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Mercury in the Environment: Sources and Health Risks

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Mercury in the Environment: Sources and Health Risks

Summary

Concern about mercury in the environment has increased in recent years due to emerging evidence that exposure to low levels of mercury may harm the developing nervous systems of unborn children. At least five bills in the 109th Congress aim to reduce mercury emissions from coal-fired electric utilities. The various proposals and a final regulation promulgated by the U.S. Environmental Protection Agency (EPA) on March 15, 2005, differ in how much and how soon emission reduction would be required, and in whether reductions would be achieved through controls at each plant or through a nationwide cap and trade system. The latter approach could allow individual plants to continue emitting current levels of mercury, potentially worsening conditions at nearby "hot spots." Analysis of competing proposals raises questions about the sources, fate, and toxicity of mercury in the environment. This CRS report provides background information about mercury and summarizes recent scientific findings. For information about regulatory proposals to reduce environmental emissions of mercury, see CRS Report RL32868, *Mercury Emissions from Electric Power Plants: An Analysis of EPA's Cap-and-Trade Regulations.*

Mercury is a natural element found in rocks, soil, water, air, plants, and animals, in a variety of chemical forms. Natural forces move mercury through the environment, from air to soil to water, and back again. Industrial activities have increased the portion of mercury in the atmosphere and oceans, and have contaminated some local environments. Coal-fired electric utilities are the largest single source of U.S. mercury emissions, according to EPA, but mobile sources also are important. The chemical form of mercury generally determines how it moves through the environment, but mercury can and does change form relatively rapidly where bromine and other oxidizing substances (e.g., ozone) are abundant. In soil or sediments of lakes, streams, and probably oceans (especially where water is oxygen-poor and acidic, and sulfate is present), bacteria convert inorganic mercury to more toxic methylmercury, which can accumulate in fish. Newly deposited mercury seems to be more readily converted than older deposits.

People and wildlife who eat contaminated fish can be exposed to toxic levels of methylmercury. In people, methylmercury enters the brain, where it may cause structural damage. Methylmercury also crosses the placenta. The National Research Council has reported that the human fetus is sensitive to methylmercury exposure, and the current risk to U.S. women who eat large amounts of fish and seafood during pregnancy is "likely to be sufficient to result in an increase in the number of children who have to struggle to keep up in school." Some studies indicate that the cardiovascular system may be even more sensitive. Mercury concentrations generally are low, but the estimated safe blood-mercury level is exceeded in about 6% of U.S. women between the ages of 16 and 49 years. EPA and the Food and Drug Administration advise women of child-bearing age to avoid certain large fish, and to limit the amount eaten of other fish. In making choices about fish consumption, the health benefits of eating fish also should be considered. Fish-eating wildlife also are exposed to methylmercury, but it is not clear whether *typical* current levels of environmental contamination are harmful. This report will be updated as warranted by significant scientific discoveries.

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Introduction

Congressional concern about mercury in the environment has greatly increased in recent years due to emerging scientific evidence that exposure to low levels of mercury may harm the developing nervous systems of young children. At higher levels of exposure, mercury is known to be a potent neurotoxin. People in the United States are exposed to mercury primarily by eating large, predatory fish. Risks of health problems for people who consume mercury in fish have caused wide public concern and prompted the U.S. Environmental Protection Agency (EPA) and the Food and Drug Administration (FDA) to issue consumer alerts, warning women of child-bearing age and young children to avoid certain fish altogether and to limit the number of meals for other fish.

Numerous legislative proposals in the 109th Congress aim to reduce levels of mercury in the environment — in consumer products, in solid waste, in utility and other emission sources, and in surface water. Most of these proposals focus on sources of mercury emissions to air, because atmospheric mercury deposition accounts for most of the mercury in U.S. freshwater lakes and streams. At least five proposals target emissions from coal-fired electric utilities, because they are thought to be the last remaining major uncontrolled source of mercury emissions. These various proposals and a final regulation promulgated by the U.S. Environmental Protection Agency (EPA) on March 15, 2005, differ in how much and how soon emission reduction would be required, as well as in the extent to which reductions would be distributed geographically across the United States.

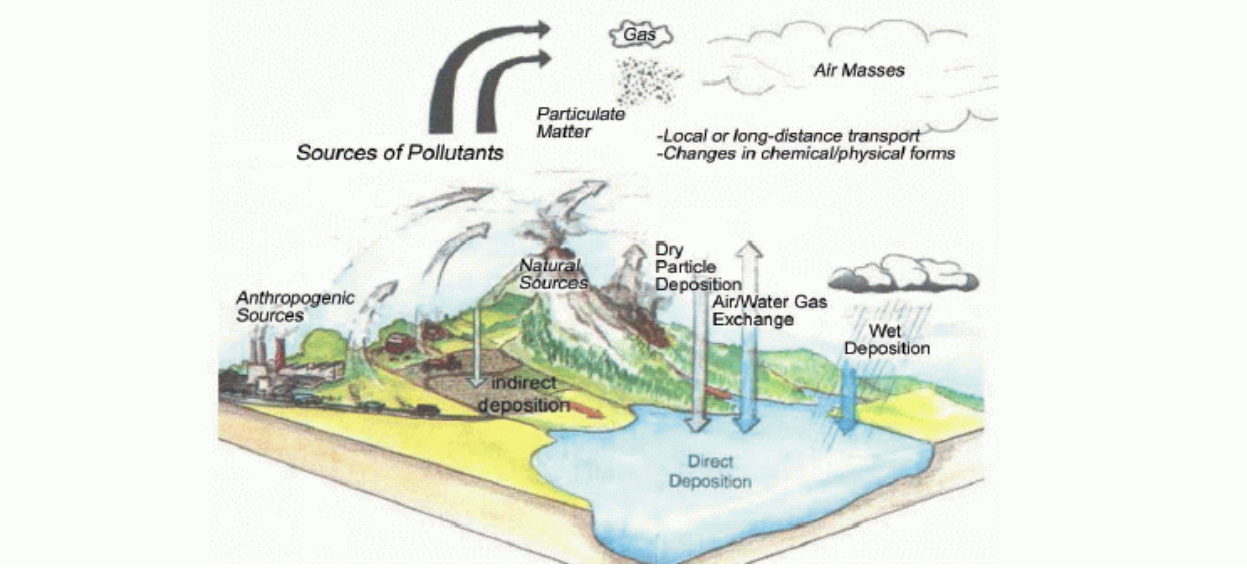
Analysis of the competing policy proposals for reducing mercury emissions raises questions about the urgency of a need for emission controls, the likelihood that they will reduce mercury contamination of fish, and the possibility that overall reductions might be achieved at the expense of local "hot spots" of mercury contamination. To answer such questions requires an understanding of the sources, fate, and toxicity of mercury in the environment — an understanding that is growing quickly as the results of numerous scientific studies are being reported. This CRS report provides background information about mercury, and summarizes recent scientific findings. It discusses the sources (i.e., natural versus industrial, historic versus modern) and chemical forms of mercury in the environment; how mercury moves through the environment and concentrates in fish (i.e., the fate of mercury); and the risks to human health and wildlife of mercury exposure through fish consumption. Each of these major sections of the report aims to summarize scientific evidence relevant to specific arguments and questions that have emerged in the policy context. For example, the section on mercury in the environment addresses the question "Are utility emissions deposited locally or regionally, or do they rise to merge with the global atmospheric mercury pool?" For information about specific

regulatory proposals to reduce environmental mercury, see CRS Report RL32868, *Mercury Emissions from Electric Power Plants: An Analysis of EPA's Cap-and-Trade Regulations*; CRS Issue Brief IB10137, *Clean Air Act Issues in the 109th Congress*; or CRS Report RL31908, *Mercury in Products and Waste: Legislative and Regulatory Activities to Control Mercury*.

Sources of Mercury in the Environment

Mercury is a natural element, a silver-colored, shiny, liquid metal that is found in a variety of chemical forms in rocks, soil, water, air, plants, and animals. Sometimes mercury occurs in its elemental, relatively pure form, as a liquid or vapor, but more commonly mercury is found combined with other elements in various compounds, which may be inorganic (e.g., the mineral cinnabar, a combination of mercury and sulfur) or organic (e.g., methylmercury).[1]

Figure 1. Emission and Deposition of Pollutants



Source: EPA, *Frequently asked questions about atmospheric deposition: A handbook for watershed managers*, EPA-453/R-01-009, Sept. 2001, p. 4, at [http://www.epa.gov/oar/oaqps/gr8water/handbook/airdep_sept_2.pdf].

Natural forces move mercury through the environment, from air to soil to water, and back again. Volcanoes and deep sea vents release tons of mercury to the atmosphere and oceans. Mercury in the air falls to earth with dust, rain, and snow. Mercury evaporates from the oceans, leaves of plants, and other surfaces back into the air.[2] Depending on geologic and meteorologic conditions, the relative amounts of mercury in the atmosphere, surface water, or soil may vary from one year, decade, century, or millennium to another.

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[1]Organic compounds consist of carbon combined with other substances. Organic compounds, such as methylmercury, are created by, and generally are more readily absorbed by, living things.

