Technical Support Document

Revision of December 2000 Regulatory Finding on the Emissions of Hazardous Air Pollutants From Electric Utility Steam Generating Units and the Removal of Coal- and Oil-Fired Electric Utility Steam Generating Units from the Section 112(c) List: Reconsideration

October 21, 2005

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1. Introduction

In the Technical Support Document (Effectiveness TSD) for the Revision of December 2000 Regulatory Finding on the Emissions of Hazardous Air Pollutants From Electric Utility Steam Generating Units and the Removal of Coal- and Oil-Fired Electric Utility Steam Generating Units from the Section 112(c) List (Revision Rule)(US EPA 2005a) the Agency analyzed the mercury (Hg) exposure due to coal-fired electric utility steam generating units ("power plants" or "utilities") as defined in the Revision Rule remaining after implementation of the Clean Air Interstate Rule (CAIR) and after implementation of both CAIR and the Clean Air Mercury Rule (CAMR). This analysis included a partly quantitative, partly qualitative treatment of the exposure from recreational and subsistence caught freshwater fish. Other pathways including commercial freshwater fish, estuarine fish, and marine fish were treated qualitatively.

The quantitative component of the analysis focused on the recreational and subsistence freshwater fish pathway because, as described below, it is this pathway that leads to the greatest individual exposure due to utility-attributable mercury emissions. Based on a qualitative analysis, EPA concluded that the other pathways lead to smaller individual exposure levels and that the combined exposure from all the pathways would not be materially different than the exposure due solely to the recreational/subsistence fish pathway for the individuals most highly exposed to utility-attributable mercury. See 70 FR at 16,102.

This document expands on information presented in the Revision of December 2000 Regulatory Finding on the Emissions of Hazardous Air Pollutants From Electric Utility Steam Generating Units and the Removal of Coal- and Oil-Fired Electric Utility Steam Generating Units from the Section 112(c) List: Reconsideration regarding the Agency's assessment of utility attributable mercury concentrations in commercial freshwater fish, aquaculture, estuarine fish, and marine fish exposure pathways, including why EPA believes these pathways are not reasonably anticipated to result in a hazard to public health after reductions in power plant emissions due to CAIR and, independently, CAMR.

2. Marine (Open Ocean) Cycling

The inclusion of the exposure pathway associated with marine (open ocean) fish in the original freshwater quantitative analysis supporting the Revision Rule does not materially change the results because the impact of power plant emissions on mercury concentrations in open ocean environments is limited. First, over half of the U.S. commercial fish supply is imported. Second, the majority of domestic commercial fish is caught in open ocean regions that have not been impacted by anthropogenic mercury releases to the same extent as the atmosphere, near-shore, or inland systems. This means that current fish tissue concentrations likely do not reflect present day atmospheric mercury concentrations or deposition rates (Mason and Gill, 2005; Kraepiel et al., 2003). EPA could not support extension of the Mercury Maps freshwater modeling framework to marine systems. Application of the Mercury Maps model to marine environments would be an extension of the modeling framework beyond the realm for which it was intended for application and has not been empirically evaluated. The power plant contribution to mercury in these fish (discussed in more detail below) is difficult to quantify with confidence at this time and is expected to be relatively small in pelagic marine species based on the analysis described below.

Predatory marine fish are a significant source of methylmercury exposure for the U.S. population (Carrington et al. 2004). Exposure is a function of both the amount of mercury in fish and the quantities of fish consumed in the U.S. High levels of methylmercury in fish are generally the result of bioaccumulation in larger, older fish. In the case of marine fish, higher trophic level species tend to have comparable concentrations to top predators in freshwater ecosystems. In addition, the quantities of marine fish consumed by humans are larger than the quantities of freshwater fish. Based on the rationale described in detail below, EPA expects that utility-attributable mercury in these fish is a small fraction of their overall burden.

It is extremely difficult to determine the response of oceans to changes in mercury emissions from human sources due to limited scientific understanding at this time. However, the best available science suggests that the significance of changes in marine fish mercury concentrations in response to reductions in power plant Hg emissions will be small and will require on the order of decades to centuries to be achieved. To further elucidate our rationale, we present the results of a sensitivity analysis below that shows the relative importance of changes in present day atmospheric deposition on the

magnitude and timing of changes in ocean mercury concentrations in the Atlantic and Pacific Oceans.

In the original analyses supporting the final Section 112 rule, EPA quantified reductions in methylmercury concentrations in *freshwater fish* associated with declining inorganic mercury emissions from coal–fired utilities but concluded that the science of Hg cycling in marine systems is not sufficiently advanced to allow for a similar quantification of this exposure pathway. Other studies (NESCAUM 2005, Trasande et al. 2005) that did quantify the marine exposure pathway used assumptions for a central tendency estimate that are not supported by the literature on marine fate and transport of Hg, likely resulting in an overestimate of the power plant contribution to marine fish by an unknown but possibly large amount. We briefly discuss some of the assumptions of the NESCAUM report as they relate to marine fish in Section 9.

When quantifying the relationship between power plant mercury emissions in deposition over coastal and marine areas the NESCAUM study relied on the REMSAD model, which appears to over-predict Hg deposition from US power plants. Next, using the approach applied in NESCAUM study for marine systems would require an additional assumption that present day concentrations in the surface ocean are tracking changes in atmospheric concentrations and deposition. This premise is not supported by the screening analysis detailed below for marine systems. The proportionality assumption is also not applicable for coastal ecosystems with significant riverine inputs or watershed areas (most coastal systems).

One of the greatest uncertainties in coastal and marine systems is the rate and location of methylmercury formation. This is important because methylmercury formation rates determine bioavailability to fish and shellfish. The NESCAUM study assumed that changes in methylmercury concentrations in marine fish would be proportional to changes in deposition of (inorganic) mercury and surface water mercury concentrations. This actually embodies two assumptions – 1) changes in the surface water concentration of total mercury are proportional to changes in the air deposition of mercury and 2) changes in the methylmercury concentration in fish are proportional to the concentration of total mercury in the water. The literature offers no clear-cut guidance on how to address the second assumption. However, irrespective of this relationship between total Hg in water and MeHg in fish, the assumption of proportionality (first assumption) between changes in deposition and concentrations of Hg in the surface ocean is not consistent with some basic physical oceanographic principles that determine the magnitude and reservoir of Hg in the world's oceans. For example, a number of studies have measured large fluxes of mercury lost through gas exchange (volatilization) at the ocean surface (Amyot et al., 1997, Mason et al., 2001, Rolfhus and Fitzgerald, 2004). In addition, monitoring data indicate that concentrations of mercury in surface waters among the different oceans are not equal, and that deep ocean water concentrations are very low and likely unaffected by anthropogenic mercury (Gill and Fitzgerald, 1988, Mason and Sheu, 2002, Laurier et al., 2004). This means that the large flows of ocean waters (laterally among oceans, oceanic upwelling and deep water formation) will dilute or add (depending on the flow) to the existing mercury in

surface waters. A conceptual model of the various inputs and outputs of mercury, in addition to atmospheric deposition, is shown in Figure 2-1. A sensitivity analysis illustrating the magnitude of this uncertainty is presented below.

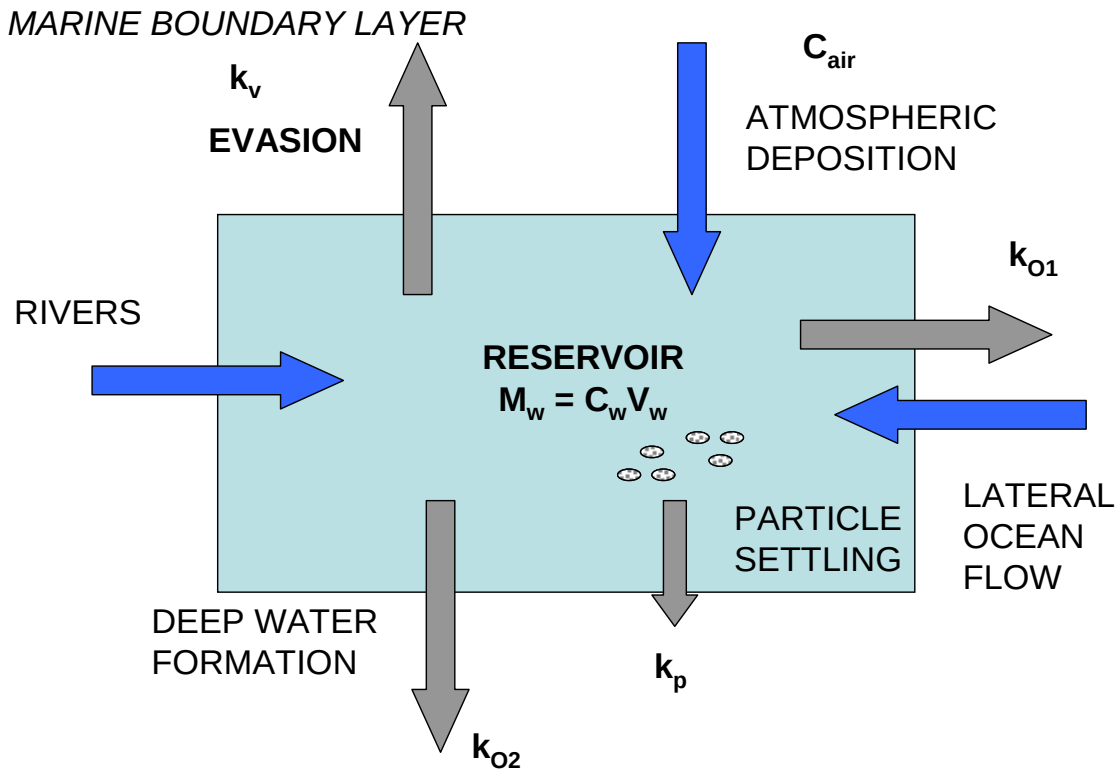

Figure 2-1. Conceptual model of mercury cycling in the surface waters of the Atlantic Ocean. Inputs are shown as blue arrows and outputs in grey arrows. The concentration of mercury in surface waters will be a function of the combined inputs and losses of mercury over time, with atmospheric deposition representing only one component of the overall flux of mercury.

Even if a proportional relationship between U.S. utility-attributable mercury deposition and marine methylmercury fish tissue concentration were a reasonable representation of the science, the saltwater species would nevertheless contribute only a very small amount of dietary utility attributable mercury because of the small contribution of the power plant emissions to the global pool. Annual emissions of mercury from U.S. utilities account for about 1.0 percent of total global emissions (including natural and recycled mercury). The total global emissions is estimated to be between 4400 and 7500 tonnes (US EPA 1997a), while total anthropogenic Hg emissions were approximately 2269 tonnes per year in 2001 (Pacyna and Munthe 2004). Overall, anthropogenic emissions from all sources in the United States still comprise less than 3 percent of the global total (UNEP 2002). Even in coastal environments where EPA's deposition models suggest that there is a higher contribution of Hg deposition by U.S. power plants, particularly the Atlantic and Gulf Coast area, the utility-attributable contribution to human mercury exposure in these regions is thought to be small as explained below.

2.1 Model of Mercury Cycling in World's Oceans

Modeling the effect the reduction in Hg deposition from U.S. power plants would have on Hg concentration in ocean fish is complicated by non-linear relationships between changes in human emissions, atmospheric Hg deposition over the oceans, water column concentrations and fish Hg concentrations. In contrast to freshwater systems, there is a paucity of field data on factors controlling MeHg formation and concentrations in marine environments and it is not clear where the majority of methylation occurs in the ocean (see review by Mason and Gill, 2005).

Mason and Sheu (2002) suggest that Hg in the ocean has only increased by 9 percent relative to pre-industrial levels even though air releases have more than doubled. Several revisions to this estimate, including Mason and Gill (2005), have suggested a larger anthropogenic contribution to mercury in the world's oceans of up to 60 percent in the Atlantic. This can be compared to the average global enrichment of the atmosphere which is between 200-500 percent. These revised estimates all support the premise that surface ocean concentrations are not tracking atmospheric deposition. Further supporting this premise are empirical data from the Pacific Ocean for water column mercury concentrations (Laurier et al., 2004) and tuna (Krapiel et al., 2004), which show no significant differences in concentrations over the last 20-30 years. Presently, there are no data to support the assumption that atmospherically deposited Hg would be preferentially converted to MeHg relative to the large reservoir of legacy Hg in marine environments. This preferential conversion would be one condition needed to achieve the assumed near-instantaneous proportional reduction in marine species MeHg concentrations described in the NESCAUM study. Based on this information, EPA concludes that the linear approach employed in the NESCAUM study will likely lead to a significant overestimation of utility attributable exposure and an even greater overestimation of the benefits of reducing power plant emissions.

At this time there are insufficient empirical and mechanistic data on the rate and location of MeHg formation in marine environments to constrain a detailed predictive model for MeHg cycling and accumulation in fish in the open ocean. However, for the purposes of developing a comparable quantitative model to the NESCAUM report and to provide a sensitivity analysis, EPA has adapted a recent modeling approach by Mason (in review) with results discussed in Mason and Gill (2005). This adapted model is used to investigate the influence of changing atmospheric deposition rates resulting from control of coal-fired utilities on concentrations of total Hg in the open ocean water. EPA recognizes that this assumption is a simplification of Hg bioaccumulation in marine environments and must be viewed as a preliminary assessment of the potential response of marine environments to changes in atmospheric Hg deposition and must be interpreted with caution.

We selected the model approach discussed in Mason and Gill (2005) to describe Hg cycling in marine environments because it represents the interconnectedness of the

different oceans through lateral transport of surface waters, upwelling, and deep-water formation in the North Atlantic. The model is based on the 16-box model for the world's oceans published by Kahana, et al. (2004) (see Figure 2-2). Kahana et al.'s model is an expanded version of Stommel's pioneering box model (Stommel, 1961), which originally described ocean circulation as a function of salinity and temperature. Inputs of Hg from major rivers globally are characterized using concentrations data from Cossa, et al. (1996), and flux data from the National Center for Atmospheric Research (NCAR) website (www.cgd.ucar.edu/cas/catalog/dai/runoff-table2-top50r.html). Losses of Hg through particulate settling and evasion are characterized using mercury-to-carbon (Hg:C) ratios for different oceans described in Mason, et al. (1994), and empirically measured values for different oceans, respectively. Overall, fluxes are constrained using empirical data on measured fluxes in each compartment and the Mason and Sheu (2002) global budget. Atmospheric deposition for each ocean was estimated from measured wet deposition rates over the oceans from existing data at that time, developed by Mason, et al. (1994), and revisited for the Mason and Gill (2005) analysis to update estimated dry deposition based on gradients in total gaseous mercury (TGM) concentrations in the marine boundary layer (MBL).

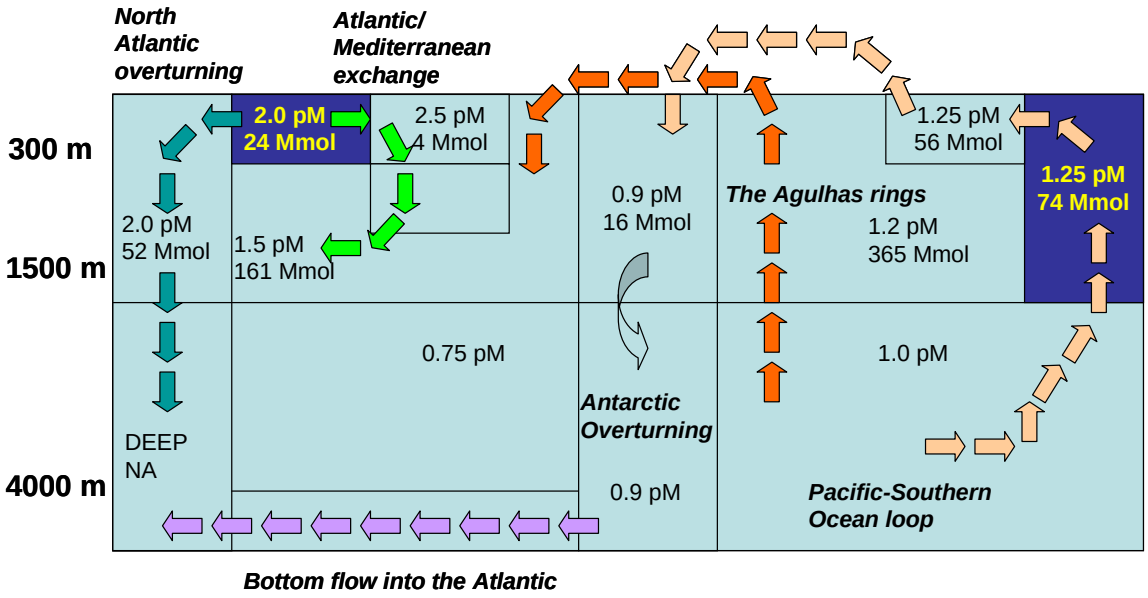

Figure 2-2. Conceptual model of the major oceanic circulation patterns adapted from Kahana et al. (2004). Also shown are average mercury concentrations (pM) and reservoirs (Mmol) of mercury in each of the ocean compartments based on the data and model discussed in Mason and Gill (2005).

