Economic Valuation of Human Health Benefits of Controlling Mercury Emissions from U.S. Coal-Fired Power Plants

February 2005

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EXECUTIVE SUMMARY

This report describes the results of a comprehensive study to estimate the health benefits of reducing mercury emissions from coal-fired power plants in the United States. Reductions in mercury emissions from coal-fired power plants are anticipated to decrease methylmercury concentrations in fish. Fish consumption is the primary pathway of human exposure to methylmercury, which is a human neurotoxicant and possibly a cardiovascular toxicant. Some babies born to U.S. residents, a population sensitive to the neurotoxic effects of methylmercury, are currently exposed to intrauterine methylmercury concentrations above the EPA's Reference Dose (RfD). (The RfD is considered by some to be an acceptable level of exposure.)

The modeling analysis is based on the assumption that equilibria currently exist between deposited mercury and fish methylmercury concentrations and between fish methylmercury concentrations and methylmercury exposures to individuals who consume these fish. Changes in the quantity of mercury deposited are assumed to lead to linear and proportional changes in fish methylmercury concentrations, assuming no other factors change. In the model, the waters from which fish are caught for human consumption are divided into eight regions. Based on broad regional patterns of mercury deposition rates, the continental U.S. is divided into five freshwater regions, the Northeast, Mid-Atlantic, Southeast, Midwest, and West. There are three saltwater regions, the Atlantic Coastal, Gulf of Mexico, and All Other Waters. Each region is modeled as an isolated compartment, connected only with the atmospheric compartment above. Thus, in each region, the predicted change in mercury deposition rate results in a proportional change in fish methylmercury levels.

Changes in mercury deposition rates associated with reductions in power plant mercury emissions are based on regional deposition modeling results from the EPA's analysis of the Clear Skies Initiative. In its analysis, the EPA simulated current mercury deposition rates and the changes in these rates that would result if power plants reduced their mercury emissions from the current rate of 49 tons per year to either 26 or 15 tons per year. We used these predictions to estimate changes in deposition rates for the freshwater regions, the Atlantic Coastal Region, and the Gulf of Mexico. Estimated decreases range from approximately 1% to 10%. The change in deposition rates to the All Other Waters region is assumed to be proportional to the change in total global emissions that would result from U.S. power plant emissions reductions, which is less than 1%.

The model accounts for human exposure through commercially and non-commercially harvested fish. Commercial and non-commercial fish consumption rates are based on data reported by the U.S. Food and Drug Administration (FDA) and the EPA. Methylmercury concentrations in commercially harvested fish are based on published reports of scientists from the FDA. Using per capita consumption rate data, we develop a weighted mean methylmercury concentration for commercial fish. This concentration changes in response to predicted deposition rate changes in the Atlantic

Coastal Region, the Gulf of Mexico, and the All Other Waters Region. Non-commercially harvested fish are caught in each of the five freshwater regions plus the Atlantic Coastal and the Gulf of Mexico Regions. Current methylmercury concentrations are estimated from EPA databases. In each freshwater region, the mix of species that is consumed is based on regional data on the frequencies at which individual species are targeted. For the Atlantic Coastal and the Gulf of Mexico Regions, the mix is assumed to be consistent with annual harvest weights for non-commercial marine fish published by the National Marine Fisheries Service (NMFS). The regional populations of non-commercial fish consuming populations are estimated using data from NMFS and the U.S. Fish and Wildlife Service. Changes in non-commercial fish concentrations under different mercury emissions control scenarios are proportional to changes in deposition in each region.

The model accounts for potential changes in two health effects: cognitive abilities and cardiovascular events. We assume that increases in a child's intelligence quotient (IQ) that result from decreases in intrauterine methylmercury exposures capture some of the neurodevelopmental delays reported in positive epidemiologic studies. We use a recent estimate of the dose-response function that integrates the results of the three primary methylmercury epidemiologic studies (from the Faroe Islands, Seychelle Islands, and New Zealand). Using a cost-of-illness approach, we estimate the value of a lost IQ point to be approximately $16,500 (year 2000 dollars). We note that the body of scientific evidence that has evaluated the relationship between intrauterine methylmercury exposures and childhood neurodevelopmental delays has been thoroughly reviewed by the National Academy of Sciences.

Credible epidemiological studies have reported an association between methylmercury exposures in males and increased risks of myocardial infarction and premature mortality. The exposed population in a group of these epidemiological studies consumed non-fatty freshwater fish. We use regression coefficients reported in these studies to estimate dose-response relationships for premature mortality and non-fatal myocardial infarction. We apply these dose-response estimates alternatively to males who eat non-fatty freshwater fish and to all fish consumers. Using a cost-of-illness approach, we estimate the value of a myocardial infarction to be approximately $50,000 (2000$). Using a willingness-to-pay approach, we estimate the value of a premature fatality to be approximately $6,000,000 (2000$). While the individual epidemiological studies have been published in the peer-reviewed literature, the body of scientific evidence concerning the relationship between adult methylmercury exposures and increases in cases of premature mortality and non-fatal myocardial infarction has not been thoroughly reviewed. Furthermore, this relationship is potentially confounded with the cardio-protective effect of fish consumption. Based on the available data, independent scientific bodies evaluating the weight of evidence for fish cardio-protective effects have recommended that adults increase their fish consumption. Whether there is an increased cardiovascular risk associated with methylmercury exposures is not clear at this time. Thus, we recommend that the predicted benefits associated with premature mortality and non-fatal myocardial infarction be viewed with caution.

Figure ES-1 describes a range of possible benefits of U.S. power plant mercury emissions controls. The benefits associated with Scenarios 1 and 2 are predicted to result when power plants face annual mercury emissions caps of 26 tons and 15 tons, respectively. While the benefits estimates increase from left to right in this figure, our confidence that the U.S. population experiences these effects decreases. For Scenario 1, the predicted annual benefit associated with IQ increases in the annual birth cohort ranges from $75 million (assuming a neurotoxicity threshold equal to RfD) to $194 million (assuming no threshold). The corresponding annual benefit predictions for Scenario 2 are $119 million to $288 million. The monetized benefits associated with avoided cardiovascular events and premature mortality are predicted to be much larger than the neurotoxicity benefits; however, there are additional uncertainties in the external generalization of the results of the epidemiologic studies upon which these estimates are based to the U.S. population. If these cardiovascular effects are only experienced by male populations that consume non-fatty freshwater fish, then the monetized annual benefits are $48 million and $86 million in Scenarios 1 and 2, respectively. If these cardiovascular effects are experienced by the whole U.S. population, then the monetized annual benefits are predicted to be $3.3 billion and $4.9 billion in Scenario 1 and Scenario 2, respectively.

This report highlights numerous mercury research needs. The following four are likely among the most important. Long-term monitoring studies that measure mercury concentrations in environmental media and biota over time are needed to determine the extent to which reductions in mercury emissions decrease mercury concentrations in the environment and reduce human exposures. Studies that evaluate the atmospheric fate of mercury emitted from coal-fired power plants need to be implemented on local, regional, and global scales. Additional studies need to be undertaken to evaluate short-term and long-term fish consumption rates in the U.S. population; these studies need to identify the types of fish consumed and their sources. Additional studies are needed to evaluate the relationship between methylmercury exposures and the development of cardiovascular effects; these should include studies of human physiological responses to fish fatty acids (the component of fish thought to be cardio-protective) and to methylmercury.

Spectrum of Health Effect Certainty

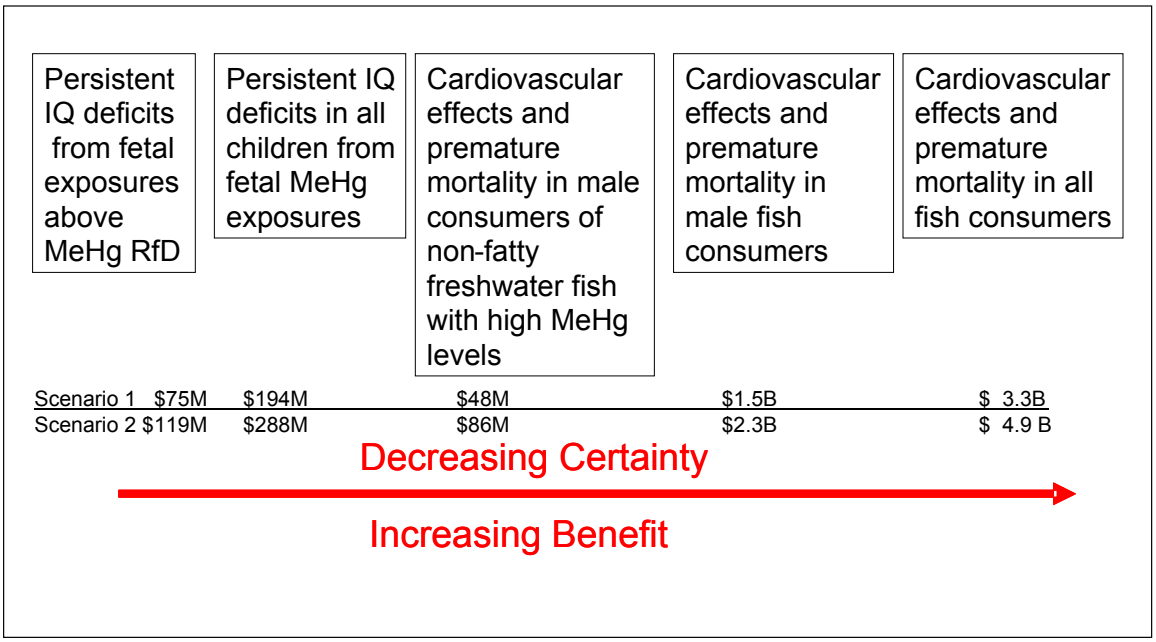

Figure ES-1

Spectrum of Certainty of Causal Association of Health Effect with Mercury Exposure with Estimated Benefit Overlay in Millions ($M) and Billions ($B) of Dollars (2000$)

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1. INTRODUCTION AND BACKGROUND

In recent years, the U.S. Environmental Protection Agency (EPA) issued final regulations mandating the reduction of mercury emissions from municipal waste combustors (1995), medical waste incinerators (1997), and hazardous waste incinerators (1999). The impetus for these federal regulations was the plausible connection between anthropogenic mercury emissions and potentially harmful methylmercury levels in consumable fish. Despite these recent regulatory actions intended to reduce methylmercury levels in fish, concerns about the safety of fish consumers persist among U.S. environmental and public health officials. This concern continues because the fetuses of some pregnant women in the U.S., a population sensitive to the effects of methylmercury, are exposed to this toxicant above the U.S. EPA's reference dose (RfD)[1] (Schober et al., 2003; Mahaffey et al., 2004; U.S. NCHS, 2003; U.S. EPA, 2001a; NRC, 2000; CDC, 2004). New federal and state legislation, regulation and policies designed to reduce mercury emissions further are being pursued. Some of these policies target coal-fired power plants because they emit over 35% of total U.S. anthropogenic mercury emissions (Table 1).

[1] The EPA defines the RfD to be "an estimate (with uncertainty perhaps spanning 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" (U.S. EPA, 1999).

Table 1. Mercury Emissions Sources

Sources to Atmosphere	Annual Emission Rate (tons yr^{-1})	Reference
Natural Emissions Land	1000 1100	Mason et al., 2002; Lamborg et al., 2002
Oceanic Evasion	2850[a] 900	Mason et al., 2002; Lamborg et al., 2002
Anthropogenic Northern Hemisphere[a]	2450	Lamborg et al., 2002
Anthropogenic Southern Hemisphere	450	Lamborg et al., 2002
Total Global Anthropogenic	2650 2850	Mason et al., 2002; Lamborg et al., 2002
Total Global Emissions	4850	U.S. EPA, 2003a
U.S. Utility Boilers Coal Oil Natural gas	48.9 (36%)[b] 48.0 0.5 0.4	U.S. EPA, 2003a
U.S. Ore Gold Ore Iron Ore Silver Ore Ferroalloy Ores, Except Vanadium	11.7 (9%) 11.5 0.2 4.0E-3 5.5E-4	U.S. EPA, 2003a
U.S. Chlorine Production	6.5 (5%)	U.S. EPA, 2003a
U.S. Municipal Waste Combustors	5.1 (4%)	U.S. EPA, 2003a
U.S. Hazardous Waste Combustion Commercial Hazardous Waste Incinerators On-Site Hazardous Waste Incinerators Hazardous Waste Incineration	5.0 (4%) 2.48 2.38 0.98	U.S. EPA, 2003a
U.S. Industrial Boilers Industrial/Commercial/Institutional Boilers & Process Heaters Stationary Combustion Turbines	3.8 (3%) 3.28 0.51	U.S. EPA, 2003a
U.S. Medical Waste Incinerators	2.8 (2%)	U.S. EPA, 2003a
Subtotal (U.S. Sources)	83.8 (61%)	U.S. EPA, 2003a
Total Point and Non-point U.S. Emissions	136.3	
Natural Emissions from U.S. [c]	64	

[a] In the Mason and Scheu (2002) model much of the mercury released to the atmosphere from the ocean re deposits into ocean.
[b] The percentage of total U.S. anthropogenic emissions as simulated in U.S. EPA (2003a) is based on 1999 emission estimates. U.S. anthropogenic emission estimates have been updated (www.epa.gov/ttn/chief).
[c] We developed this estimate based on natural global mercury emissions estimates of Lamborg et al. (2002). Using Lamborg's approach, the U.S. estimate is based on the ratio of U.S. landmass to total landmass of northern hemisphere.

Figure 1 summarizes the results of a global dynamic simulation model for mercury (Lamborg et al., 2002). In this model, anthropogenic mercury emissions (2900 tons annually) comprise roughly 60% of the total quantity emitted globally (4900 tons). Annually, 900 tons of mercury are emitted from aquatic systems and 1100 tons are emitted from terrestrial systems; these emissions consist of both re-emitted anthropogenic mercury and mercury naturally present in these environmental media. Deposition of atmospheric mercury to land accounts for slightly more than 50% of the total deposition; the remainder of the atmospheric mercury deposits to water bodies. In this model, concentrations in compartments of the biosphere (e.g., atmosphere and upper levels of the oceans) are predicted to be increasing approximately 1% per year.[2]

Estimating the human-health benefits of reductions in mercury emissions from U.S. power plants requires accounting for the atmospheric transport and deposition of mercury, methylation, uptake and bioconcentration in fish, human consumption, the relationships between human exposure to methylmercury and health effects, and the valuation of those effects. The remainder of this section provides background information on these topics.

[2] Seigneur et al. (2004) compare four global mercury simulation models, including Lamborg et al. (2002). The comparison (see Table 2 in Seigneur et al.) shows Lamborg's estimate of anthropogenic mercury emissions to be 15% higher than those associated with other models. See Table 1.

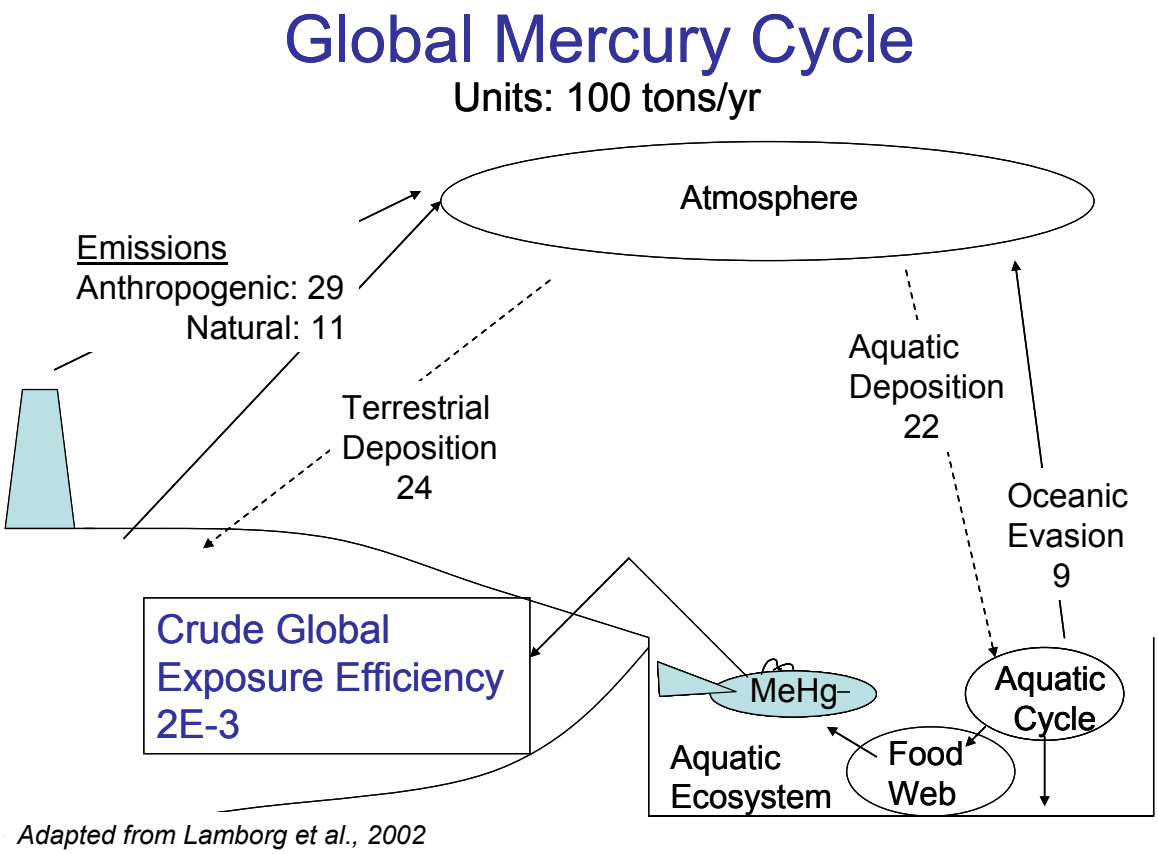

Figure 1

Global Mercury Cycle

1.1. ATMOSPHERIC MERCURY

Understanding the transport and deposition of inorganic mercury emitted to the atmosphere is fundamental to analyzing the changes in methylmercury exposures that may be associated with future mandated mercury emissions reductions. In the environment, mercury exists in two general forms: inorganic forms, which include elemental mercury, mercury oxides and mercury salts, and organic forms, which include alkylated species such as methylmercury. Inorganic forms of mercury are released to the atmosphere by natural (e.g., off-gassing from surface waters and volcanic emissions) and anthropogenic sources (Table 1). The distribution of mercury in the

environment is dominated by transport during its atmospheric phase. Although the atmospheric chemistry of mercury is extremely complex and incompletely understood, most experts agree that mercury emissions can deposit potentially causing problems both locally (i.e., close to emissions sources), regionally (i.e., within a few hundred kilometres of a source) and globally. Thus, policy makers are concerned about the impacts of local and long-range transport of atmospheric mercury.

Three different forms of inorganic mercury emissions are typically modeled in atmospheric transport models. These are elemental mercury (Hg^0), gas phase divalent mercury (Hg^{II}) (also referred to as reactive gaseous mercury), and particulate-bound divalent mercury (Hg_p). Based on the current understanding of the fate of atmospheric mercury (e.g., Petersen et al., 1995; Cohen et al., 2004; Seigneur et al., 2004), Hg^{II} and Hg_p are deposited to the surface of the earth relatively rapidly through wet and dry deposition processes.[3] Thus, when a mix of Hg^0, Hg^{II} and Hg_p is released from an anthropogenic source, Hg^{II} and Hg_p are thought to deposit locally and regionally around the source. Due to the difficulties in measuring the dry deposition of Hg^{II}, estimates of its deposition are uncertain and a source of disagreement among modelers and policy analysts. For example, U.S. EPA (1997c, 1998a) predicted that over 80% of the mercury that deposited within 50 Km of hypothetical coal-fired power plant was emitted as Hg^{II} and Hg_p,[4] although the two forms comprised only 50% of the total emitted.

[3] Hg^{II} and Hg_p are readily scavenged by atmospheric precipitation and deposited to the earth through wet deposition. Hg_p is dry deposited due to gravitational settling. Gaseous Hg^{II} also can dry deposit. Hg^{II} dry deposition velocities appear to depend on both ground cover and weather conditions; a typical deposition velocity for Hg^{II} is estimated to be 2.9 cm/s (U.S. EPA, 1997a, 1998a). While the wet deposition of mercury and dry deposition of Hg_p can be measured in a relatively direct manner, measuring the quantities of gaseous Hg^{II} that dry deposit is complex and estimates of these deposition rates are less certain (Lindberg and Stratton, 1998).

Others contend that the fraction depositing locally from such sources is much less than estimated in EPA's simulation. Because these two forms of mercury deposit near anthropogenic sources, policy makers need to take into account the local and regional nature of impacts of mercury emissions.

On the other hand, Hg^0 is volatile and much less soluble than Hg^{II}. Its estimated atmospheric half-life is approximately 1.5 years. Given this long half-life, emitted Hg^0 is unlikely to deposit locally; rather, it enters regional and global atmospheric mercury pools. In these regional and global pools, Hg^0 slowly converts to Hg^{II} and deposits (Petersen et al., 1995). For example, the results of the same U.S. EPA simulation described previously (U.S. EPA, 1997c, 1998a) predicted that only 30% of the total mercury emitted from all U.S. power plants deposits in the contiguous U.S. If these predictions are accurate, then policy makers also need to consider regional and global impacts of this form of emitted mercury.

To generate as accurate an understanding as possible of the deposition of atmospheric mercury, both the U.S. EPA and the Electric Power Research Institute (EPRI) have developed models to simulate the long-range transport and deposition of atmospheric mercury in the contiguous U.S.[5] U.S. EPA used a Lagrangian or trajectory model to estimate the fate of emitted mercury in two separate Reports to the U.S. Congress (Bullock, 1997; U.S. EPA, 1997c, d, 1998a, b). Limitations in these earlier

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[4] Very little Hg_p is emitted by power plants; however, roughly 20% of the measured mercury in power plant emissions plumes is Hg_p (U.S. EPA, 1997b). When the U.S. EPA simulated the atmospheric fate of mercury, they assumed that 20% was bound to particulate matter based on measurements in the emission plume.

[5] Other groups have also developed regional atmospheric fate models for mercury; for example, see Cohen et al. (2004) and Petersen et al. (1995).

simulation models (e.g., limited treatment of atmospheric mercury chemistry) have led both EPA and EPRI to develop regional scale Eulerian or grid-based air quality models that simulate long-term deposition fluxes of atmospheric mercury over large geographic domains

In 2002, the U.S. EPA proposed the Clear Skies Initiative (CSI) to reduce power plant emissions of mercury through the application of a market-based "cap and trade" approach over the next two decades.[6] In the analysis of the CSI, the U.S. EPA used the Regulatory Modeling System for Aerosols and Deposition (REMSAD) model to predict potential changes in mercury deposition associated with changes in power plant mercury emissions. The model predicted annual mercury deposition rates for each 36 x 36 Km grid cell in the contiguous U.S., the western Atlantic Ocean and the Gulf of Mexico.

The EPA's CSI simulation included predictions of current mercury deposition rates using the 1999 U.S. mercury emissions inventory (U.S. EPA, 2002); in this analysis 36% (48.9 tons) of the 136.3 tons of mercury emitted annually by U.S. anthropogenic sources was attributed to power plants.[7] Separate simulations were developed to compare predicted changes in mercury deposition under the CSI at two different points in time. The mercury emissions cap in 2010 for utilities subject to CSI is 26 tons. Based on these simulations, in the year 2010, the EPA (U.S. EPA, 2003a, b)

[6] In 2005, CSI was reintroduced in the U.S. Congress with a higher first phase cap of 34 TPY of mercury emissions in 2010 instead of the 26 TPY cap in the 2002 proposal. However, all analyses in this report are based on 26 TPY cap in 2010.

[7] Recent updates to the inventory estimate that power plants account for approximately 40% of mercury emitted from U.S. anthropogenic sources annually. (www.epa.gov/ttn/chief/net (See 1999 NEI data).

predicts U.S. anthropogenic mercury emissions to be 104.7 tons; if the CSI is implemented, total U.S. anthropogenic emissions are predicted to be 85.6 tons per year because power plant mercury emissions will be 29.8 tons per year (26 tons per year for power plants that are subject to CSI). By 2020, U.S. EPA (2003a,b) predicts annual anthropogenic mercury emissions to be 105.7 tons and if the CSI is implemented, total annual anthropogenic emissions are predicted to be 79 tons because power plant emissions will be at 23.2 tons (power plants that are subject to CSI would emit 18 tons in that year assuming no "safety valve.")[8] The mercury emissions cap in 2018 for utilities subject to CSI is 15 tons. In their simulations, the EPA included differences in the quantities and species of mercury emitted from power plants using a model that predicts the U.S. power sector's response (including fossil fuel consumption) to regulatory and economic changes. If the CSI is fully implemented, EPA predicts slightly more than a 10% decrease in mercury deposition across the U.S., when compared to current deposition rates.

EPRI (2003) applied the Trace Element Analysis Model (TEAM) to predict the changes in mercury deposition in the U.S. under the CSI and predicted notably different impacts than were predicted by EPA (2003a). The TEAM is a regional-scale Eulerian model that simulates the atmospheric chemistry of mercury over the North American continent using 100 x 100 Km grids. Changes in coal consumption patterns and mercury emissions by the electric power industry under the CSI were modeled. EPRI (2003) predicted that current mercury emissions from power plants account for less than

[8] The "safety valve" is a provision of the cap and trade program that allows power plants to meet their obligations by purchasing emission allowances at a predetermined cost per ounce of mercury.

8% of the mercury deposited in the U.S.; the model predicts that most deposition is the result of emissions from sources outside the U.S. The impact of mercury emissions changes was analyzed using "transfer coefficients" estimated from the model. Transfer coefficients are model parameters that relate changes in mercury emissions to changes in mercury deposition at specific locations. For example, deposition at a New York receptor is most influenced by changes in emissions in New York and New Jersey; changes in emissions from the U.S. Midwest influence mercury deposition at the New York receptor more than changes in emissions from the South (Figure 2-2 in the EPRI report). Many of the transfer coefficients were estimated for sites monitored by the National Atmospheric Deposition Program Mercury Deposition Network (NADP-MDN), although this network was designed to measure background mercury concentrations and not contributions from local anthropogenic sources. Based on their analysis of transfer coefficients at 19 receptor sites, EPRI (2003) estimates that, with CSI regulations in place, there will be a 1.5% (range of 0.1-5.3%) decrease in mercury deposition rates across the U.S. by 2020.

While the predicted mercury deposition rate decreases vary between the two models, the TEAM and REMSAD simulations predict similar geographic patterns of mercury deposition. For example, more mercury deposition is predicted in the Eastern U.S. than in the Western U.S., with high deposition rates in the Mid-Atlantic and Northeast Regions of the U.S. The EPRI model results suggest significantly smaller contributions to overall mercury deposition from power plants than do the EPA models. The EPRI report does not detail changes in deposition within the contiguous U.S.;

rather, it averages deposition changes over the entire U.S. and does not explore local and regional variability in mercury deposition.

1.2. BIOACCUMULATION OF METHYLMERCURY

Following deposition, inorganic mercury enters bodies of water where sulfate-reducing micro organisms in the water column and in the sediments convert small fractions (typically about 1 or 2%) to the form of methylmercury (Hamdy and Noyes, 1975; Gilmour and Henry, 1991).[9] Invertebrates such as zooplankton and insects that feed on organisms at the base of the aquatic food web accumulate methylmercury. Methylmercury binds to amino acids through sulfhydral bonds and is incorporated into muscle protein. Small fish feed on the contaminated aquatic invertebrates, preferentially retain methylmercury, and eventually are eaten by predatory (i.e., piscivorous) fish. Because methylmercury is highly retained in organisms, it rapidly bioaccumulates up the food web and as a result, in a body of water, the large predatory fish have the highest methylmercury concentrations in their muscle tissues.[10]

[9] Soil microorganisms can also methylate inorganic mercury (St. Louis et al., 1996). This likely occurs primarily in wetlands during anoxic periods. The mercury methylated by soil microorganisms can be transported to a water body.

[10] Inorganic forms of mercury are less efficiently absorbed through the fish gut and more efficiently eliminated from fish than methylmercury; consequently they do not bioaccumulate in aquatic organisms to the extent that methylmercury does.

There is a great deal of variability in the accumulation of methylmercury in fish across different bodies of water.[11] Thus, the methylmercury level in a fish depends on geography, geochemistry, the age and size of the fish and its position in the food web.

Understanding the fate of mercury entering a water body is fundamental to predicting the changes in methylmercury concentrations in fish that may be associated with reductions in mercury deposition. In an analysis of the applications of the primary models used to estimate methylmercury bioaccumulation in studies of specific bodies of water (Harris et al., 1996; U.S. EPA, 1997a), the Mercury Maps project (U.S. EPA, 2001d) showed that predicted changes in methylmercury concentrations in fish were proportional to changes in mercury entering the water body under steady-state assumptions. If the atmospheric mercury deposition was the dominant source of mercury to a water body, then the changes in deposition that might follow mercury emission controls would result in proportional changes over time in fish concentrations (U.S. EPA, 2001d). If non-atmospheric sources, such as mines and relic accumulations in soil and sediments, are significant sources of inorganic mercury to a body of water, then reducing atmospheric deposition of mercury is predicted to result in less than proportional (i.e., nonlinear) reductions of fish methylmercury concentrations.

[11] The following factors reportedly impact the accumulation of methylmercury in fish:

- mercury transport into the aquatic system including the fraction of a watershed that consists of wetlands (St. Louis et al., 1996),
- food chain length (i.e., short food chains result in less methylmercury bioaccumulation among apex predators) (Cabana and Rasmussen, 1994)
- breadth of lower trophic levels (i.e., dilution of methylmercury across biomass at low end of food chain leads to less accumulation among piscivorous species) (Pickhardt et al., 2002)
- demethylation rates (DisPasquale et al., 2000)
- nutrient loading (Hammerschmidt and Fitzgerald, 2004)
- water pH (Brumbaugh et al., 2000)
- dissolved organic carbon (Brumbaugh et al., 2000)
- sulfate concentration (Brumbaugh et al., 2000).

The model developed in the Mercury Maps project (U.S. EPA, 2001d) was used to conduct a national analysis at a watershed level to estimate the percent change in mercury deposition needed to remove fish consumption advisories. U.S. EPA (2001d) suggested that by using fish methylmercury concentrations reported in the National Listing of Fish and Wildlife Advisories (NLFWA) database (U.S. EPA, 2003d) and simulation results from atmospheric mercury models, the Mercury Maps model could be used to estimate the change in the number of fresh water bodies with fish consumption advisories for freshwater fish, if anthropogenic mercury emissions were reduced.

The EPRI (2003) analysis also evaluated the impact of reducing power plant mercury emissions on fish methylmercury concentrations. The analysis assumed that a linear relationship existed between reductions in mercury deposition and methylmercury levels in fish. All U.S. freshwater fish were assigned to a single model compartment and all commercial fish were assigned to a separate model compartment. Methylmercury concentrations in freshwater fish were assumed to be in equilibrium with mercury deposition across the contiguous U.S. and methylmercury concentrations in commercial fish were assumed to be in equilibrium with global mercury deposition. Decreases in mercury deposition that resulted from decreased power plant emissions were assumed to result in linear and proportional reductions in fish methylmercury concentrations. In the EPRI analysis of the CSI, U.S. exposures to methylmercury from fish consumption were predicted to change very little from current exposures.

1.3. HUMAN EXPOSURE TO METHYLMERCURY

Humans are exposed to methylmercury primarily through fish consumption (i.e., eating the contaminated fish muscle). Schober et al. (2003) and Mahaffey et al. (2004)

showed that individuals reporting some fish consumption have higher levels of blood mercury and blood methylmercury, respectively, than those who report no fish consumption. Self-reported number of fish meals is a strong predictor of blood mercury (Schober et al., 2003) and methylmercury concentrations (Mahaffey et al., 2004). The magnitude of human methylmercury exposures depends on the fish methylmercury levels and the quantity of fish consumed.

Fish consumed in the U.S. are obtained from two general sources: the U.S. commercial seafood market and non-commercial capture by individuals for personal consumption or consumption by others known to them (e.g., family members or friends). Between 70% and 90% of the U.S. population (roughly, 200 to 250 million individuals) consumes seafood from the commercial market (Carrington and Bolger, 2002). In the U.S., this market is comprised of farmed fish and wild fish that have been caught off the U.S. coasts or imported from countries around the world.

Three types of seafood, tuna, shrimp and pollock account for over 45% of the mass of fish purchased by U.S. consumers and Table 2 lists the 24 types of seafood, which combined account for 92% of the marketed fish (Carrington and Bolger, 2002). Based on Carrington and Bolger's input data, 22 of the top 24 types of fish in commerce are wild and captured in marine waters. Commercial catfish and crawfish, the two freshwater species listed among the top 24 species, are typically raised in aquaculture. In general, commercial fish consumers are unaware of the origins of the fish they consume. For example, consumers likely do not know the country from which their canned tuna was imported. They also may be unaware of the types of seafood they consume; for example, they may not know the types of fish comprising "fish sticks."

Most marine fish inhabit upwelling regions of the oceans, nutrient rich estuarine waters, or continental shelves (Ryther, 1969). Policy makers concerned about the safety of commercial seafood obtained from marine environments must consider the influence of U.S. mercury emissions on methylmercury concentrations in the food webs of the commercial seafood stocks living in these areas of the ocean. The continental shelf adjacent to the North American continent under the Atlantic Ocean is one such area of concern; the Gulf of Mexico is another.

In the U.S., the Food and Drug Administration (FDA) monitors methylmercury levels in commercial seafood. Methylmercury concentrations are typically highest in the wild finfish such as shark and swordfish (Figure 2). Carrington and Bolger (2002) developed an exposure model, based on the frequency, quantity and types of seafood consumed. The model predicts changes in methylmercury intake in the general population associated with changes in fish consumption patterns. Table 2 lists *per capita* consumption rates and mean methylmercury concentrations for the commercial fish typically consumed in the U.S. (adapted from Carrington and Bolger, 2002). Concentrations in fish raised in aquaculture are typically low; these animals are not part of the wild food web where methylmercury can accumulate.

Fish meal is a dietary component of some fish raised in aquaculture (fish-eating species raised in aquaculture include salmon and cod). Lower trophic level marine species comprise these fish meals.[12] Shellfish are low trophic level organisms and, in general, have low levels of methylmercury. While many marine species only inhabit and feed in one location, three important commercial fish, tuna, shark and swordfish, are migratory. Thus, the methylmercury concentrations of these predatory fish may reflect participation in marine food webs at a number of different locations over time.

[12] Pauly et al. (1998) reported that, based on global fisheries statistics, the mean trophic level of fish caught has declined from 1950. The decline is a consequence of limited availability of higher trophic level species and an increase in the quantity of lower trophic level fish being targeted. Many of these species are used in fish meal and fish oils fed to some aquaculture species.

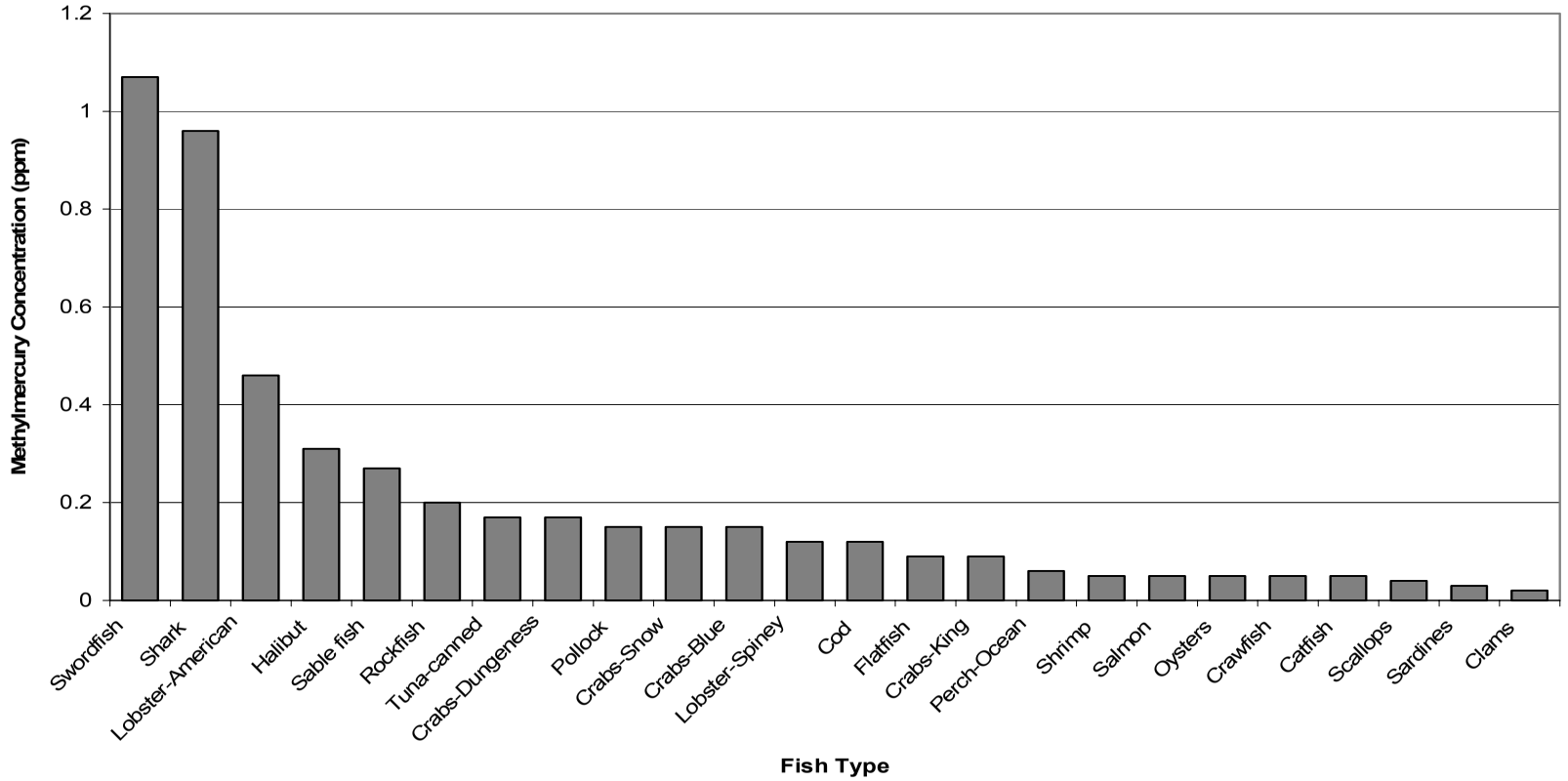

Figure 2

Average Methylmercury Concentrations for "Top 24" Types of Fish Consumed in the U.S. Commercial Seafood Market

Table 2. **The Pounds Per Capita, Methylmercury Concentration, Market Share, and Fractional Contribution of Fish in Commerce From Each U.S. Fishery or Import for Top 24 Types of Fish Consumed in U.S. (Sources: Carrington and Bolger, 2003; NMFS, 2002)**

Type	Annual Consumption Rate (Pounds per capita)	Arithmetic Mean MeHg Concentration (ppm)	Atlantic (%)	Gulf (%)	Pacific (%)	Import (%)
Tuna-canned*	3.1	0.17	migratory species			
Shrimp	2.7	0.05	1.1	10.3	2.5	86.2
Pollock	1.64	0.15	0.2	0.0	84.8	14.9
Salmon	1.299	0.05	0.0	0.0	41.7	58.2
Cod	1.057	0.12	2.2	0.0	30.5	67.4
Catfish	1.02	0.05	aquaculture			
Clams	0.46	0.02	84.0	0.4	1.8	13.9
Flatfish	0.33	0.09	9.4	0.1	41.8	48.8
Halibut	0.29	0.31	0.0	0.0	62.0	38.0
Scallops	0.25	0.04	49.5	0.0	0.3	50.3
Crabs-Blue	0.24	0.15	12.8	6.7	0.0	80.5
Oysters	0.22	0.05	4.8	34.9	15.0	45.3
Sardines	0.18	0.03	32.0	0.8	39.6	27.7
Rockfish	0.127	0.20	0.0	0.0	63.6	36.4
Crabs-Snow	0.092	0.15	0.0	0.0	0.0	100.0
Lobster-American	0.09	0.46	16.0	0.0	0.0	84.0
Lobster-Spiney	0.09	0.12	1.5	9.8	2.4	86.3
Swordfish	0.08	1.07	migratory species			
Crawfish	0.065	0.05	aquaculture			
Perch-Ocean	0.056	0.06	4.0	0.0	55.4	40.6
Crabs-Dungeness	0.054	0.17	0.0	0.0	99.8	0.2
Crabs-King	0.037	0.09	0.0	0.0	81.0	19.0
Sable fish	0.024	0.27	0.0	0.0	100.0	0.0
Shark	0.02	0.96	migratory species			

* Recent analyses have shown that canned albacore/white tuna have higher mean methylmercury concentrations (0.29 ppm) than light tuna (0.12 ppm) (FDA, 2004; www.cfsan.fda.gov).

