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Methylmercury, Fish Consumption, and the Precautionary Principle

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ABSTRACT

This paper considers several broad issues in the context of probabilistic assessment of the benefits of curtailing mercury (Hg) emissions from U.S. coal-fired power plants, based on information developed from recent literature and epidemiology studies of health effects of methylmercury. Exposure of the U.S. population is considered on the national scale, in large part because of recent questions arising from survey and experimental data about the relative importance of local deposition of airborne Hg. Although epidemiological studies have provided useful information, safe levels of Hg exposure remain uncertain, in part because of other dietary considerations in the populations that were studied. For example, much of the seafood consumed in one of the major studies was also contaminated with polychlorinated biphenyls, as are fish taken from some U.S. fresh waters. The primary epidemiological approach involves cross-study comparisons in relation to mean exposures, rather than detailed critiques of individual effects reported in each study. U.S. exposures are seen to be well below the levels at which adverse health effects are reported. This analysis supports the conclusion that unilateral reduction of Hg emissions from U.S. coal-fired power plants alone is unlikely to realize significant public health benefits.

IMPLICATIONS

Early risk assessments were based largely on the benefits of reducing local Hg deposition in U.S. freshwaters, as estimated from atmospheric modeling studies. However, local sources contribute a small fraction of total deposition, freshwater fish are only a small fraction of the fish consumed, and these fish often contain other contaminants, including polychlorinated biphenyls and lead. Although the "precautionary principle" is invoked to justify action in the face of uncertainty, responsible environmental policy requires that emission control programs be effective in terms of the health and other benefits actually realized. Global reductions in Hg will be required to accomplish this.

INTRODUCTION

Methylmercury (MeHg) is a known neurotoxicant, now virtually ubiquitous in seafood at various concentrations,[1] that originates from atmospheric compounds of inorganic Hg emitted from both natural and anthropogenic sources. These compounds are deposited in water bodies and their watersheds, where they are converted to organic forms through microbial reactions. Like some other air pollutants, the adverse health effects of mercury (Hg) first came to public attention through several unfortunate accidents in prior decades, in this case involving industrial wastewater discharges in Japan[2] and consumption of bread made from contaminated seed grain during a famine in Iraq.[3]

The U.S. Environmental Protection Agency (EPA) set its original guideline daily MeHg exposure limit (the reference dose [RfD]) at 0.3 μg/kg of body weight based on the Iraqi data. More recently, the EPA reduced the RfD to 0.1 μg/kg/day based on data from a study in the Faeroe Islands and recommendations from a National Academy of Sciences (NAS) Committee.[1] The RfD is defined as the daily exposure that is "likely to be without a risk of adverse effects when experienced over a lifetime."[1] It is

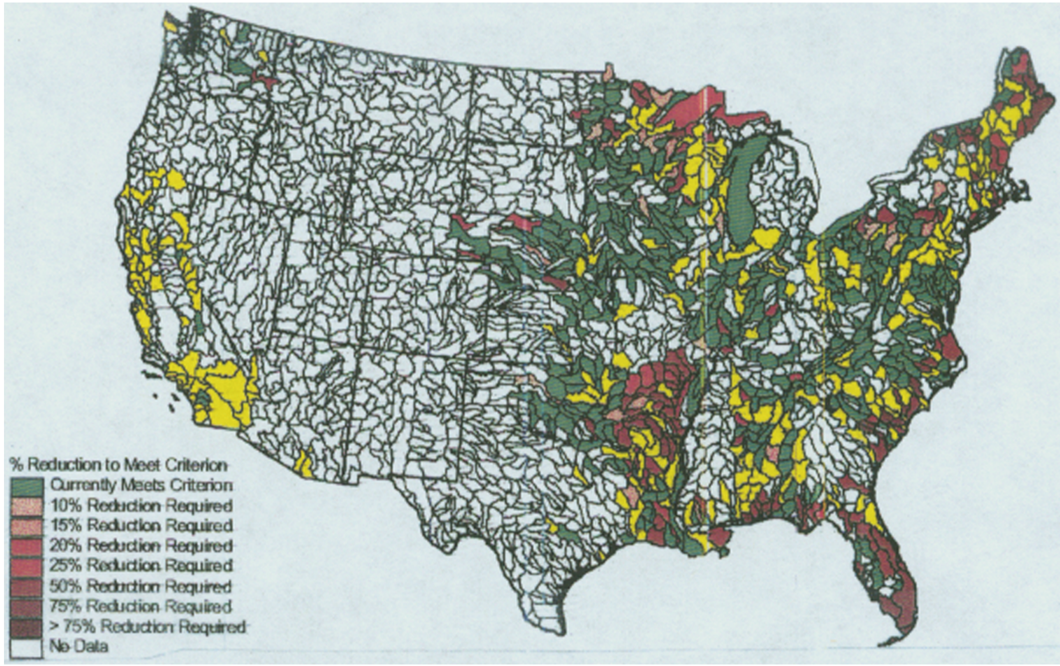

Figure 1. Percentage reduction in Hg deposition needed to meet the EPA's maximum MeHg fish concentration criterion for watersheds with no other significant Hg sources (from ref 7). Watersheds highlighted in yellow have significant nondeposition Hg sources, defined as total Hg loads >5% of estimated waterbody delivery at a deposition of 10 $\mu g/m^2/yr$. Additional reductions may be required to meet most state fish advisory levels.

