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Cover Photo:
A nesting loon covers her eggs. Methylmercury concentrations in loons and other fish-eating birds are reaching toxic levels in some parts of the world, which is impairing reproduction and may lead to population declines in some species.

Photo: M. Woodford.

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The growing interest in the sustainable use of natural resources and a general awareness of global change issues has led to increased interest in environmental research and a need for published information on the subjects involved. AMBIO aims to serve the important function of putting into perspective significant developments in environmental research, policy and related activities, and to reach specialists, generalists, students, decision-makers and interested laymen around the world with this information.

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Beginning with this issue of *Ambio* and the publishing year 2007, and continuing over the coming three (3) years, *Ambio*'s publishing position will be taking a huge step forward. To this end we have increased our cooperation with Allen Press, Lawrence, Kansas, USA, by entering a services agreement with the Alliance Communication Group (ACG) publishing division of Allen Press. The decision to make this move was largely influenced by our growing need to provide faster and more effective services to our rapidly growing numbers of authors and readers. We also needed to establish a simpler means to ensure rapid dialogue between authors, editors, and reviewers by a more efficient use of the Allentrack system. We expect this to result from this new cooperation.

The Royal Swedish Academy of Sciences will, of course, retain full ownership of the journal and our publishing policies will in no way change. Our strategy as always is to continue to be available for our authors and readers, to improve our visibility in both print and online, and to be available even for those readers who do not have purchasing power of their own. This latter is made possible by the generous financial aid provided by the Swedish International Development Agency (SIDA).

New developments in environmental science are making it necessary to weld science and societies more closely together. In order to lend credibility to the introduction of, for example, new climate policies, broad information strategies will be essential. An informed society is a prepared society. *Ambio* will continue to be one small, but effective piece of the general information puzzle.

Elisabeth Kessler
Editor-in-Chief

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Preface to the Madison Declaration and Critical Synthesis Papers on Mercury Pollution

Eighth International Conference on Mercury as a Global Pollutant Madison, Wisconsin, USA

6–11 August 2006

The International Conference on Mercury as a Global Pollutant, first convened in 1990 in Gävle, Sweden, has become a premier international forum for the presentation and discussion of advances in scientific understanding of environmental mercury pollution. In view of recent scientific advances and the ongoing consideration of policy options for addressing the mercury problem, the Eighth International Conference in Madison, Wisconsin, USA, on 6–11 August 2006 presented a timely opportunity to address key questions concerning mercury in the environment. The technical program for the conference contained a diverse and multidisciplinary assemblage of 1047 abstracts submitted by authors from 58 countries, with technical presentations spanning the global scientific effort on mercury pollution. The critical synthesis of existing scientific information into forms useful to both the scientific and policy communities was a key goal of the conference, which was hosted jointly by the University of Wisconsin–Madison, the US Geological Survey, and the University of Wisconsin–La Crosse.

Forty international experts, assembled into four panels, began this synthesis effort at a workshop in July 2005, a full year before the conference. The panels addressed a series of key policy-relevant questions concerning atmospheric sources of mercury, methylmercury exposure and its effects on humans and wildlife, socioeconomic consequences of mercury pollution, and recovery of mercury-contaminated fisheries. The findings of the panels are formally reported in this issue of *Ambio* as a series of five critical synthesis papers.

Collectively, the five synthesis papers provided the foundation for the Madison Declaration on Mercury Pollution, a summary document that was endorsed unanimously by members of the four expert panels. Each of the 1150 registered conferees assembled in Madison was provided a copy of the declaration, and each had access to peer-reviewed conference drafts of the five critical synthesis papers. Each panel presented its findings in a plenary session at the conference, and videos of these sessions remain available for viewing at the conference web site, www.mercury2006.org.

In Madison, each conferee was provided the opportunity to complete an on-line questionnaire assessing the strength of scientific support for each principal conclusion listed in the declaration. In the declaration that follows on page 63, the percentage of respondents who expressed that the current scientific evidence supports each principal conclusion in the declaration is indicated in parentheses. The majority of conferees who participated in this assessment process expressed agreement with the conclusions listed in the declaration.

The declaration and critical synthesis papers (1–5) are directly pertinent to policy discussions concerning environmental mercury pollution. We hope that these documents will facilitate the application of the best available science to policy on this geographically widespread and persistent environmental problem.

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The Panel on Health Risks and Toxicological Effects of Methylmercury:
Donna Mergler, Henry A. Anderson, Laurie Hing Man Chan, Kathryn R. Mahaffey, Michael Murray, Mineshi Sakamoto and Alan H. Stern

Methylmercury Exposure and Health Effects in Humans: A Worldwide Concern

The paper builds on existing literature, highlighting current understanding and identifying unresolved issues about MeHg exposure, health effects, and risk assessment, and concludes with a consensus statement. Methylmercury is a potent toxin, bioaccumulated and concentrated through the aquatic food chain, placing at risk people, throughout the globe and across the socioeconomic spectrum, who consume predatory fish or for whom fish is a dietary mainstay. Methylmercury developmental neurotoxicity has constituted the basis for risk assessments and public health policies. Despite gaps in our knowledge on new bioindicators of exposure, factors that influence MeHg uptake and toxicity, toxicokinetics, neurologic and cardiovascular effects in adult populations, and the nutritional benefits and risks from the large number of marine and freshwater fish and fish-eating species, the panel concluded that to preserve human health, all efforts need to be made to reduce and eliminate sources of exposure.

INTRODUCTION

The Panel on Health Risks and Toxicological Effects of Methylmercury received the mandate to describe and synthesize current scientific knowledge on methylmercury (MeHg) exposure and its effects in humans and to identify research gaps. The present paper is not intended to be a comprehensive review and presentation of all the literature on MeHg exposure and effects in humans but builds on earlier literature, other reviews, and more recent literature in highlighting the current understanding in the field and what we consider to be remaining unresolved issues. Humans are exposed to different forms of mercury (Hg), and potential health risks from forms other than MeHg can occur, including mercury vapor from dental amalgams, as well as from occupational exposures (e.g., dental offices, chloralkali plants, fluorescent lamp factories, mercury mining) and from artesanal and small-scale gold and silver mining operations (1–5), the present document does not cover these exposures, because the pathways of exposure and effects differ from those for MeHg. Here, we examine issues of MeHg exposure, studies on its health effects and major risk assessments, and conclude with our consensus statement.

MeHg Exposure

Sources of exposure. Methylmercury contamination poses a particular challenge to public health because this toxicant is mainly contained in fish, a highly nutritious food, with known benefits for human health. Moreover, fish are culturally vital for many communities and constitute an important global commodity. Although we often refer to "fish" in a generic way, all fish do not have similar amounts of mercury. As a result of bioaccumulation of MeHg through multiple levels of the aquatic food web, higher tropic-level pelagic fish can be contaminated with MeHg at concentrations in excess of 1 part per million (ppm). The concentrations of total Hg vary widely across fish and shellfish species, with the mean values differing by as much as 100-fold (6). Methylmercury is bound to proteins, as well as to free amino acids, that are components of muscle tissues, and are not removed by any cooking or cleaning processes that do not destroy muscle tissues.

Although in general, MeHg accumulates in fish through the food chain, consumption of farmed fish can also lead to MeHg exposures, in part, because of the presence of MeHg in feed (7). Some studies have shown no significant difference in MeHg levels in farmed *vs.* wild salmon, although concentrations in both cases are relatively low (8, 9). Although fish and shellfish are the predominant sources of MeHg in the diets of humans and wildlife, a few reports of other sources exist. Rice cultivated in areas contaminated with mercury can contain relatively high levels of MeHg (10). Methylmercury has also been reported in organ meats of terrestrial animals (11), as well as in chicken and pork, probably as a result of the use of fish meal as livestock feed (12). Some communities also have higher MeHg exposure because of the consumption of fish-eating marine mammals (13, 14).

Profiles of exposure. Although most reports on MeHg exposure focused on specific populations generally assumed to have high levels of fish consumption, estimates of general populations exposure exist for the United States (15, 16), Germany (17), and Japan (18) [summarized in Pirrone and Mahaffey (19)]. For populations that are not selected on the basis of high fish consumption, mean hair Hg levels generally range from >0.1 $\mu g\ g^{-1}$ to <1.0 $\mu g\ g^{-1}$ (20–25). The mean blood Hg for such populations is generally in the range of <1.0 $\mu g\ L^{-1}$ to <5.0 $\mu g\ L^{-1}$, although, worldwide there are fewer data on MeHg exposure based on blood than on hair. In the United States nationally, about 5–10% of the population of women of childbearing age have hair levels exceeding 1.0 $\mu g\ g^{-1}$ (16) and blood levels exceeding 5 $\mu g\ L^{-1}$ (26). In Japan, where more fish is consumed, 73.7% of women of this age have hair levels above 1.0 $\mu g\ g^{-1}$ and 1.7% above 5 $\mu g\ g^{-1}$ (18). In Germany, the 1998 geometric mean blood level was 0.58 $\mu g\ L^{-1}$ (17).

High levels of Hg exposure were identified in numerous fish-eating populations throughout the world [for reviews see: Pirrone and Mahaffey (19)]. Many of these live near oceans, major lakes and rivers, or hydroelectric dams, and are often dependent on local catch, with fish an integral part of their cultural traditions. In the sea islands of the Faroes and Seychelles, median mothers' hair Hg concentrations were 4.5 $\mu g\ g^{-1}$ [with 27% above 10 $\mu g\ g^{-1}$ (27)] and 5.8 $\mu g\ g^{-1}$ (28), respectively. In the river basins of the Amazon, where a large number of studies was carried out on populations for whom freshwater fish is a dietary mainstay, median hair Hg levels typically range between 5 $\mu g\ g^{-1}$ and 15 $\mu g\ g^{-1}$ (29–34).

Despite the importance of local catch, fish is also a global commodity and market fish, such as shark, tuna, and swordfish, or canned white tuna (35), consumed by persons living far away from the source can likewise have high levels of MeHg. In the United States, individuals with high blood Hg concentrations were reported among affluent urbanites who ate large quantities

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of marine fish, high in the food web (36, 37). Thus, elevated MeHg exposure is present around the globe, with no geographic, social, economic, or cultural boundaries.

Biomarkers of MeHg exposure. Hair and blood Hg concentrations are both accepted as valid biomarkers of MeHg exposure, although each provides a somewhat different reflection of exposure (38). Blood gives an estimate of exposure over the most recent one to two half-lives, with the half-life of MeHg in blood being 50–70 days, whereas hair reflects the average exposure over the growth period of the segment (28). Hair Hg is predominantly MeHg, with MeHg constituting from 80% to 98% of hair total Hg (33, 39). For populations with regular and frequent fish consumption, hair total Hg and blood MeHg are consistently correlated (40). Generally, hair is 250 to 300 times more concentrated in mercury than is blood (39). However, in populations and individuals with infrequent fish consumption or where bolus doses of MeHg occur, there can be considerable inter- and intraindividual variability in the relation between hair and blood Hg levels resulting from temporal differences in the retention of Hg by each biomarker (33, 40, 41). Segmental analyses of hair Hg can provide a chronology of exposure over time (24, 28, 29, 33). However, information on short-term peaks in exposure is not well represented by such analyses (38). Another consideration is that the growth rate of hair, generally estimated at 1 cm mo^{-1}, can have both inter-and intraindividual variability (38). Recent advances in a single hair-strand analysis (42), including measurement of Hg at micron resolution by using laser ablation (43) should yield more information on the relation between Hg uptake and Hg deposition in hair.

The Hg levels in toenails and fingernails also were used as biomarkers of Hg exposure, mostly in major studies of the cardiovascular effect of MeHg (see below) (44, 45), but to what extent these reflect organic or inorganic Hg exposures remains to be clarified (46). A recent study of women, with no history of occupational exposure to Hg, showed similar correlations between Hg intake through fish consumption and both toenail and hair Hg concentrations; however, only total Hg was assessed (47). In this study, hair, toenail, and urinary total Hg were highly correlated. Urinary Hg levels largely reflect exposure to inorganic Hg (40) and are not considered useful bioindicators of MeHg exposure. There are, however, several recent reports of positive correlations between fish consumption and urinary Hg (48–50), and investigators of these studies propose that demethylation may account, at least partially, for this observation. The relation of fish consumption and inorganic Hg in different biological tissues, and its consequence for human health still need to be elucidated.

Health effects from low to moderate levels of MeHg exposure were reported in a variety of systems and domains. Each of these effects may depend on different aspects of exposure [e.g., fish-eating patterns, time of exposure (first, second, or third trimester, childhood, adulthood)]. Therefore, the different reflections of exposure provided by hair and blood Hg concentrations may provide different information about dose-response for different exposure populations and different exposure scenarios. Few studies investigated side-by-side dose-response relations for both biomarkers. In the study in the Faroe Islands, maternal hair and fetal-cord blood predicted similar but not identical patterns of effect across various measures of neurologic performance (38).

Fish Consumption as a Predictor of MeHg Exposure

Exposure dose. Although most studies identified a clear association between the quantity and the frequency of fish consumption and Hg exposure, there is considerable interindividual and intergroup variability in the relation between the amount or the frequency of fish consumption and the levels of biomarker of MeHg exposure. Several factors mediate this relation. The MeHg concentration within and across species of dietary fish is an obvious source of variability. For example, those who eat mainly carnivorous fish and/or fish-eating mammals have relatively higher levels of Hg compared with those who eat mainly noncarnivorous fish (14, 29, 33, 51–54). Independent of the MeHg concentration, the frequency of fish consumption is also an important factor in this variability. Because biomarkers reflect the weighted average of exposure over time, short-term reporting of fish consumption may not correspond with a longer-term average of MeHg exposure. Under some circumstances, episodic exposures can result in large bolus doses of MeHg. Bolus doses can arise, for example, from the infrequent consumption of fish or fish-eating mammals with high concentrations of MeHg. Given practical limitations in sampling frequency, as well as the nature of some of the biomarkers themselves, bolus doses during putative discrete windows of sensitivity in fetal development may not be fully revealed by biomarkers of exposure.

Toxicokinetics. Although most experimental studies on the gastrointestinal absorption of MeHg indicated that nearly 100% of MeHg in fish is absorbed, recently reported animal and human data suggest that there may be substantial variability (55, 56). In animal studies, variation in absorption kinetics was related to factors such as sex and age (57). A further gap exists because human absorption studies were primarily conducted in adult male subjects.

Toxicokinetic (pharmacokinetic) models and physiologically based pharmacokinetic (PBPK) models are applied to estimate internal dose, given a known intake dose, as well as the intake dose, given a measured internal dose (38). The basic one-compartment model (39, 58, 59) is a steady-state model that is intended to predict concentration in a single compartment only (generally, blood). As such, it is less flexible than the PBPK models in predicting nonsteady state conditions and concentrations in other compartments. However, its relative simplicity has allowed it to be used with probabilistic input parameters to obtain estimates of population variability in predictions of blood concentration and intake dose (60). Estimates of concentrations in other compartments (e.g., cord blood) can be made based on empirical ratios relating mercury concentration in blood to mercury concentrations in those compartments (61).

The PBPK models have the potential to predict changes in MeHg concentration in various tissues in response to changes in MeHg intake and in response to physiological changes (e.g., pregnancy, growth). They can be used to predict short-term changes in MeHg concentrations in different compartments during intake and distribution among compartments, if the parameters are correct (62–65).

The validity of these models overall is not thoroughly established under a range of exposures to MeHg by comparison with actual human data. Although they have the theoretical advantage of making predictions under dynamic conditions, the PBPK models are computationally complex and require data on many parameters whose MeHg-specific values have not been defined. This lack of MeHg specific values is a major limitation, particularly for predicting population variability. The extent to which these models rely on coefficients derived from metabolic studies and/or physiological parameters obtained in different populations and subpopulations and studies with other metals/elements, limits their utility. Nonetheless, both simple toxicokinetic models and PBPK models have been used with reasonable consistency for setting public health guidance.

In humans, there is increasing evidence from environmental epidemiology studies of ethnic differences in the relation

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between Hg intake from fish consumption and bioindicators of exposure (56), suggesting that diet and/or metabolic differences may be influencing mercury uptake and/or excretion. As yet, such differences have not been investigated in metabolic studies. Several studies suggest that selenium (Se) may play a role in MeHg absorption or excretion (66–68), but these data are not consistent. In the Brazilian Amazon, fruit consumption was associated with lower hair Hg concentrations (69). A positive relation was reported between iron and Hg in blood samples collected from Sweden (70). Overall, little is known about the factors that may modulate Hg absorption in humans, and research is needed to better understand this complex issue.

Fetal and infant exposure. One area in which the toxicokinetic data is consistent is the finding that MeHg is actively transferred to the fetus across the placenta via neutral amino acid carriers during gestation (71, 72). Although maternal and cord blood Hg concentration is highly correlated, cord blood MeHg is consistently higher than the corresponding maternal concentration, with an average ratio of about 1.7 (24, 61, 73, 74). Consequently, biomonitoring adult women's blood MeHg as a surrogate for potential fetalexposure, the corresponding fetal level will be, on average, 70% higher than maternal blood and up to three times higher at the 95th percentile. The maternal body burden of MeHg tends to decrease during gestation consistent with hemodilution and a transfer of a portion of the maternal body burden to the fetus (24).

Neonatal and infant exposure to MeHg occurs through intake of mother's milk, which is derived from maternal plasma, has a lower level of MeHg, and is enriched in inorganic Hg relative to the whole blood (75). Thus, lactational exposure to MeHg is reduced compared with what would be expected on the basis of maternal blood MeHg. Human and animal studies showed that, after birth, there is a decline in MeHg levels, reaching 40–50% at 2–3 months of age (76–78). During this period, infant body weight increases about 1.5–2 times. Consequently, the rapid increase in body volume and the limited MeHg transfer appear to explain the dilution of MeHg in infants during breast feeding.

HEALTH EFFECTS

Neurological Endpoints

Clinical manifestations. In 1958, McAlpine and Araki (79) linked the unusual neurological disease that was associated with fish consumption from Minamata Bay to MeHg exposure. This historic recognition of the brain and nervous system as the primary target organ for MeHg poisoning, resulting in marked distal sensory disturbances, constriction of visual fields, ataxia, dysarthria, auditory disturbances, and tremor, remains unchanged (80, 81). Based on analysis of the studies of human poisoning, the World Health Organization (WHO) (39) estimated that 5% of MeHg-exposed adults would experience neurologic effects with a blood Hg level of 200 $\mu g\ L^{-1}$ (corresponding to a hair level of approximately 50 $\mu g\ g^{-1}$). This estimate, however, was called into question by a re-analysis of these studies by Kosatsky and Foran (82), who suggested that the lowest observed effect level for clinical effects is likely to be considerably lower. Indeed, anecdotal and case reports of diffuse and subjective neurologic symptoms in adults and older children with moderately elevated MeHg exposures continue to appear (36, 83). In many cases, cessation or significant curtailing of fish consumption results in improvement of symptoms in conjunction with reduction in biomarker concentrations. These suggest the possibility of clinical effects, perhaps in a sensitive subset of the general population, at levels of exposure considerably below those previously associated with clinical effects in poisoning episodes. Currently, there is no formal case description or diagnostic criteria for such effects.

Although exposures throughout the world are lower than those producing the historical epidemics of MeHg poisoning, there is growing evidence that for many populations, exposures are sufficient to alter normal functioning of several systems, constitutes an important public health problem.

Effects in neonates, infants, and children. The poisoning in Minamata brought attention to the risk from fetal exposure. Exposed to MeHg through the placenta of the exposed mother, infants showed severe cerebral palsy-like symptoms, even when their mothers had mild or no manifestation of the poisoning (84). Mental retardation, cerebellar ataxia, primitive reflexes, dysarthria, and hyperkinesias were observed. These symptoms, described over 25 years ago (80, 85), continue as the clinical hallmark of congenital MeHg poisoning. Reconstruction of maternal or fetal doses resulting in these symptoms is difficult because of a lack of concurrent sampling. An estimate of the mean maternal hair concentration, resulting in such symptoms of 41$\mu g\ g^{-1}$ ppm was proposed (86); however, a large uncertainty surrounds this estimate. Health effects observed with frank poisonings should not be confused with the more subtle, populational effects observed at lower levels of exposure.

At the subclinical and the population level, several studies in different parts of the world report poorer neurologic status and slower development in newborns, infants, and/or children exposed to MeHg *in utero* and/or during early childhood (87–98), although some studies did not observe effects (99–101). In children, MeHg exposure *in utero* is associated with lower performance on tests of language, attention, memory, and/or visuospatial and/or motor functions. Although most child studies focused on fish-eating populations with relatively high levels of MeHg exposure, in a recent study, Oken et al (90) observed an inverse relation between mercury concentration in maternal hair and infants' performance on a visual recognition memory task at levels of mercury exposure consistent with background exposure in the US population (maternal hair levels varied between 0.02–2.38 $\mu g\ g^{-1}$). Interestingly, in this study, fish consumption *per se* was associated with better performance, suggesting that some positive aspects of fish consumption, perhaps n-3 (omega-3) fatty acids, are reduced or antagonized by the MeHg contained in the same fish. A similar picture is emerging among adults for the some of the cardiovascular effects of MeHg (see below).

The two major ongoing longitudinal cohort studies on children from the Faroe Islands and the Seychelles are worthy of particular mention because they have both been following children through teenage years, assessing neuropsychological performance as a function of current, childhood, and *in utero* exposure. The Faroes study consistently observed neurobehavioral deficits associated with *in utero* exposure, even when children whose mother's hair Hg levels above 10 $\mu g\ g^{-1}$ were excluded (91). In the initial studies of the Seychelles cohort, no effects were observed (100–103). However, recent reports of the Seychelles 9-year-old cohort shows decreases in fine motor function associated with higher fetal exposure levels ($\geq$10 $\mu g\ g^{-1}$ maternal hair); the investigators suggest that adverse effects may become apparent on higher-order cognitive functions that develop with maturity (104, 105). There has been much discussion about the differences between these two well-performed studies. Factors such as type of exposure (one of the main exposure pathways in the Faroes study is through pilot whale, while in the Seychelles, it is entirely marine fish), biomarkers of exposure (cord blood *vs.* maternal hair), differences in test batteries and age of testing; cohort size and power were considered as possible explanations for the

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differences in observed outcomes. However, none of these explanations proved entirely satisfactory or clearly decisive (38, 106). Other hypotheses, such as dietary intake of nutrients that may modify Hg metabolism or toxicity, were also proposed (69). Despite whatever significant differences do, in fact, exist between the Seychelles and Faroes studies that may explain differences in results that were observed to this time, the most recent results from the Seychelles appear to indicate a convergence in findings. More work needs to be done on factors that may affect the patterns of manifestation of Hg toxicity.

Neurophysiologic studies offer strong support for nervous-system alterations associated with MeHg exposure. These studies showed mercury-related delayed latencies for auditory and visual evoked potentials (107–110). In the Faroes longitudinal study, latency delays were observed at 7 and 14 years (107, 109). No significant dose-effect relations for evoked potentials were observed in a study of Japanese children with low mercury exposure (maternal and children hair mercury levels of 1.6 $\mu g\ g^{-1}$) (111).

Nervous system endpoints in adults. Fewer studies addressed the neurotoxic effects of Hg exposure in adults. Mercury-related deficits in motor, psychomotor, visual and/or cognitive functions have been reported for different populations within the Brazilian Amazon (112–115) and for tuna consumers from the Mediterranean (116). A recent study, in the United States, of older adults (50–70 years old) with considerably lower blood Hg levels (mean, 2.1 $\mu g\ L^{-1}$) showed inconsistent evidence of effect across neurobehavioral tests (117). Studies of associations between neurobehavioral outcomes and MeHg exposure in adult populations in which frequent and lifetime fish consumption is a cultural norm, generally cannot distinguish between effects because of adult exposure and permanent developmental effects because of gestational and early childhood exposures.

Cardiovascular Endpoints

A body of evidence was developed that addresses potential associations between MeHg and a range of cardiovascular effects. These include cardiovascular disease [coronary heart disease, acute myocardial infarction (AMI), ischemic heart disease], blood pressure and hypertension effects, and alterations in heart rate variability [see Chan and Egeland (118) and Stern (119) for recent reviews]. The strongest evidence for causal associations is for cardiovascular disease, particularly AMI in adult men (44, 120–122). In general, the relative risk and the odds ratios for AMI from these studies showed a doubling in the upper range of the observed Hg exposures. Comparison of exposures in these studies to exposures in Western populations suggests that the upper percentiles of current levels of exposure in these populations may result in a significantly elevated risk of AMI. Another well-conducted study of US health professionals, however, did not find an association between Hg exposure and coronary heart disease (123). This may be because dentists with possible exposure to elemental mercury accounted for 63% of controls and had a Hg exposure more than twice that of the other groups in the cohort. It is not known whether elemental or inorganic Hg acts similarly to MeHg with respect to cardiovascular effects. In addition, two of these studies used toenail Hg as the biomarker of exposure. Because this biomarker has not been adequately compared with the more common exposure biomarkers of hair or blood Hg, it is difficult to assess the dose-response implications of these studies in relation to current exposures.

The evidence for an association between MeHg and other cardiovascular endpoints is weaker. An association was found between increased systolic and diastolic blood pressure in Faroese children at 7 years old and gestational exposure to MeHg (124). However, the association did not persist when the cohort was re-examined at 14 years old (125). Decreased heart rate variability was also associated with MeHg exposure, and this effect persisted through 14 years of age, but the implications of this effect in children for clinically significant outcomes is not clear. There are few studies that relate adult blood pressure to MeHg exposure. A recent study in the Brazilian Amazon reported that persons with 10 $\mu g\ g^{-1}$ hair Hg were three times more likely to have elevated systolic blood pressure ($\geq$130 mm Hg) (126), whereas in a study of women from the United States, no clear association was observed (127).

Reproductive Outcomes

The effect of MeHg on the sex ratio of offspring at birth and stillbirth in Minamata City, Japan, in the 1950s and 1960s, including the period when MeHg pollution was most severe, showed decreases in male birth in offspring in the overall city population, among fishing families (72, 128). An increase in the proportion of male stillborn fetuses raises the possibility that increased susceptibility of male fetuses to death *in utero* could explain the altered sex ratio.

Immune System Effects

Inorganic mercury was shown to suppress immune functions and to induce autoimmunity in multiple species (129). Both MeHg and inorganic Hg were shown to produce an autoimmune response, as well as an immunosuppressive effect in several strains of genetically susceptible mice (130, 131). However, data on the immune effects of MeHg in general are sparse, and research is required in this area.

Co-contaminants

Fish tend to accumulate halogenated organics, including polychlorinated biphenyls (PCB), dioxins, and related compounds. The neurodevelopmental effects of PCBs and, to a lesser extent, dioxins, share some similarities to those observed for MeHg (132). This can potentially present difficulties in determining causality and in constructing MeHg-specific dose-response relations. Because MeHg tends to associate more with proteins than with fats, fish species with elevated levels of MeHg are not necessarily those with elevated levels of the lipophilic halogenated organics. Thus, for fish consumption where both exposures occur, the influence of the individual contaminants can potentially be separated by statistical techniques if a variety of fish species is consumed and sufficiently precise exposure metrics are collected. In the Faroe Islands studies, both MeHg and PCBs appear to jointly affect some developmental endpoints. However, although MeHg appeared to enhance the PCB-attributable effects, the PCBs appeared to make a relatively minor contribution to the MeHg-specific effects (132, 133). Contradictory findings were observed in a study of cognitive development associated with exposures to MeHg and PCBs in the Lake Oswego area of New York State (134). In that study, elevated PCB exposure appeared to potentiate MeHg effects. However, both MeHg and PCB levels were considerably lower than in the Faroes study, and no PCB-MeHg association was observed on follow-up testing of the cohort. More work remains to be done on the joint influence of MeHg and halogenated organics, as well as other metal contaminants that may also be present in fish (135).

Elemental Hg continues to be used in dental amalgam for the treatment of dental carries. In populations with significant amalgam use, elemental Hg may account for a proportion of total Hg exposure comparable with or greater than MeHg (38).

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Table 1. Differences in decision choices between the NAS/NRC (2000) and the JECFA (2003) risk assessments for mercury intake.

Variable	NAS/NRC (2000)	JECFA (2003)
Studies	Considered Faroes, New Zealand, Seychelles. Final value based on Faroes	Faroes and Seychelles
Biomarker used as index	Cord blood, $\mu g\ L^{-1}$	Maternal hair [Hg], $\mu g\ g^{-1}$ or ppm.
BMDL selected	58 $\mu g\ L^{-1}$ cord blood	14 $\mu g\ g^{-1}$ maternal hair
Uncertainty factor	Uncertainty factor = 10. 3.2 for toxicokinetics. 3.2 for toxicodynamics	3.2 (100.5) (individual variation) × 2 for overall average interindividual variation = 6.4 No toxicodynamic factor.
Exposure limit	Reference dose of 0.1 $\mu g\ kgbw^{-1}\ d^{-1}$ (equal to 0.7 $\mu g\ kgbw^{-1}\ wk^{-1}$)	1.6 $\mu g\ kgbw^{-1}\ wk^{-1}$ (equal to 0.23 $\mu g\ kgbw^{-1}\ d^{-1}$)

It is known that elemental Hg vapor can cross the placenta and accumulate in fetal tissue (136–138), and animal data suggest that elemental Hg has the potential to cause adverse neurologic developmental effects (139). Both elemental Hg and MeHg are metabolized in the brain to the inorganic mercuric form (38). It is not known whether the ultimate neurodevelopmental toxicant of MeHg is MeHg itself, the inorganic mercuric ion, free radicals generated in the conversion to the inorganic species, or some combination of these. If the inorganic form is the ultimate toxicant of MeHg in the developing brain or if MeHg and inorganic Hg share common neurodevelopmental toxic mechanisms, then current estimates of risk based on MeHg exposure alone could underestimate the population risk. Additional research is clearly needed to address these questions.

Potential Benefits of Fish Consumption

Several investigators have addressed the issues surrounding the risks and benefits associated with fish consumption, in general and for remote communities that depend on fish traditionally and/or as their dietary mainstay (69, 140–142). Indeed, for many populations, fish is the primary source of protein and other nutrients. Moreover, some fish can be an important source of the omega-3 fatty acids, eicosapentaenoic acid and docosahexaenoic acid, that appear to have positive effects on at least some of the same systems adversely affected by MeHg. However, similar to MeHg, there is considerable variability in the occurrence of omega-3 fatty acids across species (143). Fatty fish have higher levels of omega-3s compared with lean fish, and freshwater fish largely have lower levels of omega-3 fatty acids compared with ocean fish (15). There is no association between MeHg concentration of the fish or shellfish species and the omega-3 fatty acid level of the species (15). Several fish and shellfish species that are low in MeHg are high in omega-3 fatty acids (e.g., anchovies, herring, salmon), whereas others that are high in MeHg can be comparatively low in omega-3 fatty acids (e.g., shark, swordfish, pike) (15).

