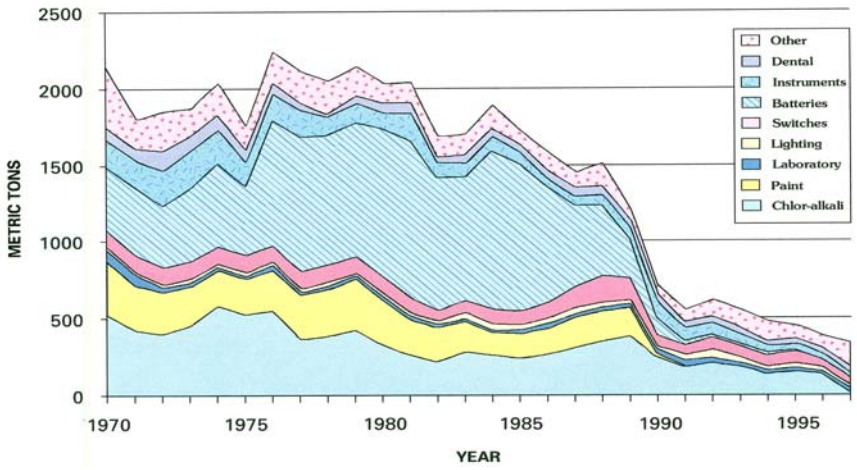
The Annapolis Center
For Science-Based Public Policy

Mercury in the Environment: The Problems, the Risks, and the Consequences

Major Summary Points:

- Mercury in mining and production in the United States stopped in 1991. Since then, industrial consumption of mercury has dropped by more than 50 percent.



- Most of the mercury deposited in U.S. water bodies comes from natural or man-made emission sources outside of our nation's borders. Therefore, mercury is a global issue, and reducing U.S. anthropogenic emissions (particularly coal-fired generating plants) will not significantly decrease the amount of mercury in fish harvested from U.S. waters.

- Women of child-bearing age can eat a variety of fish species to help them maintain good nutrition. Women in this category can safely consume 12 ounces a week (2 meals per week) of most fish species.

"Our greatest responsibility is to be good ancestors."
…Jonas Salk

Promoting responsible energy, environmental, health, and safety decision-making

It is my pleasure to send you the latest report by The Annapolis Center, entitled "Mercury in the Environment: The Problems, the Risks, and the Consequences".

Major points of the report include the following:

- Mercury in mining and production in the United States stopped in 1991. Since then, industrial consumption of mercury has dropped by more than 50 percent.

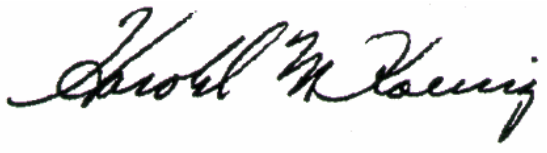
- Most of the mercury deposited in U.S. water bodies comes from natural or man-made emission sources outside of our nation's borders. Therefore, mercury is a global issue, and reducing U.S. anthropogenic emissions (particularly coal-fired generating plants) will not significantly decrease the amount of mercury in fish harvested from U.S. waters.

- Women of child-bearing age can eat a variety of fish species to help them maintain good nutrition. Women in this category can safely consume 12 ounces a week (2 meals per week) of most fish species.

You may be interested that after going to print, an exhaustive study was released of 643 children from before birth to 9 years of age showing no detectable risk from the low levels of mercury their mothers were exposed to from eating ocean seafood. This study by scientists at the University of Rochester Medical Center, is the latest in a series of updates on children who have been studied since their birth in 1989 and 1990 in the Republic of the Seychelles, an island nation in the Indian Ocean. The children have been evaluated five times since their birth, and no harmful effects from the low levels of mercury obtained by eating seafood have been detected. (The study appeared in the May 16 issue of The Lancet.)

Please do not hesitate to contact the Center if you have any questions about this report.

Sincerely,



Harold M. Koenig, M.D.
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Mercury in the Environment: The Problems, the Risks, and the Consequences

EXECUTIVE SUMMARY

In an atmosphere in which the public is regularly exposed to press reports citing the health risks of mercury exposure emanating from coal-fired power plants and other industrial sources, this report attempts to rationally discuss the dispersion and cycling of mercury in the environment, its sources, and its impacts. It also describes how methylmercury makes its way into the aquatic food chain and into the fish that we consume. This report will detail the concern over the adverse health effects seen as a result of methylmercury poisoning incidents in Iraq and Japan, and affects of prenatal exposure observed in three major epidemiological studies. In the U.S., Federal agencies have created health guidance levels for methylmercury exposure in humans. This paper's concluding thoughts point out that attempts to reduce methylmercury in fish will require actions on a global scale, and would be seriously flawed if they only focused on emissions from utilities in the U.S..

Since the late 18th century, humans have found many uses for mercury, including light bulbs, pesticides, batteries, paint, and thermometers and barometers. The ubiquitous and persistent nature of mercury has made it an environmental and human health concern over the past few decades. Because of this realization, laws were passed in the United States to protect its citizens from this toxic pollutant.

As a result, the nation's demand for mercury has significantly declined, and the mine production of primary mercury in the United States ceased in 1991. The closure of these mines has resulted in a significant reduction of the mercury released into the environment from the milling and roasting of the ores. However, a considerable amount of mercury is produced, traded and used internationally.

Mercury occurs naturally, and is dispersed into the environment by both natural and anthropogenic processes. The natural bio-geochemical global cycling of mercury involves degassing the element from surface waters and soils, transporting it through the atmosphere, depositing back into the land and water, absorbing into the soil and sediment, and then its revolatilization from the land and water.

Approximately 2,700 – 6,000 tons of mercury are released annually into the atmosphere from the naturally-occurring degassing of Earth's oceans and crust. Another 2,000 – 3,000 tons are emitted annually by human activities. An estimated total of 144 tons of mercury entered the United States' environment in 1996 as a result of our nation's anthropogenic emissions representing about 3 percent of the total mercury released globally from human activities. On the other hand, Asia accounts for nearly half of the anthropogenic mercury emitted globally, and China's coal-fired power plants alone represent approximately 22 percent of these emissions. U.S. coal-fired electric utilities, the largest source of human related mercury emissions in this country, release approximately 40 tons annually. Although this accounts for slightly more than 30 percent of the anthropogenic mercury produced by this nation's point source emissions, the U.S. utility industry contributes less than 1 percent to the existing global pool of mercury each

year. Other major sources are municipal waste combustors, medical waste incinerators and hazardous waste combustors.

Once this pollutant is released into the air, mercury vapor travels long distances and impact distant locations. Approximately two-thirds (107 tons) of U.S. generated mercury emissions are transported outside of our nation's borders. Roughly 60 percent of the total mercury deposited on the nation's soils and water bodies comes from U.S. anthropogenic air emissions. The remaining 40 percent comes from international human-made mercury emissions, natural sources and reemitted mercury from historic U.S. sources. The amount of mercury deposited over the United States increased rapidly from 1900 to 1950, and then declined about 2-3 fold between 1950 and the 1990s. Since 1995, however, even though mercury emissions from incinerators and other sources had decreased over the past 10 years, mercury deposition in most areas of the country has remained fairly constant.

The Electric Power Research Institute (EPRI) conducted a source attribution study using a global model to assess which continents contribute to the mercury deposition at three locations in the United States (Wisconsin, Florida and New York State). Over 50 percent of the mercury deposition in all three locations was attributed to background/natural emissions. The model shows the next largest contributions being from North America (with 20 – 25 percent of the total mercury emissions) and Asia (with 12 – 15 percent).

Because of all the unknowns and uncertainties in the environmental fate of mercury, there is no quantification of how much of the methylmercury in fish is directly a result of the atmospheric emissions of mercury from electric utility plants or any of the other mercury source category. In addition, there are a host of factors that reduce the certainty of the values produced by the environmental fate and transport of mercury analyses and models.

Although the total amount of mercury delivered to a water body is quite small, it is readily absorbed by the organic material, such as bacteria and plankton, floating in the water. Its methylated form, mercury is ingested by the small fish that consume the methanogenic microorganisms, and these fish (and the methylmercury) are then eaten by larger fish and so on up the food chain. The amount of methylmercury in the organism bioaccumulates at each level of this chain, and such bioaccumulation can result in high levels of methylmercury in some fish. In general, however, methylmercury levels in fish range from less than 0.01 parts per million (ppm) to 0.5 ppm.

Food consumption surveys found that persons 14 years and older had a daily mean intake of fish and shellfish of 0.03 – 0.04 ug/kg/day. Women who are among the top 5 percent of fish/shellfish consumers in the childbearing age category eat just over 100 grams per day, and have methylmercury exposures of about 0.16 ug/kg/day.

States, territories and Native American tribes have the primary authority in protecting citizens from the health risks of eating contaminated fish and wildlife. These governing bodies place consumption advisories on water bodies that contain high levels of toxic chemicals, such as mercury. Forty-four states issued mercury related fish advisories in 2001.

A spectrum of adverse health effects have been observed in humans who were exposed to

methylmercury. The severity of these effects is largely dependent on the magnitude of the dose. When methylmercury is ingested, through eating contaminated fish for example, the toxin is almost completely absorbed into the bloodstream, and then distributed to all the tissues, including the brain.

During the 1950s and 1960s, two major episodes of methylmercury poisoning resulted from the long-term consumption of high levels of methylmercury in fish. The first occurred in the early 1950s among people in Minamata City, Japan. As a result of this exposure, 111 Japanese died or suffered nervous system damage symptoms that were referred to as "Minamata Disease." These children displayed severe psychomotor retardation while their mothers' showed either minor manifestations of poisoning or none at all. The second incident occurred in Niigata, Japan in 1965 where 120 people were poisoned.