frequently based on the most sensitive indicator of response found in the most critical study, after consideration of uncertainties. For MeHg, the EPA reduced the RfD by a factor of 10 to allow for uncertainties. Thus, the RfD level of exposure does not necessarily represent an experimentally determined threshold for the onset of adverse health effects.

In this paper, we use the usual elements of risk analysis: risk characterization (is there a problem?), risk analysis (how serious is it likely to be?), and risk management (what can be done about it?). We use recent survey data to estimate exposures to the U.S. population, including those who consume self-caught fish, and contrast these exposures with those of the various populations that have been considered in epidemiology studies. We then evaluate the likely outcome of reducing Hg emissions from coal-fired power plants in the context of the "precautionary principle".

RISK CHARACTERIZATION

Relative Importance of U.S. Sources

The risks from U.S. sources of atmospheric mercury, such as coal-fired power plants, depend directly on their relative contributions to atmospheric mercury levels and deposition. To date, such contributions have been considered on two different spatial scales: local/regional and national/global.

Local/Regional Hg Deposition. On the basis of atmospheric dispersion models, local/regional deposition has been estimated to result primarily from emissions of reactive forms of gaseous Hg (RGM), including compounds such as $HgCl_2$, which are then either washed out in precipitation or dry-deposited,[4-6] usually within ~50 km of the plant. However, recent empirical evidence does not support the hypothesis that coal-fired power plants represent a major source of Hg deposition on local or statewide scales:

1. Using the data reported in the EPA "Mercury Maps" project,[7] we found no excess (e.g., average Hg >0.3 ppm) Hg in fish in the Appalachian region, where the density of U.S. coal-fired power plants is high (Figure 1).
2. Measurements in a coal-fired power plant plume found reduction of reactive Hg rather than oxidation of elemental Hg,[8] suggesting a diminished role for RGM.
3. A study of wet deposition in Connecticut[9] found excesses in urban areas. Specific urban sources were not identified, but incinerators were suspected. A recent study[10] reported that 46% of the Hg in urban stormwater runoff was attributable to deposition on rooftops, which also suggests the existence of urban Hg sources.
4. No significant relationship is apparent between Hg concentrations in freshwater fish and measured

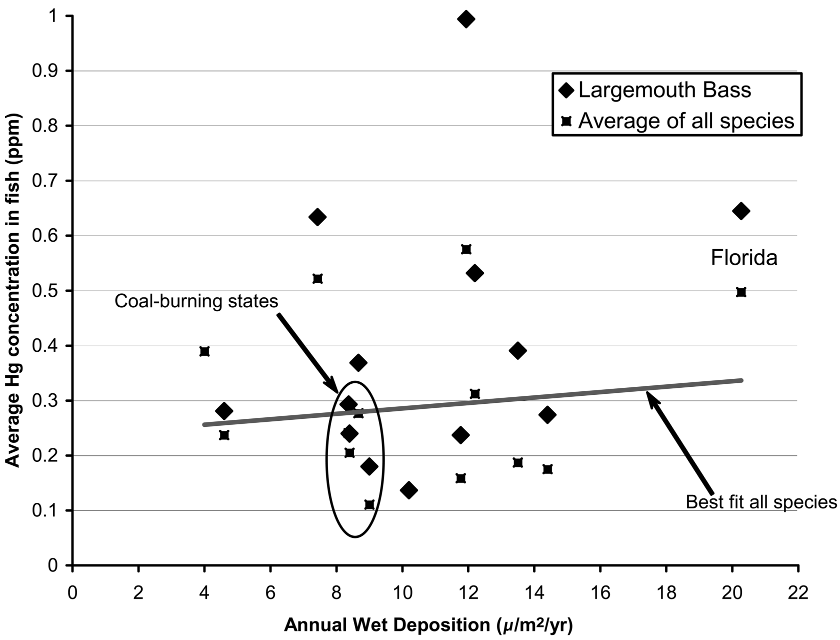

Figure 2. Statewide average Hg concentrations in fish vs. estimated statewide average wet deposition of Hg.

wet Hg deposition data from the Mercury Deposition Network (MDN)[11] (Figure 2). Furthermore, the coal-burning states (Pennsylvania, Illinois, Wisconsin, and Minnesota) that have the data needed for this plot have among the lowest fish Hg concentrations and deposition rates. Although the relationship between fish Hg and deposition is weakly positive, it is not statistically significant ($r = 0.28$ for bass [$p = 0.35$]; $r = 0.14$ for the average of all species [$p = 0.6$]). This suggests that the Hg in freshwater fish is governed mainly by factors other than current rates of wet deposition, such as aqueous chemistry, dry deposition in and subsequent releases from watersheds, or releases from aquatic sediments.