[2]Depending on the mercury compound, either a liquid or solid may turn into gas. In the latter case, the correct term is sublime, rather than evaporate.

During the past 500 years or so, human activities have released mercury from its relatively stable and water-insoluble form (cinnabar) in rocks and soil through mining, fossil fuel combustion, and other activities, and so have increased the portion of mercury that is actively cycling through the atmosphere, surface waters, plants, and animals as it changes chemical and physical form. Released mercury may enter the air, persist in the atmosphere and travel great distances or be deposited locally, dissolve in water droplets, settle back onto the land or water, re-enter the air (i.e., be re-emitted), be buried in lake or ocean sediments, or be taken into plants and animals. The generally accepted estimate is that roughly three to five times as much mercury is mobilized today as was mobile before industrialization.[3] However, the author of one recent study argues that the mercury deposited from the atmosphere today is at least 10 times the amount of mercury that was being deposited 500 years ago.[4]

In 1995, about 1,913 metric tons (roughly 2,104 U.S. tons)[5] of mercury were *newly* emitted globally as a result of stationary combustion, metal production, cement production, and waste disposal.[6] Roughly another 514 metric tons (565 U.S. tons) were emitted from other human sources, including chlor-alkali plants, gold production, and mercury uses.[7] Thus, 2,427 metric tons (2,670 U.S. tons) of mercury were released due to human activities in 1995, according to recent estimates.[8] These and other mercury emissions from human activities (past and present) account for at least 50% and perhaps as much as 75% of current, annual, global mercury emissions from all sources (including natural sources), but a large, unknown portion of those mercury emissions is due to past rather than current human activities, according to EPA estimates.[9] The most recent estimates of global, natural mercury emissions

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[3]Tom Atkeson and Don Axelrad, *2004 Everglades Consolidated Report* (2003), Chapter 2B, "Mercury Monitoring, Research and Environmental Assessment," p. 2B-7; C. H. Lamborg, H. Balcom, D. R. Engstrom, et al., "Modern and historic atmospheric mercury fluxes in both hemispheres: Global and regional mercury cycling implications," *Global Biogeochemical Cycles*, v. 16, n. 4 (2002), pp. 51-1 to 51-11.

[4]R. Bindler, "Estimating the natural background atmospheric deposition rate of mercury utilizing ombrotrophic bogs in southern Sweden," *Environmental Science & Technology*, v. 37, no. 1 (2003), pp. 40-46.

[5]A metric ton is 1,000 kilograms (one million grams), or about 2,200 pounds.

[6]E. G. Pacyna and J. M. Pacyna, "Global emission of mercury from anthropogenic sources in 1995," *Water, Air, and Soil Pollution*, v. 137 (2000), pp. 149-165.

[7]J. M. Pacyna, E. G. Pacyna, F. Steenhuisen, et al., "Mapping 1995 global anthropogenic emissions of mercury," *Atmospheric Environment*, v. 37, supp. no. 1 (2003), pp. S109-S117.

[8]Corrected emission data for 1995 and new data for 2000 are available on the internet, but have not yet been published. These indicate that global mercury emissions for 1995 totaled 2,317 metric tons (2,549 US tons). For 2000, total global emissions were estimated to be 2,188 metric tons (2,407 US tons). See J. Pacyna and E. Pacyna, "Global Anthropogenic Mercury Emission Inventories for 2000 and 1995," *Journal of Air and Waste Management Association* (in prep. 2005), at [http://amap.no/Resources/HgEmissions/HgInventoryDocs.html], visited August 12, 2005.

[9]EPA, *Mercury Study Report to Congress*, vol. 1, Executive Summary, EPA-452/R-97-003, (Washington: GPO, 1997), pp. 3-4.

range between roughly 1,600 and 3,200 metric tons (1,960 and 3,520 U.S. tons) per year.[10]

People have released mercury to the environment primarily through mining and smelting of minerals, burning of fossil fuels (e.g., coal, oil, and diesel fuel), use and disposal of mercury, certain industrial processes (e.g., chlorine production and cement production), and burning of municipal and medical wastes. In some parts of the world such activities are increasing, but in the United States, annual mercury emissions are decreasing. Most of the largest and most direct sources of U.S. mercury releases to water and air have been eliminated. Among the remaining U.S. industrial sources, coal-fired electric utilities are the most important, accounting for about 40% of current U.S. mercury releases.[11]

Three estimates of U.S. national emissions are presented in **Table 1**. The first two estimates were made by EPA for the National Emissions Inventory.[12] CRS added 12 tons of emissions from gold mines to the EPA emission inventory that was conducted for 1995, at the suggestion of EPA.[13] EPA was unaware of the emissions from that source at the time the inventory was conducted. The "other" category encompasses emissions from various unidentified industries, including most iron and steel mills. EPA advised CRS to note that there are some sources not accounted for in the 1999 EPA inventory, such as iron and steel production using mercury-contaminated scrap, which probably accounts for 7-10 tons of emissions per year.[14] These emissions are not included in the "other" category. EPA also does not include mobile source emissions in its inventory, although these might be significant, because the agency is still developing an estimate.

Since the time that EPA completed its 1999 inventory, the medical waste incinerator rules promulgated under the Clean Air Act have been fully implemented, which may have further reduced emissions from that source, and gold mining emissions have decreased due to a voluntary project. Chlorine production emissions also may have declined since the 1999 inventory, because some facilities closed, but

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[10] C. Seigneur, K. Vijayaraghavan, K. Lohman, et al., "Global source attribution for mercury deposition in the United States," *Environmental Science & Technology*, v. 38, n. 2 (2004), pp. 555-569.

[11]However, emissions from two additional sources are not well quantified and may be larger contributors: mobile sources and chlor-alkali plants. In a recent regulatory action, EPA stated that 65 tons of mercury were consumed by nine chlor-alkali plants but were not reported to have been released from chlorine production plants in the year 2000. That amount of mercury is greater than the amount released by all coal-fired utilities annually, and is equivalent to 124 gallons of mercury per plant. Although industry personnel claim that a large proportion of the "consumed" mercury condenses and accumulates in pipes, tanks, and other equipment, EPA considers the discrepancy between mercury purchased, consumed, and released to be unexplained (68 *Federal Register* 70920, Dec. 19, 2003).

[12]EPA, National Emissions Inventory, at [http://www.epa.gov/ttn/chief/net/1999inventory.html#final3haps], visited April 9, 2004.

[13]Alexis Cain, personal communication, March 12, 2004.

[14]Ibid.

one additional facility was identified and included in emission estimates by Seigneur et al.,[15] which appear in the third column. These latter estimates were calculated by researchers with Atmospheric & Environmental Research, Inc., and published in 2004, but represent emissions in the year 1998. It is not clear why the Seigneur estimates for 1998 emissions from waste incineration are so much larger than EPA estimates for emissions from that category in 1999. Seigneur included emissions from landfills and electric arc furnaces in the "other" category. The Electric Power Research Institute (EPRI) provided the estimates used in that article for utility emissions. Both the EPRI calculations and the EPA estimate for 1999 utility emissions were based on measurements of mercury content in coal and stack emissions that were collected for the year 1999, in response to an information collection request issued by EPA.[16]

Table 1. Estimates of U.S. Mercury Emissions from Major Sources
(U.S. tons per year)

Source	EPA 1995	EPA 1999	Seigneur et al. 1998
Electric Power	51	48	46
Industrial Boilers	12	12	14
Gold Mining	12	12	7
Waste Incineration/ Combustion	77	15	32
Chlorine Production[17]	8	7	7
Mobile Sources[18]	—	—	27
Other	37	25	34
Total	197	118	167

Source: EPA, National Emissions Inventory, at [http://www.epa.gov/ttn/chief/net/1999inventory.html#final3haps], visited April 9, 2004.

Fate of Mercury Released to the Environment

Transport, Deposition, Re-emission, and Transformation. Chemical form generally determines the ease with which mercury moves through the air, water, and soil and over distances. For example, elemental mercury emissions may remain airborne for more than a year, traveling around the world as part of the so-called "global pool" of atmospheric mercury. About 95% of atmospheric mercury is

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[15]Seigneur et al., 2004.

[16]*Recommendations for the Utility Air Toxics MACT Final Working Group Report*, Oct. 2002, at [http://www.epa.gov/ttnatw01/combust/utiltox/wgfinalreport10_02.pdf].

[17]See footnote 9, above.

[18]Ibid.

elemental. Particulate and reactive gaseous mercury (both organic and inorganic) are found in the atmosphere in smaller amounts, because they travel shorter distances from the point of emission and are more quickly deposited. Reactive gaseous mercury typically is deposited within about 100 kilometers of the point of emission.[19] Coal-fired electric utility emissions vary depending on the technology and coal used at each plant, but are roughly 50% elemental mercury, according to EPA.[20]

However, the chemical form of mercury emissions can and does change in the atmosphere, making it difficult to predict the fate of particular emissions, including utility emissions. Elemental mercury emitted to the atmosphere can attach to particles or change to a water-soluble form (i.e., a reactive gas) that more easily combines with other chemicals and deposits relatively quickly. Reactive, gaseous mercury is more likely to form (and to be deposited) in the presence of sunlight. This explains why measured concentrations of atmospheric mercury generally are lower during the day than they are at night.