Omega-3 fatty acids are associated with beneficial effects on neurologic development in some studies (15), as has fish consumption in general, possibly as a correlate of omega-3 intake (90). However, not all studies found such a benefit (15, 144). Omega-3 fatty acids also were linked to a reduction in the risk of cardiovascular disease (44), although such an association recently were called into question in a comprehensive review (145). For both endpoints, there is some evidence suggesting that, in addition to its intrinsic toxicity, MeHg also antagonizes the beneficial effects of the omega-3 fatty acids (44, 119, 146). Because intake of both substances arises from the same food source, this suggests that the risk-benefit analysis for either the omega-3s or MeHg will depend on an understanding of this complex interaction.

Some animal studies suggest that micronutrients that are normally found in high levels in seafood, such as Se and vitamin E, may protect against Hg toxicity without specifically modulating MeHg absorption or excretion (55). For Se, differences across studies in the forms of Se and Hg, and the route and duration of exposure make interpretation difficult. Although there is some evidence showing protection against inorganic Hg toxicity by selenite, there is almost no evidence showing protection against MeHg toxicity by the organic Se compounds, such as selenomethione or selenocysteine, that are the forms of Se commonly found in the human diet. There is no human data that support a protective role for Se with respect to Hg neurotoxicity. For vitamin E, there is a suggestion that its antioxidant properties may protect against some of the adverse effects of MeHg (147, 148). However, there are few *in vivo* studies, and no epidemiological studies have addressed vitamin E intake.

RISK ASSESSMENT FOR MeHg

The risk assessment process for chemicals in foods is based on hazard identification, exposure assessment, dose-response evaluation, and risk characterization. The most commonly used paradigms for risk assessment are those reflecting the processes developed by the National Academy of Sciences/National Research Council (NAS/NRC) in the United States (149) and a similar process used internationally by the Joint Expert Committee on Food Additives and Contaminants (JECFA) under the Food and Agriculture Organization and the WHO (150). The NAS/NRC provided recommendations on MeHg in 2000, and JECFA continues to evaluate MeHg after their evaluation published in WHO Food Additives Series Number 52 (151).

In the risk assessment for MeHg, both NAS\NRC and JECFA used a benchmark dose approach based on a predetermined change in response rate of an adverse effect. Both used the benchmark dose lower limit (BMDL), which is the statistical lower confidence limit on the dose. Because these two major risk assessments recommend different intake levels [0.1 μg kg-body-weight $(bw)^{-1}\ d^{-1}$ and 0.23 $\mu g\ kgbw^{-1}\ d^{-1}$, respectively], here we examine the choices throughout the process that lead to these differences (Table 1):

i. Choice of study. Currently both rely on neurodevelopment effects of MeHg as the adverse health effect used in their respective risk assessments. The NAS/NRC based their analyses on the Faroes Islands study as the primary source of epidemiological data and relied on the studies from New Zealand (87) and the Seychelles as secondary sources and derived a BMDL, based on cord blood of 58 $\mu g\ L^{-1}$. The JECFA excluded the New Zealand study and, basing their BMDL calculation only on the Faroe Islands and the Seychelles studies, derived a BMDL of 12 $\mu g\ g^{-1}$ in maternal hair.
ii. Biomarker of exposure. The NAS/NRC based their analyses on cord blood, and the JECFA used maternal hair. Because some of the critical studies for these risk assessments measured only one of these biomarkers converting between cord blood and maternal hair concentration (or vice versa)

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involves uncertainty. Furthermore, as the most critical period(s) of gestation for the neurodevelopmental toxicity of MeHg are not yet known, it is not clear which lengths of maternal hair are most appropriate to measure

iii. Uncertainty factor. This factor accounts for adequacy of the pivotal study, interspecies extrapolation, interindividual variability in humans, adequacy of the overall data base, and the nature of the toxicity. These are not "safety factors" in that they are intended to factor in quantitatively to address areas of uncertainty in the risk assessment rather than provide "safety" *per se*. The magnitude of the uncertainty factors is intended as an estimate of the influence of these uncertainties, rather than the application of an arbitrary layer of safety. In the assessment conducted by the NAS/NRC committee, a composite uncertainty factor of 10 was used to account for variability and uncertainty in toxicokinetics and toxicodynamic, as well as database insufficiency for endpoints possibly more sensitive than neurodevelopmental (e.g., cardiovascular endpoints). The JECFA used an overall uncertainty factor of 6.4 to address variability in both toxicokinetics and toxicodynamics. The toxicokinetic portion accounts for a factor of 3.2 based on a generalized estimate of intraspecies toxicokinetic variability (152). The toxicodynamic portion likewise accounts for a factor of 2.0 based on a generalized estimate of interindividual variability in response.

The starting points for derivation of their respective recommended intakes differ both with respect to the actual values and the approaches taken. The JECFA Committee estimated that a steady-state intake of 1.5 μg kgbw^{-1} d^{-1} would be an exposure that would have no appreciable adverse effects on children, in contrast to the NAS/NRC determination of a BMDL of 1.0 μg kgbw^{-1} d^{-1}, which is an effect level. However, neither of these assessments reflected bioconcentration of MeHg across the placental circulation from the mother to the fetus (61). This bioconcentration and its population variability suggests that the full toxicokinetic variability is significantly larger (60, 153) than previously estimated (38, 151, 154).

The NAS/NRC used cord blood mercury for their BMDL of 58 μg L^{-1}, as did the US Environmental Protection Agency in 2001. However, the subsequent increased recognition that cord blood mercury is, on average, 60% to 70% higher in Hg than maternal blood, coupled to the coefficient of variation around the mid-point of 1.7 described by Stern and Smith (61) as 0.56 with a 95th percentile of 3.4, supports the use of a blood mercury concentration in the mid-30 μg L^{-1} range to recognize this fetal-maternal blood mercury difference (152, 155). By contrast, assessments based on association of maternal hair Hg with adverse neurobehavioral outcomes in the child after *in utero* exposures to MeHg need no such adjustment for MeHg concentration.

PANEL CONSENSUS CONCLUSIONS

Methylmercury is a potent toxicant, bioaccumulated and concentrated through the aquatic food chain, placing at risk humans who consume high-end aquatic predators or for whom fish is a dietary mainstay. Elevated levels of MeHg exposure occur worldwide and are not restricted to isolated populations. Rather, exposure to MeHg at levels above those that can be considered clearly safe and without risk of adverse effect occur throughout the globe and across the socioeconomic spectrum.

Hair and blood Hg concentrations (including cord blood Hg concentrations) are valid biomarkers of MeHg exposure. Each conveys somewhat different information on exposure. The most useful picture of exposure is likely to be obtained by data from both biomarkers, along with specific dietary information on fish consumption and other dietary data. Urinary Hg concentration is a biomarker of inorganic Hg. More research characterizing the relations between toenail Hg, hair Hg, blood Hg, and urinary Hg, and the relations between MeHg and inorganic Hg should be considered a priority. Single-strand and, particularly, continuous single-strand hair analysis of Hg concentration should be pursued as the best method for elucidating dynamic changes in MeHg exposure. This is particularly relevant for studies of the effect of *in utero* exposure to MeHg to assess the significance of bolus doses.

Total fish consumption without differentiating fish species is not necessarily a dependable metric for estimating MeHg exposure. To be useful for such purposes, valid data on the MeHg concentration of each species, as well as the frequency and the amount of consumption for each species must be included.

There is sufficient evidence to state that MeHg is a developmental neurotoxin, and developmental or fetal neurotoxicity has constituted the basis for risk assessments and public health policies. Although uncertainties in the risk assessment for the neurodevelopmental effects of MeHg remain, there is sufficient evidence to warrant a public health response based on prudent selection of fish species in the diet. Development of a formal case description and diagnostic criteria for the clinical effects of MeHg observed in some adults and older children with moderately elevated MeHg exposure should be a priority for clinicians involved with MeHg research.

Current studies suggest that present levels of exposure to MeHg have the potential to result in an elevated risk of cardiovascular disease to a significant fraction of the population. However, additional studies in other populations would clarify this picture. Quantitative dose-response assessment of existing studies should be undertaken. The potential effect of MeHg on the immune system should be investigated with respect to adverse effects on immune response, as well as with respect to individual sensitivities to MeHg, potentially including autoimmune responses.

To date, it has been possible to statistically separate the neurodevelopmental effects of MeHg and PCBs in key studies where both exposures occur in the fish-consuming population. However, knowledge of the mechanisms and interactions of PCBs and other halogenated organics with MeHg is an important missing piece in understanding the overall risk for fish consumption. Research into the potential interactions of inorganic Hg and MeHg should be considered a priority. Although the possible interactions between Se and MeHg are a fruitful area for further research, there is currently no clear evidence that dietary Se can modulate the toxicity of MeHg.

Because the intake of both omega-3 fatty acids and MeHg occurs from fish consumption and because MeHg appears to antagonize the beneficial effects of the omega-3s as well as exerting its own intrinsic toxicity, a proper assessment of risks and benefits for the combination of the two must address their complex interaction. Currently, there are insufficient data on this interaction to describe a coherent picture. Despite the lack of a clear picture of the interaction of the omega-3 fatty acids and MeHg, there are fish with high levels of omega-3s and relatively low levels of MeHg. Consumption of fish with low levels of MeHg and organic contaminants constitute a "win-win" situation and should be encouraged regardless of the underlying nature of the omega-3-MeHg interaction.

To preserve human health, all efforts need to be made to reduce and eliminate sources of exposure, through regulation and dissemination of information. In addition to documenting the multiple health hazards associated with exposure to MeHg throughout the lifespan, research needs to focus on identifying factors that influence the uptake and the toxicity of MeHg and

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on examining the potential benefits of different fish species. These studies will provide information on maximizing nutritional intake from consumption and minimizing risk from exposure to MeHg.

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Effects of Environmental Methylmercury on the Health of Wild Birds, Mammals, and Fish

Wild piscivorous fish, mammals, and birds may be at risk for elevated dietary methylmercury intake and toxicity. In controlled feeding studies, the consumption of diets that contained Hg (as methylmercury) at environmentally realistic concentrations resulted in a range of toxic effects in fish, birds, and mammals, including behavioral, neurochemical, hormonal, and reproductive changes. Limited field-based studies, especially with certain wild piscivorous bird species, e.g., the common loon, corroborated laboratory-based results, demonstrating significant relations between methylmercury exposure and various indicators of methylmercury toxicity, including reproductive impairment. Potential population effects in fish and wildlife resulting from dietary methylmercury exposure are expected to vary as a function of species life history, as well as regional differences in fish-Hg concentrations, which, in turn, are influenced by differences in Hg deposition and environmental methylation rates. However, population modeling suggests that reductions in Hg emissions could have substantial benefits for some common loon populations that are currently experiencing elevated methylmercury exposure. Predicted benefits would be mediated primarily through improved hatching success and development of hatchlings to maturity as Hg concentrations in prey fish decline. Other piscivorous species may also benefit from decreased Hg exposure but have not been as extensively studied as the common loon.

INTRODUCTION

Numerous studies document the toxic effects of methylmercury (MeHg) in individuals of various vertebrate species. However, it is less clear whether current environmental levels of MeHg pose health hazards to free-living fish and wildlife, and especially to populations of animals rather than to individuals. Here we present a brief synthesis of the scientific state of knowledge regarding current levels of MeHg exposure and its toxic effects in fish and wildlife. Because of the paucity of information on the toxicology of MeHg in reptiles and amphibians, our report focuses on fish, birds, and mammals.

Under most conditions, fish and wildlife are exposed primarily to MeHg rather than to other chemical forms of Hg, and the route of exposure is primarily through the diet. Thus, our report focuses on the effects of MeHg at ecologically relevant levels of dietary exposure. Our report is not intended to be a comprehensive critical review of the literature, and we pay particular attention to recent studies that have not been included in previous reviews. For a more detailed discussion of various aspects of Hg exposure, accumulation, and toxicology in fish and wild birds and mammals, the reader is directed to prior reviews (1–6).

EXPOSURE

Species and Habitats at Greatest Risk

Because of biomagnification of MeHg, long-lived piscivorous or other top predatory animals feeding in aquatic food chains are at greatest risk for elevated dietary MeHg exposure, accumulation, and toxicity. These species include large predatory fish, such as walleye (*Sander vitreus*), northern pike (*Esox lucius*), and lake trout (*Salvelinus namaycush*); mammals, such as mink (*Mustela* spp.), otter (*Lutra* spp.), polar bears (*Ursus maritimus*), and seals (*Phocidae* and *Liliaceae spp.*); and piscivorous birds, such as common loons (*Gavia immer*), bald eagles (*Haliaeetus leucocephalus*), osprey (*Pandion haliaetus*), kingfishers (*Alcedo* spp.); and some seabirds, such as albatross (*Diomedeidae*) and certain Arctic species (7). Conversely, terrestrial nonpiscivorous species (e.g., granivorous and insectivorous birds) typically demonstrate relatively low Hg exposure (<0.5 mg kg^{-1} wet weight in blood) (8, 9) and are generally not considered to be at risk for MeHg toxicity (10). High, potentially toxic concentrations of Hg (>5 mg kg^{-1} wet weight in brain, or >20 mg kg^{-1} in liver) have occasionally been reported in a variety of predatory wildlife species (5). Factors influencing Hg exposure in fish and wildlife, and concerns regarding tissue sampling and analysis have recently been reviewed (11, 12)

Piscivorous wildlife living in inland freshwater habitats often experience higher Hg exposure than the same species from nearby estuarine or marine habitats. In Maine, bald eaglets sampled at nests on inland lakes had higher blood-Hg concentrations than did eaglets raised in nests on rivers, estuaries, or marine (coastal) habitats; and a similar trend was found for belted kingfishers (*Ceryle alcyon*) nesting in the same region (8). Higher Hg concentrations were reported in feathers from chicks of both great egrets (*Ardea alba*) and white ibises (*Eudocimus albus*) at a variety of inland freshwater sites throughout peninsular Florida than in feathers of the same species from coastal colonies (13). Tissues of otter from inland habitats in Nova Scotia, Canada, had higher Hg concentrations than those from corresponding marine coastal habitats (14).

Piscivorous fish and wildlife living near local point sources of environmental Hg contamination may experience elevated Hg exposure (15, 16), which may persist long after new inputs of Hg have ceased (17–19). Some formerly important sources of environmental Hg pollution that resulted in toxicity and death of wild birds and mammals in the past (e.g., use of organomercurials as seed dressings and effluents from Hg-cell chloralkali plants) have been eliminated or greatly reduced.

Environments remote from point-source releases of Hg can also contain fish and piscivorous wildlife with elevated Hg concentrations. For example, some common loons and river otters (*Londra canadensis*) from the interior of Nova Scotia, Canada, and from several New England states (US) have among the highest tissue Hg concentrations reported for these species (8, 20, 21). Elevated Hg concentrations have also been reported in some predatory marine mammals and birds, especially from relatively remote northern locations (7, 22–26). In general, regions that receive relatively high atmospheric Hg loadings and are characterized by a high proportion of Hg-sensitive aquatic ecosystems in which Hg methylation rates are relatively high (low-alkalinity, low-pH lakes; surface waters with large upstream or adjoining wetlands; waters with adjoining or upstream terrestrial areas subjected to flooding; and dark-water lakes and streams) pose the greatest risk for

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piscivorous wildlife, because it is in these environments that trophic transfer of Hg is high and fish accumulate the greatest concentrations of Hg (5, 27, 28).

Temporal and Geographic Trends in Exposure

Where they were studied, temporal trends of Hg in wildlife were found to vary by region and by the magnitude of the time period chosen for analysis. Studies of trends in areas affected by industrial activity have sometimes reported declining Hg levels in recent years (21, 29); however, more long-term analyses, especially in remote locations, such as the North Atlantic and the Arctic, where global atmospheric inputs of Hg likely predominate, indicate increasing Hg concentrations in piscivorous wildlife over the past 30–150 years (24, 30, 31). In Florida, Hg in fish-eating birds increased substantially during the 1990s, compared with earlier decades, reflecting trends in local source deposition, which then later declined as local emissions were controlled (13, 32).

Extensive sampling of common loon feathers, blood, and eggs indicates a general west-to-east gradient of increasing Hg exposure in this species across North America, roughly consistent with patterns of atmospheric Hg deposition (33, 34). However, within specific regions, there can be wide variation in Hg exposure in loons, associated with differing land-use patterns, and physical and chemical characteristics of local watersheds—for example, the degree of lake acidification (8).

Demethylation and Interactions with Selenium

Most fish and wildlife are exposed to Hg primarily as MeHg through diet. However, in at least some predatory aquatic wildlife species, after tissue accumulation of MeHg, a portion of the body's MeHg burden may be demethylated. The resulting inorganic Hg often accounts for a significant, yet highly variable, fraction of the total Hg present in certain tissues, especially in liver and kidney (and perhaps brain). Some other tissues, such as skeletal muscle; fur; feathers; and eggs show little or no evidence of demethylation. Methylmercury in liver generally predominates when total Hg concentrations are less than ~10 mg kg^{-1} wet weight; however, with greater Hg accumulation, an increasingly high proportion of the total liver Hg is often present as an inorganic species. Frequently, animals with the highest liver-Hg accumulation have the lowest MeHg fraction, despite being exposed to Hg primarily as MeHg in fish (35, 36). Although there is some uncertainty regarding the toxicity of inorganic Hg derived from demethylation, a detoxification mechanism for MeHg in seabirds and marine mammals has been proposed that entails demethylation by reactive oxygen species and subsequent formation of high molecular weight Hg-selenium (Se)-protein compounds, which subsequently undergo degradation in lysosomes, creating an insoluble Hg-Se compound (35, 37). Ultimately, insoluble mercuric selenide (tiemannite) or other stable, insoluble Hg-Se-protein fragments accumulate in the liver through time (23, 38). The cellular mechanisms by which this process occurs and the energy costs involved are not well understood.

Different species exhibit differences in their apparent ability to demethylate Hg, which may affect their relative sensitivity to MeHg toxicity; however, this has not been explicitly studied. For example, free-living river otters generally accumulate higher concentrations of Hg than mink; however, in otter brains, only ~74% of total Hg was in the organic form (MeHg), compared with ~90% for mink (39, 40), indicating that otters may be better able to metabolize organic Hg into an inorganic form. Also, a significant correlation between Hg and Se was reported in brains of otter but not mink (41). A positive correlation between Hg and Se levels in the brain was also observed in monkeys exposed to MeHg, with no exposure to Se other than through their regular diet (42). In addition, fish and wildlife living in environments with elevated Se levels exhibit lower-than-expected Hg accumulation (43, 44).

Diets supplemented with Se, including organic forms of Se present in biological materials, can protect against or delay the onset of MeHg-induced neurotoxicity (45). However, although selenomethionine supplementation ameliorated the neurotoxic effects of dietary MeHg exposure in adult mallard ducks (*Anas platyrhynchos*), reproductive impairment was more severe in ducks that consumed a diet supplemented with both MeHg and selenomethionine than in birds consuming diets with elevated levels of MeHg or selenomethionine alone (46). Differences in the dietary intake of Se by different wildlife species, or the same species in different environments, may contribute to variability in the expression of MeHg toxicity. The extent to which different wildlife species demethylate MeHg, the consequences of this process on the toxicology of MeHg, and the role of Se in MeHg accumulation and toxicity in wildlife at ecologically relevant doses require further investigation.

EFFECTS

Fish

Compared with humans and mammalian and avian wildlife, relatively little is known of the toxicological significance to fish of environmentally realistic exposures to MeHg (4, 5). Laboratory studies typically exposed fish to aqueous concentrations of MeHg that are 10^4- to 10^5-fold greater than those in natural waters (4), rather than using realistic dietary exposure conditions. The route of administration of Hg in these studies was also unrealistic, because diet, not water, is the main source of MeHg exposure in wild fish (47, 48).

Overt effects on fish growth and survival occur only at high tissue Hg concentrations (6–20 mg kg^{-1} wet weight in muscle) (4), observed primarily in fish from highly contaminated environments, such as Minamata Bay, Japan (49, 50), and Clay Lake in the English-Wabigoon River system, Ontario, Canada (51). However, several laboratory studies demonstrated MeHg impairment of fish behavior, gonadal development, production of sex hormones, and reproduction at concentrations more typical of those in fish from flooded, low alkalinity, or other Hg-sensitive habitats (52–55). Suppression of gonadal development, egg production, and spawning were reported in juvenile fathead minnows (*Pimephales promelas*) fed MeHg-contaminated diets until sexual maturity (56). Mated pairs of fish with mean carcass concentrations of 0.71 mg kg^{-1} wet weight (males) to 0.86 mg kg^{-1} (females) experienced a 39% reduction in spawning success. Similar carcass concentrations were also associated with disrupted reproductive behavior of male fathead minnows (57), suppressed plasma estradiol and testosterone, and reduced reproductive success (52). Suppressed estradiol was likely due to increased numbers of apoptotic follicular cells caused by MeHg exposure (58). Significant inverse relationships between concentrations of MeHg and estrogen, testosterone, and 11-ketotestorone was also observed in white sturgeon (*Acipenser transmontanus*) from the lower Columbia River (59). Methylmercury concentrations that suppressed sex hormones, altered reproductive behavior, and impaired reproduction in fish were similar to mean concentrations in carcasses of white perch (*Morone americana*; 0.78 mg kg^{-1}), walleye (0.71 mg kg^{-1}), and northern pike (*Esox lucius*; 0.56 mg kg^{-1}) from northeastern North America (60) and less than those measured in axial muscle of fish from many lakes and reservoirs (Fig. 1).

Although maternal transfer of dietary MeHg bioaccumulated during oogenesis is the primary mechanism of MeHg

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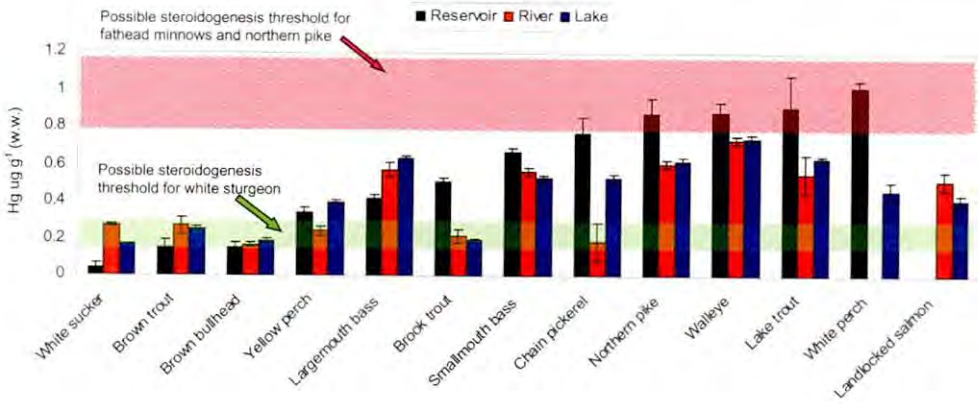

Figure 1. Mean Hg concentrations in fillets of standard-length freshwater fish of various species from across northeastern North America (data adapted from [60]), combined with estimated Hg concentration thresholds associated with steroidogenic effects. Threshold ranges adapted from (52) [fathead minnows]; Sandheinrich, M., Drevnick, P., Wiener, J., Knights, B., and Jerimison, J. unpubl. data [northern pike]; and (59) [white sturgeon].

exposure to fish embryos (61), there is limited information on the effects of maternally transferred MeHg in fish. Aqueous exposure of eggs of rainbow trout (*Oncorhynchus mykiss*) to inorganic Hg resulted in total Hg concentrations of 0.07–0.10 mg kg^{-1} wet weight in eggs and significantly increased embryonic mortality (62). These concentrations are within the range of total Hg measured in eggs of yellow perch (*Perca flavescens*) from semi-remote lakes in northern Wisconsin, US (63). Thus, based on laboratory studies that examined the effects of dietary MeHg in fish, combined with data on concentrations of MeHg in free-living fish, it is plausible that MeHg at environmentally relevant concentrations may affect reproduction in wild populations of fish. Differences among species in their reproductive sensitivity to MeHg, as well as the population-level consequences of impaired reproduction in individual fish remain to be determined.

Mammals: Suggested Toxicity Thresholds

Mink and otter are the mammalian wildlife species for which the greatest amount of information exists regarding Hg exposure and toxicity. Data from several studies indicate that consumption of diets that contained Hg (as MeHg) $\geq$1 mg kg^{-1} wet weight caused neurotoxicity and death in adult mink and otter (5). The US Environmental Protection Agency (USEPA) estimated lowest observable adverse effect level (LOAEL) for mink is currently 0.18 mg kg^{-1} body weight d^{-1}, or 1.1 mg kg^{-1} wet weight (approximately 3–4 mg kg^{-1} dry weight) MeHg in the diet (64). Neurological signs in MeHg-intoxicated mammals typically include lethargy, ataxia, limb paralysis, tremors, convulsions, and ultimately death.

Brain Hg (probably primarily as MeHg) concentrations in otter and mink with overt MeHg poisoning are typically >5 mg kg^{-1} wet weight (5). After exposure to MeHg, brain-Hg concentrations in the range of 12–20 mg kg^{-1} wet weight during postnatal development in a variety of small mammal species used in medical research were associated with blindness, spasticity, and seizures, whereas 3–11 mg kg^{-1} were associated with more subtle effects on behavior and cognition, including increased activity, poorer maze performance, abnormal startle reflex, impaired escape and avoidance behavior, and abnormal visual evoked potentials (65). Numerous individual free-living mink or otter from various locations in North America have brain-Hg concentrations sufficiently high (>3 mg kg^{-1}) to be associated either with clinical MeHg intoxication or with more subtle neurological impairments that could detrimentally affect survival (5, 21).

Recently, various neurochemical changes in brains of free-living otter (66) and mink (67) were correlated with brain-Hg concentrations <5 mg kg^{-1} wet weight; and similar effects were demonstrated in captive mink fed diets that contained ecologically realistic concentrations of MeHg (68) (Fig. 2). It is probable that the current level of MeHg exposure of free-living mink and other piscivorous mammals in a number of Hg-sensitive environments is sufficiently high to have subtle neurotoxic and other consequences (69, 70). Aqueous MeHg concentrations likely exceed the USEPA derived mammalian wildlife criteria for mink (57 pg MeHg L^{-1}) (71) in many aquatic ecosystems; however, it is currently unclear whether documented environmental concentrations and toxic effects on individual animals have population-level impacts in mink or other mammalian species.

Birds: Suggested Toxicity Thresholds

In adult birds, Hg concentrations (as MeHg) >15 mg kg^{-1} wet weight in a variety of tissues, including brain, are associated with overt signs of MeHg intoxication and death (5). Nonlethal effects of lower Hg concentrations have been less well studied, except for effects on reproduction. Egg-Hg concentrations >1 mg kg^{-1} wet weight are associated with impaired hatchability and embryonic mortality in a number of bird species, and brain-Hg concentrations >3 mg kg^{-1} wet weight are associated with mortality in developing bird embryos (5). In free-living common loons, diets that contained >0.3 mg kg^{-1} wet weight Hg (as MeHg) were associated with severely reduced reproductive success, mainly as a result of decreased egg laying and territorial

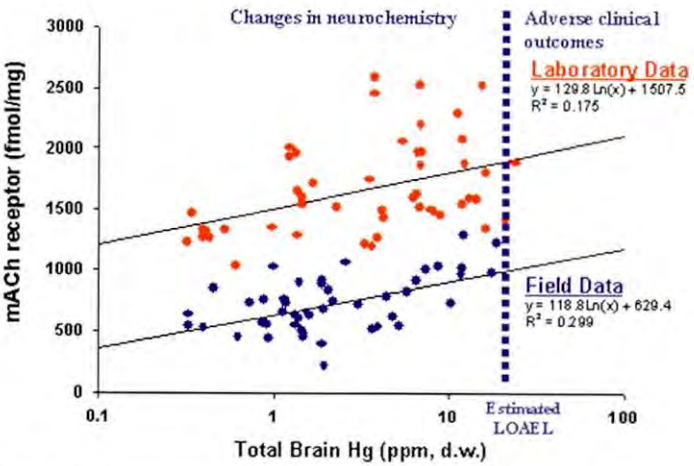

Figure 2. Concentration of muscarinic acetylcholine receptors in brains of free-living wild mink and captive mink fed diets with different levels of MeHg as a function of brain Hg concentration. Changes in receptor concentrations occur at Hg concentrations below those associated with overt toxicity. Data adapted from Ref. 68. Estimated LOEL for overt MeHg intoxication (~20 mg kg^{-1} dry weight or ~5 mg kg^{-1} wet weight Hg in brain) based on (98).

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fidelity by breeding adults (15). Many lakes in North America have fish with Hg concentrations that exceed 0.3 mg kg^{-1} wet weight (60, 72).

The MeHg effect threshold for common loon chicks was established via subchronic dose-response studies (73–75). Loons were dosed daily from hatch through day 105 with fish diets that contained control, 0.08, 0.4, or 1.2 mg kg^{-1} wet weight as MeHg chloride. No overt signs of toxicosis or significant reductions in growth or food-consumption rates were observed in any dose group (75), but there was evidence of reduced immune response and histological changes (central nervous system demyelination) in chicks that received ecologically relevant doses of MeHg (0.4 mg kg^{-1} diet wet weight) (76). A preliminary loon chick LOAEL was estimated at 0.4 mg kg^{-1} wet weight in diet (fish), whereas a preliminary no observable adverse effect level (NOAEL) in diet was estimated at 0.08 mg kg^{-1} wet weight (76).

Birds: Recent Field Studies

Correlations between MeHg exposure, reproductive impairment, and other effects have been examined in common loons breeding in the northern United States and eastern Canada (8, 15, 20, 76–80) and in wading birds nesting in south Florida and the Everglades (13). Although these correlational studies cannot be used to establish rigorous exposure thresholds of effect, they can identify populations where significant statistical relations exist between MeHg exposure and demographic and/or physiological parameters, and can assist in establishing effects thresholds.

