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Mercury in the Environment: A Volatile Problem

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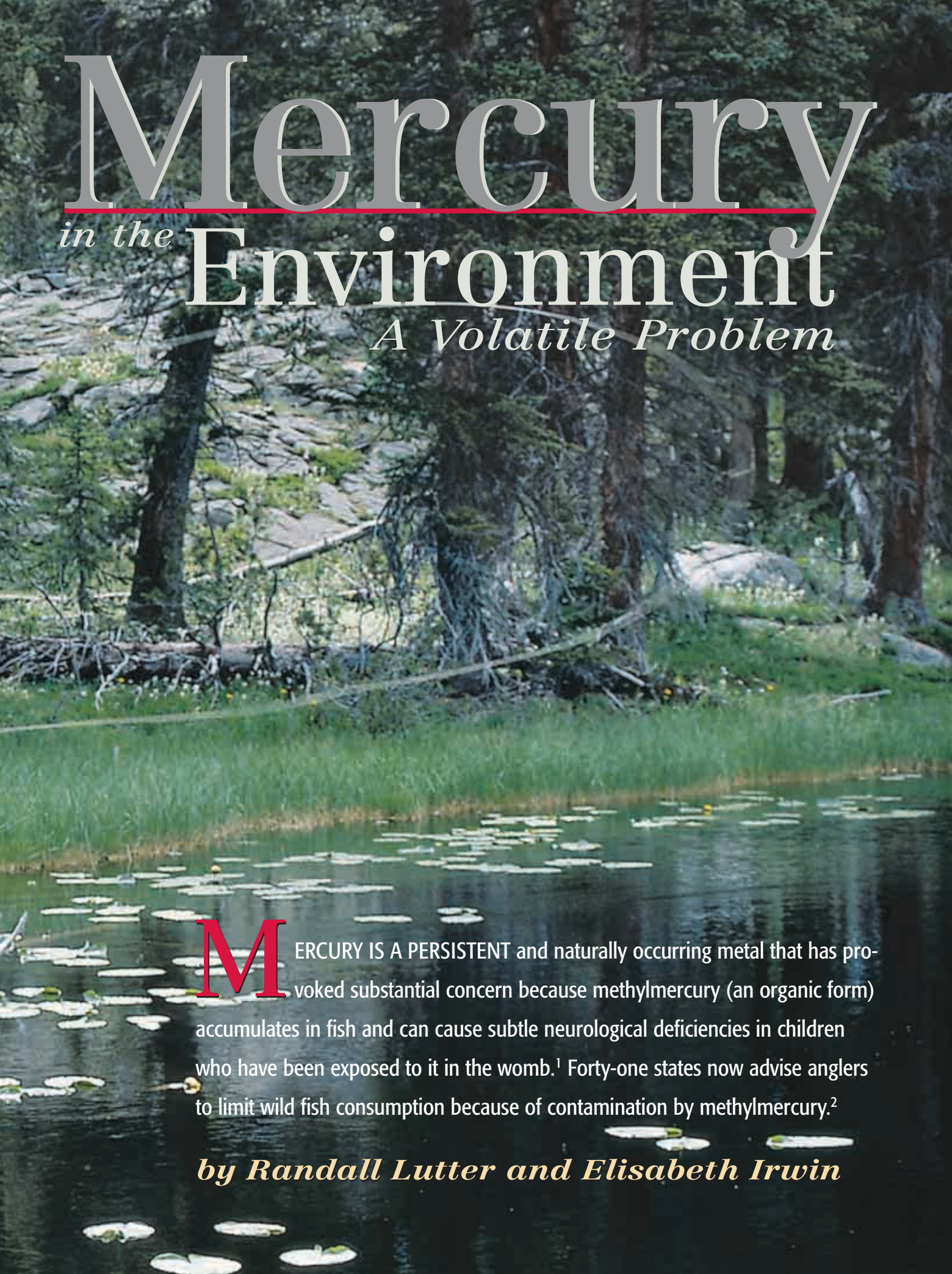


Mercury in the Environment

A Volatile Problem

MERCURY IS A PERSISTENT and naturally occurring metal that has provoked substantial concern because methylmercury (an organic form) accumulates in fish and can cause subtle neurological deficiencies in children who have been exposed to it in the womb.[1] Forty-one states now advise anglers to limit wild fish consumption because of contamination by methylmercury.[2]

by Randall Lutter and Elisabeth Irwin

Accordingly, the U.S. Food and Drug Administration is under pressure to reduce its permissible level of mercury in fish sold in the United States.[3] To address concern about mercury, several senators and President George W. Bush have proposed new legislation to cut emissions of pollutants from power plants, which are the biggest anthropogenic source of mercury in the United States.[4]

However, these different parties disagree about how mercury should be regulated. The controversy is likely to grow through December 2004, which is the deadline for the U.S. Environmental Protection Agency (EPA) to regulate emissions following the maximum achievable control technology (MACT) provisions of the Clean Air Act.[5] This proposed regulation has faced some opposition because it is relatively costly.

In general, established U.S. regulatory policy suggests that regulatory decisions to manage risks should carefully assess the costs of controls and the resulting improvements to human health and the environment.[6] However, attempts to implement this policy in the case of mercury are complicated by significant scientific uncertainty about the role of natural and anthropogenic sources of environmental mercury, how mercury is transported through the environment and where it eventually rests, the processes that produce methylmercury, the effects of methylmercury on ecosystems, and the nature and scope of mercury-related risks to human health.

The neurotoxic effects of methylmercury on children's health evoke dread and prompt reform proposals based on emotion rather than on science. However, the magnitude of risk depends on the degree of exposure to mercury, and fortunately, exposure generally appears small in the United States. Because of the uncertainty about these factors, identifying emissions limits that balance costs and benefits will be challenging to policy makers. Nonetheless, the available evidence suggests that even deep cuts in mercury emissions are unlikely to bring large benefits to public health or ecosystems. A stringent and costly cap on emissions may not be the best policy.

In examining the problem of mercury, it is necessary to review the current state of scientific understanding. Weaknesses and strengths should be identified—as should areas in which additional information would be of high value to policy makers. This article discusses mercury in the environment, human health effects, and ecological effects, and it examines the merit of past and prospective regulatory programs.

USDA PHOTO—KEN HAMMOND

Much of the largemouth bass eaten in the United States has mercury concentrations that exceed the U.S. Environmental Protection Agency's water-quality standard.

Mercury in the Environment

Mercury is present in the environment due to human activity as well as natural sources such as volcanoes and forest fires. The scope of mercury releases relevant to determining the causes of contamination in U.S. waters may be regional or even global rather than local, because mercury travels long distances in the air.[7] Table 1 on page 27 summarizes key properties and the transport and fate of different forms of mercury. Forty percent (32 metric tons (t)) of the mercury deposited from the air onto U.S. water and soil in 1995 came from the global mercury reservoir (the amount of mercury circulating worldwide at any one time); the other 60 percent came from anthropogenic sources in the United States.[8] Both natural and anthropogenic emissions contribute to the global mercury reservoir, and although significant uncertainty exists as to the length of time that some forms of mercury persist in the atmosphere, an authoritative estimate of annual global input to the reservoir is about 4,900 t.[9]

There is also substantial uncertainty about the share of worldwide emissions that originates from human activity. One study concluded that natural sources, industrial sources, and the "recycling" of anthropogenic mercury each account for about one-third of the mercury burden in the global atmosphere at a given moment.[10] Recycling occurs when mercury in water volatilizes and contributes to the buildup of atmospheric mercury concentrations.[11]

Mercury releases from ongoing human activity in the United States can be divided into four broad categories. These include "area sources" such as landfills, dental preparations, and laboratory use; combustion processes, including coal-fired power generation,

medical waste incinerators, and municipal waste combustors; some manufacture of metals, alkali, and cement; and other various industrial processes, from pigment manufacture to geothermal power generation. EPA estimated that emissions from these source categories generated 2.76 t, 112.3 t, 12.67 t, and 1.16 t of environmental mercury per year, respectively, in 1994–95.[12] Thus, combustion sources accounted for nearly 90 percent of total U.S. anthropogenic emissions. In recent years, EPA has regulated medical waste incinerators, municipal waste combustors, and hazardous waste combustors (see Table 2 below).[13] When these regulations are fully implemented in the fall of 2002, EPA estimates annual mercury emissions of about 54 t lower than they would be without these regulations.[14]

Mining in the past has raised mercury concentrations in some areas. Releases of quicksilver mercury in the hydraulic placer-gold mines of the Sierra Nevadas added 1,360 t to 3,630 t of mercury to the environment from the 1860s through the early 1900s.[15] According to the U.S. Geological Survey (USGS), "High mercury levels in fish, amphibians, and invertebrates downstream of the hydraulic mines are a consequence of historic mercury use."[16]

Methylation

Bacteria in water bodies transform water-borne elemental, divalent, or inorganic mercury into methylmercury (CH_3Hg^+), the organic form that accu-

Table 1. Forms of mercury

	Elemental or metallic (Hg^0)	Divalent or mercuric (Hg^{2+})	Methylmercury (CH_3Hg^+)
Key properties	95 percent of atmospheric mercury is Hg^0 vapor.	Bound to airborne particles Comprises 5 percent of atmospheric mercury Found in soil and water as a number of complex ions May form inorganic mercuric salts	Lipophylic ion produced by bacteria in the water column or sediment Nearly all mercury in fish is methylated.
Transport and fate	Tends to remain airborne Not easily deposited May travel long distances before conversion to other forms and deposition	Easily deposited to Earth's surface in dry form or in precipitation Once in water, may volatilize or partition into particulates and be transported to sediment	Enters food chain through aquatic biota and uptake into fish tissue Bioaccumulates as it travels up the food chain, reaching highest concentrations in organisms at highest trophic level

SOURCE: U.S. Centers for Disease Control, Agency for Toxic Substances and Disease Registry, "Toxicological Profile: Mercury" (1999), available at http://www.atsdr.cdc.gov/toxprofiles/tp46-c5.pdf; and U.S. Environmental Protection Agency, *Mercury Study Report to Congress*, EPA-452/R-97-003 (Washington, D.C., 1997), vol. 1.