Non-commercial consumers eat fish caught by individual anglers in saltwaters and freshwaters. Unlike the fish consumed from the commercial market, which may be caught in different locations, the fish eaten by non-commercial fish consumers are typically obtained from a small geographic area. Recent surveys by the National Marine Fisheries Service (NMFS, 2003) and the U.S. Fish and Wildlife Service (U.S. FWS, 2003) indicate that over 9 million people fish U.S. saltwaters (Table 3). Based on their National Survey of Fishing, Hunting, and Wildlife-Associated Recreation, U.S. FWS (2003) estimated that there were 28.4 million freshwater anglers. If the fish in a geographic area have high levels of methylmercury (for example, due to elevated mercury deposition rates associated with emissions from local or regional sources), then exposures in these fish consumers may be elevated. U.S. EPA policy makers are concerned about elevated methylmercury exposures from consumption of fish caught in these potential mercury "hot spots" in marine and freshwater environments.

Table 3. Estimates of the Size of the Saltwater Angler Population Annually in the U.S. and the Number of Consumers of Recreationally-Caught Saltwater Fish

Waters Fished	Population Size	Source	Estimated Number of Consumers Recreationally-caught Marine Fish
U.S. Saltwaters	9,051,000 10,577,000	U.S. FWS (for year 2001) NMFS (for year 2002)	--
Gulf of Mexico	3,138,000 2,655,000	U.S. FWS (for year 2001) NMFS (for year 2002)	5,793,000
Atlantic Ocean	4,766,000 5,258,000	U.S. FWS (for year 2001) NMFS (for year 2002)	10,024,000

Non-commercial fish caught in marine and estuarine waters along the U.S. coasts are monitored through the annual National Marine Recreational Fisheries Statistics Survey (NMFS, 2003). Their surveys ascertain the numbers, weights and types of fish caught recreationally. Tables 4 and 5 list the 10 most frequently captured fish, based on total mass of fish caught for consumption by recreational anglers in the Atlantic Ocean and in the Gulf of Mexico. The estimated total catch weights of these 10 types of fish comprise roughly 70% of the estimated total catch weight of all fish caught recreationally in the Atlantic and in the Gulf. The NMFS data are limited to finfish. U.S. EPA (2003a) has compiled methylmercury concentration data for marine species targeted for recreational capture.

Methylmercury concentrations in U.S. freshwater fish are compiled in the National Listing of Fish and Wildlife Advisories (NLFWA) Mercury Fish Tissue Database (U.S. EPA, 2003d). States voluntarily report information to this database, which contains approximately 58,000 samples reporting methylmercury concentrations. In addition to the methylmercury concentration, many of the entries include the type of fish, its length and weight. The U.S. FWS does not directly monitor the types of freshwater fish individuals consume, but conducts surveys to identify the types of fish anglers target and the amount of time they spend targeting specific fish.

Table 4. Median, Mean, and Maximum Methylmercury Concentrations (µg/g) Reported Fish Species Harvested via Recreational Angling in the Atlantic Ocean

Type	Median	Mean	Maximum	Number of Samples	Harvest (lbs)[a]
Striped Bass	0.1	0.15	0.8	215	12,919,000
Summer Flounder	0.03	0.04	0.1	34	12,523,000
Bluefish	0.35	0.4	1.6	174	12,334,000
Other Tunas/Mackerels[b]					8,135,000
Blackfin Tuna	1.16	1.16	1.2	1	
Cero Mackerel	0.15	0.19	0.3	3	
Dolphins	0.06	0.07	0.2	14	7,676,000
Atlantic Croaker	0.06	0.09	0.6	58	7,913,000
King Mackerel	0.67	0.98	3.5	118	4,789,000
Weakfish	0.2	0.27	0.8	61	4,045,000
Black Sea Bass	0.15	0.15	0.2	2	1,514,000
Scup	0.03	0.03	0.1	10	875,000
Subtotal (for 10 species listed)					72,721,000
Total Recreational Catch					105,215,000

[a] NMFS (1998) Data

[b] Note that we divided the estimated harvest weight for the category of other tunas and cero mackerels evenly between the two types of fish.

Table 5. Median, Mean, and Maximum Methylmercury Concentrations (µg/g) Reported Fish Species Harvested via Recreational Angling in the Gulf of Mexico

Type	Median	Mean	Max	# Samples	Harvest (lbs)*
Red Drum	0.19	0.5	4.62	590	8,522,000
Spotted Seatrout	0.28	0.32	1.5	546	8,256,000
Red Snapper	0.11	0.09	0.16	13	4,259,000
Dolphins	0.06	0.13	0.49	29	4,246,000
Groupers (myctera)	0.29	0.37	1.4	94	4,146,000
King Mackerel	0.86	1.09	4.47	385	3,933,000
Sheepshead	0.12	0.18	1.73	224	3,471,000
Black Drum	0.15	0.44	6.62	233	2,146,000
Spanish Mackerel	0.47	0.53	2.9	204	1,910,000
Sand Seatrout	0.45	0.48	1.2	99	1,815,000
Subtotal (for 10 species listed)					42,705,000
Total Recreational Catch					62,548,000

*Source of marine recreational catches: NMFS, 1998.

Fish consumption rate data have been collected by several U.S. federal agencies depending on the source of the fish. The FDA has evaluated commercial fish consumption rates (Carrington and Bolger, 2002). Table 2 reports per capita consumption rates for selected types of commercial fish based on FDA dietary surveys of the U.S. population. Based on an extensive analysis of NMFS survey data, U.S. EPA (1997a) estimated the distribution of finfish consumption rates for Atlantic and Gulf recreational anglers. U.S. EPA (1997a) also reports the distribution of freshwater fish consumption rates for U.S. recreational anglers. These values were based on studies of freshwater anglers fishing in the states of Maine, New York and Michigan (U.S. EPA, 1997a citing Ebert et al., 1993; Connely et al., 1996; West et al., 1989). Some individuals and groups catch and consume large quantities of non-commercial fish. For

example, the Columbia River Inter-Tribal Fish Commission Report (1994) describes consumption rates among a group of Native Americans who frequently consume large quantities of fish.[13] Policy makers are concerned about methylmercury exposures in individuals who either consume high quantities of commercial and non-commercial fish or consume contaminated fish.

1.4. TOXICOKINETICS

Once ingested, roughly 95% of the methylmercury entering the gastrointestinal tract is absorbed. This compound passes through the lining of the gut to the liver and enters the blood stream where it primarily binds red blood cell proteins, quickly distributing from blood to the liver, as well as the kidney and skin. Methylmercury appears to pass the adult blood brain barrier by binding to a thiol group on cystiene, which is then recognized by a neutral amino acid carrier protein (Aschner, 1989); entry into the brain tissues can lead to adult neurotoxicity. In pregnant females, methylmercury can also bind and pass through the placenta tissues; it may also enter placental tissues through an active transport process via an amino acid carrier protein. Stern and Smith (2003) report that methylmercury levels in cord blood are higher than levels in maternal blood; the reason(s) for this observation have not been fully explained. The blood brain barrier does not effectively occlude methylmercury from the brain tissue of the fetus. Entry into these brain tissues likely leads to the neurocognitive deficits observed in some children exposed to methylmercury. The possible

[13] While described as a study of "subsistence" angler consumption (U.S. EPA, 1997b), there is not a specific intake quantity or percent of total daily caloric intake or protein intake that results in a categorization of "subsistence."

cardiovascular effects associated with increased methylmercury exposures may result from the oxidative properties of mercury in the bloodstream (Salonen et al., 2000).

The mean half-life of methylmercury in humans is estimated to be between 47 and 72 days. Methylmercury is demethylated and converted back to inorganic forms primarily by gut microflora; the liver and brain also demethylate methylmercury. Demethylation in the brain may, in effect, trap inorganic mercury in the brain, because inorganic mercury passes out of the adult brain more slowly than it enters (if it passes out at all). It is unlikely that the fetus can demethylate methylmercury (Dock et al., 1994). Nearly all of the ingested methylmercury (90%) is eventually eliminated as inorganic mercury through the feces via the bile and exfoliated gastrointestinal epithelial cells. Methylmercury in the bile can be reabsorbed by the gall bladder and the small intestine; this apparent recycling is a factor in the long half-life of methylmercury in the body. Methylmercury also appears to bind sulfhydral groups in amino acids that comprise hair (Farris et al., 1993; U.S. EPA, 1997e). The binding of methylmercury to hair is an additional elimination pathway from the body.

While some researchers have used blood sera and toenails to analyze mercury exposure, measures of mercury concentrations in whole blood and hair are the most common biomarkers used in analyses of human exposures (Grandjean et al., 1999). Blood and hair mercury concentrations can be used to estimate the quantity of methylmercury ingested through the “methylmercury 1-compartment model.” This model provides reasonable approximations of intake, blood, and hair mercury levels under steady-state conditions.

$$d = c * b * v / (a * f * Bw) \quad \text{(Eq. 1)}$$

where:

d = oral dose (µg MeHg/kg-day)

c = blood concentration (µg/L)

b = elimination constant (0.014 day^{-1})

v = blood volume (5 L)

a = gastrointestinal absorption factor (0.95)

f = fraction of absorbed dose found in blood (0.059)

Bw = body weight (kg)

The parameter values above were obtained from U.S. EPA (1997e, 2001a, 2001c). The ratio of hair mercury concentration to blood mercury concentration is typically considered to be approximately 250:1 (e.g., 8 µg/L blood = 2 ppm hair) (U.S. EPA, 2001c). The mercury hair-to-blood ratio appears to be highly variable across individuals and studies.

1.5. HUMAN HEALTH EFFECTS

This discussion of mercury health effects focuses on neurological effects[14] observed in children that result from intrauterine methylmercury exposures and adult myocardial effects associated with methylmercury exposures through fish consumption. The risks of incurring these two effects are quantified in Section 3 of this report and they likely account for a large fraction of the total damage to humans that is associated with

[14] In laboratory animal studies, ingested methylmercury binds placental tissue and diffuses across these membranes, entering the fetal blood (U.S. EPA, 1997d). Methylmercury may also be actively transported across placental membranes by binding to cystiene. Methylmercury causes diffuse damage throughout the entire developing brain. When compared to normal brains, affected brains weigh less, have fewer cells, and exhibit a less organized structure (Burbacher et al., 1990). This systematic damage may result from brain cell death.

methylmercury exposures. We also discuss two additional effects that have been observed in children and associated with intrauterine methylmercury exposures, increased blood pressure and decreased heart rate variability. We do not quantify these risks, because the increased blood pressure does not appear to persist and the clinical significance of changes in heart rate variability of otherwise healthy children is not known.

The following reported effects associated with intrauterine methylmercury exposures and several reported effects associated with adult methylmercury exposures are also not addressed in this report.

- Delays in auditory brainstem evoked potentials that were observed in Faroese children at ages 7 and 14 years and associated with intrauterine methylmercury exposures are not evaluated because the clinical significance of such small delays in these evoked potentials is unknown (Murata et al., 1999, 2004).
- Intrauterine methylmercury exposures have also been associated with decreases in muscle tone and reflexes in 2-week-old children (McKeown-Eyssen et al., 1983), and with decreased neurological optimality scores in male offspring at ages 12 and 30 months (Steurwald et al., 2000). These possible effects of intrauterine methylmercury exposures are not addressed due to unknown significance of the endpoints in young children (e.g., does the effect persist? does it cause a significant decrease in the quality of life?).

- Cordier et al. (2002) reported that decreased leg coordination in males between the ages of 5 and 12 years and decreases on a Copying Test, which evaluates reasoning and visuospatial organization, in children ages 5 to 7 years was associated with intrauterine methylmercury exposures. We did not model the leg coordination because the significance of such decreases is unknown. We did not evaluate the Copying Test results because they would be a component of IQ decreases which are evaluated (Section 1.5.1) and because they did not appear to persist.

- Adult neurological effects possibly associated with methylmercury exposures through fish consumption have been reported. These include reduced motor control (Mergler and Dolbec, 1998) and reduced function on tests of fine motor speed and dexterity (Yokoo et al., 2003) and on tests of verbal memory (Yokoo et al., 2003), difficulty with accuracy and

sharpness of visual fixation and pursuit in dynamic eye movements (Beuter and Edwards, 2003) and restricted visual fields (Mergler and Dolbec, 1998). These effects were not modeled because of their unknown economic significance and impacts on individuals' quality of life.

1.5.1. Neurological Decrements Associated with Intrauterine Methylmercury Exposures.

The need for increased scientific understanding of the relationship between exposures to organic forms of mercury and human neurological decrements was initially identified through epidemiologic investigations of poisoning episodes in Japan (Harada, 1995) and Iraq (Bakir et al., 1973; Amin-Zaki et al., 1974). The victims were poisoned by methylmercury through consumption of contaminated fish in Japan and by an organic mercury compound through consumption of treated seed grain in Iraq (summarized in Clarkson, 2002). Exposures to these high levels of mercury led to severe neurological effects in adults, children and fetuses; however, the neurotoxicity that was associated with intrauterine exposures (i.e., severe cerebral palsy, delayed walking and talking) occurred at lower doses than the doses that resulted in adult effects (summary discussions in NRC, 2000; U.S. EPA, 2001b,c). For example, asymptomatic mothers (i.e., mothers in whom no neurological effects were observed) bore affected children indicating that the developing fetal nervous system was more vulnerable to methylmercury than the mature maternal nervous system.

While the effects associated with exposures to environmental levels of methylmercury in fish continue to be the subject of intensive scientific investigation and there have been additional epidemiologic studies of such exposures (U.S. EPA, 2001b), epidemiologic studies conducted in New Zealand, the Seychelle Islands and the Faroe Islands have been the subject of most recent scientific interest. All three studies

evaluated maternal methylmercury exposures during pregnancy and have examined the relationship between these exposure measures and pediatric neurological test results in the mothers' children during multiple developmental stages. The National Research Council (NRC, 2000), which evaluated all three studies, reported that none of the studies "appear to have serious flaws." Two of the studies, the Faroe Islands and New Zealand studies, showed an association between elevated *in utero* methylmercury exposures and adverse neurological outcomes in the offspring (e.g., IQ decreases), while the Seychelle Islands study has not reported such associations. At present, no adequate explanation has been offered for the differences in the results between the Faroe Islands and New Zealand studies and those found in the Seychelle (NRC, 2000).

Kjellstrom et al. (1986, 1989) conducted a prospective case-control study in New Zealand to examine the neurological development of children who had elevated intrauterine methylmercury exposures. From the 10,930 mother-child pairs enrolled in the cohort, 935 mothers who reported eating more than three fish meals per week while pregnant were identified. The fetal exposure measure was the mercury level in maternal hair over the entire pregnancy. The high-exposure group (n=31 children) was defined to be women with hair mercury levels greater than 6 mg/kg (or ppm) hair. Each child in this high exposure group was matched with a reference child based on maternal ethnicity, the child's age and birth hospital. The average maternal hair mercury concentrations in the high-exposure and reference groups were 8.8 ppm and 1.9 ppm, respectively. At 4 years of age, the children were administered a group of tests (Table 6). Based on the Denver Developmental Screening Tests (DDST) results, a statistically significant difference existed between the neurological functions of the high-exposure

and reference group. Developmental delays (primarily in the motor and language domains) were observed in 52% (16/31) of the children in the high-exposure group and in 17% (5/30) of the reference group.

The second phase of the study, undertaken when the children in the cohort were 6 years of age, employed additional neuropsychological and scholastic tests (Table 6) (Kjelstrom et al., 1989). Based on the child's gender, and the mother's age, ethnicity, residence, smoking status, and residence time in New Zealand, each child (n=61) in the high-exposure group was matched with three children whose mothers had lower hair mercury concentrations during their pregnancies. The average maternal hair mercury concentrations during pregnancy in members of the two control groups that reported frequent fish consumption were less than 3 ppm and between 3 and 6 ppm. A statistically significant association was reported between high maternal mercury exposure and decreased test scores on the McCarthy Scale of Children's Abilities in the perceptual and motor domains. Results of the linear multiple regression analyses showed significant associations between high maternal mercury exposure and decreased performance on three neurological tests. Kjellstrom and collaborators reported no differences in the two groups based on “observed” behavior (i.e., in the absence of sophisticated neurological testing).

Table 6. Tests Employed by Kjellstrom et al. (1986, 1989) in the New Zealand Studies

Developmental Domain	4 Years of Age	6 Years of Age
Vision	vision test	
Sensory	sensory test	
Academic attainment		Clay Diagnostic Survey Concepts Letter Test Word Test Reading Accuracy
		Burt Word Recognition Test Age Equivalent Score
		Key Mathematical test Grade Score
Language Development	Denver Developmental Screening Tests (DDST)	Test of Language Development (TOLD) Grammar completion Grammar understanding Oral Vocabulary Picture Vocabulary Sentence Imitation Spoken Language Quotient (TOLD-SL)
		Peabody Percentile Rank Standard Score Stanine
Motor Coordination	DDST-gross and fine	McCarthy Scales Motoric (MCC-MOT)
Intelligence		McCarthy Scales Verbal Quantitative Memory General Cognitive
		Wechsler Intelligence Scale for Children-Revised Verbal IQ Performance IQ Full scale IQ
Visuospatial/ Visuomotor		McCarthy Scales Perceptual (MCC-PP)
Personal-social	DDST	

Crump et al. (1998) reanalyzed the Kjellstrom et al. (1989) study conducting

Multiple regression analyses on the reported average maternal hair mercury concentrations during pregnancy as a continuous variable (i.e., unlike Kjellstrom et al. they used the reported values of hair mercury concentrations in their analyses rather than as a categorical variable as Kjellstrom had done). They did not identify a statistically significant relationship between maternal hair mercury levels and the neuropsychological test scores unless they omitted the single subject whose mother had the highest average maternal hair mercury concentration in the cohort, which was over four times higher than the second highest measurement. The maternal hair mercury variable was a statistically significant predictive variable in six tests. The regression coefficients for five of the tests are shown in Table 7.

Table 7. Regression Coefficients and 95% Confidence Intervals for Hair Mercury Concentrations (ppm) Calculated by Crump et al. (1998)

	Test of Language Development - Spoken Language Quotient	Wechsler Intelligence Scale for Children-Revised Performance	Wechsler Intelligence Scale for Children-Revised Full scale IQ	McCarthy Scales Perceptual	McCarthy Scales Motoric
1st Regression Analysis [a,b]	-0.60 (-1.2,-0.03)	-0.54 (-0.45,0.21)	-0.53 (-1.1,0.069)	-0.53 (-0.95,-0.11)	-0.01 (-0.02,0.003)
2nd Regression Analysis [a,b,c]	-0.42 (-0.98,0.13)	-0.47 (-1.1,0.16)	-0.42 (-1.1,0.18)	-0.50 (-0.92,-0.08)	-0.01 (-0.02,0.002)

[a] Omitted maternal-infant pair with highest maternal hair mercury level
[b] Statistically controlled for smoking, alcohol intake, social class, birth weight, maternal age, breastfeeding, gender, ethnicity, residence, residence time in New Zealand, and other siblings.
[c] Statistically controlled for age of child at testing and parental education levels

The Seychelles Child Development Study (SCDS) examined the effects of low-dose fetal exposure to methylmercury from maternal fish consumption. The main prospective study consisted of a cohort of 740 mother-infant pairs that were selected between 1989 and 1990. Average maternal hair mercury levels over the entire pregnancy (range 0.5 to 26.7 ppm; median = 5.9 ppm) were used as the marker of fetal mercury exposure. The cohort has been evaluated at 6.5, 19, 29, and 66 months of age (Marsh et al., 1995; Myers et al., 1995) and 9 years of age (Myers et al., 2003). The main prospective study reports no statistically significant associations between environmental prenatal methylmercury exposures (primarily through maternal saltwater fish consumption) and adverse neurological outcomes in Seychellois children over a 9-year period (Myers et al., 2000, 2003; Axtell et al., 2000).[15] Myers et al. (2003) reported an association with decreased performance in the peg board test with males, but noted that, given the total number of tests, this association could have been by chance alone. Table 8 indicates the developmental domain tested and age and type of tests conducted in the SCDS. Based on the results of these studies, it is plausible that *in utero* exposures to environmental concentrations of methylmercury through fish consumption are not associated with neurological decrements. Given the large sample size in the main Seychelles cohort, the SCDS appears to have sufficient statistical power to detect neurological effects and reject the null hypothesis. However, the NAS (2000) report cautions that a large epidemiologic study, such as the SCDS, may lack adequate power to detect adverse associations if a relatively small number of subjects

[15] Myers et al. (1995) noted a statistically significant association between *in utero* mercury exposure and the DDST-R (when the abnormal and questionable scores were combined); however, this association was observed in the pilot study only.

are exposed at levels where effects are likely to be found (i.e., the upper end of the exposure distribution).[16]

Table 8. Tests Employed in the Seychelles Islands Child Development Study

Developmental Domain	Age of Child (months)				
	6.5	19	29	66	168
Marsh et al. (1995)					
Global-cognitive	DDST-R	BSID, MDI	BSID, MDI	MSCA, GCI	
Visual-perceptive		Kohen-Raz	Kohen-Raz	Bender-Gestalt, MSCA Perceptual	
Speech language	DDST-R			MSCA Verbal PLS Total Language Aud. Comprehension Verbal Ability	
Memory	Fagen Infantest			MSCA Memory	
Visual Attention	Fagen Infantest				
Neuromotor exam	Neurological DDST-R	BSID PDI	BSID PDI	Bender-Gestalt MSCA Motor	
Behavioral	DDST-R		BSID IBR	CBCL	
Learning-achievement				Woodcock-Johnson	

[16] The NAS report states "power analyses that are based on total sample size can be misleading if adverse effects occur primarily among the most heavily exposed children, who typically comprise a very small proportion of the sample. Although the sample size of 700 children in the SCDS would seem to be more than adequate, only about 35 children were exposed at 15 µg/g or higher. Because multiple regression analysis examines associations that are averaged across the entire distribution of exposure, associations that hold only for the most highly exposed children can be difficult to detect. Thus, if adverse effects of prenatal MeHg exposure occur primarily in the upper range, the power to detect them will be limited…"

Developmental Domain	Age of Child (months)				
	6.5	19	29	66	168
Auditory response				Audiometry Tympanometry	
Davidson et al. (1998)					
Global-cognitive				MSCA, GCI	
Visual-perceptive				Bender-Gestalt	
Speech-language				PLS Total Score	
Behavioral				CBCL	
Learning-achievement				Woodcock-Johnson Letter and Word Recognition, Applied Problems	
Myers et al. (2003)					
Global-cognitive					WISC-FSIQ
Speech-language					BNT
Memory					visual memory subtest of the wide-range assessment of memory and learning
Sustained Attention					Connors Continuous Performance Test
Behavioral					Connors Teacher

Developmental Domain	Age of Child (months)				
	6.5	19	29	66	168
					rating scale, parent-child behavior checklist
Learning-achievement					Woodcock-Johnson Letter and Word Recognition, Applied Problems CVLT
Motor functions					finger-tapping, trail making, grooved peg board, Bruininks-Oseretsky,
Visual motor integration					Beery-Buktenica, test of haptic matching

Adapted from: U.S. EPA, 2000
Symbols and Abbreviations: BSID = Bailey Scales of Infant Development; IBR = Infant Behavior Record; MDI = Mental Developmental Index; PDI = Psychomotor Developmental Index; CBCL = Child Behavior Checklist; DDST-R = Denver Developmental Screening Test - Revised; GCI = General Cognitive Index; MSCA = McCarthy Scales of Children's Abilities; PLS = Preschool Language Scale. WISC-FSIQ= Wechsler intelligence scale for children- full scale IQ, BNT= Boston naming test, CVLT= California Verbal Learning Test
Sources: Marsh et al., 1995; Davidson et al., 1998; Myers et al., 2003

A longitudinal prospective study was conducted in the Faroe Islands to evaluate the relationship between maternal methylmercury exposure through seafood consumption during pregnancy (including episodic pilot whale consumption) and neurodevelopment in the offspring (Grandjean et al., 1995, 1997, 1998, 1999, 2003; Dahl et al., 1996; Murata et al., 1999, 2004). Following a pilot study (Grandjean et al., 1992), a cohort of 1022 singleton births was assembled during a 21-month period

between 1986 and 1987. The exposure measures relevant to intrauterine exposures were maternal hair mercury concentrations and cord blood mercury concentrations at childbirth. In the main study (Grandjean et al., 1997), the geometric mean cord blood concentration was 22.8 µg/L (interquartile range 13.4-41.3 µg/L) and the geometric mean maternal hair mercury concentration was 4.27 µg/g (interquartile range 2.6-7.7 µg/g). No mercury-related abnormalities were identified during clinical examination of the cohort. Intrauterine methylmercury exposures were statistically significantly associated with decreased scores on a number of neurological tests in the children. Table 9 indicates the developmental domain tested, subjects' ages, and type of tests conducted in the Faroe Islands study. Grandjean et al. (1997) conducted multiple regression analyses on the cord blood mercury concentrations. The cord blood mercury concentration was a statistically significant predictive variable in six tests. The regression coefficients for five neuropsychological tests appear in Table 10; the coefficients were calculated for those children born to mothers whose hair mercury concentrations were less than 10 ppm.

In summary, NRC (2000) and, more recently, a subset of the committee (Stern et al., 2004) have concluded that, based on evidence from two of the three studies, low dose *in utero* exposures to methylmercury likely lead to subtle but measurable neurological effects in children. Because the three studies used different test batteries, it is difficult to directly compare their results. In the future, there may be opportunities to evaluate confidence intervals associated with significant and non-significant test results at the level of developmental domains across the three studies (see first column in Tables 6, 8, and 9 for listing of the domains evaluated in the three studies). This type of

evaluation may provide alternative measures of consistencies between the three studies. Additional evaluations of potential confounding effects such as the intake of selenium are needed.

Table 9. Tests Employed in Studies of Faroese Children

Developmental Domain	Age of Child		
	12 months Grandjean et al. (1992)	7 years Grandjean et al. (1997) - Main Prospective Study; Grandjean et al. (1998) - Nested Case Control Study; Dahl et al. (1996); Murata et al. (1999)	14 years- Murata et al. (2004)
Developmental milestones	sitting creeping standing		
Motor coordination		Hand-Eye Coordination	
General cognitive		WISC-R Similarities	
Visuospatial		WISC-R Block Designs Bender Motor Visual Gestalt Test	
Attention		NES2 Continuous Performance WISC-R Digit Spans Forward	
Speech-language		Boston Naming Test	
Memory		California Verbal Learning Test	
Motor speed		NES2 Finger Tapping NES2 Hand-Eye Coordination NES2 Tactual Performance	
Personal-social		Nonverbal Analogue Profile of Mood States	
Neuropathological Abnormalities		Brain-stem auditory evoked potentials, visual-evoked potentials	Brain-stem auditory evoked potentials

Table 10. Coefficients for Logarithmic Transformation of Cord Blood Mercury Concentrations on Selected Neuropsychological Tests (only for mothers with hair mercury concentrations less than 10 ppm) (Grandjean et al., 1997)

Test	Regression Coefficient	p-value
Wechsler intelligence scale for children-Revised	-0.31	0.05
Bender Visual Motor Gestalt Test Reproduction	-0.43	0.02
Boston naming test, No cues	-1.42	0.01
Boston naming test with cues	-1.57	<0.01
California Verbal Learning Test-Children short term reproduction	-0.74	<0.01
Statistically controlled for age of child at testing, gender, maternal cognitive function as measure by scores on Raven's Progressive Matrices, major medical risk factors, smoking, alcohol intake, parental education levels, father's employment status, current residence, child's computer acquaintance, day care, and other siblings. NRC (2000) presents a summary table (Table 7-1) that provides estimates of the regression coefficients for all of the subjects.		

1.5.2. Myocardial Effects Associated with Adult Methylmercury Exposures.

In this section, we discuss adult myocardial effects associated with methylmercury exposures (also, see recent review by Stern, 2004). While scientific panels have evaluated the methylmercury neurologic studies as a group, similar panels have not evaluated the group of studies that have examined the relationship between adult methylmercury intakes and myocardial effects. Studies conducted by Salonen et al. (1995, 2000), Rissanen et al. (2000), and Guallar et al. (2002) suggest that methylmercury exposures may be a risk factor for myocardial events and premature deaths. Studies by Tamashiro et al. (1986), Ahlqwist et al. (1999) and Yoshizawa et al. (2002) did not observe such a relationship, indicating that it may not exist. We note that cardiovascular diseases have been associated with many other behavioral, genetic, and

dietary risk factors. Typically, in adult populations, the primary source of methylmercury exposure is consumption of contaminated fish and, in general, the consumption of fish and fish oils has been consistently associated with reduced risk of myocardial events (e.g., Kromhaut et al., 1985; Daviglus et al., 1997; Hu et al., 2002, see also summary by Bouzan et al., in preparation). For example, the study of Daviglus et al. (1997), which examined 1,822 U.S. males over 30 years, showed that, when compared to those reporting no fish consumption, individuals who reported consuming 35 or more grams of fish per day had roughly a 40% reduced risk of incurring a fatal myocardial event. The strength of this substantial body of evidence on the cardiovascular benefits of fish consumption has led to U.S. nutritional recommendations that individuals consume multiple fish meals per week (Kris-Etherton et al., 2002). However, this benefit is not observed in every study of fish intake (Curb and Reed, 1985; Vollset et al., 1985, Morris et al., 1982; Folsom and Demissie, 2004). It is plausible that fish contaminants such as methylmercury could attenuate the cardiovascular benefits of fish intake. That is, one explanation for the fish consumption studies that show no fish consumption-associated cardiovascular benefits or even adverse cardiovascular effects could be that the cardiotoxic effect of methylmercury offsets or exceeds the cardioprotective effect of the polyunsaturated fatty acids in fish. Of course, there may be other contaminants and other factors in these studies that lead to these observations. We note that these positive epidemiologic studies appear to be well conducted controlling for many known cardiovascular risk factors and the study results appear to be credible.

Next, we describe the four studies that have shown an association between adult methylmercury intakes and myocardial events. Based on the study by Salonen et al.

(1995), we develop dose-response relationships between adult methylmercury exposures, as measured by hair mercury concentrations and acute myocardial infarction (AMI) and all cause mortality (ACM)[17] in Section 2. We also describe the studies that do not report a statistically significant association between methylmercury and myocardial events.

A study of 1,833 males in Eastern Finland, aged 42-60 years, evaluated the relationship between methylmercury intake via fish consumption and the incidence of AMI and all cause mortality (Salonen et al., 1995). Upon study entry, the subjects were free of cancer, coronary heart disease, stroke and claudication. Mercury exposures were measured using hair and urine mercury concentrations. Hair mercury concentrations reflect long-term average mercury exposures (months) and urinary mercury concentrations are influenced strongly by concentrations of inorganic mercury in blood, which reflect short durations of exposure (e.g., the past 45 days). Among the study participants, mean fish intake was 46.5 g/day and the mean daily dietary intake of methylmercury was estimated to be 7.6 µg/day. The mean mercury concentration in hair was 1.92 ppm and the mean daily urinary excretion rate was 1.18 µg/day. Table 11 provides additional details of these measures. In this study, the mean and maximum follow-up periods were 5 years and 7.75 years for cardiac events and 6 years and 8.75 years for deaths, respectively.

Salonen et al. used Cox proportional hazards models to evaluate the relationship between hair mercury levels and AMI and premature death.

[17] All cause mortality (ACM) is an epidemiologic term that refers to all deaths occurring in a study cohort regardless of the cause of death.

They modeled hair mercury concentrations as a continuous variable and statistically controlled for a number of risk factors of myocardial events (Table 12 lists these for both Cox models used). We use the values estimated in the second model, (which controls for additional risk factors associated with myocardial events) and are more likely to estimate the isolated effects of methylmercury exposure than those estimated by the first model. The second model estimates that for each 1 ppm increase in hair mercury, the 5-year risk of non-fatal and fatal AMI increased by 6.8% and the 5-year risk of all cause mortality increased by 9.0% (Table 12, see results of the second set of Cox models). We note that the observed increase in AMI risks is not statistically significant (p=0.17).

Table 11. Measures of Cohort Methylmercury Intake Rates Reported in Salonen et al. (1995)

	Mean	Standard Deviation	Minimum	Maximum
Self-reported fish intake g/day	46.5	55.5	0	619
daily dietary intake of mercury µg/day	7.6	7.7	1.1	95
Hair mercury concentration ppm	1.92	1.98	0	16
Urinary excretion rate µg/day	1.18	1.1	0	5

Table 12. Results of Cox Proportional Hazards Models Reported by Salonen et al. (1995)

	Fatal and Nonfatal AMI			All Cause Mortality		
Model 1	RR	p value	95% CI	RR	p value	95% CI
Hair Mercury (ppm)	1.094	0.037	1.01, 1.19	1.132	0.001	1.05, 1.22
Hair Mercury (>2 ppm)	1.96	0.005	1.23, 3.13	2.26	0.001	1.43, 3.56
Statistically Controlled for age, exam year, ischemic exercise ECG, maximal oxygen uptake						
Model 2						
Hair Mercury (ppm)	1.068	0.175	0.97, 1.18	1.09	0.043	1.003, 1.186
Hair Mercury (>2 ppm)	1.69	0.038	1.03, 2.76	1.93	0.007	1.2, 3.10
Statistically Controlled for Same variables as Model 1 + family CHD history, smoking, systolic blood pressure, diabetes, socioeconomic status, residence, dietary iron intake, serum apolipoprotein B, HDL2 cholesterol, and ferritin concentrations						
The Cox proportional hazards model is described in Equation 6 in Section 2.5.1.						

Salonen et al. also reported an analysis that compared risks to subjects with hair mercury concentrations of 2.0 ppm or more with those that had less than 2.0 ppm. In the group with hair mercury concentrations of 2.0 ppm or more, the authors reported a 69% (p=0.038) greater risk of fatal or nonfatal AMI and a 93% greater (p=0.007) risk of premature death from all causes.

Based on an additional multivariate analysis of a small subset (n=187), the authors reported that mercury levels in the hair and urine were statistically significantly associated with immune complexes containing oxidized low density lipoprotein (LDL). These complexes are believed to be related to adverse myocardial events. The authors

posited that the increased incidence of myocardial events may be caused by oxidation of serum lipids. Of the variables analyzed, hair mercury levels were the strongest predictor of serum immune complexes containing oxidized LDL.[18]

Two follow-up studies (Salonen et al., 2000; Rissanen et al., 2000) were implemented to further analyze these results. The Salonen et al. (2000) study (n=1014) examined the association between the progression of arteriosclerosis, as measured by changes in carotid artery thickness over a 4-year period, and hair mercury concentration. Based on the results of a multivariate model, hair mercury concentration was a statistically significant predictor of the progression of common carotid artery thickening; based on this finding, the follow-up study further supported the possibility that methylmercury exposures may oxidize serum lipids.

The second follow-up study (n=1871) (Rissanen et al., 2000) evaluated whether hair mercury concentrations modified the relationship between acute myocardial events and two serum fatty acids derived from α-linoleic and linoleic fatty acids in fish, docosahexaenoic acid (DHA, also identified in the biochemical literature as 22:6 n-3) and docosapentaenoic acid (DPA, also identified in the biochemical literature as 22:5 n-6). Although evaluated, no significant associations were observed between a third fish-derived fatty acid, eicosapentaenoic acid, and the risk of acute coronary events. The mean hair mercury concentration increased with the proportion of the serum fatty

[18] Salonen et al. suggested three possible mechanisms that might explain the association between tissue methylmercury levels and increased incidence of myocardial events:
1. Mercury could catalyze the formation of free radicals that would lead to oxidized lipids.
2. Due to the affinity of mercury for sulhydrol groups, it could reduce the antioxidative capacity of the plasma by binding to proteins such as glutathiols.
3. When bound to mercury, selenium does not function as a cofactor in peroxide-scavenging enzymes.

acids that was comprised of DHA and DPA; this was expected because fish intake is the source of all three compounds.

To analyze the relationship between the two fatty acids and acute coronary events, the cohort was divided into quintiles based on the proportion of serum fatty acids comprised of DHA and DPA. Rissanen et al. used Cox proportional hazards models and statistically controlled for a number of factors related to cardiac events (Table 13). Men in the highest quintile of DHA and DPA had a 44% lower risk of acute coronary events (95% confidence interval 11%, 65%; p=0.014) when compared to subjects in the lowest quintile. To evaluate whether mercury exposure might modify the relationship between fish-derived serum acids and myocardial events, the data were stratified based on hair mercury content and a dichotomous analysis was developed that compared risks to subjects that had hair mercury concentrations of 2.0 ppm or more with those that had less than 2.0 ppm. Men in the highest quintile for serum DPA and DHA and the lower level of hair mercury content had a 67% (p= 0.016) reduced risk of acute coronary events, when compared to subjects in the lowest DHA and DPA quintiles having hair mercury levels above 2.0 ppm. Within each quintile, subjects with higher hair mercury concentrations had higher risks of acute coronary events, suggesting that mercury exposure may attenuate the cardioprotective effects of fish-derived fatty acids.

Table 13. Relative Risk of Acute Coronary Events in a Middle-Aged Finish Male Cohort Based on Serum Fatty Acid Composition, Stratified by Hair Mercury Levels (Rissanen, 2000)

	Quintiles, by Proportion of Serum Fatty Acids comprised of DHA and DPA				
Hair mercury concentration	<2.38%	2.38%-2.73%	2.74%-3.07%	3.08%-3.58%	>3.58%
< 2 ppm	0.85	0.50	0.48	0.41	0.33
> 2 ppm	1.00	0.83	0.63	0.76	0.76
Statistically Controlled for age, examination year, ischemic exercise ECG, maximal oxygen uptake, family CHD history, smoking, systolic blood pressure, diabetes, body mass index, socioeconomic status, serum insulin, ADP-induced platelet aggregation, residence, dietary iron intake, dietary energy intake, serum apolipoprotein B, HDL2 cholesterol, and ferritin concentrations.					

In a case-control study, Guallar et al. (2002) evaluated the association between toenail mercury concentrations, adipose tissue DHA concentrations, and AMI risk in men of no more than 70 years of age from nine different countries.[19] The relationship between toenail mercury levels, the biomarker of mercury exposure in this study, and blood mercury levels, although assumed to be related in the same manner that hair mercury concentrations are related to blood concentrations, is not known. The cases consisted of 684 men having first-time diagnosis of myocardial infarction confirmed by electrocardiology and blood enzyme analysis; the controls consisted of 724 age-matched males, with no history of myocardial infarction and from the same study center catchment areas as the cases. DHA and mercury concentrations were correlated (correlation coefficient = 0.34). Toenail mercury levels among the cases were 15%

[19] Cases and controls were obtained from each of 9 countries. The total number of cases and controls (i.e., the sum) from any one site numbered no more than 200. The countries from which cases and controls were obtained included Spain, Norway, Russia, Germany, Israel, the Netherlands, Finland, Switzerland and the United Kingdom.

higher than those of the controls after adjustment for DHA levels and coronary risk factors (95% confidence interval 5%, 25%); the case-control ratio was 1.10 (95% confidence interval 1.03, 1.18). After adjusting for mercury concentrations, DHA levels were inversely associated with risk of AMI; when highest and lowest DHA quintiles were compared, the odds ratio (OR) was 0.59 (95% confidence interval 0.3, 1.19; the trend test was statistically significant).

In a follow-up study of the Japanese population poisoned by methylmercury, Tamashiro et al. (1986) studied mortality patterns based on death certificates between 1970 and 1981 for individuals who resided in the section of Minamata that had the highest reported number of cases of Minamata disease.[20] The frequencies of heart disease in the study population were lower than those of the general Japanese population.

In a prospective cohort study, Ahlqwist et al. (1999) examined the association between mercury concentrations in blood sera and myocardial infarction, among 1,462 Swedish women 38 to 60 years of age at recruitment who were followed for up to 25 years. Serum mercury concentrations, which disproportionately reflect inorganic mercury exposures, were measured at cohort initiation. No association between serum mercury concentrations and myocardial infarctions was found.

In a prospective nested case-control study, Hallgren et al. (2001) examined the relationship between first time myocardial infarction and erythrocyte mercury concentrations. They reported an inverse association between the risk of a first

[20] Adequate death records were not maintained prior to 1970.

myocardial infarction and erythrocyte mercury concentrations and plasma polyunsaturated fatty acid concentrations.