Methylmercury poisoning also occurred in two separate incidents in Iraq involving the consumption of seed grains. The symptoms resulting from these Iraqi poisonings primarily involved the nervous symptoms. More than 6,500 Iraqis were hospitalized and 459 died. Both adults and children were affected.

Despite an association between the neurological problems and mercury exposure in Japan and Iraq, these examples are of relatively little relevance to the consumption of fish in the United States.

However, extrapolating from data collected from the high-dose exposure incidents in Japan and Iraq, the U.S. EPA derived a reference dose (RfD) for the amount of methylmercury that is safe to consume based on the developmental neurological effects observed in the children born to mothers exposed to these high doses. The U.S. EPA's reference dose is an estimate (with uncertainty) of a daily exposure to the population (including sensitive subgroups) that is likely to not cause an appreciable risk of deleterious effects during a lifetime. The U.S. EPA's RfD is 0.1 microgram per kilogram body weight per day (0.1 ug/kg/day).

In an attempt to establish a dose-response relationship between the severity of symptoms of mercury poisoning to the amount of fish consumed, large prospective epidemiological studies were conducted in New Zealand, the Faroe Islands and the Republic of the Seychelles. These three studies examined prenatal methylmercury exposure levels that are within the range of the general U.S. population exposures, and evaluated the "subtle end points of neurotoxicity." As a result, the body of knowledge on brain development following the long-term exposure of small amounts of methylmercury has substantially increased. Although the Seychelles Islands main study found no significant association, investigators in both the Faroe Islands and New Zealand studies found that increased prenatal methylmercury exposure was associated with lower performance on neuropsychological tests.

After reviewing these studies, the NAS *Toxicological Effects of Methylmercury* Study determined that this RfD of 0.1 ug/kg/day is a "scientifically justifiable level" for the protection of the public's health. Based on the new information, the U.S. EPA revised how it now bases its RfD value for methylmercury on data from Faroe Island study. This RfD value includes a composite uncertainty factor of 10 to account for the

pharmacokinetic variability and uncertainty in estimating an ingested mercury dose from cord blood mercury concentrations (UF = 3), and pharmacodynamic variability and uncertainty (UF = 3). (Note that these two factors only account for 3 x 3 = 9 UF, although the composite UF = 10).

In 1979, the FDA established an action level of 1.0 ppm in fish (which is based in part on an acceptable or tolerable daily intake of about 0.4 ug/kg/day). This action level limits consumers' exposure to methylmercury levels that are 10 times lower than the lowest levels associated with adverse effects - a safety factor of 10. In January 2001, the FDA issued recommendations for pregnant women and women of childbearing age suggesting that they avoid fish species with the highest concentrations of methylmercury.

The Agency for Toxic Substances and Disease Registry (ATSDR) set a methylmercury exposure concentration of 0.3 ug/kg/day for its minimal risk level (MRL). (U.S. EPA's RfD is three times more stringent than ATSDR's MRL.) An uncertainty factor of 4.5 has been applied to account for the uncertainty for human pharmacokinetic variability (1.5 UF), domain specific findings of the Faroe Islands study (1.5 UF) and human pharmacodynamic variability (1.5 UF).

There is little doubt that mercury exposure can cause toxic effects, but like any other substance, it is a matter of dosage. Arguments calling for reduction in the amounts of methylmercury in fish through regulations on mercury emissions from electric utilities and other emission sources would need to be based upon conclusions that current methylmercury concentrations in fish are harmful, and further, that these emission sources contribute significantly to these methylmercury levels in fish. However, as discussed throughout this White Paper, U.S. emission sources (particularly utilities) probably do not appreciably affect methylmercury levels in fish.

Further complicating the relationship between reducing mercury air emissions and the lowering of methylmercury concentrations in fish is the global nature of mercury in that it can travel great distances before being deposited, and much of the mercury deposited within our nation's borders is from international sources. Therefore, attempts to reduce mercury loads in particular bodies of water, or methylmercury concentration in fish, would require actions to reduce mercury emissions on a global scale, rather than a local or regional scale. If we are concerned about reducing the amount of anthropogenically produced mercury in the environment, our efforts should be focused primarily on reducing the emissions of those countries that emit the most mercury, Russia and the region of Southeast Asia. Also worth noting, because such a significant amount of the total mercury emitted globally is from naturally-occurring sources, even if anthropogenic mercury emissions were drastically reduced, this may not produce the declines in mercury deposition and methylmercury levels in fish that are desired.

These arguments all need to be considered when deciding at what level of methylmercury in fish is deemed to be a justifiable level for the public health's protection. In addition, because many fish species in our nation's waters already exceeding the U.S. EPA's RfD of 0.1 μg/kg/day, using this health action level as a guide for our nation's environmental laws related to methylmercury would most likely produce more stringent standards for anthropogenic emissions of mercury. These tougher standards would most likely produce high costs for little benefit because of the complex, non-straightforward cause-and-effect,

nature of mercury air emissions and methylmercury concentrations in fish. On the other hand, while the FDA's Action Level of 1.0 ppm and Fish Advisory ensure the public's health, the resulting mercury emissions and other mercury related standards would not be costly as those resulting from using the U.S. EPA's RfD as regulatory guidance. In addition, with the uncertainties in our knowledge and the models of the environmental fate and atmospheric deposition of mercury, at this point in time, we should use the FDA Action Level for guidance in our nation's environmental regulations and standards for mercury. Then, as we fill in the gaps of our knowledge and improve the models, this issue can be revisited to determine if basing mercury regulation on the FDA Action Level is developing beneficial results, or if it is necessary switch to the U.S. EPA's more stringent RfD as a mercury lawmaking guideline.

In the meantime, the need for burdensome and costly regulations to reduce a yet uncertain risk to public health is neither necessary nor prudent.

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Since the late 18th century, "the dawn of the Industrial Revolution,"[i] humans have found many uses for mercury (Hg), including in light bulbs, pesticides, batteries, paint, and thermometers and barometers. Since 1989, the largest component of the United States' consumption of mercury has been the use of this element in the production of chlorine and caustic soda (the chlor-alkali industry).

But, the ubiquitous and persistent nature of mercury, combined with its toxic effect on humans, has made it a significant environmental and human health concern over the past few decades. Because of this, laws were passed in the United States to protect its citizens from this toxic pollutant by controlling the amount of mercury emitted into our environment, banning the use of mercury in products such as batteries and paint, and banning the disposal of mercury-containing wastes.

As a result, the nation's demand for mercury has significantly declined and the U.S. mine production of primary mercury ceased in 1991. All currently operating primary mercury-producing mines are now in countries other than the U.S. The closure of U.S. mines resulted in a significant reduction of the mercury released into the environment from the milling and roasting of the ores. The total global production of mercury decreased 38 percent by 1996 from its 1990 levels.[ii] (See Figure 1.)

A significant amount of mercury is produced, traded (1,395 tons in 1996) and used internationally.[ii] The major mercury exporting countries include western Europe, the former Soviet Union and Africa while the main importers are Asia, South America and eastern Europe. (See Figure 2.)

MERCURY CYCLING IN THE ENVIRONMENT

Mercury occurs naturally, and is dispersed into the environment by both natural and anthropogenic processes. Mercury occurs in the environment primarily in three forms – elemental (Hg^0), organic and inorganic (Hg^{+2}). The natural bio-geochemical global cycling of mercury involves degassing of the element from surface waters and soils, its transport through the atmosphere, deposition to land and water surfaces, absorption into soils and sediments, and then sequestration into or revolatilization from the surface. A portion of this cycle may take a small fraction of mercury via living systems, such as uptake in aquatic food webs or by plants. (See Figure 3.) This constant cycling makes tracing mercury to its sources very difficult. Mercury cannot be created or destroyed, but over time, its ultimate sink is in soil or in lake and ocean sediments.

Approximately half of the mercury emitted around the world is believed to be in the Hg^{+2} (ionic) form, with the remainder being primarily released as elemental mercury and with a small portion as particulate mercury.[iii] Some of the gaseous Hg^0 may be converted into Hg^{+2} by oxidation. (Ozone, chlorine and other atmospheric constituents play roles in the oxidation process.) Inorganic mercury occurs in the atmosphere mainly associated with particulate matter. Fine particles have an atmospheric lifetime of several days, during which they disperse regionally on a scale of thousands of kilometers.[iv] Very fine

particles can transport globally. They are removed from the atmosphere by precipitation or by transfer to the surface in the absence of precipitation, in a process called dry deposition. Lake sediment records from North America, the Polar Regions, Europe and the Southern Hemisphere provide compelling evidence that even remote areas on Earth receive significant inputs of mercury from long-range atmospheric transport.[v]

The wet deposition flux, resulting from a combination of precipitation patterns and spatial distributions of ionic and particulate mercury concentrations, occurs highest in East Asia and over the northwestern Pacific Ocean. This deposition pattern reflects the fact that Asia accounts for nearly half of the world's anthropogenic mercury emissions. Areas with high levels of dry mercury deposition (Asia, South Africa, northeast America and Europe) correspond to regions with high anthropogenic emissions. Deposition of particulate mercury is greater in Europe than North America because Europe emits more anthropogenic mercury, and a larger fraction of these emissions occur in the particulate form.[vi]

Many studies have found remote lakes across the United States containing fish whose mercury concentrations are above federal health advisory levels. Because there are no current local sources of mercury near these lakes, the long-range transport and subsequent deposition of mercury into the watersheds is the probable primary contributor of mercury to these water bodies currently.[vii] Compounds of mercury are the predominant forms occurring in soil and surface water bodies. A small fraction of the inorganic mercury present in surface waters may be methylated by bacteria to form monomethylmercury compounds (abbreviated MeHg) primarily occurring as monomethylmercuric chlorine. This methylmercury is introduced into the plants and animals forming the lowest levels of the food chain where it may bioaccumulate through higher levels to concentrations of concern to humans and wildlife (discussed later in this paper).