Data on Average Hg Deposition Rates in the United States. To assess the relative importance of U.S. power plants, we compared estimates of their annual Hg emissions with the total annual Hg deposition estimated for the country as a whole, without regard to detailed location. Such total deposition estimates have not appeared in the literature, except for the EPA's initial estimate of 10 μg/m^2/yr.[6] The MDN[11] provides data from 1998 to the present on wet deposition in selected states. Using interpolated data from MDN data maps, we estimated a national average (area-weighted) value of 9.2 μg/m^2/yr for wet deposition alone. Dry deposition of Hg depends on the mercury species present (elemental [Hg^0], RGM, or particulate); the types of surfaces receiving the deposition may also have an effect. Dry deposition of particulate Hg is difficult to measure and poorly quantified; it is often assumed to be negligible. Dry deposition rates for Hg^0 are much lower than for RGM and may reflect either deposition or re-emission.[12]

Various techniques have been used to measure total Hg deposition, including mass balances around defined catchments,[13,14] peat bog cores,[15] deposits on vegetation and measurements of throughfall under a forest canopy,[16] and use of moss as a deposition collector.[17] These techniques have provided reasonably self-consistent estimates of total annual Hg deposition in the range 54–72 μg/m^2/yr, implying large contributions from dry deposition. A dry deposition flux of 16 μg/m^2/yr was estimated for RGM in the Florida Everglades[18] compared with a wet deposition of 20–23 μg/m^2/yr, and even larger contributions from Hg^0 were estimated in Tennessee.[16] A range of 20–30 μg/m^2/yr total Hg deposition was estimated for Onondaga Lake, near Syracuse, NY.[19]

An alternative method of estimating dry deposition of an air pollutant involves separate consideration of air concentration and dry deposition velocity (v_d). Typical v_d values for RGM and Hg^0 are ~2 and 0.02–0.09 cm/sec, respectively. Lindberg and Stratton[16] also estimated that ambient RGM tends to average ~3% of ambient Hg^0. Combining these estimates with a typical average ambient concentration of Hg^0 in the United States (1.7 ng/m^2) results in dry deposition flux estimates of 32 μg/m^2/yr for RGM and 27 μg/m^2/yr for Hg^0. However, there is considerable uncertainty about the net flux of Hg^0 because of the likelihood of re-emission.

Taking into account all of the information, we consider 20 μg/m^2/yr to be a reasonable lower estimate for total Hg deposition to the United States as a whole and 40 μg/m^2/yr to be a reasonable upper estimate. Multiplying by the surface area of the contiguous United States (7.8×10^6 km^2) yields an estimated range in total Hg deposition of 156–312 metric tons (t)/yr.

Accounting for U.S. Hg Emissions. Anthropogenic emissions of Hg were recently estimated at 176 t/yr for the 48 contiguous states.[20] Of that total, 43 t/yr were attributed to power plants, with another 14 t/yr attributed to industrial combustion, 45 t/yr to municipal waste combustion, and 23 t/yr to smelting processes. Because modeling results indicate that only about one-half of the Hg emitted from the United States will deposit there,[12] sources other than power plants appear to contribute between 134 and 291 mt/yr of the total Hg deposition. For example, Hanisch[21] estimated that China contributes ~1000 mt/yr to the global total, some of which likely deposits in the United States; because of the forecasted increases in Chinese coal usage, this contribution is likely to grow in the future. It may also be possible that the U.S. contribution to global Hg is larger than 176 mt/yr because of uncertainties in the inventory and the possibility of unreported urban sources. In any event, the net reduction in Hg deposited within the Unites States that might be realized by controlling U.S power plants is seen to be a small fraction (~5–10%) of the total Hg deposited annually in the conterminous United States (affecting freshwater fish) and an even smaller fraction (~1%) on a global basis (affecting marine species).

Estimating the Effects of Deposition on the Hg Content of Freshwater Fish. Assuming that the (nonsignificant) slope of Figure 2 is appropriate for all fish species, reducing Hg deposition by 5 or 10% would reduce the average Hg content of freshwater fish by only ~0.9–1.8% (~0.003 ppm). This estimate is much smaller than that obtained by assuming a linear relationship between Hg deposition and fish Hg content (~40%[4,5]), which illustrates the large uncertainty involved in assessing the potential benefits from reducing local Hg emissions.