Mercury deposition in North America increases in spring and peaks in summer, according to data from the mercury deposition network.[21] Higher summer deposition probably results, at least in part, from the increase in solar energy that is available to spark key chemical reactions (i.e., oxidation). For example, scientists have shown that in the lower layers of the atmosphere (i.e., roughly 400 meters of land or 1,000 meters of the ocean surface), elemental mercury gas may be quickly oxidized by bromine, chlorine, ozone, or hydroxide in the presence of sunlight, leading to local "mercury depletion events."[22] In such cases, concentrations of elemental gaseous mercury in the atmosphere decrease rapidly as the oxidized forms of mercury are deposited to the surface in dry deposits (i.e., without the help of rain or snow).[23] This has been observed during the summer in the Arctic and Antarctic regions, and over the oceans.

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[19]Gwendolyn Judson (undated), "Analysis of mercury speciation profiles currently used for atmospheric chemistry modeling," Wisconsin Department of Natural Resources, Madison, WI, at [http://www.dnr.state.wi.us/org/aw/air/staff/hganalysisteam/docs/hgspeciation.pdf], visited April 5, 2004.

[20]69 *Federal Register* 4674, Jan. 30, 2004.

[21]Environment Canada, Ecological Monitoring and Assessment Network, *Meeting the Challenges of Continental Pollutant Pathways*, Mercury Case Study, at [http://www.eman-rese.ca/eman/reports/publications/99_mercurywkshp/intro.html].

[22]P. A. Ariya, A. Ghalizov, and A. Gidas, "Reactions of gaseous mercury with atomic and molecular halogens: Kinetics, products studies and atmospheric implications," *Journal of Physical Chemistry*, v. 106 (2002), pp. 7310-7320; B. Pal and A. P. Ariya, "Studies of ozone initiated reactions of gaseous mercury: Kinetics, product studies, and atmospheric implications," *Physical Chemistry and Chemical Physics*, v. 6, n. 3 (2004), pp. 572-579; I. M. Hedgecock and N. Pirrone, "Chasing quicksilver: Modeling the atmospheric lifetime of HgO(g) in the marine boundary layer at various latitudes," *Environmental Science & Technology*, v. 38, n. 1 (2004), pp. 69-76.

[23]United Nations Environment Programme, Inter-Organization Programme for the Sound Management of Chemicals, *Global Mercury Assessment* (2003), p. 28, at [http://www.chem.unep.ch/mercury/Report/final-report-download.htm.

Summer mercury deposition also might be a result of increased oxidation by ozone. Higher ozone concentrations occur in summer, also due to the action of sunlight.[24]

Mercury that is deposited onto plants or soil can be re-emitted to air, attached to soil, dissolved, washed away, buried, or ingested. It may again change chemical form. Mercury often attaches to soil particles, especially humus. Recent research indicates that soil may be a repository for the largest portion of mercury emitted in the past.[25]

Mercury may be delivered to surface water bodies by air, in soil, or in streams and rivers. For many isolated lakes, very large lakes, and the oceans, atmospheric deposition (wet and dry) accounts for the largest portion of mercury contamination.[26]

Mercury deposited or delivered to surface water may be re-emitted to air, remain suspended or dissolved in the water column, be deposited in sediments, or absorbed or ingested by living things. Re-emission rates from the ocean surface to air may be very large.[27] For example, some experts believe that as much as 90% of the mercury deposited to the ocean surface might be re-emitted.[28] Nevertheless, the concentration of mercury in the mixing layer of the deep oceans probably is increasing by a few percent per year.[29]

Mercury in the air eventually will fall back to land or surface water. A recent analysis of deposition data collected for both hemispheres indicates that total gaseous mercury increased in the late 1970s, peaked in the late 1980s, decreased somewhat until the mid-1990s, and has remained constant since then.[30] At present, approximately 5,000 metric tons (5,500 U.S. tons) of mercury are deposited globally each year.[31]

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[24]P. Weiss-Penzias, D. A. Jaffe, A. McClintick, et al., "Gaseous elemental mercury in the marine boundary layer: Evidence for rapid removal in anthropogenic pollution," *Environmental Science & Technology*, v. 37, n. 17 (2003), pp. 3755-3763.

[25]R. P. Mason and G. R. Sheu, "Role of the ocean in the global mercury cycle," *Global Biogeochemical Cycles*, v. 16, n. 4 (2002), pp. 40-1 to 40-14; James G. Wiener, et al., "Ecotoxicology of Mercury," in David J. Hoffman, et al, *Handbook of Ecotoxicology*, 2nd ed. (Boca Raton, FL: Lewis Publishers, 2003), p. 418.

[26]Mason and Sheu.

[27]M. S. Landis and G. J. Keeler, "Atmospheric mercury deposition to Lake Michigan during the Lake Michigan mass balance study," *Environmental Science & Technology*, v. 36, n. 21 (2002), pp. 4518-4524.

[28]Robert Mason, personal communication, April 1, 2004.

[29]Mason and Sheu.

[30]R. Slemr, E. G. Brunke, R. Ebinghaus, et al., "Worldwide trend of atmospheric mercury since 1977," *Geophysical Research Letters*, v. 30, n. 10 (2003), p. 1516.

[31]Ibid.

Layered samples (known as cores) of glaciers and peat provide historical records of mercury deposits that clearly show the contemporary impact on land of major trends in mercury emissions. That is, cores record the historical rise in mercury emissions and deposition due to mining and industrialization. However, while such records inform us about relative changes in global, regional, and local emissions over a scale of years, even centuries, they provide little information about the precise relationship between particular emissions and particular deposits. This is because the path and time taken by emitted mercury to cycle through environmental media depends on its chemical form, as well as on physical conditions like height of emission, temperature, sunlight, wind speed and direction, humidity, and the presence of certain other substances, such as ozone.

Atmospheric deposition tends to be greater in areas closer to emission sources and in locations with more rainfall. Thus, EPA has estimated that about 60% of mercury deposited in the United States is from local or regional U.S. sources, and deposition increases from west to east.[32] Local or even regional deposition can result in areas of relatively high deposition, or "hot spots." Deposition of mercury in particular cases varies, however, depending on many factors, including regional and local climate and weather patterns, soil types, topography, vegetation, and local or regional sources of mercury emissions.[33] Thus, mercury may be deposited near to or far from an emission source.[34]

The relative contribution of various sources to mercury deposition also can change over time. For example, the record of mercury deposition in ice cores from Fremont Glacier, Wyoming, shows peaks of high mercury deposition following volcanic eruptions in the northern and southern hemispheres, as well as during the California Gold Rush.[35] Such cores are difficult to interpret, however, because they reflect local as well as global influences.

Only a few ecosystems have been studied in sufficient detail to determine the sources of mercury contamination. However, additional information about emission sources and deposition is being gathered through monitoring and modeling across the continental United States, particularly as states undertake detailed analyses of steps needed to restore the quality of waters that are impaired by mercury. According to EPA, more than 700 bodies of water throughout the United States are listed as impaired by mercury; in most cases, the source of the mercury contamination is air deposition. To address these impairments, states are developing Total Maximum Daily Loads (TMDLs), which are plans to bring those waters into attainment with

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[32]EPA (undated) *Draft Report, Mercury Sources and Regulations, 1999 Update*, "The Binational Toxics Strategy — Canada and United States," p. 4, at [http://www.epa.gov/glnpo/bns/mercury/stephg.html], visited Apr. 9, 2004.

[33]Ibid.

[34]*Everglades Consolidated Report*, p. 2B-7; Seigneur et al.

[35]U.S. House of Representatives, Committee on Science, Subcommittee on Environment, Technology, and Standards, *Mercury Emissions: State of the Science and Technology*, hearing, Nov. 5, 2003, statement of David P. Krabbenhoft, at [http://www.house.gov/science/hearings/ets03/nov05/krabben.htm], visited Apr. 14, 2004.

water quality standards. The Florida Everglades and Devil's Lake in Wisconsin were selected as pilot TMDL projects for mercury.

Scientists studying the Florida Everglades have estimated that at least half of the mercury deposited in the Everglades is emitted locally, while between 5% and 29% is emitted regionally (from within the southeastern United States). The remainder derives from sources outside the United States.[36] EPA has estimated that 80% of deposition to Pines Lakes, New Jersey, comes from U.S. sources.[37] In contrast, almost all of the mercury found in remote regions of the Arctic is believed to have traveled from distant sources.[38]

Methylmercury Formation and Accumulation. The most biologically significant transformation of mercury occurs in soil or sediments of lakes or streams, where bacteria (primarily sulfate-reducing bacteria) are capable of converting inorganic mercury to methylmercury.[39] The significance of methylation is that relative to inorganic mercury, methylmercury is more easily absorbed by living tissues, more likely to be ingested in food, and much more toxic to animals. Methylmercury is easily absorbed by the digestive tract and accumulates in the bodies of fish and other animals, when it is ingested faster than it can be excreted. Because methylmercury tends to be stored in muscle tissue (i.e., the edible meat of fish and other animals), animals higher on the food chain tend to have higher levels of exposure. Predatory fish (e.g., walleye, large-mouthed bass, or tuna), fish-eating birds (e.g., loons, ospreys, or eagles), and fish-eating mammals (e.g., raccoons, otters, or mink) which top the longest food chains accumulate the greatest concentrations of methylmercury. In the Florida Everglades, methylmercury concentrations in fish are up to ten million times greater than concentrations of mercury in water.[40] Inorganic mercury is not easily transferred through the food chain and does not concentrate to higher levels with each nutritional link.