Table 2. Recent U.S. mercury regulations

	Municipal waste combustors	Medical waste incinerators	Hazardous waste combustors
Date issued	25 August 1997	15 September 1997	13 September 1999
Compliance date	19 December 2000	15 September 2002	30 September 2002
Projected annual emissions cuts	38 metric tons	13 metric tons	3 metric tons
Annual emissions cuts as a percentage of preregulation levels	78%	94%	55%

SOURCE: U.S. Environmental Protection Agency, *Deposition of Air Pollutants to the Great Waters Third Report to Congress 2000* (Washington, D.C., 2000): Table III-2.

mulates as it moves into higher levels of the food chain. Methylation occurs both in sediment and in the water column after mercury has entered the aquatic environment through methods such as atmospheric deposition, runoff, and groundwater flow from the upper levels of soil.[17] However, not all mercury that enters a water body undergoes methylation. Rates of methylation vary with the aquatic environment and—due to limited understanding of the factors that influence methylation—are subject to significant scientific uncertainty.[18]

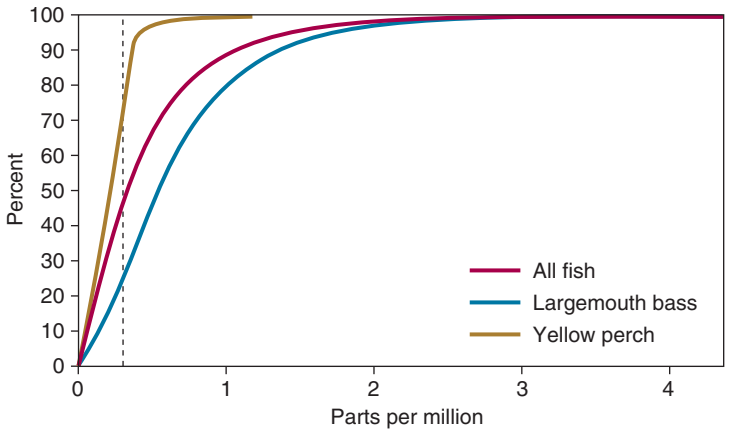
Although there is a large degree of doubt about the processes that methylate mercury, various studies suggest that the most important factors influencing methylation may be chloride, sulfate, dissolved organic carbon, calcium, and pH.[19] A study published in *Environmental Science & Technology* concludes that methylmercury principally accumulates in phytoplankton as it takes up uncharged chloride complexes.[20] Phytoplankton is subsequently ingested by noncarnivorous fish, beginning the journey of methylmercury up the food chain and its process of bioaccumulation.

This study also cites pH as a primary determinant of methylmercury. Low pH may increase bacterial methylation rates, therefore increasing methylmercury levels.[21] Typically, higher methylmercury levels in fish tissue are found in more acidic waters.[22] Sulfate appears to be a critical factor in the methylation process because sulfate-reducing bacteria stimulate methylmercury production. Recent USGS studies in the Florida Everglades region have found a correlation between sulfate levels and methylmercury concentrations.[23] While various processes can methylate mercury, USGS reports that "scientists generally agree that methylation by sulfate-reducing bacteria is most important."[24] EPA has listed dissolved organic carbon as a characteristic of water bodies that affects both methylation and demethylation (the process that reverses methylation) in the water column.[25] However, USGS reports, "Depending on local conditions, the amount of dissolved organic carbon-mercury binding can either increase or reduce mercury uptake by organisms."[26] Finally, high calcium levels may reduce the bioavailability of methylmercury because calcium cations may compete with metals such as mercury for cellular binding sites.[27]

Land use also affects the mercury concentrations that are available for methylation. Land under cultivation may be a proxy measure of erosion, which transports mercury from the soil to local water bodies. EPA notes that "[b]oth watershed erosion and direct atmospheric deposition can be important sources of mercury to the water body, depending on the relative sizes of the water body and the watershed."[28] In addition, the amount of land under cultivation may serve as an indicator of the degree to which fungicides containing mercury are used for agriculture.[29] In EPA's summary of studies in Lake Champlain, it reported that "urban and agricultural systems may retain less atmospheric mercury than forested systems," thus transporting more mercury to water, where it can be methylated.[30]

Methylmercury, which constitutes the vast majority of all mercury found in fish, accumulates at higher concentrations at higher levels in the food chain because of its affinity for fatty tissues.[31] Figure 1 on this page depicts the cumulative distribution of mercury concentrations in all fish, all largemouth bass, and all yellow perch based on an EPA study of mercury in nearly 82,000 fish caught from 1990 to 1995 in 5,000 locations in 3,200 water bodies.[32] These two broadly distributed and commonly eaten species illustrate how concentrations differ across species. Although 45 percent of all fish and 75 percent of yellow perch have mercury concentrations less than 0.3 parts per million (ppm), only 25 percent of largemouth bass have concentrations less than this value. EPA's water-quality standard for methylmercury is 0.3 ppm, which the agency recently established to "protect consumers of fish and shellfish among the general population."[33]

Figure 1. Mercury concentrations in fish



NOTE: The vertical line marks 0.3 parts per million, the fish tissue concentration that corresponds to the U.S. Environmental Protection Agency's (EPA) water-quality standard. This figure maintains EPA's conservative assumption that mercury concentrations too low to detect are equal to the limit of detection. These limits of detection vary among states.

SOURCE: EPA, *The National Survey of Mercury Concentrations in Fish: Data Base Summary 1990–1995*, EPA-823-R-99-014 (Washington, D.C., 1999): 4–7.

Transport

Atmospheric deposition appears to be the primary means of environmental mercury contamination.[34] Recent research

demonstrates how levels of mercury deposition have varied over time with volcanic eruptions, gold mining, and industrialization (see Figure 2 on this page). Although mercury deposition can occur in both wet and dry forms, EPA has concluded that deposition through precipitation is the primary means of transport between the atmosphere and the Earth's surface.[35] Studies of Lake Michigan suggest that about 80 percent of total deposition is wet.[36]

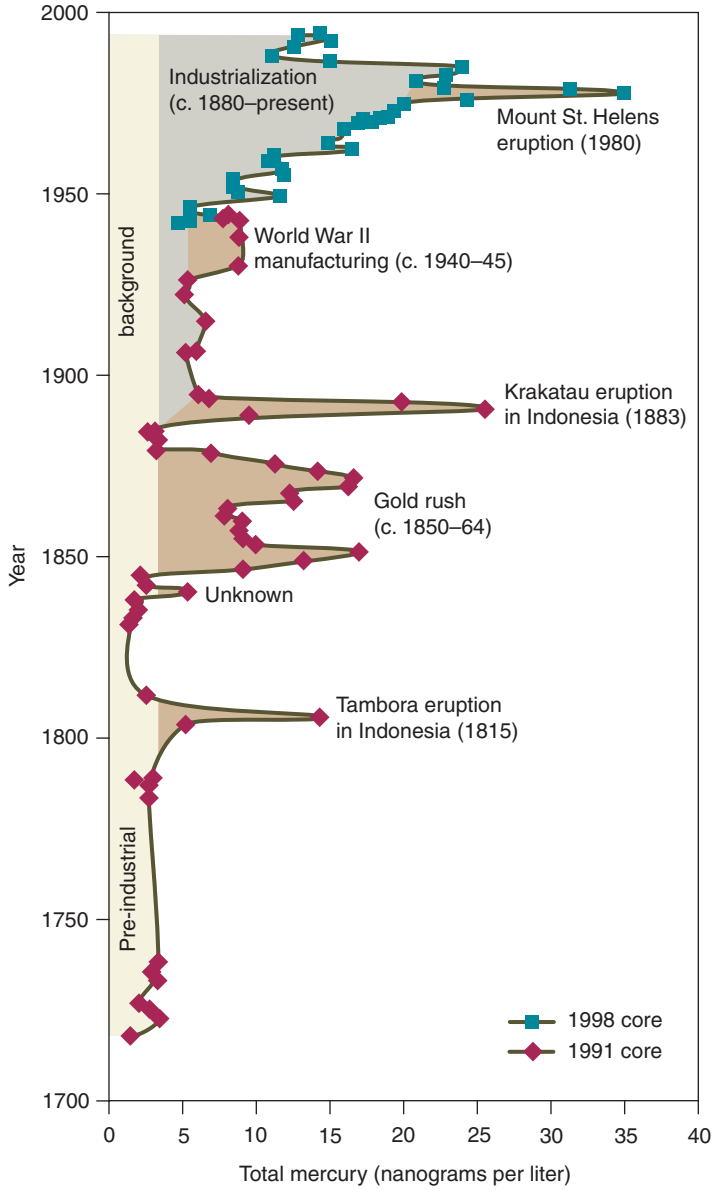
However, the determinants of wet mercury deposition in the United States are poorly understood. The National Acid Deposition Program takes field measurements of wet mercury deposition in 55 locations.[37] It is interesting to compare this program's data from the late 1990s with predictions from EPA's Regional Lagrangian Model of Air Pollution (RELMAP) modeling, which EPA derived using emissions from 1994–95 and weather from 1989.[38] Figure 3 on page 30 shows that field measurements of the annual average wet mercury deposition and RELMAP estimates for wet and dry deposition have little correlation and, thus, that mercury deposition is not predictable.