Common Loon Reproduction in New Hampshire and Maine, US. Evers et al. (77) measured common loon productivity, behavior, and biochemical markers in relation to Hg exposure in 212 breeding territories and used adult blood-Hg concentrations to assign breeding territories to categories of Hg toxicity risk. Loon territories where adult blood Hg levels exceeded 3.0 mg kg^{-1} produced 40% fewer fledged young than territories where adult blood Hg was less than 1.0 mg kg^{-1}; these territories were categorized as "high risk." Territories where adult loon blood Hg exceeded 4 mg kg^{-1} were classified as "extra-high risk," because these concentrations were associated with impaired productivity, elevated levels of corticosterone in blood, developmental asymmetry in flight feathers, and adverse changes in essential breeding behaviors. On average, circulating corticosterone hormone levels increased 14.6% for every mg kg^{-1} increase in blood Hg ($n = 239$). Paired secondary feathers (one from each wing) from adults on high or extra-high risk territories had greater differences in mass than feathers sampled from territories with low Hg exposure risk ($n = 227$). Adult loons in high-risk territories also left eggs unattended 14% of the time, compared with 1% in lower-risk territories. A significant negative relation was found between adult blood Hg and foraging behavior, and a significant positive relation was observed between adult blood Hg and brooding behavior.

The majority of loon eggs collected at nests in New Hampshire and Maine ($n = 448$) contained elevated Hg concentrations. In Maine, 11% of eggs had Hg concentrations between 1.3 and 2.0 mg kg^{-1} wet weight, and 4% were >2 mg kg^{-1} wet weight (77). Egg Hg concentrations >1 mg kg^{-1} wet weight are associated with impaired hatchability in a number of avian species (3, 5, 20).

Common Loon Reproduction in Kejimkujik National Park, Nova Scotia, Canada. Common loons in some areas of Atlantic Canada have among the highest mean blood Hg concentrations in North America (20, 33). Concentrations were highest at Kejimkujik National Park, Nova Scotia, where adult blood-Hg levels averaged >4 mg kg^{-1} wet weight and where lower-than-normal loon productivity has been observed for many years (81, 82). At Kejimkujik, 92% of adult loons sampled had blood Hg concentrations in the "extra-high risk" category (>4 mg kg^{-1} wet weight) suggested by Evers et al. (77). The Hg exposure was related to impaired loon productivity and altered breeding behavior in loons in Atlantic Canada (83). The majority of common loon eggs collected at Kejimkujik nests had potentially toxic Hg concentrations (>1 mg kg^{-1} wet weight). Mercury concentrations in both loons and fish in Kejimkujik lakes were correlated with lake pH; however, prey fish abundance was not correlated with lake pH, indicating that pH-related prey depletion was not occurring and was not a confounding factor influencing loon productivity at Kejimkujik.

Common Loon Reproduction in Wisconsin, US. Field studies conducted in Wisconsin showed reduced common loon reproductive performance on acidic lakes where fish Hg concentrations and loon Hg exposure levels were elevated (79, 80). Subsequent studies found that, on acidic lakes, loon chick food intake rates and survival were lower, adult foraging behavior was altered, and blood Hg of adults and chicks, as well as prey Hg concentrations were higher than on circumneutral lakes (78). However, several environmental parameters, such as lake pH, co-varied with Hg. Thus, a laboratory approach was deemed necessary to directly test the effects of Hg exposure on common loon reproductive performance and to estimate LOAELs and NOAELs (75, 76).

One-hundred-fifteen adult loons from Wisconsin were assessed for Hg exposure via blood samples during 2002–2004, and 7 (6% of those sampled) had a blood Hg level >3.0 mg kg^{-1} wet weight (76), within the high-risk Hg exposure category of Evers et al. (77). Fifty-four percent of unhatched loon eggs collected from 33 nests in Wisconsin during 1996–2000 were categorized as "background" (0.0–0.6 mg kg^{-1} Hg wet weight) and 46% as "elevated" (0.6–1.3 mg kg^{-1} wet weight); none exceeded 1.3 mg kg^{-1} wet weight. All adult loons and loon eggs with elevated Hg concentrations sampled in Wisconsin were from acidic lakes (pH < 6.3).

A Wisconsin common loon demographic model was developed to evaluate population performance within a 5000 km^2 risk-assessment region of northern Wisconsin (76). The 5-stage deterministic projection matrix model predicts an annual growth rate of 0.9988 by using adult survival, fertility, and juvenile recruitment rates measured in the risk-assessment region during 2002–2004. Controlled dosing studies with loons (chick survival and hatching rates), combined with exposure assessment of the Wisconsin loon population, indicate that 10% of northern Wisconsin loon chicks have exposure levels associated with toxicity in the laboratory (Fig. 3), and 10% of adult female loons have exposure levels associated with a 30% reduction in egg hatching rate (76) (Fig. 4). The benefits of decreasing the Hg content of fish can be simulated by increasing the hatching and survival rates to account for the impairment assumed to be caused by elevated Hg exposure. If all chicks that are at risk for toxicity within the assessment region die before transition to adult stage (a conservative assumption), then reductions in Hg emissions to levels that result in Hg concentrations in loon prey <0.08 mg kg^{-1} wet weight (loon chick NOAEL from dosing study) should increase juvenile survival by 10%; and hatching success could be increased by 30% in 10% of the reproducing females. Inclusion of these adjustments to hatching success and chick survival in the projection matrix model results in a predicted improvement in annual population growth rate from 0.9988 to 1.011. When assuming a 10% improvement in chick survival and breeding success, the population growth rate within the risk-assessment region is predicted to increase by 1.7% annually. When assuming a total adult population in the study area of

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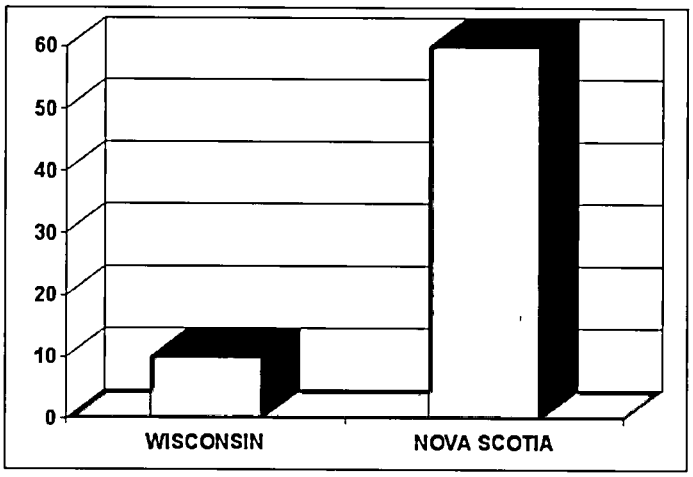

Figure 3. Percentage of loon chicks from Wisconsin, US, and Nova Scotia, Canada, having blood Hg concentrations associated with toxic effects (76) (>0.3 μg/m, as determined in controlled laboratory dosing studies [75]).

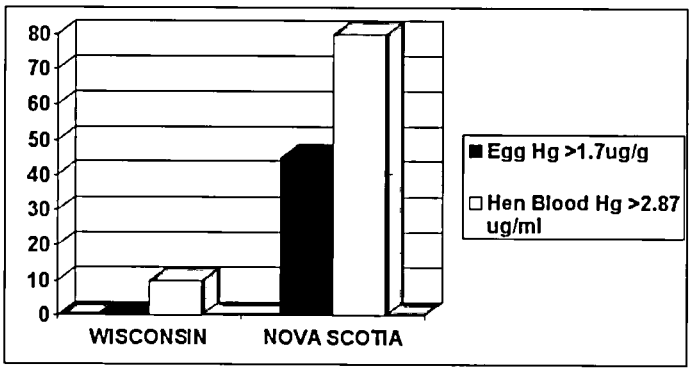

Figure 4. Percentage of regional common loon populations in Wisconsin, USA, and Nova Scotia, Canada, having egg Hg or hen blood Hg greater than thresholds associated with reduced hatching success.

approximately 1200 individuals (estimated population size in 2003), a 1.7% increase in annual growth rate would translate into 20 additional adults being recruited into the breeding pool each year. This K-selected species has an estimated lifespan of 25 years and an estimated annual survival rate of 0.92, thus the increase could plausibly translate into measurable improvement in population performance. Mercury emission reductions are predicted to have a much greater beneficial impact on the annual growth rate of the New England and Nova Scotia common loon populations where much larger proportions of chicks and hens are exposed to Hg levels associated with toxicity (Fig. 3), as determined in controlled dosing studies.

Aquatic Bird Population Decline in South Florida and the Everglades. Water bird populations (primarily wading bird species in the order Ciconiiformes) have declined precipitously in south Florida and the Everglades National Park since the mid 1930s. There was a ≥90% reduction in the numbers of nesting pairs of wood storks (*Mycteria americana*), great egrets (*Ardea albus*), white ibises (*Eudocimus albus*), and snowy egrets (*Egretta thula*) (84, 85). Loss of habitat, changes in hydroperiod, marsh compartmentalization, and salinization of estuarine feeding areas are thought to be primary factors responsible for the decline (84–86). However, MeHg toxicity has been proposed as an additional contributing factor (87) and as a potential impediment to the recovery of these species. Elevated liver Hg concentrations were found in 30–80% of breeding-age birds of various species found dead in the Everglades during 1987–1991 (88). In prebreeding female white ibises in the Everglades, estradiol concentrations were negatively correlated with Hg concentrations, as was the number of nesting attempts, suggesting that Hg exposure may cause fewer birds to nest or more birds to abandon nests because of subacute effects on hormone systems (89). Similar effects of Hg exposure on nesting behavior were previously documented for common loons in northwestern Ontario, Canada (15).

In recent years, dramatic declines in wading bird Hg exposure were documented in the Everglades. By using a previously established predictive relation between Hg consumption in food and feather Hg concentration for great egrets, Frederick et al. (13) estimated that average Hg concentrations in the diet of egrets declined by 67% between 1994 and 2000, and concluded that the Everglades underwent a biologically significant decline in Hg availability during that time period, probably due mainly to decreased local Hg inputs.

Bald Eagles and Osprey. Although both bald eagles and osprey are large piscivorous birds that experience elevated Hg exposure in some environments, these species have not been well studied with respect to potential effects of Hg on reproductive success or other population parameters. Nevertheless, the existing published reports indicate a lack of association between Hg exposure and productivity of free-living eagles or osprey in different locations in North America (19, 90–92).

Immunotoxic Effects

It has long been recognized that exposure to Hg may have autoimmune consequences. In wild birds, there is also circumstantial evidence that environmental MeHg exposure may be associated with a higher potential for infection by disease organisms. Dead loons found in an emaciated condition or with more parasitic infections generally had higher tissue-Hg concentrations than loons found dead in good body condition (93). In Florida, herons that died from chronic disease had significantly higher liver Hg concentrations (9.76 ± 2.40 mg kg^{-1}) than those that died in good body condition (1.77 ± 1.79 mg kg^{-1}) (87). Similarly, a number of immunological parameters were affected in egrets fed fish that contained an environmentally relevant concentration of Hg (0.5 mg kg^{-1}) (94). Low concentrations of both inorganic Hg and MeHg inhibited avian white blood cell phagocytosis *in vitro*; however, similar effects were not observed in blood of birds exposed to elevated MeHg *in vivo* (95). For mammals, harbour porpoises (*Phocoena phocoena*) demonstrated a relation between infections and elevated Hg accumulation (96, 97). The immunotoxicological effects of dietary MeHg exposure require further scientific research.

CONCLUSIONS

There is consistent evidence across a number of species that wild populations of fish, birds, and mammals in some regions of North America consume diets with MeHg concentrations sufficiently high to be toxic to individuals as determined from controlled dosing studies. Some wildlife species that feed at high trophic levels in aquatic food chains, especially species that feed primarily on fish or on other piscivorous species, are at particularly high risk for elevated exposure and potential toxicity. Laboratory dosing studies with fish and with piscivorous birds and mammals, indicate that ecologically relevant MeHg exposures can cause significant behavioral, physiological, immunological, neurochemical, reproductive, and histological changes. For fish and amphibians, field studies are limited and direct evidence of altered reproduction or other population parameters because of MeHg exposure in free-living

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populations are not presently available. However, for some wild piscivorous bird species, field studies demonstrated significant positive relations between MeHg exposure and numerous indicators of MeHg toxicity, including reproductive impairment, at ecologically relevant levels of Hg contamination. The weight of evidence indicates that reproduction is the demographic parameter most affected by exposure to MeHg in birds (and plausibly for fish and mammals as well). Potential population impacts will vary as a function of species life history, as well as regional differences in MeHg exposure because of regional and local variability in fish Hg concentrations. Modeling indicates that some regional common loon populations may benefit significantly from reductions in Hg emissions, which would result in decreasing concentrations of Hg in prey fish, and increased reproductive success mediated primarily through better hatching success and survival of hatchlings. Although the risk-assessment approach used for wildlife often emphasizes sustainability of wildlife "populations" rather than the health of individuals, conserving habitat quality is also a major consideration. Environmental Hg contamination results in degradation of Hg-sensitive aquatic ecosystems, because inorganic Hg is methylated, rapidly enters the food web, and biomagnifies to potentially toxic concentrations in fish and their predators.

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Panel on Source Attribution of Atmospheric Mercury:
Steve Lindberg, Russell Bullock, Ralf Ebinghaus, Daniel Engstrom, Xinbin Feng, William Fitzgerald, Nicola Pirrone, Eric Prestbo and Christian Seigneur

A Synthesis of Progress and Uncertainties in Attributing the Sources of Mercury in Deposition

A panel of international experts was convened in Madison, Wisconsin, in 2005, as part of the 8th International Conference on Mercury as a Global Pollutant. Our charge was to address the state of science pertinent to source attribution, specifically our key question was: "For a given location, can we ascertain with confidence the relative contributions of local, regional, and global sources, and of natural *versus* anthropogenic emissions to mercury deposition?" The panel synthesized new research pertinent to this question published over the past decade, with emphasis on four major research topics: long-term anthropogenic change, current emission and deposition trends, chemical transformations and cycling, and modeling and uncertainty. Within each topic, the panel drew a series of conclusions, which are presented in this paper. These conclusions led us to concur that the answer to our question is a "qualified yes," with the qualification being dependent upon the level of uncertainty one is willing to accept. We agreed that the uncertainty is strongly dependent upon scale and that our question as stated is answerable with greater confidence both very near and very far from major point sources, assuming that the "global pool" is a recognizable "source." Many regions of interest from an ecosystem-exposure standpoint lie in between, where source attribution carries the greatest degree of uncertainty.

BACKGROUND

The importance of attributing the sources of mercury (Hg) in deposition is now widely recognized, and it is critical for policy development. A panel of international experts was convened in Madison, Wisconsin, in July 2005, to address the state of science pertinent to source attribution. Our key question was this: For a given location, can we ascertain with confidence the relative contributions of local, regional, and global sources, and of natural *versus* anthropogenic emissions to Hg deposition?

Because several of the current participants were panel members at a similar workshop a decade ago (1), we agreed to develop this paper as an update and synthesis of important progress in measurements and modeling since that time. The major conclusions of our synthesis, and the rationale for reaching these conclusions are described in this paper.

Definitions

Mercury cycling research has matured, and papers now appear in all major Earth science journals. Because the Hg biogeochemical cycle is similar to those of carbon (C), sulphur (S), and nitrogen (N), the panel suggests that some earlier Hg cycling jargon is no longer useful, confusing to outside researchers, and perhaps should be avoided in new studies. The panel also agreed that the widespread use of some common terminology is confusing the development and implementation of Hg control policy. This is especially true of the phrase "global background Hg." This term may imply that the global pool is entirely "natural," although it is widely accepted that the majority (perhaps two-thirds) consists of Hg that was put into the global cycle by human activity. To facilitate the following discussion, the panel felt it was important to agree on this brief set of definitions (derived and updated from Expert Panel 1994):

- **Anthropogenic emissions**: The direct emissions of Hg from current human activities.
- **Atmospheric half-life**: The time required for the initial concentration of a chemical species to decrease by 50% (for a first-order removal process, half-life ≈ 70% of the lifetime).
- **Atmospheric lifetime** (or **residence time**): Mean time that a chemical species remains in the atmosphere before being removed.
- **Atmospheric mercury depletion events**: Periods during which rapid atmospheric oxidation reactions reduce the concentration of gaseous elemental Hg (Hg^0) (sometimes to concentrations below 0.1 ng m^{-3}), while producing oxidized gaseous and particulate Hg, and leading to elevated deposition.
- **Bidirectional surface fluxes**: The phenomenon by which the flux of *gaseous* Hg^0 across the air/surface interface (soil, vegetation, water) occurs in both directions. Regardless of whether the flux is directed up (emission) or down (deposition), it is basically the same phenomenon, though sometimes controlled by different factors.
- **Global sources, global background,** and **global pool**: The phrase *global background Hg* creates confusion regarding the contribution of anthropogenic emissions to deposition. The global atmospheric pool of Hg is a mixture of Hg emitted from all sources (both natural and anthropogenic) and is dominated by primary and secondary anthropogenic emissions, even at the most remote locations. *Global background concentration* refers to the average sea-level atmospheric concentration of Hg^0 at remote sites and is currently taken as ~1.5–1.7 ng m^{-3} in the Northern Hemisphere, ~1.1–1.3 ng m^{-3} in the Southern Hemisphere. The term "global sources" should be avoided, because "all sources are local" but have the capacity to contribute to the global pool.
- **Mercury re-emission:** The panel recommends against the continued use of this term. It was defined in the 1994 report as the emission of Hg into the atmosphere by biotic and abiotic processes from land and water surfaces, drawing on a pool of Hg that was previously deposited to Earth's surface after mobilization by anthropogenic or natural activities. *Re-emitted* Hg is *emitted* Hg, and, in most cases, it will be impossible to accurately state the true source of the Hg leaving the surface.
- **Mercury speciation:** A largely operationally defined distinction among various chemical and physical forms of atmospheric Hg. These include gaseous elemental Hg (Hg^0); reactive gaseous Hg (RGM), consisting of various

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oxidized gaseous Hg(II) compounds; and particle-bound Hg (Hg_p), consisting of various (commonly unknown) Hg compounds.

- **Natural source emissions:** The mobilization into the atmosphere of geologically bound Hg, undisturbed by human activities, by natural biotic and abiotic processes; after ~2000 years of Hg mobilization by human activity, it is doubtful that such emissions can be directly measured without some degree of interference from previously deposited anthropogenic Hg, except perhaps in new volcanic activity.

THE GLOBAL MERCURY CYCLE AND SOURCE ATTRIBUTION

Attributing the sources of Hg deposited to sensitive receptors requires a clear understanding of the concepts of the global Hg cycle. Global-scale Hg cycling models (2) provide a useful perspective on the behavior and the fate of Hg in the environment. The last decade yielded insights into the dynamics and the chemistry associated with atmospheric Hg cycling. One of the major advances is the experimental observation of the presence and the production of RGM at remote locations, ranging from polar regions to the open ocean (3–6). A sense of the importance of more dynamic cycling of Hg between the Earth's surface and the lower atmosphere can be gained by modification of the Mason Fitzgerald Model (MFM) simulation (7).

The principal features of these global models remain, with the atmosphere and its chemical and physical processes, providing the primary medium for Hg mobilization and exchange in the environment. Uncertainties associated with the fluxes and reservoirs of the Hg cycle are part of the updated perspective. These simulations are usually considered, at best, factors of 2 scaling exercises, which can provide an uncertain but useful framework for generating hypotheses and planning experiments.

Given the caveats, the fluxes derived from the Mason and Sheu model show substantial changes compared with the 1994 MFM version (Table 1). For example, anthropogenic Hg emissions are about 50% smaller than the 20 and 10 Mmol y^{-1} used previously, in agreement with new estimates for source strengths and speciation (8, 9).

Uncertainties of ±30% are given for total anthropogenic Hg emissions. The overall confidence in the portion of the human-related emissions entering the global pool and that depositing locally is around ±50%. Terrestrial Hg emissions account for an estimated land to air flux of 8 Mmol y^{-1}, which is enhanced compared with the value of 5in the prior MFM simulation. Given the difficulties of scaling results from studies conducted in limited geographic regions to the entire terrestrial realm (10), these categories are highly uncertain (greater than ±50%).

The RGM/Hg° cycling at the air-sea interface appears to be significant, with 5.7 Mmol y^{-1} of the 13 Mmol y^{-1} estimated to be evading from the oceans returning to the sea surface in deposition. Comparable RGM/Hg° dynamics in the terrestrial realm are contained in the combined wet and dry deposition fluxes. Approximately 3 Mmol y^{-1} are attributed to remote dry deposition to land, and this term includes fluxes associated with polar depletion events. Mercury depositional fluxes associated with precipitation might exhibit uncertainties of about ±30–50% for both the oceanic and continental reservoirs; dry deposition is more uncertain.

The atmospheric Hg burden shows little change in the updated global Hg model. The average residence time for Hg in the global pool is approximately 0.75 y (compared with 1 y for the MFM model), and the uncertainty in this estimate is about a factor of 2. This global perspective provides an interesting contrast to recent reports of fast Hg oxidation kinetics, leading to estimates of much shorter residence times for Hg in certain situations [e.g., polar regions, marine boundary layer (MBL), upper troposphere], as discussed in the Deposition Mechanisms and Source Attribution section below. This highlights the complexity of the global cycle and suggests the importance of Hg emissions from natural surfaces in maintaining the global pool. It has been suggested that the lifetime of gaseous Hg should be considered a regional, not global, variable (11).

Historic Anthropogenic Trends in the Global Cycle

The preindustrial simulation from Mason and Sheu (12) is juxtaposed with the modern one in Figure 1. Because such simulations are internally consistent, it is likely that the relative strengths of sources and sinks for Hg may be better constrained than the actual fluxes. Accordingly, the global Hg cycle today has been greatly altered by human-related transfer of Hg from deep geological stores to Earth's surface, especially since the onset of the industrial period (ca. 1850).

The evidence for the long-term change comes from historic records of Hg deposition preserved principally in lake sediments but also in peat deposits and glacial ice. Although these historic archives are complex recorders of atmospheric deposition, there is a large body of experimental and observational evidence for their reliability (13). A growing number of these core records from remote sites in both the Northern and Southern hemispheres demonstrates about a threefold increase (2×–4×) in Hg deposition since preindustrial times (14–17). This convergence of data is significant, because it suggests that anthropogenic emissions have altered Hg fluxes to about the same degree throughout the world, even though contemporary Hg deposition exhibits large temporal and spatial variability.

By accepting the assumptions of these models and that preindustrial Hg deposition was less than current deposition by a factor of three, the global Hg budget in preindustrial times

Table 1. Model estimates of current global Hg fluxes.

Emissions	MS* (units Mmol y^{-1})	MFM† (units Mmol y^{-1})	Deposition	MS* (units Mmol y^{-1})	MFM† (units Mmol y^{-1})
Direct anthropogenic	12	20	Wet deposition to ocean	9.7	10
Land emissions‡	8	5	Dry deposition to ocean	5.7	
			Remote wet deposition to land	10.0	15
Ocean emissions (evasion) ‡	13	10	Remote dry deposition to land	2.9	
			Local deposition to land	4.7	10
Total	33	35	Total	33	35

* From Ref. 7.

† From Ref. 2

‡ Land and ocean emission terms include a substantial portion of Hg (~50%) originally emitted from anthropogenic sources.

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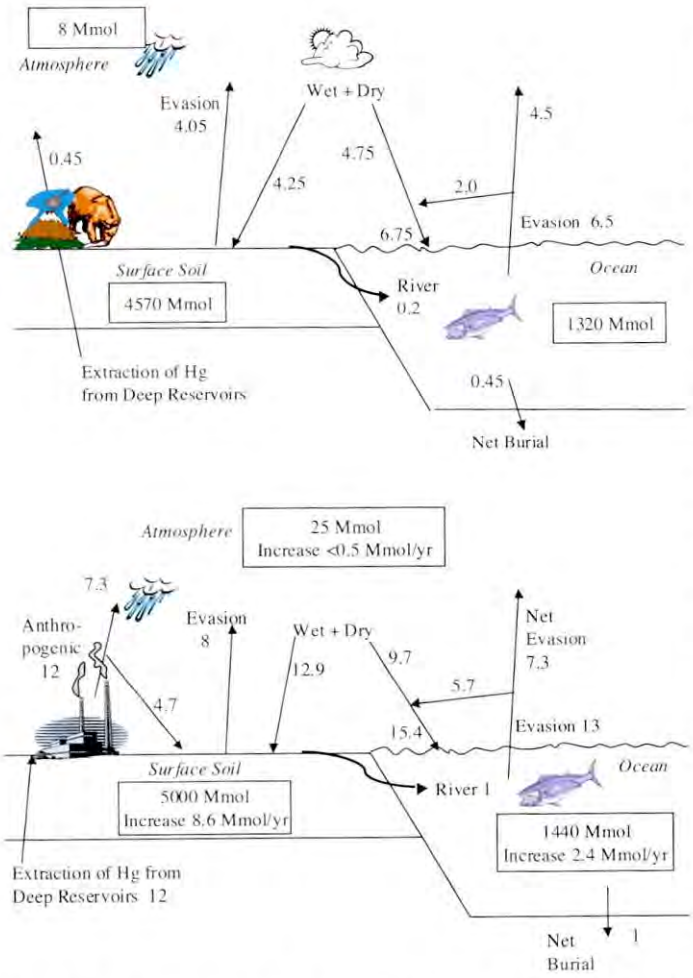

Figure 1. Global mercury fluxes and reservoirs in preindustrial (upper) and modern (lower) times; all fluxes are in Mmol y^{-1} (from Mason and Sheu (12): the term "evasion" from land should be "emission"). Reproduced by permission of the American Geophysical Union.

was dominated by natural evasional fluxes. A comparison of these 2 models (modern and preindustrial) suggests that about one half of the current evasional fluxes from land and ocean is ultimately of anthropogenic origin. This Earth-surface recycling of anthropogenic Hg accounts for about a third of the total present-day atmospheric inputs, whereas new point-source releases and natural emissions (land and ocean) each represent another third.

The models assume no change in atmospheric residence time; however, it is possible that increased concentrations of atmospheric ozone have contributed to higher rates of contemporary Hg deposition, at least regionally (17) and perhaps globally (however, other oxidants such as OH radicals may not have increased similarly). If so, this would imply a slower atmospheric cycling in preindustrial times (relative to today), which, in turn, would require a proportionally larger pool of atmospheric Hg than currently represented in the preindustrial model.

The magnitude of change in Hg deposition to remote terrestrial areas caused by human activity is fairly well constrained by lake-sediment records, with an uncertainty of about 30% (e.g., a 3 ± 1× increase).With far fewer records, trends in the Southern Hemisphere are less secure, and additional sites are needed to solidify current estimates for this part of the world. Moreover, some European peat-core records and a single ice-core profile from the Freemont Glacier (Wyoming, US) suggests an earlier and larger increase in deposition (10×–30×), implying significant preindustrial changes in Hg emissions, possibly related to earlier Hg mining and metallurgy (18–20).

In the final analysis, core records are complex processors of atmospheric Hg and need to be interpreted with caution. Sedimentary processes, such as post-depositional migration and diagenesis, dating uncertainties, and natural variability among (and within) sites, make it unwise to conclude too much from one, or even several, cores. This is especially true for longer (millennial) time scales where factors controlling Hg burial may change.

Deposition Response to Reduced Emissions on the Global Scale

Because of emission of Hg from large stores at the Earth's surface, reductions in anthropogenic inputs will not produce a linear decrease in Hg deposition, especially at remote locations that are dominated by input from the global Hg pool. Deposition declines are expected to lag emission reductions, depending on the response time of the global Hg reservoirs to changing inputs. Because of rapid cycling of Hg in the atmosphere and surface ocean (0–100 m), these pools should achieve a steady state within a few years with respect to reduced loadings. The larger and more slowly cycling Hg reservoir in the ocean thermocline could delay this response by 1 to 2 centuries. However, the net effect of this lag on atmospheric fluxes should be small given the modest (20%) anthropogenic Hg enrichment of the thermocline (17).

The response of the terrestrial soil pool to reduced Hg emissions is less clear. Although the net change in Hg storage in the reservoir of the global soil profile from anthropogenic inputs is probably not large (10% for the entire industrial period), most of the actively cycling Hg likely resides in the top few millimeters to centimeters of soil (21) where anthropogenic enrichment is far greater than for the soil reservoir as a whole (1). If Hg emission rates from this near-surface pool are high, then changes in terrestrial emissions could lag reductions in anthropogenic loading by many years. On the other hand, if terrestrial surface emission is driven largely by Hg^0 production from contemporary deposition—as suggested by recent experiments with Hg isotope additions (22–24)—then the surface soil reservoir may actually represent a long-term sink for Hg inputs, and Hg surface emission can be expected to follow deposition reductions in step.

The response of Hg deposition rates to emission reductions close to anthropogenic sources is expected to be much more rapid than that at remote locations, largely because near-field Hg deposition is probably dominated by local Hg(II) emissions. Indeed, there is good historical evidence from lake-sediment records for rapid and large (30–50%) declines in Hg deposition from urban areas in the United States and Europe (14, 25–26), as well as more widespread declines for the northeastern United States (27). These declines occurred over the last 1 to 3 decades and correspond with known reductions in local and regional Hg emissions for the same areas. However, archives provided by lake sediments are not especially responsive to changes in Hg deposition in remote locations over the recent past (e.g., ~10–20 years). The natural variability, typically low sedimentation rates, and slow Hg transport through catchment soils (27) limit their applicability within this recent time frame.

Final Perspectives on Global Cycling

It is evident that essential global-scale information for the modern cycling of Hg and its behavior and fate over the past 3 decades is lacking. New and innovative approaches are needed to address potential inconsistencies and to answer the question

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as to whether Hg is increasing or decreasing in the biogeochemically active reservoirs at the Earth's surface. For example, Mason and Gill (28) recently reported that Hg in the upper ocean near Bermuda declined between 1979 and 1999–2000. Asian Hg emissions are suggested to be increasing in a dramatic fashion, yet neither the long-term measurements of Hg in rain by the Mercury Deposition Network (MDN) network in North America, nor those of total gaseous mercury (TGM) in the air at Mace Head, Ireland, yet reflect the signal from these new emissions (29, 30). The relative constancy of the modern atmospheric pool at 25 Mmol is an important constraint for global Hg simulations and budgets. This boundary condition places limits on the likely ranges for fluxes and their uncertainties. However, there are documented recent increases in the oxidation potential of the atmosphere, which might account, at least in part, for the discrepancy between TGM trends (steady or decreasing) and Hg emission inventories (increasing). It is reasonable to ask just how uncertain are the inventories.