Generally, the more mercury that is added to an ecosystem, through direct discharge to water, runoff from the surrounding watershed, or deposition from air, the more mercury that will be found in fish.[41] However, the rate of methylmercury formation and accumulation is highly variable, even within relatively small geographic areas, because it depends on many factors, in addition to the abundance of inorganic mercury. Recent research indicates that some ecosystems are particularly sensitive to relatively small mercury inputs, and are more likely to

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[36] *Everglades Consolidated Report*, p. 2B-11.

[37] Seigneur et al.

[38] Krabbenhoft statement.

[39] There are several processes by which mercury can become methylated and demethylated. The role of sulfate-reducing bacteria generally is thought to be the most important, however. J. G. Wiener, D. P. Krabbenhoft, G. H. Heinz, et al., "Ecotoxicology of mercury," in D. J. Hoffman, B. A. Rattner, G. A. Burton, Jr., et al. (eds.), *Handbook of Ecotoxicology*, 2nd ed. (Boca Raton, FL: Lewis Publishers, 2003), pp. 420-421.

[40] *Everglades Consolidated Report*, p. 2B-16.

[41] Ibid., p. 2B-2.

experience high rates of methylmercury production and accumulation. Sensitive ecosystems include low-alkalinity (i.e., low capacity for neutralizing acid) and humic lakes and streams (which are characterized by an abundance of dissolved, decomposed, plant or bacterial matter), wetlands, surface waters connected to wetlands, and waters linked to areas subjected to flooding.[42] Methylmercury formation by sulfate-reducing bacteria and bioaccumulation is favored in ecosystems:

- that are oxygen-poor and acidic;
- that contain sulfate (the most common form of sulfur in surface waters), but not too much sulfide (the form of sulfur rendered by sulfate-reducing bacteria;[43] and
- in which mercury is recently deposited, rather than older mercury.[44]

In a Wisconsin lake, researchers found that levels of both sulfate and mercury determined levels of production and bioaccumulation of methylmercury, and that "modest changes in acid rain or mercury deposition can significantly affect mercury bioaccumulation over short-time scales."[45] In response to a significant decrease in mercury deposition between 1994 and 2000, methylmercury in yellow perch decreased by roughly 30% (5% per year).

The link between industrial emissions and mercury levels in the oceans is less clear, because the role of the oceans in mercury cycling is poorly understood. On the one hand, significant quantities of reactive inorganic mercury are deposited in the oceans, and methylmercury is found in marine fish and their predators, sometimes at very high concentrations. And, although methylmercury levels are very low in the surface layer of the open oceans, concentrations are greater, perhaps as much as three-fold higher than they were prior to industrialization (assuming that insignificant amounts descended to the ocean depths).[46] So we know that organic (methyl) mercury is formed in the oceans. What we do not know is where the mercury in ocean fish originated — in industrial emissions deposited to the oceans or in the natural reservoir of the ocean depths — nor where it was transformed into methylmercury.

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[42]Wiener et al., p. 440.

[43]*Everglades Consolidated Report*, pp. 2B-11-12, 16-18; J. M. Benoit, C. C. Gilmour, and R. P. Mason, "Sulfide controls on mercury speciation and bioavailability to methylating bacteria in sediment pore waters," *Environmental Science & Technology*, v. 33, n. 6 (1999), pp. 951-957.

[44]*Everglades Consolidated Report*, appendix 2B-3, p. 2; H. Hintelmann, R. Harris, A. Heyes, et al., "Reactivity and mobility of new and old mercury deposition in a boreal forest ecosystem during the first year of the METAALICUS study, *Environmental Science & Technology*, v. 36, n. 23 (2002), pp. 5034-5040.

[45]T. R. Hrabik and C. J. Watras, "Recent declines in mercury concentration in a freshwater fishery: Isolating the effects of de-acidification and decreased atmospheric mercury deposition in Little Rock Lake," *The Science of the Total Environment*, v. 297 (2002), pp. 229-237.

[46]Robert Mason, personal communication, April 1, 2004.

Some scientists believe that methylmercury probably is formed in the deep sediments of oceans or in the areas surrounding deep thermal vents in the ocean floor.[47] In that case, they argue, deposition of atmospheric mercury cannot account for current methylmercury levels in ocean fish, given the relatively large size of the deep sea reservoir of mercury and the time it takes for the ocean depths to mix with the surface layers where fish feed, an estimated 400 years. If all the mercury deposited into the oceans due to human activities over the past hundred years were mixed into the ocean even to its greatest depths, the mercury concentration of ocean water would have increased only an estimated 1% to 10% over pre-industrial concentrations.

Other scientists believe that sulfate-reducing bacteria form methylmercury in coastal sediments where it is taken up by tiny plants and animals at the bottom of aquatic food webs. Small fish and other animals feeding in near-shore waters concentrate the mercury, then venture far enough from shore to be prey for larger fish, seabirds, and mammals.[48]

At this time, not enough information is available to determine whether mercury levels in ocean fish and fish-eating marine mammals have increased or decreased over the past hundred years or so, much less whether levels rose and declined as a result of changes in atmospheric emissions.[49] Although most scientists who study mercury agree that deposition of atmospheric mercury has increased, and therefore the total amount of mercury in the oceans probably has increased, particularly in the surface layer, and one study (described below) has found increased mercury levels in feathers of fish-eating seabirds,[50] measurements of mercury in ocean water and fish are lacking or inconclusive. In part, this lack of data is due to the difficulty of measuring mercury: measurement of methylmercury has only been possible since about 1985, and past measurements of total mercury often were inaccurate because samples were so easily contaminated.

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[47]A. M. L. Kraepiel, K. Keller, H. B. Chin, et al., "Sources and variations of mercury in tuna," *Environmental Science & Technology*, v. 37, n. 24 (2003), p. 5551-5558.

[48]J. K. King, J. E. Kostka, M. E. Frischer, et al., "A quantitative relationship that demonstrates mercury methylation rates in marine sediments are based on the community composition and activity of sulfate-reducing bacteria," *Environmental Science & Technology*, v. 35, n. 12 (2001), pp. 2491-2496.

[49]A famous 1972 study by G. E. Miller et al. (*Science*, v. 175 (1972), pp. 1121-1122) purported to lend support to the contention that mercury in ocean fish derived from natural sources. However, because Miller reported the study in a letter to the editor of *Science*, it was not peer-reviewed, and is an insufficient basis for drawing any conclusions: A single swordfish head preserved in 1946 was the source of the "historical" data for swordfish; while five skipjack tuna, one bluefin tuna, and one albacore tuna, all less than two-thirds meter long, originating from two different oceans, served as the historical reference specimens for tuna. The modern specimens were one fresh albacore tuna, one fresh skipjack tuna, three cans of albacore tuna, and six fresh swordfish. No information was provided about the age or length of these specimens.

[50]L. R. Monteiro and R. W. Furness, "Accelerated increase in mercury contamination in North Atlantic mesopelagic food chains as indicated by time series of seabird feathers," *Environmental Toxicology and Chemistry*, v. 16, n. 12 (1997), pp. 2489-2493.

A recent study that compared total mercury concentrations in yellowfin tuna captured in 1971 with methylmercury in yellowfin tuna caught in 1998, both in the vicinity of Hawaii, found no significant differences in mercury concentrations.[51] However, the significance of these measurements is unclear, given the historical trend in atmospheric deposition, which peaked in the mid 1980s.

Another study compared feathers over time from two kinds of fish-eating birds that live in the northern Atlantic Ocean.[52] Feathers were obtained from museum specimens taken as long ago as 1885. The study found a significant increase in concentrations of methylmercury over time. Among birds that eat fish living near the ocean surface, concentrations of methylmercury in feathers increased at an estimated rate of 1.1% annually between 1885 and 1994. According to study authors, this increase is consistent with the estimated three-fold increases in concentrations of mercury in the atmosphere and surface oceans due to human industry over the same period of time. Among birds that eat fish living in a deeper, darker ocean layer, methylmercury concentrations increased at an estimated rate of 3.5 to 4.8% per year.[53]

Risks of Methylmercury Poisoning

Toxicity of Methylmercury. Methylmercury is highly toxic to the central nervous system of humans and many animals. The observed effects of toxic levels of exposure generally have been similar in laboratory animals, domestic pets, wildlife, and people. Typically, there is a lag time of weeks or even months between exposure to mercury and the onset of health effects.[54]

In human adults, absorbed methylmercury is dispersed throughout the body in blood and enters the brain, where it may cause structural damage. The physical lesions may lead to tingling and numbness in fingers and toes, loss of coordination, difficulty in walking, generalized weakness, impairment of hearing and vision, tremor, and finally loss of consciousness and death. At high levels of exposure, effects on the brain are easily observed and irreversible. Damage to the brain may exist, however, in the absence of these observable symptoms of toxicity.[55] Nervous system damage (indicated by tingling and/or numbness in the fingers and toes) has been estimated to occur in about 5 % of adults whose hair is found to contain 50 parts of methylmercury per million parts of hair (ppm).[56] This condition is predictive of

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[51]A. M. L. Kraepiel, K. Keller, H. B. Chin, et al., "Sources and variations of mercury in tuna," *Environmental Science & Technology*, v. 37, n. 24 (2003), pp. 5551-5558.