Also uncertain is the process by which mercury is removed from the aquatic environment. Mercury can be carried downstream or volatilize into gaseous elemental mercury from its divalent form (Hg^{2+}). EPA found that sedimentation may be the dominant process by which mercury leaves a water body.[39]

Effects on Human Health

The health effects of mercury are presumed to be irreversible. The U.S. Centers for Disease Control recently reported new data on levels of mercury in the blood and hair of young children and women of childbearing age, based on the 1999 National Health and Nutrition Examination Survey (NHANES), which was random and nationally representative.[40] The 90th percentile of mercury in hair for 702 women was 1.4 ppm, an estimate in line with earlier surveys of hair mercury levels in nonrandom samples of U.S. residents.[41] The study reports that "approximately 10 percent of women have mercury levels within one tenth of potentially hazardous levels indicating a narrow margin of safety for some women and supporting efforts to reduce methyl mercury exposure."[42] A more precise statement about the number of women exposed to elevated levels is difficult because NHANES does not provide estimates of mercury exposure in certain highly exposed groups such as some Native Americans and others who eat large amounts of fish.

Figure 2. A 270-year record of mercury deposition



NOTE: 1991 and 1998 cores refer to sections of ice cores that were collected from the Upper Fremont Glacier in Wyoming's Wind River Range in those years.

SOURCE: P. F. Schuster et al., "Atmospheric Mercury Deposition During the Last 270 Years: A Glacial Ice Core Record of Natural and Anthropogenic Sources," *Environmental Science & Technology* 36, no. 11 (2002): 2,303–10.

Careful epidemiological studies of children exposed in the womb to higher methylmercury concentrations than those typically found in the United States have reached sharply different conclusions. A study of children in the Seychelles Islands found no association between methylmercury and a broad variety of health effects.[43] A study in the Faroe Islands found statistically significant associations in the domains of language, attention, memory, and—to a lesser extent—visuospatial and motor functions, but it did not examine broader measures of performance such as IQ tests.[44] A study of New Zealand children found declines in summary measures of neurological performance, but only if a single highly exposed and apparently healthy child is omitted from the analysis.[45]

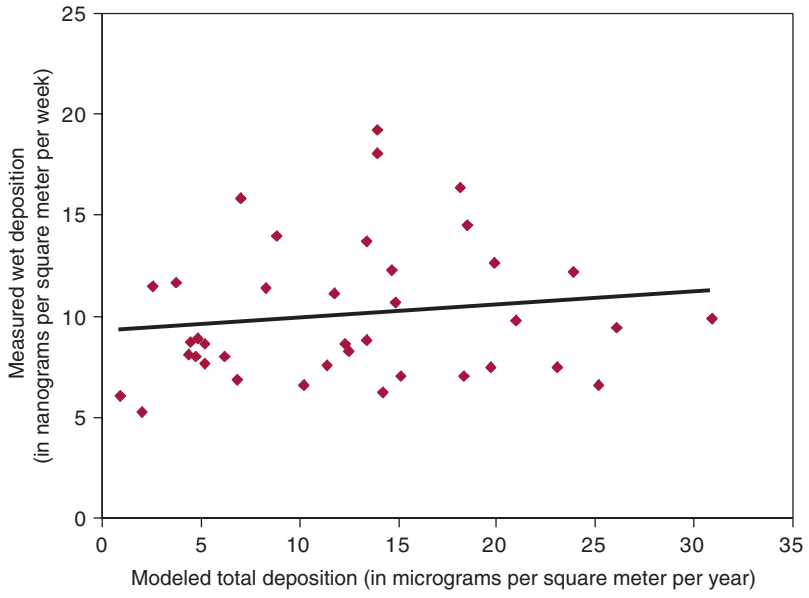
In evaluating why such studies have different outcomes, the National Research Council (NRC) reported that there "do not appear to be any serious flaws in [their] design and conduct."[46] In a 2000 review of these studies, NRC estimated that in the United States, "60,000 newborns annually might be at risk for adverse neurodevelopmental effects from in utero exposure to methylmercury."[47] NRC derived this estimate as the product of the number of female fish-consumers in the United States aged 15 to 44 years, 5 percent (thought to correspond to those consuming more than 100 grams per day of fish), and the birth rate.[48] This widely cited estimate is intended to represent the number of children born to mothers who may be exposed to mercury at levels exceeding the current reference dose of 0.1 microgram (μg) per kilogram body weight per day.[49] However, the chair of NRC's Committee on the Toxicological Effects of Methylmercury, Robert Goyer, cautioned, "That number should not be interpreted as an estimate of the annual number of cases of adverse neurodevelopmental effects."[50]

An earlier survey, published in 1997, provides estimates of the decline in mercury exposure in U.S. women that would result from the hypothetical elimination of mercury in fish. These results, which are similar to those of the NHANES survey, indicate that geometric mean mercury concentrations in the hair of respondents reporting some seafood consumption and no seafood consumption were 0.36 ppm and 0.24 ppm, respectively (with geometric standard deviations of 2.5 ppm and 2.6 ppm, respectively).[51]

Because 55 percent of the women who responded to the 1997 survey reported eating fish, and approximately 3.9 million children are born annually in the United States, these distributions imply that the elimination of mercury from fish would reduce the number of children born to women with hair mercury levels of 1–2 ppm by 100,000, children born to women with levels of 2–4 ppm by 30,000, and children born to women with levels greater than 4 ppm by 6,000.[52] Presuming that mercury exposure lower than 1 ppm has negligible effects, children who would have been born to women with levels of 1–2 ppm would experience reductions in potentially hazardous in-utero exposure of 0.5 ppm on average. Exposure of children who would have been born to mothers with mercury levels of 2–4 ppm would be reduced by 1.8 ppm, and exposure among children who would have been born to mothers with levels of greater than 4 ppm would be reduced by 4.4 ppm. Because these exposure levels are for the hypothetical total elimination of mercury in fish, actual reductions from foreseeable regulations would be smaller.

Assessing potential health improvements for children requires information

Figure 3. Mercury deposition values: measured vs. modeled



NOTE: The line represents the regression line (line of best fit). Only data from field monitors with at least 48 weeks of data are presented.

SOURCE: R. Lutter and E. Irwin's calculations of data from U.S. Department of Commerce, National Oceanic and Atmospheric Administration, Air Resources Laboratory, "Mercury Deposition Network" (NRSP-3), National Atmospheric Deposition Program web site (2000), accessed via http://nadp.sws.uiuc.edu/mdn/ on 23 July 2002; and EPA, *Mercury Study Report to Congress*, EPA-452/R-97-003 (Washington, D.C., 1997), vols. 2 and 3, p. 4-2.

The eruption of Mount St. Helens in 1980 contributed to atmospheric mercury concentrations. Volcanoes and forest fires are natural sources of mercury that add to the global mercury reservoir circulating through the atmosphere.

about the relationship between exposure and health effects. A 1999 study by Phillipe Grandjean and colleagues and a 1998 study by Kenny Crump and colleagues provide estimates of these relationships for neurological performance measures of finger-tapping, hand-eye coordination, reaction time, delayed recall of words, and two specialized tests: the Boston naming test and McCarthy's Perceptual Performance Scale.[53] According to these studies, reducing mercury in a mother's hair by 4.4 ppm—reduction to the "negligible" level from the average level experienced by the 6,000 children in the most exposed group—would improve reaction times by only 22 percent of a standard deviation.[54] (In assessing these health effects, it should be noted that four standard deviations represents a typical range of behavior in a population given a normal distribution.) All of the other health effects for which Grandjean or Crump report quantitative exposure response information would improve by less than 20 percent of a standard deviation if children were exposed in utero to mercury at negligible levels rather than levels greater than 4 ppm. For the children originally exposed at less than 4 ppm, reducing mercury exposure further would result in even smaller benefits.