Fish Consumption and MeHg Exposure

Fish Consumption Rates in the United States. Because MeHg is found almost exclusively in fish and shell fish, seafood consumption rates give an overall measure of population exposures to MeHg. Data from the U.S. Statistical Abstract indicate that total seafood consumption rates per capita have been stable over the past decade, at ~19–24 g/day. The contribution of commercial freshwater fishing is much smaller, even on a regional basis. For example, assuming that one-half of the Michigan freshwater catch is edible and consumed in Michigan, total per capita consumption there would be increased by only ~0.8 g/day.

Assuming that everyone eats at least some fish and an average portion size of 140 g, the above total national rate of consumption would average out to one meal per week, or ~182 g/portion if only 77% of the population ever eats fish. These rates are consistent with a previous analysis.[4,5] However, although a recent survey indicated that 87.5% of the U.S. population eats fish at least once over the course of a year,[22] older data[23] suggest that in the early 1970s, 40–50% of the population reported eating fish "seldom or never", which would increase the effective rate of consumption by "users" to ~27 g/day at that time. These estimates are also reasonably consistent with those of Stern et al.[24] for New Jersey; their data indicate a mean portion size of 168 g and a median consumption rate of ~1.3 meals/week, yielding a median per capita consumption rate of ~30 g/day. Other comparisons of recent data on fish consumption rates include 19–24 g/day in Greece,[25] <27 g/day in Native American freshwater fishers,[26] and an assumed value of 32 g/day for a study in the lower Mississippi River area,[27] but substantially higher values in Hong Kong (~150 g/day).[28]

Average Exposures to MeHg. At a consumption-weighted average MeHg concentration in fish of 0.15 ppm,[22] a consumption rate of 19 g/day, and a median body weight of 71.4 kg,[4,5] the corresponding population-average MeHg exposure rate would be ~0.039 μg/kg/day, which is below the EPA-recommended limit of 0.1 μg/kg/day. This estimate is also consistent with the data of Stern et al. (median = 0.043 μg/kg/day)[24] and those of Dougherty et al.[29] for total Hg in all food sources (0.04–0.08 μg/kg/day, depending on how values below the minimum detection limit are handled). However, persons eating more than three fish meals per week (11% of the New Jersey population[24]) or with a diet limited to fish having substantially higher Hg content would be likely to exceed the EPA guideline. Carrington and Bolger[22] indicated that, nationwide, only ~7% of consumers are likely to exceed three fish meals per week. More recent data collected in the National Health and Nutrition Examination Survey (NHANES) IV study suggest that 8% of the population is above the EPA reference dose based on blood biomarkers that reflect all exposures to Hg.[30]

Recent survey data on consumption of home-produced foods[31] indicate that only ~2% of the U.S. population consumes home-caught fish, with a median consumption rate of 1.1 g/kg/day for the child-bearing ages of 20–39. If this rate of consumption is consistent with that of all fish consumers, there would be no need to treat consumers of home-caught fish as a separate special case, which would greatly simplify the risk analysis. Testing this hypothesis requires estimates of the distribution of fish consumption for the general public.

Previous research[4,5] indicates that this distribution is log normal with a geometric SD of 2.5. Thus, if the median intake is 0.27 g/kg/day, the 95th percentile would be ~1.2 g/kg/day and the 99th percentile would be ~2.3 g/kg/day. In Table 1, we compare the distributions of fish consumption for the general public with that for self-caught fish.[31] For this purpose, our working hypothesis was that the distribution of consumption of self-caught fish, which comprises 2% of that sample, corresponds to the upper 2% of the distribution of all fish eaters. If this is true, the median consumption rate for self-caught fish

Table 1. Distributions of fish consumption rates and biomarkers for MeHg exposure.

	Fish Consumption (g/kg/day)		Blood Hg (μg/L)		Hair Hg (ppm)	
Percentile	All Consumers[a]	Self-Caught Fish[b]	NHANES[c]	High End[d]	All[e]	High End[d]
50	0.27		0.93		0.31	
95	1.2		7.1			
96.5	1.4		8.1	11.2		3.7
98.5	1.95	0.64	13.6			
99	2.2	1.3	16.6		3.4	
99.8	3.7	4.7	32	22	<6.3	<15

[a]Based on a geometric SD of 2.5; [b]Ages 20–39 years,[31] including 0.2 g/kg/day for commercial fish (n = 59; 2% of the sample); [c]National sample of 1709 women;[30] [d]Sample of 89 high-end consumers;[36] [e]Ref 34.

should correspond to the 99th percentile for all fish consumers (100% − 2%/2), and the other points of the distribution of self-caught fish should correspond similarly, as shown in Table 1. To make this comparison, we assumed that consumers of self-caught fish also consume commercial fish at the national median rate. As seen in Table 1, these two distributions corresponded reasonably, given that only 59 consumers of self-caught fish were included in the sample. The distributions of Hg biomarker data are discussed below.

Stern et al.[24] surveyed the consumption of self-caught fish in New Jersey; 4–5% of fish meals were from noncommercial sources, but only 13% of these were freshwater species, ~0.5% all meals. It is likely that the freshwater portion of self-caught fish might be higher in the more interior sections of the United States; however, because the average Hg contents of marine and freshwater species are roughly similar, that distinction is not critical for the purposes of this assessment.