[52]Monteiro and Furness.

[53]Ibid.

[54]T. W. Clarkson, "The three modern faces of mercury," *Environmental Health Perspectives*, v. 110 (2002), supp. 1, p. 11-23.

[55]National Research Council, *Toxicological Effects of Methylmercury* (Wahington: National Academy Press, 2000), 344 pp.

[56]World Health Organization, *Environmental Health Criteria 101: Methylmercury* (1990).

more severe toxicity. Lower levels of exposure may have more subtle adverse impacts on coordination, ability to concentrate, and thought processes.[57]

Methylmercury readily crosses the placenta of pregnant women.[58] Levels of methylmercury in the fetal brain are roughly five to seven times the levels in maternal blood.[59] Compared to the adult brain, the fetal brain is more sensitive to methylmercury. In the fetus, methylmercury exposure can affect brain development, as evidenced during childhood by a child's ability to learn and function normally after birth.[60] Human poisoning incidents in Iraq and Japan caused severely exposed children to be born with cerebral palsy and mental retardation, and in a few cases infants died. In Japan, poisoning occurred because local fish were poisoned by industrial mercury releases to Minamata Bay. The average mercury content of fish samples there ranged from 9 to 24 ppm. Recent research indicates that exposure to much lower levels of methylmercury also leads to developmental effects on cognitive development.[61]

There is general agreement that as little as 10 ppm methylmercury in maternal hair indicates a level of exposure that may produce prenatal effects.[62] Some believe effects occur at even lower exposure levels. At very low levels of exposure, effects may be very subtle, and detectable only on a population basis — for example, by an increase in the proportion of an exposed population that falls below a level of function defined as impaired.

In response to a mandate from the U.S. Congress, EPA contracted with the National Research Council (NRC) to review available research on methylmercury toxicity. The NRC Committee issued a report in 2000.[63] It concluded that scientific studies have demonstrated the sensitivity of the human fetus to pre-natal methylmercury exposure, and that the risk to women who eat large amounts of fish and seafood during pregnancy is "likely to be sufficient to result in an increase in the number of children who have to struggle to keep up in school."[64]

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[57]E. M. Yokoo, J. G. Valente, L Grattan, et al., "Low level methylmercury exposure affects neuropsychological function in adults," *Environmental Health: A Global Access Science Source*, v. 2, n. 1 (2003), pp. 8-19, at [http://www.ehjournal.net/content/2/1/8], visited April 19, 2004.

[58]Wolfe, Schwarzbach, and Sulaiman, p. 149.

[59]E. Cernichiari, R. Brewer, G. J. Myers, et al., "Monitoring methylmercury during pregnancy: maternal hair predicts fetal brain exposure," *Neurotoxicology*, v. 16, n. 4 (1995), pp. 705-710.

[60]EPA, *Mercury Study Report to Congress*, vol. 1, pp. 3-23.

[61]P. Grandjean, P. Weihe, R. F. White, et al., "Cognitive deficit in seven-year-old children with prenatal exposure to methylmercury," *Neurotoxicology and Teratology*, v. 19, n. 4 (1997), p. 417.

[62]Clarkson.

[63]National Research Council, *Toxicological Effects of Methylmercury*.

[64]Ibid. p. 325.

A study published in 2003 strengthened and extended the findings of the single major study of children which failed to find any adverse effects in children exposed to mercury before they were born.[65] However, one NRC Committee member testified before a House subcommittee in November 2003 that although those findings had not been published at the time, they only confirmed results already considered and would not have led to a different Committee conclusion.[66] This conclusion has since been confirmed in a peer-reviewed publication by four members of the original NRC committee.[67]

Human sensitivity to cardiovascular toxicity might be even greater than to developmental neurotoxicity, given recent research results. For example, a study of 1,833 Finnish men found that those who had at least 2 ppm of mercury in hair had twice the risk of acute myocardial infarction compared to men with less mercury in hair.[68] (Two ppm of methylmercury roughly corresponds to the upper 10th percentile of current methylmercury exposure among adult men in the United States.)

A follow-up study of the Finnish men also looked at levels of fish-derived fatty acids. Results suggested that the adverse effect of mercury exposure resulted from its interference with the protective effect of fatty acids in the fish. Men who ate fish appeared to benefit from a protective effect of the acids against heart disease, but among those with more than 2 ppm mercury in their hair the protective effect was reduced by half.[69] Other studies generally are consistent with these results, but one major study failed to find an association between total mercury exposure (measured in toenail clippings) and cardiovascular disease.[70] More research is needed to explore interactions among the various risk factors, fish-derived fatty acids, and mercury exposure with respect to heart disease.

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[65]G. J. Myers, P. W. Davidson, C. Cox, et al, "Prenatal methylmercury exposure from ocean fish consumption in the Seychelles child development study," *Lancet*, v. 361 (2003), pp. 1686-1692.

[66]U.S. House of Representatives, Committee on Science, Subcommittee on Environment, Technology, and Standards, *Mercury Emissions: State of the Science and Technology*, hearing, Nov. 5, 2003, statement of Thomas A. Burke, at [http://www.house.gov/science/hearings/ets03/nov05/burke.htm], visited Apr. 14, 2004.

[67]A. H. Stern, J. L. Jacobson, L. Ryan, et al., "Do recent data from the Seychelles Islands alter the conclusions of the NRC report on the toxicological effects of methylmercury?" Commentary, *Environmental Health: A Global Access Science Source*, v. 3, n. 1 (2004), pp. 2-5, at [http://www.ehjournal.net/content/3/1/2], visited April 19, 2004.

[68]J. T. Salonen, K. Saponin, K. Nyyssonen, et al., "Intake of mercury from fish, lipid peroxidation, and the risk of myocardial infarction and coronary, cardiovascular, and any death in Eastern Finnish men," *Circulation*, v. 91, n. 3 (1995), pp. 645-655.

[69]T. Rissanen, S. Voutilainen, K. Nyyssonen, et al., "Fish oil-derived fatty acids, docosahexaenoic acid and docosapentaenoic acid, and the risk of acute coronary events: the Kuopio ischaemic heart disease risk factor study," *Circulation*, v. 102, n. 22 (2000), pp. 2677-2679.

[70]K. Yoshizawa, E. B. Rimm, J. S. Morris, et al., "Mercury and the risk of coronary heart disease in men," *New England Journal of Medicine*, v. 347, n. 22 (2002), pp. 1755-1760.

Environmental Methylmercury Exposure. People may be exposed to mercury by eating or drinking, inhaling, or simply absorbing it through their skin. The level of recent (within a month or two) individual exposure to mercury may be determined based on measured concentrations of mercury in blood. For a slightly longer exposure history (e.g., over several months), mercury concentrations in human hair several inches from the scalp may be useful. However, there is no way to measure exposure that occurred more than a few years ago, because methylmercury breaks down in the bodies of animals, and both organic and inorganic mercury are excreted over time.

Although rates of physiological processes vary widely among individuals, in general, people eliminate about half the mercury taken in within a period of roughly 44-80 days.[71] In this way, mercury differs from many other pollutants such as lead, which may be measured in the bone or teeth years after exposure has ceased. If mercury exposure ends (because mercury is excreted) before a toxic amount of mercury has accumulated in the body, adverse health effects would not be expected to occur. However, effects would not necessarily subside after excretion, if a toxic level of exposure had occurred.

The 1999-2002 National Health and Nutrition Examination Survey (NHANES) collected data on blood mercury levels for a representative sample of U.S. women of child-bearing age. The results for the first two years (1999-2000) are summarized in **Table 2**. Because mercury is present in much lower levels in blood than in tissues such as hair, concentrations are expressed as parts of mercury per billion parts blood (ppb), by weight. Based on these data, the Centers for Disease Control and Prevention (CDC) concluded that mercury concentrations generally were low among women of child-bearing age and children in the U.S. population. These results were confirmed by data collected in 2001-2002.[72] However, study authors noted that the survey was designed to gather baseline data, and that there were too few people interviewed to provide reliable estimates of blood mercury levels for individuals at the highest levels of exposure.[73]

In the United States, most people are exposed to mercury primarily through eating the flesh (muscle) of fish.[74] People who eat a lot of predatory fish, such as bass, pike, tuna, or swordfish, which may be highly contaminated, may increase the risk of adverse health effects for themselves or, in the case of women who become pregnant, for any unborn children.[75] Thus, NHANES 1999-2000 found that women who ate three or more servings of fish within a month had almost four times the level of mercury in

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[71]EPA, *Mercury Study Report to Congress*, vol. 1, pp. 3-23.

