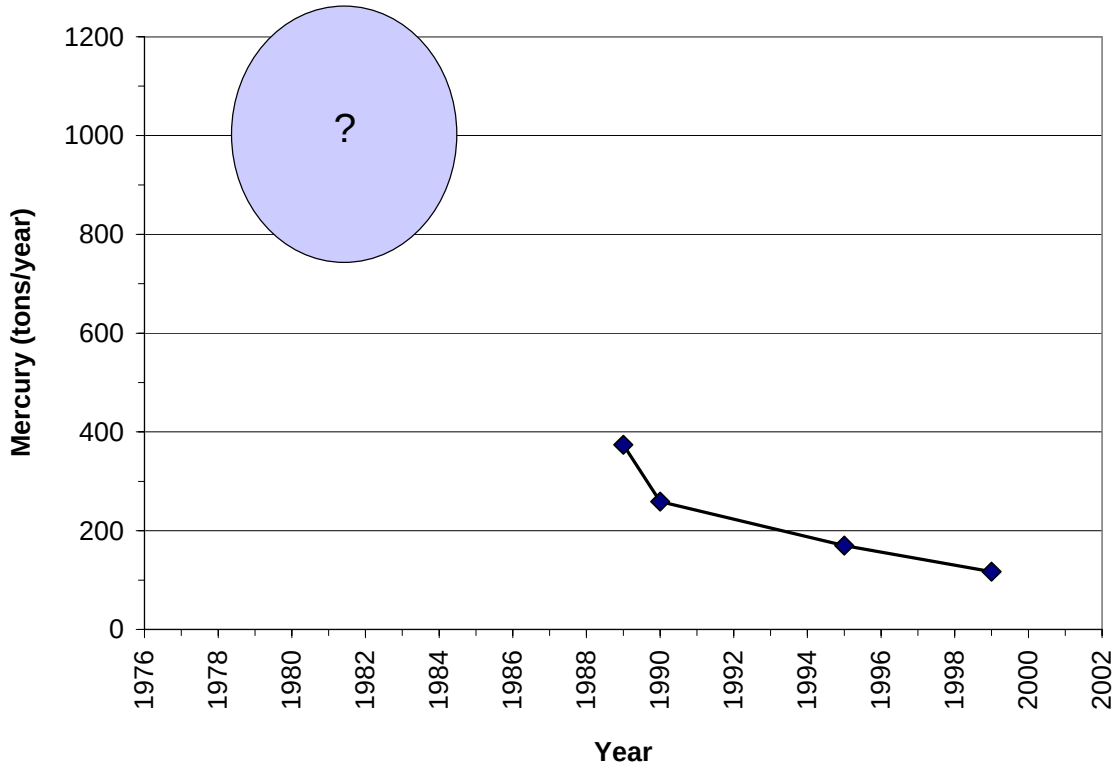
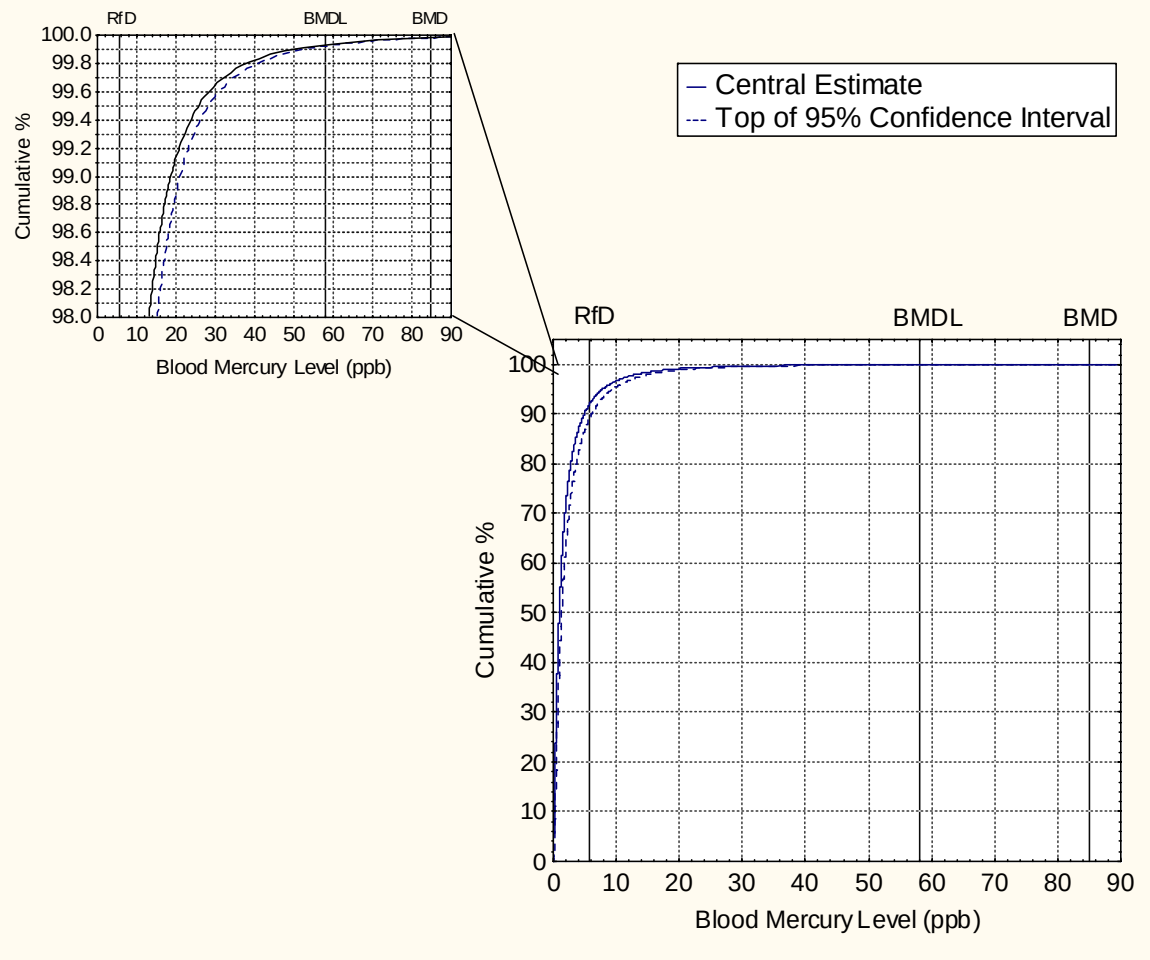


Figure 3. Comparison of Blood Mercury Levels in Women Aged 16-49 with Levels Associated with Subtle Health Effects in the Faroe Islands Study



RfD = Reference Dose, BMD = Benchmark Dose, BMDL = bottom of the 95% confidence interval of the BMD.

Figure 4. Percent of Women with Blood Mercury Greater than Given Levels