ATMOSPHERIC EMISSIONS AND SOURCE ATTRIBUTION

Background

Modeling the transport and deposition of Hg to receptors and understanding its source attribution requires improved data on the spatial and temporal trends in atmospheric emissions. Mercury is emitted into the atmosphere primarily as elemental gaseous Hg° and divalent Hg(II) species that can take both gaseous or particulate forms. Emission of gaseous dimethylmercury is also known to occur [e.g., in landfill gas (31) and possibly from the ocean surface (32)]. Although it may explain observations of methylmercury in rain and snow, dimethylmercury represents a very small fraction of emitted Hg. On a global scale, the atmospheric Hg cycle is dominated by elemental Hg vapor. However, the emission speciation of Hg is determined by the source characteristics and consequently shows large regional variability. Atmospheric fate and, especially, Hg deposition are strongly linked to the speciation of Hg released into the atmosphere. The deposition pathway is dominated by the flux of emitted Hg(II) compounds [termed "reactive gaseous mercury" (RGM)], and the oxidation of emitted elemental Hg vapor to Hg(II).

Anthropogenic Emissions and Regional Trends

Accurate emission inventories of Hg are critical, because they are used to drive atmospheric chemical-transport and source-receptor models for the prediction of Hg deposition rates. These atmospheric Hg models are particularly sensitive to the accuracy of the emissions speciation profile, because the different species can be wet or dry deposited at vastly different rates.

As a result of the global interest in Hg, we are beginning to develop a much clearer picture of trends in emissions. Between 1990 and 2000, the trends were derived for continental anthropogenic Hg emissions as summarized in Table 2.

Uncertainties

Policy decisions based on source attribution must be developed with a good understanding of the levels of uncertainty that are involved. The uncertainty of emission estimates assigned to individual source categories in this work are listed in Table 3 (8). A conservative assessment of uncertainties in both emission factors and speciation (9) was used to assess overall uncertainties in the continental inventories for the year 2000 (Table 4).

Among Asian countries, China is now regarded as the largest anthropogenic Hg emission source. However, there are very limited data on Hg emission factors from different source categories in China, which are typically adopted from studies conducted in Europe and North America with similar sources. Because the processes and pollution-control techniques used in the industrial activities in China may differ dramatically from those in developed countries, the Hg emission factors in China could significantly differ from the implied assumptions.

Emission inventories are necessarily retrospective and, therefore, current emissions are less certain. However, it can be assumed that current North American and European emissions will remain constant or decrease slightly over time. Emissions from the other continents, especially with highly dynamic economies, may continue to increase.

Agreement of Emission Inventories with Observational Data

Variations in the global atmospheric Hg pool and its interhemispheric distribution provide essential information regarding the cycling of Hg and its relations to natural and anthropogenic sources, and act as a key constraint for models. Although atmospheric Hg monitoring stations have increased, the database is sparse, especially in remote locations. Slemr et al. (33) attempted to reconstruct the worldwide trend of atmospheric Hg (TGM) concentrations from long-term measurements of known documented quality at 6 sites in the Northern Hemisphere, 2 sites in the Southern Hemisphere, and multiple ship cruises over the Atlantic Ocean made since 1977 (Fig. 2).

Table 2. Mercury emission trends 1990–2000 (all emissions are approximate).*

Continent	1990–1995	1995–2000
Africa	Twofold increase	Constant level: 400 t y^{-1}
Asia	Twofold increase	Constant level: 1200 t y^{-1}
Australia	Twofold increase	Constant level: 100 t y^{-1}
Europe	Decline from 550 to 300 t y^{-1}	Further reduction to 200 t y^{-1}
North America	Slight decline	Fairly constant: 200 t y^{-1}
South America	30% increase	Fairly constant: 80 t y^{-1}

* From Ref. 9.

Table 3. Uncertainty of Hg emission estimates by source category.*

Industrial source	Uncertainty
Stationary fossil fuel combustion	±25%
Nonferrous metal production	±30%
Iron and steel production	±30%
Cement production	± 0%
Waste disposal and incineration	as much as 5×†
Mercury and gold production	? †

* From Ref. 9.

† The information on emissions from Hg production, gold production, waste disposal, and the use of Hg for various purposes is incomplete.

Table 4. Uncertainty of Hg emission estimates by continent.

Continent	Uncertainty (±%)
Africa	50
Asia	40
Australia	30
Europe	30
North America	27
South America	50

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The authors interpreted this information to suggest that the TGM concentrations in the global atmosphere had been increasing since the first measurements in 1977 to a maximum in the late 1980s, after which Hg concentrations decreased to a minimum in 1996 and then remained constant at a level of about 1.7 ng m^{-3}. It was also hypothesized that the observed temporal profile was primarily the result of the trends in global Hg use, supply, and emissions. The temporal trend displays a qualitative relation to changes in the global Hg supply (e.g. see Swain et al., in this issue, p. 46). Although these estimates do not quantitatively reflect the reported emission decrease between 1990 and 1996 (34), this trend may be complicated by trends in the oxidation potential of the atmosphere (33).

This analysis includes several significant assumptions, which make a null hypothesis (i.e., there has been little change in TGM since 1977) equally likely. First, it is assumed that stations on or bordering the Atlantic Ocean provide trend data for TGM that is representative of the worldwide pattern. Second, the principal data for the hypothesized global increase in TGM during the mid-1980s is provided by 1 station, Rörvik, near Gothenburg, Sweden, which is likely to have been affected by regional Hg sources (i.e., eastern Europe before the breakup of the USSR) and perhaps should not be included in a global sense. As noted in Slemr et al. (33), major declines in Hg wet deposition were reported for southwestern Sweden from 1987–1989 to 1997 and are attributed to the large reductions in Hg emissions from eastern Europe (35). If Rörvik were excluded, then the data suggest there has been little change in total gaseous Hg levels in the atmosphere between 1977–1978 and 2002 for most of the stations bordering the Atlantic Ocean.

Additional support for the null hypothesis is provided by TGM measurements for the Southern Hemisphere. The Southern Hemisphere data are seen in the upper portion of Figure 2, along with additional results from several investigations conducted in the south and equatorial Pacific Ocean between 1980 and 1990 (5, 36–38). These combined TGM results for the Southern Hemisphere do not suggest that there has been much change in TGM levels in the global remote atmosphere over the past 25–30 years. Although it may appear that these competing hypotheses on atmospheric TGM levels in recent times would be disconcerting, this situation is not unusual and often aids the development of research strategies. For example, the value of long-term atmospheric Hg monitoring stations and the need for additional sites is obvious, especially in the remote Southern Hemisphere.

Other atmospheric chemistry measurements at the receptor can also provide useful information on source attribution. Mercury isotope mass spectrometry is now capable of detecting differences in the natural isotopic signatures between different sources of Hg (different cinnabars, biota *versus* coal, or sediments of different origin), though the method has yet to be tested in ambient air (39). Similarly, such methods as real-time aerosol mass spectrometers hold promise for rapid measurement of tracers of convenience for use with near real-time measurements of Hg° and Hg_p near source areas. Similar methods proved useful in source attribution (40). Another useful comparison involves measurements of the ratio of 2 or more gaseous compounds or substances in the atmosphere to derive quantitative information on emission source strengths. It assumes that the emission ratio of 2 compounds at their source is reflected in their relative ambient concentration at a distance after mixing and dilution. This approach further assumes *i)* no chemical or physical losses (or comparable losses), *ii)* a constant emission source with fixed emission ratio, and *iii)* a constant background concentration. Although the measurements encompass almost exclusively elemental Hg whose anthropogenic emission is only a portion of the entire emission loading, and

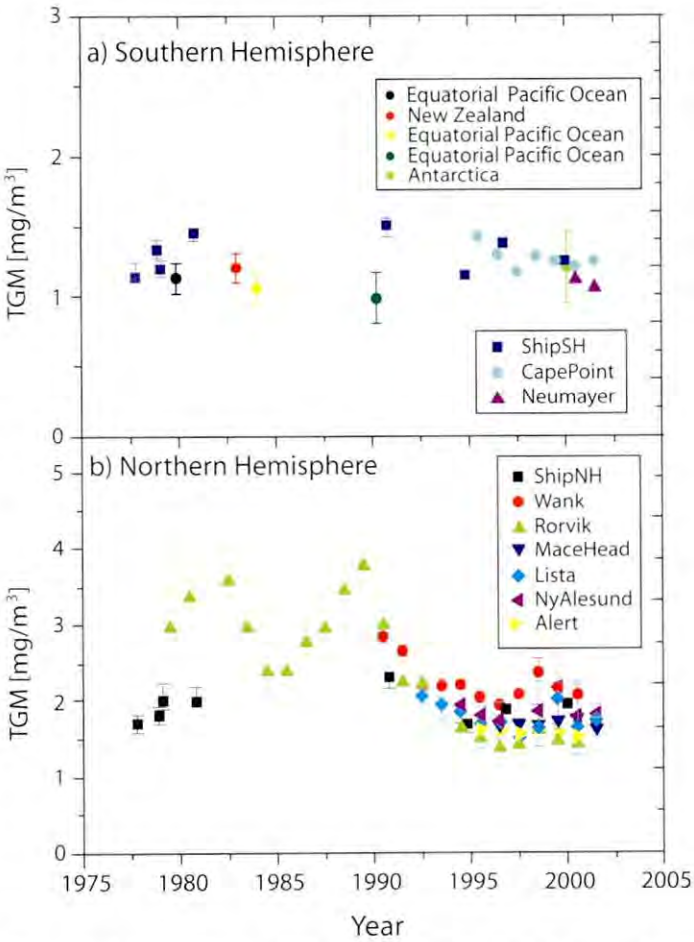

Figure 2. A comparison of trends in concentrations of airborne Hg° measured at several land-based monitoring stations and on ship cruises in the southern and Northern Hemispheres (also see text for additional data sources); adapted from Slemr et al (33).

emissions estimated from correlations with trace gases also include emissions of geologic and anthropogenic Hg from natural surfaces, these are promising approaches worthy of further investigation. For example, Hg emissions calculated from such ratios are in reasonable agreement with the estimated anthropogenic total Hg emissions for Asia (41).

Natural Emissions

Mercury is emitted to the atmosphere from numerous sources, and current anthropogenic and natural sources are thought to contribute about equally to the global tropospheric pool. Gustin and Lindberg (10) suggest that early estimates of natural source strengths, such as background soil and geologic sources, are low. Other new studies also suggest that volcanic emissions may also have been underestimated (42). The assessment of truly natural Hg sources and their relative importance compared with direct anthropogenic emissions and emissions from natural surfaces is a fundamental problem in studying the global cycling of Hg. Natural emissions cannot readily be reduced or controlled, and, when deposited, Hg, whether from a natural or anthropogenic source, enters an environmental compartment, it is subject to the same transport and transformation processes as Hg from other sources.

Emissions from Natural Surfaces

Approaches for measuring Hg° flux include controlled laboratory exposure chambers, field soil and foliage chambers, and

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micrometeorological methods (43). These methods operate across various spatial scales and have the ability to quantify bidirectional fluxes over land and water, but they can be subject to artifacts (44). Recent developments in analytical and flux technology (4, 45, 46) generated significant new data that influence the conventional wisdom regarding the global Hg cycle. For example, oxidized Hg (which exhibits a shorter lifetime), is readily reduced to Hg° after deposition to various surfaces (4, 47), giving it the ability to be rapidly emitted back to the atmosphere as a gas. New estimates of short-term surface emission rates measured with Hg isotopes suggest that on the order of 5–40% of deposition can be emitted soon after deposition, depending on the surface (22, 23). This rapid "redox recycling" gives Hg the appearance of exhibiting a longer lifetime in the atmosphere than it actually has, because all "sinks" are not permanent.

Wildfires have the potential to volatilize previously deposited Hg in terrestrial organic pools into the atmosphere, and several new studies are developing data from which global emission estimates are possible (48). Even when using the more conservative estimates (range, ~400-1300 T y^{-1}), this must be considered an important global source, perhaps the major source in the Southern Hemisphere. When considering the huge pools of anthropogenic Hg currently "stored" in surface soils (>500 000 T by some early estimates), land use and climate changes, as well as fire also have the known ability to mobilize some portion of these pools, both to air and water (49). For example, the "reservoir effect" increases Hg mobilization into biota after flooding of forests (50).

A similar situation exists for the oceans, where evasion is sensitive to altered climate and circulation patterns. Mercury evasion over water can be measured by using chambers, but it is more commonly estimated by using a thin-film model with measurements of dissolved gaseous Hg. However, our understanding of the evasion process limits the accuracy of the evasion models, especially under moderate or greater wind/wave conditions. New approaches should be developed to accurately extrapolate from measured small-scale fluxes in both oceanic and terrestrial systems. The experience of research communities studying the global cycles C, N, and S suggests that scaling is best done with models based on the mechanisms that control exchange over water, soil, and vegetation. More thorough studies of these mechanisms for Hg remain a major research need.

THE ROLE OF CHEMICAL TRANSFORMATION MECHANISMS IN SOURCE ATTRIBUTION

Background

Because of its very low solubility, Hg° is removed slowly from the atmosphere via wet and dry deposition, whereas Hg(II) species (i.e., RGM) are removed rapidly because of their high solubility and reactivity with surfaces. Thus, chemical transformations between these 2 oxidation states directly affect the atmospheric lifetime of Hg: oxidation of Hg° to Hg(II) increases atmospheric deposition, whereas reduction of Hg(II) to Hg° decreases atmospheric deposition. It, therefore, is essential to understand the reduction/oxidation (redox) processes that govern the speciation of Hg in the atmosphere and the sorption of gaseous Hg species to particulate matter (PM), because gas/particle conversion also affects Hg deposition.

Our overall knowledge of the chemistry of atmospheric Hg today (51, 52) compared with a decade ago is depicted in Figure 3 (1, 53). Our knowledge has improved significantly, but major uncertainties remain, which affect our ability to predict source-receptor relations.

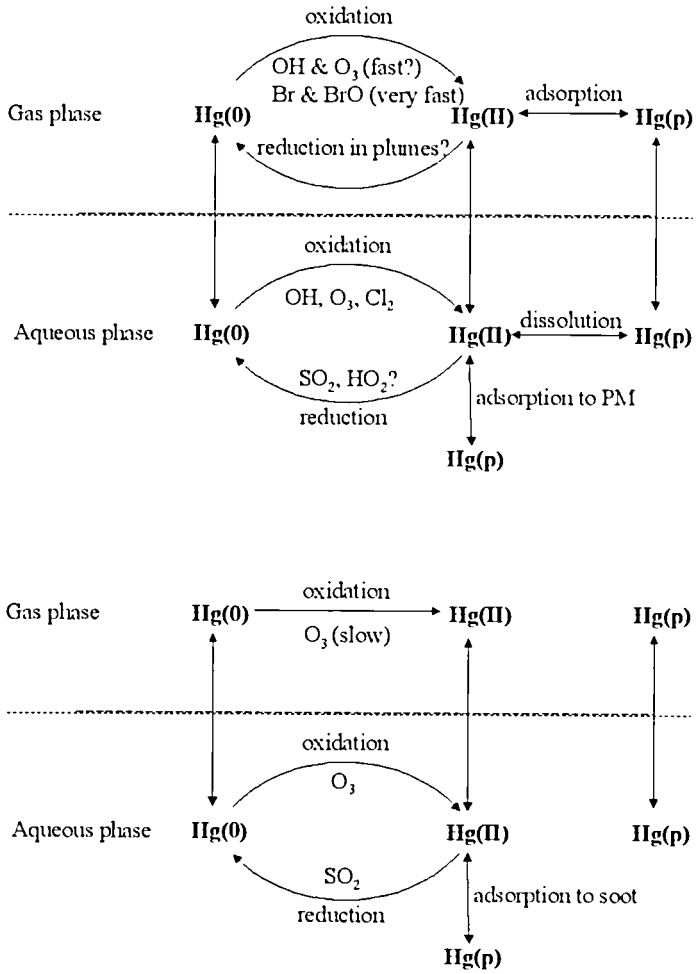

Figure 3. Evolution of knowledge of atmospheric Hg chemistry over the past decade: top, current (see text for details); bottom, former (1).

Oxidation of Hg°

In 1994, the only major Hg° oxidation reactions that were identified were the gas-phase (54) and aqueous-phase (55) reactions with ozone (O_3). The kinetics of those reactions led to a global chemical half-life of Hg° of about 1 y. Since then, several gas- and aqueous-phase reactions were identified that exhibit much faster oxidation kinetics, depending on the environmental conditions. New laboratory data on the gas-phase O_3 reaction suggested a faster kinetics (56), although it is possible that this fast kinetics results from heterogeneous reactions on the reactor vessel walls (57). Such heterogeneous reactions may also occur in the atmosphere (e.g., on particles and surfaces), but their effect on O_3 reaction kinetics is unknown. Major new gas-phase reactions have also been identified, including reactions with OH, Br, and BrO. The fast kinetics of the OH reaction reported by 2 laboratories would lead to a chemical half-life of Hg° of about 2 to 4 months (58, 59); however, theoretical calculations raise questions regarding the laboratory conditions, and this reaction may be significantly slower in the ambient atmosphere (57).

The oxidation of Hg° by halogens was investigated to explain the Hg depletion events observed in the Arctic (see below). The oxidation of Hg° by chlorine compounds was found to be slow, but the oxidation reactions with Br and BrO (and possibly other bromine species) suggested by several Arctic research teams were confirmed to be fast (60). Such reactions lead to a chemical half-life <1 d and can significantly affect the atmospheric residence time of Hg at the poles, possibly also in the MBL (6)

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and in the upper troposphere, where those radicals are present in sufficient concentrations (61). Two new Hg° oxidation reactions in the aqueous phase have also been identified, including those with OH radicals (62, 63) and dissolved chlorine (Cl_2) (64). The latter reaction could significantly affect Hg° oxidation in the MBL because of its fast kinetics.

Reduction of Hg(II)

In 1994, only 1 major pathway (aqueous-phase reaction of Hg^{2+} with dissolved SO_2) was identified for the reduction Hg(II) to Hg° in the atmosphere (65). The kinetics of this reaction were revised (66, 67), and its importance is limited by the fact that the formation of other complexes, e.g., $HgCl_2$, occurs preferentially. The aqueous-phase reaction of Hg(II) with HO_2 was proposed as a significant Hg(II) reduction pathway (68); however, the validity of this experimental result was proved incorrect (69). Evidence of measurable Hg(II) reduction in power plant plumes was obtained from Hg speciation measurements at ground-level sites downwind of power plants, from aircraft in power plant plumes, and from dilution chambers approximating plume dispersion at several power plants (70, 71).The exact mechanism leading to gaseous Hg(II) reduction is unknown and requires further investigation.

Adsorption of Hg Species to Particulate Matter

The original concept of Hg(II) adsorbing to soot in cloud droplets was extended to adsorption of Hg(II) to PM in both the gas phase and the aqueous phase (72). In addition, Hg° may adsorb to PM, and both Hg° and Hg(II) may undergo heterogeneous reactions at the surface of particles (73). Large uncertainties remain regarding *i)* the partitioning of Hg species between the bulk gas/aqueous phase and PM, and *ii)* the kinetics of heterogeneous reactions.

Polar Hg Depletion Events

Field measurements in the Arctic and the Antarctic during polar spring showed that airborne Hg° can be rapidly converted to Hg(II) (Fig. 4). These events coincide with O_3 depletion and the presence of halogen species, and new experimental evidence suggests that Br and BrO, which participate in O_3 depletion, lead to this oxidation of Hg°. The oxidation products involve Hg(II) and possibly unstable Hg(I) intermediates (74, 75), and these species rapidly deposit to snow. Some deposited Hg is reduced to Hg° and emitted back to the atmosphere; however, there is clear evidence of Hg deposition, and model calculations suggest that depletions could account for ~100–150 Mg y^{-1} of Arctic deposition (4, 52, 76). Empirical reconstruction of atmospheric Hg fluxes with dated lake sediments from the North Slope of Alaska (Toolik Lake Field Station environs, 68°38' N, 149°38' W), suggests that these phenomena increased current deposition at Toolik by about 75% (77).

Marine Boundary Layer

The chemistry of Hg may be affected in the MBL by the presence of sea salt halogens and deliquescent particles. In the presence of sunlight and low temperature, rapid oxidation of Hg° by halogen species may occur in the open ocean MBL leading to Hg(II) formation and deposition to the water surface (6, 11). Mercury can later be emitted again as Hg°, thereby leading to a dynamic exchange of Hg between the MBL and marine waters. In warm coastal waters such as off the coast of Florida, Hg(II) formation may not be as significant (78), possibly because the kinetics of Hg(II) formation via halogen chemistry is favored at lower temperatures. Therefore, Hg

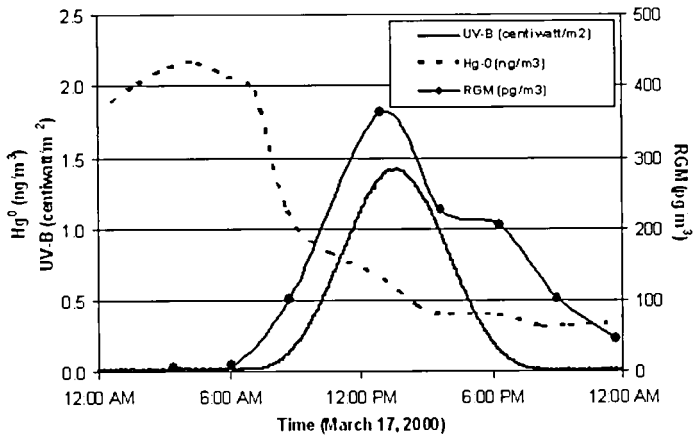

Figure 4. Time series of gaseous Hg measured at Barrow, Alaska, during a mercury depletion event, illustrating how Hg° is rapidly converted to Hg(II) (RGM) during full sunlight (4).

chemistry within the MBL is likely to vary spatially and temporally, depending on meteorological and sea conditions.

Upper Atmosphere

The chemical speciation of Hg differs in the free troposphere and lower stratosphere from that in the boundary layer, because of lower temperature, the lack of Hg(II) removal by dry deposition, and different concentrations of oxidants (e.g., halogen atoms and ozone in the stratosphere) in the upper atmosphere. For example, high concentrations of Hg(II) were observed in the free troposphere (79) and particulate-bound Hg(II) has been observed to dominate Hg speciation near the tropopause (61). Because the intermediate HgBr species tends to decompose back to Hg(0) as the temperature increases (75), the oxidation of Hg(0) to Hg(II) by Br is favored at the low temperature of the upper atmosphere; this could explain, in part, the high Hg(II) concentrations observed near the tropopause. Gas-to-particle conversion of Hg(II) may also be favored by low temperature, which would explain the observation of particulate-bound Hg(II) in the upper troposphere and lower stratosphere (61). Such Hg(II) present in the upper atmosphere could be removed via wet deposition during deep convective storms.

Atmospheric Hg and Climate Change

Temporal variations in deposition and in the cycling of Hg between the atmosphere and ecosystems can result from changes in emissions of Hg, as well as of other atmospheric contaminants (e.g., NO_x, SO_2), and from climate change. Primary effects include increases in air and sea temperatures, wind speeds and variation in precipitation patterns, and secondary effects that are related to increases in O_3 concentration and aerosol loading, a decrease of sea ice cover in the Arctic, and changes in plant growth regimes. All these effects will influence the atmospheric residence time of Hg; its deposition; and, ultimately, its dynamics on local, regional, and global scales. This will lead to increases in the overall uncertainty in the assessment of ambient concentrations and deposition rates of Hg compounds (11).

DEPOSITION MECHANISMS AND SOURCE ATTRIBUTION

Background

One of the most critical measurements needed to understand the Hg biogeochemical cycle and to verify source attribution models is its rate of atmospheric deposition. The measurement

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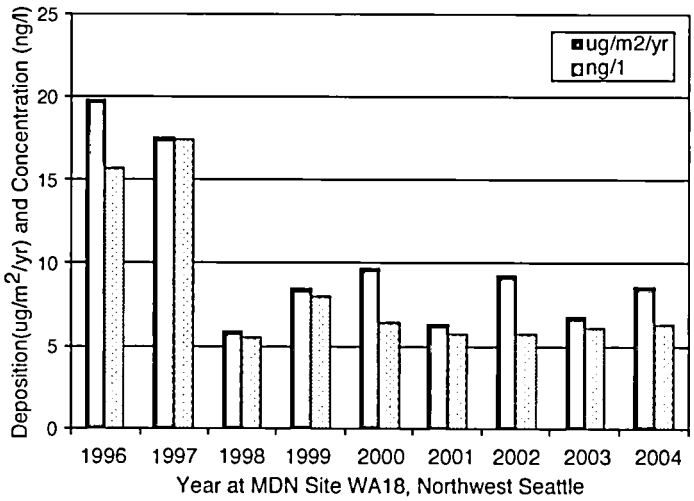

Figure 5. The closure of several medical waste incinerators in Northeast Seattle in 1998 was captured by the MDN WA18 site, where average deposition dropped from 18.7 ± 1.6 $\mu g\ m^{-2}\ y^{-1}$ in 1996–1997 to 7.9 ± 1.5 $\mu g\ m^{-2}\ y^{-1}$. The trend analysis indicates that it is significant ($p < .001$) (107).

of Hg wet deposition is now straightforward, and accepted standardized methods are used by national (e.g., the US Mercury Deposition Network, in operation since about 1995) (29, 80) and regional networks (35, 81, 82). In contrast, the accurate measurement (and modeling) of Hg dry deposition is difficult and remains the most challenging gap in our understanding of fluxes.

Wet Deposition

The global pool of atmospheric Hg is continually being depleted by wet deposition. Except for the polar regions, and possibly the MBL, the primary mechanisms for wet deposition are in-cloud oxidation of Hg^0 by O_3, and the gas-phase oxidation of Hg^0 by OH and O_3 followed by cloud-droplet uptake. Particle-bound Hg also contributes to wet deposition through cloud-droplet activation and precipitation scavenging. Wet deposition rates can be enhanced close to major Hg sources by the direct cloud-droplet uptake of emitted Hg(II) and Hg_p (35, 83). The dominant form of Hg in wet deposition is dissolved and particulate Hg(II). Methylmercury is commonly found in rain and snow at concentrations ~0.5–2.5% of total Hg, with excursions of >10% for single events (32), and some investigators have argued that wet deposition could be an important source of methylmercury to certain ecosystems (84).

Dry Deposition

Dry deposition by definition occurs continually except during periods of rainfall (typically, ~2–5% of the time for the mid-latitudes). Thus, Hg dry deposition is occurring up to 98% of the time and can be a significant input to aquatic, marine, and terrestrial ecosystems. Dry deposition of Hg occurs via two processes. One is the direct deposition of the gas-phase compounds Hg^0 and RGM and, to a lesser extent, deposition of atmospheric particles containing Hg. Vegetation covers a significant portion of the Earth's surface and is known to enhance dry deposition. The rate-limiting step for gas-phase Hg^0 dry deposition to plants is stomatal uptake (85). However, for RGM, which has a high sticking coefficient, the rate-limiting step is the turbulent transport of gases to the leaf surface (this would also hold for gaseous dimethylmercury). For Hg_p, impaction and sedimentation are the controlling mechanisms. All of these processes are challenging, both to measure and model, and they depend on meteorological phenomena, such as temperature, surface wetness, and wind speed, as well as surface geomorphology. Considerable work remains to be done to accurately quantify Hg fluxes over plant canopies, especially because they can also emit Hg^0 (86).

Unique Environments

New data suggest that local atmospheric conditions lead to enhanced deposition in several unique ecosystems. Enhanced polar springtime deposition to snow is driven by the reactive halogen cycle (87, 88). Some of the deposited Hg is emitted again during snowbelt, and some remains in runoff (4, 32, 89). In the MBL, observations and new models indicate fast *in situ* photochemical production of RGM (6, 11), which may lead to enhanced deposition in coastal and estuarine areas (12). In urban areas, where there are abundant Hg-containing particles generated by mobile and industrial sources, both wet and dry Hg deposition rates can be dominated by Hg_p removal (90, 91). Finally, based on a few direct measurements, but significant research on other atmospheric constituents, montane ecosystems receiving direct cloud-water deposition will likely exhibit enhanced Hg loading relative to nearby lower elevation sites (18, 92, 93). These observations suggest that gradients in total Hg deposition may be greater than perhaps previously understood.

Measurement Methods and Uncertainty

Wet Deposition. Four key components are required for accurate Hg wet-deposition measurements: *i)* a trace-clean sample train with an HCl preservative in the collection bottle, *ii)* a temperature-controlled collector with an automated rain sensor, *iii)* a rain gauge to verify the rainfall depth collected, and *iv)* a cold-vapor atomic fluorescence spectrometer system to measure the Hg concentration (80, 82, 94). Manual event-based Hg wet-deposition sampling is also possible for intensive studies (83). When comparing results of the analysis of the same sample by qualified independent laboratories, the uncertainty is typically less than 10% (e.g., co-located sites in the National Atmospheric Deposition Program (NADP) MDN showed median absolute differences in Hg wet-deposition rates of 6–14% (95). Measured wet-deposition rates are not likely a major factor in the uncertainty in source attribution analyses.

Dry Deposition. Unlike wet deposition, where the flux occurs over a short period at relatively high and easily detectable rates, dry deposition occurs over much longer periods at relatively low rates. Further complicating the development of measurement methods is the fact that there are at least 3 distinct atmospheric species of Hg that contribute to dry deposition, particulate-bound (Hg_p), RGM, and Hg^0. Not only do these 3 species have different chemical and physical properties that affect their dry deposition rates, they are also present at <1 ppt ($Hg^0 \sim 5 \times 10^6$ molec cm^{-3}, and Hg_p or RGM $\sim 5 \times 10^4$ molec cm^{-3}). Finally, once dry deposited, Hg may be emitted again in the same or a different chemical form (22, 96, 97), which further increases the measurement challenge when considering the sample integration time and frequency.