Mercury in the Atmosphere

The amount of mercury released to the atmosphere from the Earth's surface each year is still uncertain – estimates range from approximately 2,700 to 6,000 tons annually due to degassing of the Earth's oceans and crust. Mercury is emitted from natural sources primarily in a gaseous form as elemental mercury. In addition, some mercury originating from natural sources (such as soil erosion and volcanoes) is bound to particulate matter. Forest fires can lead to significant emissions of inorganic mercury to the atmosphere.[viii] The spatial distribution of natural mercury emissions is largely unknown. However, because these emissions are primarily in the elemental form of mercury, deposition from natural emissions of mercury are more uniformly distributed than anthropogenic mercury emissions.[vi] Approximately 2,000 – 3,000 tons are emitted annually by human activities.[viii]

An estimated total of 144 tons of mercury are emitted to the atmosphere annually by anthropogenic emissions in the United States.[ii] Globally, the "best estimates to date suggest that human activities have about doubled or tripled the amount of mercury in the atmosphere, and the atmospheric burden is increasing by about 1.5 percent per year."[ix] Based on the United States Environmental Protection Agency (U.S. EPA) estimate of 5,500 tons for the total global input of mercury annually from all natural and

anthropogenic emissions, the United States contributes about 3 percent of the total mercury released globally from human activities.[1,v] U.S. EPA's 1997 *Mercury Study Report to Congress*[x] reported that coal-fired electric utilities, the largest source of human related mercury emissions in the U.S., release approximately 40 tons annually. Although this accounts for slightly more than 30 percent of the anthropogenic mercury produced by this nation's point source emissions,[xi] the U.S. utility industry contributes less than 1 percent to the existing global pool of mercury each year.[xii] (Other major point sources are municipal waste combustors, medical waste incinerators and hazardous waste combustors.) Asia accounts for nearly half of the anthropogenic mercury emitted globally, and China's coal-fired power plants alone represent approximately 22 percent of these emissions.[vi]

In its Report to Congress, the U.S. EPA admitted that the current state of knowledge about mercury emissions has created uncertainties and a less than accurate assessment of natural and anthropogenic mercury emissions.[x] For one, although it is believed that one-third of the total global mercury cycles between the atmosphere and oceans, and a major fraction of the mercury in oceans is thought to be anthropogenically produced, these both are essentially estimates. To further illustrate its point, the U.S. EPA documented the degree of uncertainty associated with the measured amounts of emitted mercury from various anthropogenic point source categories.[2] Major sources of uncertainty related to anthropogenic mercury emission estimates have been credited by the U.S. EPA, and others, to situations, such as: emissions test data that is either of poor quality or based on very few samples; the lack of data for some source categories which then led to either estimations based on mass balance calculations or engineering judgment; and the variability in measurements because the survey procedures are not uniform and are done over different time periods.[x] **Because of these uncertainties, and numerous others that were not mentioned, readers of this White Paper should not necessarily focus on the values of different anthropogenic and natural emission sources, but instead, compare the magnitudes of these various sources.**

As one example of the local versus regional and global contributions of mercury occurring in a given area, calculations were performed to determine the proportion of airborne mercury in the state of Michigan that is a result of anthropogenic sources located in various source areas. These calculations estimated that human-produced emissions within Michigan only account for approximately 10 percent of the total mercury concentrations in the ambient air within the state. The bulk of the mercury originates from anthropogenic releases outside of the state's borders, and from natural sources. Therefore, if Michigan completely eliminated all of its emissions from human activities,

[1] This 5,500 tons estimate is on the lower end of the range of 4,700 – 9,000 tons previously stated in this paper for the annual amount of mercury emitted globally. Using the logic that U.S. EPA did to derive the 3 percent estimate, the 144 tons of anthropogenic emissions the U.S. contributes is actually 1.6 – 3.1 percent of the total amount of mercury released annually.

[2] Categories of point source mercury emissions and their degree of uncertainty: utility boilers = medium, residential boilers = high, commercial/industrial boilers = high, wood-fired boilers = medium, municipal waste combustors = medium, hazardous waste combustors = medium, medical waste incinerators = medium, sewage sludge incinerators = high, and crematories = high. Medium degree of uncertainty, as defined by the U.S. EPA's Report to Congress is an emission estimate that is thought to be accurate within +/- 25 percent. High degree of uncertainty is an emission estimate that is thought to be accurate within +/- 50 percent.[x]

the amount of mercury deposited within the state would be reduced by only 10 percent at most.[xiii]

In another simulation, the Electric Power Research Institute (EPRI) modeled what effect reducing Asian emissions of mercury by 50 percent would have on the United States. The largest reduction (approximately 22 percent) was predicted for Texas. Decreases in mercury deposition along the western and southern borders of the U.S. generally ranged between 10 and 20 percent, while declines for the Midwest and southeast ranged between 5 and 10 percent. Because of the patterns in which the global atmospheric winds would carry the mercury particles, reducing Asian mercury emissions would have a negligible effect on the northeast U.S.[vi] Both of these examples demonstrate one of the major issues related to the complexities facing those who attempt to reduce global pollutants, such as mercury. Therefore, it is necessary to create environmental control laws and reduction schemes that are as global as possible.

The various environmental control laws related to mercury that have been passed over the years have had a positive impact on reducing the amount of this toxin in the environment. (See Appendix B.) Overall, the amount of mercury released into the nation's atmosphere in 1996 declined by 97 tons from 1990 levels (144 tons versus 241 tons).[ii] There has been a significant reduction of mercury in mined coal by coal cleaning processes prior to delivery, and in exhaust gases by control devices for other pollutants. In addition, coal-fired power plants have installed pollution control devices for control of particulate matter and oxides of sulfur and nitrogen that also currently remove, on average, 40 percent of the mercury in the coal they use.[xiv] The amount of mercury in municipal and medical wastes has been reduced as a result of the federal mandates eliminating the use of mercury-containing batteries and mercury-containing medical equipment (which were replaced by electronic instruments). In addition, the amount of mercury disposed in landfills was 61 percent less in 1996 than in 1990. Because fewer mercury-containing products were entering waste streams, and emission controls on incinerators became more efficient, mercury releases from incineration decreased by 47 percent from 1990 to 1996.[ii]

Mercury Deposition

Because roughly half of the mercury emitted globally is in the ionic form, it will be deposited near its source, while the remaining portion of mercury emissions (elemental and particulate) will become part of the global background.[iii] Once released into the air, mercury (Hg^0) vapor has an average lifetime of about one year. With its long residency time in the atmosphere, and therefore, its ability to travel long distances and impact distant locations, mercury is considered a global pollutant. Approximately 98 percent of the elemental mercury emitted by U.S. combustion sources is transported outside of our borders.[xv] The National Atmospheric Deposition Program's Mercury Deposition Network (MDN) observed the highest rates of deposition within the United States occurring in the northeast, the southern Great Lakes, the Pacific Northwest and scattered locations across the southeast.[xvi] The amount of mercury deposited over the United States increased rapidly from 1900 to 1950, and then declined about 2-3 fold between 1950 and the 1990s. Since 1995, however, even though mercury emissions from incinerators and other sources have decreased over the past decade, mercury deposition in most areas of the country has remained fairly constant.

EPRI conducted a source attribution study using a global model to assess which continents contribute to the mercury deposition at three locations in the United States (Wisconsin, Florida and New York State). Over 50 percent of the mercury deposition in all three locations was attributed to background/natural emissions. The model shows the next largest contributions being from North America (with 20 – 25 percent of the total mercury emissions) and Asia (with 12 – 15 percent).[vi]

When viewing and analyzing modeled data, it is important to consider the effects limitations and uncertainty have on the results of these simulations. For one, although models that EPRI, the U.S. EPA and others utilize in their analyses of mercury deposition are best matched for their functional ability to simulate certain situations[3] and the minimization of their limitations, even the best of matches still leaves room for uncertainty. EPRI's mercury deposition study provides an illustrative example: the global grid cells used in their analysis were large (approximately 900 km resolution), local emission sources tended to be diluted, which resulted in an underestimation of their contributions.[vi] Other variables that create uncertainty in mercury deposition models, as described in Vijayaraghavan et al.'s "Modeling Deposition of Atmospheric Mercury in Wisconsin" study, arise as a result of inexact inventories of mercury, uncertain chemical reactions in power plant plumes and unknown mercury chemistries.[xvii] (These are just a few select factors that could influence the models and their results; however, there are too many to list.) Additionally, the differences between models, and their parameters, create situations where the results of one model are not equivalent to the results of another. Therefore, it would be inappropriate to compare the data from any two, and make judgments based on this comparison.