Using a nested case-control design, Yoshizawa et al. (2002) studied the association between toenail mercury concentrations and risks of coronary heart disease in male health professionals aged 40 to 75 years. They excluded from the study subjects reporting myocardial infarction, angina, coronary artery by-pass surgery, stroke, diagnosis of cancer or having a very high or very low daily caloric intake. Based on several different analyses (e.g., odds ratios, multivariate regression), there was no statistically significant difference between the mercury levels of cases and controls. A separate multivariate analysis that excluded dentists (due to occupational exposure to inorganic mercury) compared the highest and lowest mercury quintiles. A relative risk (RR) of 1.27 (95% confidence interval 0.62, 2.59) was observed, suggesting an association of toenail mercury with risk of coronary heart disease, but the association was not statistically significant. The number of subjects included in this separate analysis was roughly half of the total in the main analysis.

In summary, this group of studies does not definitively show whether methylmercury exposures increase the risk of incurring adverse myocardial events. The evidence that supports the cardioprotective effects of fish consumption, in general, is the strongest. The questions are whether co-exposure to methylmercury attenuates the cardiovascular benefit of fish consumption and, if so, under what conditions and by how much. We have noted that some epidemiologic studies that evaluated the relationship between fish consumption and cardiovascular effects did not report a positive effect. When compared to the body of epidemiologic data indicating that fish consumption may

reduce the risk of myocardial events (e.g., Daviglus et al., 1997), the epidemiologic studies showing an association between methylmercury exposures and cardiovascular effects are comprised of a relatively small number of subjects and only three independent cohorts. Only one study evaluated cardiovascular disease and mercury exposures in women (Ahlqwist et al., 1999) and this study did not report a statistically significant association. The exposure measure used in this study may be inappropriate for inferring whether there is an association between methylmercury intake and fish consumption. Serum levels of mercury, which were used as a measure of exposure in this study, may be a biomarker for inorganic mercury exposures rather than exposures to methylmercury, which binds to red blood cells (Stern, 2004). The Tamashiro et al. study, which also did not report a statistically significant association, evaluated methylmercury poisoning, and the effects of the large methylmercury doses in these individuals may differ substantially from those exposed to typical environmental levels. Finally, the Yoshizawa et al. data showed a positive but not statistically significant association between toenail mercury concentrations and cardiovascular effects, if dentists were not included.

In Section 2, we use the regression coefficients of Salonen et al. to estimate methylmercury-related risks of AMI and ACM, because the studies appear well-conducted and the results appear to be credible. The studies that have evaluated the relationship between methylmercury exposures and adult cardiovascular effects have, to date, not been subjected as a group to such an independent evaluation. We are not as confident in our external generalization of the AMI and ACM data as we are in our external generalization of the neurotoxicity studies. The neurotoxicity studies have

been independently evaluated as a group. The studies reporting the cardiovascular benefits of fish consumption have also been subjected as a group to thorough re-evaluation.

1.5.3. Elevated Childhood Blood Pressure and Cardiac Rhythm Effects Associated with *In Utero* Methylmercury Exposures.

In this section, we review the results of the Sorensen et al. (1999) and Grandjean et al. (2004) studies. We also discuss human blood pressure reports from the methylmercury poisoning in Minamata, Japan and several relevant rodent bioassays. Although blood pressure increases in children can be detrimental to health, typically these are not as significant as effects in adults. We note that because the increased childhood blood pressure among children aged 7 years that was reported by Sorensen et al. (1999) did not persist (Grandjean et al., 2004), it is unlikely to be of clinical significance. Finally, we discuss the association of *in utero* methylmercury exposures with decreased heart rate variability. This association was first observed in a cohort of children aged 7 years (Sorensen et al., 1999) and was shown to persist in this cohort Grandjean et al. (2004) until 14 years of age. The clinical significance of this observation in children is not known.

1.5.3.1. Childhood Blood Pressure —

In the Faroe Islands birth cohort (Grandjean et al., 1995), Sorensen et al. (1999) reported statistically significant associations between elevated cord blood mercury levels or maternal hair mercury levels and increased diastolic and systolic blood pressures for 7-year-old children (n=917). Obstetrical charts and interviews were used

to obtain pregnancy and pertinent maternal information. A single measure of blood pressures was made for each 7-year-old child in the cohort standardized conditions.

A series of multiple regression analyses were conducted for maternal and child risk factors and elevated blood pressure. As anticipated, both increasing body weight (regression coefficient = 0.54 and 95% CL=0.4, 0.68) and increasing height (regression coefficient = 0.25 and 95% CL=0.15, 0.36) were statistically significantly associated with increases in diastolic blood pressure (DBP) and systolic blood pressure (SBP). Adjusting for body weight and maternal hypertension, the authors report that DBP increased on average by 13.9 mmHg (95% CL=7.4, 20.4) as mercury levels increased from 1 to 10 µg/L cord blood. SBP increased on average by 14.6 mmHg (95% CL=8.3, 20.8) over the same range of mercury levels in cord blood. These increases were observed only in the range of 1 to 10 µg/L; above 10 µg mercury/L cord blood, no additional increase in blood pressures was observed and the effect appeared to plateau.

The mean cord blood mercury concentration among all subjects was 32 µg/L and the standard deviation was 29 µg/L; the cord blood concentration was below 10 µg/L in 145 children. Birth weight was noted to be a possible effect modifier for the association between mercury levels in cord blood and blood pressure. When the association was evaluated in children below the median birth weight (3.7 kg), a cord blood mercury increase from 1 to 10 µg/L was associated with an average increase of 24.4 mmHg and 20.9 mmHg in DBP and SBP, respectively. Smaller increases in diastolic (6.7 mmHg) and systolic (9.6 mmHg) blood pressures were observed in the cohort whose birth weights were greater than the median.

Blood pressure increases were also associated with increasing maternal hair mercury concentrations. Increases from 0.2 to 2 μg/g in hair mercury concentrations resulted in a 3.6 mmHg (95% CL=4.3, 11.5) increase in DBP and SBP increased 8.6 mmHg (95% CL=0.9, 16.2). Potential maternal risk factors such as smoking, alcohol intake, age, weight and height and other potential child risk factors such as breast-feeding duration and parity did not appear to be significant predictors of the subjects' blood pressures. The authors also evaluated birth weight, placenta weight, and gestational age simultaneously and found that these coefficients were not statistically significant predictors for DBP or SBP. The authors then divided current body weight, birth weight and placenta weight into quartiles and found no statistically significant effects. Assessment of mercury exposure using a biomarker such as cord blood mercury levels is indicative of fetal exposures over a relatively short time period (between 1 and 2 months). Hair mercury concentrations integrate a broader period of exposure than cord blood measures (Grandjean et al., 1999).

Blood pressure data were collected again in this cohort at 14 years of age (Grandjean et al., 2004). At that age, no effect of intrauterine mercury exposure was observed on systolic or diastolic blood pressure. Shifts in childhood blood pressure, while potentially important, do not appear to be as consequential as those that persist into adulthood.

Other epidemiologic data describe associations between methylmercury exposures and hypertension. Tsubaki and Irukayama (1977) describe a retrospective epidemiological study designed to estimate the prevalence of Minamata disease, which was caused by exposures to high levels of methylmercury, among residents of three

Japanese cities around Minamata Bay. From 1932 to 1968 a factory polluted the bay with mercury leading to elevated concentrations of methylmercury in the fish consumed by the residents (e.g., marine products from Minamata Bay had 5.61 to 35.7 ppm mercury (Harada, 1995)). Residents in cities of Minamata (n=965), Goshonoura (n=1723) and Ine (n=608) were evaluated. Fish in the Minamata area were the most heavily contaminated by methylmercury; those in Goshonoura were considered moderately contaminated, and those in Ine were unlikely to be contaminated by the factory. This is supported by the reported frequencies of Minamata disease diagnosis in the three cities; in Minamata 28.5% of the individuals examined had Minamata disease, compared to 1.8% in Goshonoura, and 0.2% in Ine. Tables 14 and 15 identify the frequencies with which individuals with Minamata disease and those in the control group were diagnosed as hypertensive. In this study, the criteria for a diagnosis of hypertension were a SBP greater than 160 mm Hg or a DBP greater than 95 mmHg; these criteria are slightly higher than the current criteria for hypertension of 140/90 mmHg. The incidence of hypertension in residents of Minamata and Goshonoura who were not diagnosed with Minamata disease is not given. Discussion of the six individuals with congenital Minamata disease (born in Minamata between 1955 and 1962) did not identify hypertension as a “main symptom.” While the co-occurrence of hypertension and Minamata disease is interesting, these individuals experienced methylmercury exposures that were much greater than those occurring through fish consumption currently in the U.S. and in the Faroe Islands.

Table 14. Co-Occurrence of Minamata Disease Diagnoses and Diagnosis of Hypertension and the Occurrence of Hypertension in the Control Group from the City of Ine

Disease Status	Hypertensive/ Total Disease*	
	Minamata	Goshonoura
Minamata Disease	83/269 (31%)	19/34 (56%)
Suspected Minamata Disease	7/19 (37%)	12/31 (39%)
Deferred Diagnosis	5/15 (33%)	10/29 (34%)
	Hypertensive/ Total Evaluated	
No Disease (Residents of Ine)	109/608 (18%)	

*Data exclude congenital Minamata Cases

Table 15. Comparison of Hypertensive Diagnoses Between Those with and Without Minamata Disease in Two Different Age Categories

Minamata Disease Status/City/Age category	Hypertensive/Total Examined
Positive Diagnosis/Minamata/>39 Years old	79/214 (36.9%)
Positive Diagnosis/Minamata/<39 Years old	4/55 (7.3%)
Disease-Free/Ine/>39 Years old	107/378 (28.3%)
Disease-Free/Ine/<39 Years old	2/230 (0.9%)

In the section of Minamata that had the highest reported number of cases of Minamata disease, Tamashiro et al. (1986) studied mortality patterns based on death certificates between 1970 and 1981. The frequency at which the cause of death was listed as hypertension or hypertensive heart disease did not differ significantly from their comparison population that was not exposed to elevated mercury levels. As noted previously, the frequencies of heart disease and cerebral infarction in the study

population were lower than those of the general Japanese population but the frequency of cerebral hemorrhage, a sequela of elevated blood pressure, was significantly higher. Adequate death records were not maintained prior to 1970. Further analyses of the Minamata disease cases do not appear to address the incidence of hypertension (e.g., Fukuda et al., 1999).[21]

1.5.3.2. Childhood Cardiac Rhythms —

Sorensen et al. (1999) also reported that heart rate variability decreased with increased *in utero* methylmercury exposures. Specifically, in the children aged 7 years, the variation for the R-R interval was decreased (i.e., variability of cardiac electrical impulses was decreased).[22] At age 14 years, the heart rate variability was evaluated again in the same cohort (Grandjean et al., 2004). The autonomic nervous system controls heart rate function through both low frequency and high frequency activities.

[21] Two relevant studies that evaluated the effect of methylmercury on blood pressure in rodents were identified. Tamashiro et al. (1986) administered methylmercury (2 mg/kg-day) to a 7-week-old spontaneously hypertensive strain of rats for 26 days (n=10 males and 10 females). Relative to untreated controls, the SBP of the treated females was significantly higher during weeks 3 and 5 of the 5 week experiment. The blood pressures in the treated males were lower than controls during weeks 3 and 4, but only 2 males survived through week 4 and no males survived week 5. Wakita (1987) administered 0.5 mg/kg methylmercuric chloride via oral gavage to four groups of male Wistar rats (age and exact number of animals (likely n < 8) in each group is unknown) for 23 to 28 days (total dose = 11.5-14 mg/kg), 120 days (total dose = 60 mg/kg), 180 to 210 days (total dose = 90-105 mg/kg), or 220 to 240 days (total dose = 110 to 120 mg/kg). SBP of the treated animals did not differ from controls (n=9) until after day 60. After this point the SBP of all treated rats were statically significantly elevated above those in the control group. The last blood pressure measurement of the low dose group occurred at 120 days. Blood pressures were measured to approximately 450 days in the other dose groups and the controls; SBP in these groups were also statistically significantly higher than the controls. Small numbers of experimental animals, short study durations, high doses, and inadequate data reporting render further interpretation of the relationship of these studies to the association reported by Sorensen difficult. A series of chronic methylmercuric chloride feeding studies did not report blood pressure measures in the treated animals (Mitsumori et al., 1983, 1984, 1990). In conclusion, the bioassay data, like the data from the Minamata disease cases, suggest that high levels of methylmercury may be associated with blood pressure increases, but neither body of data directly address the issue of fetal methylmercury exposures.

[22] Each heartbeat is a discrete event affected by both sympathetic and parasympathetic influences. When the heart beats, electrical impulses are discharged. Electrocardiography can be used to record these electrical discharges as a series of defined waves. One heart beat consists of a P wave, a QRS wave, a T wave, and finally a U wave. The R-R wave intervals are used to define the period of time between sequential heart beats.

Both low frequency and high frequency activities decreased with increasing intrauterine exposures in a statistically significant manner. Heart rate variability also declined with increasing exposures in a statistically significant manner. While there is clinical significance to these declines in heart rate variability among the elderly and those who have suffered a heart attack, the clinical significance of such decreases in apparently healthy children remains an active research area in pediatric cardiology but is unknown at this time. Given that the decreased heart rate variability persisted until age 14 years, and that this effect is prognostic of sudden death from arrhythmia in adults, additional research into this possible effect of intrauterine methylmercury is warranted.

1.6. REGULATION AND CONTROL OF MERCURY EMISSIONS FROM POWER PLANTS

The proposed CSI, a bill introduced in February 2003 in the U.S. House of Representatives (HR 999) and the U.S. Senate (S. 485), would create a mandatory program that would reduce power plant emissions of mercury, sulfur dioxide and nitrogen oxides. Under Section 473 of this proposal, mercury emissions from the U.S. coal-fired power plants would be reduced in two steps. In the years 2010 and 2018, annual mercury emissions from power plants would be capped under a national market-based "cap and trade" approach. The U.S. power plants subjected to CSI are estimated to currently emit 49 tons of mercury per year; under CSI they would face a cap of 26 and 15 tons per year in 2010 and 2018, respectively (U.S. EPA, 2003a). Other bills that would reduce mercury emissions have been introduced to the US Congress and EPA is considering regulations under the Clean Air Act as well.[23]

[23] As we note in the Introduction, CSI has been reintroduced in the U.S. Congress in 2005 with a major change in mercury control requirements: the first-phase cap in 2010 now is 34 TPY instead of 26 TPY,

1.7. PURPOSE OF REPORT

Previous studies that evaluated the impact of reducing mercury emissions from power plants either did not evaluate the potentially resultant changes in methylmercury exposure in the U.S. population (U.S. EPA, 2003b) or, if exposure was evaluated, were unable to adequately evaluate differences in sources of fish in U.S. diets (EPRI, 2003). Our goals were to characterize the impact that reductions in mercury emissions from power plants would have on methylmercury exposures in the U.S. population and to estimate quantitatively the economic benefit of the plausible improvements in the health of the U.S. population.

1.8. OVERVIEW OF ANALYSIS

We investigate five U.S. mercury emissions scenarios. The first scenario is based on current mercury emissions, two scenarios are based on U.S. EPA projections of changes in mercury emissions that are projected to occur in 2010 and 2020 under current U.S. regulations (taking into account factors such as economic growth), and the final two are based on likely emissions if the CSI passes into law.[24]

To estimate the change in exposure, we identify eight geographic regions that are sources of consumable fish and estimate the quantity of fish from each region that is consumed, noting the current population exposures and the size of the consuming

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the second-phase cap remains at 15 TPY in 2018. The analysis in this report, however, is based on the 2003 version of the CSI.

[24] Specifically, the geographic distribution and total mercury emissions, as predicted by U.S. EPA's application of the Integrated Planning Model, were used as inputs to the REMSAD model. U.S. EPA simulated mercury deposition using the REMSAD model. The Agency used this model to predict current atmospheric deposition levels and to project mercury deposition levels corresponding to the two future "base case" (without CSI controls) and "control case" (with CSI controls) scenarios for the years 2010 and

population. The impact of decreased mercury emissions from power plants on mercury deposition in these regions is estimated and related to decreases in methylmercury concentrations in these fish. Based on data that describe the types of fish consumed and current fish consumption rates, we estimate changes in methylmercury exposure in the U.S. population.

Next, we assign monetary estimates to reductions in human health effects plausibly associated with exposures to methylmercury through consumption of U.S. commercial and non-commercial fish. The monetary estimates are primarily based on human capital approaches. We also estimate these values based on health-related quality of life measures. To estimate the economic impact, we estimate the number of children that may incur neurodevelopmental effects of the type that have been observed in epidemiologic studies of pregnant women and their children. We note that this is the primary health concern associated with methylmercury intake from fish (Grandjean et al., 1997; Kjellstrom et al., 1986, 1989). We also estimate cases of adult myocardial infarction and premature death. These effects are valued using a cost-of-illness and a willingness-to-pay approach, respectively.

Our model for estimating current exposures and the changes in U.S. methylmercury exposures and the economic impacts that might result from decreased mercury emissions from power plants is presented in Section 2. In Section 3, the results of the modeling effort is presented and in Section 4 these results are discussed.

2020. The model simulated deposition of elemental, divalent particulate mercury, and gaseous divalent mercury at a 36 Km x 36 Km grid resolution for the continental USA and the surrounding waters.

2. METHODS

Figure 3 depicts our conceptual model for estimating the impact of decreases in U.S. power plant mercury emissions on methylmercury intake in the U.S. population. Mercury emissions sources are divided into two categories: coal-fired power plants and all other sources (depicted as ovals). Mercury emissions from all non-U.S. sources are assumed to remain constant.[25] The arrows represent the movement of mercury from environmental media to fish and fish to humans (depicted as boxes). Because changes in power plant mercury emissions result in spatially varying decreases in the predicted quantities of mercury deposited, the sources of wild fish for U.S. consumers are divided into eight geographic regions (Figure 4). In the model, each region is an isolated compartment, connected only with the atmospheric compartment. There are five freshwater regions in the contiguous U.S.; these are depicted as a single box in Figure 3 and geographically in Figure 4. The boundaries of the five freshwater regions follow.

1. Northeast Region includes New England and New York State,
2. Mid-Atlantic Region includes the States of Virginia, West Virginia, Pennsylvania, New Jersey, Maryland and Delaware,
3. Southeast Region includes the States of Arkansas, Tennessee, and North Carolina and the States to the south of these States,
4. Midwest Region includes the States of Kentucky and Missouri and the States to their North including Ohio and Minnesota, and
5. West Region includes the States of Texas, Oklahoma, Kansas, Nebraska, Iowa and the Dakotas and States to the west in the conterminous U.S.

[25] While there is evidence that global mercury emission and deposition rates are decreasing (Schuster et al., 2002; Slemr et al., 2003), the model results of Lamborg et al. (2002) indicate that atmospheric concentrations are increasing, suggesting that anthropogenic emissions may be increasing globally.

The three saltwater regions are depicted as separate boxes in Figure 3 and also geographically in Figure 4.

1. Atlantic Coastal Region includes the continental shelf adjacent to the eastern seaboard of the U.S. This region is bounded on the west by the U.S. coastline from Maine to the eastern coast of Florida and extends east 200 miles in width from the U.S. coast.

2. Gulf of Mexico Region includes the continental shelf under the Gulf. This region is bounded by a point 50 miles south of Florida extending west to Mexico and from that line north to the continental U.S., and,

3. All Other Waters Region includes all other oceans and seas and the parts of the Atlantic Ocean not included in the Atlantic Coastal Region.

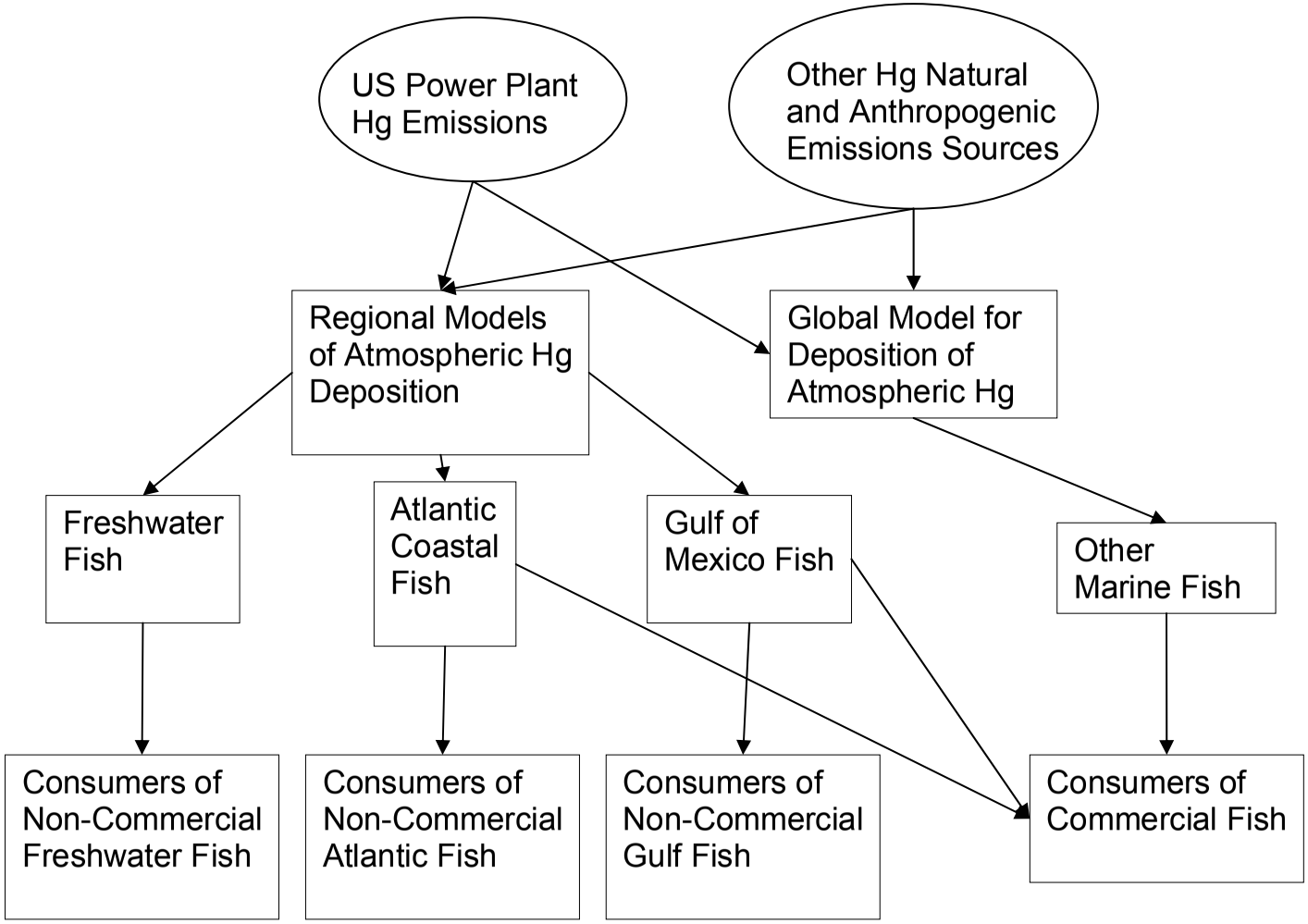

Figure 3

Conceptual Model of Human Mercury Exposures

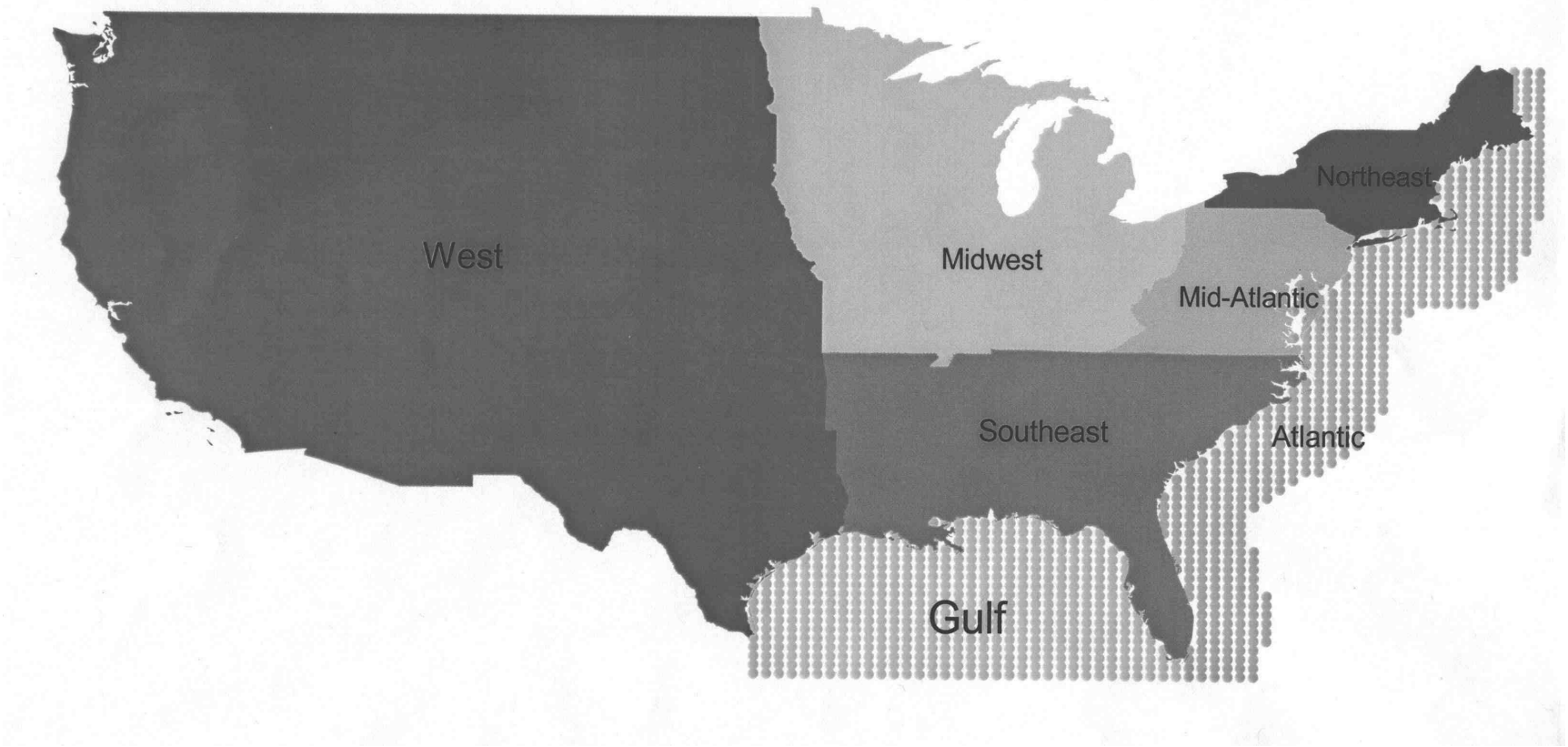

Figure 4

Regions Considered in Model

The model is based on two primary assumptions:

1. Equilibria exist between total deposited mercury and fish methylmercury concentrations and between the fish methylmercury concentrations and methylmercury exposures in the individuals who consume these fish.

2. Changes in the total quantity of mercury deposited lead to proportional changes in fish methylmercury concentrations[26], assuming no other factors change (i.e., *ceteris paribus*).

As a consequence of these two assumptions, changes in mercury deposition rates result in proportional changes in human methylmercury intakes, because there are no changes in other factors including the regions where fish are captured and types or quantities of fish that humans consume.

The first assumption is implemented, in part, through Equation 2, which is a general equation applicable to the commercial and non-commercial fish included in the model.

$$Cf_{ij} = Cc_{ij} \times Df_j / Dc_j \qquad \text{(Eq. 2)}$$

where:

Cf_{ij} = mean methylmercury concentration in the i^{th} fish type in the j^{th} region (μg/g) under an alternate emissions scenario

Cc_{ij} = current mean methylmercury concentration in the i^{th} fish type in the j^{th} region (μg/g)

Df_j = mean annual mercury deposition rate in the j^{th} region (μg/m^2/yr) under an alternate emissions scenario

Dc_j = current mean annual mercury deposition rate in the j^{th} region (μg/m^2/yr)

The premise of Equation 2 is that separate equilibria exist between the mercury deposition rates in each of the eight regions and the mean methylmercury

[26] U.S. EPA (2001d) and EPRI (2003) invoke a similar assumption.

concentrations in the different types of fish which inhabit these regions. Changes in mercury emissions lead to changes in mercury deposition rates that are specific to each region. Based on assumption 2, in each region the changes in the methylmercury concentrations in each type of fish are proportional to the changes in the mercury deposition rates. Thus, the estimate of alternative mean methylmercury concentration in each type of fish in a region is the product of the current mean concentration and the ratio of the alternative to current mercury deposition rate.

2.1. CHANGES IN MERCURY DEPOSITION

For each region, the changes in mercury deposition that result from additional control of U.S. power plant mercury emissions are based on U.S. EPA's analysis of the impacts of the CSI (U.S. EPA, 2003a,b). Based on REMSAD simulations, the U.S. EPA estimated mercury deposition rates corresponding to the following five emissions scenarios: **current emissions**, mercury emissions in 2010 (**Baseline 1**) [this includes projected changes in U.S. mercury emissions], mercury emissions in 2010 with a 47% reduction in U.S. power plant emissions associated with implementing the CSI (**Scenario 1**), mercury emissions in 2020 (**Baseline 2**), and mercury emissions in 2020 with a 69% reduction in U.S. power plant emissions associated with implementing the CSI (**Scenario 2**) (U.S. EPA, 2003a,b).

For the five freshwater regions, the Atlantic Coastal Region and the Gulf of Mexico Region, we summed all forms of mercury predicted to deposit in each 36 x 36 Km grid cell under each of the five emissions scenarios (i.e., Current, Baseline 1, Scenario 1, Baseline 2, and Scenario 2). Then, for each grid cell in each region, we developed four ratios of mercury deposition. The total annual mercury deposition

associated with current emissions, as predicted by EPA's REMSAD model, was used as the denominator in each ratio. The numerators were the predicted total mercury deposited in the grid cell in Baseline 1, Scenario 1, Baseline 2, and Scenario 2, based on the relevant REMSAD simulation modeling. Thus, the four ratios are Baseline 1/Current, Scenario 1/Current, Baseline 2/Current, and Scenario 2/Current.

We assume the waters in the Atlantic Coastal and Gulf of Mexico Regions to be well mixed. There are 539 and 511 REMSAD grid cells in the Atlantic Coastal and the Gulf of Mexico Regions, respectively. For each of these two regions, we estimated the mean value of each of the four ratios. These eight mean ratio values were used in subsequent calculations to estimate fish methylmercury concentrations under different emissions scenarios.

For each of the five freshwater regions, using Crystal Ball® (Decisioneering, Inc., Denver, CO), we developed unique distributions of these four ratios (i.e., Baseline 1/Current, Scenario 1/Current, Baseline 2/Current, and Scenario 2/Current); thus, we developed a total of 20 unique distributions of these ratios (the product of 5 regions and 4 ratios in each region). A distribution was estimated from the vector of equally-weighted grid cell values. In subsequent calculations, by sampling with replacement, these distributions are used to estimate the variability of changes in regional freshwater fish methylmercury concentrations associated with mercury emissions reductions.

Because U.S. mercury emissions are transported large distances to reach remote areas encompassed by the All Other Waters Region (e.g., Eastern Atlantic Ocean and Pacific Ocean), we assume that U.S. anthropogenic emissions become well-mixed with mercury emitted from other sources in the atmosphere during long-range

transport. Under the well-mixed assumption, we assume that the mercury emitted from U.S. power plants deposits in these distant areas, at the same rate as the rest of the mercury in the atmosphere. Thus, in the All Other Waters Region, mercury emitted from U.S. sources is estimated to contribute to mercury deposition in proportion to its contribution to global anthropogenic and natural emissions. (Current mercury emissions from the U.S. utility sector are approximately 1% of total mercury emitted globally and total U.S. anthropogenic emissions are roughly 3% [Table 1].)

2.2. METHYLMERCURY EXPOSURES THROUGH FISH CONSUMPTION

Our model simulates exposures for consumers of fish from the U.S. commercial seafood market (Section 2.2.1), the most common source in the U.S., and consumers of fish not caught commercially (Section 2.2.2). The model assumes commercial fish consumers obtain their fish from a mix of four sources: the coastal Atlantic, the Gulf of Mexico, All Other Waters and aquaculture (or farm-raised fish).[27] Non-commercial fish consumers are assumed to eat a mix of fish caught in an individual region and a mix of commercial fish. The sizes of the fish-consuming populations are estimated in Section 2.2.3.[28]

[27] Clearly, there may be regional sources for some commercial fish. Under these assumptions, the variability associated with the consumption of commercial fish from a small region will not be captured in this model.

[28] The assumption that non-commercial fish consumers eat a mix of the most common types of fish caught or targeted for capture reduces the variability in the model. Some consumers may target specific species for consumption (or conversely avoid certain species); methylmercury intakes may vary markedly from the weighted means calculated for each region.

2.2.1. Methylmercury Exposures Through Commercial Seafood Consumption.

Carrington and Bolger (2002) adjusted their simulations of human methylmercury intake through commercial seafood consumption to match the distribution of measured U.S. blood methylmercury levels (Mahaffey et al., 2004, based on NHANES survey data, Table 16). Given that 70%-90% of the U.S. population consumes commercial fish (Carrington and Bolger, 2002), we assume that the distribution of blood methylmercury concentrations reported by Mahaffey et al. (2004) is a consequence of commercial fish consumption. Some individuals consume no fish. These individuals are represented in the blood methylmercury distribution reported by Mahaffey et al. (2004). In the model, we assume these individuals (i.e., those that consume no fish) to be in this population.

Table 16. Blood Methylmercury Concentrations (µg/L) in U.S. Women Aged 16 to 49

Population	n	Geo.[b] Mean	5th [c]	10th	25th	50th	75th	90th	95th
0 fish and shellfish meal in previous 30 days [a]	480	0.39	--	--	--	--	0.44	1.1	1.6
1-4 fish and shellfish meals in previous 30 days	780	0.7	--	--	--	0.6	1.29	2.9	4.7
5-8 fish and shellfish meals in previous 30 days	230	1.33	--	--	0.43	1.29	3.29	6.1	9.9
>8 fish and meals in previous 30 days	153	2.46	--	0.44	1.15	2.75	5.2	11.1	12.1
Total	1707	0.8	--	--	--	0.6	1.7	4.44	6.73

Source: Mahaffey et al., 2004
[a] Fish meal - self-reported number of fish meals in the 30 day period prior to study participation.
[b] Geo. Mean – reported geometric mean.
[c] 5th, 10th, … 95th – percentiles of total blood methylmercury concentration

Based on assumption 1 (see introduction to Section 2), we assume the reported blood methylmercury distribution in U.S. commercial consumers to be in equilibrium with the mean methylmercury concentration in commercial fish weighted by per capita consumption rates. This implies that the fish that commercial fish consumers eat are well mixed with respect to the types of fish consumed and the regions in which fish are caught. We use Equation 3 to develop a current weighted mean methylmercury concentration for commercial fish.

$$Cmc = \frac{\sum_{i=1}^{24} w_i \times Cc_i}{\sum_{i=1}^{24} w_i} \qquad \text{(Eq. 3)}$$

where:

Cmc = current weighted mean methylmercury concentration for all commercial fish (µg/g)

w_i = estimated annual per capita consumption rate of the ith fish type (kg/person year)

Cc_i = current mean methylmercury concentration of the ith fish type

Carrington and Bolger (2002) report current per capita consumption data and mean methylmercury concentration data for 24 types of commercial fish (Table 2), which account for over 90% of the mass of fish in the U.S. commercial market. We assume that the methylmercury concentrations and the per capita consumption rates of the remaining 10% of the mass of commercial fish to be similar to those reported by Carrington and Bolger.

Based on the first two assumptions, the estimated annual per capita consumption rate of the i^{th} fish type, w_i, remains constant over time; thus, Equation 3 can be modified to estimate the weighted mean methylmercury concentration for all commercial fish

under alternative emissions scenarios with corresponding alternative regional mercury deposition rates.

$$Cmf = \frac{\sum_{i=1}^{24} w_i \; x \; Cf_i}{\sum_{i=1}^{24} w_i} \qquad \text{(Eq. 4)}$$

where:

Cmf = weighted mean methylmercury concentration for all commercial fish under alternative emissions scenario (μg/g)

w_i = estimated annual per capita consumption rate of the i^{th} fish type (kg/person year)

Cf_i = mean methylmercury concentration of the i^{th} fish type (μg/g) under alternative emissions scenario (See Eq. 5)

In this model, the mean concentration of i^{th} fish type ($Cf_{i)}$ under an alternative deposition scenario depends on the change in mercury deposition in the region. While the regional harvest data can be estimated for individual fish types (NMFS, 2002), the methylmercury concentration data on commercial fish in each region are not available. Only the mean methylmercury concentration data are reported for each type of commercial fish. For an individual type of commercial fish, we used Equation 5 to estimate the mean methylmercury concentration in fish under alternative deposition scenarios.

$$Cf_i = Cc_i x \sum_{j=1}^{n} \left[\frac{Q_j}{\sum_{j=1}^{n} Q_j} x \frac{Df_j}{Dc_j} \right] \qquad \text{(Eq. 5)}$$

where:

Cf_i = mean methylmercury concentration of the i^{th} fish type (μg/g) under alternative emissions scenario

Cc_i = current mean concentration of the i^{th} fish type (μg/g)

Q_j = quantity of fish harvested annually from the j^{th} region (kg/yr)

Df_j = mercury deposition rate in the j^{th} region in which the fish are captured under alternative emissions scenario (μg/m^2/yr)

Dc_j = current mercury deposition rate in the j^{th} region in which the fish are captured (μg/m^2/yr)

In Equation 5, for each region in which commercial fish are captured, we calculate, based on mass, the fractional contribution of the individual harvest from each regional source to the total annual quantity of the type of fish harvested (Q_j/ ΣQ_j). Next, the ratio of Df/Dc provides an estimate of the change in the mercury deposition rate in the region. We compute the product of the fractional contribution of the fish caught in each region and the ratio of the alternative and current deposition rates. By summing this product across the regions where fish are caught and multiplying by the current mean fish methylmercury concentration, an alternative mean methylmercury concentration can be estimated for a specific type of fish.

In this model, commercial fish are obtained from four regions, the coastal Atlantic, the Gulf of Mexico, the All Other Waters Region, and aquaculture. We merged the fish raised in aquaculture and those inhabiting All Other Waters into a single group. After estimates of Cf are calculated for each fish, Equation 4 is used to estimate the change in the weighted mean methylmercury concentration for commercial fish.

Next, we determine the quantity of each type of commercial fish caught in the three saltwater regions. We assume that the methylmercury concentrations of all fish imported into the U.S. are in equilibrium with the mercury deposited in the All Other Waters Region. Because tuna, shark and swordfish are highly migratory and may

consume prey over broad geographic ranges, their methylmercury concentrations likely are influenced by mercury deposition over broad geographic areas. We assume the methylmercury concentrations in these three types of fish to be in equilibrium with mercury deposited to the All Other Waters Region.[29] Methylmercury concentrations in the commercial freshwater species, obtained primarily from aquaculture, are also assumed to be in equilibrium with mercury deposited in the All Other Waters Region.[30]

We assume that the remaining 19 types of marine fishes that are caught by the U.S. domestic fishing fleet in the Atlantic Ocean or the Gulf of Mexico inhabit and feed on prey exclusively from the Atlantic Coastal Region or the Gulf of Mexico Region. Thus, in the model, the methylmercury concentrations in these fish are in equilibrium with mercury deposition in these regions. We used commercial fish data compiled by NMFS for year 2001 (summarized in Table 2) to estimate the fraction (based on mass) of each type of fish in the U.S. commercial market that was caught in the Atlantic Ocean, the Gulf of Mexico, the Pacific Ocean, the Great Lakes, and imported. The mass of each fish type harvested by the U.S. fishing fleet is reported by NMFS as a round weight (i.e., whole fish weight). NMFS reports imported and exported fish as product weights. To estimate the original whole fish weights of imports and exports, we

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[29] Some species of tuna and shark do not appear to migrate over broad geographic regions of the oceans. Regional mercury deposition patterns may be important sources of methylmercury in these fish. Our model does not incorporate this possible source of local variability.

[30] We note that aquaculture fish are not part of the wild foodchain. Protein sources in many farm-raised fish diets include fish meal obtained from wild-caught fish. Meal derived from plant proteins is sometimes mixed or substituted, depending on the type of fish. Methylmercury concentrations in the wild-caught fish used in the fish meal depend on the type of fish used and its location. Because sources of such fish are not completely known, we employed a simplifying assumption that the methylmercury concentrations in the fish used in fish meal were in equilibrium with mercury deposited in all other waters. If fed fish meal or fish oils, the methylmercury concentrations in these feed sources are likely low because fish meal and fish oils are made from low trophic level marine species.

converted the product weights to round weights using product specific conversion factors (provided through personal communication by Steve Koplin, NMFS). We then summed the round weights for each product that contained a specific type of fish. There are other product codes that likely contain the types of fish analyzed here, but, because they do not identify a particular type of fish, they were excluded from the calculations contributing to the imprecision in our estimates.