There are 3 methodologies for the estimation of Hg dry-deposition rates. Each carries unique advantages, yet also exhibits critical limitations that are important to understand but are too numerous to list [see the review by Hicks et al. (98)]. The first methodology is termed "direct" and includes surrogate surfaces, dew fall, litterfall (LF), throughfall (TF), and subtracting wet-only deposition from bulk deposition (78, 99–101). The second methodology is "inferential" and uses Hg species air concentration and meteorological measurements to model dry-

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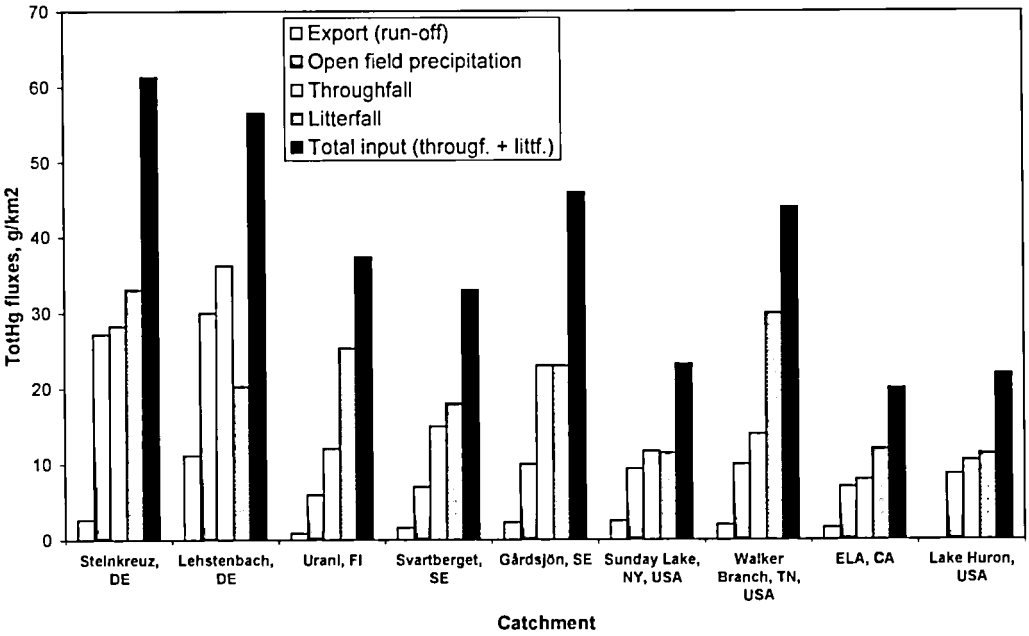

Figure 6. Annual input and output of total mercury in 9 forested catchments. The black bar is the total input estimated from the sum of LF + TF. Dry deposition can be estimated as the difference between the estimated total input (black bars) minus the measured open field precipitation (gray bars). Compiled by Munthe et al. (118) from published studies in Sweden, Germany, Finland, the United States, and Canada.

deposition rates (85, 102). The third approach is "micrometeorological" and includes gradient, modified Bowen ratio and relaxed eddy accumulation methods (46, 96, 103, 104). These methods must still be considered as experimental when applied to Hg, and flux validation is a major research need.

Observations and Trends

Wet Deposition. The atmospheric Hg record in Sweden is of sufficient quality and length to provide the clearest example on a regional scale of the link between anthropogenic emission reduction and decreasing air concentration and deposition. The rapid Hg emission reduction in northern Europe, because of the economic and political changes occurring in the early 1990s, resulted in ~40% decrease in Hg wet deposition for southern Sweden, as reflected by decreasing air concentrations, wet-deposition rates, and sediment core trends (35).

Over the last decade, North America has the most extensive measurements of Hg in wet deposition, primarily from the MDN, Canadian Atmospheric Mercury Measurement Network, and smaller research networks (29, 81, 82, 105, 106). Two sites with about a decade of precipitation data showed the following: no trend at Underhill, Vermont (1993–2003) (81) but significant decreases in wet deposition of Hg in Seattle (1996–2004) (107), coincident with decreasing emissions from local waste incineration sources (Fig. 5). As of 2006, the MDN has 93 sites in operation, with 48 operating for ≥5 y. With the advent of new Hg air-emission regulations in Canada and the United States, the MDN represents a means to measure the efficacy of future regulatory changes. This will be especially true with the recent addition of MDN sites in predicted source-influenced locations, including several with event-based sampling.

Dry Deposition. There are few measurements of Hg dry deposition in the literature (101, 108, 109), and the only large-scale estimates of Hg dry deposition come from regional and atmospheric models (110–113). Direct measurements of Hg° dry deposition generally come from studies of Hg° emission from terrestrial landscapes (96, 114), most often at very low rates at night or under conditions of elevated Hg° concentrations in laboratory studies (86). The limited field studies and modeling of the stomatal uptake suggest that foliar uptake of Hg° is characterized by deposition velocities (V_d) on the order of 0.01–0.05 cm s^{-1} (85, 103). Although these values suggest a slow uptake, the concentration of Hg° is large enough relative to other Hg species (generally representing >95% of total airborne Hg) that dry deposition of Hg° can be significant.

Complicating matters is the fact that the exchange of Hg° in vegetation is bidirectional, with Hg° exhibiting a compensation point, similar to the behavior of ammonia in green plants (86), and some have argued that there is no net accumulation of Hg with this process. However, laboratory studies clearly indicate that exposure of vegetation to elevated levels of Hg° results in a net accumulation of Hg in plant tissue (115).

Using measured values of V_d, estimates of dry deposition suggest that foliar uptake of Hg° (if retained and not emitted again) would rival or surpass wet deposition even at near-background concentrations (101). Though rarely measured in direct deposition studies, RGM and Hg_p are readily dry deposited, further adding to the dry flux. Reactive gaseous Hg is especially important, because of its solubility, and the few direct measurements published suggest a high dry-deposition velocity, similar to nitric acid vapor (>1.0 cm s^{-1}) (104, 116), whereas the V_d for Hg_p is only marginally larger than that of Hg°, suggesting generally low dry-deposition fluxes.

There are no long-term measurements of both wet and dry deposition for direct comparison. However, if one accepts the premise that LF and net TF (NTF; the flux in TF minus that in rain) reflect the net dry deposition of Hg (25, 94, 100, 117), then some crude comparisons can be made. Munthe et al. (118) compiled measurements of ecosystem fluxes of Hg at watersheds in Europe and the United States that operated for ≥1 y. In every case, the estimates of dry deposition from LF + NTF were ≥ fluxes of total Hg in wet deposition (Fig. 6). Clearly, there is a significant need to develop accepted methods to measure dry deposition and to carry out long-term intercomparison studies with all methods at sites with ongoing wet-deposition measurements. These would be especially valuable at locations with a history of wet-deposition data, such as at the US NADP MDN sites. Without these data and a better understanding of dry-deposition mechanisms, we are missing a major component of the source attribution puzzle.

MODELING ATMOSPHERIC TRANSPORT AND ATMOSPHERIC MERCURY SOURCE-RECEPTOR RELATIONS

Atmospheric Hg simulation and source attribution models that operate on a wide range of spatial scales were developed by

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various research organizations (110, 112, 113, 119–124). Local-scale air-quality models (e.g., ISC-3) do not generally treat slower chemical reactions, such as those known to affect Hg but instead focus on the physical dispersion characteristics of exhaust plumes (125). For applications involving Hg, they usually simulate transport and deposition based on the chemical and physical forms of the Hg as emitted from the sources being studied (126). However, some local-scale models were developed that include chemical transformation of Hg (e.g., ROME, TRUE) (127, 128). Models capable of simulating Hg on regional and global scales (e.g., CMAQ, TEAM, GRAHM, HYSPLIT, ADOM) treat chemical and physical transformations, in addition to transport and deposition (52). As noted previously, advances in computing equipment and technology allowed the development of models treating chemical and physical processes with much greater detail. Multiscale air-quality models are now capable of simulating atmospheric flows throughout the troposphere (0–15 km height), with horizontal resolutions as fine as only a few kilometers for continental-sized domains, or resolutions of tens to hundreds of kilometers over global domains. These models are also capable of simulating time periods of 1 y or more with hourly resolution.

Regional-scale models are dependent on accurate representations of the air concentrations of the various forms of Hg at their lateral boundaries, along with similar estimates of the concentration of important Hg reactants, like ozone and hydroxyl radical. Most models assume no transfer of air or pollutants across the top boundary, if that boundary is near the top of the troposphere. These lateral boundary conditions are sometimes defined to be variable in time and are derived from global-scale model simulations (124, 129). Otherwise, estimates of the average concentrations over time are used (e.g., monthly, seasonal). In the past, these boundary conditions for Hg concentrations were often held constant in the vertical dimension but that is changing now that important vertical gradients in the various species of Hg were observed (79). Other critical inputs of such models include emissions and meteorology. Uncertainties in those inputs are reflected in the model simulation results and it, therefore, is essential to minimize those input uncertainties.

Receptor modeling techniques also were applied to Hg (40, 130). Those techniques use ambient concentration data for several chemical species (i.e., Hg and other gases) to identify the relative contributions of various source categories (some techniques assume typical chemical profiles for selected source categories, whereas others identify those chemical profiles from the raw data). Receptor modeling techniques are sometimes combined with meteorological information to construct back-trajectories and to complement the source category information with geographical information (131). Thus, better identification of possible source contributions can be obtained. Receptor modeling techniques are based on the principle of mass conservation and do not account for chemical transformation. They are most useful to ensure that all possible sources were included in the emission inventory and to corroborate the results of the regional-scale models described above. The combination of various modeling techniques can bring some valuable "weight of evidence" for source (or source region) attribution.

POLICY RELEVANCE AND THE ISSUE OF UNCERTAINTY IN SOURCE ATTRIBUTION

The question of source attribution is central to the development of control policy for environmental Hg by any authority. Without an understanding of the sources responsible for an observed Hg deposition, it is impossible to develop a cost-effective emission control policy to reduce that flux. A critical element of our question is the term "with confidence." Our scientific understanding of any environmental process is never absolute and is rarely complete. Environmental protection policy is almost always developed with some level of inherent uncertainty and political controversy. Environmental Hg control policy is no exception.

When considering the key question in a positive light, there are indeed some locations where we can say "with confidence" that atmospheric Hg deposition is predominately from local sources or predominately from the global pool. It is well known that some forms of atmospheric Hg are rapidly deposited by both wet and dry processes, and emissions of these forms of Hg, especially near ground level, are responsible for a large portion of the observed Hg deposition in the surrounding area. These more reactive forms of Hg (e.g., RGM and some Hg_p) are usually deposited from the atmosphere before they can travel long distances. However, there are special atmospheric conditions under which they can travel longer distances. Therefore, the existence of reactive Hg in a particular air sample does not necessarily indicate the existence of a local emission source. Significant air concentrations and deposition of reactive Hg can also be the result of atmospheric chemical reactions involving less reactive forms of Hg transported from distant sources (e.g., elemental Hg). In remote locations far removed from emission sources of reactive Hg, we can conclude with confidence that this latter process is primarily responsible for the observed deposition. In this case, any significant reduction of Hg deposition would have to come from global-scale Hg emission reductions and/or a reduction in the oxidation potential of the atmosphere at that remote location.

When regarding the differentiation between natural and anthropogenic sources, it is generally accepted that reactive Hg emissions arise primarily from anthropogenic sources. Natural additions to the Hg cycle and emissions of previously deposited Hg that facilitate the cycle are primarily in the less-reactive elemental form (some important exceptions may exist). Therefore, undisturbed natural sources of atmospheric Hg seldom have a localized impact, and their effects occur mostly on the global scale in concert with the atmospheric chemical reactions mentioned above. The same is true for the large-scale surface emission of Hg, which has both a natural and an anthropogenic component. A differentiation between these 2 components of Hg recycling is necessary to accurately assess the overall importance of natural *versus* anthropogenic sources, but this differentiation is generally difficult.

We can say with confidence that elemental Hg is more inert and can be transported globally and that oxidized Hg compounds are more reactive and travel much shorter distances before depositing. However, there remains considerable uncertainty regarding the chemistry and reaction rates that govern the oxidation state of atmospheric Hg. There is also some uncertainty about the rate at which emissions of reactive Hg deposit and the distance at which they contribute to the total deposition flux. These uncertainties in rates reduce the confidence with which we can assess the contribution of anthropogenic sources on the intermediate, regional scale. For example, more rapid oxidation of elemental Hg, either in air or in atmospheric waters, increases the relative importance of natural and anthropogenic emissions of elemental Hg on the global scale to the Hg deposition at any given location. More rapid deposition of emitted reactive Hg decreases the distance over which these emissions travel and influence total deposition flux. The assumption of rapid Hg oxidation and rapid deposition of reactive Hg emissions is illustrated in Figure 7 (Hypothetical Case 1). If the opposite were true, slower Hg oxidation and slower deposition of reactive Hg emissions, this

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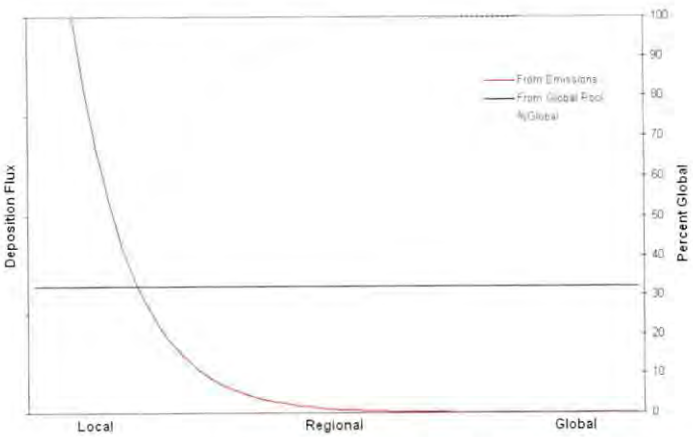

Figure 7. Illustration of Hypothetical Case 1: Rapid atmospheric oxidation of mercury and rapid deposition of reactive mercury emissions. Note that the percentage of deposition from global influences is high at the regional scale of transport from anthropogenic sources. Transport scale = X axis.

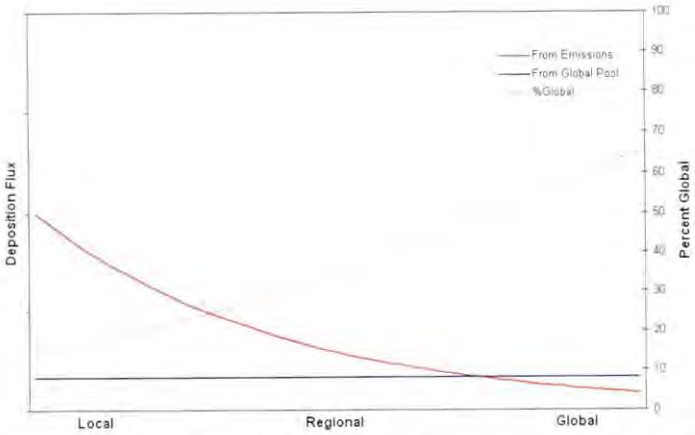

Figure 8. Illustration of Hypothetical Case 2: Slow atmospheric oxidation of mercury and slow deposition of reactive mercury emissions. Note that the percentage of deposition from global influences is low at the regional scale of transport from anthropogenic sources. Transport scale = X axis.

would result in a quite different result, as shown in Figure 8 (Hypothetical Case 2). In this hypothetical case, more of the reactive Hg that dominates the deposition flux must come directly from emission sources rather than from atmospheric oxidation of Hg^0. Without a confident understanding of atmospheric Hg chemical kinetics and Hg deposition processes, assessment of the relative importance of the intermediate, regional-scale sources is very difficult.

Another issue that must be considered by environmental policy makers is the expected time scale for decreases in atmospheric Hg deposition brought about by proposed regulatory action. Here again, uncertainty about atmospheric Hg chemical kinetics makes a confident assessment difficult. We know that the global emission flux of Hg to the atmosphere must roughly balance with the global deposition flux, because the global-average Hg burden in air is observed to be quite stable, at least on the generally accepted time scales of atmospheric Hg cycling. More rapid oxidation of atmospheric Hg implies a generally higher total deposition flux that must be balanced by the sum of all anthropogenic emissions and emissions from natural surfaces.

Although we have a relatively good understanding of primary anthropogenic emissions, this is not the case for natural and recycled (Hg emitted after being deposited) Hg emissions. Assuming vigorous atmospheric oxidation of Hg implies that vigorous emissions from natural surfaces must be compensating. In this case, reducing or even eliminating anthropogenic emissions of Hg would have a smaller than expected effect on the total emission flux. Significant reductions in deposition would not occur until a commensurate fraction of the Hg now involved in the global-scale cycle is somehow sequestered (e.g., in geologic formations, much as it was before human industrialization). However, if atmospheric oxidation of Hg is slow, then more of the observed deposition flux must be coming from anthropogenic emissions of reactive Hg. In this case, reductions in anthropogenic emissions of Hg would lead to a more rapid decrease in the total Hg deposition flux. The importance of resolving these issues for policy development is clear. Their associated uncertainties critically impact our ability to predict the outcome of any proposed emission-control strategies.

SUMMARY AND CONCLUSIONS

During our synthesis of progress in the last decade, the panel altered its opinion on the question of source attribution. Initially, the panel was divided among "yes," "no" (we cannot yet attribute sources at any location) and "maybe." By the end of our synthesis, the panel agreed that the answer is a "qualified yes." The qualification is strongly dependent upon what level of uncertainty we can ultimately accept in our answer. We agreed that the uncertainty is strongly dependent upon scale and that our question as stated is answerable with greater confidence both very near and very far (remote) from major point sources (if we accept the "global pool" as a recognizable "source"). Unfortunately, many regions of interest (from an ecosystem-exposure standpoint) lie in between, where source attribution carries the greatest degree of uncertainty. This situation will improve with a better understanding of atmospheric Hg chemical kinetics and deposition processes. The panel also agreed that all aspects of predicted climate changes have the potential to affect the Hg cycle in ways that will increase the uncertainty of source attribution.

The panel felt that answers are forthcoming from the two primary approaches: direct measurements and models, both of which are subject to uncertainty. Application of measurement approaches by using isotopic signatures and tracers of convenience (e.g., trace metals, carbon monoxide) require intensive high-resolution sampling efforts but hold promise as analytical technology improves. Although these approaches are typically applied to atmospheric concentrations, they may be adapted to provide data on source attribution for both wet and dry deposition, and they are most likely to succeed close to well-characterized sources.

The modeling "frameworks" for simulating pollutant transport, transformation, and deposition are quite mature and ready to apply for Hg, if we knew the values of the required modeling parameters (e.g., chemical kinetics rates). The models are there, but their accuracy is limited. The ability to determine the accuracy of current models is severely limited by the lack of dry deposition measurements; uncertainties in the altitudinal distributions of Hg species; and uncertainties in atmospheric, especially heterogeneous, reaction rates, emission Hg speciation, Hg emission rates in developing nations, and model parameterizations for dry deposition and meteorology.

Significant Advances

The panel noted several important advances in our understanding of atmospheric Hg over the past decade that have important implications for the question of source attribution. These include:

- An understanding of the broad uniformity of Hg deposition changes in remote areas ($3 \pm 1\times$ increase);

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- improved laboratory data on reaction kinetics and new field measurements, indicating that fast oxidation of Hg° is occurring in certain environments (e.g., seasonally at the Poles, in the MBL, and in the upper troposphere);
- improved knowledge of the lifetime of Hg°, which is not a fixed value but may vary significantly with season, latitude, and ambient concentration of atmospheric oxidants;
- widespread availability of automated instrumentation for measuring atmospheric concentrations, speciation, and surface fluxes;
- improved modeling at local, regional, and global scales;
- establishment of large-scale national Hg wet-deposition networks; and
- development of methods for accurately quantifying the isotopic signature of Hg.

Research Needs

The panel also noted the importance of further research on several fronts:

- The effects of all aspects of climate change, including wildfire, on the atmospheric Hg cycle must be understood, because this phenomenon could be the primary factor influencing the future distribution of Hg on global and regional scales;
- improved understanding of the role of halogen and OH radical chemistry involving atmospheric Hg°;
- improved measurement and modeling of the 2 largest global fluxes: Hg dry deposition and evasion across the air-water interface (especially in marine systems);
- a global Hg monitoring network is needed to assess long-term changes in atmospheric Hg concentrations and wet + dry deposition;
- an assessment of the magnitude of change in the oceanic Hg reservoir since pre-industrial times, especially in the surface ocean (0–100 m);
- an accurate determination of whether Hg is currently increasing or decreasing in the biogeochemically active reservoirs at the Earth's surface;
- a better understanding of anthropogenic Hg emissions from developing nations is needed to improve the accuracy of global Hg cycling models, and
- accurate scaling-up of air/surface exchange of Hg is not possible from small-scale field measurements alone but must also rely on large-scale modeling, which requires an improved understanding of the physical and biological mechanisms that influence these processes.

Conclusions

Finally, the major conclusions of our synthesis include the following:

Long-term Anthropogenic Changes

- Deposition, even at the most remote locations, contains a significant fraction of anthropogenic Hg.
- We are confident of the overall global increase in Hg deposition since the Industrial Revolution (by a factor of 3 ± 1).
- It is possible that the global cycling of Hg has been impacted not only by anthropogenic emissions but also by increases in the atmospheric ozone concentration since the Industrial Revolution.

Current Emission and Deposition Trends

- There has been no discernible net change in the size of the atmospheric pool of Hg in the Northern Hemisphere since the mid-1970s.
- North American and European emissions are decreasing, whereas those in Asia and Africa are increasing, but the latter changes are less well constrained.
- Decreases in local emissions have resulted in decreases in near-field deposition.

Chemical Transformations and Cycling

- There have been important developments in analytical and sampling methodology, which provide a much clearer picture of atmospheric Hg species behavior.
- Hg fluxes by dry deposition, terrestrial surface emission, and ocean evasion are globally significant but represent important gaps in measurements, data, and understanding.
- New understanding of Hg cycling in the MBL, and much more rapid cycling in the atmosphere (fast chemistry leading to a more heterogeneous atmosphere than previously imagined), is making modeling results more uncertain.
- The relative importance of Hg redox chemistry varies dramatically in space and time, and uncertainties in those redox reactions and physical transformations significantly impact the ability to develop source/receptor relations.

Modeling and Uncertainty

- There is a large discrepancy between progress made in measurement technology and the development of accurate global-emission inventories.
- Higher-resolution emission estimates would facilitate improved model verification.
- Hg isotope signatures and tracers of convenience hold promise for source attribution, particularly at the local scale.
- The uncertainty associated with current models must be relayed to and understood by policy makers.

The Bottom Line

The answer to "can we attribute sources to Hg deposition?" is a qualified "yes."

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Recovery of Mercury-Contaminated Fisheries

In this paper, we synthesize available information on the links between changes in ecosystem loading of inorganic mercury (Hg) and levels of methylmercury (MeHg) in fish. Although it is widely hypothesized that increased Hg load to aquatic ecosystems leads to increases in MeHg in fish, there is limited quantitative data to test this hypothesis. Here we examine the available evidence from a range of sources: studies of ecosystems contaminated by industrial discharges, observations of fish MeHg responses to changes in atmospheric load, studies over space and environmental gradients, and experimental manipulations. A summary of the current understanding of the main processes involved in the transport and transformation from Hg load to MeHg in fish is provided. The role of Hg loading is discussed in context with other factors affecting Hg cycling and bioaccumulation in relation to timing and magnitude of response in fish MeHg. The main conclusion drawn is that changes in Hg loading (increase or decrease) will yield a response in fish MeHg but that the timing and magnitude of the response will vary depending of ecosystem-specific variables and the form of the Hg loaded.

INTRODUCTION

There is considerable evidence that natural background levels of mercury (Hg) in the environment have increased in the past 150 y. This evidence comes from studies of lake and ocean sediment profiles, peat bogs, and ice cores, in both remote and industrialized regions. These data point toward a general increase of Hg levels concurrent with industrialization as a result of both the intentional use of Hg and from combustion of Hg-containing fossil fuels, gold mining, and processing of ores. Because Hg is transported on hemispheric or global scales via the atmosphere, contamination is not limited to locations in the vicinity of emission sources and includes remote ecosystems. In remote regions, increases are in the range 2- to 5-fold over preindustrial values, whereas locally affected sites can have levels increased by a factor of 10 or more (1, 2).

Although most Hg in the environment is inorganic, some is converted to the highly toxic monomethyl mercury (MeHg), which bioaccumulates in fish. Fish are an important dietary source in many developed and developing nations. In some communities, fish, shellfish, birds, and marine mammals constitute critical components of the diet or local economies. MeHg concentrations in fish are commonly high enough to represent a risk to the health of the fish-eating communities (3–6). Elevated MeHg concentrations may impair the long-term sustainability of aquatic ecosystems and have deleterious effects on the behavior and health of associated wildlife. Recent studies suggest that trophic transfer of Hg also occurs in terrestrial food chains (7, 8).

A critical question currently being investigated is, "Will efforts to reduce inorganic Hg inputs and mobility in the environment reduce fish Hg concentrations?" Several confounding issues have historically complicated efforts to answer this question. Individual ecosystems have different abilities to translate a given Hg load into MeHg concentrations in biota due to a range of factors affecting Hg cycling and bioaccumulation (e.g., hydrology, water quality, trophic structure, temperature) and the uncertain effects of terrestrial ecosystems on the timing and magnitude of delivering atmospheric Hg deposition to waterbodies. As a result, waterbodies close to each other and receiving the same atmospheric Hg deposition loads often have very different MeHg concentrations in biota. The conversion of inorganic load to MeHg can also be affected not only by the absolute amount but also by the form of Hg loaded to an ecosystem in cases of point-source contamination.

Despite the above complexities, it is reasonable to expect that changes in inorganic Hg loading will affect MeHg concentrations in ecosystems. A few studies provide information on historical levels of MeHg in humans and aquatic top predators (whales, seals) (9) and show that MeHg levels have increased significantly since preindustrial times. Although associated with some uncertainty, these results indicate that anthropogenic emissions in the last century have caused general increases in not only total Hg in the environment but also MeHg in freshwater and marine food chains.

In this paper, our hypothesis is that the concentration of MeHg in fish will decrease in response to decreases in Hg loading. However, the magnitude and timing of this response will vary with the type of Hg contamination and ecosystem characteristics. To test this hypothesis, we explore the current knowledge on environmental cycling and levels of Hg in the environment focusing on:

- Controls on net methylation of inorganic Hg in the environment.
- Examples of documented fish responses to both increases and decreases in Hg loading in different ecosystems receiving direct input via industrial discharges.
- Results from spatial and temporal analyses of Hg contamination in ecosystems receiving mainly atmospheric Hg.
- Results from experimental ecosystem manipulations and loading experiments.

Finally, we discuss the critical factors affecting the magnitude and timing of fish Hg responses to changes in loading of Hg and attempt to explain the variability.

CONTROLS ON ECOSYSTEM SENSITIVITY TO MERCURY LOADING

In this section, we examine the main processes involved in transporting and transforming inorganic Hg to MeHg in fish, with a focus on freshwater ecosystems. We define ecosystem sensitivity to Hg loading as the ability of that ecosystem to transform inorganic Hg load into MeHg in biota. The evaluation thus includes the movement and net methylation of Hg in watersheds and the aquatic ecosystem, as well as uptake into food chains. Direct atmospheric input of MeHg is not considered in this synthesis, because with a few notable exceptions, *in-situ* production is the dominant MeHg source to watersheds. By analyzing components of this cycling separately, we aim to illustrate the complexity of ecosystem cycling of Hg and MeHg and to describe the main controlling factors in each step.

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Factors Affecting Transport of Mercury Through Watersheds

In addition to direct atmospheric deposition, watersheds provide an input of Hg to freshwater ecosystems via runoff. Watershed characteristics control the retention of Hg in the terrestrial compartment and transport of Hg to sites of methylation via complex hydrologic, chemical, and biologic processes. Some of these key characteristics are:

- The size and topography of watersheds and the watershed-to-surface water area ratio (affecting the residence time and flow pathways of runoff and the total amount and yield of Hg to waterbodies).
- Land cover (affecting dry deposition rates and the degree of interaction between water and both methylating and non-methylating soils).
- Land use (affecting the transfer of particulate Hg load to sites of methylation and the transfer of MeHg to the aquatic compartment (10–14) and, in the case of forestry, increased transfer of MeHg from the terrestrial compartment to the aquatic (15, 16).

Watershed size and watershed-to-surface water ratios are perhaps the most important determinants of Hg retention and delivery to aquatic ecosystems. As watershed sizes increase, yields per unit area usually decline (17–19). Based on a synthesis of data from watersheds of varying size, Grigal (20) suggested that there is actually an overall decrease in Hg flux and concentrations in runoff with increasing watershed size due to less efficient transport and increased loss processes. Very large watersheds may be less responsive to changes in atmospheric Hg load than those in which the watershed area is smaller in comparison to the lake surface area. Land-use changes, anthropogenic disturbances, or local Hg sources may result in watershed Hg export many-fold larger than expected (20). Watersheds subject to significant transport of sediment in surface runoff, such as agriculturally dominated systems, contribute larger fractions of Hg to sites of methylation in aquatic sediments and wetland soils (14) than do forested watersheds (11, 21). However, because this Hg is largely associated with particulate matter, it may be less bioavailable than dissolved inputs of Hg. Other land-use impacts on Hg load and MeHg in fish have been noted, particularly in landscapes with erodable soils or developed landscapes (10), and controls on land-use practices may be used to reduce MeHg in biota. Using a modeling approach, Roué-Le Gall et al. (22) showed that when watershed characteristics such as those discussed above were coupled to information about the food web in 45 lakes, the sensitivity of biota to MeHg contamination could be predicted qualitatively (high to low).

The common factor among all of the watershed characteristics identified above is soil cover. Soils retain Hg in watersheds, and this retention is strongly coupled to organic matter (23, 20), where Hg is stored either through sorption of Hg deposited directly from the atmosphere or is associated with organic matter derived from the forest canopy or floor. Even in landscapes characterized by shallow and discontinuous soils, newly deposited Hg appears to be nearly completely retained in the short term (see The Gårdsjön Roof Study). The magnitude and timing of the release of Hg from this pool is controlled by the rate of decomposition of the soil organic matter pool and physical removal of the soil itself through erosion. The current lack of insight in the dynamics of Hg release from soils makes the greatest contribution to the uncertainty in the quantitative prediction of the magnitude and timing of the effects of a change in Hg load on MeHg in biota.

Factors affecting net mercury methylation. For Hg loads to ecosystems to result in MeHg in biota, inorganic Hg must be converted to MeHg. Net MeHg production is affected by the concentration of bioavailable Hg and by a complex system of environmental factors, most important:

- *Spatial controls:* the areal extent and connectivity of methylating and demethylating zones within the ecosystem.
- *Biogeochemical controls:* the bioavailability of Hg and MeHg to methylating and demethylating bacteria, and the relative activity of those organisms.