Advantages of this adapted modeling approach compared to previously published models of Hg cycling in the oceans include:

1. The model treats different oceans and well-mixed components of these oceans in a discrete manner and constrains concentrations and fluxes using the most recent field-data.

2. The model is based on a simplified oceanographic model allowing for various depths of the mixed layer determined by the locations of the permanent thermoclines in each ocean rather than assumption of 100 meter mixed layer for all oceans. This is important because it determines the reservoir of mercury present in each ocean and affects the temporal responses of each ocean to changes in deposition.

3. The model includes empirically based fluxes of Hg through evasion from surface waters, particulate settling and riverine inputs as well as advection resulting from major ocean circulation patterns.

To model the potential response of marine fish to changes in Hg deposition resulting from the power plant regulation the following assumptions are necessary:

1. Change in fish mercury concentrations will be proportional to the change in total mercury concentrations in the water column.

2. Power plant regulation will not significantly affect ocean concentrations other than the Surface Atlantic and North Pacific. This means that we are assuming utility attributable mercury in the U.S. will not significantly change concentrations of mercury in other oceans (e.g., Antarctic and Indian Oceans, etc.)

3. Rate constants describing the various inputs and outputs of mercury in surface waters (see Figure 2-1) can be reasonably characterized using available empirical data on measured mercury fluxes. Note that the NESCAUM modeling approach did not use rate constraints because it assumes that any change in ocean mercury concentrations will be proportional to changes in atmospheric deposition.

Table 2.1 Comparison of major assumptions used in NESCAUM analysis to this analysis based on Mason and Gill (2005)

Study Characteristics	NESCAUM Analysis	This Analysis
Assume equilibrium between Hg in the ocean and atmospheric Hg deposition	Yes	No
Assume change in total mercury concentration in the water is proportional to change in atmospheric mercury deposition	Yes	No
Based on oceanographic circulation data (e.g., to characterize depth of well-mixed surface waters impacted by atmospheric deposition)	No	Yes
Evasion of Hg^0, particulate transport, lateral advection of water (inputs and outputs of Hg), deepwater formation, upwelling included explicitly in model	No	Yes
Assumes change in methyl mercury concentration in fish is proportional to change in total mercury concentration in water	Yes	Yes

2.2 Results of Ocean Model Sensitivity Analysis

Results for EPA's sensitivity analysis are presented in tables 2.2 and 2.3. The adapted marine cycling model was first run to reflect steady state conditions in both ocean compartments (Atlantic and Pacific). The empirically constrained fluxes of mercury for both oceans indicate that neither ocean is currently at steady state. This can be seen in Figures 2-3 and 2-4 by virtue of the fact that adding all the input and output fluxes results in a net loss of mercury in the Atlantic and a net gain of mercury in the Pacific

We can also use the empirical data to estimate the time needed to reach steady state in each ocean by calculating the sum of rate constants describing inputs and losses of mercury to each ocean. Rate constants were calculated by dividing the empirically constrained fluxes by the total reservoir of mercury in each ocean compartment. Using this methodology, the Atlantic Ocean is expected to reach steady state (ocean concentrations reflect present atmospheric deposition) in approximately 3 decades with on the order of a seven percent decline in fish mercury concentrations (Figure 2-3). The North Pacific has a somewhat larger reservoir of mercury and a deeper mixed layer (see Figure 2-2 and 2-4). Thus, the time to steady state (ocean concentrations reflect present atmospheric deposition) for the North Pacific is on the order of 2 centuries and results in a ten percent increase in mercury in fish if all other factors are constant. Note that these calculations assume no changes in mercury inputs over time (i.e., present conditions remain constant).

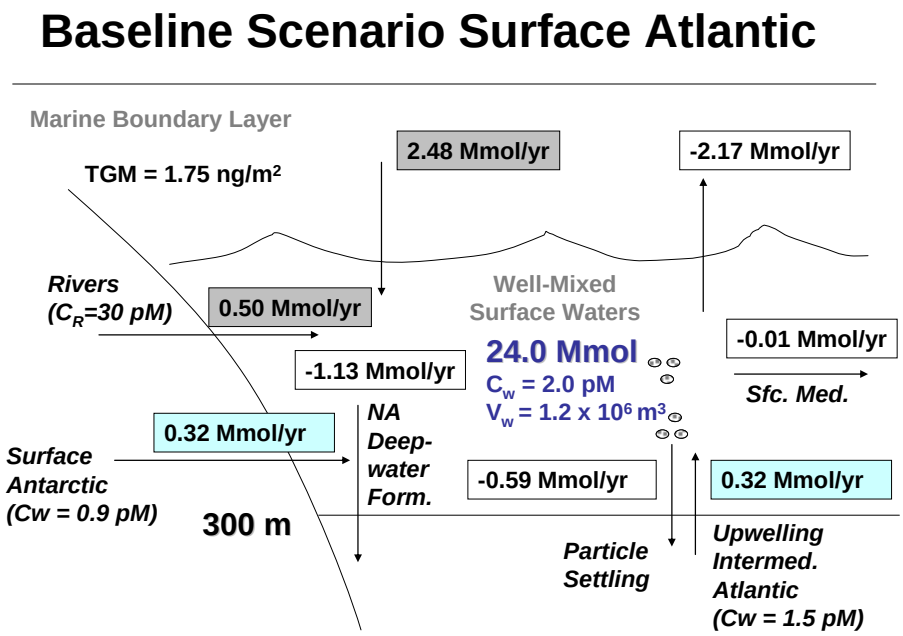

Figure 2-3. Model of mercury inputs and outputs in the surface Atlantic Ocean based on data from Mason and Gill (2005) and Mason (2005).

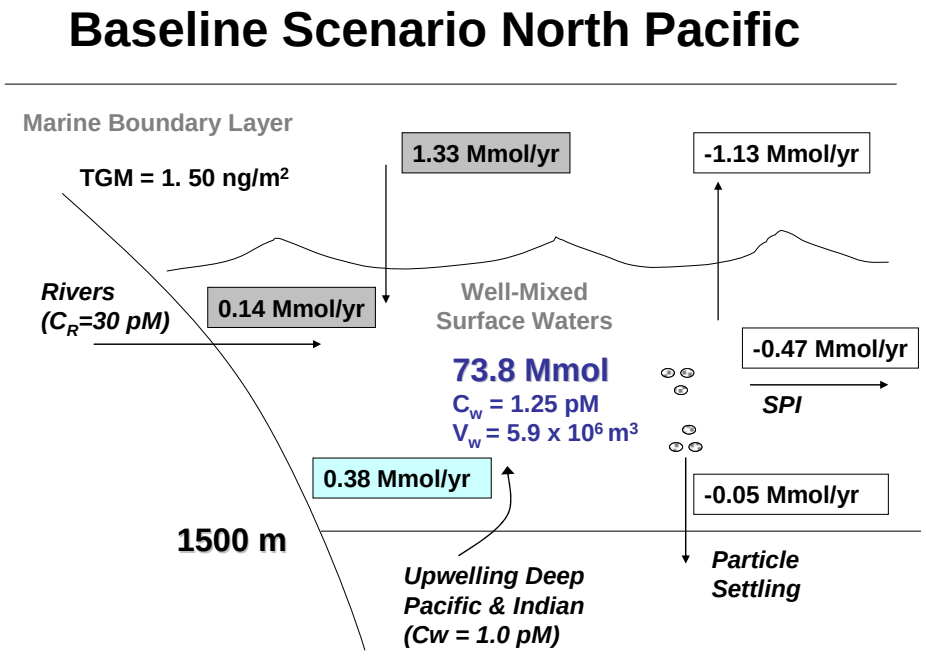

Figure 2-4. Model of mercury inputs and outputs in the North Pacific based on data from Mason and Gill (2005) and Mason (2005).

After running the model to steady state, a baseline scenario was developed to explore the potential contributions of utilities to the Atlantic and Pacific oceans. To do this, mercury inputs from atmospheric deposition were adjusted by the relative change in deposition forecasted in nearshore areas using the CMAQ model after power plant emissions are removed (2 percent in the Atlantic and 0.65 percent in the North Pacific). Because the changes in atmospheric deposition were taken from nearshore rather than offshore areas, they represent an upper bound for changes in deposition that might be attributable to U.S. coal fired utilities. In addition, changes in deposition resulting from the power plant regulation could also affect river inputs of mercury from North America to the Atlantic and Pacific Oceans. However, these changes are likely to be relatively small given the dominance of freshwater inputs to the ocean basins from South America and European rivers relative to North America. Such potential changes in river mercury concentrations have not been quantified at this time; thus, for the purposes of the sensitivity analysis we explore a range of reductions in riverine mercury inputs between zero and two percent. The upper bound of this analysis is likely a significant overestimate of potential changes in riverine mercury inputs to the oceans and should be caveated appropriately. As a sensitivity analysis, we also explore a range in potential atmospheric reductions associated with removal of U.S. utilities as a source of mercury. In the Atlantic ocean the range in atmospheric reductions explored is 0.5 percent and for the North Pacific it is 0.1 to 1 percent.

Table 2.2 Sensitivity analysis for the Atlantic Ocean to assess potential change in mercury concentrations in marine fish observed at steady state as the result of control of power plant emissions. Note that changes are expressed as a fraction of the current fish mercury concentration. The top row shows a hypothetical, upper bound range for the change in mercury concentrations in riverine inputs to the ocean, the left column indicates the decrease in atmospheric deposition of mercury.

		Hypothetical Decrease in Hg River Inputs						
		0.0%	**0.5%**	**0.7%**	**1.0%**	**1.5%**	**1.8%**	**2.0%**
Hypothetical Decrease in Hg Deposition[*]	**0.5%**	-0.3%	-0.4%	-0.4%	-0.5%	-0.5%	-0.6%	-0.6%
	1.0%	-0.7%	-0.8%	-0.8%	-0.8%	-0.9%	-0.9%	-1.0%
	1.5%	-1.0%	-1.1%	-1.1%	-1.2%	-1.2%	-1.3%	-1.3%
	2.0%	-1.4%	-1.4%	-1.5%	-1.5%	-1.6%	-1.6%	-1.6%
	2.5%	-1.7%	-1.8%	-1.8%	-1.9%	-1.9%	-2.0%	-2.0%
	3.0%	-2.1%	-2.1%	-2.2%	-2.2%	-2.3%	-2.3%	-2.3%
	3.5%	-2.4%	-2.5%	-2.5%	-2.5%	-2.6%	-2.6%	-2.7%
	4.0%	-2.7%	-2.8%	-2.8%	-2.9%	-2.9%	-3.0%	-3.0%
	5.0%	-3.4%	-3.5%	-3.5%	-3.6%	-3.6%	-3.7%	-3.7%

[*]Note that the ranges in atmospheric deposition used above are meant to explore the sensitivity of model forecasted water/fish mercury concentrations to changes in atmospheric deposition. EPA's analysis indicates that a 1% change in atmospheric deposition over the Atlantic Ocean is a reasonable upper bound for changes in atmospheric deposition resulting from reductions in utility emissions (see Section 3).

Table 2.3 Sensitivity analysis for the North Pacific to assess potential change in mercury concentrations in marine fish observed at steady state as the result of control of power plant emissions. Note that changes are expressed as a fraction of the current fish mercury concentration. The top row shows a hypothetical, upper bound range for the change in mercury concentrations in riverine inputs to the ocean, the left column indicates the decrease in atmost pheric deposition of mercury.

		Hypothetical Decrease in Hg River Inputs						
		0.0%	**0.1%**	**0.2%**	**0.3%**	**0.4%**	**0.5%**	**1.0%**
Hypothetical Decrease in Hg Deposition	**0.1%**	-0.1%	-0.1%	-0.1%	-0.1%	-0.1%	-0.1%	-0.1%
	0.2%	-0.1%	-0.1%	-0.1%	-0.2%	-0.2%	-0.2%	-0.2%
	0.3%	-0.2%	-0.2%	-0.2%	-0.2%	-0.2%	-0.2%	-0.3%
	0.4%	-0.3%	-0.3%	-0.3%	-0.3%	-0.3%	-0.3%	-0.4%
	0.5%	-0.3%	-0.4%	-0.4%	-0.4%	-0.4%	-0.4%	-0.4%
	0.6%	-0.4%	-0.4%	-0.4%	-0.4%	-0.5%	-0.5%	-0.5%
	0.7%	-0.5%	-0.5%	-0.5%	-0.5%	-0.5%	-0.5%	-0.6%
	0.8%	-0.6%	-0.6%	-0.6%	-0.6%	-0.6%	-0.6%	-0.6%
	0.9%	-0.6%	-0.6%	-0.7%	-0.7%	-0.7%	-0.7%	-0.7%
	1.0%	-0.7%	-0.7%	-0.7%	-0.7%	-0.7%	-0.7%	-0.8%

2.3 Conclusions regarding Ocean Mercury Cycling

In summary, empirical data suggest that oceans are not at steady state. The analysis described above suggests that the Atlantic Ocean can be expected to reach steady state in approximately 3 decades if atmospheric deposition were to be held constant, while the North Pacific may take 200 years or more to reach steady state if atmospheric deposition were to be held constant. In addition, if all other environmental factors remain the same, our sensitivity analysis suggests that at steady state mercury concentrations will decline by approximately 7% in the Atlantic and increase by slightly more than 10% in the Pacific. This is consistent with observed declines in anthropogenic emissions in North America over the past several decades and increasing emissions from Asian countries affecting the Pacific Oceans (see Mason (2005) for more information). In both areas, EPA expects that for a given decrease in deposition, the decrease in fish tissue concentration will be less than the decrease in deposition (i.e., linear assumption is an overestimate). Furthermore, EPA believes that there is currently not enough information to conduct a quantitative assessment of the central tendency of the impact of changes in emissions from power plants on changes in marine fish Hg concentrations. Because of the inherent uncertainties in some of our assumptions and calculations presented above, we do not link the sensitivity analysis calculations presented above to the human exposure scenarios discussed in section 3 (below). Instead we use the results of the sensitivity analysis to aid in interpreting the results of the following sections.

3. Utility-Attributable Mercury in Marine Seafood Diet

EPA does not believe that there is a proportional relationship between air deposition and MeHg concentrations in marine fish tissue. In fact, as stated in Section 2,

EPA expects that for a given decrease in deposition, the decrease in fish tissue concentration will be less than the decrease in deposition. However, due to the scientific uncertainty on this point, EPA feels that it is not appropriate at this time to use the results from Section 2 to create IDI values for marine fish. Instead, we conduct a bounding analysis of the utility attributable mercury in marine fish assuming a proportional relationship between utility-attributable mercury deposition decreases and MeHg fish tissue concentration. This is the same assumption as was used for the self-caught freshwater analysis and, for reasons stated in the Section 2, this likely overstates the utility-attributable contribution. Even with this upper bound assumption, the marine fish pathway results in only a small contribution to methylmercury consumption due to the small contribution of the U.S. power plant emissions to open ocean environments.

While the EPA CMAQ model does extend into the Atlantic and Gulf Coast region, and could potentially be used to determine the utility-attributable portion of mercury deposition in 2020 after the implementation of CAIR, the modeled deposition in the open ocean region is quite small. Using CMAQ would indicate that the average post-CAIR utility attributable portion for the Atlantic and Gulf Coast Ocean in 2020 would be less than one percent. Another measure of open ocean mercury deposition may be drawn from the U.S. power plant contribution to the total global mercury pool. U.S. anthropogenic Hg emissions are estimated to account for roughly three percent of the global total, and emissions from the U.S. power sector are estimated to account for about one percent of total global emissions (US EPA 1997a). Since both estimates are in the range of one percent, EPA believes that changes in power plant Hg emission in this rule will have a very small impact through this exposure pathway.