The weights of re-exported fish (i.e., fish imported into the U.S. and then exported) were subtracted from the total import weight of the particular type of fish. Exports of U.S. caught fish are assumed to be proportional to the fraction of the fish type caught by the U.S. fleet in each region. For example, in 2001, 1.7 million pounds of U.S. clams were exported; over 95% of these exported clams were assumed to have been collected from the Atlantic and the remainder collected in the Pacific and the Gulf of Mexico.

We assume that the distribution of U.S. blood methylmercury concentrations in females of reproductive age (Mahaffey et al., 2004 in Table 16) is in equilibrium with the weighted mean methylmercury concentration for commercial fish.[31] The blood methylmercury concentrations reported in U.S. women of reproductive age are assumed to be representative of the concentrations in this U.S. population. The data of Mahaffey et al. consist of commercial fish-consumers and non-fish consumers and are assumed to be representative of this population in the model.

[31] Implementation of this assumption decreases inter-individual variability in the model results. While some consumers may eat a mix of fish, others may consume a subset of the 24 species (e.g., some may consume only tuna or salmon).

Equation 6 was used to estimate the alternative distribution of U.S. blood methylmercury concentrations among commercial seafood consumers; this estimate is the product of the ratio of the alternative and the current mean weighted methylmercury concentrations in commercial fish (Cmf/Cmc) and the distribution of the current methylmercury blood concentration in the U.S. population. Through an *ad hoc* procedure, we determined that a gamma distribution (scale parameter = 1.28 and shape parameter = 0.65) provided a reasonable fit to the centile data reported by Mahaffey et al.

$$Bf = Bc * (\frac{Cmf}{Cmc}) \qquad \text{(Eq. 6)}$$

where:

Cmf = weighted mean methylmercury concentration in U.S. commercial fish under alternative deposition scenario (µg/g)

Cmc = current weighted mean methylmercury concentration in U.S. commercial fish (µg/g)

Bf = blood methylmercury concentration under alternative deposition scenario (µg/L)

Bc = current blood methylmercury concentration (µg/L)

Thus, the projected change in methylmercury concentrations of commercial fish results in proportional shifts in the distribution of blood methylmercury concentrations in the commercial fish consuming populations. Hair mercury levels are predicted based on blood methylmercury concentrations (see Equation 1).

Because the NHANES data reported by Mahaffey et al. are specific to U.S. females of reproductive age, we estimated male methylmercury exposures by adjusting inputs to the methylmercury one-compartment pharmacokinetic model (see Equation 1).

Using the gamma distribution developed for the blood methylmercury concentrations in U.S. females of reproductive age, male blood methylmercury concentrations were estimated based on a form of the "methylmercury one-compartment model" (Equation 7) (Ginsberg and Toal, 2000; NRC, 1999; U.S. EPA, 2001c). Between the genders, we assume that there are no differences in the mean methylmercury concentrations of the commercial fish consumed and in methylmercury toxicokinetics. Thus, using the one-compartment model, the differences between the genders' blood methylmercury levels reduce to differences in fish consumption rates, body weight and blood volume. Average values for these differences are shown in Table 17. We assume the elimination constant, the gastrointestinal absorption factor, and the fraction of the absorbed methylmercury dose entering the blood to be same for both genders (see Equation 1).

$$C_{Bm} = C_{Bf} X \frac{E_m / E_f \; x \; Bw_m / Bw_F}{V_m / V_f} \qquad \text{(Eq. 7)}$$

where:

C_{Bm} = Male blood methylmercury concentration (µg/L)

C_{Bf} = Female blood methylmercury concentration (µg/L)

E_m = Male oral exposure (µg/kg-day^{-1})

E_f = Female oral exposure (µg/kg-day^{-1})

V_m = Male blood volume (L)

V_f = Female blood volume (L)

Bw_m = Male body weight (kg)

Bw_f = Female body weight (kg)

Using the parameter values reported in Table 17 implies that mean blood methylmercury concentration is 4.3% less in males than in females.

Table 17. Comparison of Body Weight, Blood Volume and Fish Intake Between U.S. Males and Females

	Body Weight[a] (Kg)	Total Blood Volume[a] (L)	Mean Reported Fish Intake (g/person/day) Uncooked Fish Weight[b] (Age= 15-44 years)
Adult Female	60	3.9	0.29118
Adult Male	73	5.3	0.30978

[a] Source: ICRP, 1975, 2003.
[b] U.S. EPA, 1997b. For general U.S. population.

2.2.2. Methylmercury Exposures Through Consumption of Non-Commercial Seafood.

U.S. residents are also exposed to methylmercury through consumption of non-commercial fish. We estimated the distribution of daily methylmercury intake for non-commercial fish consumers in each saltwater region and freshwater region separately. We assumed "recreational" fish statistics to be a reasonable proxy for non-commercial fishing. We assumed that individuals consumed a mix of non-commercial fish caught in a specific region.[32] We also assume that these non-commercial fish consumers eat a mix of commercial fish. No individuals are assumed to consume non-commercial fish from more than one region. For example, no individuals are assumed to consume non-

[32] This assumption, like the similar one employed for commercial fish consumers, reduces the inter-individual variability captured in this model.

commercial marine fish and non-commercial fresh water fish because they would be obtained from two different regions.

2.2.2.1. Non-commercial Consumers of Marine Fish —

The methylmercury intake for non-commercial consumers of marine fish caught in the Atlantic and Gulf was estimated in a manner similar to that of the commercial fish intake. Tables 4 and 5 list the mean methylmercury concentrations reported in Atlantic and Gulf fish as reported in U.S. EPA (2003c). We assumed these estimates of the mean concentrations to be representative of mean methylmercury concentrations in tissues of fish typically caught non-commercially in U.S. saltwaters and consumed. Because the EPA document does not identify the sizes or weights of the sampled fish, it is not possible to evaluate whether the reported methylmercury concentrations for the fish in this database are representative of fish consumed recreationally. Additionally, there are a small number of samples reported for several types of fish. Equations 4 and 5 are used to estimate mean weighted methylmercury concentrations for current conditions and under alternative emissions scenarios. To apply these equations, the w_i term is a measure of the quantity of each fish type harvested non-commercially. Using the mean methylmercury concentration data and the estimated mass of the top 10 types of fish caught for consumption by non-commercial fishers (NMFS, 2002) in Tables 4 and 5, we estimated a current weighted mean methylmercury concentration for non-commercial fish in the Atlantic and the Gulf.

Non-commercial fish consumption rates for Atlantic and Gulf fish are reported separately in U.S. EPA (1997a). Equation 8 is used to estimate the current methylmercury intake rates among these consumers. By substituting the mean

weighted methylmercury concentration under alternative emissions scenarios into Equation 8 (based on results of Equation 5) methylmercury intake rates (I_c) are estimated for consumers under these scenarios. This distribution of methylmercury intake rates was converted to mercury hair concentrations using the one-compartment pharmacokinetic model (see Equation 1).

$$I_c = (CR_F \times Cmc_{nm}) / Bw \qquad \text{(Eq. 8)}$$

where:

I_c = oral intake of methylmercury from current fish consumption (µg/kg-day)

CR_F = consumption rate of fish (g/day)

Cmc_{nm} = current weighted mean methylmercury concentration in non-commercial marine fish (µg/g)

Bw = body weight (70 kg)

2.2.2.2. Non-commercial Consumers of Freshwater Fish —

We estimate methylmercury intakes through consumption of non-commercial freshwater fish based on a consumption model similar to Equation 8.

We used data from the National Listing of Fish and Wildlife Advisories (NLFWA) Mercury Fish Tissue Database (U.S. EPA, 2003d as of October 31, 2003) to estimate methylmercury concentrations in U.S. freshwater fish. We initially identified a total of 57,678 samples in the database that reported methylmercury concentrations. We selected sample data that were identified in the database as fillets, skin-on fillets, skin-off fillets, and composite fillets (NLFWA Sample ID numbers 4, 5, 6, and 7). Based on the fish samples' State of origin, we categorized the sample data by U.S. regions (Figure 4).

We then subdivided the regional data into nine categories corresponding to the U.S. FWS data describing the types of fish targeted by anglers.[33] Table 18 identifies the U.S. FWS category and examples of the types of fish we grouped under each category. No steelhead trout were identified in the NLFWA database; consequently, we did not use that category. We also ignored the U.S. FWS categories of "anything" and "other type of fish." Finally, we assumed that only fish above a certain length were retained for consumption following capture. Samples from fish shorter than the minimum length requirements prescribed by the State of Pennsylvania (2003) and samples where fish length was not reported were excluded (Table 18). (Comparison with several other State regulations indicates relatively consistent size restrictions across States.) We note that minimum length requirements are not identified for certain types of fish in Pennsylvania; we imposed a minimum size of 5 inches for the sample to be considered, because people typically do not consume small fish. We assume that the fish tissue methylmercury concentrations reported in the database are typical of those of fish captured and consumed.

[33] The U.S. FWS (2001) national survey asked anglers to identify the type(s) of fish they targeted during their fishing and how much time (in days) they spent targeting particular types of fish. The survey did not evaluate the types of freshwater fish consumed following capture. They report 12 categories of freshwater fish targeted for capture by freshwater anglers (listed in first row of Table 19).

Table 18. Fish Size Restrictions Imposed on Model Data

U.S. FWS Designation	Minimum Length[a] (inches)	Examples of Types of Fish Included from NLFWA Database
Crappie	5[b]	Black and white crappie
Panfish	5[b]	Rock bass, bluegill, sunfishes, perch
White Bass, Striped Bass, Striped Bass Hybrids	12	White bass and striped bass
Black Bass	12	Largemouth, smallmouth and spotted bass
Catfish, Bullheads	5[b]	Bullheads, channel catfish, fathead catfish, white catfish, flathead catfish
Walleye/Sauger	15/12	Walleye and sauger
Northern Pike/Pickerel/ Muskie, Muskie Hybrids	24/15/30	Muskellunge, chain pickerel, and Northern Pike
Trout	7	Rainbow, lake, brook, splake, and brown trout
Salmon	7	Coho salmon, Atlantic Salmon, Lake Whitefish, cisco, Chinook salmon

[a] State of Pennsylvania (2003).
[b] The State of Pennsylvania has no minimum length requirement for crappie, panfish and catfish; we imposed a minimum length of 5 inches for fish to be included in these categories.

Using Crystal Ball®, we developed unique distributions for each type of fish in each U.S. Region based on the individual methylmercury concentrations. For a sample size of n, the i^{th} measurement was given a weight of 1/n when developing a distribution.

Within each U.S. Region, targeting weights were developed for each type of fish. For each State in a region, we multiplied the percent days spent fishing in that state by the percent days targeting a specific type of fish in each State. For a given type of fish, we summed these products across all the States in the Region to develop target weights specific to each type of fish in each region.[34]

[34] This is parameter T in Equation 9.

Table 19. Percentage of Fishing Days Targeting Selected Species

State	Crappie	Panfish	White and Striped Bass	Black Bass	Catfish	Walleye and Sauger	Northern Pike, Pickerel and Muskie	Steelhead	Trout	Salmon	Anything	Other Species
AL	18	16	10	36	11	0	0	0	1	0	5	3
AZ	5	5	12	24	13	0	0	0	28	0	13	0
AR	22	9	13	21	22	0	0	0	3	0	5	4
CA	4	4	10	15	10	0	0	0	35	13	7	3
CO	5	2	3	7	9	2	4	0	60	3	5	0
CN	0	10	12	32	2	0	4	0	31	0	8	0
DE	10	10	15	29	9	0	0	0	8	0	19	0
FL	13	15	8	27	14	0	0	0	1	0	13	9
GA	15	15	13	18	23	0	0	0	4	0	7	3
ID	2	3	2	12	7	0	0	11	54	10	0	0
IL	15	20	9	15	23	6	0	0	4	0	9	0
IN	16	27	11	23	16	3	0	0	0	0	4	0
IA	17	15	9	15	18	15	5	0	3	0	3	0
KS	19	9	7	21	25	9	0	0	2	0	7	0
KY	20	13	13	24	19	4	0	0	1	0	6	0
LA	20	1	9	22	18	0	0	0	2	0	5	5
ME	0	5	7	24	0	0	9	0	34	16	4	1
MD	4	8	12	30	13	4	0	0	13	0	7	9
MA	0	6	12	36	4	0	6	0	23	0	11	2
MI	7	34	5	14	0	9	9	3	7	3	7	0
MN	16	20	2	9	1	32	18	0	2	0	1	0
MS	18	17	9	18	27	0	0	0	2	0	9	0
MO	21	15	8	24	22	0	0	0	5	0	3	1
MT	0	5	0	6	2	12	12	0	49	4	9	1

Table 19 cont.

State	Crappie	Panfish	White and Striped Bass	Black Bass	Catfish	Walleye and Sauger	Northern Pike, Pickerel and Muskie	Steelhead	Trout	Salmon	Anything	Other Species
NE	13	9	9	19	18	15	5	0	2	0	9	2
NV	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
NH	0	8	3	28	0	0	7	0	32	5	14	2
NJ	0	13	10	36	6	0	7	0	16	0	4	8
NM	2	3	7	9	11	0	0	0	58	7	3	0
NY	3	11	3	28	4	10	10	2	19	4	7	0
NC	19	11	12	24	17	0	0	0	5	0	8	5
ND	3	16	0	2	2	52	18	0	1	1	5	1
OH	11	17	7	31	16	11	0	0	3	0	4	1
OK	20	6	8	31	20	4	0	0	2	0	9	0
OR	1	2	2	5	3	0	0	14	41	21	6	7
PA	5	7	10	25	7	7	3	0	26	0	9	0
RI	0	9	19	26	0	0	0	0	30	0	17	0
SC	16	15	9	25	22	0	0	0	2	0	8	3
SD	5	18	2	5	6	42	12	0	3	0	7	0
TN	17	15	10	27	15	6	0	0	7	0	4	0
TX	18	5	14	27	30	0	0	0	2	0	4	0
UT	0	4	3	11	4	1	0	0	68	0	6	3
VT	0	13	3	10	3	7	7	0	34	9	11	3
VA	14	11	14	30	14	0	0	0	10	0	7	0
WA	1	3	2	10	0	0	0	18	39	21	2	4
WV	7	9	6	28	17	0	0	0	22	0	11	0
WI	11	29	3	17	1	17	15	0	4	0	3	0
WY	0	0	0	0	0	1	0	0	78	0	0	3

ND= No data reported from State

U.S. EPA (1997a) reports the mean and 95th percentile fish consumption rates for recreational freshwater anglers to be 8 and 25 g/day, respectively, based on integrating the data reported from studies of freshwater anglers fishing in the states of Maine, New York and Michigan (U.S. EPA, 1997a citing Ebert et al., 1993; Connely et al., 1996; West et al., 1989). We assumed that these data were lognormally distributed and calculated the standard deviation of these data to be 10.3 g/day.

Some individuals and groups consume large quantities of fish. To estimate exposures among these high-end consumers, we used information from the Columbia River Inter-Tribal Fish Commission Report (1994). The Report describes consumption rates among a group of Native Americans who likely consume fish at the upper end of the distribution.[35] The mean and 95th percentile fish consumption rates were 59 and 170 g/day, respectively. We assumed that these data were lognormally distributed and calculated a standard deviation of 64 g/day.

We estimated methylmercury exposures to consumers of non-commercial U.S. freshwater fish as the product of the distribution of the weighted fish methylmercury concentrations and the distribution of the freshwater fish consumption rate (Equation 9). The change in mercury deposition in each region under alternative emissions was based on the unique distributions of the four ratios described in Section 2.1. We implemented a Monte Carlo approach using Crystal Ball®. Each simulation consisted of 50,000 iterations.

[35] While described as a study of "subsistence" angler consumption (U.S. EPA, 1997b), there is not a quantitative point or percent of total daily caloric intake or protein intake that results in a categorization of subsistence.

$$I = \frac{CR_F x \sum_{i=1}^{n} \left[T_i x C_{ik} \right] x \frac{Df_L}{Dc_L}}{Bw} \qquad \text{(Eq. 9)}$$

where:

I = oral intake of methylmercury from fish consumption (µg/kg-day)

CR_F = fish consumption rate (g/day)

T_i = relative target frequency for i^{th} fish type (unitless)

C_{ik} = methylmercury concentration in the k^{th} randomly selected fish of the i^{th} fish type (µg/g)

Df_L = future mercury deposition rate of the L^{th} randomly selected grid cell (µg/m^2/yr)

Dc_L = current mercury deposition rate of the L^{th} randomly selected grid cell (µg/m^2/yr)

Bw = body weight (kg)

n = n different types of non-commercial freshwater fish routinely targeted in a region

In each iteration, a single REMSAD model output from a randomly selected grid cell in a region was used to estimate the ratio of the future to current mercury deposition rate (Df_L/Dc_L). This ratio was multiplied by the fish methylmercury concentration of a randomly selected fish. We made no attempt to estimate the time needed for a change in deposition to result in a change in fish concentrations. In each of the 50,000 iterations, each type of fish ($n \leq 10$) was selected once and weighted based on the relative target frequency for that fish type. The consumption rate data were also analyzed using the Monte Carlo approach.

Thus, each non-commercial freshwater fish consumer is assumed to eat a mix of non-commercial fish caught in a specific region. The methylmercury content of each

fish in the mix is obtained by randomly sampling fish methylmercury concentrations for each type of fish from a given region. These values are independent of fish consumption rates. Changes in methylmercury concentrations of fish are based on randomly sampling from the ratios developed in Section 2.1. For each of the populations of non-commercial fish consumers, the daily methylmercury intake rates were converted to changes in blood mercury concentrations using a one-compartment methylmercury model (see Equation 1). The changes in mercury concentration in hair were estimated assuming a ratio of hair methylmercury concentration (µg mercury/g hair) to steady-state blood methylmercury concentration (µg mercury/L blood) of 0.25:1 (U.S. EPA, 1997a, 2001a).

2.2.2.3. Commercial Fish Consumption Among Those also Consuming Non-Commercial Fish —

Non-commercial fish consumers are also exposed to methylmercury through the consumption of commercial fish. Connelly et al. (1996) developed estimates of total fish consumption and non-commercial fish consumption among Lake Ontario anglers. They randomly drew a sample of 2,500 names from 1990-1991 New York fishing license records purchased in six counties bordering Lake Ontario. From the initial sample, 1,202 stated that they intended to fish Lake Ontario in 1992 and were willing to participate in the study and 853 participated in the final study. Table 20 identifies the quantities of total and non-commercial fish consumed. In the Monte Carlo simulation model, non-commercial fish consumers were categorized based on the quantity of non-commercial fish they consumed each day. Total commercial fish intake in individuals that consumed less 0.6 g/day was estimated by subtracting the daily fish intake value

from 8.8 grams/day, the daily intake of total seafood corresponding to the lowest category of recreational fish intake. Individuals consuming more than 39.8 grams of non-commercial fish per day were assumed to consume no commercial fish.[36] The remaining individuals in the simulation, who consumed between 0.6 and 39.8 grams of non-commercial fish per day were categorized into 1 of 5 groups depending on the quantity of recreational fish they consumed; the groups were 0.6-2.2, 2.2-6.6, 6.6-13.2, 13.2-17.9, and 17.9-39.8 (g/day). In these groups, total commercial fish intake was estimated by subtracting the non-commercial intake from the average of the total fish intake values for the same stratum of the population reported by Connoly. For example, to predict commercial fish consumption among individuals that consumed between 0.6 and 2.2 grams per day of non-commercial fish, the quantity of non-commercial fish consumed was subtracted from 11.5, the average of 8.8 and 14.1 grams/day.

Table 20. Commercial Fish Intake Rates Among Consumers of Recreationally Caught Fish

	Percentile					
Source: Conolly et al., 1996	25	50	75	90	95	99
Recreational fish intake g/ day	0.6	2.2	6.6	13.2	17.9	39.8
All fish intake g/day	8.8	14.1	23.2	34.2	42.3	56.6

2.2.3. Estimates of Population Size.

This section describes the method used to estimate the sizes of the U.S. population consuming non-commercial and commercial fish. Both the U.S. FWS and NMFS have developed estimates of the number of recreational fishers in the Gulf of

[36] The value of 39.8 g non-commercial fish/day is the 99th percentile in the Conolly study.

Mexico and the Atlantic Ocean (Table 3). We averaged these two estimates, assumed that 80% of the anglers consumed non-commercial fish (U.S. EPA, 1997a), and multiplied this value by 2.5 (U.S. EPA, 1997a notes that anglers typically share their catch with 1.5 other people, thus, on average, 2.5 individuals share a catch) to estimate the number of consumers in the Atlantic Coastal and Gulf of Mexico Regions.

In 2001, U.S. FWS estimated that there were 28.4 million freshwater anglers, based on their National Survey of Fishing, Hunting, and Wildlife-Associated Recreation. Because of our interest in the source of the freshwater fish, rather than freshwater fish consumers' residence state, we estimate the number of freshwater fish consumers in each Region as the product of the total population predicted to consume recreationally-caught fish and the percentage of days fishing (relative to the U.S. total) in each U.S. Region (Table 21). Because roughly 80% of anglers report eating their catch (U.S. EPA, 1997a), we adjusted our initial estimates by a factor of 0.8. Because, on average, anglers share their catch with 1.5 additional people, we multiplied the regional estimates by 2.5. We summed the total number of non-commercial fish consumers from each of the seven regions and subtracted this from the total U.S. population to estimate the population that consumes no fish or only commercial fish.

Table 21. Fishing Days by U.S. Region and Estimated Number of Consumers of Fish Caught in Each U.S. Freshwater Region

Region	Days Fishing	Percent of Total	Estimated Number of Consumers (thousands of fishers)
Northeast	36,685,000	8.7	2,965
Mid-Atlantic	3,053,000	0.7	247
Southeast	109,505,000	25.9	8,852
Midwest	150,895,000	35.7	12,197
West	122,953,000	29.1	9,939
Total	423,091,000	100	34,200

The demographic distribution of each population was assumed to be the same as the general U.S. population. We assume that 22% of the non-commercial fish consumers are women of child-bearing age and that 6.2% of these women bear children in a given year (U.S. Census Bureau, 2004, based on a 5-year average). The U.S. population above the age of 39 years is at higher risk for adverse myocardial events than the population below age 39. Based on the 2000 Census data (U.S. Census Bureau, 2004), there were 55,681,000 males and 63,705,000 females above the age of 39 years in the U.S. in 2000; males and females above this age comprise approximately 19.8% and 22.6% of the total U.S. population, respectively. There is no attempt to adjust this annual estimate to reflect possible demographic changes (e.g., changes in the fraction of women of child-bearing age or birth rate) over time (e.g., 2010 or 2020).

2.3. NEUROTOXICITY

Changes in IQ associated with intrauterine methylmercury exposures were estimated using Equation 10. Cohen et al. (submitted) developed a slope estimate of

-0.7 IQ points per 1 ppm increase in hair mercury concentration. Cohen et al. used a weighting procedure for the neurological test results from the Seychelle Islands, Faroe Islands, and New Zealand studies. In a re-analysis of the New Zealand cohort study (Kjellstrom et al., 1989), Crump et al. (1998) reported a decrease of 0.5 IQ points on the Wechsler Intelligence Scale for Children-Revised Full Scale IQ for each increase of 1 ppm methylmercury in average maternal hair during pregnancy. We note that in the analysis by Crump et al., the coefficient for the Wechsler Intelligence Scale for Children-Revised Full scale IQ was not statistically significant. At this time, there is not a single best estimate of the slope of this dose-response function. A range of plausible slope estimates might include values close to 0 (i.e., essentially no effect) up to values of -1. In this analysis an estimate of -0.6 IQ points per 1 ppm increase in hair mercury concentration is used. We assume that this estimate approximates a central tendency value for the slope. Section 1.5.1 describes the range of neurodevelopmental delays. While IQ, a rather blunt neurological assessment tool, would correspond to some of the domains identified, other neurodevelopmental delays may not be captured through an IQ assessment.

$$IQ_D = -0.6 \text{ IQ points} \times Hg_h \qquad \text{(Eq. 10)}$$

where:

IQ_D = IQ point lost given maternal mercury exposure

Hg_h = maternal hair mercury concentration (ppm)

Next, we discuss the implications of a toxicity threshold for neurodevelopmental effects from methylmercury exposures. We identify a plausible surrogate value for a population toxicity threshold for methylmercury. The plausible threshold value is used in

subsequent sections that discuss the human health benefits associated with reducing methylmercury exposures.

The risk functions developed by Cohen et al. and Crump et al. did not use a threshold. This is consistent with a statement made by EPA in the discussion of the methylmercury oral reference dose (RfD); U.S. EPA (2001a) noted "no evidence of a threshold arose for methylmercury-related neurotoxicity within the range of exposures…" of the epidemiologic studies analyzed.

2.3.1. Population Toxicity Threshold: Background.

A toxicity threshold for a population implies the existence of a contaminant intake rate that is without risk of adverse health effects (i.e., "a safe dose" to which any person could be exposed without deleterious effects). The existence of a toxicity threshold implies the following functional form for estimating the risk associated with an oral intake of a contaminant (Equation 11).

$$\begin{aligned} &\textit{If} \quad D \leq T, \quad P(D) = 0 \\ &\qquad\quad D > T, \quad P(D) = m \times (D - T) \end{aligned} \qquad \text{(Eq. 11)}$$

where:

P(D) = Probability of effect at dose D

T = Population toxicity threshold (µg /kg-day)

D = average dose (µg/kg-day)

m(*) = slope of the dose-response function observed for the contaminant (per µg/kg-day)

We used the same value for m (0.6 IQ points/ppm hair) in both the threshold and non-threshold analysis. Other slopes could be developed using other assumptions regarding the slope of the dose-response relationship around the threshold.

RfDs have served as surrogates for, or approximate values of, toxicity thresholds. The RfD is defined as "an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily oral dose exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime" (U.S. EPA, 1988). Based on this definition, doses at the RfD are not necessarily without risk. Based on the formula for estimating the RfD (Equation 12), the RfD is an estimate of a No-Observed-Adverse-Effect Level (NOAEL) that would be observed in a well-conducted, chronic epidemiologic study in the most sensitive human population (Swartout et al., 1998), assuming that other relevant toxicologic data exist; under these conditions, each uncertainty factor (UF) in the RfD equation would be reduced to 1.[37] Identification of a NOAEL depends on the background incidence rate in the unexposed population, spacing of the dose groups, the number of organisms on study, and study quality including identification of a biologically or statistically significant LOAEL in the study. The true value of a population toxicity threshold is likely between a study NOAEL and a LOAEL. A toxicity threshold must be below a LOAEL and it could be less than a study NOAEL for a sensitive individual (or for a sensitive subpopulation not considered during the development of an RfD).

$$RfD = NOAEL/UF \qquad \text{(Eq. 12)}$$

[37] The EPA adjusts NOAELS or Lowest Observed Adverse Effect Levels (LOAELs) identified in animal bioassays by dividing by the product of 5 different uncertainty factors (UF) that account for 1) differences between species, 2) differences within a population, 3) differences between a subchronic and chronic study, 4) the use of a study LOAEL instead of a NOAEL, and 5) an insufficient toxicologic database.

2.3.2. Population Toxicity Threshold for Methylmercury Exposures.

The U.S. EPA's oral RfD for methylmercury is based on epidemiologic studies conducted in the Faroe Islands and New Zealand (U.S. EPA, 2001a; NRC, 2000). EPA considered the continuous tests employed to evaluate the subjects' neurologic function using a benchmark dose (BMD) analysis. EPA interpreted a child's neurodevelopmental test response to be abnormal, if the test result was below the value associated with the score achieved by the 5^{th} percentile of the general population. Doses corresponding to a 5% increase above background in the probability of having a test result below the 5^{th} percentile of the general population were identified as the BMD_{05}. For combinations of tests and subgroups within the Faroese cohort, the values reported for BMD_{05} ranged from 41 to 393 ppb mercury in blood. A benchmark dose lower limit ($BMDL_{05}$) (the lower 95% confidence limit of the BMD_{05}) was calculated for each test in the three studies (listed in Table 2 of U.S. EPA, 2001a). The estimates of the values for the $BMDL_{05}$ converged around average methylmercury intakes of approximately 1 µg/kg-day. U.S. EPA's (2000) draft Benchmark Dose Guidance summarized a series of studies showing the $BMDL_{05}$ for reproductive and developmental toxicity data to approximate the NOAEL.

EPA applied an intraspecies UF of 10 to the $BMDL_{05}$ when developing the methylmercury RfD. The Agency cited inter-individual toxicokinetic variability associated with the relationship between the ingested dose and the cord blood mercury concentrations reported in the Faroes. The Agency also cited possible toxicodynamic

variability and uncertainty, noting the high degree of homogeneity in the Faroese population. The EPA oral RfD for methylmercury is 0.1 µg/kg-day.[38,39]

In the subsequent analysis, we estimate benefits alternatively assuming a population toxicity threshold for methylmercury does and does not exist. The data are insufficient to exclude either possibility. To date, there does not appear to be substantial toxicologic evidence of a biological population threshold, but its possibility cannot be eliminated.

Based on the EPA's BMD analysis, a range of plausible population toxicity thresholds exists. The range of values likely extends from 0 µg/kg-day (i.e., no apparent threshold) to doses between the BMD_{05} and the $BMDL_{05}$. EPA's RfD (0.1 µg/kg-day) is used as a surrogate based on convenience; we note that this value may be a conservative (smaller) estimate of a threshold, if one exists.

Methylmercury is a neurodevelopmental toxicant. The threshold applies to maternal methylmercury intake during pregnancy and for a period of approximately 1.5 to 3 months prior to conception. The half-life of methylmercury in human blood is reported to be 44 days (Clarkson, 1997); thus, exposures prior to conception could affect the developing fetus.

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[38] Typically, noncancer environmental human health risk assessments use the hazard quotient (HQ). The HQ compares the dose (D) to the reference dose (RfD), but does not quantify risk *HQ = D/RfD*. RfDs are derived from a NOAEL, LOAEL, or benchmark dose, with uncertainty factors generally applied to reflect data limitations such as uncertainty associated with inter-individual variability. An HQ of greater than 1 implies that an individual is exposed above a 'safe' level, but does not predict risk or identify a threshold.

[39] One component of the uncertainty factor used to estimate the EPA's RfD was inter-individual toxicokinetic variability associated with the relationship between the ingested dose and the cord blood mercury concentration. The data of Grandjean et al., (1999) describe the relationship between cord blood methylmercury concentrations and neurological tests. Stern and Smith (2003) published a quantitative analysis of this relationship showing the ratio of methylmercury concentrations in cord blood to maternal blood to be approximately 1.7. Because the EPA assumed that this ratio was 1, (i.e., maternal blood and cord blood methylmercury concentrations are the same), this study suggests RfD should be reduced by a factor of 1.7, unless the EPA would modify the UF for inter-individual variability.

2.3.3. Analysis of Risk Assuming Threshold.

2.3.3.1. Estimating Size of Population Above Threshold —

The number of births above the possible neurotoxicity threshold in each population was estimated as the product of the number of births in each population and the fraction of females of childbearing age in each population above the RfD. We note that Mahaffey et al. (2004) report that roughly 8% of children may be born to women whose methylmercury intake exceeds the threshold of 0.1 μg/kg day.

2.3.3.2. Methylmercury Intake Rate in Population Above RfD —

For the fraction of each population with an intake rate above the possible threshold (i.e., the methylmercury RfD (0.1 μg/kg day), we estimated the conditional daily mean methylmercury intake rate. This rate was converted to a blood mercury concentration (see Equation 1) and then a conditional mean hair methylmercury concentration. Based on Equation 11, the average concentration of methylmercury in the hair at the RfD was subtracted from the populations' conditional mean hair methylmercury concentrations. This estimate of incremental (above threshold) concentration was combined with the slope in Equation 10 to estimate the IQ loss for the average individual in each annual birth cohort. To estimate the population impact, the average IQ changes per child were multiplied by the number of births occurring in a population to women whose methylmercury intake exceeded the threshold.

2.4. DOSE-RESPONSE FOR ACUTE MYOCARDIAL INFARCTION (AMI) AND ALL CAUSE MORTALITY (ACM)

The predicted myocardial risks associated with methylmercury exposures should be interpreted with caution. Most of the evidence of such risks is based on observations

from a single cohort. Additionally, a great deal of evidence indicates that fish consumption in general protects individuals from incurring adverse cardiac events.

If any of the three cardiotoxic modes of action for methylmercury that were proposed by Salonen et al. (1995) are true (see Footnote 18), then myocardial risks likely increase with blood mercury concentrations, which in turn are highly influenced by fish consumption. Human studies were not designed to examine whether a dose-response threshold exists for the effects evaluated. In Section 2.4.1, we develop dose-response functions for increased incidence of myocardial infarction and all cause mortality. We use the regression coefficients estimated by Salonen et al. (1995) when they modeled risk using the mercury concentration as a continuous variable with the statistical controls using the Cox Proportional Hazards model (model 2 in Table 12).

In Section 2.4.2, we discuss annual background rates of myocardial infarctions and deaths in the U.S. We also discuss the application of the dose-response models developed in two ways; first, we assume that the models apply only to males that consume non-fatty freshwater fish (similar to those described by Salonen) and, second, we assume that, if any of the three potential mechanisms of action proposed by Salonen are true, they could be generalized to and occur in the adult male and female population as well. Thus, we assume the regression coefficients observed in this cohort can be externally generalized to the U.S. population despite differences between these populations and the types of fish consumed. We note that, if there is an antagonistic relationship between methylmercury and polyunsaturated fatty acids then the slope likely would be less steep for consumers of fish containing higher levels of polyunsaturated fatty acids. We also assume that the myocardial events and the

premature deaths observed in this Finnish male cohort are related and use the information on the age of myocardial events to estimate the age at which a premature death occurred.

2.4.1. Estimating the Dose-Response Functions.

Using a Cox Proportional Hazards model, Salonen et al. (1995) estimated a relative risk for non-fatal and fatal myocardial infarctions to be 1.068 per 1 ppm hair mercury. The Cox Proportional Hazards Model, presented in Equation 13, assumes that changes in levels of the independent variables, such as hair mercury concentrations, will produce proportional, time independent, changes in the hazard function.

$$h(t) = h_o exp(\beta C_h) \quad \text{(Eq. 13)}$$

where:

- h(t) = hazard function at time t
- h_o = baseline hazard
- β = regression coefficient (ppm^{-1})
- C_h = exposure measure hair mercury concentration (ppm)

The relative risk can then be estimated by the following equation:

$$RR = \frac{h(t)}{h_o} = exp(\beta C_h) \quad \text{(Eq. 14)}$$

Rearranging,

$$ln(RR) = ln\left(\frac{h(t)}{h_o}\right) = \beta C_h \quad \text{(Eq. 15)}$$

Thus,

$$\beta = \frac{ln(\frac{h(t)}{h_o})}{C_h} \quad \text{(Eq. 16)}$$

When RR = 1.068 and C_h = 1 ppm, then β = 0.066 ppm^{-1}.

We substitute R_{AMI} for h_o and y for *h(t)*. For a given level of hair mercury, the incidence rate of acute myocardial infarctions (AMI) is calculated by the following equation:

$$y = R_{AMI} X exp(\beta C_h) \quad \text{(Eq. 17)}$$

where:

y = incidence rate (yr^{-1})

R_{AMI} = AMI background incidence rate (yr^{-1})

C_h = mean hair mercury concentration (ppm)

β = regression coefficient (ppm^{-1} yr^{-1})

We estimate the increased incidence of all cause mortality (ACM) in a similar manner. Using a Cox Proportional Hazards model, Salonen et al. (1995) estimated the relative risk for ACM to be 1.09 per 1 ppm increase in hair mercury concentration. Using Equation 16, we estimate a value for β of 0.086 ppm^{-1}, when C_h is 1.

2.4.2. Applying the Dose-Response Functions.

In this section, we discuss annual background rates of myocardial infarctions (R_{AMI}) and death (R_{ACM}) in the U.S. Then, we discuss the application of the dose-response models.

To avoid double-counting deaths, we include fatal myocardial infarctions with all cause mortality, and report separately non-fatal myocardial infarctions. In 2001, the AHA (2004) estimated that there were 865,000 new and recurrent myocardial infarctions in the U.S. population. Given that these myocardial infarctions lead to 184,800 deaths in 2001, roughly 21% of all AMI resulted in death. Specifically, among males, there were estimated to be 520,000 new and recurrent myocardial infarctions and these

resulted in 95,900 deaths (18.44%). Thus, in males, we estimate the non-fatal AMI by reducing the total AMI estimate by 18.44 %. Among females, there were estimated to be 345,000 new and recurrent myocardial infarctions and these resulted in 88,900 deaths (25.77%). Thus, we estimate non-fatal AMIs by reducing the total number of AMI by 25.77%.

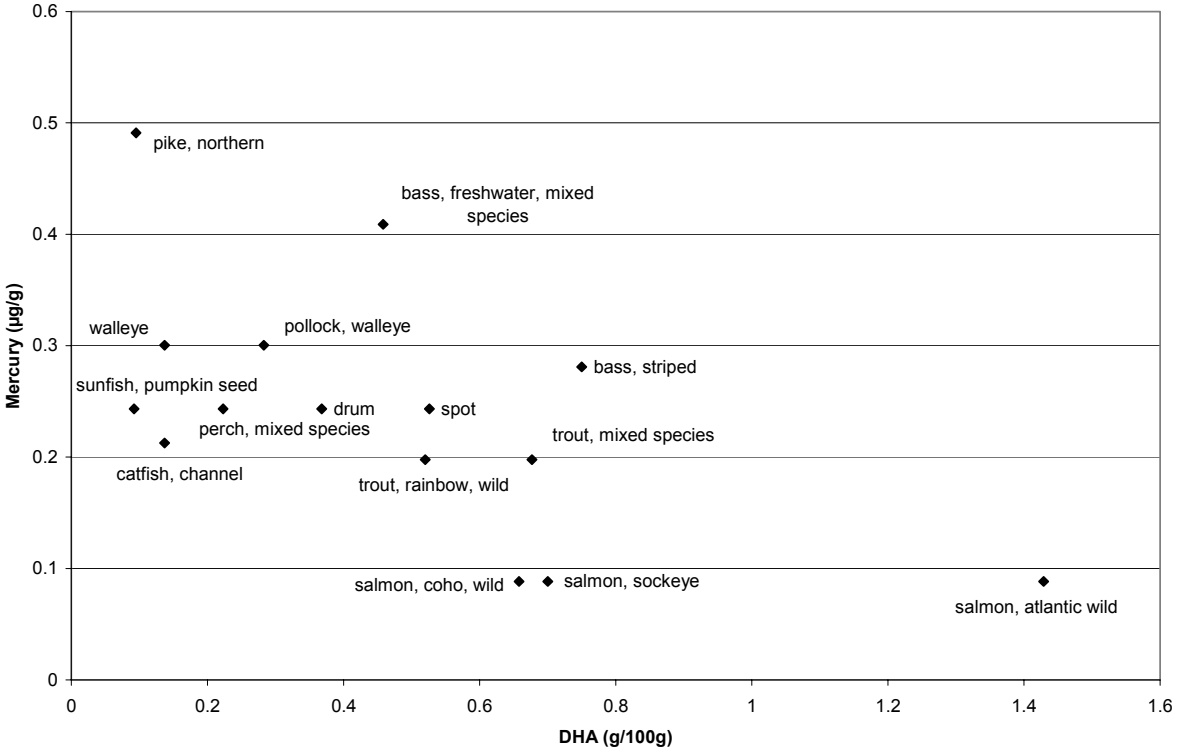
The average age of a first MI is 65.8 among men and 70.4 among women (AHA, 2004). We make the simplifying assumption that AMI only occur in people above age 39. Based on the 2000 Census data (U.S. Census Bureau, 2004), there were 55,681,000 males and 63,705,000 females above the age of 39 years in the U.S. in 2000; males and females above this age comprise approximately 19.8% and 22.6% of the total U.S. population, respectively. If all of the estimated 520,000 non-fatal AMI that occur annually in males occur in this population group, then the annual background rate of AMI in males above 39 years of age is roughly 9×10^{-3}. If all of the estimated 345,000 non-fatal AMI that occur annually in females occur only in this population group, then the annual background rate in females above 39 years of age is roughly 5×10^{-3}.

In 2001, life expectancy, conditional on living to age 65 (males) and 70 (females) was 86 years and 81 years for U.S. females and males, respectively (Arias et al., 2003). If we assume that the age of death coincides with the average age of first myocardial infarct, then, on average, women and men would lose 16 years of life due to increased mortality associated with methylmercury. We emphasize that this is extremely uncertain and conducted as a bounding exercise. We note that the age of the males at the initiation of the Salonen cohort was less than 65 years.