Before the creation of the National Atmospheric Deposition Program's (NADP) Mercury Deposition Network (MDN), in 1996, the U.S. EPA had only limited data describing the spatial and temporal distribution of mercury deposition. Therefore, the U.S. EPA utilized RELMAP and ISC3 computer models to describe the environmental fate of mercury.[xviii] As described above, these models have a host of factors which reduce the certainty of the values produced by these environmental fate and transport of mercury analyses. In addition to those factors already mentioned, uncertainty arises from situations where models create hypothetical locations/conditions in generalized areas to mimic that of real settings and conditions. The U.S. EPA did this when designing their environmental fate of mercury models, using a hypothetical eastern and western U.S. site for their simulation, as described in their Report to Congress.[x]

The exposure pathway that served as the focal point of these U.S. EPA environmental fate simulations: atmospheric deposition of mercury → mercury in the watershed → mercury/methylmercury in the waterbody → methylmercury in the fish.[x] However, because of all the unknowns and uncertainties, there is no quantification of how much of the methylmercury in fish is directly a result of the atmospheric emissions of mercury from electric utility plants or any of the other mercury source category.[xix] Another reason for this is the concentration of total mercury (which primarily in the elemental gaseous

[3] Brief description of the three main models used in mercury transport studies and their functions. The Regional Lagrangian Model of Air Pollution (RELMAP) is utilized when assessing regionally scaled atmospheric transport of mercury. Industrial Source Code (ISC3) Model analyzes mercury transport on a local scale. Trace Elements Analysis and Modeling (TEAM) System simulates atmospheric transport, physical and chemical transformations, and deposition of mercury.

form) cycling in the environment is generally a poor predictor of the amount of methylmercury that occurs in receiving waters.[xx] Inorganic mercury has a high affinity for sediments, and a major portion of the mercury in fresh water is transported quickly into sediments. In Wisconsin seepage lakes, about 90 percent of the mercury becomes part of the sediment layer.[xxi] The distribution of inorganic mercury in the environment seems to be controlled primarily by the transport, sorting and sedimentation processes that are related to the hydrologic cycle. The amount and rate of production of methylmercury, however, largely depends on the activity of sulfate-reducing bacteria populating a particular area. These microorganisms convert mercury into methylmercury. Other controlling variables include waterway activity, dissolved organic carbon, oxygen content and turbidity.[xxii]

The divalent fraction of mercury in a water body is absorbed by the life forms, such as bacteria, plankton, and associated organic material. Only about 1 percent (or less) of the total mercury in a water body is methylated. These microorganisms are either consumed by organisms higher up in the food chain, or die and settle on the bottom where they are then incorporated into the bottom sediments. Studies of sediment cores from these water bodies show that the younger sediments have mercury concentrations that are 3 – 5 times that of historical sediments.

METHLYMERCURY AND FISH

In its methylated form, mercury is ingested by the small fish that consume the lower trophic level organisms that have absorbed the methylmercury. These fish (and the MeHg) are then eaten by larger fish and so on up the chain. The amount of methylmercury in the organism bioaccumulates at each level of this food chain. Such bioaccumulation can result in relatively high levels of methylmercury in some fish compared to the methylmercury concentrations in the water column itself. Bioaccumulation occurs because humans and other organisms uptake/ingest/inhale contaminants (such as mercury) faster than their bodies are able to eliminate them. As a result, the contaminants accumulate in their bodies over time. The extent of bioaccumulation varies by water body, and by the complexity and number of trophic levels present. However, if a period of time passes where the organism does not intake any more of that contaminant, then its concentration will decline. The human body can eliminate about half of the mercury in its system in roughly 70 days if no additional mercury is ingested during that time period.[vii]

In general, methylmercury levels in fish range from less than 0.01 parts per million (ppm) to 0.5 ppm. However, large, predatory fish species contain more methylmercury than smaller fish species do because as this accumulation process continues in the food chain, the amount of mercury also magnifies at each level. Shark, tilefish, king mackerel and swordfish (saltwater fish), and bass, pike and walleye (freshwater fish), being at the top of their respective aquatic food chains, contain the highest levels of methylmercury among sampled fish. (For more details, see Appendix B.) Biomagnification occurs as the concentration of mercury incrementally increases at each level of the food chain. (See Figure 4.) Therefore, bioaccumulation can produce concentrations of methylmercury in

large piscivorous (fish-consuming) fish on the order of 10,000 – 100,000 times those concentrations found in the ambient water.[iii]

Mercury concentrates in the muscle tissue of the fish, but unlike some other organic contaminants that concentrate in the fat or skin of the fish, preparation or cooking cannot remove the mercury from the consumed fish. Studies have shown that methylmercury concentrations in shellfish and fish are approximately 1,000 to 10,000 times greater than in other foods, such as milk, meats, poultry, potatoes, cereals, eggs, fruit and vegetables.[xxiii] This is why the major source of human exposure to methylmercury is from eating contaminated fish and seafood.

Saltwater fish, which are the primary source of fish and methylmercury in the human diet, respond much slower to the anthropogenic emissions of mercury that deposit as raindrops into the oceans because of the hundreds, and even thousands, of years of retention time. Therefore, saltwater fish are not expected to be significantly impacted by controls on mercury emissions.

Freshwater farm-raised fish represent the second largest class of fish consumed by humans, but contain very low methylmercury levels because the artificial diet in which they are fed is largely devoid of methylmercury. The remaining 10 percent of fish consumed by humans are wild freshwater fish, which have the potential to respond to changes in mercury loading. However, this change is expected to be trivial.

Consumption of Fish & Related Methylmercury Exposure in U.S.

Approximately 85 percent of American adults eat fish and/or shellfish at least once a month, according to the National Center for Health Statistics of the Centers of Disease Control (NCHS CDC) 1988 – 1994 National Health and Nutrition Examination Survey (NHANES III). The median fish/shellfish consumption values collected from various national dietary surveys, such as NHANES and the United States Department of Agriculture's (USDA) Continuing Surveys of Food Intakes by Individuals (CSFII), were between 73 and 79 grams per day (g/day)[4], based on single day estimates. Analyses based on the U.S. Food and Drug Administration's (FDA) Total Diet Survey methods determined that persons 14 years and older had a daily mean intake of 0.03 – 0.04 μg/kg/day. Asian/Pacific Islander Americans, Native Americans and anglers reported more frequent fish/shellfish consumption than other NHANES III survey participants. Only 1 – 2 percent of the 19,000 adult respondents of the food frequency data portion of NHANES III reported consuming fish and/or shellfish almost daily.[vi] Those individuals reporting consumption levels in the top 5 percent of daily fish/shellfish consumption ate an average of 222 grams per day, according to the USDA's 1994 - 1996 CSFII.[xxiv] Because the developing fetus is especially sensitive to methylmercury exposure, women of childbearing age (15 - 44 years) are of special interest in these daily intake/food consumption surveys. Those women who are among the top 5 percent of fish/shellfish consumers in the childbearing age category eat just over 100 grams per day, and have methylmercury exposures of about 0.16 μg/kg/day.[viii] It is at the 93rd percentile of this population where the methylmercury exposure equals that of the U.S. EPA reference dose of 0.1 μg/kg/day.

[4] 28 grams = 1 ounce

Fish Advisories

States, territories and Native American tribes have the primary authority in protecting citizens from the health risks of eating contaminated fish and wildlife obtained outside commercial channels. These governing bodies place consumption advisories on water bodies that contain fish exhibiting high levels of toxic chemicals, such as mercury. Consumption advisories are often issued for the general population, as well as for those individuals who are among a more susceptible population, such as pregnant women, children and nursing mothers. The advisories also name which bodies of water and/or which fish to limit or avoid eating. (See Figure 5.) Interested individuals can access the National Listing of Fish and Wildlife Advisories (NLFWA) at the U.S. EPA's website http://www.epa.gov/waterscience/fish. A total of forty-four states issued mercury related fish advisories in 2001.[xxv]

ADVERSE HEALTH EFFECTS

A spectrum of adverse health effects has been observed in humans who were exposed to methylmercury, the severity of which is largely dependent on the magnitude and rate of the exposure. When methylmercury is ingested, through eating contaminated fish for example, the toxin is almost completely absorbed into the bloodstream, and then distributed to all the tissues, including the brain. The first frank symptom of methylmercury poisoning among adults is often parasthesia, characterized by the numbness and tingling sensations of the fingers, toes and lips. At greater exposure levels, symptoms can include difficulty in articulating words, a stumbled gait, impaired hearing, and a narrowing of the vision fields that ultimately leads to tunnel vision. Generalized muscle weakness, headache, irritability, and inability to concentrate often occur at successfully higher exposures. Acute high-level exposures can result in impaired central nervous system function, gastrointestinal damage, kidney damage and failure, cardiovascular collapse, shock, and even death. (The lethal dose of methylmercury is estimated to be at 10 – 60 mg/kg.[xx]) Clearly, these effects occur at very high exposure levels. We will consider the effects at these levels, and try to estimate where today's community exposures might cause an effect.