[72]Centers for Disease Prevention and Control. MMWR Weekly. Nov. 5, 2004, v. 53, n. 43, p. 1018-1020.

[73]S. E. Schober, T. H. Sinks, R. L. Jones, et al., "Blood mercury levels in U.S. children and women of childbearing age, 1999-2000," *JAMA*, v. 289, n. 13 (2003), pp. 1667-1674.

[74]Note that mercury is stored in muscle rather than skin, fat, or bone, and so it cannot be avoided by removing those parts before eating.

[75]J. M. Hightower and D. Moore, "Mercury levels in high-end consumers of fish," *Environmental Health Perspectives*, v. 111, n. 4 (2003), pp. 604-608.

their blood as women who ate no fish that month.[76] Nevertheless, 95% of the 448 women who ate fish relatively frequently (at least three times during the previous 30 days) had blood mercury levels less than about 11 ppb. About 25% of the study population ate no fish or shellfish at all.[77] Generally, their blood contained levels of mercury that were below 2 ppb.

Table 2. Geometric Mean and Selected Percentiles of Total Blood Mercury Concentrations (ppb) for U.S. Children Aged 1-5 Years and Women Aged 16-49 Years

Group	Geometric Mean	Selected percentiles					
		10th	25th	50th	75th	90th	95th
Children	0.3	< 0.14	< 0.14	0.2	0.5	1.4	2.3
Women	1.2	0.2	0.5	1.2	2.7	6.2	7.1

Source: 1999-2000 National Health and Nutrition Examination Survey.[78]

The amount of mercury in fish varies with the species, age, and size of the fish. Uncontaminated fish contain less than 0.01 ppm methylmercury in muscle, while very contaminated swordfish in U.S. waters have more than 3 ppm mercury.[79] (Grossly contaminated fish in Minamata Bay, Japan, contained between 9 and 24 ppm mercury.)[80] Even higher levels have been found where there is a local source of water pollution. Diverse species of fish differ in sensitivity to mercury. Significant toxic effects and death are associated in adult fish of various species with between 6 ppm (e.g., for walleyes) and 20 ppm (e.g., for salmon) in muscle tissue. However, individual fish within species also differ in sensitivity, and fish seem able to tolerate higher concentrations of mercury if it is accumulated slowly.[81] In general, older, larger fish of the same species will have more mercury. **Table 3** provides the average concentration found in recent years in selected species popular with American consumers. Concentrations are given in parts of mercury per million parts of fish (ppm). Freshwater fish are in italic type. Methylmercury levels in particular species of fish are highly variable, however, reflecting the chemistry and methylation potential of the bodies of water in which they live.

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[76]S. E. Schober, T. H. Sinks, R. L. Jones, et al.

[77]Ibid.

[78]Ibid; K. R. Mahaffey, R. P. Clickner, and C. C. Bodurow, "Blood organic mercury and dietary mercury intake: National Health and Nutrition Examination Survey, 1999 and 2000," *Environmental Health Perspectives*, v. 112, n. 5 (2004), p. 568.

[79]FDA, Mercury Levels in Commercial Fish and Shellfish, at [http://www.cfsan.fda.gov/~frf/sea-mehg.html].

[80]FDA, "Mercury In Fish: Cause for Concern?" *Consumer Magazine*, Sept. 1994, as updated May 1995.

[81]Wiener et al., p. 427.

Table 3. Mercury Concentrations in Some Popular Fish (ppm)

Species	Average Mercury Level (ppm)
Shrimp	<0.01
Tuna, white, canned (solid and chunk albacore)	0.36
Tuna, light, canned (chunk)	0.12
Salmon	0.01
Pollock	0.06
Catfish	0.05
Cod	0.11
Crab (blue, king, snow)	0.06
Clams	<0.01
Tilapia	0.01
Flatfish (flounder, sole, plaice)	0.05
Scallops	0.05
Halibut	0.26
Perch	0.14
Swordfish	0.97

Sources: FDA websites Mercury Levels in Commercial Fish and Shellfish, at [http://www.cfsan.fda.gov/~frf/sea-mehg.html] and Mercury in Fish: FDA Monitoring Program (1990-2003), at [http://www.cfsan.fda.gov/~frf/seamehg2.html].

Note: *Italics* indicate freshwater fish. All other fish are marine.

Recommended Exposure Limits. A key question for Congress is whether there is currently a potential for adverse health effects among individuals who regularly consume fish. Federal agencies have estimated the risk associated with methylmercury exposure at current levels of environmental (i.e., fish) contamination. Of particular relevance is the reference dose (RfD) set by EPA, which is discussed in some detail below. Because there has been some controversy surrounding the EPA RfD, it is compared to two other maximum allowable concentration levels established by federal agencies, the minimum risk level (MRL) set by the Agency for Toxic Substances and Disease Registry, and the Acceptable Daily Intake (ADI) level established by the Food and Drug Administration. As explained below, the apparent inconsistency among the FDA, ATSDR, and EPA estimates of a "safe" exposure level for methylmercury is primarily due to the agencies' diverse responsibilities and actions that are triggered when contamination is found to occur.

EPA Reference Dose for Methylmercury. The EPA Reference Dose (RfD) is a risk assessment tool, used to estimate daily intake levels of chemicals that are expected to be "without an appreciable risk of deleterious health effects,"[82] even if exposure persists over a lifetime. The risk associated with exposure to methylmercury above the RfD is uncertain, but likely to increase with increasing exposure levels. The RfD is intended to account for sensitive members of the human population, such as pregnant women and infants, but not individuals with unusual sensitivity due to conditions such as genetic disorders or severe illness. To calculate the RfD, EPA generally uses a "no observed adverse effect level" (NOAEL), which may be observed or estimated using a model. A NOAEL estimates the threshold level of exposure below which adverse effects do not occur. Then the RfD is established by dividing the NOAEL by uncertainty factors which account for the need to extrapolate from limited data sets to the general U.S. population.

In 1985, EPA established its first RfD for people who eat methylmercury-contaminated fish at 0.3 micrograms of methylmercury (μg) per kilogram of body weight (kg_{bw}) per day. This is equivalent to about 126 μg of methylmercury per week (roughly the amount in two 7-ounce servings of fish containing 0.3 ppm mercury) for a person weighing 132 pounds. This dose is based on the lowest level of exposure that produced adverse effects on the nervous systems (i.e., numbness and tingling in the extremities) of adult Iraqis after they were poisoned by eating contaminated grain during 1971-1972 and adult Japanese who ate contaminated fish from Minamata Bay during the mid-1950s.

Two years after EPA set its RfD, data were published showing adverse effects of maternal mercury exposure on the development of Iraqi children who were exposed in the womb. In 1995, EPA revised its RfD, basing it on these developmental effects. This second RfD of 0.1 $\mu g/kg_{bw}$/day (42 μg per week for a person weighing 132 pounds) remains in effect. This level would be exceeded if a 132-pound person ate more than one fish meal per week, and the fish contained more than 0.21 ppm of mercury.

To calculate the current RfD, EPA used a benchmark dose approach. The benchmark dose for methylmercury estimates the level of exposure that has a 5% chance of doubling the number of children (from 5% to 10% of the exposed population) who function at an abnormally low level on a standardized measure. In 1997, the benchmark dose calculated was 11 parts methylmercury per million parts maternal hair (ppm), by weight, based on all the adverse health effects observed in Iraqi children who were exposed to methylmercury before birth. The findings of other human studies as well as toxicity data collected from animals in scientific laboratories, supported the validity of the EPA calculated benchmark dose.[83] Benchmark doses calculated based on data from studies of island populations with heavy seafood consumption produced similar values (11 to 17 ppm).[84]

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[82]EPA Fact Sheet, *Mercury Update: Impact on Fish Advisories*, June 2001, at [http://www.epa.gov/ost/fishadvice/mercupd.pdf].

[83]EPA, *Mercury Study Report to Congress*, vol. 1, pp. 3-26.

[84]Lynn R. Goldman and William H. Farland, letter, *Science*, v. 279, n. 5351, pp. 640-641.

EPA used the benchmark dose to conclude that consumption of 1.1 $\mu g/kg_{bw}$/day of methylmercury probably was safe for the unborn children of women who ate contaminated grain in Iraq. At this level of mercury intake, Iraqi women who weighed an average of 60 kg (about 132 pounds) had about 11 ppm mercury in maternal hair and 44 µg methylmercury per liter of blood. (However, individual ratios of hair to blood concentrations varied widely.) EPA divided that daily dose (1.1 $\mu g/kg_{bw}$/day) by an uncertainty factor of 10, accounting for the lack of data on reproductive effects and differences among individuals, to establish the RfD at 0.1 $\mu g/kg_{bw}$/day. At this level of exposure, a mercury concentration of approximately 4 to 5 parts mercury per billion parts blood (ppb), by weight, and 1 part mercury per million parts of hair (ppm), by weight, would be expected to accumulate in an adult.