Exposures Indicated by Biomarkers

Data on individual MeHg concentrations in blood and hair permit population exposures to be estimated much more accurately, compared with conventional air pollutants that rely on complex mathematical modeling to infer individual exposures. Sources of such biomarker data have increased in recent years, notably with the inclusion of Hg measurements in the NHANES.[30,32] NHANES IV[30] included ~2400 samples of blood MeHg from 1999 and 2000, in male and female children and in females up to age 49. The median total Hg concentration in the blood of children up to age 6 was 0.23 μg/L (i.e., ppb), with a 95th percentile value of 2.3 μg/L, similar to the previous NHANES estimates;[32] the new values for women, however, are significantly lower than reported earlier. The median value for females weighted by the distribution of births by age in 2000[33] was 0.93 μg/L. This estimate is ~20% lower than that implied by the ~1982 national estimate[34] of hair Hg of ~0.31 ppm for 2820 women of child-bearing age, based on a hair/blood ratio of 270. This suggests a decreasing trend in U.S. population exposures, which would be consistent with the decreasing trend in deposition seen in ice-core samples[35] and in fish concentrations.[4,5]

For an example of a subset of the population that might be heavily exposed to MeHg, we turn to the data of Hightower and Moore,[36] comprising 89 patients (66 females) who tested high for blood Hg, presumably because of habitually frequent consumption of predatory fish such as swordfish. They reported an average of 16 fish meals per month, or a mean consumption rate of 1.42 g/kg/day, which places the mean of this group at the 96.5 percentile of the whole population. According to our working hypothesis, this subset should thus correspond to the upper 7% of the national population. In Table 1, we compare their blood Hg levels with the NHANES data for females with the available data on the high-end consumers; the comparison is reasonable, given the small sample at the high end, suggesting that such highly exposed, specially selected groups may also be considered as a part of the general public. In addition, the 99th percentile (3.4 ppm) in the distribution of hair Hg reported by Smith et al.[34] is consistent with the hair value (3.7 ppm) derived from the high-end consumers, for whom the maximum hair Hg was 15 ppm.

In summary, when combined, these disparate survey data provide a reasonably consistent picture of MeHg exposure for the entire U.S. population, although the responsible sources of MeHg have not been identified with certainty. For example, according to Smith et al.[34], only approximately one third of the MeHg in hair could be attributed to seafood consumption. This ratio is also consistent with the NHANES data, based on blood Hg levels in 429 women who denied eating seafood during the 30 days before examination (~2 ppm).

RISK ANALYSIS

Decisions about the extent of risks for the U.S. population involve comparing these biomarker levels with those of populations in which health effects have been reported; such levels vary considerably. Some exposure data from major published fish-based epidemiology studies of the effects of prenatal exposures to MeHg[37–50] are shown in Table 2 Mean or median contaminant levels are shown under the simplifying assumption that frequency distributions are likely to be similar in most studies. Although

Table 2. Exposures to fish contaminants in recent major epidemiological studies.

			MeHg			PCBs				
Reference	**Location**	**Subjects**	**Hair**	**Blood**	**Cord**	**Cord**	**Blood**	**Milk**	**Blood Pb**	**Effects**
Jacobson and Jacobson[37]	Lake Michigan	212 mothers and children	1 (child; age 11)			3	6	841	6	High PCBs = lower IQ; some effects of Pb and Hg
Lonky et al.[38]	Lake Ontario	559 mothers and babies				(Grouped by implied PCB exposure)				Those with high PCBs scored worst
Stewart et al.[39]	Lake Ontario	293 mothers and children	0.50	0.53						Those with heavy Cl PCBs were worst; no effect of Hg or Pb
Stewart et al.[40]	Lake Ontario	212 children	0.50	0.52	153				1.7	Effects of PCBs; interaction with Hg
Darvill et al.[41]	Lake Ontario	247 mothers and children	0.50	0.53	153				1.7	High PCBs = lower score; no effect of MeHg
Schantz et al.[42]	Michigan	572 adults who ate fish	2–4		17				3.9	High PCBs = lower recall
		419 adults who ate no fish	<2		6				3.5	No effect of Hg or Pb
Marsh et al.[43]	Peru	131 mothers and infants	7 (mothers)							No effects
Kjellstrom et al.[44]	New Zealand	73 mothers and infants	10 (mothers)						5.6	Lower IQ for highest Hg exposures
Davidson et al.[45] and Myers et al.[46]	Seychelles	711 mothers and children	6.8 (mothers); 6.5 (child, age 6)			Not detectable in blood			2	No effects
Grandjean et al.[47]	Faeroe Islands	435 mothers and children	4		26ppb	1.9				Neurological effects; possible interaction between PCBs and Hg
Steuerwald et al.[48]	Faeroe Islands	182 births	4		20ppb	1.1	1520			Hg = low neonatal neurological function
Weihe et al.[49]	Greenland	43 mothers and children	16 (mothers); 5 (children)							Hg effects not significant
Murata et al.[50]	Madeira	149 mothers and children	9.6 (mothers); 3.8 (children)							Effects when maternal hair Hg > 10 ppm