METHYLMERCURY POISONING INCIDENTS OF JAPAN & IRAQ

During the 1950s and 1960s, two major episodes of methylmercury poisoning resulted from the long-term consumption of high levels of methylmercury in fish. The first occurred in the early 1950s among people, and even fish-consuming domestic animals and wildlife, living in Minamata City, on the shores of Minamata Bay, Japan. The source of contamination was the effluent from a chemical factory using mercury as a catalyst and discharging its wastes containing methylmercury into this bay. The fish and shellfish that make up the dietary staple of this population became contaminated. (The average fish consumption was reported to be greater than 300 grams per day, an amount that is 20 times greater than is typical for recreational fishers in the United States.)[xx] As a result of this exposure, 111 Japanese died and several hundred others suffered nervous system damage symptoms that were referred to as "Minamata Disease." Children displayed severe psychomotor retardation while their mothers showed either minor manifestations of poisoning or none at all. The second incident in which 120 people were poisoned

occurred in Niigata, Japan in 1965. The average concentration of methylmercury in fish samples from Minamata and Niigata ranged between 9.0 parts per million (ppm) and 24 ppm.[i,5]

Methylmercury poisoning also occurred in two separate incidents in Iraq involving the consumption of seed grains. Seed grain treated with a fungicide containing organic mercury caused the first outbreak prior to 1960. Then, in the early 1970's, imported mercury-treated seed grains that were baked into bread served as the source of second outbreak. Unlike the long-term exposures seen in the Japanese cases, the methylmercury poisoning in Iraq was shorter in duration, lasting about six months. The symptoms resulting from these Iraqi poisonings were based on measurements and subject reporting more than a year after exposure. More than 6,500 Iraqis were hospitalized and 459 died.[vi] Both adults and children were directly affected. A study led by Thomas Clarkson, a toxicologist at University of Rochester (NY), in the 1970s, looked at 81 Iraqi infant-mother pairs who were exposed to methylmercury during the Iraqi poisoning outbreak. It found that the toxin level in the hair of the mother was predictive of the adverse effects seen in the infants. Some of these infants born to mothers who consumed this contaminated grain (especially during their second trimester) displayed nervous system damage even though their mothers were only slightly affected or had no symptoms resulting from the poisoning.

By extrapolating from data collected from the high-dose exposure incidents in Japan and Iraq, the U.S. EPA derived a reference dose (RfD) for the amount of methylmercury that is considered safe to consume. It was based on the developmental neurological effects observed in the children born to mothers exposed to these high doses. The U.S. EPA's RfD based on these data was 0.1 microgram per kilogram body weight per day (0.1 μg/kg/day).[xiv] The current (2002) RfD is identical, although based on different, more contemporary studies.

EPIDEMIOLOGY STUDIES

In an attempt to establish a dose-response relationship between the severity of symptoms of mercury poisoning and the amount of fish consumed, three large prospective epidemiological studies were conducted. These studies examined prenatal methylmercury exposure levels that are within the range of exposure of the general U.S. population, and evaluated the "subtle end points of neurotoxicity."[ix] As a result, understanding of brain development following the long-term exposure of small amounts of methylmercury has substantially improved. One study has reported results for more than 1,500 children aged over 66 months in the Republic of the Seychelles, a multi-island nation in the Indian Ocean, off of the coast of Africa. The second study examined 917 seven-year-old children of the Faroe Islands, located in the North Atlantic. The third study was conducted using 237 six- and seven-year-olds living in New Zealand. These three locations were chosen because fish and marine mammals served as a dietary stable for the residents of these island nations. This seafood also served as their ongoing source of methylmercury.

[5] 1 ppm = 1 μg/g

(Information related to studies on the Seychelles Islands, Faroe Islands, and New Zealand can be found in Appendix A.)

FEDERAL HEALTH GUIDANCE LEVELS (U.S. EPA, FDA & ASTDR)

U.S. EPA's Revised RfD

After reviewing these studies, the NAS panel reporting its results in *Toxicological Effects of Methylmercury* determined that this RfD of 0.1 μg/kg/day is a "scientifically justifiable level" for the protection of the public's health.[ix] Based on the NAS analysis of the new epidemiological studies, the U.S. EPA revisited how it derived its RfD value. Since the Seychelles Islands study found no evidence showing adverse effects resulting from methylmercury exposure, U.S. EPA decided it could not be used. The data from Faroe Island study was selected over the New Zealand study because New Zealand's cohort was relatively small by comparison to the other studies. The advantages U.S. EPA found with the Faroe Islands study were that it had a large sample size; endured extensive scrutiny in the epidemiological literature; used two different biomarkers of exposure (maternal hair and cord blood); had good statistical power; had statistically significant observations which remained even after adjusting for potential effects from PCB exposure; utilized a comprehensive and focused neurological assessment; and was conducted at an age and state of development when the effects on complex neuropsychological functions are most likely detectable.[xxvi]

Before getting into details about the derivation of this RfD level, it is important to note the definition of a reference dose – it is "an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime."[xxvii] In accordance with this definition, when calculating the RfD, the U.S. EPA looked at data related to the most sensitive target organ, the nervous system. The first step done to determine the RfD, was to conduct a benchmark dose analysis to find the lower confidence level benchmark dose (BMDL). In this analysis, U.S. EPA decided to use the k-power model, and a P_o value of 0.05 (one-sided 95th percentile confidence limit), to create a cutoff for abnormal responses at the lowest 5 percent of children. The BMDLs, in terms of blood cord mercury levels, for the various neuropsychological tests performed on the Faroese children ranged between 24 ppb and 103 ppb, with the median value being 48 ppb. The BMDL is then converted into the ingested daily amount that would result in exposure to the developing fetus at the BMDL in terms of parts per billion (ppb) in blood using the formula,[xxviii]

$$d = \frac{(c)(b)(V)}{(A)(f)(bw)}$$

where d = daily intake rate (expressed in μg/kg/day)
c = concentration in blood which corresponds to the BMDL
b = elimination constant = 0.014 days^{-1} (corresponds to methylmercury's half-life of 70 days as noted above)
V = volume of blood in the body = 5 liters
A = fraction of mercury in diet that is absorbed = 0.95
f = fraction of absorbed dose found in blood = 0.059
bw = body weight = 67 kg

Therefore, the dose conversion formula used by the U.S. EPA to determine the ingested daily amount is:

$$d = \frac{\text{BMDL } \mu g/L \times 0.014 \text{ days}^{-1} \times 5L}{0.95 \times 0.059 \times 67 \text{ kg}}$$

Inputting the range of BMDL values into the above formula produces daily intake rates of 0.447 μg/kg/day – 1.920 μg/kg/day, with the median value being 0.895 μg/kg/day. Then, a composite uncertainty factor of 10 is placed on these values to account for the pharmacokinetic variability and uncertainty in estimating an ingested mercury dose from cord blood mercury concentrations (UF = 3), and pharmacodynamic variability and uncertainty (UF = 3). (Notice that these 2 uncertainty factors only account for 3 x 3 = 9 UF, although the composite UF = 10). The RfD is then obtained when the composite uncertainty factor reduces the daily intake rates by a factor of 10. Although based on the range of daily intake rates, and reducing them by a factor of 10, the RfD values would range from 0.05 – 0.2 μg/kg/day, majority of the RfD values are at or close to 0.1 μg/kg/day. Based on this analysis, U.S. EPA decided that rather than choosing a single endpoint (observed in the neuropsychological analyses) for the RfD, it will be based on a composite of several endpoints. In fact, similar calculations were done with the data from New Zealand study, and an integrative analysis of all three studies, and this also supports U.S. EPA's RfD of 0.1 μg/kg/day.[xxviii,6]

Hair Mercury Concentrations in U.S. Not Comparable to Faroese Population

Results from the NHANES IV (NHANES 1999) measurements of blood mercury concentrations in women aged 16 – 49 years showed a mean concentration of 1.2 ppb, and a mean blood mercury concentration of 0.3 ppb for children aged 1 – 5 years. The blood concentrations of women and children in the 90th percentile were 6.2 ppb and 1.4 ppb, respectively. Most values for the hair mercury concentration were lower than the limit of detection, so no mean values were calculated for this biomarker. However, the 90th percentile values for hair mercury levels (women = 1.4 ppm and children = 0.4 ppm) were attained. In addition, there were no blood or hair methylmercury concentrations that were greater or equal to the 58 ppb methylmercury concentration in cord blood (corresponding to 12 ppm methylmercury concentration in maternal hair) that the NAS review recommended for the BMDL from the Faroe Islands study to be used to determine U.S. EPA's RfD.[xxviii]

FDA's Action Level

In 1979, the FDA established an action level of 1.0 ppm in fish (which is based in part on an acceptable or tolerable daily intake of about 0.4 μg/kg/day).[xxix] Levels that caused parasthesia in the Japanese methylmercury poisoning incidents, the first adult symptom of methylmercury poisoning (as discussed above), were selected as a basis for the FDA's action level. The levels of exposure at which parasthesia occurred were lower than the lowest level found to affect developing fetuses. Therefore, this 1.0 ppm level provides them more protection.[xxx] This was set at 1.0 ppm to limit consumers' exposure to methylmercury levels that are 10 times lower than the lowest levels associated with adverse effects[vi] - a safety factor of 10. This action level ensures the public health's safety in two ways. First, as the agency that oversees the safety of commercial fish in interstate commerce, the FDA uses this measure to make recommendations for legal

[6] U.S. EPA's RfD corresponds to a hair methylmercury concentration of 1.1 μg/g and a cord blood concentration of approximately 5.5 μg/L.[xxiv]

action when an edible portion of a fish sample from a shipment exceeds the 1.0 ppm level. In addition, this level serves as a way to assess risk to human health and as guidance in creating recommendations.