3.1 Index of Daily Intake for Average Seafood Consumption by the General U.S. Population

In order to describe the utility-attributable portion of Hg in the typical seafood diet, we used the Index of Daily Intake (IDI) values, as was described in the Effectiveness TSD (U.S. EPA, 2005a). The IDI is an index of exposure to Hg due solely to power plants. An IDI of 1 or greater indicates that an individual exposure to mercury from power plants is equal to or exceeds the EPA reference dose (RfD) for mercury due solely to utility-attributable mercury exposure.

This index uses the RfD for Hg of 0.1 microgram per kilogram body weight per day (ug/kg-bw/day) as a reference point. The IDI is defined as the ratio of exposure due solely to power plants divided by the reference dose. The Hg exposure due solely to power plants is calculated as

Equation 3.1: Exposure due to power plants

$$\left\{\begin{array}{c}\text{Exposure}\\ \text{due to}\\ \text{power}\\ \text{plants}\end{array}\right\} = \frac{\left\{\begin{array}{c}\text{consumption}\\ \text{rate}\end{array}\right\} \text{X} \left\{\begin{array}{c}\text{methylmercury}\\ \text{concentration}\\ \text{due to power plants}\end{array}\right\} \text{X} \ (1.5)}{(\text{bodyweight})}$$

Consumption rates are generally given in ounces of cooked fish, therefore, the 1.5 in this equation reflects the increase in the concentration of Hg from cooking. It should be noted that this implies a concentration factor of .667 of initial fish weight. Carrington et al. (2004) report less concentration of methylmercury occurring from cooking (i.e. a concentration factor between 1 and .737) , making the use of a concentration factor of .667 an upper bound assumption of the amount of methylmercury in cooked fish. This exposure value is then divided by the RfD of 0.1 to obtain the IDI.

The Effectiveness TSD (EPA 2005a) lists the IDI values for freshwater fish consumption at various rates of consumption in Table 6-4. To create an equivalent table for marine fish consumption, we require the concentration of methylmercury in seafood and consumption rates. Carrington et al. (2004) provide a table (Table 3.2) of the mercury concentration for 42 commercial finfish and shellfish seafood species. This is a relatively comprehensive list, containing 99 percent of the market share for commercial seafood, and includes fish in both open ocean and near shore areas. The Hg concentration for the seafood diet of the general population can then be estimated by summing the product of Hg concentration times market share for each species. This produces an estimate of a mean Hg concentration of 0.104 ppm for the seafood diet of the general population. Note that this is the Hg concentration from *all* sources in all countries; U.S. power plants account for a small fraction of this total.

Consumption rates of marine fish can be obtained from the Exposure Factors Handbook (US EPA 1997b). The mean intake of all fish species (marine, freshwater, estuarine, and aquaculture) for the general population is 20.1 grams per day (g/day) with an estimate of the 95 percent consumption level of 63 g/day. For marine fish alone, the best estimate of general population consumption is 14.1 g/day. If we assume the distribution of marine fish is proportional to total fish consumption, then the 95th percentile of marine fish consumption would be approximately 44 g/day.

The 95th percentile consumption value of 65 g/day of all fish (marine, freshwater, estuarine, and aquaculture) is generally consistent with Carrington and Bolger (2002) estimate of seafood consumption, based on data from the U.S. Department of Agriculture Continuing Survey of Food Intake by Individuals. Adjusting their data to account for the consumption of high-frequency consumers, they estimate a 95th consumption rate for all seafood[1] of 62.7 g/day. They also estimate consumption rates of 6.8, 41.5, 62.7, 123.2, 141.3, and 206.6 g/day for the median, 90th, 95th, 99th, 99.5th, and the 99.9th percentile, respectively. This information can be used to estimate the consumption rate at very high levels of consumption.

Table 3.1 below lists the mercury exposure, in µg/kg-bw/day, from <u>all</u> sources at the various rates of consumption described above. Values of 0.1 or greater, which occur

1 Carrington and Bolger (2002) use records of seafood-consumption events. We assume that this includes consumption from all fish (marine, freshwater, estuarine, and aquaculture). As such, the tables in this section that include high-end consumption values must be interpreted as if all of the consumer's fish diet came from marine fish.

at consumption levels between 90 and 95 percent, imply that some individuals are experiencing levels of Hg above the RfD due to fish consumption. It is very important to note, however, that this is the Hg exposure from all sources. This includes natural sources as well as man-made sources, both U.S. and from other countries. It also assumes that the mercury concentration in fish tissue consists entirely of MeHg, biasing the estimates upward.

Table 3.1 Mercury Exposure from Seafood Consumption from all Sources (units in µg/kg-bw/day)

Consumption Rate	g/day	
Mean Marine Fish Intake	*14.1*	0.03
95% Long-Term Marine Fish Intake	*44*	0.10
Mean Total Fish Intake	*20.1*	0.05
95% Long-Term Total Fish Intake	*63*	0.15
Median Carrington & Bolger Seafood Consumption	*6.8*	0.02
90% Carrington & Bolger Seafood Consumption	*41.5*	0.10
95% Carrington & Bolger Seafood Consumption	*62.7*	0.15
99% Carrington & Bolger Seafood Consumption	*123.2*	0.29
99.5% Carrington & Bolger Seafood Consumption	*141.3*	0.33
99.9% Carrington & Bolger Seafood Consumption	*206.6*	0.48

Note: Mean fish tissue methylmercury concentration from all sources is 0.1 ppm.

The scaling method used for freshwater fish assumed a proportional relationship between reductions in air deposition of Hg and MeHg concentrations in fish. This relationship has been documented in the MMaps approach (US EPA 1997a, US EPA 2001). The MMaps model assumes that, in steady-state, changes in MeHg concentrations in fish are proportional to changes in Hg inputs from atmospheric deposition. This solution only applies to situations where air deposition is the only significant source of Hg to a water body, and the physical, chemical, and biological characteristics of the ecosystem remain constant over time. These conditions do not hold in open ocean systems. As discussed in the previous section, assuming proportionality would most likely overstate the change in MeHg concentration in marine systems given a change in the atmospheric deposition of Hg. This means that assuming proportionality to calculate the marine fish Hg levels due to utilities would likely overstate the contribution of those utilities. This assumption could be useful, however, to produce an estimate that likely overstates the utility-attributable Hg levels in marine fish. If this utility attributable estimate is deemed not to represent a hazard to public health, we can reasonably conclude that a more realistic estimate, although not currently quantifiable, would also not represent a hazard to public health. Using the bounding assumption of proportionality between reductions in Hg deposition and reductions in fish tissue concentrations, then we can produce an approximation of the IDI for the 2001 base case.

Broadly speaking, global Hg deposition comes from three sources, approximately one-third comes from natural sources (e.g., volcanoes), one-third comes from the

emissions of modern man-made sources, and one-third from redeposition.[2] Mercury emissions from the U.S. power sector are estimated to account for about one percent of total global emissions (US EPA 1997a). We use this one-percent as the bounding estimate of the open ocean fish concentration reductions that could be expected from a 100 percent reduction in power plant emissions in 2001.[3] This assumes proportionality between emissions and open ocean fish concentrations, likely an overestimate, and so is used in this bounding calculation .[4] Multiplying the exposure factors in Table 3.1 by this one percent and dividing by the RFD of 0.1 produces the IDI values for marine fish consumption in Table 3.2 below. Since all values in Table 3.2 are all well below one, we can conclude that Hg emissions from U.S. power plants do not cause a health concern and do not significantly contribute to the U.S. general public's exposure to Hg due to marine fish consumption.

Table 3.2 IDI values for Seafood Consumption for the General U.S. Population (units in IDI values) (2001)

Consumption Rate	g/day	
Mean Marine Fish Intake	*14.1*	0.00
95% Long-Term Marine Fish Intake	*44*	0.01
Mean Total Fish Intake	*20.1*	0.00
95% Long-Term Total Fish Intake	*63*	0.01
Median Carrington & Bolger Seafood Consumption	*6.8*	0.00
90% Carrington & Bolger Seafood Consumption	*41.5*	0.01
95% Carrington & Bolger Seafood Consumption	*62.7*	0.01
99% Carrington & Bolger Seafood Consumption	*123.2*	0.03
99.5% Carrington & Bolger Seafood Consumption	*141.3*	0.03
99.9% Carrington & Bolger Seafood Consumption	*206.6*	0.05

Note: Mean fish tissue methylmercury concentration from all sources is 0.1 ppm. The utility attributable fraction of that concentration is used for calculating IDI values.

3.2 IDI for Consumption of Species with Very High Methylmercury Concentrations

The results above use a mean fish tissue MeHg concentration of an average seafood diet, proportional to the market share for commercial seafood. This understates the Hg exposure from an individual who eats proportionally more high-Hg fish than the average consumer. As an extreme assumption, we consider the MeHg concentration for a

2 see http://www.epa.gov/mercury/control_emissions/global.htm

3 Another possible measure would be data from the CMAQ model used for the Revision Rule. Using the difference between Hg deposition in the coastal Atlantic and the Gulf of Mexico region in the base case and the 2020 zero-out CMAQ run, it is estimated that U.S. power plants contribute an average of less than one-percent to total deposition in this area. Averaging the entire Atlantic and Gulf Coast ocean area modeled by CMAQ implies an average change in atmospheric deposition of 0.65 percent in 2020 using a "zero out" of utility mercury emissions from a post-CAIR baseline. We use the estimate of the contribution of U.S. power plant emissions to the global pool of one percent rather than the value from the CMAQ model because it provides an upper bound estimate.

4 By 2020 after CAIR and furthermore after CAMR, this fraction would decline.

fish with a concentration of 1ppm.[5] This is one of the highest Hg concentrations from marine fish and represents a type of fish that a person might potentially eat on a regular basis.

As an extreme assumption, we consider an individual whose fish consumption comes exclusively from a marine species with a methymercury concentration of 1.0 ppm. Table 3.3 uses this MeHg concentration of 1.0 ppm, the one percent change in MeHg concentration, and the various consumption levels from Table 3.1. Again, the IDI value never exceeds one, even for a 99.9 percent consumption level. Given that this assumes someone exclusively eating a fish with one of the highest marine Hg concentrations and eating at the highest consumption rate, it is reasonable to conclude that U.S. power plants do not significantly contribute to the U.S. exposure to Hg from marine fish consumption.

Table 3.3 IDI values for Exclusive Consumption of Marine Species with High Methylmercury Concentration
(units in IDI values)

Consumption Rate	g/day	
Mean Marine Fish Intake	*14.1*	0.03
95% Long-Term Marine Fish Intake	*44*	0.10
Mean Total Fish Intake	*20.1*	0.05
95% Long-Term Total Fish Intake	*63*	0.15
Median Carrington & Bolger Seafood Consumption	*6.8*	0.02
90% Carrington & Bolger Seafood Consumption	*41.5*	0.10
95% Carrington & Bolger Seafood Consumption	*62.7*	0.15
99% Carrington & Bolger Seafood Consumption	*123.2*	0.29
99.5% Carrington & Bolger Seafood Consumption	*141.3*	0.33
99.9% Carrington & Bolger Seafood Consumption	*206.6*	0.48

Note: High fish tissue methylmercury concentration from all sources is 1.0 ppm. The utility attributable fraction of that concentration is used for calculating IDI values.

3.4 Conclusions Regarding the Marine Pathway

In summary, EPA believes that the utility-attributable portion of Hg in the seafood diet is not significant. However, EPA is soliciting comments on the IDI constructed above as well as other methods to account for commercially important marine fish that have relatively high Hg concentrations.

4. Estuarine Near-Shore Exposure Pathway

5 Carrington, Montwill, and Bolger (2004) report a mean Hg concentration of 0.97 ppm and a median concentration of 0.86 ppm for swordfish.

Despite the lack of a comprehensive quantitative analysis, EPA finds that the available data support our position that the utility attributable U.S. population wide exposure to methylmercury from estuarine fish and shellfish will likely be small relative to that from recreational self-caught freshwater fish described in previous sections of the Effectiveness TSD. In general, coastal finfish species of the same age/size and trophic level as freshwater fish have relatively lower mercury concentrations (Mason and Gill, 2005).

Overall methylmercury exposure from coastal fish and shellfish is likely smaller than exposure from a comparable amount of freshwater fish. First, coastal fish of the same age/size and trophic level as freshwater fish generally have relatively lower mercury concentrations (Mason and Gill, 2005). Second, shellfish tend to have a lower fraction of methylmercury relative to their total mercury burden than freshwater fish (Bloom et al. 1992, Joiris et al. 2000, Mikac et al. 1985), which further lowers methylmercury exposure from all coastal species.

When compared to overall landings of marine fish in the U.S., domestically caught estuarine fish and shellfish (defined as those harvested within 3 miles from shore) make up only 38 percent of the total 2001 commercial fish and shellfish landings in the U.S. (NMFS 2002) Based on the average mercury content of estuarine fish and shellfish, a conservative (high) estimate of total mercury exposure is approximately 23 percent of the total mercury intake from the U.S. Commercial Seafood Market (described below). A comparably larger fraction of the seafood consumed in the U.S. originates from open ocean/marine areas discussed in section 2. The utility attributable fraction of this 23 percent will also be very small as indicated by data showing that: 1) Utility attributable mercury deposition in near-shore areas is small relative to inland areas where recreational fishing occurs; 2) Legacy mercury sources and inputs from watershed areas in most coastal systems will further lower the percentage of utility attributable mercury in estuarine fish and shellfish.

The following sections outline EPA's rationale for concluding that utility attributable human exposure resulting from consumption of estuarine fish is small. First, we discuss the major scientific uncertainties in the estuarine exposure pathway. Second, we review the data on consumption of estuarine fish and shellfish in the United States and the relative exposure to mercury from this source compared to other pathways. Finally, we review the utility attributable deposition estimates in near-shore area of the United States and discuss how this may affect concentrations in estuarine species.

4.1 Uncertainty in the Near-Shore Fish Exposure Pathway

EPA believes that the state of the science currently does not support a national-scale quantitative analysis for this component of the exposure pathway. The most important difficulty in conducting this analysis is determining the response of coastal and estuarine systems to changes in atmospheric mercury deposition. Some studies have assumed an instantaneous proportional relationship between declines in deposition and concentrations in estuarine fish to complete their exposure analysis. However, such an

assumption has not been endorsed by EPA or the scientific community as an appropriate method for characterizing the effects of emissions reductions on estuarine fish concentrations.

A proportional relationship between mercury deposition and the methylmercury concentration in fish was developed and has been evaluated for application for certain freshwater water bodies across the U.S. if air deposition is the only significant source of mercury to a water body (US EPA 1997a, US EPA 2001). As described above, such a relationship is thought to be an overestimate for marine systems so can be used in a bounding calculation. Applying such a relationship to estuarine systems extends this assumption beyond the realm of application for which this model was developed for freshwater systems and, unlike marine systems, it is not known whether it would likely be an overestimate for all estuaries. External sources of methylmercury to estuaries, like other aquatic systems, are typically small such that *in situ* methylmercury production accounts for the majority of mercury in fish (Mason and Benoit 2003). Mercury cycling in estuarine areas differs significantly from both the open-ocean and freshwater environments. Because estuaries are much shallower than open ocean areas, active methylmercury production in estuarine sediments is an important source of mercury for fish and shellfish residing in these regions (Hammerschmidt et al, 2004, Cossa and Gobeil, 2000, Lawson et al., 1998). Unlike freshwater systems, most estuaries have significant inputs of tidal water, which generally dilutes incoming mercury from rivers, streams, and direct atmospheric deposition. In addition, because of the saline environments found in estuaries, the geochemistry of these systems differs significantly from inland lakes, affecting the rate and magnitude of methylmercury production (Heyes et al., 2005). There is, however, a paucity of data and modeling describing mercury cycling in coastal areas compared to freshwater and marine environments. This limits our ability to develop a comprehensive quantitative assessment of the exposure pathway at this time.