Note: Hair Hg levels are in ppm, blood Hg levels are in μg/L, milk PCB levels are in ng/g of lipids, blood Pb is in μg/dL.

there are questions about many of these studies in terms of exposure measurements and interpretation of the various types of neurological endpoints that have been used, a "macro" examination across the studies in Table 2[34] may be informative. Some key facts from the major studies are as follows:

1. No adverse effects have been found in the Seychelles,[45,46] where the children are healthy (and even precocious), diets are balanced, and levels of polychlorinated biphenyl (PCB) and Pb exposure are low. Median Hg levels in hair there are quite high compared with those in the United States (Table 1) but similar to those found in Hong Kong.[28]
2. Pilot whale, which contains high levels of contaminants, particularly PCBs, is a major feature of the Faeroese diet.[51]

3. In the Michigan studies,[42] Hg was low, Pb and PCBs were higher, and adverse effects were found.
4. In the recent Lake Ontario study,[40] PCB concentrations in umbilical cord blood were statistically significant predictors of reduced cognitive development, and an interaction was seen between Hg and PCB exposures, but hair Hg (median = 0.5 ppm) was not a significant predictor in its own right.
5. Average lead levels were slightly elevated in New Zealand children.[44] In addition, it is important to note that these 73 children were selected from a much larger cohort (~10,000) on the basis of high exposures.
6. In all other locations, maternal Hg levels exceeded those in the Seychelles, and adverse effects were reported. Unfortunately, concentrations of all other contaminants were often not reported.
7. The NAS review[1] apparently did not consider the Great Lakes studies.[37–42]

Determination of Thresholds

Finding a dose–response threshold is often difficult for a weak effect because random noise tends to obscure any departures from linearity. Such difficulties are compounded when the measure of exposure (such as hair Hg) is a surrogate for the actual agent (brain Hg).[52] In this study, we tried to deduce the existence of a threshold on a population rather than an individual level, on the basis of the absence of statistically significant (linear) responses. The data in Table 2 suggest that a population threshold for Hg effects may exist around the mean level of the Seychelles cohort, i.e., ~7 ppm in hair, which is similar to a previous WHO advisory level.[44] According to the analysis of Carrington and Bolger,[22] only ~0.2% of U.S. females 16–49 of age would exceed this threshold. By contrast, ~8% of the female population exceeds a hair level of 1.1 ppm, which corresponds to the EPA's reference dose for MeHg. The findings of adverse effects in the Faeroe Islands and in Michigan are likely to be the result of other contaminants (PCBs and Pb), perhaps in combination with Hg.[53,54] Note that it is not necessary for a contaminant other than Hg to be a confounder (i.e., correlated with Hg) to have an effect on the outcome of an epidemiological study. Given the likely presence of a threshold for neurological effects, the other contaminants could reinforce the effects of Hg by acting as an effect modifier. It is more difficult to establish threshold levels for PCBs or Pb relative to normal levels, but Hoover[55] cited a value of 238 ng/g for PCBs in Canadian breast milk and Pirkle et al.[56] found a mean Pb level of 2.3 μg/dL for U.S. blood samples. Many of the known exposure levels in Table 2 exceed these reference values, especially the PCBs in the Faeroese population.[57]

Summary of Risks

An important conclusion from Table 2 is that MeHg is not likely to be the only contaminant in seafood in many situations; careful measurements of all exposures will be required to partition any adverse effects. For now, the Great Lakes studies may provide the best indications of effects in the United States, and the mean Hg levels in those studies (0.5–1 ppm in hair) were well above the most recent U.S. estimates (median = 0.2 ppm).[32] For example, using an average hair/intake ratio of 18,[52] the EPA's intake guideline (RfD) of 0.1 μg/kg/day translates to a blood concentration of 5.8 μg/L (1.8 ppm in hair). According to the NHANES exposure data,[30] 8% of U.S. females exceed this guideline. Furthermore, the WHO guideline of 6 ppm in hair corresponds to an Hg intake of 0.3 μg/kg/day (the previous EPA RfD); based on the most recent NHANES blood data, the MeHg exposures of ~99.5% of the U.S. population would be below this limit, as well as ~75% of those eating home-caught fish.

In characterizing risks, it is important to distinguish between a public health advisory level, such as the EPA RfD, and exposure levels that actually relate to observed risks (Table 2). To ensure a margin of safety, public health advisory levels are deliberately set well below any observed thresholds. As a result, predictions of risk or harm based on such advisory levels may be misleading. This caveat certainly applies to the NAS prediction of large numbers of American children at risk for "adverse neurodevelopmental effects (see p. 327 in reference 1).