According to EPA's independent advisory group, the Science Advisory Board (SAB), the1997 EPA RfD was strongly supported by multiple studies based on different ethnic populations and species, exposures, and developmental endpoints, all suggesting similar RfDs.[85] However, the SAB advised EPA to consider an additional uncertainty factor to account for the need to extrapolate from the observed effects of an acute, short-term exposure to effects that might result from low-level, life-long exposure; the difficulty of detecting subtle population effects; and evidence from animal and human studies suggesting possible neurological degeneration in the elderly and high mercury exposure of the fetus compared to the mother's exposure.

Soon after the results of long-term studies were published, the NRC recommended that EPA base its RfD on a evidence of chronic toxicity among island dwellers who were exposed to methylmercury through fish and other seafood.[86] The NRC panel concluded in its 2000 report that there is a 5% chance that maternal exposure to1.0 $\mu g/kg_{bw}$/day of methylmercury would double the proportion of children functioning at an abnormally low level. Mothers eating that amount of mercury (in contaminated fish), on average, would have about 12 ppm methylmercury in their hair (and 58 ppb in their blood); fetuses would be exposed to about 58 ppb in cord blood. Recent analyses indicate that these numbers may need to be revised to incorporate research results indicating that the relationship between cord blood and maternal mercury intake is highly variable.

Based on the NRC report, EPA revised the RfD for methylmercury. The value of the RfD did not change from 0.1 $\mu g/kg_{bw}$/day, but the basis for the RfD was updated using the most current data and analyses. This RfD is considered to be protective of all populations in the United States, including sensitive subpopulations. Based on that RfD, pursuant to section 304(a)(1) of the Clean Water Act, EPA established in 2001 a water quality criterion for methylmercury of 0.3 parts of methylmercury per one million parts of fish tissue (ppm). (This is the first time that EPA based a water quality criterion on a concentration of a pollutant in fish rather than in the water column.) EPA indicated that to protect consumers of fish and shellfish among the general population, this concentration of methylmercury in fish and shellfish tissue should not be exceeded.

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[85]Science Advisory Board, *An SAB Report: Review of the EPA Draft Mercury Study Report to Congress*, EPA-SAB-EC-98-001, Oct. 1997, p. 91.

[86]National Research Council, *Toxicological Effects of Methylmercury*, p. 325.

Agency for Toxic Substances and Disease Registry Minimum Risk Level. The Agency for Toxic Substances and Disease Registry (ATSDR), a branch of the Public Health Service, has health-related authority under the Comprehensive Emergency Response, Compensation, and Liability Act (CERCLA, better known as Superfund). One of the agency's responsibilities is to study hazardous substances found at sites on the national priority list (NPL) and to publish and periodically update toxicological profiles of those most frequently found. In revising the toxicological profile for mercury, ATSDR evaluated available data and concluded in 1999 that they supported a Minimum Risk Level (MRL) for chronic exposure to methylmercury of 0.3 $\mu g/kg_{bw}$/day. (This is the same as EPA's 1985 RfD.) ATSDR uses the MRL as a screening tool to determine when the risks posed by a hazardous waste site require additional study.

Food and Drug Administration Action Level. The Food and Drug Administration (FDA) established an action level in 1984 at a concentration of 1 ppm methylmercury in fish or seafood products sold through interstate commerce. At this level, the Acceptable Daily Intake for an adult in the general population is 0.42 $\mu g/kg_{bw}$/day, slightly higher than 0.3 $\mu g/kg_{bw}$/day, the RfD established by EPA in 1985. The FDA action level is based on the mid-point of the estimated range of the "lowest observed adverse effects level" (LOAEL), or 300 μg of methylmercury/day, at which level of exposure Japanese adults who ate contaminated fish experienced paresthesia (numbness and tingling in extremities). FDA divided this value by 10 to account for scientific uncertainties and to provide a margin of safety. FDA chose not to use the Iraqi data on the effects of fetal exposure as a basis for revising its action level, due to concerns about uncertainties (in contrast to the relative certainty of the health benefits of consuming fish.) The FDA action level is enforceable; the Administration may seize interstate shipments of fish and shellfish containing more than 1 ppm of methylmercury, and may seize treated seed grain containing more than 1 ppm of mercury.[87] For the purpose of advising the general public about fish consumption, FDA has used EPA's RfD, recommending that women of child-bearing age avoid certain fish and limit consumption of other fish.[88]

The inconsistency among the FDA, ATSDR, and EPA estimates of a "safe" exposure level for methylmercury is more apparent than real: the differences are less than the uncertainty factor, and the reference levels serve different purposes. In addition, the EPA number assumes a lifetime of exposure, while the ATSDR level is for chronic exposure of 365 days or longer, and the FDA level is for consumption of particular fish.

Table 4 consolidates the quantitative information provided above to facilitate comparisons among agencies.

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[87]Mercury formerly was used as a pesticide to treat seed grain. However, the last mercury-based pesticides registered for use in the United States (to control mold) were voluntarily canceled by the manufacturer in November 1993.

[88]EPA/FDA Joint Federal Advisory for Mercury in Fish, at [http://www.epa.gov/ost/fishadvice/advice.html], visited Apr. 26, 2004.

U.S. Fish Consumption, Methylmercury Exposure, and Health Risk. By comparing methylmercury concentrations for popular fish (**Table 3**) with federal guidelines (**Table 4**), it is possible to assess the relative safety of eating different fish and shellfish.[89] **Table 5** provides estimates of the numbers of meals of fish with different average levels of contamination that one could eat without increasing methylmercury exposure beyond the EPA RfD. It is important to note, however, that these recommendations assume that the size of meals, the age and size of particular fish, and the age and size of the consumer are "average." Generally, if other factors are held constant, risks of poisoning increase to the extent that consumers are younger or smaller than average, eat larger amounts, or eat older and larger fish (and risks decrease if the reverse is true). For example, a fish lover who consumed one 7-ounce meal of freshwater fish (roughly 200 grams) containing 0.3 ppm of methylmercury (the level permitted by the EPA water quality criterion) seven days in a row could be exposed to ten times the level of EPA's RfD, a level equal to the benchmark dose level. But, because different fish contain different levels of methylmercury, daily consumption of 7 ounces of fish could result in much lower or much higher levels of methylmercury exposure, depending on the types of fish consumed.

Table 4. Federal Upper Limits for Methylmercury Exposure

Agency	Level of Daily Exposure	Level in Fish
EPA	0.1 $\mu g/kg_{bw}$/day	0.3 ppm
ATSDR	0.3 $\mu g/kg_{bw}$/day	—
FDA	0.42 $\mu g/kg_{bw}$/day	1 ppm

Table 5. Recommended Number of Meals per Month of Fish Containing Various Methylmercury Concentrations, Based on EPA RfD[90]

Methylmercury Level in Fish (ppm)	Allowable Meals per Month (8 ounce or 232 grams)
0.1	9
0.2	4.5
0.3	3
0.5	1.8
0.9	1

[89]Guides to fish consumption may be found on the Internet. For example, see the one produced by the state of Maine at [http://www.epa.gov/waterscience/fish/forum/2004/presentations/sunday/frohmberg.pdf].

[90]*Methylmercury: EPA Update 06/02*, presentation by Rita Schoeny, Mercury Conference.

Average U.S. fish consumption is 7-14 ounces (200-400 grams) *per month*, according to EPA, when those who do not eat fish are included.[91] On average, that level of fish consumption would expose fish eaters to 4 µg of mercury per day, a level below the RfD for anyone weighing more than 88 pounds (40 kilograms). Fish consumption rates in the United States are estimated annually by the National Marine Fisheries Service (NMFS). Rates are estimated based on total fish and shellfish in commerce (edible weight) divided by the total population in the middle of the census period. No adjustments are made for waste or spoilage of the fish or for people who do not eat fish. Sport-caught fish are not included. For 2002, NMFS estimated per person consumption at 15.6 pounds of fish.[92] Of this quantity, 11 pounds were fresh or frozen, including 6 pounds of finfish and 5 of shellfish. Cured fish accounted for 0.3 pounds and canned fish for 4.3 pounds per capita. Seventy-seven percent of the fish consumed was imported.

Consumption rate estimates are higher when only those who eat fish are considered. Unfortunately, data are limited. In the *Mercury Study Report to Congress*, EPA estimated that:

> 85% of adults in the United States consume fish and shellfish at least once a month with about 40% of adults selecting fish and shellfish as part of their diets at least once a week (based on food frequency data collected among more than 19,000 adult respondents in the NHANES III conducted between 1988 and 1994). This same survey identified 1-2% of adults who indicated they consume fish and shellfish almost daily.[93]

Data from NHANES 1999-2002 indicates that exposure to methylmercury is greater than the RfD for approximately 6% of women of child-bearing age.[94] This percentage is based on four years of data; it is lower than was found by NHANES in the first two-year reporting period,1999-2000. However, a declining trend should not be inferred, because the difference is not statistically significant. At least two more years of data are needed to determine whether the apparent decline in blood mercury levels is a real trend.