A 1995 study of 1,833 middle-aged and older Finnish men with average hair mercury levels of 1.9 ppm found mercury exposure to be associated with elevated risks of heart attacks and death.[55] Study authors Jukka Salonen and colleagues estimated that subjects with hair mercury levels greater than 2 ppm had a 70-percent greater risk of suffering acute myocardial infarctions than subjects with lower hair mercury levels.[56] They also reported that the risk of death for men with hair mercury levels greater than 2 ppm was 93 percent higher than that of men with lower levels.

There are several reasons for skepticism about the results of this study.[57] It has not been independently replicated in other samples, so the results may reflect something peculiar to the Finnish population.[58] In addition, the study did not show that relative risk increases systematically with exposure, as should generally be the case.[59] Indeed, the paper's conclusions are contrary to those of an epidemiological study published in 1996 of people exposed in Japan during the infamous 1950s Minamata episode.[60] The 1996 study found no excess risk of overall mortality or noncancer deaths, even though exposure to mercury was vastly greater than that of Finnish men in the 1995 study.

Finally, the Finnish study did not assess whether mercury in hair was predominantly methylmercury—an important issue because nearly all mercury in fish is methylmercury. Mercury from other sources could have played a significant role, as is suggested by some aspects of the data. Salonen and colleagues report hair mercury levels that were 260 percent higher than those reported by a 1996 study of New Jersey women (1.9 ppm versus 0.53 ppm)—but levels of mercury in the diet that were only 20 percent greater (7.6 µg per day versus 6.3 µg per day).[61] Because fish intake among the Finnish men correlates only weakly with mercury in hair, some of the variation in hair mercury levels may be due to factors other than fish consumption.[62] Thus, heart attack risks from mercury exposure merit greater attention from researchers—but not from policy makers at this point.

It is difficult to compare the health effects of mercury with potential health problems caused by other environmen-

Erosion—evident on farmland near Cedar Bluff Reservoir in Kansas—transports mercury that has been deposited on the soil to local water bodies, where it can be methylated. Some chemicals used for agriculture contain mercury.

tal factors, but the effects described above seem less severe than some others such as cancers, fatalities from respiratory ailments, or IQ deficits related to lead poisoning. Efforts to reduce the risk for health effects such as these are currently being pursued through environmental policies.

Effects on the Environment

Assessing the environmental effects of mercury is intrinsically difficult because of the complexity of ecosystems, the existence of multiple sources of stress, and the many endpoints where mercury might have adverse effects. In its 1997 *Mercury Study Report to Congress*, EPA described environmental effects by noting that environmental mercury may adversely affect reproduction of loons—fish-eating birds in which effects should be most easily detectable because of the bioaccumulative properties of methylmercury. However, common loon populations grew at an annual rate of 2.6 percent between 1966 and 1999.[63] Populations of other fish-eating birds grew at even faster rates.[64] Such high growth rates and the understanding that there appears to be no correlation between areas of low population growth and areas of high mercury deposition suggest that the ecological benefits of reducing mercury emissions may not be great.[65]

Florida panthers also may be at risk of methylmercury poisoning, because high mercury concentrations in their habitat have caused high mercury concentrations in their prey. According to EPA, "Mercury levels found in tissues obtained from dead panthers are similar to levels that have been associated with frank toxic effects in other feline species."[66] Over the last decade, however, the Florida panther population apparently has increased to 80 from between 30 and 50 as a result of introducing new panthers into the area to reduce inbreeding.[67]

Although a recent screening-level assessment of mercury in southern Florida food webs suggested that 100 percent of alligators are exposed to methylmercury at concentrations that exceed toxic reference values for sensitive bird and mammal species, no data on its toxicity to alligators are available.[68] No studies that quantitatively estimate the effect of methylmercury on the population of alligators or other indicator species are apparent.

Mercury Controls

Policy makers should take into account the environmental and health effects of mercury when they design new regulations to limit mercury releases. In the past several years, EPA has issued three regulations to limit mercury emissions (as shown in Table 2). The U.S. government has regulated a diverse set of possible uses of mercury under other laws, as listed in Table 3 on page 33. Concern about the environmental effects of mercury has prompted a variety of industry and state efforts to restrict the sale of certain products and implement disposal programs; these measures reduced industrial demand for mercury by 75 percent between 1988 and 1997.[69] To date, 18 states have proposed 49 bills that would restrict or prohibit products that contain mercury; limit mercury emissions from power plants; or establish product-labeling requirements, health-warning standards for water bodies, or waste-management programs.[70] Currently, only Indiana, Maryland, Minnesota,

and Maine have enacted these laws, but some states have instituted special programs to remove from use thermometers or other devices that contain mercury.[71] The box on pages 34 and 35 provides an overview of international regulation.

EPA expressed its recent water-quality criteria for methylmercury in terms of fish tissue concentration—0.3 ppm—because fish consumption is the primary means of human exposure to methylmercury. EPA intends for its water-quality criteria program to provide a science-based maximum level for methylmercury that can be used by states and localities to promulgate local water-quality standards and by EPA as a basis for federal rulemaking to reduce mercury discharges under the Clean Water Act. States are not forced to adopt the EPA criteria, but under the Clean Water Act, they must adopt water-quality standards that are adequate for each water body's designated use—such as swimming—and commensurate with "sound scientific rationale."[72] In addition, EPA must approve states' standards. Thus, the practical impact of EPA's criteria is likely to be high even though, strictly speaking, the criteria are not legally binding. However, because 55 percent of fish have mercury concentrations that exceed EPA's water-quality criteria and there is no model predicting how much mercury emissions must be cut to reduce concentrations in fish tissue, compliance with EPA-approved water-quality standards might be quite difficult.[73]

Prospective Regulations

Because coal-fired power plants are the largest unregulated source of anthropogenic mercury emissions in the United States, they are a favorite target of new regulation and legislation. A bill introduced by Senator Jim Jeffords (I-Vt.), chair of the Environment and Public Works Committee, would reduce

Table 3. U.S. mercury product regulations

Product	Role of mercury	Regulation or program
Agricultural products	Pesticide, bactericide, disinfectant, fungicide	Restricted and/or banned under U.S. Insecticide, Fungicide, and Rodenticide Act
Paints	Biocide to control microbial growth in paint cans and mildew on painted surfaces	Registrations for use of mercury in latex paint cancelled in 1990–91; mercury in antifouling paint for marine use banned in 1972
Pigments and dyes	Red coloring for plastic	Domestic production ceased in 1988, imports allowed
Cosmetics	Preservative, antimicrobial	Limited to eye area and concentrations under 65 parts per million[a]
Fluorescent lamps	Mercury vapor fluoresces under ultraviolet light	Spent lamps were added in 1999 to the Universal Waste Rule, implementing the Resource Conservation and Recovery Act (RCRA) to reduce risks that might pose threats over time in municipal solid waste landfills[b]
Batteries	Mercury offers high and lasting voltage in alkaline-manganese and zinc-carbon batteries, commonly used for cameras	Included in 1995 Universal Waste Rule of RCRA to divert them from municipal solid waste landfills[c]
Dental equipment	Forms alloys, binds compounds to form restorative material	Under the U.S. Food, Drug, and Cosmetic Act, dental mercury and amalgam alloys regulated separately by the U.S. Food and Drug Administration as Class I and II devices

[a] Code of Federal Regulations, U.S. Department of Health and Human Services, 21CFR700.13 (2001).
[b] U.S. Environmental Protection Agency (EPA), "Hazardous Waste Management System: Modification of the Hazardous Waste Program—Hazardous Waste Lamps," *Federal Register* 64, no. 128 (6 July 1999).
[c] EPA, "The Universal Waste Rule" information sheet, available at http://www.epa.gov/epaoswer/hazwaste/id/univwast.htm (last updated 24 June 2002).

NOTE: Battery manufacturers have reduced mercury use by more than 90 percent since 1988.

SOURCE: U.S. Environmental Protection Agency, Great Lakes Program, Virtual Elimination Pilot Project, "Mercury Sources and Regulations" (1994): Appendix C, available at http://www.epa.gov/grtlakes/bnsdocs/mercsrce/images/9409merc.pdf.

International Mercury Regulation

An overview of mercury regulation in the European Union, Japan, and the United States reveals a variety of different approaches. Promoting uniformity, the 1998 United Nations Protocol on Heavy Metals set prescriptive numerical emissions limits for hazardous and municipal waste incinerators and directed parties to the protocol to set limits for medical waste incinerators.[1] The European Union (EU) has approved the protocol, and the United States has accepted but not ratified it. The protocol does not mention the use of economic approaches such as the tradable mercury emissions permits proposed in the United States as part of the Bush administration's Clear Skies Initiative. Because the protocol does not address mercury emissions from electricity generation or the regulation of mercury in lakes and rivers, it leaves room for ample differences in approaches across countries. See the table below for some approaches to mercury regulation in the United States, the EU, and Japan.