Although the action level still remains at this level, the recommendations for women who are pregnant and of childbearing age was revised in January 2001.[xxxi] In creating the current advisory, the FDA looked at the large-scale epidemiological studies of methylmercury exposure on human populations in New Zealand, the Faroe Islands and Seychelles Islands. Data regarding fish consumption rates in the U.S. and mercury concentrations of fish found in U.S. water bodies, the healthy benefits of eating fish, and feedback from focus groups that provided suggestions about different types of consumer messages was taken into account. As a result of all their research, the FDA created recommendations for pregnant women and women of childbearing age to avoid identified fish species with the highest concentrations of methylmercury (shark, swordfish, tilefish and king mackerel), rather than limiting their consumption to a single serving per month, as was stated in the old advisory. (Focus groups noted they preferred a simple recommendation of avoidance rather than limitation to once per month because they felt the limitation was "essentially a recommendation to abstain."[xxxii]) This advisory also encourages women in this population to eat a variety of "safe" fish species - those that are not on the higher end of the methylmercury concentration range - to help them maintain good nutrition while keeping their exposure to the toxic substance low. The FDA states that even this vulnerable population can safely eat an average of 12 ounces a week of most fish species. (A typical serving size for fish is 3 – 6 ounces.[xxxiii]) In addition, this amount is consistent with the American Heart Association's recommendations about the benefits of fish consumption, especially the cardiovascular benefits.[xxxiii] The FDA notes that though this advisory is mainly targeting those women of childbearing age and pregnant women, it also includes nursing mothers and young children because of the uncertainties associated with the potential adverse effects from methylmercury exposure on the developing nervous system of newborns. It is added that, "these latter groups are being included as a matter of prudence."[xxxiii]

ATSDR's Minimal Risk Level

The Agency for Toxic Substances and Disease Registry (ATSDR) set a methylmercury exposure concentration of 0.3 μg/kg/day for its minimal risk level (MRL). (U.S. EPA's RfD is three times more stringent than ATSDR's MRL.) This level is based upon the No Observed Adverse Effect Level (NOAEL) obtained in the Seychelles Islands study of 15.3 ppm for the highest exposure group of 66-month-olds, which is in accordance with the Agency's methodology of deriving MRLs. Both the Seychelles Islands and Faroe Islands studies were taken into consideration when the current MRL was derived. An uncertainty factor of 4.5 has been applied to account for the uncertainty for human pharmacokinetic variability (1.5 UF), domain specific findings of the Faroe Islands study (1.5 UF) and human pharmodynamic variability (1.5 UF). The ATSDR uses MRLs, such as the one for methylmercury, as an assessment tool for whether any additional evaluation of potential human exposure to hazardous waste sites and emergency response incidents is warranted. They are not intended to set regulatory standards. Instead, they have been designed to (as their name implies) establish substance-specific levels which pose minimal risk to the public's health.[xxxiv]

CONCLUDING REMARKS

The NAS *Toxicological Effects of Methylmercury* Study, and others, suggest that because of the health benefits from eating fish, our nation's long-term goal needs to be a reduction in concentrations of methylmercury in fish rather than having the public replace fish with other foods in their diet. (In the meantime, it is suggested that the public abide by the state and local fish advisories to minimize the consumption of fish known to have high levels of methylmercury.) Arguments calling for reducing the amounts of methylmercury in fish through regulations on mercury emissions from electric utilities and other emission sources would need to be based upon conclusions that current methylmercury concentrations in fish are harmful, or that these emission sources contribute significantly to these methylmercury levels in fish. However, as discussed throughout this White Paper, U.S. emission sources (particularly utilities) probably do not appreciably affect methylmercury levels in fish. For one, there are a multitude of factors, such as the activity of sulfate-reducing bacteria, and the multitude of human and natural mercury sources, that confound the any expectation of a simple relationship between reducing air emissions from a particular source type and lowering methylmercury concentrations in fish. Because the relationship between amount of mercury loaded into water bodies and that are methylated are non-linear. Drastically reducing the mercury that is inputted into these water bodies will most likely only produce a minute change in the methylmercury levels found in fish.

Case in point, although our nation's mercury emissions have decreased and mercury deposition in most areas of the continental U.S. have declined, methylmercury levels in fish do not show any clear or consistent patterns. Computer models of northern Wisconsin lakes produced information suggesting that reducing the amount of mercury emitted into the atmosphere probably is not enough to make a difference. These models predict that if mercury emissions were reduced by 5 percent, it would take 8 years for any observable changes in methylmercury concentrations become evident in the fish populations. This decrease in methylmercury would be small.[vii]

Further complicating the relationship between reducing mercury air emissions and the lowering of methylmercury concentrations in fish is the global nature of mercury. It can travel great distances before being deposited. Much of the mercury deposited within our nation's borders is from international sources. Therefore, attempts to reduce mercury loads in particular bodies of water, or methylmercury concentration in fish, would require actions to reduce mercury emissions on a global scale, rather than a local or regional scale.

If we are concerned about reducing the amount of anthropogenically produced mercury in the environment, our efforts should be focused primarily on reducing the emissions of those countries that emit the most mercury - Russia and the region of Southeast Asia. Attempts to reduce the U.S. produced mercury emissions, through measures that tend to be costly, would be fairly insignificant in the overall picture. Because such a significant amount of the total mercury emitted globally is from natural sources, even if anthropogenic mercury emissions were drastically reduced, they may not produce the

desired declines in mercury deposition and methylmercury levels in fish.

These arguments all need to be considered when deciding what level of methylmercury in fish is justifiable for the public health's protection. In addition, because many fish species in our nation's waters already exceed the U.S. EPA's RfD of 0.1 μg/kg/day, using this health action level as a guide for our nation's environmental laws related to methylmercury would most likely produce more stringent standards for anthropogenic emissions of mercury. These U.S. EPA's tougher standards would most likely produce high costs for little benefit due to the complex, non-straightforward cause-and-effect, nature of mercury air emissions and methylmercury concentrations in fish. On the other hand, while the FDA's Action Level of 1.0 ppm and Fish Advisory (targeting primarily women of childbearing age and pregnant women) works to ensure the public's health, the resulting mercury emissions and other mercury related standards would not be as costly as those resulting from using the U.S. EPA's RfD as regulatory guidance. In addition, with the uncertainties in our knowledge and the models of the environmental fate and atmospheric deposition of mercury, we should use the FDA Action Level for guidance in our nation's environmental regulations and standards for mercury. Then, as we fill in the gaps of our knowledge and improve the models, the real risk issue can be revisited to determine if basing mercury regulation on the FDA Action Level is developing beneficial results, or if it is necessary switch to the U.S. EPA's more stringent RfD as a mercury lawmaking guideline.

Appendix A

Seychelles Islands

Myers et al.'s pilot study utilized the Denver Developmental Screening Test – Revised (DDST-R), deep tendon reflexes, overall neurological and muscle tone examinations to assess the effects prenatal methylmercury exposure had on 789 Seychellois infants and toddlers between 1 and 25 months of age. A relationship between fetal exposure and scores on the DDST-R was found using regression analyses. (The results of the DDST-R are scored as normal, questionable or abnormal.) However, the relationship appeared only if the children who scored in the "abnormal" were combined with those whose scored were rated as "questionable." This association disappeared when those questionable scores were treated as passes. In addition, no relationship was found between neurological examination scores and fetal mercury exposure. Although a second evaluation conducted on a subset of 217 of these children at 66 months found a significant relationship between fetal exposure and language function, global intellectual function and perceptual ability. These associations disappeared with all tests except auditory comprehension when a small number of outliers (?) and influential scores were removed. The Seychelles Islands main study assessing the development of 779 children at 6 ½, 19 and 29 months of age found no significant association between fetal methylmercury exposures and mental and physical development. However, an inverse relationship between boys at 29 months and activity was observed - boys with higher methylmercury exposures had lower activity levels. (It is important to note that this was detected using a more subjective behavioral test, and the examiner was the one who judged the level of activity.)[1]

Faroe Islands

The neuropsychological tests Grandjean et al. chose to administer to the Faroese children included tasks that had been described as being affected by the neuropathologic abnormalities in congenital methylmercury poisoning incidents. Children were asked to partake in tests of fine motor tasks, attention measures, executive function tests, language tests, short-term memory tests and visuospatial tasks. Maternal hair mercury concentration was measured when the child was born (mean (M) = 4.27 μg/g, interquartile range (IR) = 2.6 – 7.7μg/g). In addition, the child's hair methylmercury levels were measured at 12 months (M = 1.12 μg/g, IR = 0.7 – 1.9 μg/g), and 7 years of age (M = 2.99, IR = 1.7 – 6.1 μg/g). Child's cord blood (M = 22.9 μg/L, IR = 13.4 – 41.3 μg/L) and child's blood at 7 years (M = 8.82 μg/L, IR = 4.8 – 18.2 μg/L) served as additional biomarkers. Multiple regression analyses found that cord blood concentrations showed the clearest associations with decreased attention, memory and language development.[1] In addition, investigators determined that deficits seen in several brain function domains were related to increased prenatal methylmercury exposures, while postnatal exposures appeared to have less of an effect.[1] This study, and many post-study analyses performed by other researchers with these data, concluded that these adverse effects were not confounded by the relatively high levels of polychlorinated biphenyls (PCBs) that this population is also exposed to from consuming pilot whale blubber, the site of methylmercury in the animal.[ix]

New Zealand

Kjellstrom et al. conducted a battery of 26 psychological tests, behavioral tests, and tests of aptitude on New Zealand children to assess their general intelligence, academic attainment, language development, social adjustment, and fine and gross motor coordination. Hair mercury levels were quantified on a binary scale where the variable either was >6 mg/kg or 3 – 6 mg/kg. Multiple regression analyses performed found statistically significant associations between maternal hair mercury and poorer scores on full-scale IQ, visual-spatial skills, language development and gross motor skills. Based on their data, Kjellstrom et al. concluded that there was an "apparent consistent association" in the New Zealand cohort between prenatal exposure to high levels of methylmercury and decreased performance on scholastic and psychological tests.[1]