Spatial controls on net methylation. The ecosystem compartments that support net MeHg production are reasonably well defined, except for the oceans. Because net methylation is favored under anoxic conditions (24), the areal extent of wetlands and hydric (water saturated, partially anaerobic) soils (11, 21, 25) is a strong determinant of MeHg export from watersheds. Wetlands can be particularly active zones of MeHg production (e.g., the Florida Everglades [26], tidal marshes [27]). Methylation in bogs (28), fens (29, 30), and riparian zones (19, 31, 32) is highly dependant on flow paths and hydrologic connectivity that govern the location and extent of zones of microbial activity. Forest disturbance (clear cutting and mechanical damaging to soil cover by tractors) has been shown to increase the load of MeHg to the aquatic ecosystem (15, 16), including biotic concentrations (33, 34), thus providing a potential for more direct anthropogenic influence on MeHg levels in aquatic biota.

In freshwater aquatic ecosystems, warm, shallow, organic-rich lake sediments are often important zones of net methylation (35–37) and therefore a real extent of these sediments in a lake impacts the conversion of Hg to MeHg (38). MeHg concentrations and production rates in sediments are generally maximal at or near the sediment surface (39). The presence of anoxic bottom waters in stratified lakes significantly enhances net MeHg bioaccumulation, often through *de-novo* MeHg production in bottom waters (40, 41) and also through enhanced flux of MeHg from sediments to the anoxic water column.

Flooding increases the production of MeHg due to increased rates of decomposition of terrestrial organic matter, which stimulates development of poor oxygen conditions and activity of Hg-methylating bacteria (30, 42, 43). Soil drying and rewetting cycles also strongly affect Hg methylation (44). Oxidation of reduced sulfur during drying leads to a pulse of sulfate reduction and Hg net methylation when soils rewet (45). Changes in water levels in lakes and wetlands can significantly affect MeHg levels in fish (45, 46). Several examples of this behavior from reservoirs are also available (42, 47–51).

Although the MeHg contamination of ocean fish has the greatest impact on human health globally (52), information on the sources of MeHg to estuaries, coastal zones, and particularly the oceans is limited. In the limited number of estuaries studied, MeHg production in sediments appears to be its major source (28, 53–55). Although net methylation in salt marshes can be high (56, 57), their importance to estuarine and coastal MeHg budgets has not been quantified. In coastal zones, shallow sediments (54, 58–61) also appear to be the major sources of MeHg. There are also unidentified sources of marine MeHg in the high Arctic (62). The source of MeHg to open ocean fish remains a subject of debate (63), with little available information, although there is a documented accumulation of MeHg in deep ocean waters with an unknown source (64).

Factors affecting the uptake of MeHg by the food web. There are important physical, biological, and chemical controls on MeHg bioaccumulation that dramatically affect the transfer of Hg load into MeHg in fish and subsequent predators.

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Box 1
Biogeochemical controls on net methylation in aquatic ecosystems

In addition to the impact of Hg loadings on MeHg production, some key biogeochemical cycles also have large effects on net MeHg production:

- **Sulfur.** The Hg and sulfur cycles are intimately linked, thus linking acid precipitation to the Hg cycle. The balance between sulfate and sulfide is a key control on Hg net methylation rate in many ecosystems. Sulfate stimulates Hg-methylating sulfate-reducing bacteria, while excess sulfide creates Hg complexes that are not bioavailable for methylation (57, 65, 66). Sulfate stimulation of methylation has been demonstrated in studies that range from pure culture (67–69), to sediment and soil amendments (70–72), to field amendments to lakes and wetlands (73–77). Among these studies, and in the field, the optimal concentration for methylation ranges from 10 to about 300 μM sulfate, while the optimal sulfide concentration is quite low, about 10 μM (26, 78). Factors such as iron and organic matter concentration that affect Hg and S complexation change these optima. Sulfate, along with pH and dissolved organic carbon, have been identified as parameters that relate Hg levels in fish among water bodies (75).
- **pH.** Many studies have linked lake acidity to increased MeHg bioaccumulation (75, 79–81). This observation has important implications for management of fish Hg contamination. Although several mechanisms have been hypothesized, the main process controlling this phenomenon is not evident. This pattern could be driven by effects on bioaccumulation, for example, decreases in aquatic productivity with decreases in pH. However, the effect could also be linked to MeHg production. Acidity linked to sulfate deposition may stimulate the activity of Hg-methylating sulfate-reducing bacteria. Additionally, uptake of Hg by micro-organisms that use facilitated transport for Hg uptake is enhanced with decreasing pH (82).
- **Organic matter.** Both the character and concentration of dissolved organic matter affect the complexation and potential bioavailability of Hg and MeHg for methylation/demethylation (83–85). Higher molecular weight dissolved organic matter can limit Hg availability to bacteria (86), probably through the formation of complexes that are too large to assimilate, whereas small organic ligands can enhance Hg uptake through facilitated transport (87). Recent work suggests that dissolved organic matter is an important ligand under sulfidic conditions, through interactions with Hg sulfur complexes (88, 89). The relationships between dissolved organic matter and methylation need to be further explored before they can be adequately modeled. Further, organic matter in sediments and soils often stimulates the activity of microorganisms, making it an important control on net methylation rates (90).
- **Iron.** Like sulfur and decayed organic matter, the impact of iron on net methylation appears to be concentration and environment dependent (14, 91, 92). Iron may affect net methylation through impacts on Hg complexation, solid phase partitioning, or microbial activity. A few strains of iron(III)-reducing bacteria are now known to be capable of Hg methylation (93, 94), but the impact of iron on Hg net methylation in the environment needs further study.
- **Hg "aging."** Recent Hg-amendment studies in lakes and wetlands show that the bioavailability for methylation of recently loaded Hg differs from that of the overall Hg pool in the ecosystem (95–97). Understanding these differences will be a key to modeling ecosystem responses to changes in Hg load. The "aging" effect could include both chemical changes in Hg complexation and partitioning with time and changes in the relative distribution of newly loaded Hg *versus* Hg that has been in the system for longer periods (e.g., newly loaded Hg could be more or less focused in zones of methylation).
- **Type and activity of bacteria.** Although Hg methylation can be measured in almost any soil or sediment under reducing conditions, only a few sulfate-reducing bacteria (24) and a few closely related iron-reducing bacteria (93, 94) have demonstrated methylation ability in pure culture. There are a variety of microbial demethylation mechanisms, including the *mer* operon detoxification system that is spread widely among microorganisms in contaminated environments, and may serve to limit MeHg accumulation at high Hg concentrations. The oxidative demethylation pathway (98) appears to be the dominant demethylation pathway in sediment and soil environments where the *mer* operon is not operating. It is anaerobic, driven by sulfate-reducing bacteria and methanogens, and linked to one-carbon metabolic pathways rather than to Hg levels. However, the processes of methylation and oxidative demethylation may be differently affected by temperature, organic substrates, and redox chemistry (24). For example, sulfide (57, 58, 99), organic matter (90), and redox (100) appear to affect methylation more than they do demethylation. A better understanding of the controls on demethylation is needed to improve assessments of net methylation in different environments.

Differences in these processes among water bodies may result in varying responses to Hg loading.

Biophysical controls. MeHg uptake is affected by the nature (pelagic *versus* benthic) and structure of the food web (101). MeHg bioaccumulation in lakes occurs through both benthic and pelagic routes. MeHg produced in sediments can be directly accumulated into benthic food webs or flux into overlying water and into pelagic food webs. Stratified lakes where MeHg accumulates in anoxic bottom waters (40, 41) often provide a pulse of MeHg to surface waters and plankton after fall turn over (102). In large lakes and coastal marine environments, MeHg accumulation appears to occur predominantly around near-shore areas of sedimentary MeHg production (55, 100, 103). Zones of MeHg production that are disconnected from aquatic food webs (e.g., isolated bogs) may have little impact on Hg in aquatic biota but can affect terrestrial food webs (104, 105).

Biological controls. Food web structure, fish population age structure, growth rates, and physiological controls on uptake all affect the bioaccumulation of MeHg. Any examination of

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Table 1. Observations of fish mercury concentrations for selected point-source contaminated ecosystems.

Site	Primary Hg source	Fish species	Loading trend	Fish Hg response	Reference
Clay Lake, Canada	Chlor-alkali facility	Walleye *(Sander vitreus)*	10 tonnes of inorganic Hg released 80 km upstream, primarily from 1962 to 1969.	Clear increase in response to contamination and remediation. Fast then slower recovery after remediation (Fig. 1).	118
Haifa Bay, Israel	Chlor-alkali facility	Soldierfish *(Sargocentron rubrum)*	Tonnes of Hg released from 1950s to 1970s.	~80% decline from late 1970s to 1990.	120, 121
Princess Royal Harbour, Australia	Super-phosphate processing plant	Several marine fish species	14 kg y^{-1} for ~30 y, stopped in 1984.	50% decline from 1984 to 1993. Fast response at first, slower later.	122
Lake Vänern, Sweden	Chlor-alkali	Northern pike *(Esox lucius)*	Load ~3 tonnes per year from 1920s to 1960s, reduced to ~3 kg y^{-1} during 1970s to 1980s.	50% decline within a decade, then stabilized.	161
Onondaga Lake, NY, USA	Chlor-alkali	Small-mouth bass and several other freshwater species	75 000 kg discharged in the 1940s to 1970, followed by greatly reduced loading and closure.	No statistically significant decline after load reductions; fish concentrations remain elevated.	162
East Fork Poplar Creek, Oak Ridge, TN, USA	Inorganic Hg used in lithium isotope separation at national security complex in Oak Ridge	Red-breasted sunfish *(Lepomis auritus)*	250 tonnes of inorganic Hg released from 1950s to early 1960s, then an 80% decline in Hg load and concentrations in creek near facility.	After remedial actions, fish Hg decreased at a site near point of release but not a short distance downstream.	125

change in MeHg in fish through time must examine potential concomitant changes in food web and fish population structure. Changes in Hg bioaccumulation patterns after the invasion of a fish species provide an example of how food web structure affects Hg bioaccumulation (106). Food web structure and composition also affect bioaccumulation through food quality (42, 107) and gut chemistry (108) effects on uptake efficiency. Faster fish growth rates have the potential to reduce fish Hg levels when comparing concentrations standardized to a given size of fish (109, 110). Growth differences in multiple levels of the food web (e.g., plankton, prey fish, predatory fish) could result in cumulative growth effects across a food web.

Chemical controls. The bioavailability of MeHg to the plankton and benthic invertebrates at the bottom of food webs is affected by MeHg partitioning and complexation. Higher organisms, such as zooplankton (111) and fish (112), accumulate MeHg mainly from their diet. Thus, accumulation of MeHg by these organisms is affected strongly by bioavailability to lower trophic levels. The direct or indirect inhibition of MeHg accumulation by higher molecular weight dissolved organic matter has been well documented for fish and zooplankton (113). Particulate matter can play largely the same role, sorbing MeHg and making it less available for uptake by lower organisms through dissolved routes. The role of organic matter overall in Hg transport, methylation, and bioaccumulation is complex with competing influences but central to the physical and biogeochemical behavior of Hg in watersheds. For example, dissolved organic carbon in runoff may enhance the delivery of inorganic Hg and MeHg to waterbodies via binding and cotransport but may inhibit the biological uptake of MeHg within a waterbody. An inverse relationship between pH and MeHg bioaccumulation is also well documented, such as for loons (114), benthic invertebrates (115), and fish (116), although pH may affect both methylation and bioaccumulation.

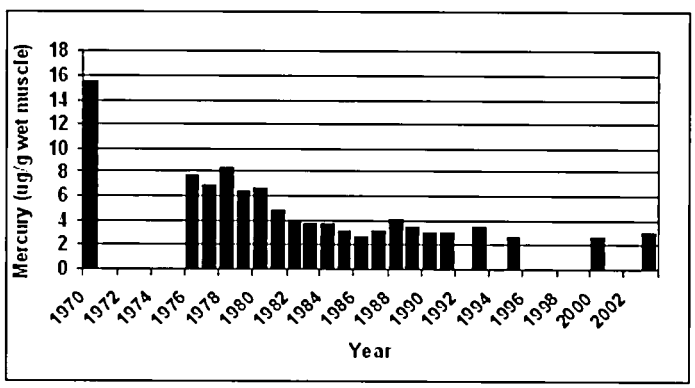

Figure 1. Observed decline in walleye Hg concentrations in Clay Lake, Canada, after reduced releases of Hg from a chlor-alkali facility 80 km upstream (results standardized to 50 cm fish) (118).

Summary of controls. Mercury concentrations in fish can vary by an order of magnitude or more in regions with similar atmospheric Hg deposition rates. This is because there are many factors that affect the movement, methylation, and bioaccumulation of Hg in different environmental settings. The form of Hg being loaded into an ecosystem can also affect the production and bioaccumulation of MeHg. These considerations often obscure a relationship between Hg loading and fish Hg levels when looking at multiple sites, even if the fish Hg levels are standardized to a particular species and size. In the next section, however, we present evidence suggesting that although each ecosystem has its own efficiency to translate an inorganic Hg load into MeHg in biota, Hg loading is still an important determinant of fish Hg concentrations.

EVIDENCE

Here we examine data from remote areas and point-source–contaminated sites that help characterize the response of fish Hg concentrations to changes in Hg loading rates. Studies can be grouped into four categories according to Carpenter (117), three of which are used in this synthesis: i) time series measurements, *ii)* spatial analysis/gradient studies, and *iii)* experimental manipulations.

Time Series Measurements

Temporal data from point-source–contaminated sites. Freshwater and marine sites with large inputs of Hg from local industrial discharges clearly show that Hg loading rates can

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affect fish Hg concentrations (Table 1). Clay Lake, Canada, for example, is downstream of a chlor-alkali facility that released approximately 10 tonnes of inorganic Hg to receiving waters, primarily from 1962 to 1969 (118). Mercury concentrations in walleye in Clay Lake in 1970 exceeded 15 $\mu g\ g^{-1}$ wet muscle (50 cm standard fish), far higher than concentrations measured in systems without point-source Hg inputs. Walleye Hg concentrations quickly declined when Hg releases were curtailed, although the rate of recovery slowed in later years (Fig. 1). These observations can only be explained if Hg inputs associated with the chlor-alkali facility contributed to higher fish Hg concentrations.

Minamata Bay, Japan, is a very well-known case of Hg release to a marine environment in the 1950s but atypically involved the direct industrial release of MeHg (119). Other examples of Hg concentrations changing in marine fish in response to Hg releases or remedial actions associated with point sources include Haifa Bay, Israel (120, 121); Princess Royal Harbour, Australia (122); Dutch Wadden Sea and Ems Estuary (123); and Liverpool Bay, UK (124) (Table 1).

The response of MeHg concentrations at point-source-contaminated sites is not always proportional to the increase in inorganic Hg concentrations and, by inference, the rate of inorganic Hg loading. At East Fork Poplar Creek, Tennessee, USA, 250 tonnes of inorganic Hg were released to water and soils from a U.S. Department of Energy facility in the 1950s and early 1960s. Total Hg concentrations in surface waters and sediments were two to three orders of magnitude higher than natural levels (e.g., >1 $\mu g\ L^{-1}$ in surface waters during the early 1990s), while MeHg concentrations in surface waters and Hg concentrations in fish increased less, by roughly one order of magnitude (125). This suggests that at a sufficient level of contamination either Hg concentration is no longer a limiting factor for gross MeHg production or rates of demethylation are enhanced to a greater degree than methylation, resulting in a less than proportional increase in net methylation. Similarly, decreases in Hg loading are not always followed by proportional decreases in MeHg levels. Elevated discharge of Hg from a chlor-alkali facility to Onondaga Lake, New York, USA, in the 1940s through 1970 contributed to elevated concentrations of Hg in the water column and fish (126, 127). However, cessation of Hg discharge from the facility has not resulted in changes in fish Hg concentrations after 30 y. This condition is likely due to continued high Hg loading in this urban lake due to a combination of external ongoing sources and resuspension of contaminated sediments, high natural sulfate loading, high loading of labile carbon, and highly anoxic conditions in the lower waters that promote high rates of methylation.

In contrast, significant reductions in fish Hg concentrations (50% or greater) have been observed at freshwater and marine sites with point-source contamination within a decade after remedial actions (118, 122–124). This initial response can be followed by a slower second phase with the potential to last decades. Multiple-phase temporal responses may occur due to residual loading (e.g., leaking equipment) or because some compartment(s) in the ecosystem accumulates Hg during the contamination phase (e.g., sediments or terrestrial system) and feed Hg back to the water column and biota for long periods after the original source of contamination is curtailed.

Fish Hg concentration trends in grossly contaminated areas may also be influenced by bioaccumulation of inorganic Hg. Although fish Hg burdens are primarily in the form of MeHg at remote sites (128), it is reasonable to question whether fish in ecosystems receiving very large inputs of inorganic Hg may accumulate larger proportions of inorganic Hg (59, 129). This would result in more rapid declines after load reductions,

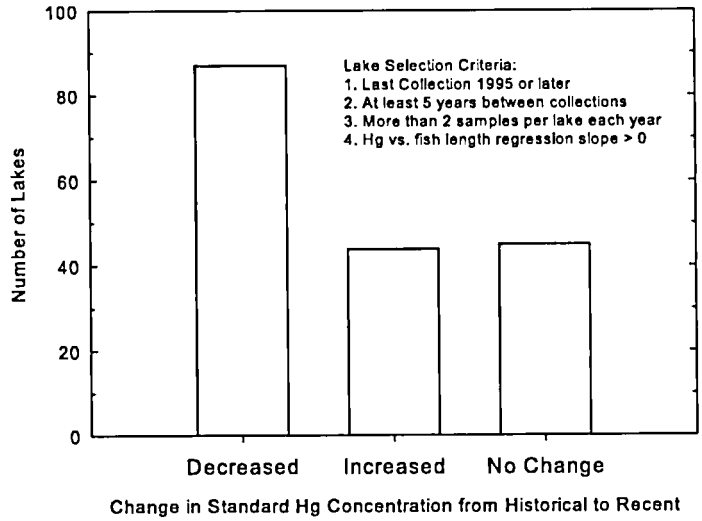

Figure 2. Comparison between recent and historical fish Hg concentrations in 176 lakes in Minnesota. Fish are northern pike *(Esox lucius)* and walleye *(Sander vitreus)* standardized to 55 and 40 cm, respectively (138).

because inorganic Hg is depurated from fish more quickly than MeHg (130).

The use of Hg in artisanal gold mining can be treated as a point-source situation with large amounts of Hg being released to the aquatic ecosystem, but very little data exist on temporal trends of Hg in fish in areas affected by Hg releases from areas affected by artisanal gold mining. This use of Hg is undoubtedly responsible for marked increases of Hg concentrations in soils, water, sediments, and air near mines located in the Brazilian Amazon, but available information on fish Hg provide variable results (131–134).

Response to changes in atmospheric Hg deposition. The response of fish Hg levels to changes in atmospheric Hg deposition is typically more difficult to demonstrate than for point-source contamination because of the smaller degree of change in Hg loading, natural year-to-year variability, and changes to other environmental factors that can affect MeHg cycling to a similar extent as changes in Hg deposition (e.g., water quality, land use, climate, hydrology, trophic structure). Relationships between Hg deposition and fish Hg may also be confounded by delays in delivery of Hg deposition to waterbodies via terrestrial systems. It has long been postulated that terrestrial ecosystems may impose time lags on the transport of atmospheric Hg deposition to lakes. This hypothesis has been corroborated in recent years by forest roof experiments in Sweden (16) and initial results from a whole-watershed Hg-loading experiment (METAALICUS) in Canada (135, 136) (see also Spatial/Gradient Analysis). Finally, there are generally limitations on data describing overall Hg loading to a waterbody, including wet deposition, dry deposition, and terrestrial export (where relevant), in terms of what was measured and for how long.

Recognizing the above challenges, there are examples of Hg concentrations systematically declining in fish for remote individual waterbodies (137) or on average for groups of waterbodies (138, 139), where associated data indicate a decline in wet or bulk Hg deposition. Hrabik and Watras (137) studied a seepage lake not affected appreciably by terrestrial Hg sources (Little Rock Lake, Wisconsin, USA) and concluded that Hg levels in yellow perch (30 g standard) decreased by roughly 30% between 1994 and 2000 due to decreased atmospheric Hg loading (bulk Hg deposition). Johansson et al. (139) reported a 20% average decline in Hg concentrations for northern pike (*Esox lucius*, 1 kg standard) from 42 remote Swedish lakes

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sampled at least 3 y apart, initially between 1981 and 1987, and again between 1988 and 1995. Wet Hg deposition declined by more than 50% in southwest Sweden (less in central and northern parts) during this period. Although fish Hg concentrations declined on average for the study lakes, increases and decreases were both observed for fish Hg levels in individual waterbodies. Although declines in fish Hg and wet Hg deposition were observed for these studies, other factors were also changing that could affect MeHg production (e.g., sulfate loading), confounding the interpretation of the effects of Hg loading. In Sweden during the same period as the above study, sulfate loading and to some extent pH levels in many lakes changed. Hrabik and Watras (137) accounted for pH changes in their analysis, but the extent to which sulfate effects were considered was not clear. In another study, Hg in predatory fish was determined in 176 Minnesota lakes over a period when deposition was known to decrease (138, Fig. 2). A range of trends in fish Hg concentrations (increase in 44, no change in 45, and decreases in 87 lakes) suggested a general link to atmospheric input but also that multiple factors likely affect fish Hg concentrations. These studies demonstrate the need for carefully designed monitoring programs that monitor not just Hg but also sufficient environmental information to help explain very different trends that might emerge among individual sites (140, 141).

The timing and magnitude of the response of fish Hg concentrations in open oceans to changes in Hg loading are not clear. Trends in Hg loading, primary sources of MeHg to the mixed surface layer of oceans, and temporal patterns for marine biota are matters of ongoing investigation and debate. There are no statistically robust datasets documenting temporal trends for Hg levels in marine fishes in the open oceans at warmer latitudes. Kraepiel et al. (63) reported no increase in yellow-fin tuna caught near Hawaii in 1998 relative to 1971 values, although many factors that could have affected Hg in fish, such as growth rates and food chain structure, were not studied, and changes could have occurred in the intervening years. Monteiro and Furness (142) reported increases in Hg concentrations of 65 to 397% for samples collected before 1931 and after 1990 in feathers of seabirds breeding in Azores, Madeira, and Salvages (subtropical northeast Atlantic, 30–40°N, 8–32°W). Temporal datasets exist for Hg levels in beluga, ringed seals, moose, seabirds, and fish from the Canadian Arctic marine environment during the past 20 to 30 y but show conflicting trends (143, 144). Some animal populations exhibited significant increases in Hg concentrations while others did not.

From this analysis of temporal studies we can conclude:

- Mercury loads can affect fish Hg concentrations in cases of point-source contamination, and reductions in Hg loading in such situations usually reduces fish Hg concentrations.
- The long-term magnitude of the response of fish Hg concentrations to changes in atmospheric deposition is not yet clear.

It is also very likely that different environmental settings affect the response time of fish Hg concentrations after changes in atmospheric Hg deposition, but this has not been shown clearly using datasets from natural settings. It has been observed experimentally however, as discussed in the next section.

Spatial/Gradient Analysis

The Nordic countries. Previous studies have demonstrated a link between spatial patterns of atmospheric deposition of Hg and MeHg levels in Swedish freshwater fish (145, 146). An examination of a larger dataset of Hg in fish from more than 1500 lakes in Sweden, Finland, and Norway revealed decreasing levels from south to north but with the high concentrations also found in central Sweden and along the Swedish coast of the Gulf of Bothnia (147). The elevated concentrations in central and northern coastal Sweden may be influenced by historical emissions of Hg to both water and air. However, specific ecosystem characteristics also contribute to the variability in fish Hg between regions. This variation is particularly evident for the southernmost part of Sweden, where very low concentrations of Hg were found in fish despite a larger influence of atmospheric pollution. This region of Sweden has mainly agricultural land use. This land disturbance typically increases lake productivity, decreasing fish Hg concentrations through biodilution (148).

Northeast Ecosystem Research Cooperative. A number of spatial datasets including atmospheric deposition, surface waters, sediments, and biota were recently compiled for eastern North America as part of a Northeast Ecosystem Research Cooperative initiative (149). These observations include 831 surface water, 570 surface sediment, more than 15 000 fish, more than 4,700 bird, and more than 900 mammal records. The spatial patterns and relationships evident for this analysis are similar to others reported elsewhere. None of the organisms studied showed direct relationships with spatial patterns in atmospheric Hg deposition, except blood Hg of Bicknell's thrush, for which Rimmer et al. (8) found a strong relationship with estimates of Hg in litter fall.

Biological Hg hotspots were geographically identified using this large dataset of biotic Hg concentrations. Yellow perch and common loon were chosen as indicator species for human and ecological effects of Hg, respectively (150). Impact thresholds of 0.30 μg/g (wet weight) for yellow perch fillets and 3.0 μg/g (wet weight) in blood for common loons were used to determine the location of biological Hg hotspots. These biological Hg hotspots reflect conditions that influence ecosystem and associated biological response to atmospheric Hg deposition. In particular, three factors were identified that control the formation of biological Hg hotspots, including *i)* local elevated atmospheric Hg deposition due to proximity to emission sources; *ii)* landscape sensitivity associated with forest cover, shallow hydrologic flowpaths, the abundance of wetlands, and unproductive surface waters; and *iii)* water level manipulations.

Links between Hg in fish and Hg wet deposition across the US. Two recent studies by Hammerschmidt and Fitzgerald (151, 152) reported strong relationships between rates of wet Hg deposition, as measured by the Mercury Deposition Network (153), and Hg concentrations in mosquitoes and largemouth bass in North America. Mosquito Hg is relevant to aquatic ecosystems because these animals have aquatic life stages and accumulate primarily MeHg. Hammerschmidt and Fitzgerald (143) found that the state-wide average concentration of MeHg in largemouth bass was significantly correlated with wet atmospheric Hg deposition among many American states. Only the accumulation of large, long-term data sets over wide geographic areas made this analysis possible.

From these studies we can conclude that a growing body of studies show significant correlations between Hg deposition rates and Hg levels in fish.

Experimental Manipulations

There have been a number of recent manipulation experiments that help resolve the relationship between Hg loading and the response of Hg in fish. These experiments have been conducted in freshwaters (and associated watersheds). Most are addition experiments. None of the addition experiments has been conducted long enough to observe a recovery phase.

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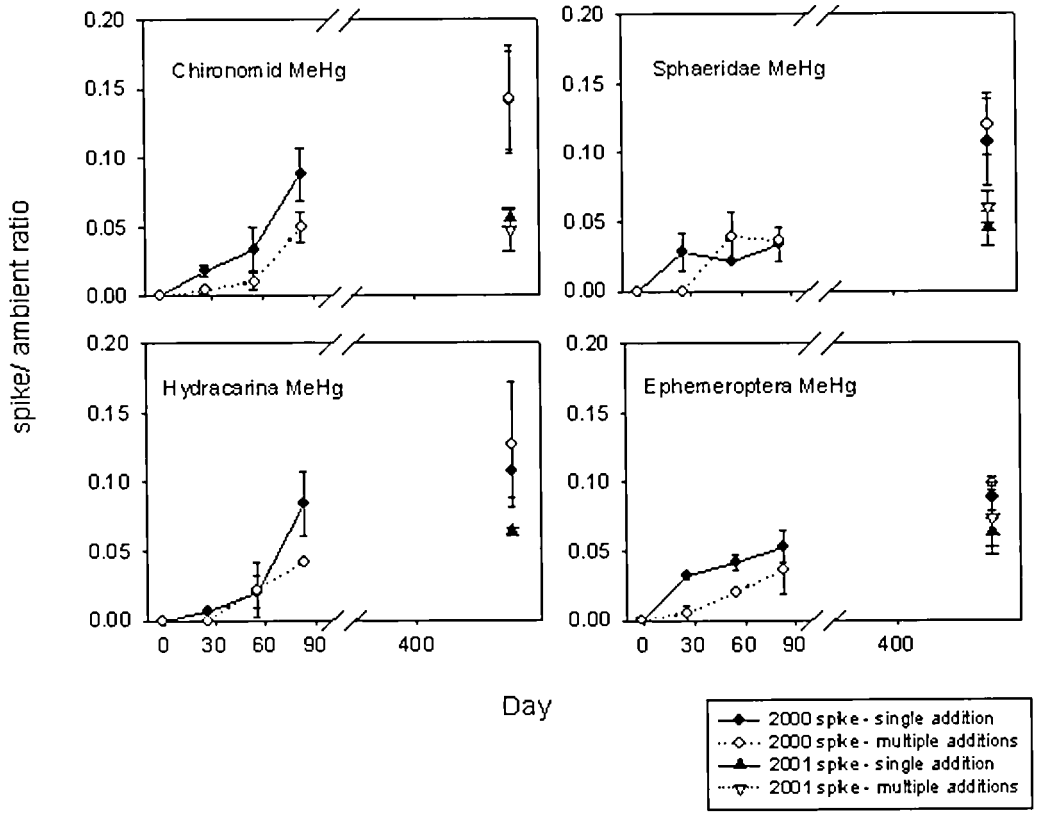

Figure 3. Response in MeHg in invertebrate biota in Lake 239 enclosures at the Experimental Lakes Area, northwestern Ontario, Canada, after additions of stable Hg isotopes in enclosures. Response is given as the ratio of spike (enriched stable isotopes) to ambient Hg (157).

Stable isotope tracer and loading experiments. The Mercury Experiment to Assess Atmospheric Loading in Canada and the US (METAALICUS) is a whole-ecosystem, Hg-loading study being conducted in a boreal, oligotrophic lake and its watershed at the Experimental Lakes Area in northwest Ontario, Canada (154, 155). The lake, upland, and wetland of the Lake 658 ecosystem are receiving Hg enriched with different stable Hg isotopes to determine the relative importance of each as sources of Hg to aquatic biota. Annual Hg additions of approximately 22 $\mu g\ m^{-2}\ yr^{-1}$ (5-fold greater than wet deposition) began in 2001 and are ongoing (through 2006).

Mercury applied to the upland and wetland catchments was strongly retained in the watershed and not observed in the lake in appreciable amounts after 3 y of treatment (136). Results to date indicate that the overall Hg load to the lake increased by nearly a factor of two due largely to the Hg isotope added directly to the lake surface ("lake spike"). This lake spike Hg began to be methylated within weeks after the addition of Hg and was quickly accumulated in biota (156). Throughout the study, the fraction of lake spike observed as MeHg in both sediment and water has been higher than for ambient Hg, although the differences have declined over the years since spikes began. This pattern demonstrates a decline in the availability of Hg deposition for methylation with time in lakes (155).