Separate from the UN protocol, in 1996 the European Council issued a directive ordering "limit values and alert thresholds" for a variety of air pollutants, including mercury.[2] The directive resembles aspects of U.S. federal regulation in that it assigns to member states the responsibility to implement the limit val-

Regulatory approaches

Jurisdiction	Air emissions	Water	Methylmercury consumption reference dose
United States	Regulated except for power utilities, for which legislation and regulation are pending.	Water-quality standard of 0.3 ppm in tissue of fish in U.S. waters Effluent limits and standards exist on mercury discharges from a broad range of industrial sources.[a]	0.1 µg/kg/day
European Union	UN protocol set limits on mercury emissions from municipal and hazardous waste incinerators.[b] Proposed ambient air-quality standard of 0.05 µg/m^3	Water Framework Directive (2000/60/EEC) reaffirms various effluent limits under 1982 and 1984 directives.[c]	EU Position Paper, written in compliance with Air Quality Framework Directive of 1996, indicates that EPA's reference dose of 0.1 µg/kg/day is also appropriate for the EU.[d]
Japan	Mercury is identified as a "substance requiring priority action" under harmful air pollutant regulations, which specify duties such as accumulation of knowledge and voluntary reductions.[e]	0.5 µg/liter for all public water bodies[f] Uniform National Effluent Standards permit 5 µg/liter[g]	Provisional tolerable weekly intake of 0.4 µg/kg/day[h]

[a] See Code of Federal Regulations, U.S. Environmental Protection Agency, 40CFR26, parts 470–71 (2001).
[b] United Nations Economic Commission for Europe, "Protocol to the 1979 Convention on Long-Range Transboundary Air Pollution on Heavy Metals," Annex V (1998), accessible via http://www.unece.org/env/lrtap/protocol/98hm_a/annex5.htm.
[c] United Kingdom Department of Environment, Food, and Rural Affairs, "Regulatory Impact Assessment of a Priority List of Substances Under Article 16 of the Water Framework Directive" (2002), accessible via http://www.defra.gov.uk/environment/water/wfd/art16-ria/pdf/art16_ria.pdf. See also European Council Directive 82/176/EEC (Official Journal L 81, 27.03.1982); and European Council Directive 84/156/EEC (Official Journal L 74, 17.3.1984).
[d] European Commission, Working Group on Mercury, *Ambient Air Pollution by Mercury: Position Paper* (Luxembourg: European Communities, 2001): page ES 3-7, accessible via http://europa.eu.int/comm/environment/air/background.htm#mercury.
[e] Government of Japan, Ministry of the Environment, "Regulatory Measures Against Air Pollutants Emitted from Factories and Business Sites and the Outline of Regulation" (last amended 1998), accessible via http://www.env.go.jp/en/lar/regulation/air.html.
[f] Government of Japan, Ministry of the Environment, "Environmental Quality Standards for Water Pollution," accessible via http://www.env.go.jp/en/lar/regulation/wp.html.
[g] Government of Japan, Ministry of Environment, "National Effluent Standards," accessible via http://www.env.go.jp/en/lar/regulation/nes.html.
[h] European Commission, note *d* above, page 5-23.

NOTE: ppm = parts per million; µg/kg/day = micrograms per kilogram body weight per day; m^3 = cubic meter.

SOURCE: R. Lutter and E. Irwin.

ues and all requisite attainment programs. In response to the directive, the European Commission proposed an ambient air-quality standard of 0.05 micrograms per cubic meter for elemental mercury to protect the general population from kidney cancer related to inhalation.[3] However, the proposed standard is rarely exceeded in Europe. The United States and Japan do not have such a standard.

The United States has regulated all significant sources of mercury emissions in a manner consistent with the UN protocol—with one important exception. Mercury emissions from power plants are not addressed in the protocol and are unregulated in the United States, although legislation and regulation are pending. The EU also has not regulated mercury emissions from power plants.

Regulatory approaches to mercury in water differ. Japan has established safe levels of mercury in lakes and streams and has set effluent standards. The U.S. Environmental Protection Agency has set water-quality criteria in terms of the mercury concentration in fish tissue. It is notable that the U.S. standard of 0.3 parts per million (ppm) is more stringent than the World Health Organization and the Food and Agriculture Organization's recommendations of 0.5 ppm.[4] The United States and the EU have set standards for mercury in the effluent of a broad range of industrial sources.

The EU is taking steps to adopt the same consumption reference dose as the United States, defining levels of mercury exposure thought to be safe. Japan's Ministry of Health and Welfare has set its own less stringent value.

1. United Nations Economic Commission for Europe, "Protocol to the 1979 Convention on Long-Range Transboundary Air Pollution on Heavy Metals," Annex V (1998), accessible via http://www.unece.org/env/lrtap/protocol/98hm_a/ annex5.htm.

2. See Summary of European Council Directive 96/62/EC (27 September 1996), Official Journal L 296, 21.11.1996, accessible via http://europa.eu.int/scadplus/leg/en/lvb/l28031a.htm.

3. European Commission, Working Group on Mercury, *Ambient Air Pollution by Mercury: Position Paper* (Luxembourg: European Communities, 2001): page ES 2-7, accessible via http://europa.eu.int/comm/environment/air/background.htm#mercury.

4. World Health Organization and Food and Agriculture Organization, "Guideline Levels for Methylmercury in Fish," CAC/GL 7-1991, accessible via http://www.who.int/fsf/Codexreview/methylmercury.htm.

utilities' mercury emissions by 90 percent, or 39 t, by 2007.[74] The Bush administration has advocated a 70-percent, or 30-t, cut in utilities' mercury emissions by the year 2018 as part of its Clear Skies Initiative to cut power plant emissions.[75] Notably, the Clear Skies proposal includes a cut of 20 t by 2008. Although progress on such legislation currently is slow because of disagreement about whether to cap utilities' carbon emissions as well, bipartisan agreement to cap mercury emissions suggests that some legislative action is likely. At the same time, EPA has begun proceedings to regulate mercury emissions from power plants as a hazardous air pollutant under the Clean Air Act.

Legislation might allow companies to trade mercury emissions permits. EPA's sulfur dioxide–emissions permit trading program has been so successful in achieving greater-than-expected emissions cuts at lower-than-expected costs that emissions permit trading has become a point of departure in most environmental policy discussions. Although trading would minimize the costs of controlling emissions, it is still controversial. Objections center around concern for "hotspots"—the chance that some companies facing high control costs might choose not to reduce emissions, thereby contributing to elevated mercury concentrations in watersheds immediately downwind. In that case, trading could be detrimental to certain areas.

Weighing Costs and Benefits

The costs of controlling emissions will bring higher electric bills to consumers. Researchers can clarify the nature of this tradeoff to inform policy makers and the public about the merits of different emissions caps and trading arrangements. EPA sets maximum achievable control technology (MACT) standards based solely on the availability and affordability of technology to control emissions, irrespective of the magnitude of resulting improvements to the environment or public health. EPA has estimated that implementing MACT requirements of the Clean Air Act might cost $1.1 billion to $1.7 billion per year.[76] The lower of these cost estimates corresponds to reductions from new mercury-specific controls and pre-existing controls for other pollutants that together cut emissions by between 60 and 95 percent. For the upper-bound cost estimate, aggregate mercury emissions reductions range from 80 to 95 percent.[77] However, EPA does not estimate how much of these reductions would come from new MACT controls.[78]

The cost of controlling mercury emissions from power plants depends critically on the controls already in place for emissions of other pollutants, including sulfur dioxide, nitrogen oxides, and particulate matter. Because mercury occurs in emissions in a variety of forms, effective controls must encompass processes that oxidize it into soluble forms, then absorb it into porous solids, and finally remove it from emissions flue gas. Particulate matter control devices such as electrostatic precipitators and fabric filters can perform this final capture of absorbed mercury particles, as can wet and dry sulfur dioxide scrubbers. EPA estimates that wet scrubbers generally capture more than 90 percent of divalent mercury emissions, which may account for 20 to 80 percent of total mercury emissions.[79] Control technologies for nitrogen oxides also may enhance control of mercury emissions.[80] EPA suggests that selective catalytic reactors and scrubbers intended to control other pollutants have the potential to reduce mercury emissions by 70 percent—at virtually no cost.[81]

EPA's analysis suggests that reducing mercury emissions from 43 t to 13 t per year under the Clear Skies Initiative will cost less than $975 million per year.[82] However, deep cuts in mercury emissions from U.S. utilities are likely to reduce exposure to mercury among U.S. residents only slightly because other factors affect their exposure. Only some mercury deposition in the United States is from U.S. anthropogenic sources, and only some U.S. anthropogenic emis-