It is also important to characterize the nature of such neurodevelopmental effects. For example, frank manifestations of mercury poisoning, such as paresthesia or loss of coordination, have never been an issue at these low exposure levels. Instead, the types of effects seen in some studies of children may include delays in first walking or talking of a few days, intelligence quotient (IQ) deficits of a few points, or small deficits in performance on tests of memory or dexterity. Furthermore, it has also been shown that such deficits in young children may not be permanent.[40]

RISK MANAGEMENT

From the outset, it hardly seems necessary to point out that removing fish from the U.S. diet is not an option. The ultimate goal must be to make fish safe to eat because seafood consumption confers many other health benefits. Risk management must therefore consider the best ways to reach this goal, which is quite separate from the traditional concerns of environmental regulation per se. Also, unwarranted concerns about low levels of fish contaminants could prove to be counterproductive to public health.

The Precautionary Principle

Given the highly toxic nature of mercury at sufficiently high doses, one might ask, "Why not eliminate all consumer uses of Hg and prohibit any and all industrial discharges, regardless of any scientific or risk assessment considerations?" This evokes the "precautionary principle", which states: "When an activity raises threats of harm to human health or the environment, precautionary measures should be taken even whether some cause and effect relationships are not fully established scientifically." However, a main tenet of any public health action should be, "First, do no harm."[58]

Four central components of the precautionary principle are (1) taking preventive action in the face of uncertainty, (2) shifting the burden of proof to the proponents of an activity, (3) exploring a wide range of alternatives to possible harmful actions, and (4) increasing public participation in decision-making.[59] Public health practitioners also use the term "primary prevention" in this context. Goldstein[60–62] has written extensively on this subject and points out that the EPA is not a public health agency in that punitive actions sometimes tend to be taken without regard to unintended consequences.[60]

In contrast, the primary public health approach is that of minimization of risk, in this case, neurological damage to the fetus from any of a number of possible toxic agents, MeHg being only one. Kriebel and Tickner[63] point out that "Precaution is relevant to public health, because it can help to prevent unintended consequences of well-intentioned public health interventions by ensuring a more through assessment of the problems and proposed solutions." Some possible unintended consequences (i.e., the "doing of harm") of large-scale removal of Hg from coal-fired power plant flue gas may include the following:

1. Increased electricity costs, with effects throughout the economy, including making air conditioning less affordable to those who may need it for health reasons.
2. Diminution of efforts to abate other sources of Hg in the environment (as listed above).
3. Conversion of the Hg removed from flue gas from very dilute levels spread over a wide area to highly concentrated levels that must be disposed of as solid waste, at a substantial cost.
4. Creating a false sense of security with respect to the safety of freshwater fish.

Other actions designed to remove Hg from the environment and their unintended consequences include

1. unwarranted fears of toxicity from amalgam dental fillings containing Hg (unnecessary removal)[64];
2. banning of Hg manometers for blood pressure measurement in hospitals, clinics, and physicians' offices (the replacement electronic devices tend to be less accurate);[65] and
3. banning of thimerosal, a preservative containing Hg, from childhood vaccines (increased costs),[64,66]

Unintended consequences could also result from the U.S. Department of Defense's reported intention to dump its mercury stockpile on the open market, which could further reduce Hg prices and end up in environmentally unfriendly uses such as gold mining.[67] Brazil's 1989 emission of Hg from gold mining was estimated to be 168 t, which is roughly equivalent to U.S. emissions from all sources. At a price of \$12.00/g and a 1:1 ratio of mercury to gold, it seems to be far cheaper to pay gold miners not to mine gold than to remove Hg from flue gases, which has been estimated to cost 3–4 times higher.[68]

Will Mercury Controls on U.S. Coal-Fired Power Plants Make the Fish Supply Safe?

The unequivocal answer is "No," but the question must be considered on two levels. First, we assumed that the EPA MeHg intake guideline of 0.1 μg/kg/day must be met to provide a large margin of safety, as discussed above. For example, if 70% of the Hg in coal-fired power plant flue gas were removed and permanently sequestered, U.S. emissions would drop by 30 t/yr, with a total Hg deposition reduction of 5–10%, depending on plant location, mercury speciation in the flue gas, and the rates of plume dilution. As seen from Figure 2, this would translate into smaller reductions in MeHg intake, but only from freshwater fish (deposition modeling suggests that increased deposition would occur only within 50–100 km of the source; therefore, marine fish are affected mainly by global Hg emissions). Lacking data on commercial freshwater fish consumption, we relied on the figures for home-caught fish, for which the median intake level was ~0.15–0.21 μg/kg/day. As seen in Figure 2, this range would be reduced by only 2% to ~0.147–0.208 μg/kg/day, changes that are clearly in the noise level but that are expected to require around \$1.4 billion of emission controls. Typical avoided health effects are subtle neurological effects, such as delays in first walking or talking of a few days.[69] However, health impacts would occur for only very few babies.