Some empirical observations of mercury cycling in coastal ecosystems often show total mercury concentrations are not necessarily good predictors of ambient methylmercury concentrations (Benoit et al., 2003). In addition, such an estimate likely overestimates the response of coastal ecosystems to changes in atmospheric deposition if all other ecosystem characteristics remain constant. A number of studies have shown that the production and bioavailability of mercury in coastal ecosystems is a function of environmental characteristics like total organic carbon, sulfides concentrations in water and sediments and temperature (Benoit et al, 1999, Sunderland et al, 2005). When the methylation potential of estuarine systems is limited by some environmental or geochemical characteristic of the ecosystem, small changes in inorganic mercury deposition from the atmosphere are unlikely to significantly impact methylation rates and ultimately fish methylmercury concentrations. Fish methylmercury concentrations in coastal ecosystems that have been significantly impacted by legacy mercury sources (e.g., San Francisco Bay, CA, Lavaca Bay, TX) are also unlikely to be affected by small changes in atmospheric deposition because they receive the majority of their mercury from watershed sources and from contaminated sediments (Bloom et al, 1999, Sager, 2002, Conaway et al., 2003).

Most coastal areas in the United States are highly populated and therefore receive significant mercury loadings from land based sources through wastewater effluents and other industrial contamination. In addition, most coastal regions are likely to have relatively deep active sediment layers relative to freshwater systems due to mixing processes that occur when rivers and tidal waters meet. Effectively, a deeper active sediment layer allows coastal systems to store legacy contaminants and allows historically released mercury to interact with the water column, undergo methylation and accumulate in the food-web, slowing the ecosystems response to changes in mercury inputs (Sunderland et al., 2004). Coastal systems with deep active sediment layers that contain large amounts of legacy Hg from past anthropogenic contamination and large contributions from watershed-based Hg sources will respond very slowly to changes in emissions from utilities, and the magnitude of this response will also likely be small.

Finally, the life cycles of marine species (fraction of time spent in near-shore relative to pelagic environments) needs to be considered to accurately model mercury bioaccumulation. Many of the coastal and estuarine species, listed in Table 4.1 below, spend a portion of their lifecycle in near-shore areas and the remainder in open-ocean regions. This behavior is likely to further lower the significance of utility-attributable mercury in the U.S. on methylmercury concentrations in these species.

Based on this, EPA believes that a simple linear relationship that assumes instantaneous changes in Hg levels in fish and shellfish across all estuarine systems in the U.S. is proportional to changes in atmospheric deposition does not appropriate reflect the best available information on mercury cycling in coastal ecosystems. As such, we cannot conduct a comprehensive, national-scale quantitative analysis of this pathway for all estuary and coastal regions.[6]

4.2 Consumption of Near-Shore Species in the United States and Associated Mercury Exposure

To assess the total mercury exposure from consumption of fish and shellfish in the United States, we analyzed commercial landings data from the National Marine

[6] EPA has distinguished between estuarine and open ocean (marine systems) in this analysis. Bounding calculations were possible for marine systems because of the relative homogeneity of human influences on mercury cycling in marine systems compared to estuarine systems. Specifically, open-ocean environments are isolated from local variability in methylmercury formation rates and biotic concentrations that are commonly observed in estuaries. This is because open ocean environments do not exhibit the same variability in geochemical properties affecting methylmercury formation as estuaries (i.e., the deep water column means that methylmercury production in the sediments has a negligible effect on water column concentrations). Finally, we believe that the analysis for the open ocean environment is justifiable because it is a sensitivity analysis taking into account the limited nature of the data on mercury fate and transport in the open-ocean. Given the regional differences in utility attributable mercury deposition to estuaries in the US, the fraction of mercury deposited that is converted to methylmercury, and fish methylmercury concentrations, we felt that it would be unrealistic to attempt to develop a national estimate of changes in mercury exposure resulting from changes in utility mercury deposition in all estuaries across the US at this time because of the heterogeneity of estuarine systems and that it would be more appropriate to present a qualitative assessment.

Fisheries Service (NMFS, 2002). For this analysis, we define estuarine species as those caught within 0-3 miles of the U.S. coastline. Total commercial fish production in 2001 was 4.4 million tonnes, with 3.9 million tonnes as finfish and 0.5 million tonnes as shellfish. Of the total commercial catch, 63% of the shellfish and 34% of the finfish in 2001 were caught within 0-3 miles from the shore. This estimate includes some species, such as menhaden, that are also used for animal feed or other purposes. As a sensitivity analysis we subtracted out menhaden, resulting in a somewhat lower percentage of finfish (excluding menhaden) coming from the near-shore area.

Using the product of mean mercury concentrations and percent of the commercial market for each species from Carrington et al. (2004), we are able to rank the Hg exposure from the top 25 species in the US commercial seafood market (Table 4.1). We then multiply each finfish or shellfish species by the overall fraction caught in coastal waters to provide a conservative estimate of the relative mercury exposure from coastal species. As illustrated in Table 4.1, the fraction of mercury exposure from fish caught in near-shore waters in the U.S. commercial seafood market is roughly 35 percent of the total. However, the utility-attributable fraction of mercury exposure from fish and shellfish will be much smaller as outlined below.

This estimate of total exposure from estuarine species is thought to be conservative (high) because it is based on total mercury concentrations in fish and shellfish rather than methylmercury concentrations, the mercury species in fish that is toxicologically most significant. It is fairly well established that shellfish in general will have a lower fraction of total mercury present as methylmercury (%MeHg) in their tissues than predatory fish. For example, Mikac et al. (1985) found that marine mussels had 5-27% of mercury in the organic form, while Mason et al. (2000) found between 50-80% of the mercury in crayfish was present as MeHg. Thus, it appears that there is considerable variation in estimates of the fraction of methylmercury contained within the tissue of invertebrates. This lowers methylmercury exposure typically associated with certain species by assuming measured total mercury concentrations are equivalent to methylmercury concentrations.

Table 4.1. Estimated mercury intake from species harvested within three miles from shore based on the Hg content of the top 25 species in the U.S. Commercial Seafood Market.

Species	% Hg Intake by Species[1]	***% Coastal Estimate*** [2]
Tuna, albacore (canned)	17.9%	6.1%
Tuna, light (canned)	15.9%	5.4%
Haddock, hake, and monkfish	8.7%	3.0%
Pollock	7.1%	2.4%
Tuna, fresh	6.5%	2.2%
Cod	6.5%	2.2%
Swordfish	3.9%	1.3%
Lobsters, American	3.8%	2.4%

Catfish	3.0%	1.0%
Crabs	2.8%	1.8%
Lingcod and scorpionfish	2.5%	0.9%
Salmon	2.2%	0.8%
Flatfish	2.0%	0.7%
Halibut	1.9%	0.6%
Shrimp	1.7%	1.1%
Bass, saltwater	1.5%	0.5%
Anchovies, herring, and shad	1.5%	0.5%
Shark	1.2%	0.4%
Orange Rougy	1.0%	0.4%
Lobsters, spiny	1.0%	0.6%
Grouper	0.9%	0.3%
Snapper, porgy, and sheepshead	0.7%	0.3%
Squid	0.7%	0.2%
Sablefish	0.7%	0.2%
Skate	0.5%	0.2%
Percent Total	**96.2%**	**35.4%**

[1] Ranking for %Hg Intake column is based on %Market and average Hg content for each species in Carrington et al., 2004.

[2] Percent coastal intake was estimated using 2001 landings data averages of 34% of all finfish harvested from 0-3 miles from shore (near-shore areas) and 63% of all shellfish (NMFS 2002).

4.3 Utility Attributable Mercury Deposition in Near-shore Areas

The significance of utility-attributable mercury for estuarine species is expected to be a small component of their overall body burden because of the prevalence of legacy contaminants, watershed based mercury sources and other atmospheric mercury sources in these regions. When analyzing mercury exposure in most coastal regions, it is apparent that the utility-attributable portion of this exposure will be limited. For example, the Pacific Coast accounts for over 65 percent of the productions of fish species listed in table 4-1 (NMFS 2002) and will be negligibly impacted by utility-attributable mercury deposition. This is reinforced by EPA's deposition modeling, which indicates that coal-fired utilities will not significantly impact Hg levels on the Pacific Coast. As a bounding analysis, if we recalculate the potential fraction of total mercury exposure that can then be affected by changes in utility deposition from Table 4.1, we are left with 35 percent of the original 35 percent of total exposure from the U.S. commercial seafood market. In other words, only slightly more than 12 percent of the total mercury exposure from the consumption of marine fish can be from the consumption of coastal and estuarine fish from the Gulf coast, southeast Atlantic and New England. Only a small fraction of that 12 percent can be attributed to utility-attributable mercury as discussed below because utility deposition comprises only a small fraction of the mercury that is present and available for methylation in the Gulf coast, southeast Atlantic and New England.

EPA's modeling of deposition due to mercury emissions from Utilities in 2001 suggests that Utilities represent between 0 and 10 percent of the total deposition in the Gulf Coast, 5 to 20 percent in the Southeast Atlantic, 0 to 10 percent in the New England coastline, and 20 to 30 percent in the Chesapeake Bay area. In 2020 after CAIR (and furthermore after CAMR) the deposition due to mercury emission from utilities falls to a range between 1 percent and 12 percent for the Gulf and Southeast Atlantic coasts. Most Gulf and Southeast Atlantic Coast areas will experience approximately 2 percent of total deposition from utilities. Most areas of the New England coastline will experience slightly over 2 percent of total deposition from utilities, with a maximum utility-attributable deposition of 7 percent. After CAIR in 2020, utility attributable deposition will be highest close to shore off the Northeastern States but will still be less than 7 percent, compared to up to 19 percent in the highest freshwater fishing areas (see Table 2.6 of the Effectiveness TSD).

The Chesapeake Bay represents 6.5 percent of total U.S. landings (NMFS 2002) and is likely one of estuarine ecosystems most sensitive to atmospherically deposited mercury from US power plants because of the significance of coal fired power plants (20-30 percent of total in 2001) to overall atmospheric mercury deposition rates and its relatively small watershed to water surface area ratio (resulting in a greater importance of the atmospheric pathway compared to many other estuary ecosystems). Utility attributable deposition after CAIR is expected to be in the range of 8.5 percent (and will be reduced even further following CAMR) of the total deposition. The percent of total deposition attributable to utilities for the Chesapeake Bay watershed is somewhat less that the 8.5 percent for the Chesapeake Bay itself. EPA's analysis of projected mercury deposition rates after implementation of CAIR and CAMR show some of the largest reductions in mercury deposition are expected to occur in the Chesapeake Bay region.

4.4 Conclusions Regarding the Near-Shore Pathway

As discussed above, populated coastal regions like the Chesapeake Bay and Baltimore Harbor (Mason and Lawrence, 1999) will receive significant land-based mercury inputs from wastewater effluents, municipal waste discharges and historical mercury contamination that are slowly leaching from the watershed. In addition, legacy mercury stored in the active sediment layers of these systems will continue to supply coastal systems with inorganic mercury for many decades. These types of inputs to coastal ecosystems lower the overall significance of small changes in atmospheric deposition to the overall magnitude of mercury in coastal systems. These estimates of utility attributable deposition following CAIR, combined with the magnitude of commercial fish landings data from the Eastern U.S. coastline further reinforces that exposure from this source is small.

Although we are not currently able to quantitatively estimate IDI values associated with various levels of consumption of estuarine fish, the available information suggests that they will be bounded by the freshwater recreational/subsistence IDI values presented in the Effectiveness TSD (US EPA 2005a). We therefore continue to use the IDI values in Table 6.4 of the Effectiveness TSD as our estimate of the maximum individual risk. In

addition, we will use the utility attributable MeHg in the 85th percentile freshwater fish as a bound of the utility attributable MeHg in estuarine fish.

5. Aquaculture Exposure Pathway

Due to the unique nature of the aquaculture pathway and gaps in the available data, it is not possible to conduct a quantitative assessment of the utility-attributable exposure from mercury in farm-raised fish. However, based on the available information we are able to conclude that the contribution of aquaculture to utility-attributable mercury exposure is small. By breaking this potential exposure pathway into three main components that are discussed below, we outline our rationale for determining that utility-attributable exposure due to consumption of aquaculture is small.

First, we find that farm-raised fish accounts for only 10 percent of total commercial fish production in the U.S (NMFS 2002), which limits the relative importance of consumption of aquaculture species harvested in the US that could potentially be affected by U.S. power plant mercury deposition compared to other fish sources. Second, the main aquaculture species are salmon and catfish, which are generally low in mercury. Third, the fraction of the mercury attributable to power plants in farm-raised fish is likely much smaller than other wild fish because their diet is specifically engineered from a number of different protein sources that may not necessarily originate in the U.S. and are therefore not appreciably affected by utility-attributable mercury. Further, the fish meal used to make fish feed is usually derived from smaller fish, which are generally lower in mercury than are larger fish. Because the mercury residue in fish tissues is mainly the result of dietary biomagnification, uptake of mercury from the water column (which may potentially be affected by power plant emissions) by farm-raised is expected to be small. Given this information, it is reasonable to conclude *utility-attributable mercury* from this particular pathway will not add significantly to the overall population body burden.

5.1 Quantity of Fish Produced in Marine and Freshwater Aquaculture

In 2001, total aquaculture production in the U.S. was 371,470 metric tons, compared to total commercial finfish that were domestically caught in the same year of 3,738,769 metric tons (NMFS 2002).7 Because only U.S. aquaculture is relevant from the standpoint of assessing utility-attributable mercury exposure, these numbers indicate that U.S. aquaculture is at most 10 percent of the other fish landings within the country. In short, potential utility-attributable mercury exposure from U.S. aquaculture is small relative to other types of fisheries.

5.2 Mercury in Aquaculture Fish from Direct Deposition versus Fish Feed

It is well known that the major pathway of mercury accumulation in fish is through the diet rather than uptake from water (e.g., see review by Rodgers 1994). The

7 This estimate includes some species, such as menhaden, that are also used for animal feed or other purposes.

process whereby contaminants accumulate in organisms to many times the concentrations found in the ambient environment as larger organisms eat smaller organisms is known as "biomagnification." For example, it is not uncommon to see concentrations of mercury in predatory wild fish that are a million times higher than the methylmercury concentration in water because of dietary biomagnification. Direct uptake of a contaminant from the ambient environment is known as "bioconcentration" and both of these processes together are known as "bioaccumulation."

Because the major pathway of mercury accumulation in fish tissue occurs through biomagnification of methylmercury, concentrations of mercury in the diet of aquaculture species are most relevant for determining their ultimate tissue Hg residue. In order for utility-attributable mercury to be significant in farm-raised fish, a significant fraction of the diet would need to be composed of fish that are affected by utility-attributable mercury. This is not the case as illustrated below.

Two key ingredients of fish feed, for which economic data exist, are fish meal and fish oils. In 2002, approximately 28 percent (148 million pounds) of the U.S. supply of fish meal was imported and the remaining 72 percent (389 million pounds) was domestically produced (NMFS, 2003, p. 83). Data on the U.S. supply of fish oils shows that it is roughly 50 percent imported and 50 percent domestically produced (NMFS, 2003, p. 83). The mercury concentration in domestically produced fish meal and fish oil depends on where the fish were caught. Only 16 percent of U.S. domestic commercial landings occur on the Atlantic coast, versus 83 percent on the Gulf and Pacific coasts (NMFS, 2003, p. 6). Of U.S. commercial landings, only 36 percent occur within 0 to 3 miles from U.S. shores, while 61 percent occur between 3 and 200 miles of U.S. shores (NMFS, 2003, p. 19). Because Gulf coast, U.S. Pacific coast, internationally landed fish, and Atlantic coast fish caught greater than 3 miles off shore are expected to see a very small decrease in exposure to U.S. utility-attributable Hg emissions, only a small fraction of the U.S. supply of fish meal and fish oil is expected to see a significant change in Hg exposure. Therefore, while the dynamics of Hg contained in imported fish meal and fish oil is largely unknown, the mercury contained in this fish is likely from the global pool, of which US power plants represent about 1 percent.

In contrast to dietary mercury accumulation from fish oil and meal, much smaller contributions are expected from ambient Hg concentration and wild food sources and ingredients in fish food other than fish oil and fish meal. Any geographic analysis of deposition with regard to fish farm location would assume that reductions in atmospheric deposition would directly impact farm-raised fish Hg loads through bioconcentration, which is not the case as described in detail above. Regional changes in Hg emissions and subsequent atmospheric deposition resulting from the Revision Rule are not expected to have a direct impact on the content of Hg in farm-raised fish.

In summary, EPA finds that it is the location of the fish caught to make fish feed that is relevant, as opposed to the location of aquaculture farms.