Data for certain areas of the California coast indicate that although half of all consumers surveyed ate 21 grams per day or less, 5% of consumers ate more than 161 grams per day (more than 10 pounds per month) of fish that consumers caught themselves.[95] At 0.3 ppm methylmercury, such consumers would be taking in about 48 µg per day of methylmercury, an amount close to the benchmark dose. Similarly, a 1988 study of Michigan anglers who eat the fish they catch found that they ate *on*

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[91]EPA, *Mercury Study Report to Congress*, vol. 1, pp. 3-23.

[92]National Marine Fisheries Service, *Fisheries of the United States — 2002*, at [http://www.st.nmfs.gov/st1/fus/current/09_percapita2002.pdf], visited Apr. 27, 2004.

[93]EPA, *Mercury Study Report to Congress*, vol. 4, p. ES-2.

[94]Centers for Disease Prevention and Control, *MMWR Weekly*, Nov. 5, 2004, v. 53, n. 43, pp. 1018-1020.

[95]Office of Environmental Health Hazard Assessment, *Chemicals in Fish: Consumption of Fish and Shellfish in California and the United States*, Final Report, Pesticide and Environmental Toxicology Section, Office of Environmental Health Hazard Assessment, California Environmental Protection Agency, Oakland, CA, 2001, p. 92.

average 45 grams of freshwater fish per day, but 5% of those surveyed ate 98 grams per day.[96] That amounts to 1.5 pounds of fish per week per person, much more than is recommended for contaminated species of fish, but not an implausibly large amount. **Table 6** illustrates the general relationship between plausible levels of fish consumption and methylmercury exposure for various segments of the U.S. population, assuming that fish contain methylmercury at the level of the water quality criterion established by EPA.

Table 6. Various Estimates of Fish Consumption and Mercury Exposure, Assuming a Concentration in Fish of 0.3 ppm

Population	Mean Monthly Consumption Rate (ounces)	Methylmercury Exposure per Day (μg/day)[97]
Per Capita U.S. (All)	21	6
Average U.S. Fish Consumer	7 - 14	2 - 4
Average MI Angler/ Consumer	48	14
Upper 5% CA Sport Fish Angler/Consumer	170	49

In making choices about fish consumption, factors other than, or in addition to, methylmercury concentration should be considered. In particular, the health benefits of eating fish high in omega fatty acids are important, for cardiovascular health and fetal development in particular. **Table 7** provides relative fatty acid content of some popular fish.

Wildlife Exposure and Health Effects

Fish consumption also is the dominant pathway for wildlife exposure to methylmercury. Fish-eating predators in North America generally have relatively high concentrations of mercury. Toxic mercury levels have been found in individual mink, otters, loons, the Florida panther, and other U.S. birds and wildlife. However, it is not clear whether *typical* levels of environmental contamination are stressful for wildlife.

Fish-eating birds annually eliminate much of their accumulated methylmercury when they form new feathers. Moreover, seabirds seem to be able to demethylate methylmercury, rendering it less toxic. Nevertheless, methylmercury exposure may harm sensitive species at levels found in certain local environments. Many scientists suspect that the immune system is weakened as a result of methylmercury exposure. The most likely adverse impact on birds of methylmercury exposure is impaired ability to reproduce.[98]

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[96]Ibid.

[97]Assumes chronic exposure at a constant level, which may be invalid.

[98]Wiener et al., p. 430.

Table 7. Relative Fatty Acid Content and Mercury Concentration in Some Popular Fish

Species	Average Mercury Level (ppm)	Relative Fatty Acid Content
Shrimp	<0.01	Moderate
Tuna, white, canned (solid and chunk albacore)	0.36	High
Tuna, light, canned (chunk)	0.12	Moderate
Salmon	0.01	High
Pollock	0.06	Moderate
Catfish	0.05	Low (Channel) Moderate (Brown Bullhead)
Cod	0.11	Low
Crab (blue, king, snow)	0.06	Moderate
Clams	<0.01	Low
Flatfish (flounder, sole, plaice)	0.05	Low
Scallops	0.05	Low
Halibut	0.26	Moderate
Trout	0.03	Moderate (Rainbow) High (Lake)
Swordfish	0.97	Low

Sources: FDA websites Mercury Levels in Commercial Fish and Shellfish, at [http://www.cfsan.fda.gov/~frf/sea-mehg.html], and Mercury in Fish: FDA Monitoring Program (1990-2003), at [http://www.cfsan.fda.gov/~frf/seamehg2.html]; Purdue University, Food Safety and Quality, Angling Indiana, 2004 Fish Consumption Advisory, Nutritional Content of Fish, at [http://fn.cfs.purdue.edu/anglingindiana/NutritionalContentofFish/omega3.pdf], visited April 26, 2004.

In common loons, which have been studied extensively, concentrations of mercury in blood correlate with mercury levels in the fish they eat. Mercury levels in loon blood increase from west to east in Canada, with the highest levels being found in southeast Canada.[99] A recent study of mercury in 577 loon eggs collected across eight U.S. states from Alaska to Maine found a similar trend of increasing mercury concentrations from west to east.[100] These blood and egg concentrations are consistent with the pattern

[99]D. C. Evers, J. D. Kaplan, M. W. Meyer, et al., "A geographic trend in mercury measured in common loon feathers and blood," *Environmental Toxicology and Chemistry*, v. 17, n. 2 (1998), pp. 173-184.

[100]D. C. Evers, K. M. Taylor, A. Major, et al., "Common loon eggs as indicators of methylmercury availability in North America," *Ecotoxicology*, v. 12 (2003), pp. 69-81.

of mercury deposition for North America (i.e., increasing from west to east).[101] A study reported in 2003 declining egg volume, but no effect on fertility, with increasing mercury concentrations in New England. However, eggs were collected only if abandoned, which might have biased the results.[102] Reduced egg laying has been associated with concentrations greater than 0.4 ppm methylmercury in prey fish.[103]

Mink and otter exposed over a long period of time to more than 1 ppm methylmercury in their diets exhibit classic signs of poisoning and may die. Higher concentrations cause earlier but similar health effects.[104] Less than half that concentration is not lethal; data are lacking for more subtle effects on mink of mercury exposure. There are no field data indicating that the wildlife species most at risk (because they eat fish) currently are experiencing adverse health effects from mercury exposure.

Conclusion

Current scientific knowledge can inform the debate about competing legislative and administrative proposals to reduce mercury emissions from utilities, but it cannot provide firm answers to all of the specific questions that have been raised. Neither can science resolve policy controversies that revolve around value judgments, for example, questions about how urgent the need is for utility emission controls. However, recent scientific studies have provided potentially useful information for policy makers, about chemical changes to mercury emissions that may take place in the atmosphere; rates of mercury deposition to, and re-emission from, the earth's surface; the relationship between mercury emissions and mercury levels in freshwater fish in various specific ecosystems; and the potential effects of low level, chronic exposure to methyl mercury through fish consumption.

Scientific studies have clearly demonstrated that levels of mercury in the atmosphere and in deposits to earth have at least doubled and probably tripled due to human activities, even in places that are remote from human influence. Although most of the largest and most direct U.S. sources of mercury releases to water and air have been controlled, and levels of U.S. mercury deposition are declining, levels of mercury in fish continue to be a concern. Electric utilities are the only uncontrolled major stationary source of U.S. mercury emissions. As a result, control of utility emissions might be the most direct step that could be taken to reduce mercury deposition in the United States. However, there are uncertainties in chemistry and transport, leading to current debates among policy makers.

Local and regional emissions from various sources have caused mercury deposition to increase as much as tenfold in some locations, indicating that there is a possibility that local "hot spots" of mercury contamination might persist, despite overall reductions in mercury emissions. In sensitive experimental lakes and wetlands, when local and

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[101]EPA, *Mercury Study Report to Congress*, vol. 7, "Characterization of human health and wildlife risks from mercury exposure in the United States."

[102]Evers et al., 2003.

[103]Wiener et al., p. 430.

[104]Wiener et al., p. 435.

regional mercury emissions decreased, deposition decreased proportionately, and levels of methylmercury in freshwater fish dropped quickly. This indicates that controls on mercury emissions from electric power plants (particularly those plants with emissions that tend to be deposited locally) could lead to substantial reductions in deposition at some nearby hot spots. It remains to be determined whether there is a link between mercury emissions and mercury in ocean fish. However, scientists have shown that significant quantities of emitted mercury are deposited in the oceans; methylmercury is found in marine fish and predatory seabirds, sometimes at very high concentrations; and sulfate-reducing bacteria are active in coastal sediments.

As yet unquantifiable but potentially significant risks from emissions exist, to people and wildlife locally, but also in areas distant from emission sources. Research continues to find evidence of subtle impacts on human health of low levels of methylmercury exposure, levels close to current levels of exposure for people who eat large amounts of certain large, predatory fish. In considering the potential adverse effects of mercury, however, the potential nutritional benefits of eating fish that are not heavily contaminated by mercury should not be overlooked.