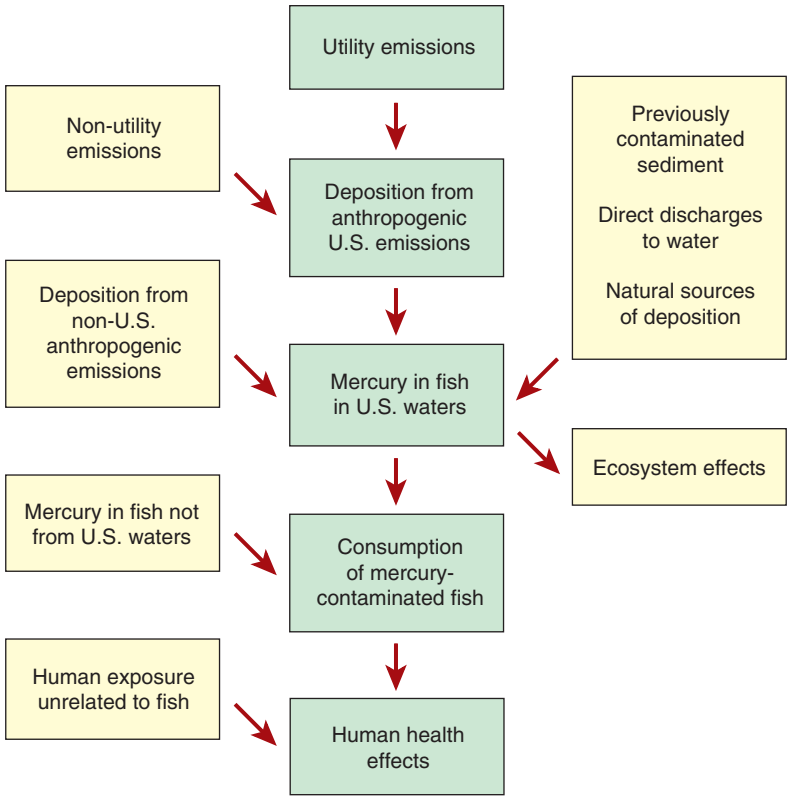
sions are from utilities. Figure 4 below illustrates how sources of mercury are linked to human health effects in the United States. EPA estimated that about three-fifths of mercury deposited in the United States in 1994–95 was emitted from U.S. anthropogenic sources and that the rest came from global or nonanthropogenic sources.[83] The pending regulations discussed earlier would significantly reduce U.S. anthropogenic emissions below these levels.

Another factor to consider in efforts to limit exposure is that, despite important new research about how mercury enters the food chain, there is no accepted quantitative estimate of the relationship between mercury deposition and mercury concentrations in fish.[84] To the authors' knowledge, there is no quantification of such a link even for particular types of lakes. Although statistical studies have suggested that acidity, prevalence of wetlands, and mercury in the water column are associated with mercury concentrations in fish, to date they have not shown an association between mercury deposition and measures of mercury in fish.[85] An innovative ongoing study in Canada that deliberately adds trace amounts of mercury to the area surrounding a small lake might elucidate this link, but its broader applicability to other ecosystems is unclear.[86]

Finally, deposition in the United States affects only some of the fish consumed by U.S. residents. Mercury levels in marine fish from distant waters and in farmed fish that eat commercial feed are not affected by changes in U.S. emissions. Even mercury levels in seafood caught near the United States may be relatively insensitive to local deposition because of the complexity and geographic extent of marine food chains.

Some evidence suggests that most exposure to mercury, even among populations with high fish intake, comes from consumption of seafood caught in offshore waters where reductions in U.S. mercury emissions would have negligible impact.[87] In New Jersey, for example, only 4 to 5 percent of all fish eaten is obtained recreationally, and only about 13 percent of that amount is freshwater fish, according to one study.[88] (Ocean fish may be less likely to be affected by mercury from U.S. sources.) The study found that the two most commonly consumed types of seafood were tuna and shrimp, which together accounted for one-third of seafood consumption; no other category accounted for more than 12 percent.[89] Yet these fish are predominantly from waters unaffected by U.S. emissions. More than 97 percent of all tuna marketed in the United States is caught on the high seas or imported from other countries; only about 3 percent of all shrimp marketed in the United States is caught within 200 miles of the Atlantic states that are downwind from utility plants.[90] There is no assessment of how much of the mercury exposure among highly exposed individuals comes from fish sensitive to U.S. deposition.

Figure 4. Mercury emissions and human health



NOTE: The sections in green mark the chain connecting utility emissions of mercury to human health.

SOURCE: R. Lutter and E. Irwin.

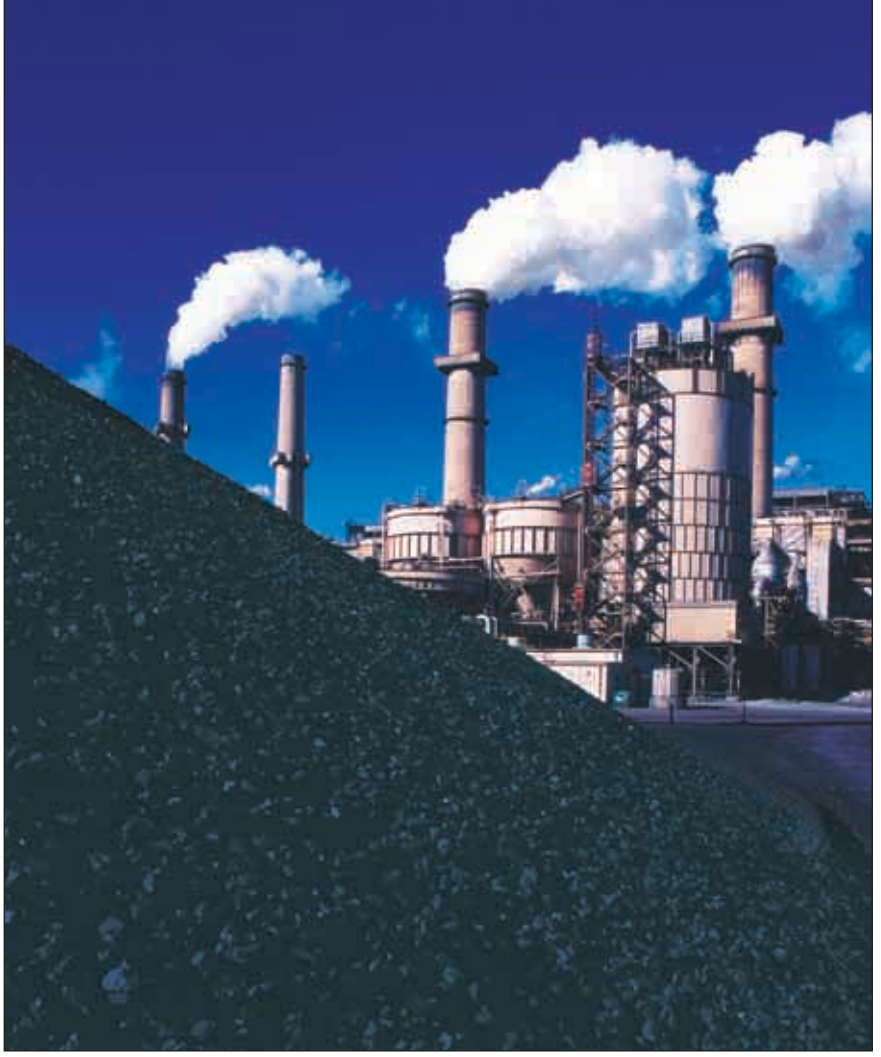
Sensible Approaches to Mitigating Risk

The uncertain and incomplete scientific understanding about how mercury emissions harm public health and ecosystems handicaps government attempts at sensible regulation. Although scientific uncertainty is ubiquitous in environmental policymaking, there is a serious lack of knowledge about mercury, despite some insightful new research. The available data suggest that sharp cuts in mercury emissions from U.S. power plants will not result in large environmental and public health gains in comparison with gains from other high-priority environmental initiatives.

Based on survey evidence linking mercury in women's hair to their fish consumption and the exposure-response relationships from epidemiological studies, the hypothetical complete elimination of mercury from fish would benefit only a relatively small number of children. Approximately 6,000 children would experience improvements in specific, narrow measures of neurological performance (between 13 percent and 22 percent of a standard deviation)—a small change.[91] Larger numbers of children would experience even smaller improvements, assuming that the exposure-response relationships are valid at lower levels of exposure. Even very sharp cuts in U.S. power plants' mercury emissions would provide much more modest benefits because they would affect only one of many sources of mercury to which U.S. women are exposed.

Measuring these health gains against those from other environmental initiatives requires a difficult apples-to-oranges comparison, because the value of such changes in neurological performance has not been quantified. Nonetheless, the scope of health benefits due to reduced mercury emissions appears smaller than that of benefits from proposed cuts in sulfur dioxide and nitrogen oxides, which EPA estimates would prevent more than 10,000 deaths annually from respiratory and other ailments.[92]

These potential benefits also appear small when considering the costs of EPA's pending technology-based mercury regulations—from $1.1 billion to $1.7 billion annually. On a per-child basis, the costs of MACT standards would be on the order of $10,000, conservatively including all affected children and completely ignoring the limited effectiveness of cuts in U.S. utility emissions in reducing exposure to mercury in fish. While such comparisons sometimes suggest unsettling tradeoffs between corporate profits and children's health, such suggestions are dubious here. The health effects are not changes in the health of identifiable children but are small differences in the risk that all children face. Moreover, utilities pass on to consumers the costs associated with emissions controls. Thus the tradeoff is really between the cost of reductions for collective U.S. consumers and the potential for subtle health improvements.



Coal-fired power plants are the largest unregulated source of U.S. anthropogenic mercury emissions. Multipollutant controls might prove effective in reducing mercury.