Increases in the amount of MeHg in water and the food chain may eventually match the percent increase in Hg load to the lake, but this had not occurred within 3 y. After the second year of additions, MeHg concentrations in zooplankton, benthic invertebrates, and small fish were more than 25% higher than would have occurred in the absence of experimentally added Hg (156). By the third year of the study, MeHg concentrations in zooplankton, benthic invertebrates, and small fish were approximately 30 to 40% higher than would have occurred in the absence of experimentally added Hg. METAALICUS clearly illustrates that biota in aquatic ecosystems respond to changes in Hg load at rates comparable to atmospheric deposition presently experienced in some regions and that different components of the ecosystem respond at different rates (96). The implication of this work is that the rate of response to changes in Hg deposition depends on ecosystem-specific characteristics.

Lake enclosure Hg addition studies at the Experimental Lakes Area. A number of mesocosm-scale studies associated with METAALICUS were also conducted to examine the timing and magnitude of responses to Hg loading. In the first mesocosm studies, Hg was added at a single loading rate of 30 $\mu g\ m^{-2}$ (157). In another lake enclosure study, the MESOSIM project, the dose response was examined over a range of Hg loadings (95), something that could not be done at a single loading rate and single site in the whole watershed study. Stable isotopes of Hg were added to 11 enclosures (10-m diameter) at 1 to 15x ambient wet deposition (7.1–106.5 $\mu g\ m^{-2}\ yr^{-1}$). In both mesocosm studies, newly added Hg was rapidly (i.e., weeks) detectable as MeHg in sediments and biota (Fig. 3). Importantly, isotopically labeled Hg and MeHg concentrations in sediments, water, and biota were all linearly related to loading, across concentrations representative of range of atmospheric Hg deposition (Fig. 4). These results suggest that changes in Hg loading affect MeHg concentrations in fish and other biota and that the response is linear and proportional at least in the early stages (95, 157).

Wetland Hg addition studies at the Experimental Lakes Area. A small-scale Hg loading experiment to an Experimental Lakes Area wetland also demonstrated the link between Hg deposition, methylation, and transport to surface waters (158). Over 3 mo, added isotopic Hg migrated below the water table and transported several meters horizontally to the lake margin. At one location, 6% of aqueous isotopic Hg was detected as MeHg 1 d after application. These results indicated that new inorganic Hg in atmospheric deposition could be readily methylated and transported towards the lake by shallow groundwater flow, confirming the importance of wetlands in the supply of Hg to surface waters.

Small-scale upland plot Hg addition experiment. A boreal upland subcatchment was also amended with a stable isotope of

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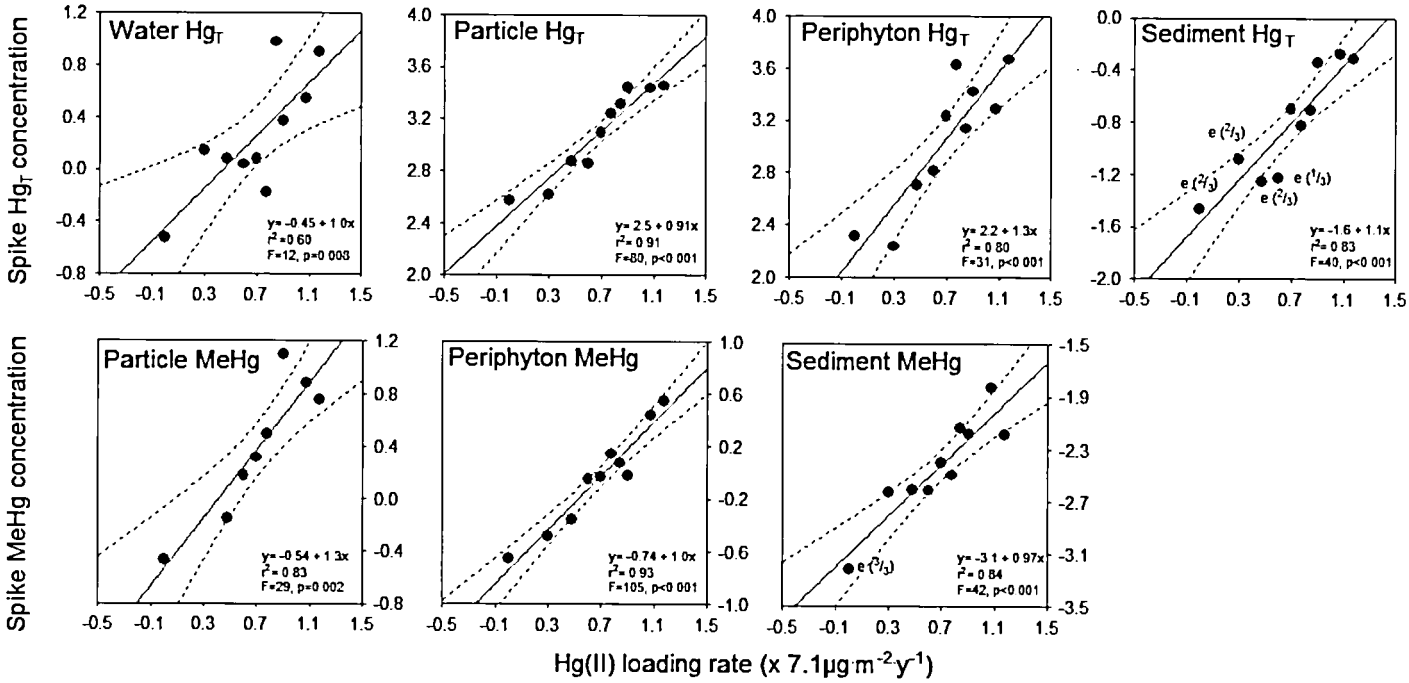

Figure 4. Dose-response relationships for the MESOSIM study, an enriched stable-isotope Hg loading study in lake enclosures at the Experimental Lakes Area, Ontario. Relationship between Hg(II) loading rate and spike HgT (top panels) or spike MeHg (bottom panels) concentration of water (ng L^{-1}), particles (ng g C^{-1}), periphyton (ng g C^{-1}), and sediments (ng g^{-1} dry weight). All variables are on a log10 scale. The regression lines are shown with 95% confidence intervals. The samples were collected approximately 2 mo after dosing. The symbol "e" indicates that one or more samples was estimated (in brackets is the number of samples with estimated concentrations/total number of samples). Reprinted with permission from (95), Copyright 2006 American Chemical Society.

Hg (135). The new Hg was found to be more reactive than old Hg for both volatilization to the atmosphere and methylation in soils. However, the yield of new Hg from the catchment was very low, less than 1% in the first year. The fraction of new Hg that was bound to vegetation was much greater than for old Hg, suggesting that the canopy delays the delivery of some atmospheric deposition to soils until after needle and leaf fall occurs.

Everglades studies. The Florida Everglades is a large, subtropical, predominantly freshwater wetland with strong spatial gradients in trophic condition, biogeochemistry, and net methylmercury production rates (24, 26, 159). Hg stable isotopes were added to small enclosures corresponding to 0.5, 1, and 2x the average annual wet deposition of Hg to the Everglades (160). At four sites, chosen to represent a wide range of water chemistry and productivity, $Me^{200}Hg$ concentrations in surface sediments, water, and fish responded linearly to Hg dose. However, the rate of the response varied significantly among the sites (Fig. 5). The results demonstrate both the response of ecosystems to changing Hg load and the variability in the magnitude of that response among ecosystems of different chemical characteristics.

The Gårdsjön Roof Study

To determine the relationship between reduced atmospheric deposition and runoff of Hg from a subcatchment, an experimentally covered catchment and a reference catchment in Sweden were monitored for total Hg and MeHg yield (16). The roof limited the atmospheric wet deposition of total Hg and MeHg (but not litterfall) to the catchment. There was no statistically significant change in the output of total Hg or MeHg as a result of the roof treatment after 10 y. In the catchments around the Lake Gårdsjön, stream Hg export (2–3 g km^{-2} yr^{-1}) and wet plus dry deposition (30–40 g km^{-2} yr^{-1}) are only small fractions in comparison to the total soil pool (10–20 kg km^{-2}). Clearly, factors other than deposition (e.g., decomposition of organic matter in soils) appear to be controlling runoff over the short term (i.e., years).

From these experiments we can conclude:

- All aquatic ecosystems that have been subjected to direct experimental additions of Hg to the surface waters begin to respond rapidly, with MeHg increases in the food web, including fish.
- Increased Hg loading directly to aquatic ecosystems at rates relevant to changes in atmospheric deposition results in increased uptake of labeled Hg in fish that is initially proportional to loading rates (i.e., experiments were not run long enough to observe steady-state responses).
- Terrestrial compartments are slow to respond to changes in Hg loading, with this response governed by the size and biogeochemistry of the soil pool and watershed hydrology. The implications of this response for the recovery of Hg-contaminated fisheries are dependent on the characteristics of the watershed. No studies have been conducted for a long enough period to understand soil Hg dynamics.

SYNTHESIS AND FINAL CONCLUSIONS

This synthesis clearly shows that fish MeHg concentrations respond to changes in Hg load. The magnitude and the timing of the response will vary depending on case-specific factors, including the timing and extent of loading, chemical form of the added Hg, and ecosystem characteristics (e.g., the efficiency of delivery of Hg load to zones of methylation, controls on net methylation, availability of MeHg to the food web, trophic transfer of MeHg), and the role of the terrestrial system in delivering atmospheric deposition to receiving waters. The main body of evidence comes from cases in which the Hg loading originates from point sources. For responses to changes in atmospheric loading, evidence is limited to a few cases that

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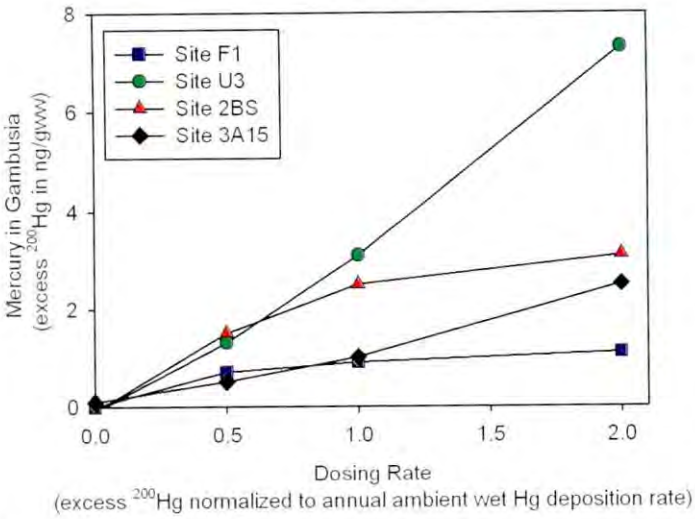

Figure 5. Response of MeHg in *Gambusia* 40 days after the addition of enriched stable Hg isotopes to the water column of 0.7 m diameter enclosures placed at four sites in the Everglades. Dosing rates are normalized to an average annual wet deposition rate of 22 μg m^{-2} y^{-1}. The sites cross a wide range of sulphur and dissolved organic matter concentrations (160).

currently limit the possibilities to draw firm conclusions. This is partly due to a lack of long-term, consistent monitoring data (Hg loading and fish Hg) and partly an expected consequence of the complex biogeochemistry of Hg, with multiple chemical, physical, and chemical factors besides Hg load that control the MeHg levels in fish. Based on the synthesis presented here, the following conclusions can be drawn:

- Numerous examples exist in freshwater and coastal marine systems where cessation of point source Hg loading have resulted in lowered Hg levels in biota, mainly from ecosystems contaminated by industrial releases.
- For freshwater ecosystems affected by atmospheric Hg deposition, a growing body of temporal and spatial data are generally consistent with a relationship between Hg deposition and MeHg in fish. Some evidence of the relationship between Hg loads and MeHg in fish exists for coastal regions. However, data for the open ocean areas are far from sufficient.
- In most ecosystems, large pools of Hg are present in soils and sediments. Much of this Hg may not be readily available for methylation. Additions of relatively small amounts of Hg that can reach sites of methylation in bioavailable forms can lead to increased MeHg production and bioaccumulation.
- All aquatic systems that have been subjected to experimental increases in direct Hg loading have begun to respond rapidly and significantly to increased Hg, as reflected in increased MeHg levels in the food web, including fish.
- Experimental studies suggest that the magnitude of fish responses to changes in direct Hg loading (within the range relevant to atmospheric Hg loadings) is initially proportional. However, experimental dose-response studies have not been conducted for long enough periods to assess the steady state conditions in response to changes in loading.
- Terrestrial ecosystems are slow to respond to increases and decreases in loading rates. Hg that has accumulated in soils from previous atmospheric Hg deposition is a source that may mask or delay the recovery of fish Hg levels when emissions of Hg decline. Mobilization of Hg from soils as well as MeHg production can be enhanced by land-use changes, such as agriculture, forestry, and reservoirs.

In summary, existing field and experimental evidence clearly indicates that Hg loading rates affect fish Hg concentrations. The magnitude and timing of the dose-to-response relationship are affected by the form of Hg loaded and by a wide range of environmental conditions. Current information suggests a very wide range of ecosystem response times to changes in Hg loading, ranging from months to decades or longer. Current knowledge is also consistent with a long-term proportional response to changes in atmospheric Hg deposition but not for cases of extreme point-source contamination. We use "consistent" rather than "clearly demonstrates" because existing data are not sufficient to draw firm conclusions regarding the timing and magnitude of the response to changing Hg loads, particularly for the oceans. The many factors affecting Hg cycling and bioaccumulation can mask the influence of atmospheric Hg deposition across a range of environmental settings, highlighting the need for carefully designed national and international monitoring programs (132), currently lacking, to detect trends for Hg levels in the environment and the response to changes in Hg emissions.

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Socioeconomic Consequences of Mercury Use and Pollution

In the past, human activities often resulted in mercury releases to the biosphere with little consideration of undesirable consequences for the health of humans and wildlife. This paper outlines the pathways through which humans and wildlife are exposed to mercury. Fish consumption is the major route of exposure to methylmercury. Humans can also receive toxic doses of mercury through inhalation of elevated concentrations of gaseous elemental mercury. We propose that any effective strategy for reducing mercury exposures requires an examination of the complete life cycle of mercury. This paper examines the life cycle of mercury from a global perspective and then identifies several approaches to measuring the benefits of reducing mercury exposure, policy options for reducing Hg emissions, possible exposure reduction mechanisms, and issues associated with mercury risk assessment and communication for different populations.

INTRODUCTION

Mercury (Hg) has historically been used in a wide variety of activities, both in compounds and as a liquid metal, resulting in widespread dispersion and the creation of some heavily contaminated sites. Geologic materials that contain high concentrations of Hg are usually found only in areas known as mercuriferous belts, such as along the west coast of the Americas. Even though most natural materials (including coal, oil, and minerals) contain very low levels of Hg, the use of large quantities of these materials releases significant amounts of Hg into the biosphere each year. Because Hg can vaporize at ambient temperatures, the Earth's atmosphere plays a major role in the dispersion of Hg. Human activities, including Hg mining, use, and waste disposal, combined with releases from the refining of other metals and the combustion of fossil fuels, have significantly increased Hg emissions to the atmosphere. Compared with preindustrial times, atmospheric deposition has increased uniformly in remote regions by a factor of about 3 (1). Deposition increases above threefold have been documented near emission sources; depositions depend on stack height, the quantity and chemistry of the emitted Hg, and local atmospheric chemistry (1). Bacteria in aquatic systems convert a small proportion of the deposited Hg to methylmercury (MeHg), which bioaccumulates in fish (inorganic Hg does not bioaccumulate). Aquatic systems vary in the efficiency with which atmospherically deposited Hg is bioaccumulated in fish. For example, the Hg concentration of fish in neighboring lakes can vary by as much as 10-fold, even when atmospheric Hg is similar (2). Nevertheless, in a given aquatic system, the production of MeHg is believed to be approximately proportional to atmospheric Hg deposition, so it is likely that historical increases in Hg emissions have increased MeHg concentrations in fish (3).

Hg use and releases are of concern because of two exposure pathways: consumption of MeHg-contaminated fish and inhalation of elevated concentrations of Hg vapor. The most common route of MeHg exposure for humans and wildlife is the consumption of fish from marine and freshwater sources. While low levels of Hg vapor are normally measured in the atmosphere, elevated concentrations can result from a variety of intentional Hg uses and accidents involving Hg, especially indoors where dilution is constrained.

In industrialized societies, although Hg was once a component in many products, it is now viewed as a material for which the risks of use generally outweigh the benefits. Hg-free substitutes are now viable for nearly all uses (energy-efficient lighting remains a notable exception), although their adoption varies greatly across jurisdictions and industrial sectors. The link between Hg emissions to the atmosphere and fish contamination has influenced policy development in industrialized countries since the mid-1990s, variously resulting in mandatory control of Hg emissions from waste incineration and other sources; restrictions on the labeling, sale, and disposal of Hg-containing products; and control of Hg emissions from coal-fired power plants as recently mandated by federal and state regulations in the United States (4, 5) and Canada (6).

Hg is also a commodity, with net flow from industrialized countries to developing countries, where its uses are less constrained. Annual global Hg consumption peaked at about 10 000 tonnes (t) in the 1960s; at that time large amounts were used in electrolytic and chemical processes, pesticides, paints, and batteries. Use gradually decreased to an estimated 3500 t in 2005 (Fig. 1a) (7–9). In 2000, however, global trade statistics reveal that at least 9000 t of metallic Hg were bought and sold across national borders, confirming that Hg is an actively traded commodity (Fig. 1b). However, the Hg market is opaque, with considerable uncertainties associated with many of the estimates presented here.

Significant and growing coal combustion, particularly in developing countries, and industrial-scale refining and smelting of metals also are responsible, in part, for the continued elevation of atmospheric Hg levels (10). Although global Hg pollution of the biosphere is likely a major contributor to the widespread MeHg contamination of fish, effective international agreements that address Hg pollution have yet to be developed.

This paper describes how certain human activities mobilize Hg from geologic stores into the biosphere and outlines the resulting social and economic consequences. We present possible policies to reduce exposure to Hg and MeHg, and explore issues associated with risk assessment and communication for different populations. We utilize substance flow analysis, which reveals not only the source and fate of a material, but also potential control points. We discuss policy options for addressing control points in the flows of Hg that could ultimately reduce fish contamination while simultaneously reducing the potential of harmful exposure to Hg vapor.

Concern about MeHg in Fish

Exposure to MeHg through consumption of contaminated fish by humans has the potential to interfere with normal neurological development and function and may increase the risk of myocardial infarction (heart attack) (11). Negative

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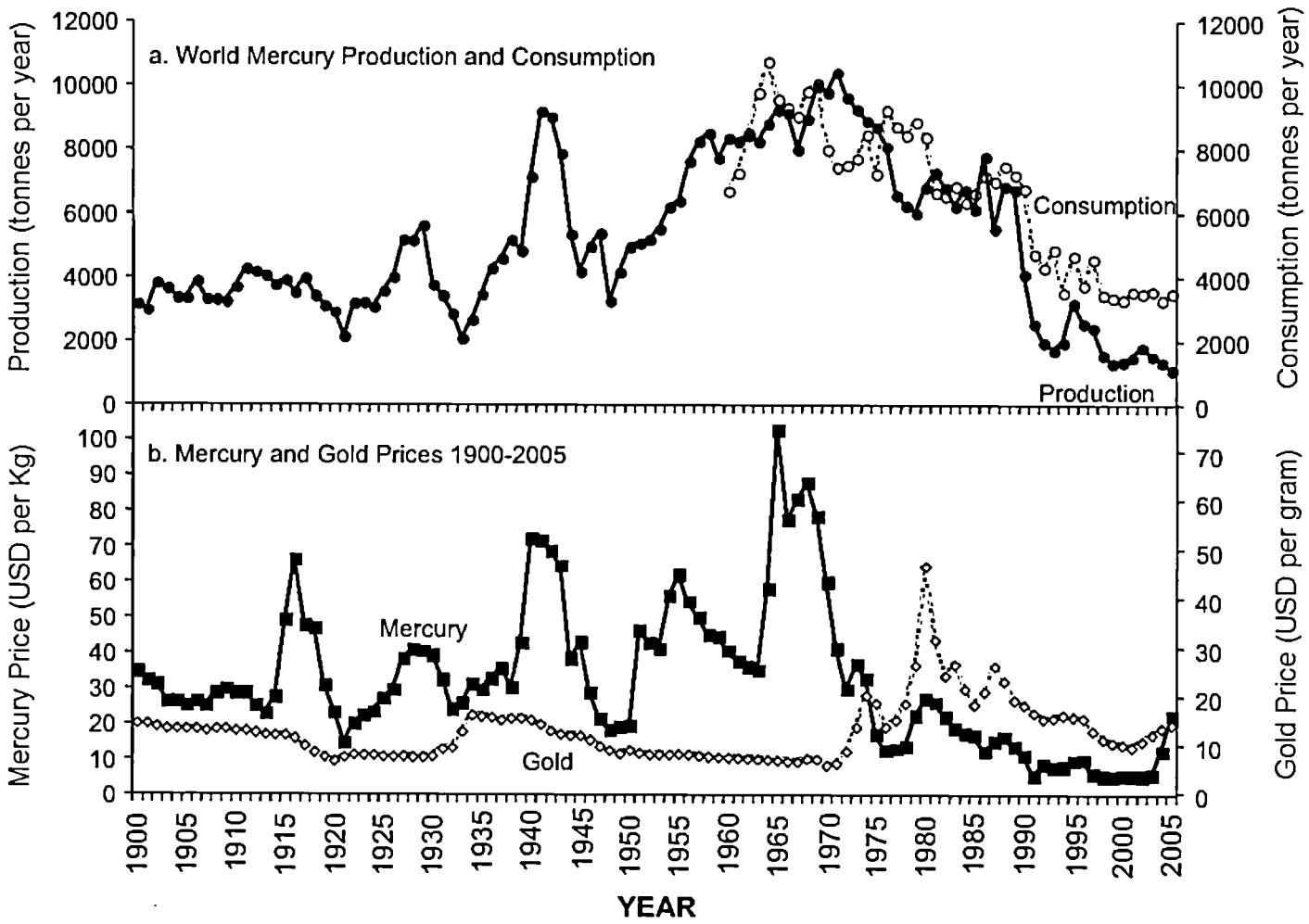

Figure 1. (a) Historical mercury production and consumption. Consumption has exceeded production since about 1990, with demand met by large supplies from government stockpiles and closed chlor-alkali plants, plus contributions from recycling (7). (b) Mercury and gold prices in the United States from 1900 to 2005, adjusted to constant 2005 US dollars (USD). The correlation between gold and mercury prices begins after 1971 with the breakdown of a fixed exchange rate system (7–9).

neurological and reproductive effects of MeHg on fish-eating wildlife—birds (e.g., loons), mammals (e.g., river otter and mink), and fish that eat other fish—are also a concern (12).

Concern about Inhalation of Hg Vapor

Absorption of liquid Hg through the skin or digestive system is quite limited, but the body retains 80% of inhaled Hg vapor (13). An indoor spill of metallic Hg, even in quantities as small as a gram, can give rise to Hg concentrations in ambient air that approach WHO recommended limits for occupational exposure. Heating metallic Hg and Hg compounds, such as cinnabar and scrap dental amalgam, has generated fatal inhalation doses (14). Although heating Hg is a rare activity in most societies, an estimated 10 to 15 million people earn their living in small-scale gold-mining operations by amalgamating gold with Hg, and then concentrating the gold by heating the amalgam, thereby releasing the Hg in vapor form. This process directly exposes the miners and can expose their families and neighbors (estimated to total about 50 million people), to elevated Hg levels (15). In 2005, small-scale or artisanal gold mining was estimated to contribute more than 10% of annual global anthropogenic Hg loading to the atmosphere (about 300 out of a total 2400 t, Fig. 2).

Concern about Hg vapor extends beyond small-scale mining to include Hg in products. One such application, dental amalgam, is the most common way that people are exposed to elevated Hg vapor, although the levels are not as high as those frequently encountered in artisanal gold mining. Even though Hg levels in autopsy brain samples correlate positively with the number of amalgam fillings, vapor concentrations from amalgam are usually well below those associated with even subtle neurobehavioral effects (13). Otherwise, Hg is usually encapsulated in consumer and commercial products, such as fluorescent lamps and thermometers. However, these devices can break during use or disposal, releasing Hg into indoor air and the environment. Historical uses of Hg left a legacy of many small bottles of liquid Hg. In the United States, Hg spills have caused expensive and disruptive clean-up efforts (16). Although adverse health effects have rarely been documented from Hg spills, we do not consider this as evidence of a low potential for harm. For example, three children were hospitalized in Michigan (United States) as a result of Hg poisoning, including one child who was no longer able to walk. Investigation revealed that exposure occurred after a small vial of Hg was spilled in the children's bedroom approximately two to three months prior to detection of the gross symptoms (17).

GLOBAL COMMERCIAL AND ENVIRONMENTAL PATHWAYS

Although somewhat simplistic, conceptually, it is useful to divide the Earth's Hg between two repositories: Hg in the biosphere and Hg stored in geological formations. The biosphere encompasses

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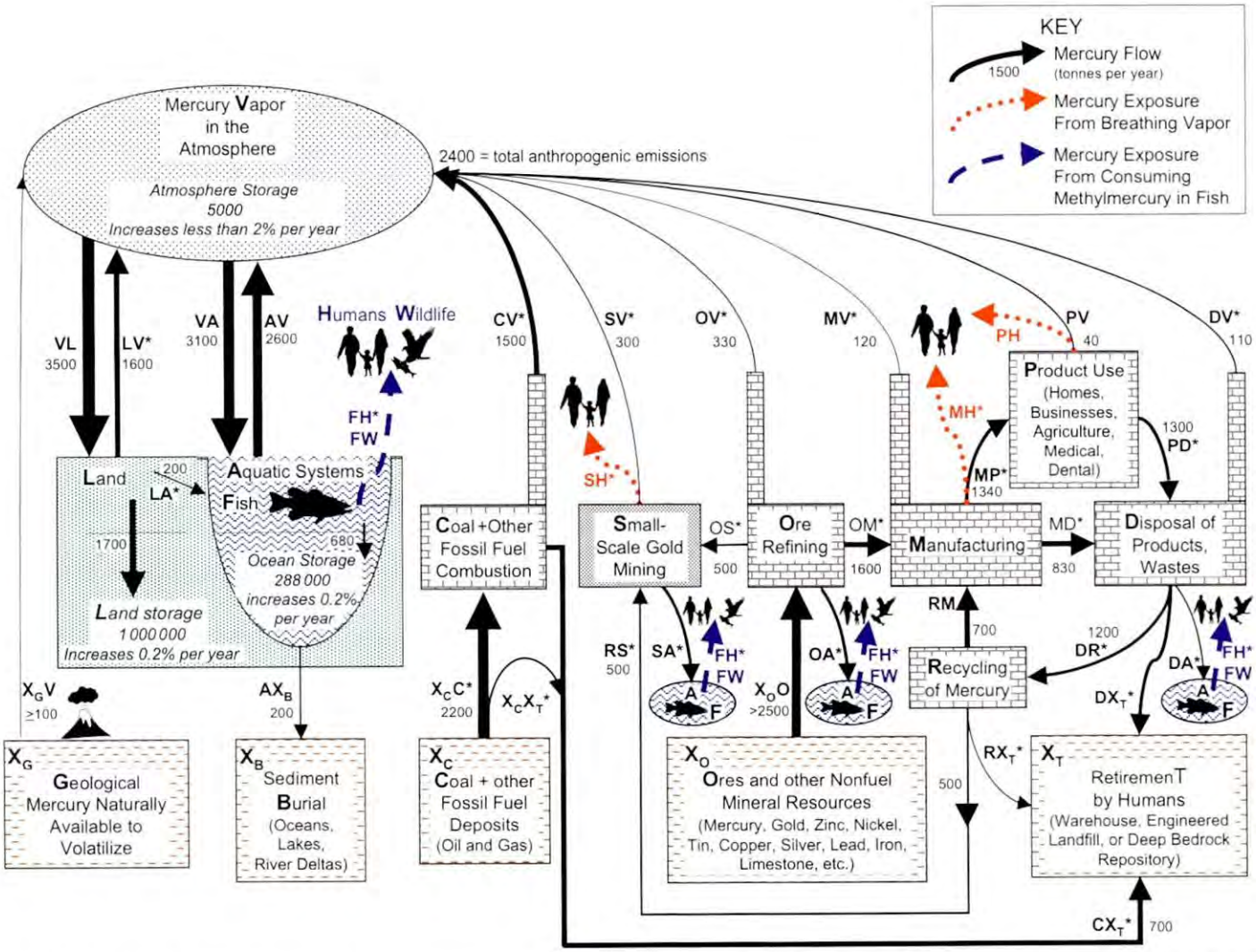

Figure 2. Important global pathways of mercury in commerce and the environment. Width of pathway line is proportional to the flow per year. See Table 1 for explanation of abbreviations and the text for discussion of the compartments and Table 2 for discussion of the pathways.

the portion of Earth and its atmosphere that supports life; geologic formations include deep sediment deposits, buried minerals, and fossil fuels. The most significant human impact on Hg is its mobilization from long-term geologic storage into the biosphere, where Hg cycles between air, soil, and water.

Assuming only two repositories is simplistic because mobilized Hg can enter subcompartments that appear to be stable, such as Hg encapsulated in a device such as a thermometer, Hg that has settled into deep ocean waters, and Hg sequestered in deep soils, such as the vast peatlands of Canada and Siberia (18, 19) or the ancient soil of the Amazon Basin (20). However, use of only two repositories is useful from a policy perspective because human activity can mobilize the Hg from apparently stable compartments of the biosphere. Thermometers inevitably break, and the Hg in soils can be mobilized through fire (19), forestry practices (21, 22), agriculture (20), climate change (18, 19, 23), and the creation of reservoirs (24). Additional research is needed to understand the fate of Hg that enters both coastal and deep-water marine systems (1).