5.3. Summary of the Aquaculture Exposure Pathway

In summary, the limited available data indicate that Hg body burdens in aquaculture fish are less than or equal to their wild counterparts, and mercury in aquaculture fish is predominantly the result of the mercury content of fish feed. The fish feed used in US aquaculture comes from a variety of sources including the Gulf Coast, the Atlantic Ocean, US Pacific Coast, and internationally landed fish – fish that have small utility-attributable mercury levels. Therefore, while there are insufficient data available to conduct a full quantitative assessment, EPA believes that the existing information support the conclusion that that utility-attributable mercury exposure from aquaculture is small and that all individuals or groups of individuals who consume aquaculture fish are likely to ingest utility-attributable methylmercury at levels that are reflective of the levels ingested from the types of marine fish that comprise aquaculture fish feed.

6. Commercial Freshwater Exposure Pathway

EPA's claim that freshwater commercial fish are not a significant pathway is valid since 17 million pounds/year (lb/yr) is small when compared to recreational freshwater fish consumption of 377 million lb/yr, or 22 times the Great Lakes commercial haul.[89] Further even though utility attributable deposition is comparatively higher around the Great Lakes and the bordering areas (including the states of Michigan, Indiana, Illinois, and Ohio and other surrounding areas) in comparison with the rest of the United States, it is still only a small percentage of mercury deposition from all sources. The typical percent of total deposition that is attributable to utilities in these areas is approximately 10 percent. Thus, following the assumptions in Mercury Maps, only approximately 10 percent of the mercury in the fish found in this area is attributable to utilities. In addition, the areas in the Great Lakes that are affected do not experience a disproportionately high deposition rate compared to the surrounding land area, where recreational freshwater fish are caught, so the commercial freshwater pathway is still expected to be small relative to the recreational/subsistence freshwater pathway.

Because exposure is determined at the population level, the determination for including this pathway is whether adding it (population exposed times exposure rate) will significantly alter the cumulative distribution of total exposure rates. As described above, the commercial freshwater harvest is small compared to recreational freshwater consumption, the percent of utility-attributable deposition in the primary commercial freshwater harvesting area is 10 percent, and those levels are not disproportionate to the areas for recreational freshwater harvest. These facts lead the EPA to conclude that including the commercial freshwater pathway in the exposure model would result in a

8 Recreational freshwater fish consumption was calculated by multiplying the population of fishers (27,900,000 (US FWS 2002)) by both the percent of recreational fishers who consume their catch (0.84 (average of values presented in West at al. 1989, Chemrisk 1991, and West et al 1993 as presented in EPA 1997b)), and the number of friends and family with whom the average recreational fisher shares his or her catch (2.5 (EPA 1997b)) and converting to pounds.

9 The Great Lakes commercial haul is 0.2% of the total commercial haul of finfish (8.2 billion pounds) (NMFS 2002). The marine haul represents the most significant fraction of the total haul and is discussed elsewhere.

relatively small change in the general population level exposure estimate. In addition, there is no reason to include this pathway to address its effect on the higher end groups (e.g. subsistence fishers) because most of the fish they eat is self-caught and it is highly unlikely, given the nature of their fishing activity, that more than a small fraction, if any, of their consumption is comprised of commercially caught freshwater fish. As such, we believe that the IDI values for this pathway are bounded by the freshwater recreational/subsistence IDI values.

7. Joint Consumption

In order to examine utility-attributable Hg exposure from total fish consumption quantitatively, it would be necessary to have information on the distribution of consumption of each type of fish – recreational freshwater, commercial freshwater, recreational saltwater, etc - as well as utility-attributable MeHg concentrations (either sufficiently accurate or upper-bound) for each type of fish. If we were able to identify the consumption of each type of fish as well utility-attributable MeHg concentration for each type of fish, then the IDI values from each type of fish could be calculated and added together to arrive at a total IDI value. Currently no such data exists. Regardless, for the reasons described above, EPA maintains that self-caught freshwater fish consumption represents the most significant exposure pathway for the populations with the highest utility-attributable exposure.

At any given total fish consumption rate noted in our analyses, introducing aquaculture, marine, or estuarine fish into the diet of a self-caught freshwater fish consumer necessarily implies reducing consumption of self-caught freshwater fish (e.g., in order to maintain the same total fish consumption rate). As discussed in previous sections, because utilities contribute more Hg to freshwater fish species than to any other fish species, such substitution implies a lower IDI than is associated with consumption of self-caught freshwater fish alone, supporting the assertion that self caught freshwater consumption represents the primary source of utility-attributable Hg exposure. As can be seen in the seventh column of Table 6-4 of the Effectiveness TSD and Table 3.2 of this document, for any given consumption rate, the estimated IDI for self caught freshwater fish consumption is higher than the estimated IDI for marine fish consumption (for fish tissue methylmercury percentiles 50 percent or greater). Hence, for any given consumption rate, consumption of self-caught freshwater fish alone leads to a higher IDI than that of any other combination of fish, supporting our decision to focus our analysis on consumption of self-caught freshwater fish.

Table 6.4 of the Effectiveness TSD (US EPA 2005a) shows an array of consumption values combined with percentiles of methylmercury concentration in freshwater fish. Results for 2020 with CAIR indicate that estimated IDIs are all well below 1 for the first three consumption rates. Estimated IDIs are over one for 99th percentile recreational fishers and mean subsistence Native Americans only when all of the fish consumed has MeHg concentrations at the 99th percent level, a convergence of

factors which is unlikely to occur.[10] See 70 FR 16024. While estimated IDIs for the 95th (170 g/day) and 99th percentile (295 (g/day) subsistence Native American consumers are above one for lower percentile MeHg concentration fish, it is unlikely that these consumers would add significant amounts of non-self-caught freshwater fish to their diets over the course of a year, but rather would substitute fish, again supporting our focusing on the consumption of self-caught freshwater fish. Finally, the IDI values for the combinations of fish consumption rates and MeHg concentrations bordering the combinations with IDIs above one are sufficiently below one that it is unlikely that a consumer in these combinations would add a sufficient amount of other fish (with lower utility-attributable MeHg concentrations than freshwater fish) to their freshwater fish diet to cause their IDI to exceed one.

Further, we have no evidence that high end consumers of self caught fish also consume other types of fish. It is highly unlikely that subsistence individuals eat 170 g/day or 295 g/day of self caught freshwater fish *and* consume significant quantities of marine fish. Even if we were to assume that these consumers do eat additional fish, the additional MeHg ingested by these consumers is small as we have shown above.

8. Health Benefits and Costs

8.1 Introduction

Below we describe a bounding analysis that includes the exposure pathways described in this TSD and self-caught freshwater fish. In this calculation, we use mean or central tendency estimates of variables when available. However, for several variables we are not able to provide such an estimate and we therefore use a conservative estimate that would overestimate the utility-attributable mercury exposure. The final calculation therefore represents a combination of central tendency estimates for some variables and conservative estimates for other variables. This analysis is a bounding analysis in the sense that the final health benefit estimate presented below is very likely to be above the true health benefits of improved neurological performance associated with reducing mercury emissions from power plants because of the compounding use of conservative estimates for certain variables.[11] The bounding analysis approach supports our reasonable belief that the costs of reducing mercury emissions beyond CAIR under section 112 from power plants outweigh the health benefits of reduced utility-attributable mercury exposure.

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10 In addition to the particular combinations shown in the table, there are a multitude of other combinations of fish consumption rates and methylmercury concentrations possible. For example, Table 6.4 shows that the 99th percentile recreational fisher consuming 47 grams per day of fish with the 99th percentile of utility attributable mercury concentration would have an IDI value of 1.12. An individual consuming slightly less than 47 grams per day of fish with the 99th percentile of utility attributable mercury concentration would also have an IDI value greater than 1. Similarly, an individual consuming 47 grams per day of fish with slightly less than the 99th percentile of utility attributable mercury concentration would also have an IDI value greater than 1.

11 Note that the assumptions in this bounding analysis are different from those in the marine cycling section and this section should not be considered as an extension of that analysis.

8.2 Outline

The benefit calculation will follow directly from the IDI values presented in Table 6-4 of the Effectiveness TSD (US EPA 2005a) and Table 3.2 of this TSD. These IDI values represent an estimate of exposure due to power plants and, equivalently, the reduction in exposure that would occur if power plant mercury emissions were eliminated. Using a dose-response relationship, we translate these IDI values into neurological improvements, using intelligence quotient (IQ) points as a surrogate. We then estimate the monetized value of these IQ point increments and discount these future monetized benefits to account for the ecosystem response time. These discounted benefits can then be compared with the discounted costs, taking into account the important uncertainties described elsewhere.

8.3 IDI values

We use the IDI values from Table 6-4 of the Effectiveness TSD and this TSD. For the purposes of estimating total benefits of reducing mercury emissions from power plants, we use the mean fish consumption rate for each pathway. The mean consumption rate is 8 g/day freshwater fish and is 14.1 g/day for the marine pathway (US EPA 1997b).

The IDI values can be converted to ppm of mercury in hair by first multiplying it times 5.4, which is a conversion factor between the mercury in blood (in ppb) and exposure of someone exposed to mercury at the RfD. In other words, an exposure of 0.1 µg/kg/day of MeHg (i.e., the RfD) is associated with 5.4 ppb in blood (see the one-compartment model for MeHg in IRIS (US EPA 2002a) for more detail[12]), and then dividing by the 4, which is the conversion ratio between blood mercury and hair mercury (250 ppb blood mercury = 1 ppm hair mercury).[13]

8.4 IQ decrements

EPA has chosen to focus on quantification of intelligence quotient (IQ) decrements associated with prenatal mercury exposure as the initial endpoint for

12 The one compartment model converts the concentration of MeHg in blood (in µg/L) to daily dietary intake (in µg/kg/day) using the equation d = (c*b*V)/(A*f*bw), where c is the blood concentration (µg/L), b is the elimination constant (days^{-1}), V is the volume of blood (L), A is the absorption factor (unitless), f is the fraction of absorbed dose taken up by blood (unitless), and bw is the body weight (Kg). Using the recommend values in IRIS (b=0.014, V=5, A=0.95, f=0,059, bw=67), substituting the RfD value of 0.1 µg/kg/day for d and solving for c, we arrive at a conversion factor of 5.4. Note that others have equated the RfD to a maternal blood equivalent of 5.8 ppb. This estimate does not take into account the fact that the RfD was rounded to one significant digit and was not based on a single measure for the RfD critical endpoint. (see Table 2 at http://www.epa.gov/iris/subst/0073.htm) Rather, EPA based this RfD for this assessment on several scores from the Faroes measures, with supporting analyses from the New Zealand study, and the integrative analysis of all three studies. We therefore take the RfD of 0.1 ug/kg/day as the starting point of this calculation and then apply the one compartment model.

13 http://www.epa.gov/iris/subst/0073.htm

quantification and valuation of mercury health benefits.[14] The IQ dose-response analysis uses data from three major prospective studies investigating potential neurotoxicity of low-level, chronic mercury exposure. Epidemiological studies of prenatal mercury exposure conducted in the Faroe Islands (Grandjean et al. 1997), New Zealand (Kjellstrom et al. 1989, Crump et al. 1998), and the Seychelles Islands (Davidson et al. 1998, Myers et al. 2003) have examined neurodevelopmental outcomes through the administration of tests of cognitive functioning.

A statistical analysis was conducted to integrate data from the three studies to produce a single estimate of the IQ dose-response relationship. Details of the analysis, including statistical model formulation, selection of input values, results and sensitivity analysis are reported in Ryan (2005). For the analysis in this section, EPA is using a linear model that goes through the origin to fit population-level dose-response relationships to the pooled data from the three studies. The application of a linear model should not be interpreted to suggest that any of the three studies used have data showing health effects from methylmercury exposure at or below the RfD.[15] It is also important to note that the use of a linear model applied to all exposed individuals is done for purposes of developing an upper bound estimate of the IQ detrimental effect of MeHg. In effect, it assumes that all exposed individuals are exposed above the RfD.16 This assumption

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14 There is limited evidence directly linking IQ and methylmercury exposure in the three large epidemiological studies that were evaluated by the NAS and EPA. Based on its evaluation of the three studies, EPA believes that children who are prenatally exposed to low concentrations of methylmercury may be at increased risk of poor performance on neurobehavioral tests, such as those measuring attention, fine motor function, language skills, visual-spatial abilities (like drawing), and verbal memory. For this analysis, EPA is adopting IQ as a surrogate for the neurobehavioral endpoints that NAS and EPA relied upon for the RfD.

In the Faroes Island Study, a full scale IQ evaluation was not conducted. However, two core subtests were evaluated (Similarities and Block design) and one supplementary test was conducted (Digit Span). The Similarities and Block Design tests are reported to be well correlated with the full WISC-R battery (0.885, see Bellinger (2005)), but how the Digit Span test relates is not reported. In the EPA analysis, we assume that it relates similarly. In the Faroes study, performance scores on the Similarities and Block Design tests were not shown to be statistically related to cord blood or maternal mercury levels; the Digit Span test did show a statistical relationship with cord blood mercury.

Both the New Zealand and Seychelles study administered the WISC IQ test (WISC III in Seychelles, WISC R in New Zealand). A reanalysis of the New Zealand data found a positive association, but it was not statistically significant. No significant associations were seen in the Seychelles study. As displayed in Figure 5 of Ryan (2005), the confidence intervals for full scale IQ in both these studies include zero. However, Ryan conducted an integrative analysis, combining results from all three studies. When combined, the statistical power of the analysis increases. While the size of the dose-response relationship declined relative to past studies with a statistically significant finding, Ryan found a statistically significant relationship between IQ and mercury. The confidence interval did not include zero.

15 The RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime (EPA 2002). EPA believes that exposures at or below the RfD are unlikely to be associated with an appreciable risk of deleterious effects. It is important to note, however, that the RfD does not define an exposure level corresponding to zero risk; mercury exposure near or below the RfD could pose a very low level of risk which EPA deems to be non-appreciable. It is also important to note that the RfD does not define a bright line, above which individuals are at risk of adverse effect.

16 From recent data (MMWR November 5, 2004 / 53(43);1018-1020, http://www.cdc.gov/mmwr/preview/mmwrhtml/mm5343a5.htm), we know that 5.7% of women of

produces an upper bound, but is not consistent with the fact that most of the U.S. population is below the RfD and at these lower levels of exposure EPA believes that there is not an appreciable risk of deleterious effects occurring.

The model makes use of dose-response coefficients for IQ. These coefficients express a central estimate of the average reduction in children's scores in tests of IQ (or other tests of cognitive performance) for a one unit change in the mercury body burden of the mother during pregnancy. The model also incorporates coefficients for other cognitive tests conducted in the studies, in an effort to obtain more robust estimates of the IQ relationship that account for within-study (endpoint-to-endpoint) variability as well as variability across studies. A Bayesian hierarchical statistical model was used to estimate the integrated dose-response coefficient. This is similar to the approach used by the NRC panel to calculate a benchmark dose value integrating data from all three studies (NRC 2000). A more technical description of these same methods has been provided by Coull et al. (2003).

The statistical analysis produced a dose-response relationship, integrating data from all three studies, with a central estimate of an IQ change of -0.13 IQ points (95% confidence interval -0.28, -0.03) for every ppm of mercury in maternal hair.

An IDI value can be converted to an IQ decrement by first converting it to a blood equivalent mercury level (in ppb) and then converting it to a hair mercury level (in ppm), as described above. The result can then be multiplied by the estimated dose-response coefficient of 0.13 to produce an IQ decrement. In other words:

$$IQ\ Decrement = \left\{ \frac{(IDI) * (\text{Blood equivalent of RfD}) * (\text{IQ decrement per 1 ppm hair})}{\text{Conversion between blood (ppb) and hair (ppm)}} \right\}$$
$$= \left\{ \frac{(IDI) * (5.4) * (0.13)}{4} \right\} = (IDI) * 0.1755$$

For example, an individual born to a mother exposed above the RfD due to non-US power plant sources and with an IDI value of 1.0 is estimated to experience a 0.1755 decrement in his or her IQ due to the utility-attributable exposure according to the equation above.

Applying this equation to all exposed individuals, in effect, assumes that all individuals are above the RfD due to non-US power plant sources and the utility-attributable exposure is in addition this level. As mentioned above, this will produce larger estimate of the IQ decrements attributable to power plants than if each individual's exposure (above or below the RfD) were known and accounted for. To produce an upper bound estimate for this analysis, we falsely, but conservatively, assume that all individuals are above the RfD from non-U.S. power plant sources.