We apply these estimated annual risk coefficients in two ways. In the first approach, we limit the application of the non-fatal AMI and all cause mortality risk coefficients to adult males that consume northern pike; this first application is based on the discussion of the characteristics of the fish implicated in the diets of Finnish male cohort in the Salonen et al. (1995) study. Like the Finnish fish consumed in the study, northern pike are caught in freshwaters and, as Figures 5-7 illustrate, relative to other freshwater species, northern pike have low levels of DPA, DHA, and total polyunsaturated fatty acids and high concentrations of methylmercury. As Figures 5-7 illustrate, other fish may contain low levels of polyunsaturated fatty acids.

Figure 5

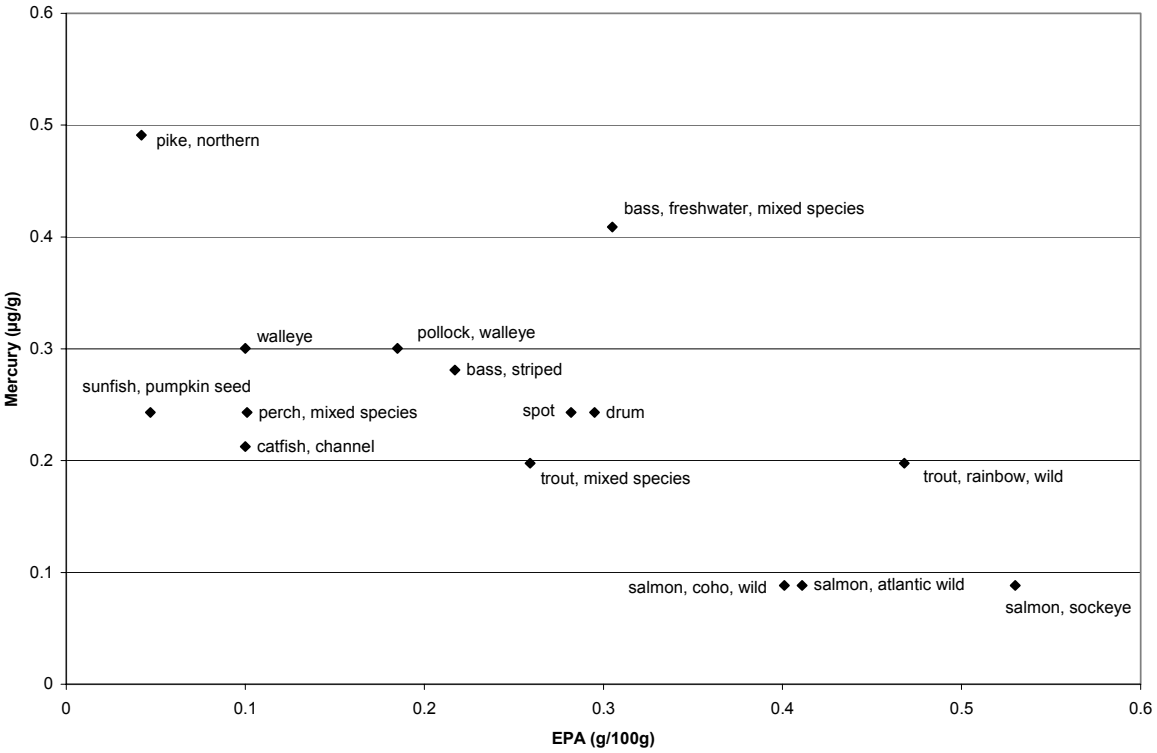
DHA and Methylmercury Levels in U.S. Freshwater Fish



Source of Mercury data: NLFWA Database, accessed 9/03
Source of DHA data: http://www.nal.usda.gov/fnic/foodcomp/, accessed 6/04

Figure 6

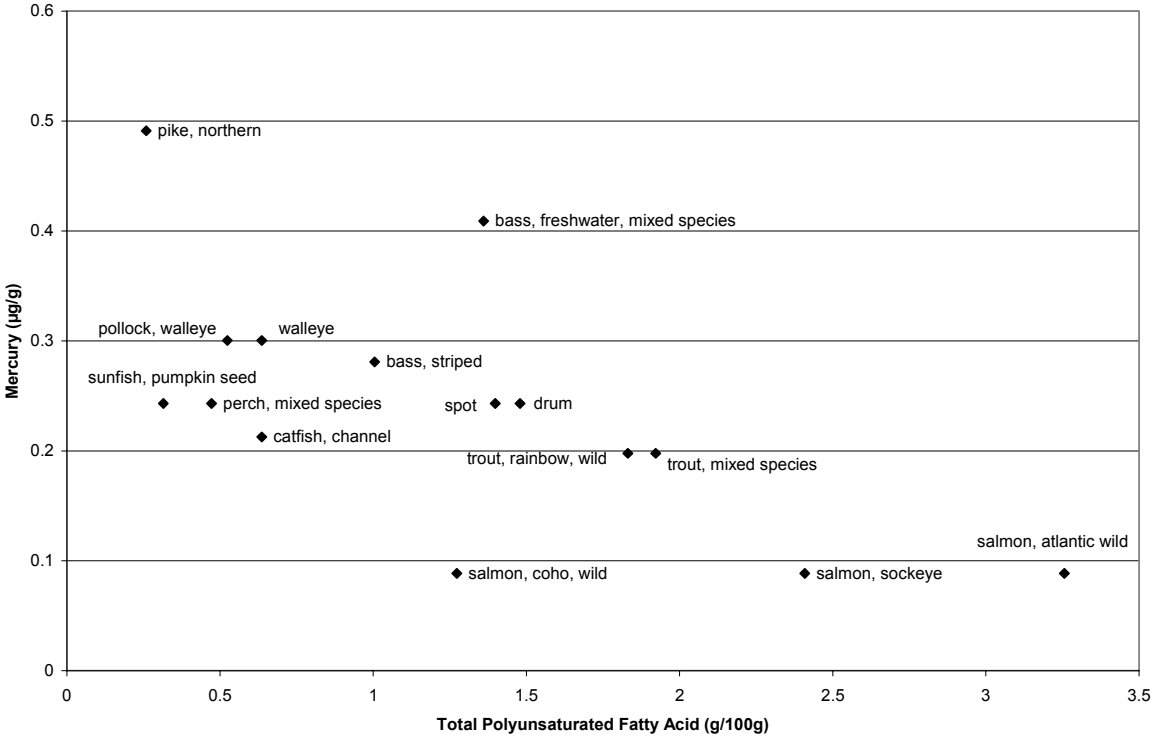
EPA and Methylmercury Levels in U.S. Freshwater Fish



Source of Mercury data: NLFWA Database, accessed 9/03
Source of EPA data: http://www.nal.usda.gov/fnic/foodcomp/, accessed 6/04

Figure 7

Total Polyunsaturated Fatty Acid and Methylmercury Levels in U.S. Freshwater Fish



Source of Mercury data: NLFWA Database, accessed 9/03
Source of Polyunsaturated Fatty Acid data: http://www.nal.usda.gov/fnic/foodcomp/, accessed 6/04

Based on the species targeting information in U.S. FWS (2001), we estimated the fraction of the freshwater fish consuming population that consumes northern pike in four of the five regions (pike are not listed among the top 10 species sought in the Southeastern Region). We assumed that the frequency at which pike were targeted corresponded to the share of the non-commercial freshwater fish-consuming population that consumes pike. Less than 10% of freshwater fishers report targeting northern pike in each of the four regions.

In a separate application, we assume that the myocardial toxicity and premature mortality associated with methylmercury is independent of fish type and polyunsaturated fatty acid levels. We apply the non-fatal AMI and all cause mortality dose response estimates to all males and females above the age of 39 years in separate calculations. Again, the external generalization of the Salonen data and the resulting application is extremely uncertain.

2.5. VALUING CHANGES IN CHILDREN'S INTELLIGENCE

Available economic research provides little empirical data for society's willingness to pay (WTP) to avoid a decrease in an infant's IQ. Models have been developed to address the monetization of *a subset* of the effects of decreased IQ. These effects would represent components of society's WTP to avoid IQ decreases. Employed alone, these monetized effects should underestimate society's WTP.

This section estimates the impact of small, permanent changes in IQ on lifetime earnings. This section has four parts. Section 2.5.1 estimates proportional impact of a 1 point change in IQ on lifetime earnings. Section 2.5.2 quantifies average lifetime earnings for individuals born in the U.S. in the year 2000. Section 2.5.3 combines the results of Sections 2.5.1 and 2.5.2 to estimate the absolute impact of a 1 point change in IQ on lifetime earnings. Finally, Section 2.5.4 considers the impact of changes in IQ on costs associated with remedial education. This last cost category reflects the impact of population shifts in IQ on the proportion of individuals in tail categories of the IQ distribution.

We note several caveats. First, our results serve as a conservative estimate (i.e., likely lower bound) of the total value individuals place on changes in IQ because

they may also value such changes independently of their impact on lifetime earnings. Second, our application of the results from this analysis to the assessment of intrauterine methylmercury exposures makes the implicit assumption that the impact of methylmercury on IQ is permanent even though epidemiological evidence of such an impact has only been reported in children up to 7 years of age (Grandjean et al., 1997, 1999).[40] Finally, our estimate does not account for the value of uncompensated labor (e.g., work in the home and volunteer labor), although it is plausible that the value of labor in these sectors could be similarly affected by changes in IQ.

2.5.1. Proportional Impact of a One-Point Change in IQ on Lifetime Earnings.

We adapt an existing cost-of-illness model (Schwartz, 1994; Salkever, 1995) to examine the influence of small changes in IQ on lifetime earnings. The original model, which was developed as part of an economic assessment of childhood lead exposures, posited that an individual's lifetime earnings (E) are equal to the product of the average wage rate (W) and the participation rate (P). Differentiating this equation with respect to IQ yields the relationship,

$$\frac{dE}{dIQ} = P\frac{dW}{dIQ} + W\frac{dP}{dIQ} + \frac{dW}{dIQ}\frac{dP}{dIQ} \qquad \text{(Eq. 18)}$$

(The term, dE/dIQ, refers to the change in lifetime earnings with respect to IQ; dW/dIQ refers to the change in annual wage rate with respect to IQ; dP/dIQ refers to the change in workforce participation rate with respect to IQ.)

[40] We note that the recent report from the Faroe Islands cohort presents evidence of neurotoxicity as measured by delays in auditory evoked potentials, but does not report results of IQ testing among children 14 years of age.

Dropping the second order term, $\frac{dW}{dIQ}\frac{dP}{dIQ}$, which we assume to be small relative to the remaining terms, and rearranging the remaining terms in Equation (18) yields

$$\frac{dE}{dIQ} = E\left(\frac{dW/dIQ}{W} + \frac{dP/dIQ}{P}\right) \qquad \text{(Eq. 19)}$$

Thus, the change in lifetime earnings equals the product of the present value of those earnings (E) and the sum of the proportional change in wages associated with the change in IQ and the proportional change in workforce participation associated with a change in IQ.

As illustrated in Figure 8, this model assumes that *in utero* methylmercury exposures affect the wage rate and participation rates both directly and indirectly (Schwartz, 1994; Rowe et al., 1995; Salkever, 1995; U.S. EPA, 1997b), via their impact on scholastic attainment and the subsequent impact of scholastic attainment on both wages and labor force participation. Salkever (1995) developed parameters to quantify these direct and indirect impacts (Table 22).

Salkever estimated these values from a series of regression analyses using information obtained in the National Longitudinal Survey of Youth (NLSY, 1990) and data provided by Schwartz (1994). The NLSY data include annual earned income, educational attainment data, and results of intelligence tests that Salkever used as a proxy for IQ. The data are drawn from surveys administered to a random sample of U.S. residents.

Figure 8

Model for Relationship Between IQ and Wages and Labor Force Participation

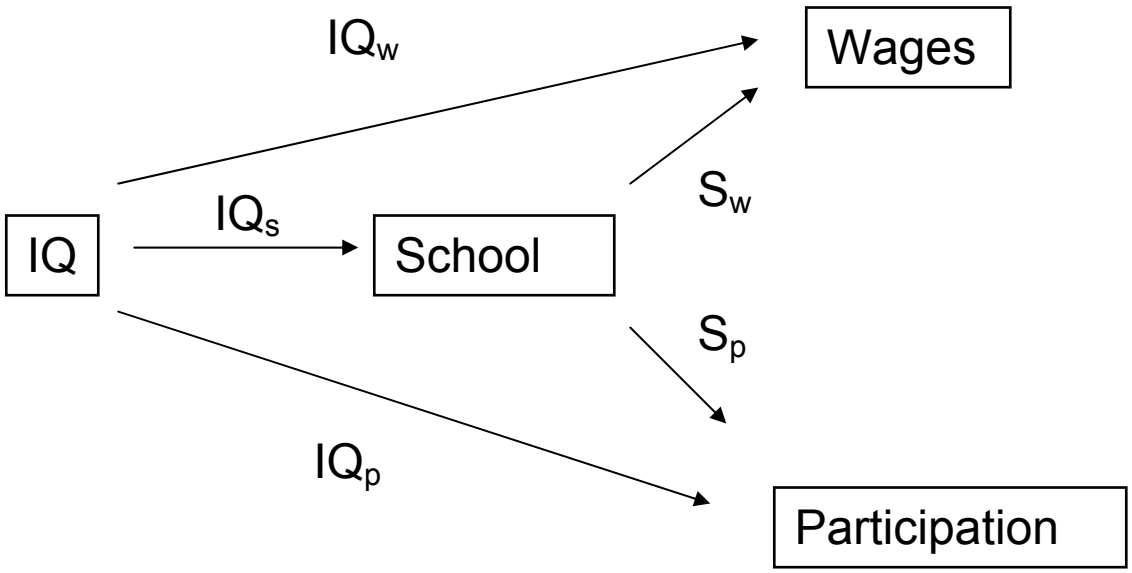


Table 22. Parameter Values Developed by Salkever (1995)

Effect	Symbol	Male	Female
Direct impact of a 1 IQ point change on:			
Years of schooling	IQ_S	0.1007	0.1007
Workforce participation probability	IQ_P	0.0016	0.0037
Wages (proportional wage change)	IQ_W	0.0124	0.014
Direct impact of a 1 year of schooling change on:			
Workforce participation probability	S_P	0.0035	0.0282
Wages (proportional wage change)	S_W	0.049	0.10

Salkever's model consists of three equations. The first model equation combines the direct and indirect effects of IQ in order to quantify its total proportional impact on labor force participation. In particular, $\frac{dP/dIQ}{P} = IQ_P + IQ_S S_P$, where the first term on the right side of the equation represents the direct effect of IQ on labor force participation, and the second term represents its indirect effect (i.e., the effect of IQ on the level of education attained [S_p]). Similarly, $\frac{dW/dIQ}{W} = IQ_W + IQ_S S_W$.[41] Substituting the right side of these two equations into Equation (19) yields

$$\frac{dE}{dIQ} = E\left[\left(IQ_W + IQ_S S_W\right) + \left(IQ_P + IQ_S S_P\right)\right] \qquad \text{(Eq. 20)}$$

Substituting the values from Table 22 into Equation 20 yields $E \times 1.9\%$ for males and $E \times 3.2\%$ for females. Using the labor force participation rates for males and females, Salkever (1995) estimated a weighted average value of $E \times 2.4\%$. This adjustment is used to estimate the influence of IQ point changes on lifetime earnings.

2.5.2. Baseline Lifetime Earnings Value (E).

This section estimates the value of E to be used in Equation 20 to quantify the impact of changes in IQ on lifetime earnings. Grosse (Appendix I in Haddix et al., 2003) estimated average lifetime earnings for each age and gender group. In his analysis, earnings were comprised of two broad components: wages/fringe benefits and household production. Wage estimates were based on the Current Population Survey (U.S. Census, 2001 Supplement as cited in Grosse, 2003) and included salary income,

[41] Derivative notation is used. For example IQ_W refers to the impact of a change in IQ on the proportional change in wages.

overtime pay, bonus pay, and self-employment earnings. Fringe benefits included health insurance and retirement pay. Grosse assumed that the average person worked 250 days per year. Household production included a number of activities such as cleaning, cooking, and child care, for which individuals are typically not compensated but are known to be valued; he assumed that household services need to be performed every day. Combining the data for men and women and using a 3% discount rate, Grosse (2003) estimated the present value of labor market earnings over a lifetime of an infant was $692,000 (2000$) (the term 2000$ refers to the value of a dollar in the year 2000). We use this estimate as a value of E in Equation 20. The discounted present value of an infant's lifetime labor market and household production was estimated to total $956,000 (2000$). As there is no evidence to suggest that decreased IQ alters the components of household production (although as previously noted it certainly may), we did not include the household production in the estimate. Previously, Rowe et al. (1995) and U.S. EPA (1997b) developed estimates of lifetime earnings. These estimates differ from those developed by Grosse because of apparent differences in discounting and annual earnings estimates.

2.5.3 The Absolute Impact of IQ Changes on Lifetime Earnings.

This section combines the findings from Sections 2.5.1 and 2.5.2 to calculate the impact of a 1 point change in IQ on the present value of lifetime earnings (evaluated at the time an individual is born). The product of the proportional change (2.39%) and baseline lifetime earnings ($691,830) yields a change of $16,500 (2000$).

We note that in their studies of lead's effect on earnings, Schwartz and Salkever included a term in their models representing the impact of that metal's toxicity on

educational attainment independent of the impact mediated by changes in IQ. We omitted this term from our model because there is no evidence suggesting that prenatal methylmercury exposures reduce educational attainment independent of IQ.

2.5.4. Remedial Education Costs and Shifts in the Population IQ Distribution.

While changes in IQ affect wages earned in a lifetime, they also change the fraction of the population that is considered to be mentally handicapped (i.e., individuals having an IQ <70).[42] We assume that when individuals fall below this threshold, additional educational resources are required. We estimate the cost of a shift in the population IQ distribution as the product of the incremental educational cost and the incremental probability of having an IQ <70.

2.5.4.1. Incremental Probability of IQ <70 —

To estimate this incremental probability, we assume that population IQ is normally distributed, and that the change in IQ is the same for all members of the population (i.e., the mean of the distribution shifts but the variance and other properties of the distribution remain unchanged). In general, the probability that a randomly selected individual has an IQ score less than 70 can be expressed in terms of the standard normal deviate Z, which is normally distributed with mean zero and standard deviation one. In particular,

$$P(IQ < 70) = P\left(Z < \frac{70 - \mu}{\sigma}\right) \qquad \text{(Eq. 21)}$$

[42] A shift in the IQ of a population would also decrease the number of highly intelligent individuals (e.g., the number of individuals with an IQ above 130). No data were identified that evaluated the economic productivity of this group or compared it to the population with IQ sources between 70 and 129, and so we make no adjustment for the change in number of people with IQs above 130.

where μ is the mean population IQ score and σ is the corresponding standard deviation. For the baseline conditions, μ = 100 and σ = 15, so that the proportion of individuals with an IQ below 70 is 2.28%. For a 1 point decrease in mean IQ (μ = 99), this proportion increases to 2.66%. Hence, a 1 point decline in mean IQ increases the probability that a randomly selected individual will have an IQ less than 70 by 0.38%. Similarly, a 1 point increase in mean IQ decreases this probability by 0.33%.

2.5.4.2. Incremental Costs Associated with IQ <70 —

We assume that individuals with IQ scores below 70 require additional educational resources compared to those with IQ scores above 70. We assume further that these incremental costs are approximately equal to the cost of providing part-time special education for children who remain in the regular classroom. U.S. EPA (1997b) applied the GNP price deflator to the annual cost of this service, as reported by Kakalik (1981) ($3,064, 1978$), to arrive at an annual cost of $6,318 (1990$). The GNP deflator does not adjust costs based on a basket of goods and services, but on the basis of all labor and property supplied by U.S. residents. Using the third quarter 2000 GNP deflator, we estimate the updated annual cost of such services to be $7,800 (2000$) (GNP Deflator 1990 = 87 and GNP Deflator 2000 = 107.08). Assuming that compensatory education begins at age 6 and continues through age 18 and that the annual discount rate is 3%, the present value of the compensatory education is $77,000 (2000$).

2.5.4.3. Cost of a One Point Shift in Mean IQ —

Multiplying the incremental probability that IQ will be less than 70 (0.38%) resulting from a 1 point drop in mean IQ and the present value at birth for compensatory

educational costs yields a cost per IQ point of $290. We note that this cost is small (roughly 2% of the total) compared to the impact on population earnings of $16,500 (2000$) (see Section 2.5.3).

2.6. HEALTH-RELATED QUALITY OF LIFE MEASURES FOR NEUROLOGICAL DECREMENTS

Health-related quality of life measures, such as Disability Adjusted Life Years (DALYs) and Quality Adjusted Life Years (QALYs), have been applied to neurological effects from methylmercury exposures. For example, QALYs are utility functions that may describe individual preferences for longevity and health-related quality of life associated with either temporary or permanent morbid conditions. The health-related quality of life is assigned a quantitative weight described as a utility weight. The utility weight is typically a value between 0 and 1. A value of 1 represents a perfect health state and 0 represents a health state equivalent to dead. For an individual's lifetime, the product of the utility weights and the duration of time an individual experiences these utility weights yields an estimate of an individual's QALYs. The value of reducing the health decrement associated with lowering *in utero* methylmercury exposures can be measured as the difference in QALYs with and without such a reduction in exposures over time (Hammitt, 2002; Pliskin et al., 1980). In Sections 2.6.1 and 2.6.2, we discuss the development of a utility weight for the neurologic decrements associated with methylmercury exposures in the New Zealand and Faroe Islands studies and we describe an exposure-response function for the health-related quality of life measures.

2.6.1. Utility Weight for Health-Related Quality of Life Associated with Neurodevelopmental Toxicity.

In this section, we examine the appropriate utility weight for neurological changes associated with methylmercury exposures. In the discussion of the New Zealand and Faroe Islands studies, we noted that the investigators reported no differences in the exposed and reference groups based on "observed" behaviors (i.e., in the absence of sophisticated neurological testing, the groups were indistinguishable). In these studies of environmentally relevant methylmercury exposures, the neurological effects associated with methylmercury exposures, although potentially permanent, appear to be subtle. These subtle effects are the basis of the RfD. In our literature review, we found no data that describe utility weights for the subtle neurologic decrements reported for methylmercury.

Several studies examined utility weights associated with cognitive decrements. Ponce et al. (2000) used QALYs to evaluate neurological effects associated with methylmercury exposures. They evaluated the risk of delayed talking using a dose-response function based on the Iraqi poisoning episode (Marsh et al., 1987). If speech development was delayed, the utility weight assigned to that health-related quality of life was a value of 0.9; the health decrement was 0.1. This was based on the work of Kind et al. (1982). In our review of Kind et al., we could not identify a description of the relevant neurological condition. Torrance et al. (1996) and Feeney et al. (2002) also developed a series of utility weights for cognitive decrements. Tables 23 and 24 describe the cognitive decrements associated health-related quality of life and the corresponding utility weights. We note that the descriptions of the health-related quality

of life for the neurocognitive decrements in these two studies suggest impacts that are substantially more severe than those reported by the New Zealand and Faroe Islands investigators. There appear to be no survey-based studies that report utility weights for the subtle neurological effects observed in the recent epidemiological literature. Although it is not possible to know the results of a study that has not been conducted, we assume that in such a study the decrement in the utility weight for the neurological effects associated with *in utero* methylmercury exposures would be valued at no more than 0.01.

Table 23. Description of Cognitive Decrement and Associated Utility Weight Based on Torrance et al. (1996)

	Description of Levels for Health Utilities Index Mark 2: Cognition	Multiattribute Function on Dead Healthy Scale
1	Learns and remembers school work normally for age	1.00
2	Learns and remembers school work more slowly as judged by parents and teachers	0.95
3	Learns and remembers very slowly and usually requires special education	0.88

Table 24. Description of Cognitive Decrement and Associated Utility Weight Based on Feeny et al. (2002)

	Description of Levels for Health Utilities Index Mark 3: Cognition	Multiattribute Function on Dead Healthy Scale
1	Able to remember most things, think clearly and solve day-to-day problems	1.00
2	Able to remember most things, but have a little difficulty when trying to think and solve day-to-day problems	0.92
3	Somewhat forgetful, but able to think clearly and solve day-to-day problems	0.95
4	Somewhat forgetful and have a little difficulty when trying to think and solve day-to-day problems	0.83

2.6.2. Exposure-Response Function for Health-Related Quality of Life Weights.

There are no studies that describe an exposure-response relationship appropriate for the health-related quality of life weights. We assumed that all children born to mothers above the RfD would incur a neurodevelopmental effect and that this health effect would persist throughout their lives. We assume these IQ losses to cause similar neurodevelopmental effects that would be valued at no more than a utility weight decrement of 0.01. We assume that the life expectancy of children born is 77.2 years (CDC/NCHS, 2003). Combining these values yields an estimated loss of 0.77 QALYs for each child born to a mother with exposure above the RfD.

2.7. VALUING CHANGES ASSOCIATED WITH ADVERSE MYOCARDIAL EVENTS AND ALL CAUSE MORTALITY

Section 2.7 is divided into three subsections. Section 2.7.1 discusses the costs of lost productivity and medical costs associated with a non-fatal and fatal myocardial infarct. Section 2.7.2 discusses the value of a statistical life, an estimate of a group's

willingness-to-pay to reduce the risk of death. Section 2.7.3 discusses the utility weights associated with non-fatal and fatal myocardial infarctions.

2.7.1. Cost-Of-Illness Estimates For Myocardial Infarctions and All Cause mortality.

Then a non-fatal myocardial infarct occurs, individuals and societies incur medical costs for both urgent and continuing treatment and costs associated with lost productivity while the individual convalesces. Blake et al. (2003), citing an Appendix in Gold et al. (1996), estimate the lifetime costs of a myocardial infarction to be between $37,030 and $83,290 depending on the age and sex of the case. These costs, which are discounted at 3%, include hospitalization, physician costs, procedural costs, and annual follow-up costs. As an analytic simplification, we assume that the myocardial infarct occurs at ages 65 and 70 in males and females, respectively. The specific costs for a 65-year-old male and a 70-year-old female were estimated to be $46,000 and $43,000 (2000$), respectively (Blake et al., 2003).

Grosse (2003) estimated the value of a lost day to be $150/day (2000$). This estimate included annual earnings and the value of annual household services; he divided these annual estimates by 365 days to estimate loss of a typical day. For simplification, we assume that individuals who survive a myocardial infarct are unable to perform these tasks for 6 weeks. Thus, we estimate the lost productivity associated with a non-fatal myocardial infarct to be $6100 (2000$). For cases of nonfatal myocardial infarctions, we sum the medical and lost productivity costs.

As a simplification, we assume that the men and women, who are predicted to die in the analysis due to methylmercury exposure, die at ages 65 and 70 years,

respectively. These are the average ages for first myocardial infarct in the U.S. population (AHA, 2004). For men aged 65 years and women aged 70 years, Grosse estimated the present value of earnings and household production, discounted at 3%, to be $274,000 and $151,000 (2000$), respectively. We add the medical costs associated with the nonfatal cases (likely an overestimate of medical costs) and use these estimates in our cost-of-illness estimate for males and females.

2.7.2. Value of a Statistical Life.

The value of a statistical life (VSL) can also be used to estimate the value of a fatality. The VSL is a measure of an individual's willingness to pay to avoid incurring a small change in the risk of dying (Hammitt, 2000). The EPA currently treats the VSL as independent of age,[43] and the societal VSL is the Agency's standard approach to estimating a dollar value of mortality benefits of environmental regulations. In this analysis, we update the VSL to $5.9 million (2000$). This is based on an update of previous EPA estimates of $5.6 million (U.S. EPA, 1997b). Estimates of appropriate values for VSL generally vary between $3 million and $9 million (Mrozek and Taylor, 2002; Viscusi and Aldy, 2003).

2.7.3. Health-Related Quality of Life Utility Weights for Myocardial Infarctions.

In this section, we discuss estimates of utility weights for individuals incurring non-fatal myocardial infarctions. Blake et al. (2003) present a utility weight of 0.9 for survivors of myocardial infarct. We assume this decrement of 0.1 to the affected

[43]The EPA Guidelines for Preparing Economic Analyses (U.S. EPA, 2000) suggest that adjusting the VSL based on age may be appropriate.

individuals' utility persists. Given our assumptions about the age at which a heart attack occurs and that life expectancy of men aged 65 is 81 years of age and women age 70 is 86 years of age, men and women incurring a non-fatal myocardial infarct are estimated to lose 1.6 QALYs over their remaining lifetimes. Based on typical life expectancy in the U.S., males predicted to die of all cause mortality lose 16 QALYs and females lose 16 QALYs.

2.8. SENSITIVITY ANALYSES

Finally, we developed four sensitivity analyses that were limited in scope. In the first analysis, we evaluate the impact of freshwater fish consumption advisories. We assume that fish consumption advisories are issued for all bodies of water where fish methylmercury concentrations exceed 0.3 ppm. We assume that freshwater fish consumers are fully compliant with the advisories; that is, non-commercial fish having a methylmercury concentration above 0.3 ppm are not consumed by members of this population.

In the second, we estimated the distribution of methylmercury intakes among non-commercial freshwater fish consumers, assuming that individuals eat only one type of fish. We note that recreational anglers may target specific species for consumption and may not consume a representative mix of non-commercial species. Thus, some consumers targeting specific types of fish may have methylmercury intakes well above the average. In this sensitivity analysis, we assume no correlation between species consumed and quantity of fish consumed.

In the third, we evaluate the impact of alternatively doubling or halving the decrease in deposition that was predicted in Scenario 1 on methylmercury intakes in

freshwater fish consumers in a single region. The Northeast Region was selected for the example. Doubling the decrease might account for a response in fish that is greater than linear with respect to mercury deposition or possible underprediction of the deposition change associated with the REMSAD modeling. Halving the predicted decrease might account for a less than linear response in fish to decreased mercury deposition rates or a possible overestimate of the deposition rate change associated with the REMSAD simulation.

In the fourth sensitivity analysis, we re-evaluate the slope of the relationship between neurotoxicity and exposure in the case where a threshold exists. We estimated a slope of -0.6 IQ points per 1 ppm increase in mercury in hair. This estimate is based on three epidemiologic studies in which the reported hair mercury concentrations are generally between 0.5 and 30 ppm (These studies are reviewed in Section 1.5.1; see also NRC, 2000). If there is a threshold below which maternal methylmercury exposures have no effect on the developing fetal nervous system, the slope of the exposure-response function between the threshold and the maternal hair mercury concentration where responses are observed must be larger than if there is no threshold. To evaluate the sensitivity of our results to this possibility, we note that a relatively low level of maternal hair mercury at which effects may be observed is approximately 3 ppm. At this level, the estimated effect under the no threshold model is a loss of approximately 1.8 IQ points in the affected fetus (i.e., the product of -0.6 IQ points/ppm and 3 ppm). Under an alternative model in which there is a threshold around 1.3 ppm hair methylmercury, the slope of the exposure-response function between 1.3 and 3 ppm hair methylmercury must be -1.1 IQ points per increase of 1

ppm hair methylmercury in order to achieve the same effect (i.e., -1.8 IQ points per 1.7 ppm increase in hair methylmercury. We use this alternative slope value in the fourth sensitivity analysis.

3. RESULTS

This section has six parts. For each region, Section 3.1 describes the predicted current mercury deposition rates and the predicted changes in annual mercury deposition rates associated with the alternative emissions. Section 3.2 quantifies both the current and predicted changes in fish methylmercury concentrations. Section 3.3 describes both the current and the estimated changes in human exposures that are predicted to result from the alternative emissions scenarios. Section 3.4 quantifies the decreased risks of IQ point loss, as a consequence of the alternative mercury emissions. Cost-of-illness (COI) and QALY approaches are used to evaluate the averted cases. Section 3.5 quantifies the decreased risks of nonfatal AMI and ACM and uses COI, willingness-to-pay, and QALY approaches for valuation of the averted outcomes. Finally, Section 3.6 summarizes the results and provides a summary of the limited sensitivity analyses.

3.1. CHANGES IN ANNUAL MERCURY DEPOSITION RATES IN THE 8 REGIONS

The alternative emissions scenarios were predicted to result in decreased mercury deposition in each Region. For the All Other Waters Region, the Coastal Atlantic Region, and the Gulf of Mexico Region, the predicted changes in annual mercury deposition rates are summarized in Table 25. Table 26 summarizes the decreased deposition rates in the five freshwater regions. Under the alternative emissions scenarios, the largest decreases in mercury deposition are predicted in the Mid-Atlantic Region (9% and 10% decreases under Scenarios 1 and 2, respectively). Modest decreases are predicted for the Northeast, Midwest, and Southeast Regions (decreases range from 3% to 6%). The predicted decreases in the West, Atlantic

Coastal and Gulf of Mexico Regions are relatively small (roughly 1%). The reported standard deviations indicate predicted variability in deposition across grid cells within a region. These estimates of the spread of the predicted changes in deposition are highest in the Southeast and Mid-Atlantic regions.

Table 25. Predicted Percent Decreases in Mercury Deposition to the Coastal Atlantic Ocean Region, the Gulf of Mexico Region, and the All Other Waters Region Under CSI

	Coastal Atlantic Ocean	Gulf of Mexico	All Other Waters
Current Deposition Rate ($\mu g/m^2/yr$)	22.6	22.1	NA
Baseline 1	5.87%	3.52%	0.6%
Scenario 1	7.04%	3.89%	1%
Baseline 2	6.00%	3.54%	0.6%
Scenario 2	7.53%	4.29%	1.2%

Notes: Percent decreases are based on 2001 deposition levels.

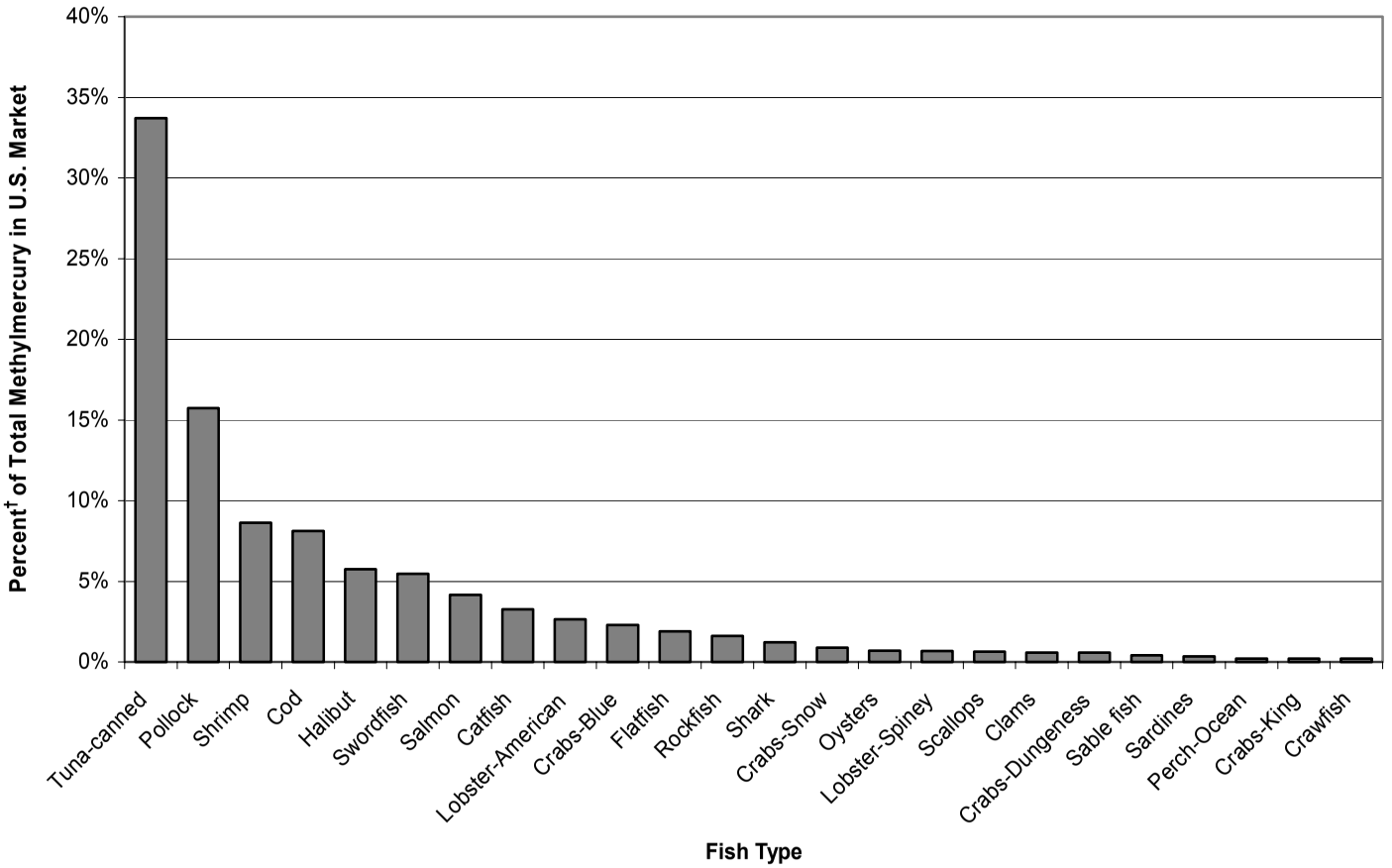
Table 26. Predicted Percent Decreases in Mercury Deposition in the Five Freshwater Regions Relative to Current Emissions

	Baseline1	Scenario 1	Baseline 2	Scenario 2
Northeast	Current deposition rate: 12.6 μg/m^2/yr (199 Receptors)			
Average Decrease	9%	12%	9%	13%
Standard deviation	9%	9%	9%	9%
MidAtlantic	Current deposition rate: 14.1 μg/m^2/yr (201 Receptors)			
Average Decrease	22%	31%	24%	34%
Standard deviation	12%	12%	12%	12%
Southeast	Current deposition rate: 10.2 μg/m^2/yr (661 Receptors)			
Average Decrease	17%	20%	18%	24%
Standard deviation	12%	12%	13%	12%
Midwest	Current deposition rate: 12.5 μg/m^2/yr (841 Receptors)			
Average Decrease	9%	12%	9%	14%
Standard deviation	7%	9%	8%	10%
West	Current deposition rate: 6.5 μg/m^2/yr (3001 Receptors)			
Average Decrease	3%	4%	3%	4%
Standard deviation	5%	5%	5%	6%

3.2. CHANGES IN FISH METHYLMERCURY CONCENTRATIONS IN THE 8 REGIONS

For commercial fish, a weighted mean methylmercury concentration was developed by weighting the mean methylmercury concentrations in each of the 24 types of fish (Figure 2) by the per capita consumption rates (Table 2). We calculated a weighted mean methylmercury concentration of 0.12 μg/g for current U.S. commercial fish. Figure 9 shows the percent contribution of each commercial fish type to total methylmercury in the U.S. market. The figure shows that, based on the product of the

methylmercury concentration and the per capita consumption rate, canned tuna, pollock, shrimp, cod, halibut and swordfish have the most influence on the weighted mean and that the remaining types of fish contribute minimally to the current per capita methylmercury intake.