In a reanalysis of the New Zealand data, Crump et al. performed benchmark dose (BMD) calculations and additional regression analyses using the maternal hair mercury levels as a continuous scale (rather than a binary one). With P_o fixed at 0.05, just as U.S. EPA did in its methylmercury analyses described below, the benchmark dose lowest limits (BMDL, which is the 95 percent lower limits of the BMD curve) ranged from 17 mg/kg to 24 mg/kg. The regression analyses found no statistically significant associations between methylmercury exposure and test scores. However, both the BMDL values and the regression analyses were heavily influenced by an outlier, a single child whose mother had a hair mercury concentration of 86 ppm, more than four times as high as any other mother. When they excluded this child from the analyses, Crump et al. found the BMDLs ranged from 7.4 kg/mg to 10 mg/kg, and there were significant associations between maternal hair concentrations and scores on 6 tests related to language development, general cognition, reading and perceptual performance.[xxvi]

Appendix B: Key Pieces of Legislation & Agreements Related to Mercury

Year	Legislation
1971	Hg designated as hazardous pollutant.
1972	Federal Insecticide, Fungicide, Rodenticide Act banned many pesticides containing Hg. Federal Water Pollution Control Act authorized EPA to regulate the Hg discharge into waterways.
1973	Hg designated as toxic pollutant. Standards for Hg ore processors and chlor-alkali plants enacted. Dumping Hg and Hg compounds into the ocean was prohibited.
1978	Resource Conservation and Recovery Act established regulations for the disposal of Hg waste.
1992	EPA banned land disposal of high Hg content wastes generated by chlor-alkali facilities.
1993	EPA canceled the registrations for the last 2 Hg-containing fungicides at the manufacturer's request.
1994	Congress suspended the sale of National Defense Stockpile Hg because of EPA's concerns with environmental problems related to the toxin.
1995	EPA's new regulations on municipal waste combustors are designed to reduce Hg emissions from these facilities by 90% from 1990 emission levels.
1996	The Mercury Containing and Rechargeable Battery Management Act prohibited batteries being sold without recyclability or disposal labels and phased out most batteries containing Hg.
1997	EPA's new standards for medical waste incinerators will reduce Hg emissions from these facilities by 94% from 1990 levels once fully implemented in 2002.
1997	The U.S./Canadian Great Lakes Bi-National Toxics Strategy was created. This agreement sets a goal to significantly reduce human use and release of Hg in the Great Lakes Basin by 2006.
1998	The 1998 Protocol on Heavy Metals of the Convention on Long Range Transboundary Air Pollution involves the U.S., Canada and all European nations.
1999	EPA's new standards for hazardous waste combustors are designed to reduce Hg emissions from these facilities by 50% from 1990 emission levels.
2000	EPA lowered the threshold level for reporting Hg emissions to the Toxic Release Inventory. Phase II North American Regional Plan on Mercury, under the North American Agreement on Environmental Cooperation, involves U.S., Canada, and Mexico.
2004	EPA developing emission standards for small sources of air toxins (including Hg). These standards are expected to be issued in 2004.
2005	The Chlorine Institute has committed to reducing the amount of Hg used in the chlor-alkali industry by 50% by 2005. Initially, this will occur through tighter controls and later, closing these manufacturing facilities will shift private stocks into market supply line.

Appendix C: Mean Mercury Concentrations in Selected Fish & Shellfish Species

SPECIES	MEAN (PPM)	RANGE (PPM)	# SAMPLED
Tilefish	1.45	0.65-3.73	60
Swordfish	1.00	0.10-3.22	598
Shark	0.96	0.05-4.54	324
King Mackerel	0.73	0.30-1.67	213
Eastern Chain Pickerel	0.61	0.014-2.81	N/A
Red Snapper	0.60	0.07-1.46	10
Orange Roughy	0.58	0.42-0.76	9
Largemouth Bass	0.52	0.0005-8.94	N/A
Walleye	0.43	0.005-16	N/A
Northern Pike	0.36	0.005-4.4	N/A
Smallmouth Bass	0.32	0.005-3.34	N/A
Tuna (fresh or frozen)	0.32	1.30 (max.)	191
Lobster Northern (American)	0.31	0.05-1.31	88
Lake Trout	0.27	0.005-2	N/A
Trout (Saltwater)	0.27	1.19 (max.)	4
Halibut	0.23	0.02-0.63	29
Sablefish	0.22	0.70 (max.)	102
Pollock	0.20	0.78 (max.)	107
Cod (Atlantic)	0.19	0.33 (max.)	11
Dungeness Crab	0.18	0.02-0.48	50
Ocean Perch	0.18	0.31 (max.)	10
Blue Crab	0.17	0.02-0.50	94
Haddock (Atlantic)	0.17	0.07-0.37	10
Whitefish	0.16	0.31 (max.)	2
Tanner Crab	0.15	0.38 (max.)	55
Herring	0.15	0.016-0.28	8
Spiny Lobster	0.13	0.27 (max.)	8
Perch (Freshwater)	0.11	0.10-0.31	4
Perch (Saltwater)	0.10	0.10-0.15	6
King Crab	0.09	0.02-0.24	29
Catfish	0.07	0.31 (max.)	22
Scallop	0.05	0.22 (max.)	66
Flounder/Sole	0.04	0.18 (max.)	17
Salmon (fresh, frozen or canned)	No Data	0.18 (max.)	52
Oysters	No Data	0.25 (max.)	33

Shaded = fish species mentioned in paper as having highest concentrations of methylmercury

Figure 1: U.S. Industrial Reported Consumption of Mercury (1970 – 1997)