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childbearing age have blood Hg levels above the RfD-equivalent level used by the CDC. Thus the assumption that all exposed individuals are above the RfD is extremely conservative.

8.5 Monetized value of IQ decrements

The valuation approach for assessing losses associated with IQ decrements is based on an approach used by EPA to assess benefits for reductions in lead exposures (EPA, 2000a). For that analysis, EPA used results from a study by Salkever (1995) to estimate the effects of IQ loss on expected future earnings and years of education. Salkever analyzes data from the National Longitudinal Study of Youth (NLSY) and uses a three equation regression model to estimate the relationships between IQ levels, educational attainment, and expected future earnings. The results of this study indicate that the average effect (for men and women combined) of a one point decrease in IQ is:

1. A 2.379 percent decrease in future earnings; and
2. A 0.1007 decrease in years of schooling.

To estimate the expected monetary value these effects, EPA first estimated the average present value of future earnings at the time of birth for a person born in the U.S. Using earnings data from the 1992 Current Population Survey (CPS) and discounting at a 3 percent annual rate, this present value was estimated to be $366,021 in 1992 dollars. EPA then estimated the average direct and indirect costs associated with one additional year of schooling. Based on Department of Education data, the average annual expenditure per student was estimated to be $5,500, and the average annual opportunity cost (lost income from being in school) was estimated to be $10,925. Assuming that these costs were incurred at age 19 (based on an average of 12.9 years of education among those over age 25 in the U.S.) the combined present value of these two costs at time of birth (discounted at 3 percent) were estimated to be $9,367 per additional year of schooling in 1992 dollars.

Combining these estimates with the results from the Salkever (1995) study summarized above implies that the average present value of net earnings losses associated with a one point decrease in IQ is $7,765 in 1992 dollars. This value is calculated as the average present value of lost earnings per IQ point loss ($8,708 = $366,021 * 0.2379) minus the partially offsetting change in average education costs per IQ point loss ($943 = $9,367 * 0.1007). Corrected for inflation using the GDP deflator, the average present value of net earnings losses per IQ point loss is $8,807 in 1999 dollars. The value per IQ estimate using a 7 percent discount rate is $1,580 per IQ point.[17] In keeping with our desire to produce an upper-bound estimate and given the uncertainty in the appropriate value per IQ point, we use the estimate of $8,807.

8.6 Undiscounted Benefits

17 EPA acknowledges that lost earnings from IQ loss is not the conceptually correct metric for valuing benefits of reduced mercury exposure besides the fact that IQ is being used as a surrogate for other subtle neurobehavioral endpoints. Ideally, we should use a measure of willingness-to-pay (WTP) to avoid these endpoints caused by mercury exposure. However, there is a lack of informative research on which to form an estimate of WTP.

Multiplying this value times the IQ decrement for any given IDI value produces the estimated undiscounted economic value for that IDI. In other words, this is the economic value that the individual would gain if he or she were no longer exposed to any utility attributable mercury, which is why it is listed as a benefit. This benefit value can also be viewed as the economic loss associated with utility-attributable, post-CAIR mercury exposure.

8.7 Total Benefits

Since there is a lag between the decrease in deposition and the decrease in methylmercury exposure, this economic value must be discounted over some number of years to arrive at the economic value of eliminating utility-attributable mercury deposition. For freshwater systems, this lag is between 10 and 50 years. As a conservative estimate, we can assume a 15 year lag and a 3% discount rate. Mathematically, this means that we should divide our undiscounted value by approximately 1.6 (= 1.03^15) to arrive at the present value of *eliminating* utility-attributable mercury emissions[18]. In other words:

$$Monetary\ Value = \left(\frac{1}{1+Discount\ Rate}\right)^{Discount\ Period} * \left(\begin{matrix}Monetary\ Value\\ of\ \ IQ\ Decrement\end{matrix}\right) * (IQ\ Decrement)$$

$$= \left(\frac{1}{1.03}\right)^{15} * (\$8{,}807) * (IQ\ Decrement) = \$5{,}552 * (IQ\ Decrement)$$

For example, an individual born to a mother exposed to methylmercury at an IDI of 1 would suffer an economic loss of, roughly, $1,000, assuming non-power plant exposures are already above the RfD. Table 8.1 below lists the economic loss associated with individuals exposed to various levels of utility-attributable mercury and at various consumption levels.

Table 8.1: Economic Loss from IQ Decrements associated with Mercury Exposure due to Freshwater Fish Consumption in 2020 after CAIR

2020 with CAIR *Discounted Benefits (in dollars)*	**MeHg** **%** **ppm** **g/day**	**5th** **0**	**10th** **0.001**	**15th** **0.002**	**25th** **0.004**	**50th** **0.01**	**75th** **0.02**	**85th** **0.027**	**90th** **0.035**	**95th** **0.052**	**99th** **0.102**
EPA EFH Mean Recreational Fisher	**8**	$ -	$ 2	$ 4	$ 7	$ 19	$ 37	$ 50	$ 65	$ 97	$ 190
EPA OW 90th Percentile General Population	**17.5**	$ -	$ 4	$ 8	$ 16	$ 41	$ 81	$ 110	$ 142	$ 212	$ 415
EPA EFH 95th Percentile Recreational Fisher	**25**	$ -	$ 6	$ 12	$ 23	$ 58	$ 116	$ 157	$ 203	$ 302	$ 593
EPA EFH 99th Percentile Recreational Fisher	**47**	$ -	$ 11	$ 22	$ 44	$ 109	$ 219	$ 295	$ 382	$ 568	$ 1,115
EPA EFH Mean Subsistence Native American	**60**	$ -	$ 14	$ 28	$ 56	$ 140	$ 279	$ 377	$ 488	$ 725	$ 1,423
EPA EFH 95th Percentile Subsistence Native American	**170**	$ -	$ 40	$ 79	$ 158	$ 395	$ 791	$ 1,067	$ 1,383	$ 2,055	$ 4,032
EPA EFH 99th Percentile Subsistence Native American	**295**	$ -	$ 69	$ 137	$ 274	$ 686	$ 1,372	$ 1,852	$ 2,401	$ 3,567	$ 6,996

At a consumption rate of 8 grams per day, the mean rate for a freshwater fisherman, the economic loss from IQ decrements associated with utility-attributable,

18 Using a 3 percent discount rate produces a higher benefits estimate, which is desirable to produce an upper bound estimate. Conducting an additional analysis using a 7 percent discount rate, which would be standard in a regulatory economic analysis, would require dividing by 2.8.

post-CAIR mercury exposure from freshwater fish consumption ranges between zero and $190, depending on the amount of deposition. For example, in a watershed with the 85th percentile utility-attributable mercury deposition, self-caught freshwater fish are assumed to have a utility-attributable methylmercury concentration of 0.027 ppm. This translates to a discounted economic loss of approximately $50 per birth.

A similar analysis can be done using the IDI values for marine fish consumption and estuarine and coastal fish consumption. For marine fish consumption, we use the IDI values reported earlier in this TSD. Since the marine environment produces a longer time lag between deposition reductions and exposure reductions, the discount factor for this calculation will be slightly different. We use a 3 percent discount rate over 30 years, which is the shorter of the two lag times described in Section 2.3, the Marine Cycling section, of this TSD. The results of this calculation are reported in Table 8.2.

Table 8.2: Economic Loss from IQ Decrements associated with Mercury Exposure due to Marine Fish Consumption in 2020 after CAIR

2020 with CAIR ***Discounted Benefits (in dollars)***	**MeHg** **g/day**	**ppm**	**Mean 0.1**
Mean Marine Fish Intake	14.1		$ 2
95% Long-Term Marine Fish Intake	44		$ 7
Mean Total Fish Intake	20		$ 3
95% Long-Term Total Fish Intake	63		$ 9
Median Carrington & Bolger Seafood Consumption	6.8		$ 1
90% Carrington & Bolger Seafood Consumption	42		$ 6
95% Carrington & Bolger Seafood Consumption	63		$ 9
99% Carrington & Bolger Seafood Consumption	123		$ 18
99.5% Carrington & Bolger Seafood Consumption	141		$ 21
99.9% Carrington & Bolger Seafood Consumption	207		$ 31

Assuming a maternal fish consumption rate of 14.1 grams per day of marine fish, which is the mean marine fish intake rate, an individual prenatally exposed to utility-attributable methylmercury in 2020 would suffer a lifetime economic loss of around $2 from IQ decrements.

8.8 Aggregate Benefits

As described above, the benefits of *eliminating* mercury emission from U.S. power plants in 2020, after the implementation of CAIR, can be estimated by summing the economic loss associated with their damage. This is done by multiplying the economic loss value times the numbers of births to mothers in each consumption range. If we assume that consumption is log-normally distributed, then we can do this by multiplying the economic loss for the mean consumption rate times the total number of births.

According to the U.S. Census, the U.S. population in 2001 was roughly 280 million people (BOC 2005), and there were approximately 4 million births according to the CDC's National Center for Health Statistics (CDC 2002). However, because the consumption self-caught fish represents the highest level of utility-attributable exposure, it is important to make a distinction between births to self-caught freshwater fishermen and those to the general public. Assuming that the ratio of births to individuals for the general population holds for the 58 million self-caught freshwater fishers described in CAMR, this implies approximately 830,000 births to self-caught freshwater fishers and approximately 3.2 million births to the rest of the general public.

The consumption rate for these two groups can be obtained from the U.S. EPA's Exposure Factors Handbook (US EPA 1997b), which recommends using a mean consumption rate for the general population of 20.1 grams of fish per day, with 14.1 grams associated with marine fish and 6 grams per day of freshwater or estuarine fish (including aquaculture consumption). These are the consumption rates we assume for the general public. The recommended mean consumption value for freshwater anglers is 8

grams per day of self-caught freshwater fish and we will assume a value of 14.1 grams per day from the consumption of marine fish. Multiplying the number of births for each group times the economic loss value produces an estimate of the economic benefits of *eliminating* the post-CAIR mercury emission from U.S. power plants.

The economic loss for an individual consuming self-caught freshwater fish is given in Table 8.1. It would be reasonable to conduct a population level analysis using the average MeHg exposure at the average consumption level from Table 8, which would imply a loss value of $19. However, to maintain a clear upper bound estimate, we use the economic loss value $50 for the mean consumption value for the watershed with the 85th percentile of mercury deposition.[19] Multiplying $50 times the 830,000 births to freshwater fishers implies an upper bound aggregate economic loss of $41.5 million for the consumption of self-caught freshwater fish.

The upper bound estimate of the individual loss value for marine fish consumed by both freshwater fishermen and the general public is $2, as given in Table 8.2. Multiplying $2 times the 4 million births from both groups implies an upper bound aggregate economic loss of $8 million for the consumption of marine fish. $1.6 million of this accrues to freshwater fishers and $6.4 to the rest of the general public.

Table 8.3 summarizes the fact that the upper bound estimate of the aggregate economic benefits of reduced IQ decrements from eliminating utility-attributable mercury exposure in 2020 after CAIR are approximately $50 million plus some additional amount from the consumption of commercial freshwater, estuarine, and aquaculture fish by the general public. The best estimate of the individual economic loss from the consumption of these fish is not known because a best estimate of the IDI values for these pathways has not been estimated. Therefore a best estimate of the aggregate economic loss is unknown. However, we are able to calculate an upper bound estimate as described below.

Table 8.3: Upper Bound Estimate of the Aggregate Economic Benefits of Reduced IQ Decrements from *Eliminating* Utility-Attributable Mercury Exposure in 2020 after the Implementation of CAIR

	Population	**Births**	**Consumption Rate (g/day)**	**Value**	**Benefits**
Freshwater Fishers	58 million	830,000			
Freshwater Fish			8	$50	$41.5 million
Marine Fish			14.1	$2	$1.6 million

19 If fish were consumed equally from all watersheds, it would be appropriate to use the mean utility attributable methylmercury concentration for this calculation. The mean utility attributable methylmercury concentration based on available information is .016 ppm. To allow for the possibility that fish are generally consumed from areas with a higher utility attributable methylmercury concentrations than the mean, we use a utility attributable methylmercury concentration of 0.027 ppm, corresponding to the 85th percentile. It is highly unlikely that consumption of freshwater fish would be so skewed towards waterbodies with such high methylmercury. Therefore this assumption is thought to overestimate the actual utility attributable exposure from freshwater fish consumption.

General Public	222 million	3.2 million			
Freshwater, Estuarine, and Aquaculture Fish			6	**$V**	**B**
Marine Fish			14.1	$2	$6.4 million
				Total =	**$49.5 million + B**

As described in Section 4, EPA finds that the utility attributable exposure to methylmercury from estuarine fish and shellfish will likely be small relative to that from freshwater fish and so the IDI values for this pathway is bound by the freshwater recreational/subsistence IDI values presented in the Effectiveness TSD (US EPA 2005a). Therefore the bound for the IDI value from estuarine consumption for the mean consumption rate of 6 g/day will be 75 percent (6g/8g) of the IDI value for the recreational fisher consuming fish from the 85th percentile watershed at the mean consumption level.

Similarly, as described in Section 5, EPA believes that the utility-attributable mercury exposure from aquaculture is small and exposure to utility-attributable MeHg from aquaculture fish will be lower than the levels ingested from the types of marine fish that comprise aquaculture fish feed. Therefore, the $50 value for 8 g/day of freshwater fish consumption and the $2 value for 14.1 g/day of marine fish consumption can be used to create an upper bound our estimate of **$V** above.

As described above, the U.S. EPA's Exposure Factors Handbook (US EPA 1997b) recommends a consumption rate of 6 g/day of freshwater, estuarine, and aquaculture consumption for the general public. Table 8.4 lists the possible combinations of marine equivalent and freshwater equivalent fish that could be used to make up the 6 g/day.

Table 8.4: Upper Bound Estimates of Economic Loss from IQ Decrements associated with Mercury Exposure due to Freshwater, Estuarine, and Aquaculture Consumption by the General Public in 2020 after CAIR

		Freshwater equivalent consumption (g/day)						
		0	1	2	3	4	5	6
Marine equivalent consumption (g/day)	0							$38
	1						$31	
	2					$25		
	3				$19			
	4			$13				
	5		$7					
	6	$1						

For example if all of the consumption were marine and aquaculture fish, then the economic loss value would be $1. This is because 6 g/day is about 40% of the 14.1 g/day of marine fish consumption with an associated economic loss of $2. 40% of $2 is (rounding up) about $1. In contrast, if all of the fish were estuarine or freshwater fish, then the economic loss would be $38. This is because 6 g/day is 75% of the 8 g/day for freshwater fishers with an associated economic value of $50. So 75% of $50 is $38.

Given this, the highest estimate possible for **$V** in Table 8.4 is $38. Substituting this value in Table 8.3 above would produce a total upper bound estimate of $168 million. This, however, is a dramatic overestimate of the upper bound estimate of benefits. It assumes that the 6 g/day of commercial freshwater, estuarine, and aquaculture fish consumed by the general public is as contaminated as the 85th percentile of self-caught freshwater fish. In other words all aquaculture fish and all estuary fish, including fish from the estuaries on the Pacific coast, are as contaminated as self-caught fish. It is, however, useful to state that the aggregate economic benefits of reduced IQ decrements from *eliminating* utility-attributable mercury exposure in 2020 after the implementation of CAIR can not be higher than $168 million.

8.9 Costs

The methodology for estimating the total annual monetized benefits of CAMR are described in the Cost TSD (US EPA 2005b). Under the base-base assumptions, the total annual monetized cost of the CAMR is estimated to be approximately $750 million in 2020.[20]

8.10 Summary

As can be seen from this analysis, the total monetized costs of CAMR exceed the total monetized benefits presented here of eliminating all utility-attributable mercury emissions remaining in 2020 after the implementation of CAIR. It should again be pointed out that the analysis estimates the upper bound monetized benefits associated with the potential neuro-toxicity (represented by IQ decrements as a surrogate) of low level, chronic mercury exposure. This is the endpoint about which we have the most certainty and which we can monetize (see Section 8.4 above). Furthermore, these estimates were based on a number of assumptions intended to produce an upper bound estimate of the benefits for this endpoint. In contrast, the cost estimate is based on the estimated cost of the cap-and-trade program of CAMR which does not eliminate all mercury emissions from U.S. power plant. Furthermore, it is generally accepted that, all else equal, the cost of cap-and-trade programs are less than other regulatory approaches. By 2020 CAMR reduces between 16 percent and 32 percent of the remaining mercury emissions, depending upon the species, but does not eliminate all mercury emissions from U.S. power plants (See table 3 on page 4 of US EPA 2005c). Given that the monetized costs of reducing 1/3 of the current emissions (by one of the lowest cost emission reduction schemes available) exceeds the upper bound of monetized benefits of reduced IQ decrements, it is reasonable to conclude that the cost of requiring further

20 See table 7-19. The cost associated with monitoring emissions, reporting, and record keeping for affected sources is not included in these annualized cost estimates, but EPA has done a separate analysis and estimated the cost to be about $76 million (see final CAMR preamble Section VI.B. Paperwork Reduction Act). Under the sensitivity analysis described in the Cost TSD, the total annual monetized costs are estimated to be $560 million in 2020 (See Cost TSD). We use the estimate of $750 million in 2020 because it reflects our best estimate, although we note that the conclusions that annual costs exceed annual health benefits would equally apply if we were to use the sensitivity analysis cost estimate of $560 million.

reductions in U.S. power plant Hg emissions beyond CAIR would outweigh the benefits presented here.