†Estimate based on the product of per capita fish consumption rates and mean methylmercury concentrations of each type of fish (Carrington and Bolger, 2002)

Figure 9

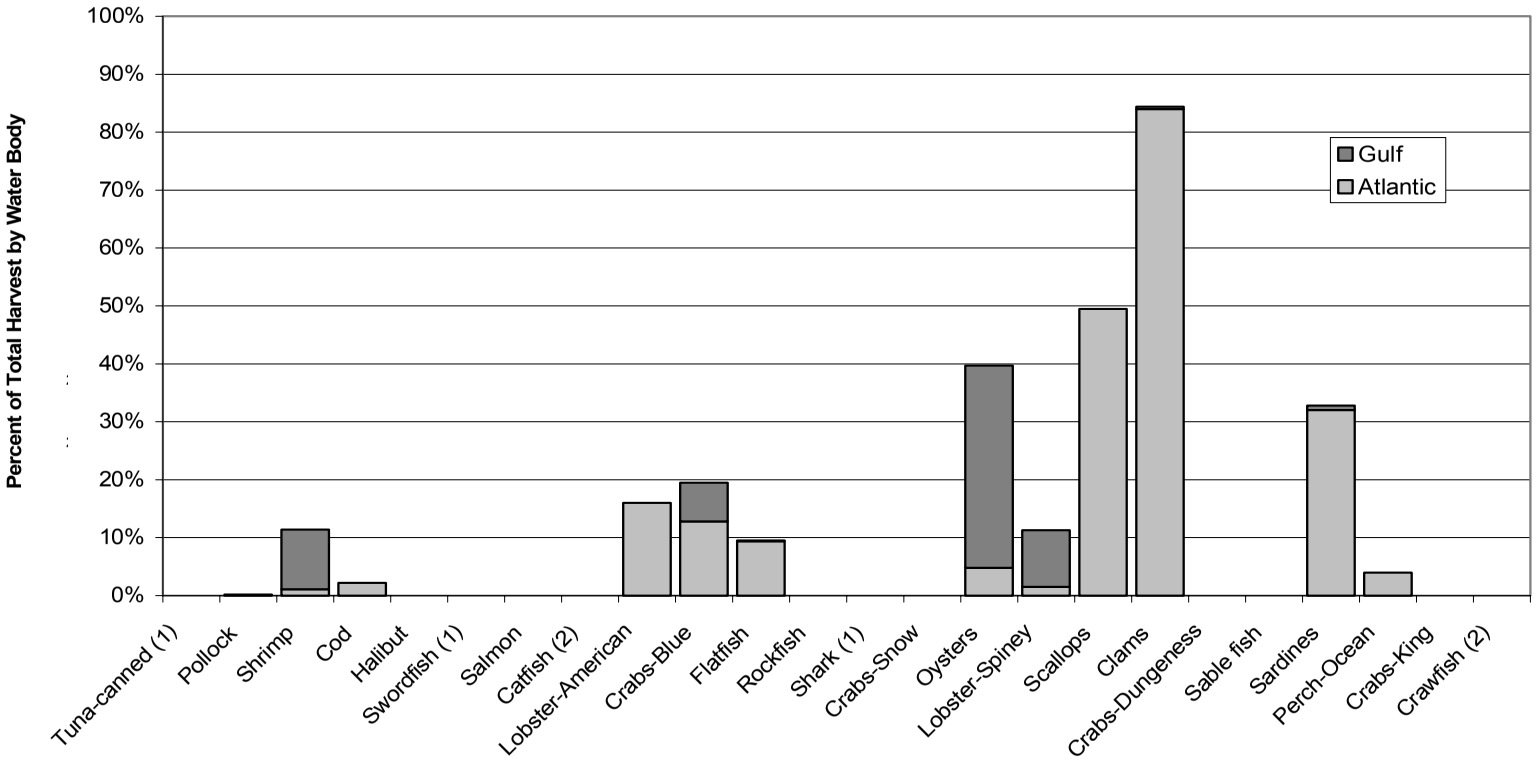
For "Top 24" Types of Fish in U.S. Commercial Seafood Market, the Percentage of Methylmercury Contributed by Fish Type

Table 27 shows the weighted mean methylmercury concentrations in commercial fish under current emissions and under each of the baselines and the alternative U.S. mercury emissions scenarios. This estimate was based on weighting mean methylmercury concentrations in each of the 24 types of fish by the quantity of the individual types of fish harvested from each of the four source waters, (i.e., the Coastal Atlantic Ocean, the Gulf of Mexico, All Other Waters and Aquaculture) and the predicted change in mercury deposition in each region (Table 25).

Table 27. Weighted Mean Methylmercury Concentrations in Commercial Fish

Commercial Fish	Concentration (µg/g)	Percent Change
Current	0.116	
Baseline 1	0.115	0.8%
Scenario 1	0.114	1.5%
Baseline 2	0.114	1.5%
Scenario 2	0.113	2.4%

Decreases of less than 3% are predicted in the weighted mean methylmercury concentrations of the commercial fish under the alternative scenarios. Most methylmercury exposures in typical commercial fish consumers (>75%) are a consequence of consuming six types of fish, canned tuna, pollock, shrimp, cod, halibut, and swordfish. (For example, based on methylmercury levels in tuna and the per capita consumption rate estimates, tuna accounts for 33% of per capita exposure.) For these six types of fish, Figure 10 shows the fractional contributions of the total catch from different bodies of water to total marketed quantities (NMFS, 2003).



(1) Migratory Species. Tuna, shark and swordfish are migratory species. The regions from which these fish were captured were assumed to not necessarily serve as a good predictor of change in methylmercury concentration.
(2) Aquaculture. Marketed catfish and crawfish are assumed to be raised in aquaculture.
Source: NMFS (2002)

Figure 10

Percent Contribution of the Atlantic Ocean and Gulf of Mexico Harvests to U.S. Commercial Market by Fish Type

Mercury concentrations in highly migratory fish,[44] like tuna and swordfish, are likely influenced by U.S. contributions only to the extent that U.S. emissions influence global atmospheric mercury pools, given the atmospheric mixing that likely occurs over the distance that U.S. mercury emissions must be transported to deposit into such remote food webs. Most pollock are caught in the Pacific Ocean. Given the prevalence of west-to-east atmospheric transport patterns in the U.S., the influence of U.S. power plant mercury emissions on pollock methylmercury levels is also likely proportional to the power plants' contribution to global atmospheric mercury pool. Shrimp and cod are primarily imported; thus, U.S. sources likely have a small contribution to methylmercury levels in these fish. Halibut, like pollock, are typically caught in the Pacific. Roughly 70% of marketed marine fish are imported into the U.S. (NMFS, 2003) and, thus, are assumed in this model to be harvested from the All Other Waters Region. The remaining 30% are harvested primarily from the U.S. continental shelves that extend roughly 200 miles off shore in the Atlantic Ocean, the Pacific Ocean, and the Gulf of Mexico. The small predicted changes in the methylmercury concentrations of most commercial fish result from the small influence of U.S. emissions sources on the methylmercury concentrations in our model; specifically, the top five sources of methylmercury in the diets of commercial seafood consumers per capita are likely influenced very little by U.S. mercury emissions. Figure 10 shows that, of the top 24 types of fish in the U.S. market, only clams and scallops are primarily harvested from

[44] Highly migratory species may consume prey over broad geographic ranges; consequently, we assumed that their methylmercury concentrations are influenced by mercury deposition over broad geographic areas and participation in foodwebs located in these different geographic areas. Note that we assumed that changes in mean methylmercury concentrations in these highly migratory species are based on estimated changes in global emissions as a result of the CSI. See Chapter 2.

waters that are likely to be heavily influenced by mercury emissions from U.S. sources. Thus, from a policy perspective, changes in U.S. emissions likely exert small changes in deposition rates of mercury at sites distant from U.S. shores and in the Pacific Ocean. Consequently, U.S. mercury emissions reductions exert small changes in the methylmercury concentrations of most commercial fish and, therefore, small changes in U.S. methylmercury exposures.

For non-commercial marine fish, we again assumed that consumers ate a mix of non-commercial marine fish obtained from either the Atlantic or Gulf. The specific mix was weighted by the estimated harvest weights for the top 10 types of fish caught recreationally in the Atlantic and Gulf (Tables 4 and 5, respectively). Table 25 shows the predicted change in deposition in the two regions and Table 28 shows the resulting predicted changes in the weighted mean methylmercury concentrations of non-commercial fish in the Atlantic and Gulf regions. Current fish methylmercury concentrations are predicted to be lower in the Atlantic fish than the Gulf fish, based on data in U.S. EPA (2003c). The predicted decrease in mercury deposition in the Atlantic Ocean Region (roughly 1.1% and 1.5% under Scenarios 1 and 2, respectively) is greater than the Gulf Region (less than 1% under both scenarios) (see Table 25). The assumption that each consumer eats a mix of 10 fish species results in an underestimation of the variability in the predicted intake distribution.

Table 28. Predicted Weighted Mean Non-commercial Fish Methylmercury Concentrations (µg/g)

	Atlantic Ocean	Gulf of Mexico
Current Fish Methylmercury concentration (µg/g)	0.28	0.40
Baseline 1 Fish Methylmercury concentration (µg/g)	0.26	0.39
Scenario 1 Fish Methylmercury concentration (µg/g)	0.26	0.38
Baseline 2 Fish Methylmercury concentration (µg/g)	0.26	0.39
Scenario 2 Fish Methylmercury concentration (µg/g)	0.26	0.38

Summary statistics for each of the types of fish from each of the five U.S. freshwater regions are presented in Tables 29-33, including the number of samples of each type of fish, the mean fillet methylmercury concentration and fish length. After adjustment for fish size (i.e., excluding fish smaller than are usually eaten), the NLFWA fish database is assumed to reasonably depict individual methylmercury concentrations of fish in different regions. Tables 29-33 also identify the assumed consumption frequency for each type of fish in each region; these frequencies were estimated from the U.S. FWS fish targeting data (see note at bottom of Tables 29-33). Given the assumption that consumers eat a mix of freshwater fish, these consumption frequencies were used (factor T in Equation 9) when developing the intake mix for individual Monte Carlo iterations in the simulation. The assumption that each consumer eats a mix of eight or nine types of fish results in an underestimate of the likely variability in the predicted intake distribution. Based on the predicted changes in deposition in the five freshwater regions (Table 26), the largest decreases in mercury deposition are predicted in the Mid-Atlantic Region, under the alternative emissions scenarios. There is very little decrease in the West Region (see discussion in Section 3.1).

Table 29. Northeastern Fish Consumption Data

Statistic	Crappie	Panfish	White and Striped Bass	Black Bass	Catfish	Northern Pike, Pickerel and Muskie	Trout	Salmon
Consumption Frequency* (unitless)	0.02	0.11	0.07	0.33	0.04	0.10	0.29	0.06
Sample Size	3	1131	121	755	237	34	173	8
Mean MeHg Concentration (ppm)	0.38	0.37	0.35	0.57	0.17	0.66	0.31	0.22
Standard Deviation MeHg Concentration (ppm)	0.18	0.28	0.22	0.35	0.17	0.37	0.24	0.20
Mean Length (inches)	10.03	8.08	24.61	14.58	9.84	18.42	16.97	12.56
Std. Dev. Length (inches)	1.48	1.78	5.32	1.88	2.12	3.63	5.90	5.57

*Consumption frequency refers to the relative targeting frequency for region. This was used as a surrogate for consumption frequency.

Table 30. MidAtlantic Fish Consumption Summary Data

Statistic	Crappie	Panfish	White and Striped Bass	Black Bass	Catfish	Walleye and Sauger	Northern Pike, Pickerel and Muskie	Trout
Consumption Frequency* (unitless)	0.07	0.10	0.12	0.32	0.12	0.04	0.03	0.21
Sample Size	25	20	190	161	186	5	49	17
Mean MeHg Concentration (ppm)	0.14	0.14	0.18	0.69	0.14	0.10	0.80	0.22
Std. Dev. Concentration (ppm)	0.08	0.13	0.21	1.03	0.20	0.02	0.67	0.27
Mean Length (inches)	7.68	6.18	25.19	14.68	15.78	14.73	18.86	16.84
Std. Dev. Length (inches)	1.82	0.54	6.34	1.80	4.12	2.32	3.12	5.42

*Consumption frequency refers to the relative targeting frequency for region. This was used as a surrogate for consumption frequency.

Table 31. Southeastern Fish Consumption Data

Statistic	Crappie	Panfish	White and Striped Bass	Black Bass	Catfish	Walleye and Sauger	Trout
Consumption Frequency* (unitless)	0.19	0.16	0.12	0.28	0.20	0.01	0.04
Sample Size	952	1914	99	5431	1335	14	43
Mean MeHg Concentration (ppm)	0.26	0.25	0.21	0.62	0.29	0.28	0.04
Std. Dev. MeHg Concentration (ppm)	0.32	0.24	0.17	0.53	0.37	0.22	0.04
Mean Length (inches)	11.55	7.94	18.64	15.42	15.99	18.48	9.11
Std. Dev. Length (inches)	8.90	3.43	4.70	5.18	8.49	2.34	2.48

*Consumption frequency refers to the relative targeting frequency for region. This was used as a surrogate for consumption frequency.

Table 32. Midwest Fish Consumption Summary Data

	Crappie	Panfish	White and Striped Bass	Black Bass	Catfish	Walleye and Sauger	Northerns, Pickerel and Muskie	Trout	Salmon
Consumption Frequency* (unitless)	0.15	0.23	0.07	0.19	0.11	0.14	0.07	0.03	< 0.01
Sample Size	905	1824	100	1446	617	5163	2448	1404	322
Mean MeHg (ppm)	0.17	0.15	0.25	0.35	0.22	0.45	0.50	0.24	0.10
Std. Dev. MeHg conc(ppm)	0.12	0.12	0.17	0.21	0.36	0.34	0.37	0.22	0.09
Length (inches)	10.76	7.84	13.57	14.58	15.91	19.30	28.17	20.24	20.88
Std. Dev. (inches)	17.72	1.51	1.18	1.84	5.44	3.12	3.37	5.78	4.82

*Consumption frequency refers to the relative targeting frequency for region. This was used as a surrogate for consumption frequency.

Table 33. Western Fish Consumption Summary Data

	Crappie	Panfish	White and Striped Bass	Black Bass	Catfish	Walleye and Sauger	Northern Pike, Pickerel and Muskie	Trout	Salmon
Consumption Frequency* (unitless)	0.10	0.05	0.08	0.18	0.16	0.05	0.02	0.30	0.07
Sample Size	153	132	105	353	461	122	48	236	338
Mean MeHg Concentration (ppm)	0.32	0.30	0.41	0.45	0.25	0.67	0.46	0.21	0.12
Standard Deviation MeHg Concentration (ppm)	0.24	0.25	0.29	0.38	0.21	0.59	0.26	0.35	0.22
Mean Length (inches)	9.60	7.80	16.26	14.54	16.03	20.11	31.06	12.45	22.43
Std. Dev. Length (inches)	2.53	1.88	3.91	2.04	4.62	4.34	4.74	4.47	6.31

*Consumption frequency refers to the relative targeting frequency for region. This was used as a surrogate for consumption frequency.

Table 34. Estimated Population Sizes

	Estimated Angler Population	Estimated Non-commercial Fish Consumer Population	Estimated Women of Child-bearing Age	Estimated Annual Childbirths	Men >39 Years of Age	Women >39 Years of Age
Atlantic	5,012,000	10,024,000	2,205,000	137,000	1,983,000	2,269,000
Gulf	2,897,000	5,793,000	1,274,000	79,000	1,146,000	1,311,000
Northeast	2,471,000	4,942,000	1,087,000	67,000	978,000	1,119,000
Mid-Atlantic	206,000	411,000	90,000	6,000	81,000	93,000
Southeast	7,376,000	14,753,000	3,246,000	201,000	2,919,000	3,340,000
Midwest	10,164,000	20,329,000	4,472,000	277,000	4,022,000	4,602,000
West	8,282,000	16,565,000	3,644,000	226,000	3,277,000	3,750,000
General Population (Total Population = 281,421,906)	NA	NA	45,893,000	2,845,000	41,274,000	47,221,000
Total	36,409,000	72,817,000	61,913,000	3,839,000	55,681,000	63,705,000

NA=not applicable

3.3. CHANGES IN METHYLMERCURY EXPOSURES AMONG FISH CONSUMERS IN THE 8 REGIONS

3.3.1. Population Data.

Table 34 summarizes the estimated sizes of populations consuming the fish caught in each region. The second column identifies the size of the angler population that fishes in each region. The third column identifies the total number of consumers of each type of fish including children. Most individuals in the U.S. consume commercial fish. The size of the population that consumes no fish or only commercial fish is estimated by subtracting the non-commercial fish consuming population (72.8 million) from the total U.S. population (281 million). We assume that the population demographics of non-commercial fish consumers in each region reflect those of the U.S. population. Specifically, we assumed that 22% of the consumers are women of child-bearing age and that roughly 6% of these women give birth each year. We also assume that roughly 20% of the individuals in the populations are males over 39 years of age and 23% are females over 39 years of age (U.S. Census, 2004).

3.3.2. Predicted Exposures in General U.S. Population.

The male and female blood methylmercury concentrations that are assumed to result from commercial fish intake are presented in Table 35.[45] We assumed that the distribution of U.S. female blood concentrations reported by Mahaffey et al. provides a reasonable estimate of the distribution of blood methylmercury concentrations in members of the population that consume commercial fish or no fish. We noted

[45] The distribution was fitted using the NHANES analysis published by Mahaffey et al. (2004) (see second row of Table 35).

previously that the weighted mean methylmercury concentration of the 24 types of fish reported by Carrington and Bolger is 0.116 μg/g. We also noted that the per capita seafood consumption rate for these 24 types of fish is 13.5 pounds per year, which is roughly 16.8 g/day. Combining the weighted mean fish methylmercury concentration and the daily intake fish rate and dividing by the average body weight yields an average daily intake of 0.03 μg/kg-day per capita. Based on Equation 1, the corresponding average blood methylmercury concentration is estimated to be 1.56 μg/L. The mean of the distribution used to fit the Mahaffey data was 1.64 μg/L. Because our calculated mean closely approximates the observed U.S. blood methylmercury concentrations reported by Mahaffey et al., it provides support for the use of the predicted changes in the weighted mean methylmercury concentrations of commercial fish to predict the associated changes in the distribution of the blood methylmercury concentration in the U.S. population.

Table 35. Predicted Tissue Methylmercury Concentrations in Commercial Fish Consumers

General Population	Mean MeHg Blood Conc. (µg/L Blood)	Mean MeHg Hair Conc. (µg/g hair)	Percent Population Below RfD	Conditional Mean MeHg Blood Concentration (µg/L Blood) for those above RfD
Current [a] Female	1.64	0.41	92.1%	8.82
Baseline 1 Female	1.63	0.41	92.2%	8.80
Scenario 1 Female	1.62	0.40	92.4%	8.79
Baseline 2 Female	1.62	0.40	92.4%	8.79
Scenario 2 Female	1.60	0.40	92.5%	8.77
Current Male[b]	1.56	0.39		
Baseline 1 Male	1.55	0.39		
Scenario 1 Male	1.54	0.38		
Baseline 2 Male	1.54	0.38		
Scenario 2 Male	1.53	0.38		

[a] The data in this row results from fitting the female blood methylmercury concentrations reported by Mahaffey et al. (2004). For the total sample population, the 50th, 75th, 90th and 95th percentile values were 0.6, 1.7, 4.4 and 6.7 µg/L, respectively. See Table 16 for additional details of the Mahaffey data set.
[b] Male blood data are based on fitted distribution for female blood methylmercury concentrations and Equation 7.

Using our distribution, which was based on an approximation of the distribution reported by Mahaffey et al., tissue methylmercury levels currently exceed the value that corresponds to the methylmercury RfD in roughly 8% of U.S. females of reproductive age. Based on the data in Table 17, current U.S. male blood methylmercury concentrations are estimated to be 4.3% lower than current levels in females. Using the

methylmercury 1-compartment pharmacokinetic model, hair mercury levels were also estimated based on the blood methylmercury concentrations.

In our model, decreases in U.S. emissions resulted in small changes in methylmercury exposures. Table 35 shows that, when compared with estimates for Baseline 1 and Baseline 2, female blood methylmercury concentrations in Scenario 1 and Scenario 2 are predicted to decrease by 0.01 and 0.02 µg methylmercury/L blood, respectively. The fraction of the population exposed above the RfD was predicted to decrease by less than 0.5% and the conditional mean blood methylmercury concentration in this group is predicted to decrease by less than 0.05 µg/L.

3.3.3. Predicted Exposures in Consumers of Non-commercial Marine Fish.

Tables 36 and 37 present the results of the exposure models for non-commercial fish consumers in the Atlantic and Gulf Regions. These estimates are developed for consumers that eat a mix of non-commercial fish derived from either the Atlantic or the Gulf and a mix of commercial fish.

Table 36. Predicted Methylmercury Intake Rates (µg/kg-day) in Consumers of Non-Commercial Atlantic Ocean Fish*

Population	Mean Intake (µg/kg-day)	50th % (µg/kg-day)	95th % (µg/kg-day)	Percent Population Below RfD	Conditional Mean Intake for those above RfD (µg/kg-day)
Atlantic Current	0.048	0.038	0.123	93.7%	0.144
Atlantic Baseline 1	0.047	0.038	0.119	94.9%	0.147
Atlantic Scenario 1	0.047	0.037	0.118	94.9%	0.146
Atlantic Baseline 2	0.047	0.038	0.119	94.9%	0.147
Atlantic Scenario 2	0.046	0.037	0.117	94.9%	0.145

*This population also is assumed to consume a mix of commercial fish (see Section 2.2.2.3).

Table 37. Predicted Methylmercury Intake Rates (µg/kg-day) in Consumers of Non-Commercial Gulf Fish*

Population	Mean Intake (µg/kg-day)	50th % (µg/kg-day)	95th % (µg/kg-day)	Percent Population Below RfD	Conditional Mean Intake for those above RfD (µg/kg-day)
Gulf Current	0.065	0.044	0.187	79.7%	0.203
Gulf Baseline 1	0.063	0.043	0.182	80.0%	0.200
Gulf Scenario 1	0.063	0.043	0.181	80.1%	0.199
Gulf Baseline 2	0.063	0.043	0.182	80.0%	0.200
Gulf Scenario 2	0.063	0.043	0.180	80.2%	0.198

* This population also is assumed to consume a mix of commercial fish (see Section 2.2.2.3).

Currently, methylmercury from commercial fish is estimated to contribute approximately 64% and 55%, respectively, to predicted methylmercury intakes among consumers of non-commercial Atlantic and Gulf fish. The fractional contribution of commercial fish is higher among consumers of non-commercial Atlantic fish because the estimated weighted mean methylmercury levels in these non-commercial fish are 30% lower than those in the Gulf.

Current methylmercury intake in the Atlantic and Gulf populations is predicted to exceed the RfD in roughly 6% and 14% of these consumers, respectively. The high percentage of the Gulf population that exceeds the RfD is a consequence of both the higher rates of non-commercial fish consumption (relative to non-commercial Atlantic consumers as described in U.S. EPA, 1997a) and the higher concentration of methylmercury in the tissues of non-commercial Gulf fish (relative to non-commercial Atlantic fish) (see Tables 4 and 5). Some of the exposure variability is not captured based on the assumption that non-commercial marine fish consumers eat a mix of non-commercial species. Specifically, consumers who primarily eat species with methylmercury levels above the estimated weighted mean will have higher methylmercury exposures than the consumers that eat the mix of non-commercial fish assumed in the analysis. Conversely, others may eat a mix of fish that have a lower methylmercury concentration than the weighted mean. The assumption that commercial fish consumed by members of these populations contain mean methylmercury concentrations also does not fully capture the variability.

Under the alternative mercury emissions modeled, estimated intake in the Atlantic population declines and the fraction of the population that is exposed above the

RfD also declines. The fractional contribution of methylmercury from commercial fish consumption also is predicted to increase under the alternative emissions. This is a consequence of larger decreases in non-commercial fish methylmercury concentrations than commercial fish concentrations under these alternatives. Table 25 shows that under the emissions cases considered (i.e., the two baselines and the two alternative scenarios), the predicted decreases in mercury deposition to the Gulf and the Coastal Atlantic exceed the predicted change to the All Other Waters Region.

In the Gulf population, estimated intake and the fraction of the population that is exposed above the RfD also decline, but these changes are small. Because the decrease in methylmercury exposure due to commercial fish consumption is less than that from non-commercial fish consumption, the fractional contribution of methylmercury from commercial fish consumption also increases under the alternative emissions. However, the predicted decrease in methylmercury intake among non-commercial fish consumers of Gulf fish is smaller than that among the non-commercial Atlantic fish consuming population. This is a consequence of the relatively smaller decreases in the Gulf non-commercial fish methylmercury concentrations when compared to those in the Atlantic fish. Also, non-commercial Gulf consumers eat higher quantities of non-commercial fish than their Atlantic counterparts.

3.3.4. Predicted Exposures in Consumers of Non-commercial Freshwater Fish.

Tables 38-42 present the results of the exposure models for non-commercial fish consumers in the five freshwater regions simulated. These estimates are developed for consumers that eat a mix of non-commercial fish derived from the freshwaters of the region and a mix of commercial fish that reflects per capita consumption rates.

Table 38. Predicted Methylmercury Intake Rates (µg/kg-day) in Consumers of Non-Commercial Northeast Fish*

Population	Mean Intake (µg/kg-day)	50th % (µg/kg-day)	95th % (µg/kg-day)	Percent Population Below RfD	Conditional Mean Intake for those above RfD (µg/kg-day)
Northeast Current	0.075	0.053	0.196	79.5%	0.173
Northeast Baseline 1	0.069	0.050	0.179	82.3%	0.168
Northeast Scenario 1	0.069	0.049	0.176	82.7%	0.167
Northeast Baseline 2	0.070	0.050	0.182	81.9%	0.169
Northeast Scenario 2	0.068	0.049	0.175	82.9%	0.167

* This population also is assumed to consume a mix of commercial fish (see Section 2.2.2.3).

Table 39. Predicted Methylmercury Intake Rates (µg/kg-day) in Consumers of Non-Commercial Mid-Atlantic Fish*

Population	Mean Intake (µg/kg-day)	50th % (µg/kg-day)	95th % (µg/kg-day)	Percent Population Below RfD	Conditional Mean Intake for those above RfD (µg/kg-day)
Mid-Atlantic Current	0.066	0.045	0.171	85.3%	0.186
Mid-Atlantic Baseline 1	0.057	0.040	0.140	89.4%	0.176
Mid-Atlantic Scenario 1	0.053	0.038	0.129	91.1%	0.173
Mid-Atlantic Baseline 2	0.056	0.040	0.139	89.6%	0.175
Mid-Atlantic Scenario 2	0.052	0.037	0.125	91.5%	0.169

* This population also is assumed to consume a mix of commercial fish (see Section 2.2.2.3).

Table 40. Predicted Methylmercury Intake Rates (µg/kg-day) in Consumers of Non-Commercial Southeast Fish*

Population	Mean Intake (µg/kg-day)	50th % (µg/kg-day)	95th % (µg/kg-day)	Percent Population Below RfD	Conditional Mean Intake for those above RfD (µg/kg-day)
Southeast Current	0.067	0.048	0.170	84.1%	0.171
Southeast Baseline 1	0.059	0.044	0.147	87.6%	0.163
Southeast Scenario 1	0.058	0.043	0.143	88.3%	0.162
Southeast Baseline 2	0.059	0.044	0.146	87.8%	0.163
Southeast Scenario 2	0.057	0.042	0.138	89.0%	0.160

* This population also is assumed to consume a mix of commercial fish (see Section 2.2.2.3).

Table 41. Predicted Methylmercury Intake Rates (µg/kg-day) in Consumers of Non-Commercial Midwest Fish*

Population	Mean Intake (µg/kg-day)	50th % (µg/kg-day)	95th % (µg/kg-day)	Percent Population Below RfD	Conditional Mean Intake for those above RfD (µg/kg-day)
Midwest Current	0.057	0.042	0.137	88.4%	0.149
Midwest Baseline 1	0.054	0.041	0.129	89.7%	0.144
Midwest Scenario 1	0.053	0.040	0.125	90.4%	0.143
Midwest Baseline 2	0.054	0.041	0.128	89.8%	0.144
Midwest Scenario 2	0.053	0.039	0.123	90.6%	0.142

* This population also is assumed to consume a mix of commercial fish (see Section 2.2.2.3).

Table 42. Predicted Methylmercury Intake Rates (µg/kg-day) in Consumers of Non-Commercial West Fish*

Population	Mean Intake (µg/kg-day)	50th % (µg/kg-day)	95th % (µg/kg-day)	Percent Population Below RfD	Conditional Mean Intake for those above RfD (µg/kg-day)
West Current	0.061	0.044	0.151	86.4%	0.159
West Baseline 1	0.060	0.044	0.147	87.0%	0.158
West Scenario 1	0.060	0.043	0.146	87.1%	0.157
West Baseline 2	0.060	0.044	0.147	86.8%	0.157
West Scenario 2	0.060	0.043	0.146	87.1%	0.157

* This population also is assumed to consume a mix of commercial fish (see Section 2.2.2.3).

Based on the use of the Connely et al. (1996) data on rates of commercial fish consumption among non-commercial fish consumers, methylmercury from commercial fish is estimated to contribute roughly 50% or more to predicted current methylmercury intakes among these consumers. The fractional contribution of commercial fish is higher among consumers of less contaminated freshwater fish (e.g., Midwest or West).

When compared to consumers of non-commercial Atlantic fish, a greater percentage of non-commercial freshwater consumers are predicted to exceed the RfD. Currently, roughly 21% of the population that consumes fish from the Northeast is predicted to exceed the RfD. Between 12 and 16% of the other freshwater fish consuming populations are predicted to exceed the RfD currently. We note that the

assumption that non-commercial consumers eat a mix of non-commercial fish likely under estimates the variability in population exposures.

Under the alternative mercury emissions modeled, estimated methylmercury intakes in each of these five populations declines and the fraction of the populations that is exposed above the RfD also declines. The fractional contribution of methylmercury from commercial fish consumption also increases under the alternative emissions. This is a consequence of larger decreases in the methylmercury concentrations of the non-commercial fish, particularly in the Mid-Atlantic and Southeast Regions. Decreases in the Western Regions are relatively small compared to the other regions. Again, the predicted decrease in the methylmercury concentrations of commercial fish is smaller than predicted decreases in the non-commercial freshwater fish in each region.

For the five freshwater regions, we also evaluated high-end fish consumers. We used the data from the fish consumption survey conducted by the Columbia River Inter-Tribal Fish Commission (1994) as a surrogate for this population. For current exposures and all emissions reduction scenarios considered, methylmercury exposures in most consumers with these rates of fish consumption are predicted to exceed the RfD (Table 43). For example, our model predicts that over 80% of this population in the Northeast is currently exposed above the RfD. If the CSI is implemented, over 75% of this population will still be exposed above the RfD (see Table 43). We did not estimate the size of this population explicitly, because we found no suitable data to prepare such an estimate. Additional details are provided in Table 43.

Table 43. Predicted Methylmercury Intakes Among High-End Freshwater Fish Consumers in the U.S.

Population	Mean Intake (µg/kg-day)	Percent Population Below RfD	Conditional Mean Intake for those above RfD (µg/kg-day)
Northeast Current	0.363	18%	0.428
Northeast Baseline 1	0.324	21.6%	0.396
Northeast Scenario 1	0.318	22.1%	0.391
Northeast Baseline 2	0.330	21%	0.401
Northeast Scenario 2	0.316	22.5%	0.389
Mid-Atlantic Current	0.296	35.1%	0.426
Mid-Atlantic Baseline 1	0.230	44.6%	0.374
Mid-Atlantic Scenario 1	0.204	48.9%	0.353
Mid-Atlantic Baseline 2	0.227	45.2%	0.371
Mid-Atlantic Scenario 2	0.195	50.7%	0.345
Southeast Current	0.296	28.8%	0.392
Southeast Baseline 1	0.244	36.2%	0.351
Southeast Scenario 1	0.237	37.4%	0.345
Southeast Baseline 2	0.243	36.3%	0.350
Southeast Scenario 2	0.226	39.1%	0.335
Midwest Current	0.230	33.2%	0.314
Midwest Baseline 1	0.210	37%	0.298
Midwest Scenario 1	0.202	38.6%	0.293
Midwest Baseline 2	0.209	37.2%	0.299
Midwest Scenario 2	0.198	39.6%	0.290
West Current	0.260	30.7%	0.349
West Baseline 1	0.252	31.9%	0.343
West Scenario 1	0.251	32.1%	0.341
West Baseline 2	0.253	31.9%	0.343
West Scenario 2	0.250	32.4%	0.341

3.4. ESTIMATES OF *IN UTERO* NEUROTOXICITY RISKS ASSOCIATED WITH MATERNAL CONSUMPTION OF METHYLMERCURY IN FISH

In Section 3.4.1, we present the estimated IQ losses associated with *in utero* methylmercury exposures per annual birth cohort in the U.S. We then estimate the costs associated with IQ points lost due to methylmercury exposures; these costs are based on a Cost-of-Illness approach. In Section 3.4.2, we estimate the averted costs associated with IQ point gains per annual birth cohort if Scenarios 1 and 2 are implemented. In Section 3.4.3, we present estimates of the QALYs currently lost due to *in utero* methylmercury exposures and those losses that may be averted if Scenarios 1 and 2 are implemented.

3.4.1. IQ Points Lost and Associated Costs due to Current Methylmercury Exposures.

3.4.1.1. Current Methylmercury Impacts Using a No Threshold Model —

Current methylmercury intakes in pregnant consumers of commercial and non-commercial fish in each regional population and in the general population cause IQ decrements (Tables 44-51). Assuming no threshold, the model predicts that currently, the mean IQ point losses per child range from 0.39 to 0.60 among the consumers of non-commercial fish and the mean IQ point loss per child in the population that consumes no fish or only commercial fish is 0.25 (Table 51). When considered as a group and using these modeling assumptions, children born to women consuming only commercial fish lose roughly 59% of the total IQ point loss attributable to *in utero* methylmercury exposures in current annual birth cohorts. Figure 11 shows the fractional contribution of each population to the estimated U.S. total IQ point loss. Tables 44-51 also show the estimated costs associated with these IQ decrements.

Table 52 shows that the current total cost per annual birth cohort is estimated to be approximately $19.9 billion. This estimate is based on a cost-of-illness estimate. Cost-of-illness estimates are typically not considered valid welfare measures. Willingness-to-pay measures for IQ losses were not available, but would likely be significantly greater than the cost-of-illness estimate.

Table 44. Predicted Mean Hair Methylmercury Concentrations, Mean IQ Point Loss, IQ Losses in Annual Birth Cohort, and Estimated Monetary Value (Cost-of-Illness) (2000$) in Consumers of Non-Commercial Atlantic Ocean Fish* (Assuming No Neurotoxicity Threshold)

Population	Mean MeHg Hair Conc. (µg/g hair)	Mean IQ Loss per person	Children born per year	IQ Loss per Annual birth cohort	$ Value IQ point Loss
Atlantic Current	0.649	0.390	137,000	53,300	$894,522,000
Atlantic Baseline 1	0.630	0.378	137,000	51,700	$867,343,000
Atlantic Scenario 1	0.625	0.375	137,000	51,300	$860,587,000
Atlantic Baseline 2	0.629	0.378	137,000	51,600	$866,891,000
Atlantic Scenario 2	0.623	0.374	137,000	51,100	$857,959,000
Benefit Scenario 1		0.003		400	$6,756,000
Benefit Scenario 2		0.004		500	$8,932,000

* This population also is assumed to consume a mix of commercial fish (see Section 2.2.2.3).

Table 45. Predicted Mean Hair Methylmercury Concentrations, Mean IQ Point Loss, IQ Losses in Annual Birth Cohort, and Estimated Monetary Value (Cost-of-Illness) (2000$) in Consumers of Non-Commercial Gulf Fish* (Assuming No Neurotoxicity Threshold)

Population	Mean MeHg Hair Conc. (µg/g hair)	Mean IQ Loss per person	Children born per year	IQ Loss per Annual birth cohort	$ Value IQ point Loss
Gulf Current	0.866	0.520	79,000	41,100	$689,416,000
Gulf Baseline 1	0.845	0.507	79,000	40,000	$672,311,000
Gulf Scenario 1	0.841	0.505	79,000	39,900	$669,680,000
Gulf Baseline 2	0.845	0.507	79,000	40,000	$672,286,000
Gulf Scenario 2	0.839	0.503	79,000	39,800	$667,593,000
Benefit Scenario 1		0.002		100	$2,631,000
Benefit Scenario 2		0.004		200	$4,693,000

* This population also is assumed to consume a mix of commercial fish (see Section 2.2.2.3).

Table 46. Predicted Mean Hair Methylmercury Concentrations, Mean IQ Point Loss, IQ Losses in Annual Birth Cohort, and Estimated Monetary Value (Cost-of-Illness) (2000$) in Consumers of Non-Commercial Northeast Fish* (Assuming No Neurotoxicity Threshold)

Population	Mean MeHg Hair Conc. (µg/g hair)	Mean IQ Loss per person	Children born per year	IQ Loss per Annual birth cohort	$ Value IQ point Loss
Northeast Current	1.006	0.604	67,000	40,700	$683,094,000
Northeast Baseline 1	0.932	0.559	67,000	37,700	$632,940,000
Northeast Scenario 1	0.922	0.553	67,000	37,300	$625,877,000
Northeast Baseline 2	0.945	0.567	67,000	38,200	$641,447,000
Northeast Scenario 2	0.916	0.550	67,000	37,100	$622,248,000
Benefit Scenario 1		0.006		400	$7,063,000
Benefit Scenario 2		0.017		1,100	$19,199,000

* This population also is assumed to consume a mix of commercial fish (see Section 2.2.2.3).

Table 47. Predicted Mean Hair Methylmercury Concentrations, Mean IQ Point Loss, IQ Losses in Annual Birth Cohort, and Estimated Monetary Value (Cost-of-Illness) (2000$) in Consumers of Non-Commercial Mid-Atlantic Fish* (Assuming No Neurotoxicity Threshold)

Population	Mean MeHg Hair Conc. (µg/g hair)	Mean IQ Loss per Person	Children Born per Year	IQ Loss per Annual Birth Cohort	$ Value IQ Point Loss
Mid-Atlantic Current	0.881	0.529	6,000	3,000	$49,817,000
Mid-Atlantic Baseline 1	0.760	0.456	6,000	2,600	$42,948,000
Mid-Atlantic Scenario 1	0.713	0.428	6,000	2,400	$40,303,000
Mid-Atlantic Baseline 2	0.754	0.452	6,000	2,500	$42,597,000
Mid-Atlantic Scenario 2	0.695	0.417	6,000	2,300	$39,289,000
Benefit Scenario 1		0.028		200	$2,645,000
Benefit Scenario 2		0.035		200	$3,308,000

* This population also is assumed to consume a mix of commercial fish (see Section 2.2.2.3).

Table 48. Predicted Mean Hair Methylmercury Concentrations, Mean IQ Point Loss, IQ Losses in Annual Birth Cohort, and Estimated Monetary Value (Cost-of-Illness) (2000$) in Consumers of Non-Commercial Southeast Fish* (Assuming No Neurotoxicity Threshold)

Population	Mean MeHg Hair Conc. (µg/g hair)	Mean IQ Loss per Person	Children Born per Year	IQ Loss per Annual Birth Cohort	$ Value IQ Point Loss
Southeast Current	0.892	0.535	201,000	107,700	$1,808,796,000
Southeast Baseline 1	0.798	0.479	201,000	96,300	$1,617,526,000
Southeast Scenario 1	0.780	0.468	201,000	94,200	$1,581,019,000
Southeast Baseline 2	0.794	0.477	201,000	95,900	$1,609,977,000
Southeast Scenario 2	0.761	0.457	201,000	91,900	$1,542,468,000
Benefit Scenario 1		0.011		2,100	$36,507,000
Benefit Scenario 2		0.020		4,000	$67,509,000

* This population also is assumed to consume a mix of commercial fish (see Section 2.2.2.3).

Table 49. Predicted Mean Hair Methylmercury Concentrations, Mean IQ Point Loss, IQ Losses in Annual Birth Cohort, and Estimated Monetary Value (Cost-of-Illness) (2000$) in Consumers of Non-Commercial Midwest Fish* (Assuming No Neurotoxicity Threshold)

Population	Mean MeHg Hair Conc. (µg/g hair)	Mean IQ Loss per Person	Children Born per Year	IQ Loss per Annual Birth Cohort	$ Value IQ Point Loss
Midwest Current	0.767	0.460	277,000	127,500	$2,141,187,000
Midwest Baseline 1	0.730	0.438	277,000	121,400	$2,038,527,000
Midwest Scenario 1	0.714	0.428	277,000	118,800	$1,994,266,000
Midwest Baseline 2	0.729	0.438	277,000	121,300	$2,036,886,000
Midwest Scenario 2	0.705	0.423	277,000	117,400	$1,970,379,000
Benefit Scenario 1		0.010		2,600	$44,261,000
Benefit Scenario 2		0.014		3,900	$66,507,000

* This population also is assumed to consume a mix of commercial fish (see Section 2.2.2.3).

Table 50. Predicted Mean Hair Methylmercury Concentrations, Mean IQ point Loss, IQ Losses in Annual Birth Cohort, and Estimated Monetary Value (Cost-of-Illness) (2000$) in Consumers of Non-Commercial West Fish* (Assuming No Neurotoxicity Threshold)

Population	Mean MeHg Hair Conc. (µg/g hair)	Mean IQ Loss per Person	Children Born per Year	IQ Loss per Annual Birth Cohort	$ Value IQ Point Loss
West Current	0.824	0.494	226,000	111,700	$1,875,787,000
West Baseline 1	0.809	0.485	226,000	109,700	$1,841,484,000
West Scenario 1	0.804	0.482	226,000	109,000	$1,829,600,000
West Baseline 2	0.809	0.486	226,000	109,700	$1,842,501,000
West Scenario 2	0.801	0.481	226,000	108,600	$1,823,060,000
Benefit Scenario 1		0.003		700	$11,884,000
Benefit Scenario 2		0.005		1,100	$19,441,000

* This population also is assumed to consume a mix of commercial fish (see Section 2.2.2.3).