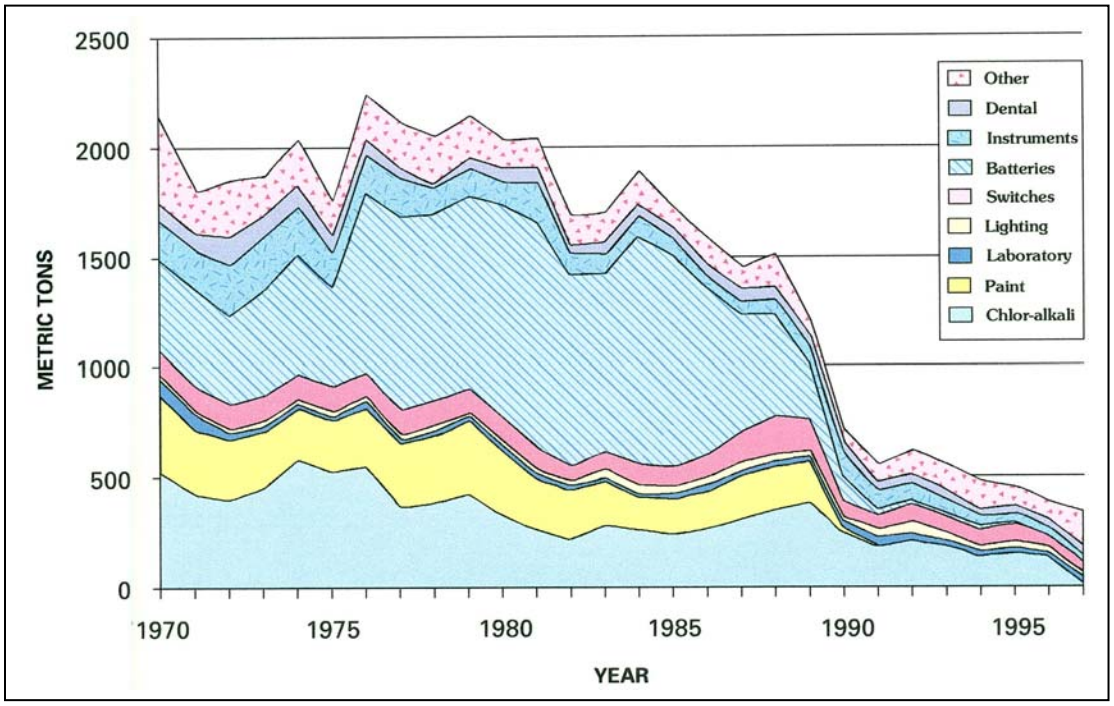


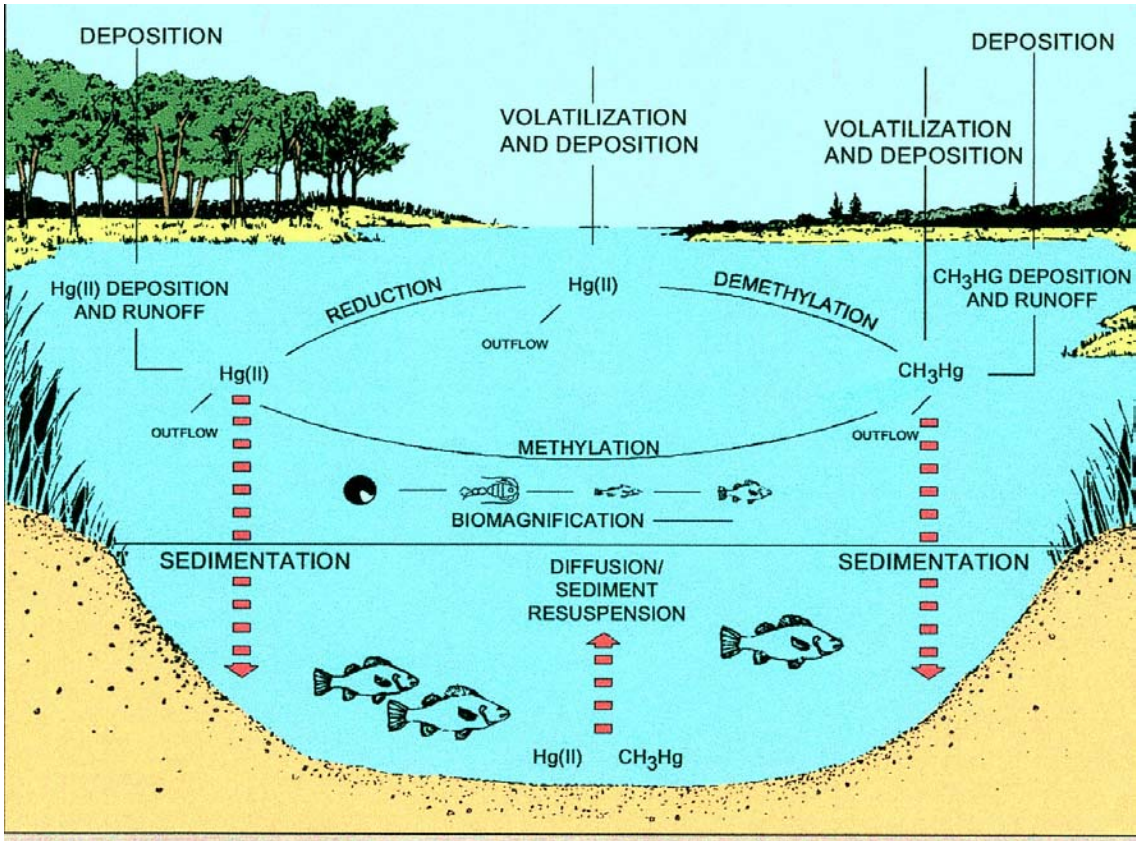
Figure 2: Global Mercury Production, Use and Flow (1990 & 1996)

In metric tons

Table 15. Global mercury production, use, and flow 1990 and 1996, in metric tons.
[n.a., not available]

Region	Production		Use								Net flow	
			Chlor-alkali		Manufactures		Artisanal gold		Stock changes			
	1990	1996	1990	1996	1990	1996	1990	1996	1990	1996	1990	1996
North America	1,297	526	319	154	553	238	n.a.	n.a.	255	84	-170	-50
South America	0	5	72	62	65	20	200	100	34	18	+371	+195
West Europe	882	1,141	1,067	631	440	177	n.a.	n.a.	-1,165	-30	-540	-363
East Europe	163	25	209	184	88	28	n.a.	n.a.	30	21	+164	+208
FSU	1,400	785	34	34	150	60	n.a.	n.a.	459	9	-757	-682
Middle East	47	0	101	81	35	18	n.a.	n.a.	7	5	+96	+104
Africa	637	347	43	36	1	9	unknown	unknown	3	2	-570	-300
India and Pakistan	0	0	138	133	66	30	n.a.	n.a.	20	16	+224	+179
NE. Asia	930	508	0	5	375	445	unknown	unknown	1,688	701	+1,133	+643
SE. Asia	0	0	20	24	25	36	unknown	unknown	4	6	+49	+66
World Total	5,356	3,337	2,003	1,344	1,818	1,061	200+	100+	1,335	832	0	0

Figure 3: Aquatic Mercury Cycle



Mercury cycling pathways in aquatic environments are very complex. The various forms of mercury can be converted from one to the next; most important is the conversion to methylmercury (CH_3Hg^+), the most toxic form. Ultimately, mercury ends up in the sediments, fish and wildlife, or evades back to the atmosphere by volatilization. Reprinted with permission from Mercury Pollution: Integration and Synthesis. Copyright Lewis Publishers, an imprint of CRC Press.

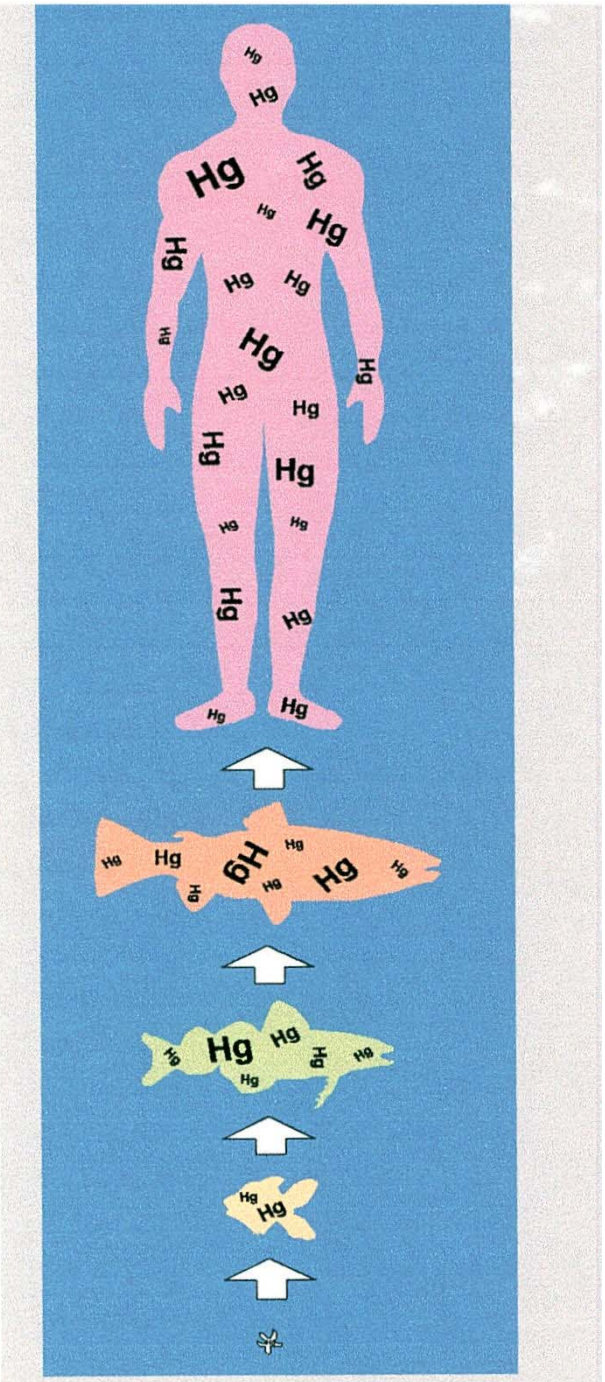


Figure 4. *Mercury (Hg) biomagnifies from the bottom to the top of the food chain. Even at very low input rates to aquatic ecosystems that are remote from point sources, biomagnification effects can result in mercury levels of toxicological concern.*

Figure 5

Summary of Statewide Advisories by Waterbody Type			
State	**Lake**	**River**	**Coastal Waters**
Alaska*	N/A	N/A	
Alabama			Mercury
Connecticut	Mercury	Mercury	PCBs
Dist. of Columbia	PCBs	PCBs	
Florida			Mercury
Georgia			Mercury
Indiana		Mercury PCBs	
Kentucky	Mercury	Mercury	
Louisiana			Mercury
Maine	Mercury	Mercury	Dioxins Mercury PCBs
Maryland	Mercury	Mercury	
Massachusetts	Mercury	Mercury	PCBs
Michigan	Mercury		
Minnesota	Mercury		
Mississippi			Mercury
Missouri	Mercury	Mercury	
New Hampshire	Mercury	Mercury	PCBs
New Jersey	Mercury	Mercury	PCBs Cadmium Dioxins
New York	PCBs Chlordane Mirex DDT	PCBs Chlordane Mirex DDT	Cadmium Dioxins
North Carolina	Mercury	Mercury	Mercury
North Dakota	Mercury	Mercury	
Ohio	Mercury	Mercury	
Pennsylvania	Mercury	Mercury	
Rhode Island			PCBs
South Carolina			Mercury
Texas			Mercury
Vermont	Mercury	Mercury	
Wisconsin	Mercury		

*Alaska's statewide advice places no restrictions on consumption of fish or wildlife.

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Mercury Biomagnification diagram. From the U.S. Department of the Interior, U.S. Geological Survey's *Mercury Contamination of Aquatic Ecosystems*. Fact Sheet FS-216-95. 1995.

Summary of Statewide Advisories by Waterbody Type chart. From the U.S. Environmental Protection Agency's *Update: National Listing of Fish and Wildlife Advisories* Fact Sheet. EPA-823-F-02-007. May 2002.

Appendices:

Appendix B: Key Pieces of Legislation & Agreements Related to Mercury table. Adapted from Szopek JL and Goonan TG *U.S. mercury reported consumption, production, price and legislation (1970 – 1997)* graph in the U.S. Geological Survey Circular 1197. Information for 1995, 1997 – 2004 obtained from the U.S. EPA's *Mercury White Paper*. Information for 2005 obtained from Szopek JL and Goonan TG U.S. Geological Survey Circular 1197.

Appendix C: Mean Mercury Concentrations in Selected Fish & Shellfish Species table. Adapted from U.S. EPA's *Mercury Update: Impact on Fish Advisories* Fact Sheet and U.S. Food and Drug Administration, Center for Food Safety and Applied Nutrition's *Mercury Levels in Seafood Species* (Tables 1-3) found at http://www.cfsan.fda.gov/~acrobat/hgadv2.pdf

Regional Popularity of Fish and Shellfish Species table. From U.S. EPA's *Mercury Study Report to Congress*. (VII):5-15.

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