Multipollutant approaches such as the Clear Skies Initiative and Senator Jeffords's proposal could reduce mercury more cost-effectively than mercury regulations alone because new controls on nitrogen oxides and sulfur dioxide provide "free" reductions in mercury emissions. However, the authors are unaware of estimates of the incremental cost of caps on mercury emissions, assuming cuts in nitrogen oxides and sulfur dioxide consistent with Clear Skies, Jeffords's proposal, or other legislative initiatives. Thus it is premature to assess the economic merit of the mercury cuts in these proposals.

Economics and risk assessment can and should play a prominent role in public debate about the merit of environmental initiatives, although Congress and EPA consider many other factors in developing new legislation. The challenge to the research community is to improve understanding of the merit of mercury controls quickly enough to help Congress and EPA develop sensible limits for mercury emissions.

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NOTES

1. National Research Council (NRC), Committee on the Toxicological Effects of Methylmercury, Board on Environmental Studies and Toxicology, Commis-

sion on Life Sciences, *Toxicological Effects of MeHg* (Washington, D.C.: National Academy Press, 2000).

2. U.S. Environmental Protection Agency (EPA), "Mercury Update: Impact on Fish Advisories," EPA-823-F-01-011 (Washington, D.C.: EPA Office of Water, 2001), available at http://www.epa.gov/ost/fishadvice/mercupd.pdf.

3. NRC, note 1 above, page 11. This report concluded that EPA's reference dose for methylmercury (0.1 microgram per kilogram body weight per day), which is more stringent than the U.S. Food and Drug Administration's (FDA) action level of 1 part per million (ppm), is "a scientifically justifiable level for the protection of public health." Publication of the report generated scrutiny of the FDA standard. See L. Neergaard, "FDA Says Mercury in Some Fish Could Harm Babies' Brains," *Detroit News*, 13 January 2001, available at http://detnews.com/2001/health/0101/13/health-175462.htm.

4. See EPA, "Regulatory Finding on the Emissions of Hazardous Air Pollutants from Electric Utility Steam Generating Units," *Federal Register* 65, no. 245 (20 December 2000): 79,825–31.

5. EPA must issue a final rule by December 2004 (EPA, note 2 above). See also EPA, "Fact Sheet: Clear Skies Initiative," at http://www.epa.gov/clearskies/clearskiesfactsheet_3_26.pdf, accessed 19 August 2002.

6. See The White House, Executive Order 12866, *Regulatory Planning and Review*, 30 September 1993, which is still used by the Bush administration.

7. EPA, *Mercury Study Report to Congress*, EPA-452/R-97-003 (Washington, D.C., 1997), vol. 1, p. 2-1. This report (volumes 1–8) is accessible via http://www.epa.gov/oar/mercury.html.

8. EPA and Environment Canada, "Draft Report: Mercury Sources and Regulations" (Washington, D.C.: EPA, 1999), available at http://www.epa.gov/grtlakes/bns/mercury/stephg.html.

9. EPA, note 7 above, vol. 1, page 0-1.

10. Ibid.

11. Ibid., vol. 2, page ES-2; and L. Poissant, M. Amyot, M. Pilote, and D. Lean, "Mercury Water-Air Exchange over the Upper St. Lawrence River and Lake Ontario," *Environmental Science & Technology* 34 (2000): 3,069–78.

12. EPA, note 7 above, vol. 2, Table ES-3.

13. EPA, "Standards of Performance for New Stationary Sources and Emission Guidelines for Existing Sources: Hospital/Medical/Infectious Waste Incinerators: Final Rule," *Federal Register* 62, no. 178 (15 September 1997); and EPA, "Large Municipal Waste Combustion Units: Emission Guidelines: Final Rule," *Federal Register* 62, no. 164 (25 August 1997).

14. Potential mercury reductions from these regulations are taken from EPA, *Deposition of Air Pollutants to the Great Waters Third Report to Congress 2000* (Washington, D.C., 2000): Table III-2.

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16. Ibid.

17. EPA, note 7 above, vol. 1, page 2-13.

18. Ibid., page 2-5.

19. Ibid.

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23. C. T. Zaneski, "Toxin Taking Toll on Wildlife in the Everglades," *Miami Herald*, 22 August 1999, 1A.

24. USGS, "Mercury in the Geochemical Cycle and Food Chain of the Everglades," *Mercury Studies in the Florida Everglades* (1999): 1, accessed via http://sofia.usgs.gov/publications/fs/166-96/printfood.html on 23 July 2002. See also S. Ullrich, T. W. Tanton, and S. A. Abdrashitova, "Mercury in the Aquatic Environment: A Review of Factors Affecting Methylation," *Critical Reviews in Environmental Science and Technology* 31, no. 3 (2001): 241–93.

25. EPA, note 7 above, vol. 1, page 3-18.

26. USGS, note 24 above.

27. EPA, *The National Survey of Mercury Concentrations in Fish: Data Base Summary 1990–1995*, EPA-823-R-99-014 (Washington, D.C., 1999): page 4-7.

28. EPA, note 7 above, vol. 3, page ES-6.

29. See, for example, ibid., page 2-6.

30. EPA, note 14 above, page 2-15.

31. EPA, note 27 above, page 1-1.

32. EPA, note 27 above.

33. EPA, "Water Quality Criteria: Notice of Availability of Water Quality Criterion for the Protection of Human Health: Methylmercury," *Federal Register* 66, no. 5 (8 January 2001): 1,344.

34. EPA, note 7 above.

35. Ibid., vol. 1, page 2-4.

36. M. S. Landis, "Assessing the Atmospheric Deposition of Mercury to Lake Michigan: The Importance of the Chicago/Gary Urban Area on Wet and Dry Deposition" (Ph.D. diss., University of Michigan, 1998).

37. U.S. Department of Commerce, National Oceanic and Atmospheric Administration (NOAA), Air Resources Laboratory, "Mercury Deposition Network" (NRSP-3), National Atmospheric Deposition Program web site (2000), accessed via http://nadp.sws.uiuc.edu/mdn/ on 23 July 2002.

38. EPA, note 7 above, vols. 2 and 3, page 4-2.

39. Ibid., vol. 3, page 2-13.

40. Center for Food Safety and Applied Nutrition, FDA; EPA; National Energy Technology Laboratory, Department of Energy (DOE); National Marine Fisheries Laboratory, NOAA; and National Center for Health Statistics, National Center for Environmental Health, Centers for Disease Control (CDC), "Blood and Hair Mercury Levels in Young Children and Women of Childbearing Age—United States, 1999," *Mortality and Morbidity Weekly Report* 50, no. 8 (2001): 140–43.

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42. FDA, EPA, DOE, NOAA, and CDC, note 40 above, page 140.

43. K. Crump et al., "Benchmark Concentrations for MeHg Obtained from the Seychelles Child Development Study," *Environmental Health Perspectives* 108, no. 3 (2000): 257–63; G. Myers et al., "Secondary Analysis from the Seychelles Child Development Study: The Child Behavior Checklist," *Environmental Research* 84, no. 1 (2000): 12–19; and C. Axtell et al., "Association between MeHg Exposure from Fish Consumption and Child Development at Five and a Half Years of Age in the Seychelles Child Development Study: An Evaluation of Nonlinear Relationships," *Environmental Research* 84, no. 2 (2000): 71–80.

44. P. Grandjean et al., "Cognitive Deficit in Seven-Year-Old Children with Prenatal Exposure to MeHg," *Neurotoxicology and Teratology* 19, no. 6 (1997): 417–28.

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46. NRC, note 1 above, page 6.

47. Ibid., page 325. A letter from Professor Robert A. Goyer, who chaired the NRC Committee on the Toxicological Effects of Methylmercury, to Joseph A. Levitt, director of the Center for Food Safety and Applied Nutrition of FDA, more fully explains the meaning of the NRC statement (R. A. Goyer, letter to J. A. Levitt, Washington D.C., 2000).

48. Goyer, ibid.

49. Ibid. The reference dose is defined as "an estimate of a daily exposure to the human population (including sensitive subpopulations) that is likely to be without a risk of adverse effects when experienced over a lifetime" (NRC, note 1 above, page 2).

50. Goyer, note 47 above.

51. J. Smith, R. Von Burg, and P. Allen, "Hair MeHg Levels in U.S. Women," *Archives of Environmental Health* 52, no. 6 (1997): 476–80. See Table 1.

52. U.S. Department of Commerce and NOAA, note 37 above, Table 91.

53. Crump, Kjellstrom, Shipp, Silvers, and Stewart, note 45 above.

54. P. Grandjean et al., "MeHg Exposure Biomarkers as Indicators of Neurotoxicity in Children Aged Seven Years," *American Journal of Epidemiology* 150, no. 3 (1999): 301–05. This study reports the estimated declines in various performance tests such as finger-tapping speed for a doubling of mercury concentrations. This information is used here to calculate improvements in health for the specified reduction in mercury exposure.

55. J. Salonen 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* 91, no. 3 (1995): Table 4.

56. Salonen et al. derive this estimate in a model that controls for age, ischemic exercise electrocardiogram, family history of coronary heart disease, cigarette-years, mean systolic blood pressure, socioeconomic status, and place of residence (urban vs. rural), among other factors (ibid.).