Table 51. Predicted Mean Hair Methylmercury Concentrations, Mean IQ Point Loss, IQ Losses in Annual Birth Cohort, and Estimated Monetary Value (Cost-of-Illness) (2000$) in Consumers of Commercial Fish and Non-Fish Consumers (Assuming No Neurotoxicity Threshold)

Population	Mean MeHg Hair Conc. (µg/g hair)	Mean IQ Loss per Person	Children Born per Year	IQ Loss per Annual Birth Cohort	$ Value IQ Point Loss
Current Female	0.410	0.246	2,845,000	700,600	$11,763,387,000
Baseline 1 Female	0.407	0.244	2,845,000	695,000	$11,668,494,000
Scenario 1 Female	0.404	0.243	2,845,000	690,100	$11,586,300,000
Baseline 2 Female	0.404	0.242	2,845,000	689,900	$11,583,590,000
Scenario 2 Female	0.401	0.240	2,845,000	684,000	$11,484,931,000
Benefit Scenario 1		0.002		4,900	$82,194,000
Benefit Scenario 2		0.002		5,900	$98,659,000

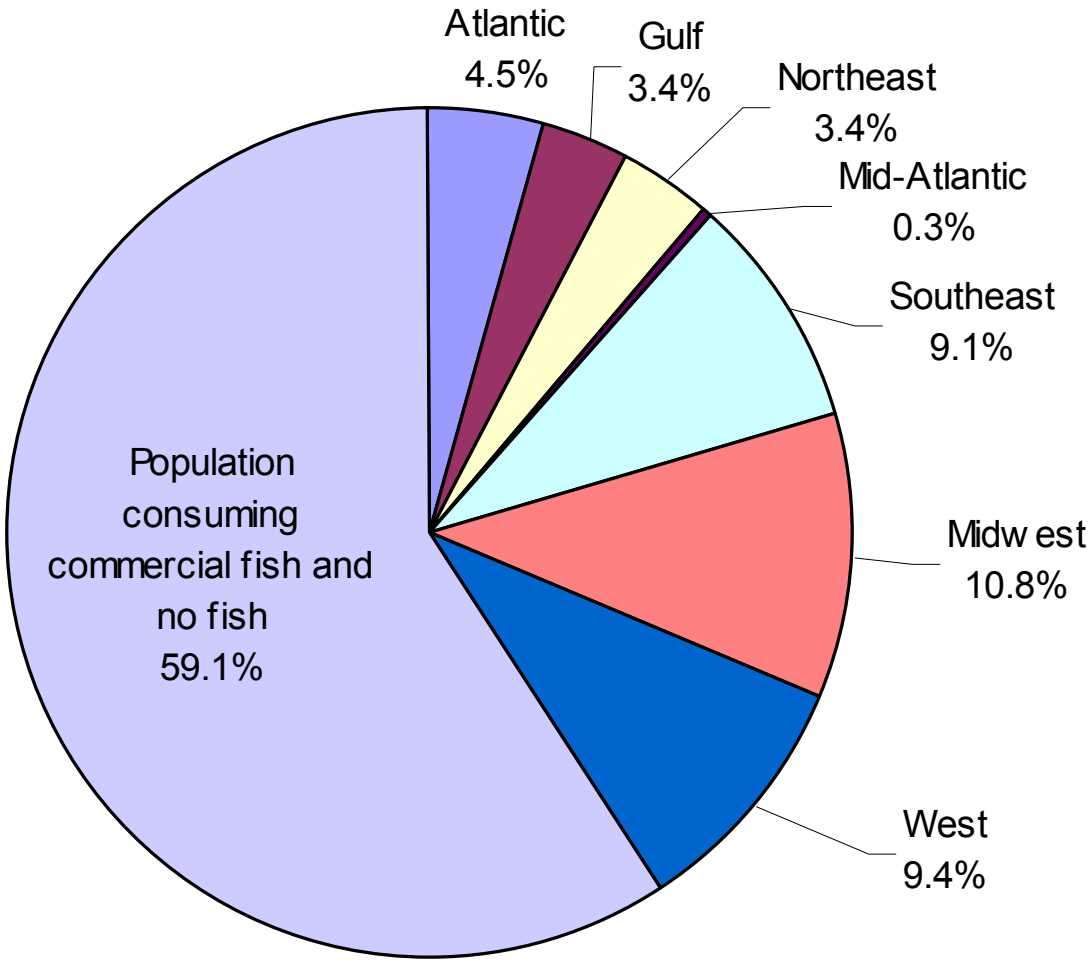


Figure 11

Fractional Contribution of Consumers of Non-Commercial Fish in Each Region and Commercial Fish to Total IQ Point Loss, Assuming No Neurotoxicity Threshold

Table 52. Summary of IQ Point Losses and Associated Costs per Annual Birth Cohort for the Entire U.S. Population (2000$)

	IQ Points Lost per Annual Birth Cohort	Monetary Value of Lost IQ Points
Assuming no Neurotoxicity Threshold		
Total Population Current	1,185,600	$19,906,000,000
Total Population Baseline 1	1,154,400	$19,382,000,000
Total Population Scenario 1	1,143,000	$19,188,000,000
Total Population Baseline 2	1,149,100	$19,296,000,000
Total Population Scenario 2	1,132,200	$19,008,000,000
Assuming a Neurotoxicity Threshold		
Total Population Current	187,000	$3,137,000,000
Total Population Baseline 1	173,000	$2,897,000,000
Total Population Scenario 1	168,000	$2,821,000,000
Total Population Baseline 2	170,000	$2,862,000,000
Total Population Baseline 2	163,000	$2,743,000,000

Table 53. Predicted Incremental IQ Gains per Annual U.S. Birth Cohort and Incremental Estimated Monetary Value of the IQ Gains (Cost-of-Illness) (2000$)

	IQ Point Gain per Annual Birth Cohort	$ Value IQ Point Gain	Number Children Born Above RfD Annually	QALY Gain per Annual Birth Cohort
Scenario 1 (Assuming No Neurotoxicity Threshold)	11,600	$193,940,000		
Scenario 2 (Assuming No Neurotoxicity Threshold)	17,200	$288,248,000		
Scenario 1 (Assuming RfD = Neurotoxicity Threshold)	4,500	$75,311,000	7,400	5,700
Scenario 2 (Assuming RfD = Neurotoxicity Threshold)	7,100	$119,002,000	9,600	7,400

3.4.1.2. Current Methylmercury Impacts Using a Model Threshold of 0.1 µg/kg-day

The estimated current methylmercury intake rates of some pregnant consumers of commercial and non-commercial fish in each regional population and in the general population exceed the RfD (Tables 54-61). Thus, if a neurotoxicity threshold of 0.1 µg/kg-day (i.e., the value of the RfD) is assumed, current methylmercury intakes in some pregnant consumers of commercial and non-commercial fish in each regional population and in the population that consumes only commercial fish cause IQ decrements. Assuming the RfD value to be a threshold, Tables 54-60 show that the predicted mean IQ point losses per child (i.e., conditional mean) range from 0.36 to 0.83 among the consumers of non-commercial fish and Table 61 shows that the mean IQ point loss per child among affected children in the population that consumes only

commercial fish is 0.52.[46] The mean IQ points lost by affected members of each population is greater under the threshold assumption than under the no threshold assumption because the conditional mean methylmercury exposures of these children are higher than the mean of the children at risk under the no threshold assumption. When considered as a group, children born to women in the population that consumes only commercial fish or no fish are estimated to lose roughly 62% of the total IQ point loss attributable to *in utero* methylmercury exposures in the current annual birth cohorts. Tables 54-61 show the estimated costs associated with these IQ decrements. The total cost per current birth cohort is estimated to be approximately $3.1 billion, roughly 16% of the associated cost of $19.9 billion estimated when using the non-threshold model (Table 52). Under the threshold assumption, these costs are borne exclusively by approximately 9% of the babies born in each annual birth cohort. We note that the models employed in the exposure analysis likely underestimate variability in population intake.

[46] This population is assumed to include individuals that consume only commercial fish and those that consume no fish.

Table 54. Predicted Mean IQ point loss, IQ Losses per Annual Birth Cohort, and Estimated Monetary Value (Cost-of-Illness) (2000$) in Consumers of Non-Commercial Atlantic Ocean Fish* (Assuming RfD is Neurotoxicity Threshold)

Population	Average IQ Loss per Person	Number Children Born Above RfD per Annual Birth Cohort	IQ Loss per Annual Birth Cohort	Value IQ Point Loss
Atlantic Current	0.36	8,610	3,060	$51,391,000
Atlantic Baseline 1	0.38	7,000	2,630	$44,127,000
Atlantic Scenario 1	0.37	6,920	2,540	$42,680,000
Atlantic Baseline 2	0.38	6,970	2,620	$43,983,000
Atlantic Scenario 2	0.36	6,920	2,510	$42,092,000
Benefit Scenario 1		80	90	$1,447,000
Benefit Scenario 2		50	110	$1,891,000

* This population also is assumed to consume a mix of commercial fish (see Section 2.2.2.3).

Table 55. Predicted Mean IQ point loss, IQ Losses per Annual Birth Cohort, and Estimated Monetary Value (Cost-of-Illness) (2000$) in Consumers Non-Commercial Gulf Fish* (Assuming RfD is Neurotoxicity Threshold)

Population	Average IQ Loss per Person	Number Children Born Above RfD per Annual Birth Cohort	IQ Loss per Annual Birth Cohort	Value IQ Point Loss
Gulf Current	0.83	16,040	13,310	$223,413,000
Gulf Baseline 1	0.80	15,770	12,650	$212,369,000
Gulf Scenario 1	0.80	15,710	12,500	$209,904,000
Gulf Baseline 2	0.80	15,770	12,640	$212,296,000
Gulf Scenario 2	0.79	15,660	12,360	$207,561,000
Benefit Scenario 1		60	150	$2,465,000
Benefit Scenario 2		120	280	$4,735,000

* This population also is assumed to consume a mix of commercial fish (see Section 2.2.2.3).

Table 56. Predicted Mean IQ point loss, IQ Losses per Annual Birth Cohort, and Estimated Monetary Value (Cost-of-Illness) (2000$) in Consumers of Non-Commercial Northeast Fish* (Assuming RfD is Neurotoxicity Threshold)

Population	Average IQ Loss per Person	Number Children Born Above RfD per Annual Birth Cohort	IQ Loss per Annual Birth Cohort	Value IQ Point Loss
Northeast Current	0.59	13,810	8,130	$136,501,000
Northeast Baseline 1	0.54	11,940	6,510	$109,278,000
Northeast Scenario 1	0.54	11,640	6,310	$105,909,000
Northeast Baseline 2	0.56	12,200	6,790	$114,047,000
Northeast Scenario 2	0.54	11,550	6,190	$103,933,000
Benefit Scenario 1		300	200	$3,369,000
Benefit Scenario 2		650	600	$10,114,000

* This population also is assumed to consume a mix of commercial fish (see Section 2.2.2.3).

Table 57. Predicted Mean IQ point loss, IQ Losses per Annual Birth Cohort, and Estimated Monetary Value (Cost-of-Illness) (2000$) in Consumers of Non-Commercial Mid-Atlantic Fish* (Assuming RfD is Neurotoxicity Threshold)

Population	Average IQ Loss per Person	Number Children Born Above RfD per Annual Birth Cohort	IQ Loss per Annual Birth Cohort	Value IQ Point Loss
Mid-Atlantic Current	0.69	820	570	$9,574,000
Mid-Atlantic Baseline 1	0.61	590	360	$6,068,000
Mid-Atlantic Scenario 1	0.58	500	290	$4,900,000
Mid-Atlantic Baseline 2	0.60	590	350	$5,896,000
Mid-Atlantic Scenario 2	0.56	470	260	$4,427,000
Benefit Scenario 1		90	70	$1,168,000
Benefit Scenario 2		110	90	$1,469,000

* This population also is assumed to consume a mix of commercial fish (see Section 2.2.2.3).

Table 58. Predicted Mean IQ point loss, IQ Losses per Annual Birth Cohort, and Estimated Monetary Value (Cost-of-Illness) (2000$) in Consumers of Non-Commercial Southeast Fish* (Assuming RfD is Neurotoxicity Threshold)

Population	Average IQ Loss per Person	Number Children Born Above RfD per Annual Birth Cohort	IQ Loss per Annual Birth Cohort	Value IQ Point Loss
Southeast Current	0.57	32,070	18,280	$306,955,000
Southeast Baseline 1	0.51	24,880	12,680	$212,816,000
Southeast Scenario 1	0.50	23,590	11,770	$197,686,000
Southeast Baseline 2	0.51	24,630	12,480	$209,572,000
Southeast Scenario 2	0.49	22,140	10,740	$180,298,000
Benefit Scenario 1		1,290	900	$15,130,000
Benefit Scenario 2		2,500	1,740	$29,273,000

* This population also is assumed to consume a mix of commercial fish (see Section 2.2.2.3).

Table 59. Predicted Mean IQ point loss, IQ Losses per Annual Birth Cohort, and Estimated Monetary Value (Cost-of-Illness) (2000$) in Consumers of Non-Commercial Midwest Fish* (Assuming RfD is Neurotoxicity Threshold)

Population	Average IQ Loss per Person	Number Children Born Above RfD per Annual Birth Cohort	IQ Loss per Annual Birth Cohort	Value IQ Point Loss
Midwest Current	0.39	32,250	12,640	$212,145,000
Midwest Baseline 1	0.36	28,590	10,150	$170,484,000
Midwest Scenario 1	0.35	26,700	9,300	$156,139,000
Midwest Baseline 2	0.36	28,390	10,130	$170,154,000
Midwest Scenario 2	0.34	25,940	8,730	$146,517,000
Benefit Scenario 1		1,900	850	$14,345,000
Benefit Scenario 2		2,450	1,400	$23,637,000

* This population also is assumed to consume a mix of commercial fish (see Section 2.2.2.3).

Table 60. Predicted Mean IQ point loss, IQ Losses per Annual Birth Cohort, and Estimated Monetary Value (Cost-of-Illness) (2000$) in Consumers of Non-Commercial West Fish* (Assuming RfD is Neurotoxicity Threshold)

Population	Average IQ Loss per Person	Number Children Born Above RfD per Annual Birth Cohort	IQ Loss per Annual Birth Cohort	Value IQ Point Loss
West Current	0.48	30,750	14,640	$245,866,000
West Baseline 1	0.47	29,420	13,700	$229,987,000
West Scenario 1	0.46	29,230	13,510	$226,851,000
West Baseline 2	0.46	29,770	13,720	$230,393,000
West Scenario 2	0.46	29,130	13,300	$223,274,000
Benefit Scenario 1		190	190	$3,136,000
Benefit Scenario 2		650	420	$7,120,000

* This population also is assumed to consume a mix of commercial fish (see Section 2.2.2.3).

Table 61. Predicted Mean IQ point loss, IQ Losses per Annual Birth Cohort, and Estimated Monetary Value (Cost-of-Illness) (2000$) in Commercial Fish Consumers* (Assuming RfD is Neurotoxicity Threshold)

Population	Average IQ Loss per Person	Number Children Born Above RfD per Annual Birth Cohort	IQ Loss per Annual Birth Cohort	Value IQ Point Loss
Current	0.52	224,330	116,220	$1,951,400,000
Baseline 1	0.52	220,860	113,840	$1,911,439,000
Scenario 1	0.51	217,330	111,800	$1,877,187,000
Baseline 2	0.51	217,220	111,740	$1,876,063,000
Scenario 2	0.51	214,090	109,310	$1,835,300,000
Benefit Scenario 1		3,530	2,040	$34,251,000
Benefit Scenario 2		3,130	2,430	$40,763,000

* This population also is assumed to include individuals that consume no fish.

3.4.2. Averted Costs Associated with IQ Point Gains per Birth Cohort Under Alternative Emissions Scenarios 1 and 2.

3.4.2.1. Results of No Threshold Model —

In this section, the results of Scenario 1 are compared to those of Baseline 1 and the results of Scenario 2 are compared to those of Baseline 2. Based on the model results, under Scenarios 1 and 2 methylmercury intakes in pregnant consumers of commercial and non-commercial fish in each regional population and in the general population will decline but continue to cause IQ decrements in children (Tables 44-51). When compared to Baseline 1 and Baseline 2, the mean IQ point losses per child across the entire population under Scenarios 1 and 2 are predicted to decrease by roughly 0.003 and 0.004 IQ points, respectively. They decrease from 0.301 to 0.298 between Baseline 1 and Scenario 1 and from 0.299 to 0.295 between Baseline 2 and Scenario 2. The decrease in IQ points lost is predicted to be highest among non-commercial fish consumers (except in the West) and lowest among commercial consumers. Table 52 summarizes the estimated IQ points lost per annual birth cohort in the entire population for each emissions scenario. Rows 2 and 3 of Table 53 show that the predicted incremental gain in average IQ between Baseline1 and Scenario1 is associated with a societal benefit of approximately $194 million and the societal benefit associated with the predicted incremental gain in average IQ between Baseline 2 and Scenario 2 is $288 million. (See last two rows in Tables 44-51 for regional benefits summaries of Scenarios 1 and 2.) Most of the cost decrease is associated with the decreased IQ point loss in the commercial fish consuming population. Among the non-

commercial consumers, most of the predicted benefit is predicted to occur in the populations that consume Southeast or Midwestern non-commercial fish.

3.4.2.2. Averted Costs Associated with IQ Point Gains per Birth Cohort Under Alternative Emissions Scenarios 1 and 2 Using Model Threshold of 0.1 µg/kg day

The estimated current methylmercury intake rates of some pregnant consumers of commercial and non-commercial fish in each regional population and in the population that consumes only commercial fish or no fish are predicted to exceed the RfD. Based on the model results, under Scenarios 1 and 2 methylmercury intakes in pregnant consumers of commercial and non-commercial fish in each regional population and in the population that consumes only commercial fish or no fish will decline resulting in fewer affected individuals in each population; however, methylmercury exposures will continue to cause IQ decrements in children (Tables 54-61) (see summary in Table 52).

When compared to Baseline 1 and Baseline 2, the mean IQ point gains per child under Scenarios 1 and 2 are predicted to be roughly 0.001 and 0.006 IQ points, respectively. When compared to the current estimate, under Scenario 2 the mean IQ point losses per child are predicted to decrease roughly 0.015 IQ points. Table 53 shows that the incremental gain in mean IQ in each annual birth cohort between Baseline1 and Scenario 1 is associated with a societal benefit of approximately $75 million. The societal benefit associated with mean IQ gain between Baseline 2 and Scenario 2 is $119 million. Most of the societal benefit is associated with the decreased IQ point loss in the population that consumes only commercial fish. Among the non-

commercial consumers, most of the benefit is predicted to occur in the populations that consume Southeast or Midwestern non-commercial fish.

3.4.3. IQ Results Considering QALYs.

We estimated QALYs lost by assuming that each child born to a mother whose intake exceeded the RfD lost a utility weight of 0.01 (see Section 2.6), and that this effect would persist throughout the child's life (life expectancy at birth is estimated to be 77.2 years) for a loss of 0.77 QALYs. We noted that both the utility weight and the exposure-response function are quite simple. The results are similar to those in Sections 3.4.2.2. Currently, children born to commercial fish consumers (i.e., the population that eats only commercial fish or no fish) are predicted to lose most of the QALYs lost due to *in utero* methylmercury exposures (Table 53). When compared to Baseline 1, under Scenario 1 the loss of roughly 5700 QALYs is averted. When compared to Baseline 2, under Scenario 2 the loss of roughly 7400 QALYs is averted.

3.5. COSTS ASSOCIATED WITH ACUTE MYOCARDIAL INFARCTIONS AND ALL CAUSE MORTALITY

3.5.1. Pike Consumers.

If it is appropriate to generalize the Salonen et al. data, then it is most plausible that the regression coefficients also apply to consumption of non-fatty fish with high methylmercury levels such as pike. The Salonen data are based on a male cohort. In this report, initially we apply the predicted rate increases for acute myocardial infarction and all cause mortality to the male pike consumers in the U.S.

To estimate the size of this cohort, we assume that those targeting pike for catch are the only consumers.[47]

Table 62 lists the predicted mean daily methylmercury intakes (µg/kg-day) and the estimated sizes of the male pike-consuming populations in each of four freshwater regions.[48] In this part of the analysis we assumed that only males that consumed pike were affected by the cardiovascular risk. The mean daily current methylmercury intakes range from approximately 0.05 to 0.07 µg/kg-day. The blood and hair concentrations are estimated using the methylmercury one compartment model (Equation 1).

The last two columns in Table 62 list the predicted annual changes in the number of cases of nonfatal acute myocardial infarctions and premature deaths due to all cause mortality, when comparing Baseline 1 to Scenario 1 and Baseline 2 to Scenario 2. The incremental decreases are the result of the change in daily methylmercury intake and the size of the affected population. Given the small sizes of the populations and the modest predicted changes in mean methylmercury intakes, annually, 4.5 cases of non-fatal acute myocardial infarctions and eight premature deaths are predicted to be avoided through implementing Scenario 1 in this population. Under Scenario 2, annually, we predict eight cases of non-fatal acute myocardial infarctions and 14.6 premature deaths to be avoided. Table 63 lists the annual avoided costs associated with these outcomes. If the VSL estimate is used for premature death, then the annual avoided costs are $49 million and $87 million under Scenarios 1 and 2, respectively.

[47] If methylmercury antagonizes the effects of n-3 fatty acids, then other fish with low to moderate n-3 concentrations and high methylmercury concentrations may cause cardiovascular effects as well. Thus, a larger population may be affected. However, they may be affected less severely than the Salonen cohort, because they will likely consume higher levels of n-3 fatty acids than the members of the Salonen cohort.
[48] Pike are not among the species routinely targeted by anglers fishing in the Southeastern Region.

Most of the benefit is realized by Midwest pike consumers. Annually, we predict 140 and 250 QALYs are gained under Scenarios 1 and 2. Most QALYs gained are the result of decreases in premature mortality.

Table 62. Distribution of Predicted Annual Cases of Acute Myocardial Infarction (AMI) and Premature Deaths (ACM) in Male Northern Pike Consumers

	Mean Daily Intake	Mean Blood Concentration	Mean Hair Concentration	Incidence Rate AMI	Incidence Rate ACM	Population	Non-Fatal AMI Cases	Premature Deaths	Non-Fatal AMI Avoided	Premature Deaths Avoided
Northeast Current	0.075	4.05	1.01	9.983E-03	1.123E-02	98,000	796	1,098		
Northeast Baseline1	0.067	3.61	0.90	9.911E-03	1.112E-02	98,000	790	1,088		
Northeast Scenario 1	0.066	3.55	0.89	9.902E-03	1.111E-02	98,000	790	1,086	0.7	1.3
Northeast Baseline2	0.069	3.69	0.92	9.924E-03	1.114E-02	98,000	791	1,089		
Northeast Scenario 2	0.066	3.53	0.88	9.898E-03	1.110E-02	98,000	789	1,086	2.0	3.7
MidAtlantic Current	0.092	4.93	1.23	1.013E-02	1.144E-02	2,000	17	23		
MidAtlantic Baseline 1	0.071	3.83	0.96	9.948E-03	1.118E-02	2,000	17	23		
MidAtlantic Scenario 1	0.063	3.40	0.85	9.877E-03	1.107E-02	2,000	16	23	0.1	0.2
MidAtlantic Baseline 2	0.070	3.78	0.95	9.939E-03	1.116E-02	2,000	17	23		
MidAtlantic Scenario 2	0.060	3.25	0.81	9.852E-03	1.104E-02	2,000	16	23	0.1	0.3
Midwest Current	0.057	3.05	0.76	9.820E-03	1.099E-02	292,000	2,340	3,211		
Midwest Baseline 1	0.052	2.78	0.70	9.777E-03	1.093E-02	292,000	2,330	3,193		

Table 62 cont.

	Mean Daily Intake	Mean Blood Concentration	Mean Hair Concentration	Incidence Rate AMI	Incidence Rate ACM	Population	Non-Fatal AMI Cases	Premature Deaths	Non-Fatal AMI Avoided	Premature Deaths Avoided
Midwest Scenario 1	0.050	2.69	0.67	9.762E-03	1.090E-02	292,000	2,326	3,186	3.6	6.4
Midwest Baseline 2	0.052	2.78	0.69	9.776E-03	1.093E-02	292,000	2,330	3,192		
Midwest Scenario 2	0.049	2.63	0.66	9.752E-03	1.089E-02	292,000	2,324	3,182	5.7	10.2
West Current	0.052	2.80	0.70	9.779E-03	1.093E-02	54,000	427	586		
West Baseline 1	0.050	2.71	0.68	9.766E-03	1.091E-02	54,000	427	585		
West Scenario 1	0.050	2.69	0.67	9.763E-03	1.091E-02	54,000	427	584	0.1	0.2
West Baseline 2	0.050	2.71	0.68	9.766E-03	1.091E-02	54,000	427	585		
West Scenario 2	0.050	2.68	0.67	9.761E-03	1.090E-02	54,000	427	584	0.2	0.4
Cases Avoided Scenario 1									4.5	8.2
Cases Avoided Scenario 2									8.1	14.6

Table 63. Using a Cost-of-Illness Approach and VSL, Annual Costs Associated with Cases of Non-Fatal AMI and Premature Death In Male Northern Pike Consumers (2000$)

	Predicted Annual decrease in cases of non-fatal AMI	Annual Avoided Costs due to reduction in non-fatal cases of AMI	Predicted Annual decrease in cases of ACM	Annual Avoided Costs (COI) due to reduction in cases of ACM	Annual Avoided Costs (VSL) due to reduction in cases of ACM	Annual Total Avoided Costs (COI) due to reduction in cases of AMI and ACM	Annual Total Avoided Costs (COI for AMI and VSL for ACM) due to reduction in cases of AMI and ACM
Change Scenario 1	4.5	$235,000	8.2	$2,624,000	$48,380,000	$2,858,000	$48,614,000
Change Scenario 2	8.1	$423,000	14.6	$4,671,000	$86,140,000	$5,094,000	$86,563,000

3.5.2. Entire U.S. Population.

In the second case, we apply the regression coefficients from the Salonen et al. data to consumption of all fish. We apply them to males and females. We note that this is an extremely uncertain extrapolation, because of potential differences between the subjects in the Salonon et al. study and the U.S. population and the potential differences in the types of fish consumed.

Tables 64-71 list the predicted cases of male non-fatal AMI and ACM by region. Currently, most cases are predicted to occur in the population that consumes only commercial fish or no fish. Among the non-commercial consumers, most cases are predicted to occur among Midwestern and Western consumers. Table 72 summarizes the benefits of Scenarios 1 and 2 in males. Under Scenario 1, annually, roughly 140 cases of non-fatal acute myocardial infarctions and 260 premature deaths are predicted to be avoided. Under Scenario 2, annually, roughly 210 cases of non-fatal acute myocardial infarctions and 380 premature deaths are predicted to be avoided. Based on the VSL estimate for premature death, the annual avoided costs are predicted to be $1.5 billion and $2.3 billion under Scenarios 1 and 2, respectively. Among males, most of the benefit is realized by the population that consumes commercial fish. Annually, we predict 4300 and 6500 QALYs are gained under Scenarios 1 and 2, respectively. Nearly 95% of QALYs gained under both scenarios are the result of decreases in premature mortality.

Table 64. Predicted Annual AMI and ACM Incidence and Valuation (2000$) in Male Consumers of Non-Commercial Atlantic Ocean Fish*

Population	Male Non-Fatal AMI Cases	Male Total ACM Cases	Male Total AMI COSTS	Costs Male Mortality (COI)	Costs Male Mortality (VSL)
Atlantic Current	15,768	21,585	$823,048,000	$6,906,000,000	$127,353,000,000
Atlantic Baseline 1	15,747	21,549	$821,981,000	$6,894,000,000	$127,136,000,000
Atlantic Scenario 1	15,742	21,539	$821,716,000	$6,892,000,000	$127,083,000,000
Atlantic Baseline 2	15,747	21,548	$821,963,000	$6,894,000,000	$127,133,000,000
Atlantic Scenario 2	15,740	21,536	$821,612,000	$6,890,000,000	$127,062,000,000
Benefit Scenario 1	5	10	$265,000	$2,914,000	$53,728,000
Benefit Scenario 2	7	12	$351,000	$3,852,000	$71,026,000

* This population also is assumed to consume a mix of commercial fish (see Section 2.2.2.3).

Table 65. Predicted Annual AMI and ACM Incidence and Valuation (2000$) in Male Consumers of Non-Commercial Gulf Fish*

Population	Male Non-Fatal AMI Cases	Male Total ACM Cases	Male Total AMI COSTS	Costs Male Mortality (COI)	Costs Male Mortality (VSL)
Gulf Current	9,243	12,710	$482,484,000	$4,066,000,000	$74,987,000,000
Gulf Baseline 1	9,230	12,686	$481,802,000	$4,059,000,000	$74,848,000,000
Gulf Scenario 1	9,228	12,682	$481,697,000	$4,058,000,000	$74,827,000,000
Gulf Baseline 2	9,230	12,686	$481,801,000	$4,059,000,000	$74,848,000,000
Gulf Scenario 2	9,227	12,680	$481,614,000	$4,057,000,000	$74,810,000,000
Benefit Scenario 1	2	4	$105,000	$1,156,000	$21,311,000
Benefit Scenario 2	3	6	$187,000	$2,062,000	$38,018,000

* This population also is assumed to consume a mix of commercial fish (see Section 2.2.2.3).

Table 66. Predicted Annual AMI and ACM Incidence and Valuation (2000$) in Male Consumers of Non-Commercial Northeast Fish*

Population	Male Non-Fatal AMI Cases	Male Total ACM Cases	Male Total AMI COSTS	Costs Male Mortality (COI)	Costs Male Mortality (VSL)
Northeast Current	7,959	10,974	$415,430,000	$3,511,000,000	$64,749,000,000
Northeast Baseline 1	7,920	10,905	$413,416,000	$3,489,000,000	$64,339,000,000
Northeast Scenario 1	7,915	10,895	$413,134,000	$3,486,000,000	$64,281,000,000
Northeast Baseline 2	7,927	10,917	$413,757,000	$3,493,000,000	$64,408,000,000
Northeast Scenario 2	7,912	10,890	$412,988,000	$3,484,000,000	$64,251,000,000
Benefit Scenario 1	5	10	$283,000	$3,126,000	$57,642,000
Benefit Scenario 2	15	27	$769,000	$8,499,000	$156,718,000

* This population also is assumed to consume a mix of commercial fish (see Section 2.2.2.3).

Table 67. Predicted Annual AMI and ACM Incidence and Valuation (2000$) in Male Consumers of Non-Commercial Mid-Atlantic Fish*

Population	Male Non-Fatal AMI Cases	Male Total ACM Cases	Male Total AMI COSTS	Costs Mortality Death (COI)	Costs Male Mortality (VSL)
Mid-Atlantic Current	657	904	$34,291,000	$289,000,000	$5,331,000,000
Mid-Atlantic Baseline 1	652	894	$34,018,000	$286,000,000	$5,276,000,000
Mid-Atlantic Scenario 1	650	891	$33,913,000	$285,000,000	$5,254,000,000
Mid-Atlantic Baseline 2	651	894	$34,004,000	$286,000,000	$5,273,000,000
Mid-Atlantic Scenario 2	649	889	$33,873,000	$284,000,000	$5,246,000,000
Benefit Scenario 1	2	3	$105,000	$1,151,000	$21,232,000
Benefit Scenario 2	2	5	$131,000	$1,439,000	$26,528,000

* This population also is assumed to consume a mix of commercial fish (see Section 2.2.2.3).

Table 68. Predicted Annual AMI and ACM Incidence and Valuation (2000$) in Male Consumers of Non-Commercial Southeastern Fish*

Population	Male Non-Fatal AMI Cases	Male Total ACM Cases	Male Total AMI COSTS	Costs Male Mortality (COI)	Costs Male Mortality (VSL)
Southeast Current	23,580	32,440	$1,230,830,000	$10,379,000,000	$191,395,000,000
Southeast Baseline 1	23,434	32,177	$1,223,213,000	$10,295,000,000	$189,845,000,000
Southeast Scenario 1	23,406	32,127	$1,221,765,000	$10,279,000,000	$189,551,000,000
Southeast Baseline 2	23,428	32,167	$1,222,914,000	$10,292,000,000	$189,784,000,000
Southeast Scenario 2	23,377	32,075	$1,220,237,000	$10,262,000,000	$189,240,000,000
Benefit Scenario 1	28	50	$1,448,000	$15,965,000	$294,402,000
Benefit Scenario 2	51	92	$2,676,000	$29,494,000	$543,875,000

* This population also is assumed to consume a mix of commercial fish (see Section 2.2.2.3).

Table 69. Predicted Annual AMI and ACM Incidence and Valuation (2000$) in Male Consumers of Non-Commercial Midwestern Fish*

Population	Male Non-Fatal AMI Cases	Male Total ACM Cases	Male Total AMI COSTS	Costs Male Mortality (COI)	Costs Male Mortality (VSL)
Midwest Current	32,225	44,219	$1,682,077,000	$14,148,000,000	$260,895,000,000
Midwest Baseline 1	32,147	44,080	$1,678,015,000	$14,103,000,000	$260,070,000,000
Midwest Scenario 1	32,114	44,019	$1,676,267,000	$14,084,000,000	$259,715,000,000
Midwest Baseline 2	32,146	44,077	$1,677,950,000	$14,103,000,000	$260,057,000,000
Midwest Scenario 2	32,096	43,987	$1,675,324,000	$14,074,000,000	$259,523,000,000
Benefit Scenario 1	33	61	$1,748,000	$19,245,000	$354,876,000
Benefit Scenario 2	50	90	$2,626,000	$28,906,000	$533,031,000

* This population also is assumed to consume a mix of commercial fish (see Section 2.2.2.3).

Table 70. Predicted Annual AMI and ACM Incidence and Valuation (2000$) in Male Consumers of Non-Commercial Western Fish*

Population	Male Non-Fatal AMI Cases	Male Total ACM Cases	Male Total AMI COSTS	Costs Male Mortality (COI)	Costs Male Mortality (VSL)
West Current	26,357	36,210	$1,375,801,000	$11,586,000,000	$213,641,000,000
West Baseline 1	26,331	36,163	$1,374,438,000	$11,571,000,000	$213,364,000,000
West Scenario 1	26,322	36,147	$1,373,966,000	$11,565,000,000	$213,268,000,000
West Baseline 2	26,332	36,165	$1,374,478,000	$11,571,000,000	$213,372,000,000
West Scenario 2	26,317	36,138	$1,373,706,000	$11,562,000,000	$213,215,000,000
Benefit Scenario 1	9	16	$472,000	$5,205,000	$95,979,000
Benefit Scenario 2	15	27	$772,000	$8,514,000	$156,997,000

* This population also is assumed to consume a mix of commercial fish (see Section 2.2.2.3).

Table 71. Predicted Annual AMI and ACM Incidence and Valuation (2000$) in Male Consumers of Commercial Fish*

Population	Male Non-Fatal AMI Cases	Male Total ACM Cases	Male Total AMI COSTS	Costs Male Mortality (COI)	Costs Male Mortality (VSL)
Current	322,603	439,302	$16,839,216,000	$140,555,000,000	$2,591,880,000,000
Baseline 1	322,536	439,182	$16,835,723,000	$140,517,000,000	$2,591,176,000,000
Scenario 1	322,478	439,079	$16,832,699,000	$140,484,000,000	$2,590,566,000,000
Baseline 2	322,476	439,076	$16,832,599,000	$140,483,000,000	$2,590,546,000,000
Scenario 2	322,406	438,952	$16,828,969,000	$140,443,000,000	$2,589,814,000,000
Benefit Scenario 1	58	103	$3,025,000	$33,069,000	$609,806,000
Benefit Scenario 2	70	124	$3,630,000	$39,683,000	$731,772,000

* This population also is assumed to include individuals that consume no fish.

Table 72. Predicted Annual Decreased AMI and ACM Incidence and Annual Benefit (2000$) in Males

	Male Non-Fatal AMI Cases	Male Total ACM Cases	Male Total AMI COSTS	Costs Male Mortality (COI)	Costs Male Mortality (VSL)
Scenario 1	140	260	$7,451,000	$81,830,000	$1,508,976,000
Scenario 2	210	380	$11,141,000	$122,447,000	$2,257,964,000

Tables 73-80 list the predicted cases of female non-fatal AMI and ACM by region. Currently, most cases are predicted to occur among commercial consumers in the general population. Among the non-commercial consumers, most cases are predicted to occur among Midwestern consumers. Table 81 summarizes the benefits of Scenarios 1 and 2 in females. Under Scenario 1, annually, roughly 150 cases of non-fatal acute myocardial infarctions and 300 premature deaths are avoided. Under Scenario 2, annually, roughly 230 cases of non-fatal acute myocardial infarctions and 450 premature deaths are avoided. Based on the VSL estimate for premature death, the annual avoided costs are $1.8 billion and $2.6 billion under Scenarios 1 and 2, respectively. Most of the benefit is realized by commercial fish consumers. Annually, we predict 5000 and 7500 QALYs are gained under Scenarios 1 and 2, respectively. Over 95% of QALYs gained under both scenarios are the result of decreases in premature mortality.

Table 82 summarizes the predicted annual benefits of Scenarios 1 and 2 in both males and females. Under Scenario 1, annually, roughly 300 cases of non-fatal acute myocardial infarctions and 550 premature deaths are predicted to be avoided. Under Scenario 2, annually, roughly 440 cases of non-fatal acute myocardial infarctions and 830 premature deaths are predicted to be avoided. Based on the VSL estimate for premature death, the annual avoided costs are estimated to be $3.3 billion and $4.9 billion under Scenarios 1 and 2, respectively. Most of the benefit is realized by the population that consumes commercial fish. Annually, we predict 9300 and 13,900 QALYs are gained under Scenarios 1 and 2, respectively (Table 83). Over 95% of QALYs gained under both scenarios are the result of decreases in premature mortality.

Table 73. Predicted Annual AMI and ACM Incidence and Valuation (2000$) in Female Consumers of Non-Commercial Atlantic Ocean Fish*

Population	Female Non-Fatal AMI Cases	Female Total ACM Cases	Female Total AMI COSTS	Costs Female Mortality (COI)	Costs Female Mortality (VSL)
Current	16,419	24,696	$813,199,000	$4,796,000,000	$145,704,000,000
Baseline 1	16,397	24,654	$812,144,000	$4,788,000,000	$145,456,000,000
Scenario 1	16,392	24,643	$811,882,000	$4,786,000,000	$145,395,000,000
Baseline 2	16,397	24,653	$812,127,000	$4,788,000,000	$145,452,000,000
Scenario 2	16,390	24,639	$811,780,000	$4,785,000,000	$145,371,000,000
Benefit Scenario 1	5	11	$262,000	$2,024,000	$61,471,000
Benefit Scenario 2	7	14	$346,000	$2,675,000	$81,260,000

* This population also is assumed to consume a mix of commercial fish (see Section 2.2.2.3).

Table 74. Predicted Annual AMI and ACM Incidence and Valuation (2000$) in Female Consumers of Non-Commercial Gulf Fish*

Population	Female Non-Fatal AMI Cases	Female Total ACM Cases	Female Total AMI COSTS	Costs Female Mortality (COI)	Costs Female Mortality (VSL)
Current	9,625	14,541	$476,710,000	$2,824,000,000	$85,792,000,000
Baseline 1	9,611	14,514	$476,036,000	$2,819,000,000	$85,633,000,000
Scenario 1	9,609	14,510	$475,933,000	$2,818,000,000	$85,609,000,000
Baseline 2	9,611	14,514	$476,035,000	$2,819,000,000	$85,633,000,000
Scenario 2	9,608	14,507	$475,851,000	$2,818,000,000	$85,590,000,000
Benefit Scenario 1	2	4	$103,000	$803,000	$24,382,000
Benefit Scenario 2	3	7	$185,000	$1,432,000	$43,496,000

* This population also is assumed to consume a mix of commercial fish (see Section 2.2.2.3).

Table 75. Predicted Annual AMI and ACM Incidence and Valuation (2000$) in Female Consumers of Non-Commercial Northeast Fish*

Population	Female Non-Fatal AMI Cases	Female Total ACM Cases	Female Total AMI COSTS	Costs Female Mortality (COI)	Costs Female Mortality (VSL)
Current	8,287	12,556	$410,458,000	$2,439,000,000	$74,080,000,000
Baseline 1	8,247	12,476	$408,469,000	$2,423,000,000	$73,610,000,000
Scenario 1	8,241	12,465	$408,190,000	$2,421,000,000	$73,544,000,000
Baseline 2	8,254	12,490	$408,806,000	$2,426,000,000	$73,689,000,000
Scenario 2	8,239	12,459	$408,046,000	$2,420,000,000	$73,510,000,000
Benefit Scenario 1	6	11	$279,000	$2,171,000	$65,948,000
Benefit Scenario 2	15	31	$760,000	$5,902,000	$179,301,000

* This population also is assumed to consume a mix of commercial fish (see Section 2.2.2.3).