9. Evaluation of NESCAUM Report

Recent analyses have attempted to account for exposures from Hg in marine fish, including "Economic Valuation of Human Health Benefits of Controlling Mercury Emissions from U.S. Coal-Fired Power Plants" (docket item OAR-2002-0056-5749) by Glenn Rice and James K. Hammitt, Harvard Center for Risk Analysis (NESCAUM Report) (NESCAUM 2005).

EPA's approach to modeling exposure and health benefits of reducing emissions from power plants differs in some important ways from the approach that NESCAUM chose. EPA believes that some of these differences simply reflect the large amount of uncertainty in the underlying science. Other differences reflect situations where the science and economics are fairly clear and EPA has concerns about the approach that NESCAUM took.

For example, as noted earlier, the NESCAUM report attempted to quantify the marine exposure pathway but used assumptions that are not supported by the literature on marine fate and transport of Hg, likely resulting in an overestimate by an unknown amount. For example, the NESCAUM study assumes that Hg deposition from the atmosphere are tracking Hg concentrations in the surface ocean and that the changes in ocean fish MeHg concentrations will be proportional to changes in total Hg concentrations in the surface ocean. This proportionality assumption was used in Section 2 to provide an upper-bound estimate to show that the marine pathway is probably not a significant contributor to the mercury exposure from U.S. power plants, but we recognized that this was an upper bound estimate. Also, NESCAUM used REMSAD modeling which appears to over-predict Hg deposition from US power plants.

The NESCAUM Report focused on estimating the benefits of reductions in mercury exposure from U.S. power plants and did not attempt to estimate upper-bound estimate of reducing mercury emissions for power plans. However, for the additional reasons noted below EPA believes that NESCAUM's approach should be interpreted as producing an upper-bound estimate of the IQ benefits of reducing Hg emissions from power plants for two reasons. First, it does not appear that the NESCAUM Report took into account the timeframe for reduced exposure to MeHg this issue into account. This omission alone leads to the benefits in the NESCAUM Report being overstated by at least factor of two. Second, EPA commissioned Harvard researchers Dr. Louise Ryan and Dr. David Bellinger to perform an integrated analysis of the three major epidemiological studies (Faroes, Seychelles, New Zealand) and used the resulting relationship between exposure and neurological problems. NESCAUM relied in part on an unpublished study and produces estimated neurological benefits four to five times EPA estimates for this reason alone.

9.1 Timeframe for costs and benefits

It is important to consider the time in an economic analysis because future costs and benefits are discounted. EPA's Revision Rule analyses took into account the timeframe for costs and benefits. As noted in Section 8, benefits were discounted to take into account the response times for the freshwater and marine ecosystems. Case studies of individual ecosystems show that the time necessary for aquatic systems to reach a new steady state after a reduction in Hg deposition rates can be as short as 5 years or as long as 50 years or more. The medium response scenarios also varied widely but were generally on the order of one to three decades. Overall, EPA concludes that the most likely appropriate response times for freshwater ecosystems to be considered in the national scale assessment range between 5 and 30 years, while recognizing that some systems will likely take more than 50 to 100 years to reach steady state. Our preliminary analysis of the temporal response of marine systems based on the model by Mason and Gill (2005) indicates that the Atlantic Ocean will take approximately three decades to reach steady state and the Pacific Ocean will take over two centuries.

At 3 percent and two to three decades, present value benefits would decrease by about half. At 7 percent, present value benefits would fall even more. This shows the importance of taking into account the time lag between emissions reductions and exposure reductions. EPA does not believe that NESCAUM accounted for this important factor in its analysis and in so doing significantly overestimates benefits.

9.2 IQ dose-response relationship

In the benefits analysis presented above, EPA chose to focus on quantification of intelligence quotient (IQ) decrements associated with prenatal Hg exposure as the initial endpoint for quantification and valuation of Hg health benefits. Reasons for this initial focus on IQ include the availability of well-designed epidemiological studies assessing IQ or related cognitive outcomes suitable for IQ estimation, and the availability of well-established methods and data for economic valuation of avoided IQ deficits, as applied in EPA's previous benefits analyses for childhood lead exposure.

There is limited evidence directly linking IQ and MeHg exposure in the three large epidemiological studies that were evaluated by the National Academy of Sciences (NAS) and EPA. Based on its evaluation of the three studies, EPA believes that children who are prenatally exposed to low concentrations of MeHg may be at increased risk of poor performance on neurobehavioral tests, such as those measuring attention, fine motor function, language skills, visual-spatial abilities (like drawing), and verbal memory. For this analysis, EPA is adopting IQ as a surrogate for the neurobehavioral endpoints that NAS and EPA relied upon for the RfD.

The NAS identified three well-designed studies (Faroes, Seychelles, New Zealand) of the neurotoxicological effects of MeHg. "Each of the studies was well designed and carefully conducted, and each examined prenatal MeHg exposures within the range of the general U.S. population exposures" (NRC 2000). In order to develop a

dose-response relationship that reflects all three studies identified by the NAS, EPA commissioned Harvard researchers David Bellinger and Louise Ryan to perform an integrated analysis, combining results from all three studies. When combined, the statistical power of the analysis increases. While the size of the dose-response relationship declined relative to past studies with a statistically significant finding, Ryan found a statistically significant relationship between IQ and Hg. The confidence interval did not include zero. EPA used Ryan's mean estimate -0.131 as a coefficient to relate changes in exposure (parts per million in hair) to IQ point changes. EPA also performed sensitivity analysis using coefficients of -0.108 and -0.233 also based on the work by Louise Ryan and David Bellinger. (Ryan 2005).[21]

NESCAUM appears to have used a coefficient of -0.60 based on an unpublished study (Cohen, et al. full reference unavailable) that is not included in the references. The paper upon which NESCAUM based its coefficient has not been submitted to the rulemaking docket, making it difficult for us to assess the NESCAUM approach. However, the reliance on a coefficient of -0.60 is not consistent with the work done by Harvard researchers Louise Ryan and David Bellinger or the other existing studies.

9.3 Conclusion

Unlike the analyses conducted for this reconsideration which provide an estimate of the upper-bound of IQ benefits associated with reduced mercury emissions from power plants, the NESCAUM Report estimates are presented as "central" or "best" benefits from marine fish Hg reductions. Further even if presented as upper-bound estimates, there is a disconnect between the derivation of quantitative results and the summary of the science since authors relied on a variety of unsupported assumptions.

10. 2010/2015 CMAQ Modeling Results

The Technical Support Document (TSD) for the Final Clean Air Mercury Rule: Air Quality Modeling, March 2005, describes in Section V.B. that the expected mercury deposition with CAIR plus CAMR in 2015 is expected to be similar to the mercury deposition with CAIR plus CAMR in 2020. Since the March 2005 CAMR TSD was prepared, a combined strategy consisting of the implementation of CAIR, CAMR and

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21 Ryan and David develop a linear model that goes through the origin to fit population-level dose-response relationships to the pooled data from the three studies. The application of a linear model should not be interpreted to suggest that any of the three studies used have data showing health effects from MeHg exposure at or below the RfD. Use of a linear model that goes through the origin, rather than one that reflects a threshold effect is technically more simple and practical. It associates an increment of IQ benefit with a given reduction in exposure. A linear model allows us to estimate the benefits of reductions in exposure due to power plants without a complete assessment of other sources of exposure. Other models would require information on the joint distribution of exposure from power plants and other sources to estimate the benefits of reducing the exposure due to power plants, which would require much more precise information about consumption patterns.

CAVR has been modeled with CMAQ for 2010, 2015 and 2020. The BART rule indicates that States that opt into the CAIR trading program do not have to do anything more for BART for their eligible utility sources. Therefore, CAIR satisfies BART for utilities for the CAIR States. Thus, in the CAIR States, there would be no further reductions in utility mercury emissions from BART. It is only, in the States where CAIR does not apply that the implementation of BART would possibly lead to additional mercury reductions over those that would occur with CAIR and CAMR.

The total utility mercury emissions for the each of the Clear Skies CAIR-CAMR-BART scenarios are shown in Table 10.1. More importantly, the utility mercury emissions of the most readily depositable form of mercury emissions (Reactive Gaseous Mercury) for each of these scenarios are also shown Table 10.1. It can be seen in Table 10.1 that there is a large (approximately 11 ton) decrease in utility reactive gaseous mercury (RGM) emissions from 2001 to 2010. The reduction in RGM emissions from 2010 to 2015 is approximately 2 tons and the reduction in RGM emissions from 2015 to 2020 is only approximately 1 ton.

The total modeled mercury deposition for 2010, 2015 and 2020 under CAIR/CAMR/BART are provided below in Figures 10.1 through 10.3. It can be seen in Figures 10.1 through 10.3 that the total mercury depositions with CAIR/CAMR/BART are very similar in 2010, 2015 and 2020. The reduction in total mercury deposition from 2001 to 2010, 2015 and 2020 with the implementation of CAIR/CAMR/BART are shown in Figures 10.4 through 10.6. It can be seen in figures 10.4 through 10.6 that the difference in the decrease in total mercury deposition between 2010, 2015 and 2020 relative to 2001 are fairly small. Figure 10.7 provides the reduction in total mercury deposition between 2010 and 2015 and Figure 10.8 provides the reduction in total mercury deposition between 2015 and 2020. It can be seen in Figure 10.7 that the reductions in total mercury deposition from 2010 to 2015 cover scattered areas of the country with the reductions less than 5 ug/m^2. It can be seen in Figure 10.8 that the reductions in total mercury deposition from 2015 to 2020 cover only scattered small areas of the country with the reductions generally less than 5 ug/m^2. It can be seen by examining Figures 10.4, 10.7 and 10.8 that there is a much larger reduction in utility attributable mercury deposition from 2001 to 2010 than from 2010 to 2015. The reduction in utility attributable mercury deposition from 2015 to 2020 is even smaller than the reduction from 2010 to 2015.

The mercury deposition reductions shown in Figures 10.4 through 10.6 look very similar to the reduction in mercury deposition that would occur from 2001 with the implementation of CAIR and CAMR in 2020, which were modeled under the CAMR rule, and are shown below in Figure 10.9. The additional reductions in 2020 total mercury depositions that would occur relative to 2001 with the implementation of BART in addition to CAIR and CAMR are provided in Figure 10.10. As can be seen in Figure 10.10, the maximum additional reduction with BART is less than 4 ug/m^2 in all locations. The additional reductions with BART are outside the eastern States covered by CAIR.

Table 10.1. Utility Mercury Emissions for Clear Skies Act CAIR-CAMR-BART Scenario (tons/year)

Year	Reactive Gaseous Mercury	Total Mercury Emissions
2001	20.6	48.6
2010	9.7	32.1
2015	7.2	28.7
2020	6.1	25.5