57. Some of these concerns first appeared in a review commissioned by NRC. See W. Rosamond and C. Poole, "A Critique of Salonen 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,'" prepared for NRC Committee on the Toxicological Effects of Methylmercury, Washington, D.C. (undated).

58. E. Guallar et al., "Mercury Levels in Toenails and Risk of Myocardial Infarction: Interim Results of an International Case-Control Study" (paper presented at the Seventh Nordic Symposium on Trace Elements in Human Health and Disease, Espoo, Finland, June 1999). This study published interim results from an international case-control study that appears to support the findings of Salonen et al. See also N. Sorensen, K. Murata, E. Budtz-Jørgensen, P. Weihe, and P. Grandjean, "Prenatal MeHg Exposure as a Cardiovascular Risk Factor at Seven Years of Age," *Epidemiology* 10, no. 4 (1999): 370–75; and J. Salonen, K. Seppanen, R. Salonen, and G. A. Kaplan, "Mercury Accumulation and Accelerated Progression of Carotid Atherosclerosis: A Population-Based Prospective Four-Year Follow-Up Study in Men in Eastern Finland," *Atherosclerosis* 148, no. 2 (2000): 265–73. The Sorensen study reports an association between mercury in maternal hair and blood pressure in seven-year-old children but do not address the clinical significance and permanence of this effect—or the nature of the relationship at relatively low doses (for example, 1 ppm to 5 ppm). The Salonen study reports an association between mercury accumulation and accelerated progression of carotid atherosclerosis among Finnish men. Although they report statistically significant associations between mercury hair content and atherosclerosis, they fail to assess the proportion of mercury that is methylmercury and therefore attributable to fish consumption.

59. This condition was one of Sir Bradford Hill's (of the London School of Hygiene and Tropical Medicine) criteria for inferring that epidemiological associations are causal (Rosamond and Poole, note 57 above).

60. See Y. Kinjo et al., "Cancer Mortality in Minamata Disease Patients Exposed to Methylmercury through Fish Diet," *Journal of Epidemiology* 6, no. 3 (1996): 134–38.

61. A. H. Stern, L. Korn, and B. Ruppel, "Estimation of Fish Consumption and MeHg Intake in the New Jersey Population," *Journal of Exposure Analysis and Environmental Epidemiology* 6, no. 4 (1996): 503–27; and A. H. Stern, M. Golchfeld, C. Weisel, and J. Burger, "Mercury and MeHg Exposure in the New Jersey Pregnant Population," *Archives of Environmental Health* 56, no. 1 (2001): 4–10. Note that these articles discuss two different surveys of New Jersey women.

62. Salonen et al., note 55 above, Table 2.

63. J. R. Sauer, J. E. Hines, I. Thomas, J. Fallon, and G. Gough, *The North American Breeding Bird Survey, Results and Analysis 1966–1999, Version 98.1* (Laurel, Md.: USGS Patuxent Wildlife Research Center, 2000), accessed via http://www.mbr-pwrc.usgs.gov/bbs/bbs99.html.

64. For osprey populations, the survey-wide trend is 6.7 percent, and for double-crested cormorants, the survey-wide trend is 7 percent (ibid.).

65. Sauer, Hines, Thomas, Fallon, and Gough, note 63 above; see trend maps.

66. EPA, note 7 above, vol. 5, page 3-3.

67. "Panthers: 80, Rising," *National Geographic Magazine*, August 2002.

68. S. Duvall and M. Barron, "A Screening Level Probabilistic Risk Assessment of Mercury in Florida Everglades Food Webs," *Ecotoxicology and Environmental Safety* 47 (2000): 298–305.

69. EPA, *Mercury White Paper* (1997). See http://www.epa.gov/oar/merwhite.html.

70. National Conference of State Legislatures, *Environmental Health Legislation Database*, accessed via http://www.ncsl.org/programs/ESNR/cehdb.htm on 23 July 2002.

71. Washington State Department of Ecology, "Draft—Washington State Mercury Chemical Action Plan Background Information," Publication No. 02-03-016 (2002), accessed via http://www.ecy.wa.gov/pubs/0203016.pdf on 15 May 2002; and Minnesota Pollution Control Agency, *Progress Report to the Minnesota Legislature* (2002), accessed via http://www.pca.state.mn.us/hot/legislature/reports/2002/mercury-02.pdf on 15 May 2002.

72. EPA, note 33 above, page 1,346.

73. EPA, note 27 above, Figure 4-2.

74. S. 556, 107th Cong., 1st sess., *Congressional Record* 147 (15 March 2001).

75. The White House, "President Bush Announces Clear Skies and Global Change Initiatives," 14 February 2002, accessible via http://www.epa.gov/clearskies/clear_skies_factsheet.pdf.

76. ICF Consulting, "Mercury Control Cost Calculations: Assumptions, Approach, and Results, EPA Contract No. 68-D7-0081, Task 011, Subtask 02" (30 September 2000). These are 1999 dollars.

77. Ibid, pages 8–9.

78. An EPA memo suggests that current controls may eliminate 43 percent of generated mercury emissions (F. Princiotta, "Control of Mercury Emissions from Coal-Fired Utility Boilers," memo from the director of the Air Pollution Prevention and Control Division to John Seitz, director of the Office of Air Quality Planning and Standards (MD-10), EPA, 25 October 2000, 6).

79. EPA, note 4 above, page 79,828.

80. Ibid.

81. See the comments of Peter Tsirigotis of EPA on 19 February 2002 at the AEI-Brookings Joint Center conference "Regulating Air Pollutants from Power Plants: What is Sensible?"

82. This upper-bound estimate of compliance costs of the mercury cap for the Clear Skies proposal is derived by conservatively assuming that EPA's estimate of the marginal cost is also the average cost, which the agency did not report. Multiplying the 30-metric ton (t) reduction in mercury emissions (from 43 t to 13 t) by the permit price estimate for the 13-t cap in 2018 ($14,460 per pound) gives $972 million. See EPA, "Clear Skies Initiative," Regional Summary Report (2002), accessed via the Clean Air Markets—Programs and Regulations page at http://www.epa.gov/airmarkets/epa-ipm/results.html#downloadresults on 23 July 2002 (download ipm2000s153d_c).

83. EPA, note 7 above, vol. 1, page 3-14.

84. J. A. Sorensen, G. E. Glass, K. W. Schmidt, J. K. Huber, and G. R. Rapp Jr., "Airborne Mercury Deposition and Watershed Characteristics in Relation to Mercury Concentrations in Water, Sediments, Plankton, and Fish of Eighty Northern Minnesota Lakes," *Environmental Science & Technology* 24, no. 11 (1990): 1,716–27; R. P. Mason, J. R. Reinfelder, and F. M. Morel, "Uptake, Toxicity, and Trophic Transfer of Mercury in a Coastal Diatom," *Environmental Science & Technology* 30 (1996): 6; and EPA, note 7 above. Sorenson provides an early and interesting article; Mason and EPA provide summaries. See also R. Renner, "Newly Deposited Mercury May Be More Bioavailable," *Environmental Science & Technology* 36, no. 11 (2002): 226A–27A. EPA has found only a "plausible link" between mercury deposition and concentrations in fish. See EPA, note 7 above, vol. 1, page 0-2; and W. Fitzgerald, D. Engstrom, R. Mason, and E. Nater, "The Case for Atmospheric Mercury Contamination in Remote Areas," *Environmental Science & Technology* 32, no. 1 (1997): 1–7.

85. W. Brumbaugh, D. Krabbenhoft, D. Helsel, J. Wiener, and K. Echols, "A National Pilot Study of Mercury Contamination of Aquatic Ecosytems along Multiple Gradients: Bioaccumulation in Fish," *Biological Science Report*, USGS/BRD/BSR-2001-0009 (USGS, September 2001). See also Qian, Warren-Hicks, Keating, Moore, and Teed, note 22 above.

86. See the Electric Power Research Institute's Mercury Experiment to Assess Atmospheric Loading in Canada and the United States (METAALICUS) project summary at http://www.epri.com/corporate/products_services/project_opps/environment/1006515.pdf, accessed on 16 August 2002.

87. EPA, note 7 above, vol. 4, page 4-37.

88. Stern, Korn, and Ruppel, note 61 above, page 505.

89. Ibid., page 510. The study indicates that average mercury concentrations were 0.17 ppm for tuna and 0.11 ppm for shrimp.

90. U.S. National Marine Fisheries Service, *Fisheries of the United States, 1999* (Silver Spring, Md.: NOAA and U.S. Department of Commerce, 2000). See the NOAA Fisheries and National Marine Fisheries Service web site at http://www.nmfs.noaa.gov. This percentage is calculated as the total catch within 200 miles of New England and the southern Atlantic states divided by the sum of all landed shrimp and net imports.

91. The authors' calculations used data from the Grandjean and Crump studies.

92. EPA, "Human Health and Environmental Effects of the Clear Skies Initiative," report prepared for the EPA Clear Skies Workshop, 19 June 2002.