Table 76. Predicted Annual AMI and ACM Incidence and Valuation (2000$) in Female Consumers of Non-Commercial Mid-Atlantic Fish*

Population	Female Non-Fatal AMI Cases	Female Total ACM Cases	Female Total AMI COSTS	Costs Female Mortality (COI)	Costs Female Mortality (VSL)
Current	684	1,034	$33,881,000	$201,000,000	$6,099,000,000
Baseline 1	679	1,023	$33,611,000	$199,000,000	$6,036,000,000
Scenario 1	677	1,019	$33,508,000	$198,000,000	$6,011,000,000
Baseline 2	678	1,022	$33,597,000	$199,000,000	$6,033,000,000
Scenario 2	676	1,017	$33,468,000	$198,000,000	$6,002,000,000
Benefit Scenario 1	2	4	$103,000	$800,000	$24,292,000
Benefit Scenario 2	2	5	$129,000	$999,000	$30,351,000

* This population also is assumed to consume a mix of commercial fish (see Section 2.2.2.3).

Table 77. Predicted Annual AMI and ACM Incidence and Valuation (2000$) in Female Consumers of Non-Commercial Southeast Fish*

Population	Female Non-Fatal AMI Cases	Female Total ACM Cases	Female Total AMI COSTS	Costs Female Mortality (COI)	Costs Female Mortality (VSL)
Current	24,553	37,114	$1,216,101,000	$7,208,000,000	$218,975,000,000
Baseline 1	24,401	36,814	$1,208,575,000	$7,150,000,000	$217,202,000,000
Scenario 1	24,372	36,757	$1,207,144,000	$7,139,000,000	$216,865,000,000
Baseline 2	24,395	36,802	$1,208,279,000	$7,148,000,000	$217,132,000,000
Scenario 2	24,342	36,697	$1,205,635,000	$7,127,000,000	$216,510,000,000
Benefit Scenario 1	29	57	$1,431,000	$11,088,000	$336,825,000
Benefit Scenario 2	53	105	$2,644,000	$20,484,000	$622,247,000

* This population also is assumed to consume a mix of commercial fish (see Section 2.2.2.3).

Table 78. Predicted Annual AMI and ACM Incidence and Valuation (2000$) in Female Consumers of Non-Commercial Midwest Fish*

Population	Female Non-Fatal AMI Cases	Female Total ACM Cases	Female Total AMI COSTS	Costs Female Mortality (COI)	Costs Female Mortality (VSL)
Current	33,555	50,591	$1,661,948,000	$9,826,000,000	$298,489,000,000
Baseline 1	33,474	50,431	$1,657,935,000	$9,795,000,000	$297,545,000,000
Scenario 1	33,439	50,363	$1,656,207,000	$9,782,000,000	$297,139,000,000
Baseline 2	33,473	50,429	$1,657,870,000	$9,794,000,000	$297,530,000,000
Scenario 2	33,420	50,326	$1,655,276,000	$9,774,000,000	$296,921,000,000
Benefit Scenario 1	35	68	$1,727,000	$13,366,000	$406,013,000
Benefit Scenario 2	53	103	$2,595,000	$20,075,000	$609,840,000

* This population also is assumed to consume a mix of commercial fish (see Section 2.2.2.3).

Table 79. Predicted Annual AMI and ACM Incidence and Valuation (2000$) in Female Consumers of Non-Commercial West Fish*

Population	Female Non-Fatal AMI Cases	Female Total ACM Cases	Female Total AMI COSTS	Costs Female Mortality (COI)	Costs Female Mortality (VSL)
Current	27,445	41,428	$1,359,337,000	$8,046,000,000	$244,427,000,000
Baseline 1	27,418	41,374	$1,357,990,000	$8,036,000,000	$244,109,000,000
Scenario 1	27,409	41,356	$1,357,524,000	$8,032,000,000	$244,000,000,000
Baseline 2	27,419	41,376	$1,358,030,000	$8,036,000,000	$244,119,000,000
Scenario 2	27,403	41,346	$1,357,267,000	$8,030,000,000	$243,939,000,000
Benefit Scenario 1	9	18	$466,000	$3,615,000	$109,810,000
Benefit Scenario 2	16	30	$763,000	$5,913,000	$179,620,000

* This population also is assumed to consume a mix of commercial fish (see Section 2.2.2.3).

Table 80. Predicted Annual AMI and ACM Incidence and Valuation (2000$) in Female Consumers of Commercial Fish*

Population	Female Non-Fatal AMI Cases	Female Total ACM Cases	Female Total AMI COSTS	Costs Female Mortality (COI)	Costs Female Mortality (VSL)
Current	336,350	503,450	$16,659,069,000	$97,781,000,000	$2,970,356,000,000
Baseline 1	336,277	503,307	$16,655,441,000	$97,753,000,000	$2,969,509,000,000
Scenario 1	336,213	503,182	$16,652,299,000	$97,729,000,000	$2,968,775,000,000
Baseline 2	336,211	503,178	$16,652,196,000	$97,728,000,000	$2,968,751,000,000
Scenario 2	336,135	503,029	$16,648,426,000	$97,699,000,000	$2,967,870,000,000
Benefit Scenario 1	64	125	$3,142,000	$24,153,000	$733,710,000
Benefit Scenario 2	76	149	$3,770,000	$28,983,000	$880,447,000

* This population also is assumed to include individuals that consume no fish.

Table 81. Predicted Annual Decreased AMI and ACM Incidence and Annual Benefit (2000$) in Females

	Female Non-Fatal AMI Cases	Female Total ACM Cases	Female Total AMI COSTS	Costs Female Mortality (COI)	Costs Female Mortality (VSL)
Scenario 1	150	300	$7,515,000	$58,018,000	$1,762,450,000
Scenario 2	230	450	$11,192,000	$86,464,000	$2,626,562,000

Table 82. Predicted Annual Decreased AMI and ACM Incidence and Annual Benefit (2000$) in Males and Females

	Non-Fatal AMI Cases	Total ACM Cases	Total AMI COSTS	Costs Mortality (COI)	Costs Mortality (VSL)
Scenario 1	300	600	$14,965,000	$139,849,000	$3,271,425,000
Scenario 2	400	800	$22,333,000	$208,911,000	$4,884,526,000

Table 83. Predicted Annual QALY Increase Resulting from Decreased AMI and ACM Incidence in Males and Females

	QALYs Non-Fatal AMI Cases	QALYs Total ACM Cases	Total QALYs
Scenario 1	470	8,900	9,300
Scenario 2	700	13,200	13,900

3.6. RESULTS SUMMARY

Tables 84 and 85 present a summary of the results. Table 84 summarizes the results of Scenario 1 and Scenario 2 in rows 2 and 3. In rows 4 and 5, Table 84 integrates the avoided costs associated with a reduction in the neurological effects (assuming no threshold), non-fatal cardiovascular events, and premature fatalities related to methylmercury exposures. Based on the results of this analysis, the potential value of the estimated human health benefits varies across a broad range depending on the health effects included in the analysis. If benefits are limited to COI estimates for persistent IQ deficits in children exposed above the RfD *in utero*, then the annual benefits of mercury control are predicted to be roughly $75 million and $119 million, in Scenarios 1 and 2, respectively. If these IQ deficits persist and occur in all children exposed to methylmercury (i.e., there is no threshold) then the annual benefits of mercury control are predicted to be roughly $194 million and $288 million, in Scenarios 1 and 2, respectively. If the cardiovascular effects associated with methylmercury are limited to males that consume non-fatty freshwater fish, such as pike, then the annual benefits of mercury control are predicted to be roughly $48 million and $86 million, in Scenarios 1 and 2, respectively. Finally, if all individuals are at risk for experiencing the cardiovascular effects associated with methylmercury, then the annual benefits of mercury control are predicted to be roughly $3.3 billion and $4.9 billion, in Scenarios 1 and 2, respectively. If the IQ benefits (no threshold) and the cardiovascular benefits for the entire population are combined, then, the annual benefits of mercury control are predicted to be roughly $3.5 billion and $5.2 billion, in Scenarios 1 and 2, respectively.

Table 84. Summary of Cost-of-Illness and Value-of-Statistical Life Approaches for Neurotoxicity and Cardiovascular Toxicity

	Neurotoxicity Threshold	No Neurotoxicity Threshold	Costs AMI+ ACM (VSL) Male Pike Consumers	Costs AMI+ ACM (COI)	Costs AMI+ ACM (VSL)
Scenario 1	$75,311,000	$193,940,000	$48,436,000	$154,814,000	$3,286,000,000
Scenario 2	$119,002,000	$288,247,000	$86,713,000	$231,244,000	$4,907,000,000
Scenario 1 Summary of neurotoxicity costs and cardiovascular toxicity costs (no threshold)			$242,376,000	$348,754,000	$3,480,000,000
Scenario 2 Summary neurotoxicity costs and cardiovascular toxicity costs (no threshold)			$374,959,000	$519,491,000	$5,195,000,000

Table 85. Summary of Estimates of QALY Gains due to Reductions in Neurotoxicity and Cardiovascular Toxicity

	Neurotoxicity	QALY Non-fatal AMI	QALY ACM	Total
Scenario 1	5,700	470	8,900	15,000
Scenario 2	7,400	700	13,200	21,300

Table 85 summarizes QALY gains associated with the reductions in the health effects due to reductions in mercury emissions. Less than half of these predicted gains are associated with decreased neurological effects. Figure 12 shows that our degree of confidence in these event estimates differs. The neurological effects associated with *in utero* methylmercury exposures are well documented and have been thoroughly evaluated by a number research and advisory groups (e.g., NRC, 2000). On the other hand, while the studies that have evaluated the association of adult methylmercury exposures with cardiovascular events and premature mortality appear to be scientifically sound and the individual study results appear to be credible, they have not been subjected to a rigorous scientific analysis as a group.

Spectrum of Health Effect Certainty

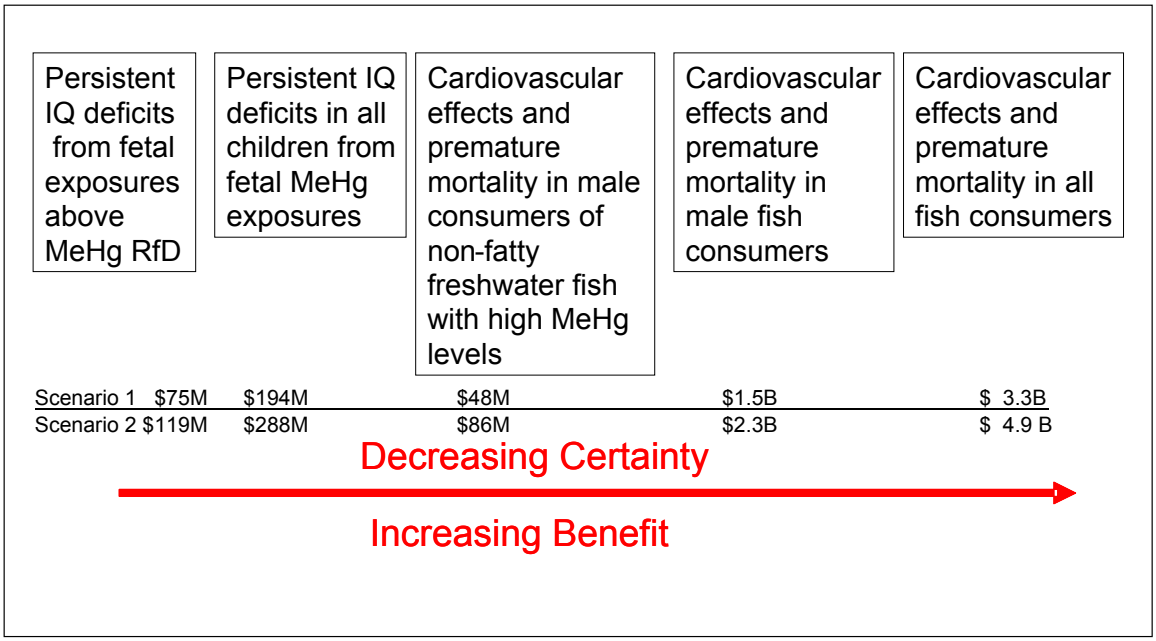


Figure 12

Spectrum of Certainty of Causal Association of Health Effect with Mercury Exposure with Estimated Benefit Overlay in Millions ($M) and Billions ($B) of Dollars (2000$)

Finally, we estimate the annual benefits (2000$) per ton of power plant mercury emissions removed. Annually, 104.7 tons of mercury are emitted in the U.S. under the conditions of Baseline 1. Under Scenario 1, 19.1 tons of power plant mercury emissions are removed and annual U.S. anthropogenic mercury emissions are estimated to be 85.6 tons. Annually, 105.7 tons of mercury are emitted in the U.S. under the conditions of Baseline 2. Under Scenario 2, 26.7 tons of power plant mercury emissions are removed and annual U.S. anthropogenic mercury emissions are estimated to be 79 tons.

We use the data in Table 84 to estimate the ratio in terms of dollar benefits per ton. If we assume a neurotoxicity threshold and assume that only neurological benefits result, we estimate the annual benefits to be $3.9 million/ton removed and $4.5 million/ton removed in Scenarios 1 and 2, respectively. If we limit the benefits to neurotoxicity but assume that there is no threshold for neurotoxicity, then we estimate the annual benefits to be $10.2 million/ton removed and $10.8 million/ton removed under Scenarios 1 and 2, respectively. If we limit the benefits analysis to male pike consumers and neurotoxicity assuming no neurotoxicity threshold, (i.e., a conservative external generalization of the Salonen data), we estimate the annual monetized benefits associated with Scenario 1 to be approximately $242 million and $375 million under Scenario 1 and Scenario 2, respectively (see column 4 in Table 84). These annual benefits are estimated to be $12.7 million/ton removed and $14 million/ton removed under Scenarios 1 and 2, respectively. Finally, if we externally generalize the Salonen data to the entire U.S. population (benefits presented in column 6 of Table 84) and include neurotoxicity assuming no neurotoxicity threshold, the annual benefits are estimated to be $182 million/ton removed and $194.5 million/ton removed under Scenarios 1 and 2, respectively. We note that there are likely to be co-benefits of controlling power plant mercury emissions; these include reduced emissions of primary PM and precursors of secondary PM. The human health benefits of reducing these emissions are not quantified in this analysis. If we use the QALY results in Table 85, we estimate benefits of 300 QALYs/ton removed and 280 QALYs/ton removed based on neurotoxicity only in Scenarios 1 and 2, respectively. In scenarios 1 and 2, we estimate

benefits to be 790 and 800 QALYs per ton removed, respectively, based on reduced neurotoxicity, non-fatal acute myocardial infarctions, and premature mortality.

3.7. RESULTS OF LIMITED SENSITIVITY ANALYSES

Sensitivity analyses of risk and economic models are typically conducted to quantify the impacts of variability and uncertainty in model parameters on the final model outputs. We did not undertake a comprehensive model sensitivity analysis in this report. We plan to conduct such an analysis. We conducted four limited sensitivity analyses to investigate of several key assumptions.

In the first limited sensitivity analysis, we investigated methylmercury intakes, if freshwater fishing was confined to bodies of water where fish methylmercury levels were less than 0.3 ppm. Under conditions where the U.S. EPA's methylmercury water criterion of 0.3 ppm methylmercury in fish is met, the methylmercury intakes still exceed the RfD in more than half of the high-end consumer populations in each region. This result suggests that methylmercury exposures in populations consuming large quantities of freshwater fish will still exceed a level considered by some to be acceptable. We note that intakes in the average freshwater fish consuming population do not exceed the RfD, if the criterion is met.

The results of the second limited sensitivity analysis, in which individual freshwater fish consumers ate only one type of fish, were indistinguishable from the results of the primary analysis. The same fraction of the populations is predicted to exceed the RfD in each freshwater region in this sensitivity analysis and in the primary analysis.

The third sensitivity analysis was conducted to evaluate the impact of an overprediction or an underprediction of the change in deposition associated with the CSI in the Northeast Region. This region was chosen only as an example to investigate the impact of changes in deposition levels in one geographical region. Table 86 compares the predicted IQ point losses per annual birth cohort if deposition rates in the Northeast decreased by two times more than predicted in Scenarios 1. If the decrease is doubled, roughly 950 additional IQ points are gained. If the decrease is halved, then roughly 300 fewer IQ points are gained in Scenario 1. The benefits estimate varies from $1.4 million (if the estimated deposition rate decrease is halved) to$22.7 million (if the estimated deposition rate decrease is doubled). Consumers of non-commercial fish were assumed to eat commercial fish; methylmercury concentrations in the commercial fish change vary little across the different mercury emissions cases used in the analysis.

Table 86. Predicted IQ Point Loss per Annual Birth Cohort in the Northeast Region and the Associated Cost-of-Illness Estimate (2000$), if Deposition Rates are Doubled or Halved

	IQ Loss per Annual Birth Cohort	Value IQ point Loss ($)
Scenario 1 Double	1350	$22,661,000
Scenario 1 Unchanged	400	$7,063,000
Scenario 1 Halved	90	$1,434,000

In the fourth sensitivity analysis, we developed an alternative estimate of the slope of the exposure-response function, if there is a neurotoxicity threshold below which the risk is 0. The results of the fourth limited sensitivity are presented in Table

87. The results suggest that substituting the value of -1.1 for the slope of the exposure-response relationship in the case where a threshold exists increases the IQ point gain by roughly a factor of 2. Under a threshold assumption, the slope value used in the sensitivity analysis results in the highest estimate of IQ points lost per annual cohort currently. The average IQ points lost per affected individual ranges from 0.6 to 1.4 IQ points across the groups of fish consumers. The estimated benefits of scenario 1 increase from an estimated 4,500 IQ points per annual birth cohort to 8,100 IQ points, in the case where a threshold exists. The estimated benefits of scenario 2 increase from 7,100 IQ points per annual birth cohort to 12,800 IQ points.

Table 87. Comparison of Predicted Incremental IQ Gains per Annual U.S. Birth Cohort and Incremental Estimated Monetary Value of the IQ Gains (Cost-of-Illness) (2000$) for 3 Neurotoxicity Models

	IQ Point Gain per Annual Birth Cohort	$ Value IQ Point Gain
Scenario 1 (No Neurotoxicity Threshold)	11,600	$193,940,000
Scenario 2 (No Neurotoxicity Threshold)	17,200	$288,248,000
Scenario 1 (Threshold; slope -0.6 IQ points per ppm)	4,500	$75,311,000
Scenario 2 (Threshold; slope -0.6 IQ points per ppm)	7,100	$119,002,000
Scenario 1 (Threshold; slope -1.1 IQ points per ppm)	8100	$135,560,000
Scenario 2 (Threshold; slope -1.1 IQ points per ppm)	12,800	$214,203,000

In summary, the results of the first sensitivity analysis indicate that methylmercury exposures in most consumers of freshwater fish will not exceed the methylmercury RfD, if they consume fish that have a methylmercury concentration above the methylmercury criterion. People who frequently consume large quantities of

fish may experience exposures to methylmercury above the methylmercury RfD. The second sensitivity analysis highlights a limitation of the approach; specifically, we did not identify data that indicated the mix of non-commercial fish that individuals consumed. The alternative assumption used changed neither the predicted mean intakes or the fraction of the population predicted whose methylmercury intake rate exceeds the RfD. The third sensitivity analysis highlights the potential impacts of changes in the air modeling results on the benefits predicted for a given region. We have noted the uncertainties in these models, particularly in the prediction of dry deposition rates. This analysis also highlights the potential importance of methylmercury exposures from commercial fish consumption to total methylmercury intake. Finally, the fourth sensitivity analysis suggests that, if there is a threshold for the neurotoxicity exposure-response function, then the impacts on those fetuses exposed above the RfD (we again note that the RfD is used as a surrogate for a neurotoxicity threshold) may be quite large (e.g., some populations may lose 1.4 IQ points on average).

4. DISCUSSION

This appears to be the first mercury analysis that attempts to develop multiple model compartments for marine fish and freshwater fish on a national level for the U.S. This model is used to estimate the impacts of mercury emissions control policies on methylmercury intake in different U.S. adult fish-consuming populations and their offspring.

The assumption that there is a proportional relationship between fish methylmercury concentrations and deposition rates has been used in previous analyses to predict changes in methylmercury concentrations in freshwater fish (U.S. EPA, 2001d, 2003b; EPRI, 2003) and marine fish (EPRI, 2003). Limited evidence from field studies suggests that decreases in deposition may result in proportional decreases in fish methylmercury concentrations in freshwater systems (U.S. EPA, 2003b). It is not known whether studies of changes in mercury deposition to salt water bodies will support this assumption or not. Methylmercury concentrations in yellowfin tuna caught between 1971 and 1998 do not appear to have changed over time despite significant increases in surface water mercury concentrations in the area where these fish were caught (Kraepiel et al., 2003). It is not known whether these data reflect a general trend for marine species or not. Clearly, if these data reflect a general trend our estimates of changes in methylmercury intake rates in the general population may be biased upward and the possibility exists that there may be no change in marine fish methylmercury concentrations as a result of mercury emissions controls. Alternatively, the cycling of mercury in ocean waters may be significantly slower than such cycles in freshwater

systems. We note that there are no data that can be compared with the Kraepiel data set.

We discuss several additional exposure model assumptions and indicate the likely direction of bias.

1. Although we assume all mercury in fish to be methylmercury, in reality, methylmercury is half of the total mercury typically measured in mollusks and 95% of the total mercury measured in piscivorous species. Thus, we may overestimate the percent decrease in methylmercury intake in commercial fish consumers because all of the mercury reported in the fish is not methylated and does not contribute to the methylmercury measured in blood. It is unlikely that we overestimate methylmercury intake among non-commercial fish consumers; however, because this component of the analysis was limited to finfish intake of large freshwater and marine fish that were likely piscivorous and primarily contaminated with methylmercury.

2. In the global emissions analysis that was used to estimate changes in mercury deposition in the All Other Waters Region, we did not reduce the input of U.S. power plant emissions to account for deposition to the Atlantic Coastal Region, the Gulf of Mexico Region and the five freshwater regions. Consequently, we overestimate the contribution of power plants to deposition in the All Other Waters Region by approximately 12%. In effect, the fraction of the emitted mercury depositing in these other regions is double counted in the All Other Waters Region. Given the small changes predicted in the All Other Waters Region, this is unlikely to change the estimate significantly.

3. We assume that mercury deposition is the only source of methylmercury in water bodies. Because there are other potential sources of contamination around water bodies, this approach may overestimate reductions in fish methylmercury concentrations that may follow emission decreases (U.S. EPA, 2001d); U.S. EPA (2001d) identifies several potential sources of mercury to freshwater bodies. Also, natural processes such as ocean currents and volatilization of deposited mercury from the ocean back into the atmosphere may also transport mercury into coastal ocean habitats that can later be methylated. If this is the case, this approach may overestimate impacts of decreased mercury emissions in the coastal Atlantic and Gulf regions. If terrestrial and riverine mercury inputs to coastal oceans substantially influence the quantity of methylmercury that accumulates in such fish, then decreases in deposition at distant inland points that influence riverine concentrations may contribute substantially to decreased mercury inputs to coastal marine habitats and this assumption regarding changes in deposition to the ocean waters may underestimate changes in fish concentrations, because the predicted decreases in mercury deposition rates

associated with power plant emissions reductions are generally predicted to be larger in terrestrial environments than in marine environments.

4. Based on the REMSAD model results, mercury emitted to the air in the U.S., in general, flows from west to east. Consequently, U.S. power plant mercury emissions influence mercury deposition into freshwater regions and the Atlantic Coastal region and the Gulf of Mexico region. We may underestimate impacts of mercury emissions reductions in the coastal Pacific region, because we assume that fish methylmercury concentrations in the coastal Pacific Ocean are not directly affected by decreases in U.S. power plant emissions. If changes in the contributions of riverine mercury to the coastal Pacific Ocean change methylmercury levels in the fish along the coastal Pacific, then this assumption may result in a significant underestimation of impacts of mercury emissions decreases. Overall, the impact of not evaluating changes in this region is believed to have little effect on the results.

5. If changes in fish methylmercury levels are not linear and proportional to changes in mercury deposition, but, instead if the changes in fish concentrations are greater than linear and proportional, then the primary analysis may underestimate impacts of emissions decreases. Conversely, if this relationship is less than linear and proportional, then it may overestimate the impacts of emissions control.

6. This analysis does not account for the impact of fish consumption advisories on choices of bodies of water fished or choices of sources of fish for consumption. Some anglers do not consume fish or consume fewer fish from bodies of water under fish consumption advisories. Thus, the model likely overestimates the number of freshwater fish consumers above the RfD. The first sensitivity analysis showed that, if the methylmercury criterion of 0.3 ppm in fish is met only populations of high-end freshwater fish consumers are exposed above the RfD.

7. In the analysis (with the exception of the analysis of pike consumption and the second sensitivity analysis) we did not address exposures to individuals that consumed specific commercial and non-commercial fish. Our assumption of consumers eating a mix of different types of fish is a simplification that reduces predicted exposure variability. The non-commercial freshwater and marine fish in the analysis, in general, have higher methylmercury concentrations than the commercial fish. There are several exceptions including commercial shark or swordfish.

In addition to the uncertainties associated with our exposure model assumptions, other uncertainties are associated with model outputs and other data utilized in the analysis. The accuracy of the results of the REMSAD atmospheric model is uncertain.

Despite the advances in scientific research into the chemistry and fate of emitted mercury, the atmospheric chemistry and the deposition of mercury, particularly dry deposition of Hg^{II}, remain uncertain. If atmospheric transport and deposition processes differ substantially from those modeled with REMSAD, then the results of this modeling analysis are likely incorrect for the freshwater regions and the Atlantic Coastal and Gulf regions. For example, the atmospheric modeling conducted for the EPRI (2003) analysis of the CSI predicts substantially smaller decreases in mercury deposition than the REMSAD model results of EPA.

Our assumption that the REMSAD outputs are, in general, correct given the limited comparisons with deposition data led to the structure of the Regions in the analysis. EPRI (2003) modeled mercury deposition to U.S. freshwaters as a single compartment (i.e., the continental U.S. was one compartment in the EPRI model); however, U.S. EPA (2003b) chose to model deposition at the individual watershed level. While we did not want to obscure potential regional differences in mercury deposition, we also did not want to base the model on changes in deposition at relatively small spatial scales (e.g., the watershed level); thus, to strike a balance, simulation of exposure at a regional level was chosen for this analysis. We also wanted to evaluate possible impacts of decreased deposition along the Coast of the Atlantic Ocean and Gulf; thus, we included these regions.

Factors that influence the aquatic chemistry of deposited mercury and the bioaccumulation of methylmercury in different aquatic environments are similarly not well understood (see footnote 7). This analysis employed a simple linear relationship; in reality, the relationships between deposited mercury and fish methylmercury levels may

vary based on a number of chemical and physical factors. Between saltwaters and freshwaters, the relationship between changes in deposited mercury and changes in fish methylmercury concentrations may differ. Although not a focus of this analysis, the time scales of mercury cycling in marine environments may differ substantially from those of freshwater systems (Lamborg et al., 2003; Kraepiel et al., 2003).

The fish methylmercury concentrations in the NLFWA database are voluntarily reported by the States to the U.S. EPA's Office of Water. The data quality criteria and sampling approaches may vary among the States. While non-commercial freshwater fish that were unlikely to be eaten were excluded, biases in the fish sampling approaches used by the States may render this database inconsistent with the types of fish caught and consumed by freshwater anglers. Also, the types of freshwater fish that anglers report targeting for capture, used as a surrogate for the types of fish consumed, may differ substantially from those actually caught and consumed. These data influence weighted mean methylmercury concentrations in freshwater fish and the estimates of the number of fetuses predicted to be exposed to methylmercury above the RfD.

Exposures to high-end consumers of non-commercial freshwater fish were simulated using a fish consumption rate distribution based on data from the Columbia River Inter-Tribal Fish Commission Report. We assumed that these individuals consumed no commercial fish. Methylmercury intakes in most of these individuals are predicted to exceed the RfD (see Table 43). Due to a lack of data, we developed no estimates of the size of this population. We assumed that the non-commercial freshwater fish consumption rate distribution developed for populations of typical non-

commercial fish consumers would include these high level consumers. We note that in this distribution, the fish consumption rate was 59 g/day at the 99.4th percentile; thus, roughly 0.5% of all non-commercial consumers of freshwater fish consume levels of fish above the mean value reported in the Columbia River Inter-Tribal Fish Commission Report in our model. To date, there have been no studies that adequately quantify the sizes of high fish consuming populations in the U.S.

There are additional uncertainties associated with the non-commercial saltwater fish data. When compared to commercial fish, there are fewer methylmercury samples of saltwater fishes intended for non-commercial consumption. There are no indicators of fish length or weight so the relevance of the samples compiled by the EPA (2003d) in the Mercury in Marine Life database to the non-commercial fish caught and consumed in the Atlantic and the Gulf is not known. Also, the Mercury in Marine Life Database does not provide information on the dispersion of the data around the central tendency estimates provided. Finally, intake of methylmercury from shellfish consumption is not evaluated.

Currently, the simulation results predict that roughly 359,000 children are born to women with blood methylmercury levels above 5.8 µg/L blood, the value corresponding to the EPA's oral RfD. Our estimate that roughly 9% of the population of U.S. females of child-bearing age have blood levels above this value is slightly greater than those developed by Mahaffey et al. (2003) based on earlier NHANES data and with recent CDC estimates (CDC, 2004). We developed an approximate distribution of the blood methylmercury concentration data reported by Mahaffey et al., (2003) and used this distribution as an exposure measure for the population that consumes only commercial

fish or no fish. We note that the distribution developed did not precisely match that reported by Mahaffey et al. Our estimate of median female blood concentration in the general population is 0.9 μg/L, which is higher than the median values reported by Mahaffey et al. (0.6 μg/L) and by CDC (2004) (0.86 μg/L). Similarly, our estimate of the current 95th percentile blood concentration in this population is 7 μg/L; Mahaffey et al. report a 95th percentile value of 6.7 μg/L and CDC (2004) reports a value of 6 μg/L. Using our assumptions, we currently estimate that 63% of the women predicted to have blood concentrations that exceed the RfD consume commercial fish only (and do not consume non-commercial fish); the remainder consume some fish caught non-commercially and may also consume fish caught commercially.

Under the decreased mercury emissions scenarios, the predicted decreases in methylmercury intake and in the number of fish consumers above the RfD are modest. For the commercial fish consuming population, this small change is caused by the large fraction (approximately 70%) of the U.S. commercial seafood that is imported and the relatively small contribution of U.S. coal-fired power plant mercury emissions to global (natural and anthropogenic) emissions under the assumptions used in this model. We assumed that the decrease in methylmercury concentrations in tuna, the most heavily consumed saltwater fish in the U.S. commercial market, was linear and proportional to the fractional decrease in global emissions. If the methylmercury concentrations in the tuna in the U.S. commercial market reflect changes in mercury deposition off the coast of North America, then we predict a slightly greater decrease (of less than 1%) in the weighted mean methylmercury concentration of commercial fish than the baseline value. Also, the analysis assumed that the methylmercury levels in freshwater

commercial fish were in equilibrium with the All Other Waters Region. If the decreased mercury deposition from other freshwater regions was substituted, it made almost no difference in the predicted weighted mean methylmercury concentration predicted in commercial fish. The use of mean methylmercury concentrations in commercial fish and the use of a mean weighted average reduced the variability in the predicted exposures.

The predicted decreases in the methylmercury concentrations of fish caught in the Atlantic Coastal, Gulf, and the freshwater regions are larger than those in the All Other Waters Region. Thus, the decreases in methylmercury intakes in individuals consuming fish from these waters are larger than in those consuming commercial fish exclusively.

Based on our model results, on a per consumer basis, the policies that control mercury emissions from U.S. power plants will likely have the largest impact on freshwater fish consumers and non-commercial saltwater fish consumers. This simulation indicates that some consumers of these fish have methylmercury intake rates above the RfD. Thus, as a result of these policies, fewer individuals, including those in the sensitive population, will be exposed above the RfD. Other anthropogenic sources including some outside the U.S. contribute to deposition of mercury in the U.S.; thus, the impact of reducing mercury emissions from a single source or a single country is limited. The effectiveness of U.S. policies for reducing exposures in the majority of the population that consumes commercial fish is limited because imported fish serve as the primary source of fish to the commercial seafood market and the relatively small contribution of U.S. power plant mercury emissions to global mercury emissions.

Thus, reduction in U.S. methylmercury exposures may best be accomplished by pursuit of both domestic mercury emissions reductions and international reductions in emissions. Global emissions reductions could have a greater impact on U.S. commercial fish methylmercury levels and likely reduce methylmercury intake among non-U.S. fish consumers globally. We note that a substantial body of evidence (summarized in U.S. EPA, 1997c; Mason et al., 1994; Lamborg et al., 2002; Mason and Scheu, 2002) indicates that significant changes in the global mercury cycle have occurred as a consequence of historical and current anthropogenic mercury emissions. From the mid-eighteenth century until the present, mercury concentrations in the biosphere have increased; for example, the mercury concentration in the atmospheric compartment is predicted to have increased by a factor of 3 over this period of time (Mason et al., 1994; Lamborg et al., 2002; Mason and Scheu, 2002). The modeling results of Lamborg et al. (2002) indicate that mercury concentrations in the biosphere continue to increase; for example, their model predicts annual mercury concentration increases of 1.4%, 1.5%, and 0.4% in the global atmosphere, mixed layer of the ocean, and ocean thermocline region, respectively. Other data, notably the work of Schuster et al. (2002) and Slemr et al. (2003) suggests that global atmospheric levels of Hg have been declining since about 1990. If the quantity of mercury in the biosphere influences the quantities in fish globally, then there is additional reason to reduce emissions globally.

Finally, there are a number of significant research needs related to the exposure assessment, that, if met, would reduce the uncertainty in future model results.

1. Long-term monitoring studies that collect mercury concentrations in air, water, sediment and fish are needed across the U.S. and around the world. Such

efforts could lead to long-term data records that can be compared to model predictions. Without such studies, it will be difficult to quantify the actual environmental responses to a change in U.S. mercury emissions and to evaluate the time needed for such responses to manifest.

2. Additional development of atmospheric fate models for mercury is needed, including analyses of the chemical reactions that control many of the processes in the atmosphere. Additional research is needed to improve the understanding of the fate of atmospheric mercury emissions from coal-fired power plants on local, regional, and global scales.

3. Additional development of models for evaluating the fate of aquatic mercury and bioaccumulation by fish is needed. Studies are needed that evaluate the factors influencing the response time between changes in deposition and changes in fish methylmercury concentrations; these studies should include water bodies of different sizes and characteristics (see footnote 7).

4. Simulation models need to be developed that account for factors that might lead to regional differences in mercury cycling.

5. Fish methylmercury sampling strategies in freshwaters and saltwaters that are designed to collect the types and sizes of fish that non-commercial fish consumers eat need to be implemented. These fish need to be collected from the source waters that are fished.

6. Additional studies should be undertaken that identify short-term (peak) and long-term fish consumption rates among high-end commercial and non-commercial fish consumers. These studies should examine the types of fish these individuals are consuming and the sources of these fish. These studies should evaluate the number of individuals in these groups. Additional studies should be undertaken to evaluate consumption rates in pregnant females, females of reproductive age, and men.

7. Studies should be undertaken to identify the locations of populations that exhibit elevated non-commercial fish consumption rates. (If many of these populations are located in areas of the U.S. that would experience high reductions in mercury deposition as a consequence of mercury control policies, then the benefits of mercury emissions control would be greater for this population).

8. Additional studies are needed to improve the understanding of the rates and types of commercial fish intake among non-commercial fish consumers.

9. The next NHANES survey should collect blood methylmercury concentrations and detailed exposure information about sources of fish for consumption and the quantities of fish consumed.

The analysis of health effects and the associated economic burden has numerous limitations and uncertainties. There may be additional neurotoxic effects associated with intrauterine methylmercury exposures that are not captured by IQ measures. These were not quantified. We did not evaluate ecological benefits of reducing methylmercury in terrestrial, freshwater, estuarine or marine ecosystems. Methylmercury appears to be neurotoxic for most chordates, thus reductions in methylmercury levels may improve ecosystem health. We did not evaluate the benefits of mercury emission reductions on recreational fisheries in the U.S. The control technologies that would be employed to reduce mercury emissions from coal-fired power plants would likely reduce other emissions such as fine particulate matter and its precursors (oxides of nitrogen and sulfur dioxide). These co-benefits were not evaluated. We also noted that our degree of confidence in these estimates differs between the neurotoxic and cardiotoxic effects. Although there are differences in the results of the three primary epidemiologic neurotoxicity studies, the neurological effects associated with *in utero* methylmercury exposures have been thoroughly evaluated by a number of research and advisory groups (e.g., NRC, 2000). As U.S. EPA noted, there is no evidence of a threshold for neurotoxicity associated with *in utero* methylmercury exposures; thus, it is plausible that there are neurotoxicity benefits associated with reducing methylmercury exposures below the RfD.

On the other hand, as Figure 12 attempts to illustrate, the studies evaluating the association of cardiovascular events with adult methylmercury exposures have, as a group, not been as thoroughly evaluated. While high doses of methylmercury are clearly associated with neurological decrements, they have not been repeatedly shown

to be associated with adverse cardiac events; in fact, fish consumption, which implies some methylmercury exposure, is recommended as protective of cardiovascular disease. Thus, we urge caution in interpreting the results related to monetized benefits associated with the cardiovascular endpoint. Interpretations of these results should include a full explanation of the assumptions used to develop the integrated benefit estimates.

The QALY gains and cost-of-illness estimates associated with myocardial infarctions and all cause mortality are highly dependent on our assumptions of when these events occurred during the course of an individual's life. For example, if we assume that the myocardial infarctions occurred 5 years earlier, then our present estimates of earnings and household production lost would be roughly 45% larger than the estimates we currently use for males and females.

The methylmercury-associated mortality events, if valid for the entire U.S. population and valued using the VSL estimate, are likely the most important economic consequence of methylmercury exposure. We note that reported estimates for the VSL vary by a wide range. Mrozek and Taylor (2002) suggest a VSL range of between $1.5 and $2.5 million (1998$). Viscusi and Aldy (2003) suggest a range between $4 and $9 million. Variability in these measures would influence the predicted range of the benefits analysis if cardiovascular and premature mortality effects are included in the benefits assessment.

Given the uncertainty surrounding this health effect and the values involved, further research is needed to clarify the relationship between adult methylmercury exposures and adverse cardiovascular events and premature mortality. Additional

research is needed to determine whether *in utero* methylmercury exposures alter heart rate variability. If methylmercury exposures change heart rate variability and these changes lead to increased incidence of adverse myocardial events, then benefits of mercury control are likely substantially underestimated in this analysis.

If the cardiovascular effects are limited to male consumers of northern pike and similar species, then avoided costs associated with IQ are comparable to those associated with avoided myocardial infarctions and premature deaths when valued using a VSL approach. The size of the population that is potentially affected by the possible cardiovascular toxicity of methylmercury is an important uncertainty.

The number of QALYs gained due to IQ increases contributes roughly one third of the total predicted QALYs gained in each of the two scenarios. Reductions in non-fatal myocardial infarctions and premature deaths account for the remainder of the total. We note the uncertainty in the utility weight associated with the neurological decrement (see Sections 2.6 and 2.6.1). If this substantially over or under estimates the actual utility weight, then the results of this comparison would change. While it is unlikely that the utility weight associated with such neurological decrements is much larger than 0.03 (a factor of 3 above our current estimate) given the data reviewed in Section 2.6.1, it is possible that the utility weight could be much lower than current value (e.g., factor of 10). (We note that there may be neurological effects associated with methylmercury exposures that are not captured by measures such as IQ and these would increase the benefits associated with mercury emissions control). We also note the uncertainty in the identification of when the myocardial infarction or death occurs in the lifespan of an

individual could have a large impact on the number of QALYs gained due to reductions in methylmercury exposures.

There are a number of significant research needs related to this risk and benefits assessment, that, if met, would reduce the uncertainty in future model results.

1. Additional analysis is needed of the persistence and magnitude of neurological decrements reported in children in the New Zealand and Faroe Islands studies. Additional epidemiologic investigations need to evaluate the relationship between *in utero* methylmercury exposures and neurotoxicity, including potential confounders and modifying factors that may influence the relationship.

2. Willingness-to-pay studies are needed on neurological decrements, such as IQ point loss in children, to improve the economic assessment of benefits of decreased methylmercury exposures.

3. Additional epidemiologic studies are needed on the relationship between methylmercury exposures in adults and the development of cardiovascular effects.

4. If increased cardiovascular effects are observed, then additional studies are needed on the physiological responses to fish fatty acid and methylmercury exposures.

5. Finally, detailed sensitivity analyses are needed on methylmercury benefits studies, such as this one, to identify the effects of factors that are most uncertain.

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