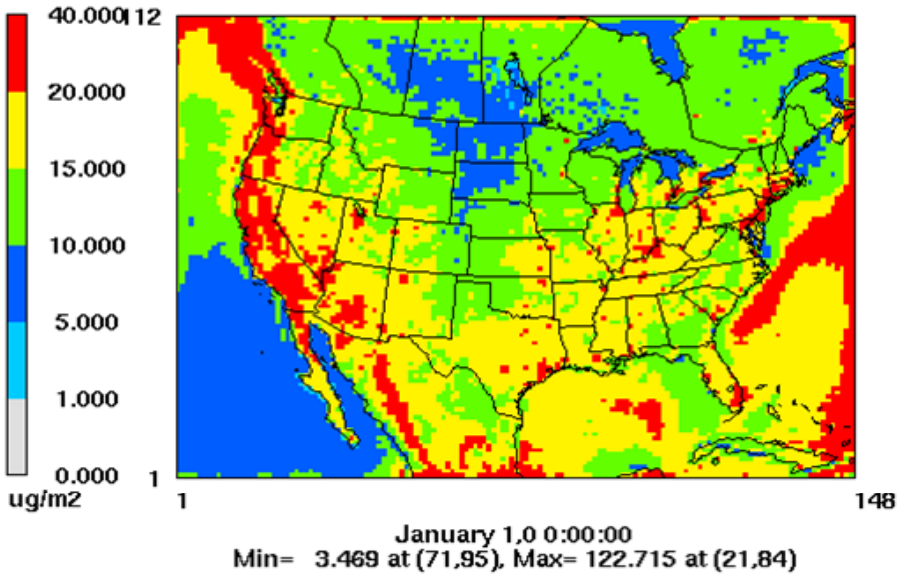

Figure 10.1. Total Mercury Deposition with CAIR/CAMR/BART: 2010

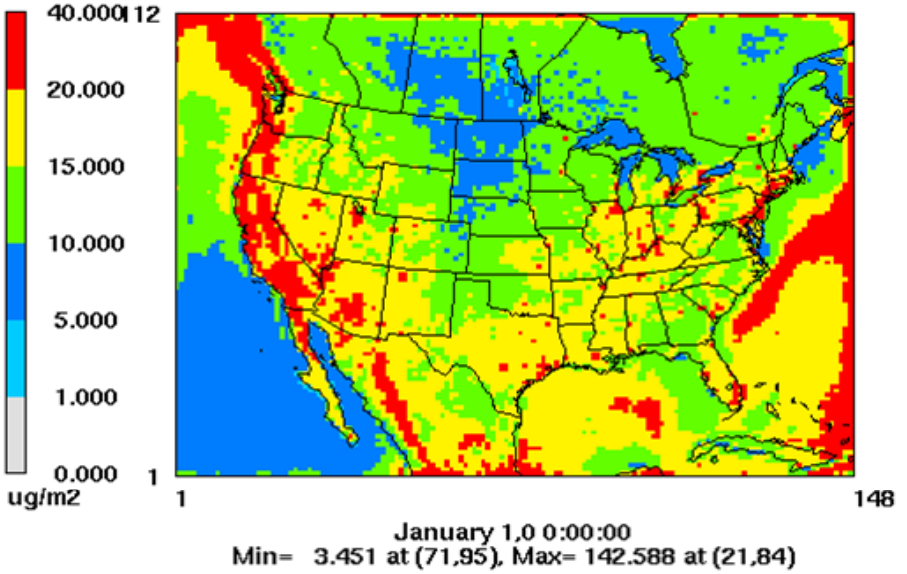

Figure 10.2. Total Mercury Deposition with CAIR/CAMR/BART: 2015

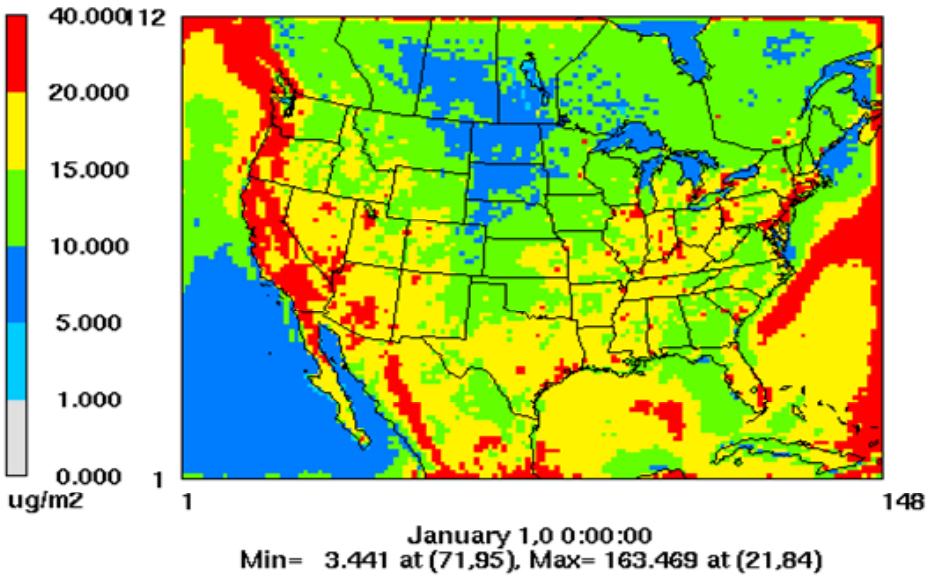

Figure 10.3. Total Mercury Deposition with CAIR/CAMR/BART: 2020

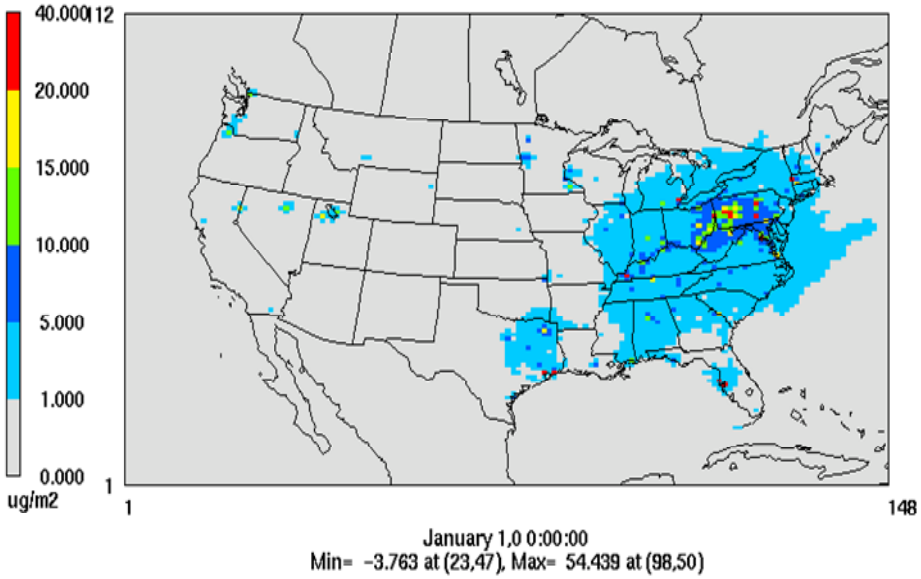

Figure 10.4. Reduction in Total Mercury Deposition: 2010 with CAIR/CAMR/BART Relative to 2001

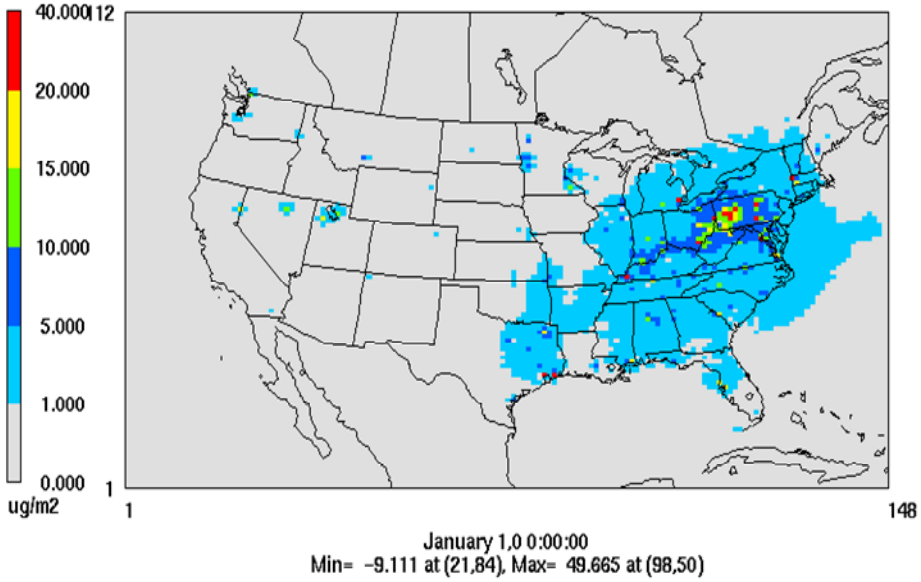

Figure 10.5. Reduction in Total Mercury Deposition: 2015 with CAIR/CAMR/BART Relative to 2001

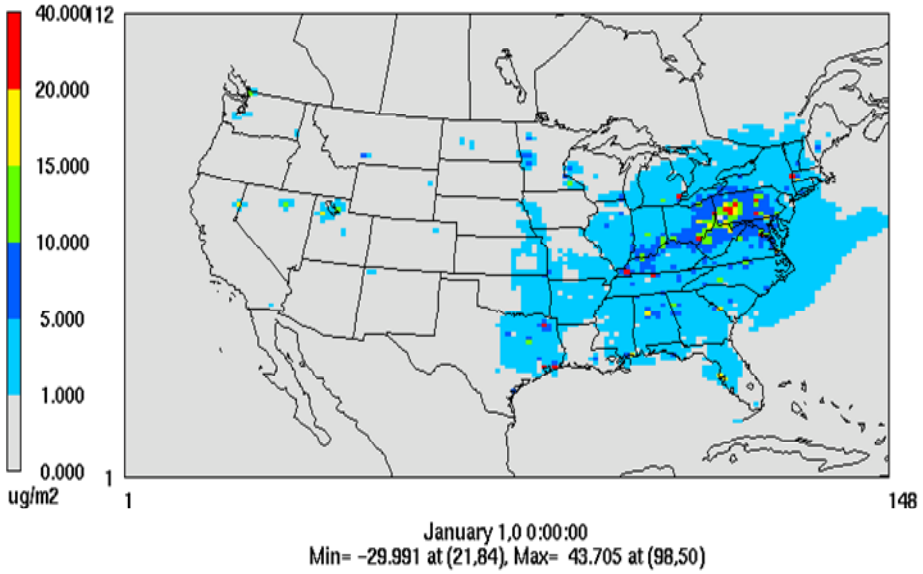

Figure 10.6. Reduction in Total Mercury Deposition: 2020 with CAIR/CAMR/BART Relative to 2001

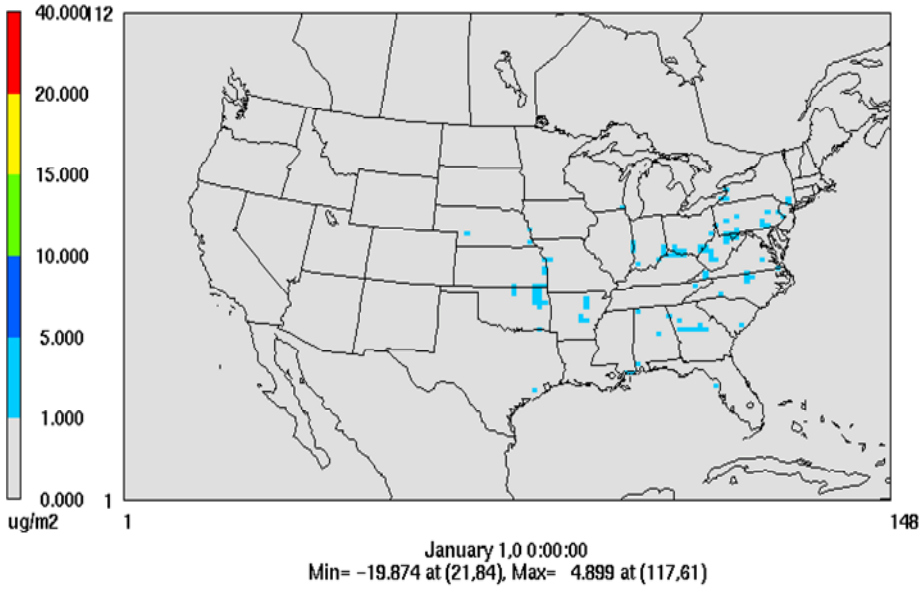

Figure 10.7. Reduction in Total Mercury Deposition with CAIR/CAMR/BART: 2010 to 2015

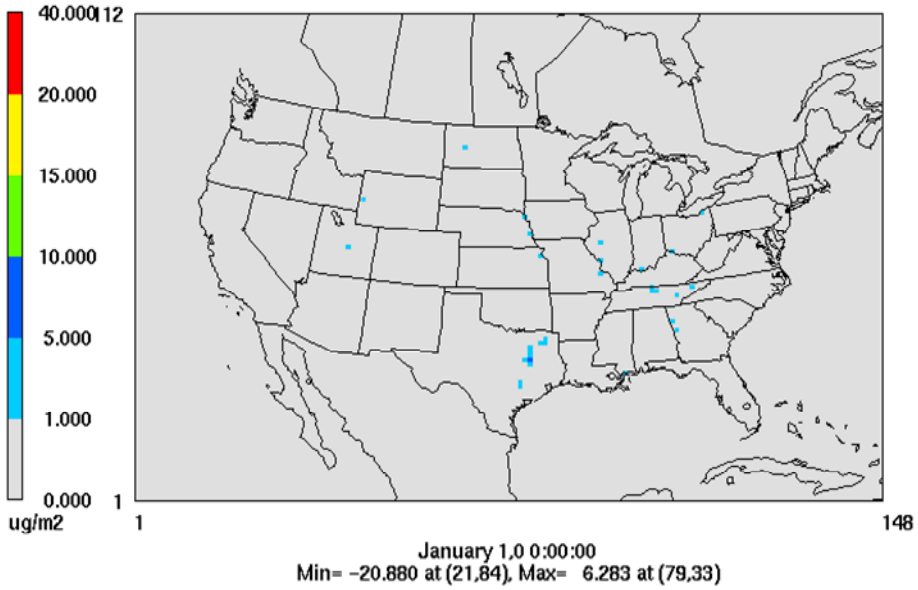

Figure 10.8. Reduction in Total Mercury Deposition with CAIR/CAMR/BART: 2015 to 2020

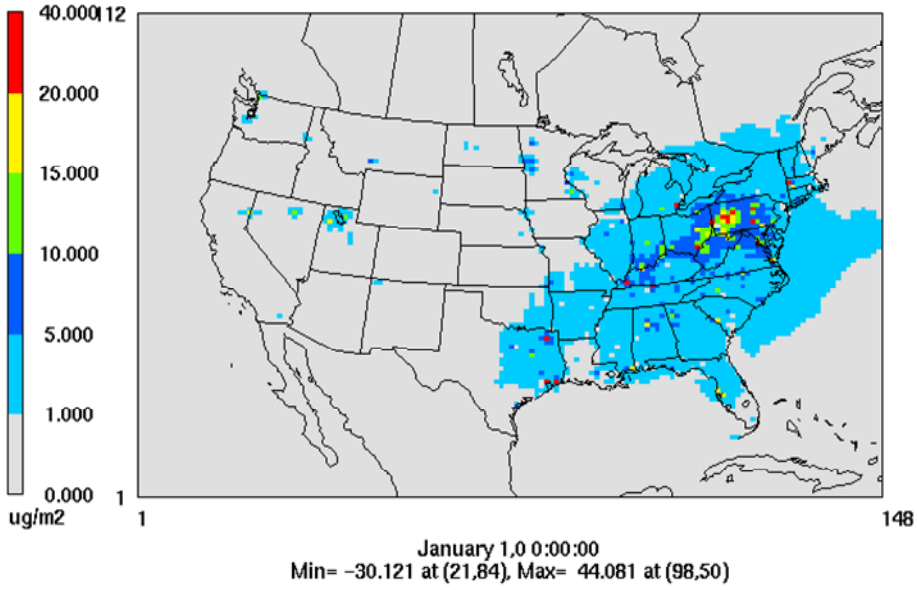

Figure 10.9. Reduction in Total Mercury Deposition with CAIR/CAMR: 2020 Relative to 2001

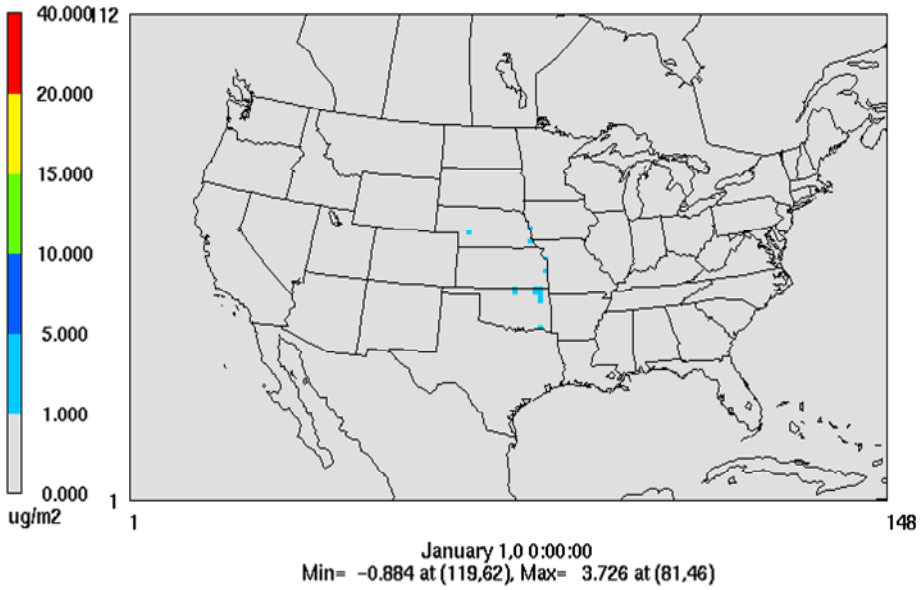

Figure 10.10. Reduction in Total Mercury Deposition with BART in addition to CAIR and CAMR: 2020 Relative to 2001

11. Global Source Impact

A CMAQ 2001 modeling run was performed after the CAMR rule was finalized to estimate the impact of global sources. In this model run, all mercury boundary condition input species to CMAQ that were obtained from the GEOS-CHEM global model were zeroed-out. By comparing this run with the 2001 base case run, which included the mercury boundary condition species input to CMAQ, the percent of total mercury deposition attributable to global sources can be estimated. The model estimated percent of total mercury deposition attributable to global sources is provided below in Figure 11.1. The scientific understanding of mercury atmospheric chemistry is still evolving. Changes in the current understanding of mercury chemistry could possibly lead to the need to change the mercury chemistry in the global GEOS-CHEM and regional CMAQ models. Thus, it should be noted that there is considerable uncertainty associated with the estimates of global source impacts.

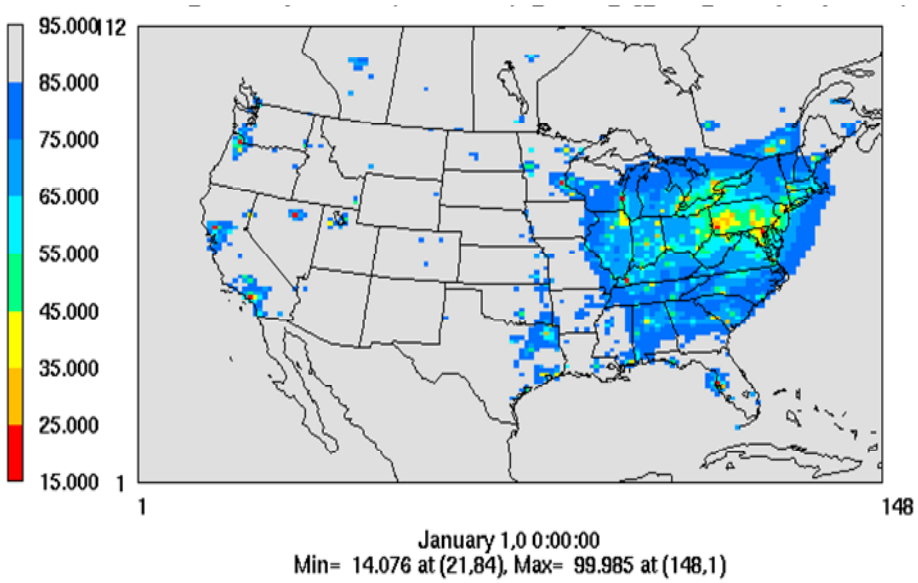

Figure 11.1. Percent of Total Mercury Deposition Attributable to Global Sources: 2001

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