The second level of consideration involves contaminants other than MeHg in seafood, including PCBs, Pb, Cd, and pesticides. The small reductions in MeHg would impact only an extremely small fraction of the population, because the maximum MeHg blood level in the

NHANES data was above the implied threshold for adverse effects for ~0.04% of the sample population. The discrepancy between the estimate of 0.04% above the threshold and the fact that 8% are above the EPA exposure guideline of 0.1 μg/kg/day is that the EPA applied an "uncertainty factor" of 10 to the risk threshold in deriving its exposure guideline. Even if we apply the reduction of a few percentages in mercury deposition to the population group above the EPA threshold, it would only minimally reduce the number of people above the threshold. The question remains, however, as to other contaminants in fish that might be harmful to the developing fetus. Overemphasizing MeHg could result in overlooking such other contaminants, particularly in freshwater species; many states have fish advisories for PCBs, and farmed salmon have higher PCB content than wild salmon.[70] Published data on other contaminants in fish and seafood include

1. Pb, Cd, Hg, and Se in fish, birds, and mammals in Greenland;[71]
2. pesticides, PCBs, polycyclic aromatic hydrocarbons, and Hg in both farmed and wild salmon;[70]
3. Hg and Se in mollusks, crustaceans, and fish;[72]
4. high blood Pb levels in Canadian consumers of sportfish;[73]
5. PCBs in Lake Michigan fish;[74]
6. PCBs, chlorinated pesticides, Se, and Hg in Quebec Inuit;[75]
7. Pb and Hg in the Arctic;[76,77] and
8. Pb, Cd, PCBs, and 1,1,1-trichloro-2,2-bis(*p*-chlorophenyl)ethane (DDT) in consumers of Great Lakes fish.[78]

Finally, the ability to distinguish the separate effects of colocated contaminants in epidemiology depends on the relative accuracy of exposure measurement;[52] statistical "transfer of causality" can occur with correlated pollutants differing in exposure reliability. Thus, the true causal agents are not likely to be identified until all contaminants are measured with equivalent care. This may have been the problem with the Faeroe Island study,[47] because only a few of the many PCB congeners were studied. Budtz-Jorgensen et al.[79] noted that the effects of MeHg in the Faeroese cohort "could be reduced to nonsignificance by assuming a large measurement error for the PCB biomarker."

CONCLUSIONS

The precautionary principle requires that a wide range of policy alternatives be considered in terms of the public health good or harm that might ensue. The current program to reduce U.S. mercury emissions should be given this same degree of scrutiny. Several elements of potential harm were listed above; elements of potential good (i.e., goals) may be examined as follows:

1. If the goal were to improve infant health in the United States, imposition of Hg controls on power plants is likely to be far less cost-effective than direct expenditures on public health, such as better access to prenatal care or programs to reduce maternal smoking and alcohol use. For example, ~17% of the 4 million U.S. births in 2001 received no prenatal care during the first trimester;[33] the annual cost of Hg controls could provide approximately \$2000 for each of those births (approximately \$100 for each biweekly visit to a clinic).
2. If the goal were to improve seafood safety, the money would be better spent on more comprehensive inspection of seafood because only a small fraction of the catch is likely to have elevated contaminant levels. Because 30% of U.S. MeHg intake comes from canned tuna, eliminating the highest tuna concentrations through factory inspections could have a substantial impact. For example, based on U.S. Food and Drug Administration data,[22] eliminating the highest 10% from the distribution of Hg in tuna would reduce the mean U.S. MeHg intake by 6.4%; for the highest 20% of tuna, the dietary benefit in reduced MeHg would be 11%. This would appear to be a much more cost-effective measure than attempting to reduce global Hg emissions by 11%, which would require a reduction of ~400 tons/yr (~10 times the U.S. power plant contribution). The power plant control costs discussed above would correspond to approximately \$0.60 per 6-oz can consumed, which could conceivably cover a thorough Hg inspection program. Such inspections should include a wide range of possible contaminants, not just MeHg. Safe freshwater fish will not be attained until all contaminants, notably PCBs, are abated.
3. If the goal were to reduce levels of airborne Hg for its own sake and regardless of any local benefits, international trends in Hg emissions must be considered, especially those from gold mining and developing countries in which substantial future growth in coal use might be expected, such as China. It might be more cost-effective to provide technical assistance in such situations than to control domestic emissions from selected source categories.

None of these perspectives provides unequivocal support for unilaterally reducing Hg emissions from U.S. coal-fired power plants. Recent epidemiological studies suggest that only subtle neurological effects are involved and that they are associated only with prenatal maternal exposures. Expectant mothers and women contemplating pregnancy are now counseled to stop smoking and to avoid alcohol; it thus seems prudent to add a list of problematic fish species to the list of proscriptions until the absence of all potentially harmful contaminants may be assured.

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