The movement of Hg into and out of the biosphere is shown in Figure 2, based on the global model of Mason and Sheu (25), a model that is representative of the current scientific understanding of global Hg cycling [see review of four models by Seigneur et al. (26)]. Although there is a considerable range of estimates in the models for specific components of the global Hg cycle and there is uncertainty regarding the scale of natural emissions and reemission of deposited Hg (1), the four models converge on a relatively tight range for total global emissions (natural and anthropogenic); that is, from 6060 to 6600 t y^{-1}, and for current anthropogenic emissions alone, from 2000 to 2400 t y^{-1}. Reemission of previously deposited anthropogenic emissions roughly equals current anthropogenic emissions (1). In general, the global models can be considered uncertain, but useful, frameworks.

Although current anthropogenic emissions in Figure 2 are constrained to 2400 t y^{-1} to conform to the Mason and Sheu model, it would be reasonable to assign an uncertainty of ±30% to total anthropogenic emissions (1), with some sectors embodying less uncertainty than average and some sectors more. It has been suggested that Hg emitted from global coal combustion has a ±25% uncertainty; nonferrous metal production ±30%; waste disposal and incineration has uncertainty up to 500%; and Hg use in artisanal and small-scale gold production is too poorly understood to allow a quantitative uncertainty factor to be assigned (1). A protocol, or "toolkit," has been developed to assist countries that wish to identify and quantify sources of Hg emissions (27). The toolkit, including extensive text and a spreadsheet template, explains how to develop detailed quantification of Hg pathways that are only broadly defined in Figure 2.

Given the uncertainties in the current understanding of global Hg pathways, Figure 2 can reasonably be used as background information for discussions of policy options that

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Table 1. Important global compartments of mercury in commerce and the environment, as diagrammed in Figure 2.

Code	Mnemonic	Definition
A	Aquatic system	Hg in wetlands, lakes, rivers, and oceans. Hg introduced to aquatic systems may become MeHg, which may be bioaccumulated by fish.
C	Coal and other fossil fuel combustion	Hg mobilized by the processing and combustion of the fossil fuels coal, oil, and natural gas (X_C).
D	Disposal	Hg in discarded products or process wastes from chlor-alkali or VCM plants.
F	Fish	Hg in fish, virtually all of which is in the form of MeHg, which is produced by naturally occurring bacteria in aquatic systems.
H	Humans	Hg absorbed by humans following exposure, generally through fish consumption or inhalation of vapor.
L	Land	Hg in soil, mostly derived from atmospheric deposition of vapor, but can be elevated from mine waste, Hg waste disposal, or geologically rare mineral deposits containing Hg.
M	Manufacturing	Hg used in the manufacture of Hg-containing products, or in processes that use Hg to make Hg-free products (e.g., chlor-alkali and vinyl chloride monomer processes).
O	Ore refining	Hg mobilized by the processing and refining of nonfuel mineral resources X_O.
P	Products	Hg contained in products, including thermometers, switches, fluorescent lamps, batteries, fungicides, preservatives, seed-coatings, pharmaceuticals, etc.
R	Recycling	Hg that is extracted from discarded products or wastes, purified, and put into commerce or retired.
S	Small-scale gold mining	Hg utilized by independent, artisanal, miners to concentrate geological gold through amalgamation.
V	Vapor	Hg vapor in indoor and outdoor air.
W	Wildlife	Hg absorbed by fish-eating wildlife, such as seal, whale, otter, mink, osprey, eagle, kingfisher, and loon.
X	Out of the biosphere	Hg in the "X" compartments are not part of the Hg cycling in the biosphere and therefore do not harm humans or wildlife. "X" Hg may be mobilized at some point in the future, but for practical purposes is permanently stored unless humans intervene.
X_B	Buried	Hg, formerly in the biosphere, that has been buried in the sediments of oceans, lakes, and river deltas.
X_C	Coal and other fossil fuel deposits	Hg in buried fossil fuel deposits such as coal, oil, and gas, that may be extracted and burned.
X_G	Geological	Hg in geological materials that release Hg vapor to the atmosphere through natural processes.
X_O	Ores	Hg in nonfuel geological resources that are subject to mining and refining, including minerals containing Hg, gold, zinc, nickel, tin, copper, silver, lead, and iron. All geological materials contain some Hg, even limestone that is heated to make lime.
X_T	Retirement	Hg permanently stored, or "retired" by humans in warehouses, engineered landfills, or deep bedrock repositories.

might be adopted to reduce Hg emissions and, ultimately, exposure from fish consumption and small-scale gold mining.

Each letter in Figure 2 codes for a compartment in which Hg is stored or from which it is released (see Tables 1 and 2). A two-letter code indicates a path from one compartment, indicated by the first letter, to another compartment represented by the second letter. For instance, VL denotes the pathway of atmospheric Hg to the Earth's surface whereas LV is the path of Hg volatilization from land to the atmosphere, with the annual flux in tonnes next to each arrow. Note that Hg is relatively mobile in the biosphere; in this model, over 80% of the Hg deposited to the oceans is reemitted to the atmosphere. In contrast, only about half of the Hg deposited to land is reemitted within a few years because Hg can associate strongly with soil.

Pathways with an asterisk (*) are amenable to manipulation to minimize Hg release or exposure at the time this manuscript was written. Therefore, we refer to these pathways as control points. For example, FH* denotes that the exposure of humans (H) to MeHg from contaminated fish (F) may be reduced through selection of fish to consume; it should be noted, however, that in some societies or regions there may be little choice of fish species to consume. The lack of an asterisk indicates that it is unlikely that present social or economic policies can affect that pathway. MP* indicates that the creation of products by manufacturers does not have to include Hg. PV indicates that once Hg is a product component, it is inevitable that some of that Hg will be released to the atmosphere as a result of breakage, no matter what policies, such as recycling, are adopted.

FOSSIL FUEL COMBUSTION: COMPARTMENT C

Fossil fuels (coal, oil, and natural gas) contain a wide range of Hg concentrations in their natural state. Some natural gas supplies are high in Hg vapor, but the Hg is routinely removed during refinement before distribution to avoid degradation of aluminum heat exchange surfaces via amalgamation in gas-processing plants (28). Few analyses have been performed on the Hg content of petroleum or oil sands, or on the fate of Hg throughout the oil exploitation and refinery process. While there is a lack of general data on Hg concentrations in oil and oil sands in their natural state, once refined, the Hg emissions from the combustion of these fuels appear to be much lower than from coal (28).

Coal combustion is responsible for about 60% of anthropogenic Hg emissions (pathway CV*, 1500 t y^{-1}; Fig. 2; 29). A

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continuous decrease in Hg emissions in Europe and North America since 1980 has resulted from the installation of emission-control equipment, particularly electrostatic precipitators (ESPs), fabric filters, and flue gas desulfurization (FGD) technologies. These existing technologies incidentally capture Hg largely to the extent that coal combustion produces divalent Hg. Metallic Hg vapor (Hg^0) is usually poorly controlled by equipment designed to capture particulates or sulfur. The emitted form of Hg (and its removal) is highly dependent on the coal type and the installed emission control equipment (30).

There are two major types of FGD systems: wet and dry. In general, removal efficiency for Hg in FGD systems ranges from 30% to 85% depending on the proportion of divalent Hg, which is related to the halogen content of the coal. The greatest removal efficiencies with existing technology can be expected when a wet FGD system is installed downstream of a fabric filter (30).

Most utility power plants in developed countries are equipped with either ESPs or fabric filters for particulate control. Removal of Hg by these devices is highly dependent on halogen content and unburned carbon in the flue gases. Generally speaking, fabric filters remove approximately twice as much Hg as ESPs under similar flue gas conditions (4, 30). As part of efforts to control Hg emissions from coal-fired power generation, significant new information is emerging on flue gas chemistry (Hg, chlorine, sulfur, and the effect of nitrous oxide, or NOx, control) that will help to improve Hg control. A wide variety of technologies are being developed (4, 31, 32) and applied (33) to reduce Hg emissions. As with other emission control technologies (e.g., SO_2, NOx, etc.), it is possible that innovations will yield commercially available options that are significantly less costly than initially estimated (34).

ORE REFINING: COMPARTMENT O

Ores of metals, especially nonferrous metals such as gold, silver, copper, lead, zinc, and nickel, often contain Hg because the geological processes that concentrate these metals typically also concentrate Hg. In particular, sulfide ores often contain significant concentrations of Hg because of a high chemical affinity between Hg and sulfur. Cinnabar—mercuric sulfide—has been the main source of Hg as a commodity for thousands of years. Despite recent mine closures in Spain and Algeria, cinnabar mining of 1300–1400 t annually remains the largest source of elemental Hg (Fig. 2; 9) and is increasing in China to meet internal demand. The processing of cinnabar is associated with elevated atmospheric Hg emissions. As global Hg demand declines, however, less expensive sources of Hg—recycling, Hg from closed chlor-alkali plants, and as a by-product from large-scale gold, zinc, and copper mining—may be expected to supply a greater proportion of Hg demand. The availability of Hg as a by-product may increase significantly if new regulations restrict Hg emissions from ore refining.

Hg IN MANUFACTURING AND PRODUCTS: COMPARTMENTS M AND P

Sources of Hg to industries include new Hg from current mining (pathway OM) as well as recycled Hg (pathway RM). Hg has been intentionally used in many products and processes, although consumption decreased by ~50% from 1990 to 1998 (Fig. 1; 35). Annual use of Hg in products in 2004 (Fig. 3) comprised mostly dental amalgams (270 t), electrical switches and relays (150 t), measuring and control equipment (160 t), energy-efficient lighting (110 t), and disposable batteries (estimated at 600 t). Smaller quantities also continue to be used in an array of other products. For example, some Hg is

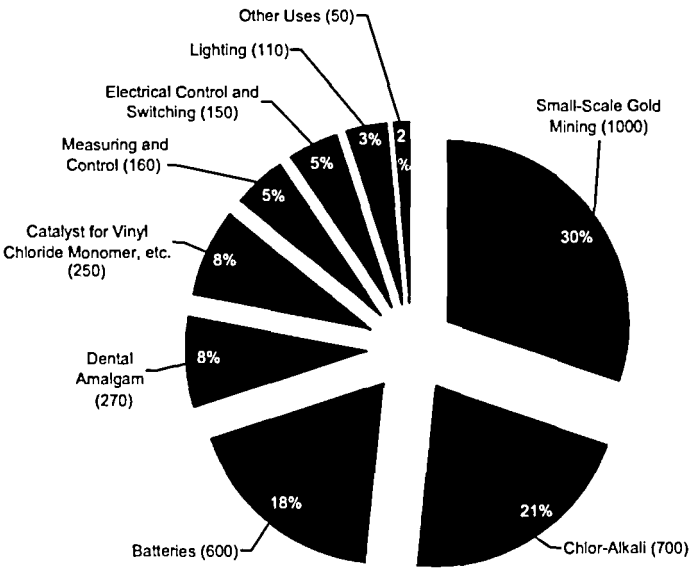

Figure 3. Estimates of global mercury consumption for 2004 (9, 36).

used in cosmetics; in the United States, up to 65 ppm is allowed. Hg continues to be used in chlor-alkali (chlorine and caustic soda) production (possibly 700 t in 2004, but is declining every year) and as a catalyst for the production of vinyl chloride monomer. The latter remains a significant use in China, India, and Russia [estimated at 250 t y^{-1} in this paper, although recent information from China puts this number as high as 600 t (36)].

Before 1990, large quantities of Hg were used in ways that dispersed Hg widely in the biosphere; these uses included fungicides used in seed coatings, the paper industry, and latex paints (37). Today, most Hg added to products is not released during normal use, with the exception of Hg-containing skin-lightening soaps and creams; vaccines containing thimerosal (a Hg-containing preservative that is injected into the body during vaccination); dental amalgams; and volatilization from Hg-catalyzed polyurethane products. The largest remaining dissipative use of Hg is in dental amalgams, which can result in direct human exposure during inhalation, occupational exposure in the dental office, releases to wastewater both from dental offices and homes, emissions during incineration of dental wastes, and flue-gas emissions during cremation (pathways PH, PV, PD*, DA*, and DV*).

Electrical switches and relays, measuring and control equipment, energy-efficient lighting, and batteries do not release Hg until disposal, except as a result of misuse or accidents (pathways PH and PV). It is possible to manufacture these products in such a way as to limit air or water emissions of Hg (pathways MV* and MH*). Therefore, the extent of Hg emissions from Hg-added products is likely a function of the method of disposal (pathways MD* and PD*). Chlor-alkali factories reportedly have greatly decreased their Hg releases in many nations (38, 39). However, considerable uncertainty remains regarding the environmental fate of hundreds of tons of Hg unaccounted for annually by the global chlor-alkali industry, much of it released within developing countries.

Polyvinyl chloride (PVC) is typically manufactured from petrochemical feedstocks. There is, however, a Hg-catalyzed method to make a PVC feedstock, vinyl chloride monomer (VCM), from coal; this method typically results in large Hg consumption. Such PVC production is a substantial contributor to Hg releases (pathways MV*, MH*, but mostly MD*), conservatively estimated at 150 t y^{-1} (9, 40), but possibly twice that amount (36). However, there remains considerable

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Table 2. Important global pathways of mercury in commerce and the environment (diagrammed in Figure 2). An asterisk (*) on the pathway code indicates that the path is amenable to manipulation by society's policies. The estimates of annual anthropogenic flow are constrained by setting total anthropogenic emissions at 2400 t y^{-1} (25).

Code	From	To	Comments	Annual flow (t)	Information source
AF	Aquatic system	Fish	Only a portion of Hg entering an aquatic system bioaccumulates in fish, partly because only a portion is converted to MeHg.		
AV	Aquatic systems	Vapor	The oceans are estimated to re-emit over 80% of atmospheric deposition; there is little binding capacity in ocean water, and photochemistry produces Hg^0, which has low solubility in water.	2600	25
AX_B	Aquatic system	Sediment burial	Some of the Hg entering an aquatic system associates with particles that settle to the bottom and is buried permanently by new sediment. Mason and Sheu (25) conclude that Hg is building up in deep ocean water and only a small proportion is buried.	200	25
CV*	Fossil fuel combustion	Vapor	Emitted Hg vapor may be elemental or divalent, which greatly affects distance traveled before deposition to earth.	1500	29
CX_T*	Fossil fuel combustion	Retirement	Hg vaporized during combustion may associate with ash or can be caught separately. Coal ash is often used in construction, which may not retire the associated Hg as permanently as putting it in a landfill.	700	Estimate
DA*	Disposal	Aquatic system	Some Hg is discharged directly into surface water, or indirectly through treatment plants. In treatment plants, most Hg associates with solids, which if not discharged are incinerated or land-applied.		
DR*	Disposal	Recycling	This category includes Hg from closed chlor-alkali plants, which is saleable as is. The Hg in product waste can be purified at relatively high cost per kilogram of Hg (100 USD to 1000 USD kg^{-1}), which is then sold for less than 20 USD kg^{-1}. Retirement of recycled Hg may be a better economic choice for societies.	1200	Estimate
DV*	Disposal	Vapor	Disposal to the solid waste stream may include incineration, which vaporizes all Hg, of which some may be caught by pollution control equipment and place in a landfill.	110	29
DX_T*	Disposal	Retirement	Manufacturing waste Hg may be retired in landfills as sludge. Hg-products might be put in landfills after breakage and spilling of Hg.		
FH*	Fish	Humans	Humans absorb most of the methylmercury (MeHg) consumed in a fish meal. The MeHg is in the protein, not the fat, of fish.	25 (as MeHg)	Estimate, assuming 0.2 ppm
FW	Fish	Wildlife	Fish-eating wildlife are particularly vulnerable to elevated Hg in fish.		
LA*	Land	Aquatic system	Of the Hg deposited from the atmosphere to land, a variable proportion (approximately 5% to 20%) is delivered to lakes and rivers draining the land. Human alteration of the landscape (e.g., agriculture and urban development) can affect the transport to aquatic systems.	200	25
LV*	Land	Vapor	The land reemits about half of the Hg deposited from the atmosphere. Human alteration of the landscape (e.g., climate change, fire, agriculture) can change the rate of reemission to the atmosphere.	1600	25
MD*	Manufacturing	Disposal	Any manufacturing process that employs Hg will have Hg waste.	830	Estimate
MH*	Manufacturing	Human vapor exposure	Any manufacturing process that employs Hg will produce Hg vapor that potentially exposes the workers.		
MP*	Manufacturing	Products	Products containing Hg are still manufactured even though cost-effective Hg-free substitutes exist for almost all uses, probably because of economic and technological inertia.	1070	Estimate
MV*	Manufacturing	Vapor	Manufacturing both makes Hg-containing products and uses Hg in processes that emit Hg (chlor-alkali and VCM).	120	Estimate
OA*	Ore refining	Aquatic systems	The processing of Hg-containing ores sometimes produce wastes or tailings that enrich aquatic systems with Hg.		
OM*	Ore refining	Manufacturing	Almost all Hg mines in the world are now closed, because the world's Hg demand is now met by by-product Hg from non-Hg ores, recycling, and the closure of chlor-alkali plants.	1600	Estimate
OS*	Ore refining	Small-scale gold mining	By-product Hg is one source for gold mining.	500	Estimate
OV*	Ore refining	Vapor	Heating ores will vaporize any Hg present, either emitting it to the atmosphere or incidentally catching it with air pollution control devices, unless special Hg control efforts are made.	330	29
PD*	Products	Disposal	Hg-containing products will eventually reach the end of their useful life and will either break or be disposed of.	1020	Estimate
PH	Products	Human vapor exposure	When Hg-containing products break and spill, they create the risk of human exposure and time-consuming clean ups.		
PV	Products	Vapor	Hg spills from broken products contribute to the atmospheric burden of Hg.	40	Estimate
RM	Recycling	Manufacturing	Recycled Hg can be used by many manufacturers.	700	Estimate

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Table 2. Continued.

Code	From	To	Comments	Annual flow (t)	Information source
RS*	Recycling	Small-scale gold mining	Hg used in mining does not have to be of a high purity, so recycled Hg, especially from chlor-alkali plants, can be used.	500	Estimate
RX_T	Recycling	Retirement	Hg need not be purified to be retired, although there may be some advantages in handling and containment. 4400 t retired in 2006 (62)		
SA*	Small-scale gold mining	Aquatic systems	Hg may be used directly in flowing water to concentrate gold, contaminating water and its sediments.	700	Estimate
SH*	Small-scale gold mining	Human vapor exposure	The gold–Hg amalgam is heated to concentrate the gold, exposing miners and their families to Hg vapor.		
SV*	Small-scale gold mining	Vapor	Hg vaporized during heating of Hg–gold amalgam adds significantly to the global atmospheric burden of Hg.	300	45
VA*	Vapor	Aquatic Systems	All atmospheric Hg eventually deposits to earth, with oceans receiving almost half.	3100	25
VL*	Vapor	Land	Hg deposition rates to the continents has increased by about a factor of 3 over preindustrial rates.	3500	25
X_CC*	Fossil fuel deposits	Combustion	Although the Hg concentration in fossil fuel is usually low, much is burned, and all Hg vaporizes during burning, but some binds to particles and is captured. Pretreatment of fuel has the potential to remove Hg prior to combustion.	3000	Estimate
X_CX_T*	Fossil fuel deposits	Retirement	Some of the Hg in fossil fuel is separated from fuel prior to combustion through coal cleaning or natural gas treatment. Oil refineries are poorly understood where the fate of Hg is concerned.	700	Estimate
X_GV	Geology	Vapor	Hg is naturally released from geological deposits, naturally enriched soils, and volcanoes. Lindberg et al. (1) point out that this path is poorly known and may be greater than 1500 t y^{-1}.	100	25
X_OO	Ore resources	Ore refining	Hg is in relatively high concentrations in sulfide ores of gold, silver, copper, lead, and zinc, and in lower concentrations in nonsulfide ores. Heat and other processes release the Hg, which enters the biosphere unless efforts are made to capture it.	>2500	Estimate

uncertainty as to what part of those releases go into the atmosphere in contrast to other waste streams.

The substantial reduction of global Hg use (Fig. 1a) is due to two factors: *i)* substitution of non-Hg products (e.g., paints, batteries, thermometers, and pesticides) and production processes (mainly chlor-alkali) and *ii)* more efficient use of Hg in production, except, typically, in cases where production has been shifted to developing countries.

SMALL-SCALE GOLD MINING: COMPARTMENT S

A combination of high gold prices and persistent poverty contributes to a proliferation of small-scale gold mining that uses Hg amalgamation to concentrate gold (41, 42). While the scale of gold production by artisanal miners is not well defined, it may constitute 20% to 30% of global gold production, ranging from 500 to 800 t of gold each year (43, 44) and occurs in over 50 developing countries (15). Because gold is easy to sell and transport, and its value remains relatively stable in countries with unstable currencies, it constitutes one of the more important extraction economies (41).

Hg is widely used in small-scale gold mining, despite laws prohibiting its use. Hg is combined with gold-containing silt or crushed ore to form a gold–Hg amalgam, simplifying recovery of the gold. Generally the amalgam is then heated with a blowtorch or over an open fire, vaporizing the Hg and leaving the gold behind (pathways SV*, SH*). Miners are estimated to lose, on average, 1 to 2 g of Hg per gram of gold produced; thus, this process annually releases approximately 1000 t of Hg to the biosphere, of which an estimated 300 t is emitted directly to the atmosphere (Figs. 2 and 3; 45). Virtually all of the Hg consumed by this activity is released somehow to the environment. The leading consumer of Hg through this activity is thought to be China (200 to 250 t y^{-1}), followed by Indonesia (100 to 150 t y^{-1}), while Brazil, Colombia, Peru, the Philippines, Venezuela, and Zimbabwe each consume an estimated 10 to 30 t y^{-1} (46–48). The unregulated trading of Hg in developing countries makes it readily available at the mine sites (pathways OS*, RS*).

Hg use in small-scale mining has left a legacy of thousands of polluted sites with impacts extending far beyond localized ecological degradation, often presenting long-term health risks to persons living in mining regions (49). Inhalation of Hg vapor is the primary exposure pathway for miners, gold shop workers, and people living near areas where the gold–mercury amalgam is produced and processed. Miners and community members often breathe air with Hg concentrations above 50 $\mu g\ m^{-3}$—50 times the World Health Organization maximum public exposure guideline. Consequently, many miners and others—particularly amalgam burners, who are often women—demonstrate tremors and other symptoms of Hg poisoning (50). Two mining practices may be increasing local Hg exposure because of inadequate information: Improper use of retorts to recover Hg may increase exposure to vapor (51) and cyanide leaching after Hg amalgamation may increase Hg bioavailability and fish contamination (14).

UNIDO efforts to reduce Hg releases have yielded new retorting techniques using readily available pipes and kitchen bowls, which allow miners to contain Hg emissions and recycle as much as 95% of the Hg from the vaporization process (52, 53). Such approaches are garnering increased attention, in particular because Hg prices increased fourfold and more between 2002 and 2005 (Fig. 1b). While the international price for 1 kg of Hg increased from less than 5 USD to more than 20 USD (all prices in constant 2005 USD), the price for 1 kg

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reached more than 100 USD in the small-scale gold mining sites in Mozambique, Zimbabwe, and Indonesia.

Hg-free alternatives to dissolve gold (cyanide) or to concentrate gold (e.g., gravity separation, magnetic sluices, and coal-oil gold agglomeration methods) are relatively costly and currently inaccessible to most miners in developing countries (54). Other promising Hg-free techniques to extract gold from concentrates include electro-oxidation and alternative methods of leaching, such as the iGoli Process (15, 55). Widespread adoption of Hg-free gold-mining methods would require substantial time and investment in both technology and social-development (55).

RECYCLING AND RETIREMENT: COMPARTMENTS R, D, AND X_T

All products eventually enter the waste stream. Hg contained in waste products may be recycled (DR*), incinerated (DV*), left in place, disposed on land, disposed into wastewater (DA*), released through breakage during use or disposal (PV), or "retired" through placement in a warehouse, engineered landfill, or deep bedrock repository (DX_T). Every time Hg moves from one compartment to another, there is some loss to the atmosphere and potential for human exposure via inhalation and eventually via deposition, aquatic methylation, and fish consumption (56–58). In some societies, regulations may protect workers from exposure, incinerators may have Hg-control devices, and diversion of Hg-containing products from the waste stream may be mandated. But in much of the world, Hg exposure and disposal are unconstrained (43). The incentive for firms in industrialized countries to recycle is regulatory in nature rather than economic because the market value of recovered Hg is usually much lower than the cost of recycling. On the other hand, recycled Hg may be considered a "cheap" Hg source because it is a product of the waste disposal process that has already been paid for.

Recycling

From a sustainability perspective, recycling of materials is generally preferred over landfill disposal because recycling obviates the need for (and costs associated with) landfills as well as the environmental costs associated with extraction of virgin material. However, unless it is integrated into a larger strategy of stable or decreasing Hg supply and demand, recycling Hg may not decrease global Hg pollution levels if the recycled Hg is merely returned to the marketplace. In the latter case, Hg recycling could have the effect of increasing the Hg supply and decreasing the price.

Under current regulations in most developed countries, it is cheaper to recycle waste containing a significant percentage of Hg and to sell the recovered Hg on the open market, than it is to dispose of Hg-bearing waste at a hazardous waste landfill (59). The generation of Hg from recycling and the recovery of Hg from decommissioned chlor-alkali plants have become increasingly significant contributors (10%–20% in recent years) to global supplies because recycling has increased and the production of mined Hg has declined. However, in the interest of eliminating surplus Hg supplies from the global market, the European Union (EU) has a draft regulation for a Hg export ban, which is presently under discussion in accordance with Actions 5 and 9 of the EU Hg strategy. This ban targets all Hg from decommissioned EU chlor-alkali plants for retirement as of 2011 (60). The supporting analysis suggests that recycled and by-product Hg (along with reduced Hg mine production, as necessary) will be more than adequate to meet global Hg demand (35).

Retirement

The alternative to marketing surplus Hg is the intentional retirement of Hg, that is, permanent storage that removes Hg from commerce and the biosphere. Hg need not be recycled, or purified, before it is retired. Unprocessed wastes or pure Hg could be stored indefinitely in warehouses or engineered land disposal sites, such as lined and capped landfills. There have been few efforts to compare the benefits of recycling and retirement, although Sweden has decided to retire Hg wastes (61).

The US government, through the Defense National Stockpile Center (DNSC), owns one of the world's larger stocks of Hg, and in the early 1990s began selling it on the international market after declaring it unneeded for future defense needs. A moratorium on sales was declared in 1994 as a result of concerns that marketing Hg may contribute to global environmental contamination. The relative merit of selling *versus* retiring the Hg was studied (62), and in February 2006 the US government announced that the stockpile of some 4400 t of Hg would be stored indefinitely in a warehouse.

Hg IN FISHERIES AND HUMAN COMMUNITIES: COMPARTMENTS F & H

The consumption of fish is the primary route of exposure to Hg in the form of MeHg (pathway FH*). Patterns of human exposure to MeHg are largely determined by the global distribution of fisheries, the trade in fish, and fish consumption.

Concentrations of MeHg in fish tissues vary by over a factor of 10 (63) because of variation in the environmental biogeochemical pathways of Hg and in aquatic food webs. Efforts to quantify the benefits of reducing anthropogenic Hg pollution are dependent on biogeochemical and food web models relating Hg releases to fish concentration. Much of the research on the biogeochemical pathways of MeHg has focused on freshwater ecosystems; in contrast, marine fish make up 92% of the global fish harvest (64). The quantification of reduction benefits would be enhanced if research efforts more closely matched the source of fish that people eat.

The global fish harvest has increased in recent decades and appears to have stabilized at around 130×10^6 t, a figure that includes aquaculture (30×10^6 t) and fish reduced and processed for use in meal employed in both agriculture and aquaculture (20×10^6 t) (64, 65). Based on these estimates, per capita consumption is 24 and 14 kg live weight per year in developed and developing countries, respectively. In some countries (notably in the western Pacific region), annual fish consumption is appreciably greater, ranging up to 75 kg per person-year. Freshwater fish account for about 5% of the total fish harvest in developed countries and 15% in developing countries.

Over one third of the global marine harvest of approximately 85×10^6 t enters international trade. Half of the production from developing nations, including tuna and other high-value piscivores, is exported to industrialized nations. The ability to assess patterns of exposure to MeHg is limited by data needed to link Hg concentrations to trade statistics. The quantity of fish produced via aquaculture is an increasing proportion of the global fish production (some 25%), but little is known about MeHg concentrations in these fish.

Fisheries are not easily classified. Commercial, recreational, and subsistence fisheries are often closely interrelated. Subsistence fishers often sell part of their catch commercially—a practice that yields cash for goods that must be purchased, such as boats, motors, gasoline, and market foods. Small-scale production of fish appears to involve some 35 million producers and their families, of whom fewer than 5% are from developed nations (65). Subsistence fishing—fishing primarily for local

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Table 3. Summary of economic analyses that have been performed on the costs or benefits of reducing mercury emissions or just reducing exposure through fish consumption advisories (e.g., 92). All of the studies concern the United States.

Study	Scenario	Health endpoints or other endpoints	Benefits measurement tools	Costs or benefits in 2004 USD (entire US unless noted)
EPRI (77)	Utility sector cap of 15 tons by 2018 or MACT by 2008 (about 24 tons emitted).	IQ change in fraction of population above MeHg RfD.	Benefits not monetized	Cost of cap: 6×10^9 Cost of MACT: 19.3×10^9
Gayer and Hahn (78)	Utility sector cap of 15 tons by 2020 or MACT by 2008.	IQ	Parental willingness to pay for IQ increases through chelation therapy.	Cost of cap: $3.4–5.5 \times 10^9$ Benefits of cap: $60–150 \times 10^6$ Cost of MACT: $15.4–20.7 \times 10^9$ Benefit of MACT: $82–142 \times 10^6$
Hagen et al. (69)	50% reduction from all sources in Minnesota (US).	Unspecified health effects, recreational fishing, effects on wildlife	CVM	Benefits to Minnesota residents: 255×10^6 y^{-1} (1996 Minnesota population = 4.7 million).
Jakus et al. (92)	Issue mercury-related advisories on the Maryland portion of Chesapeake Bay (US)	IQ, AMI, ACM Recreational fishing Commercial fishing	COI VSL TCM	Benefits: Avoided illness 15.4×10^6 y^{-1} for consumers of Maryland–Chesapeake Bay fish. Lost recreation and commercial value: 9.1×10^6
Lutter et al. (83)	60%–90% reduction in power plant emissions (US)	Neurological deficiency	Benefits not monetized.	Total cost: $1.2–1.9 \times 10^9$ 120 000–190 000 per case averted
Palmer et al. (79)	Hg cap under CAIR for power plant emissions (US)	IQ, AMI, ACM	COI VSL	Cost: 3.4×10^9 Benefit: Same as Rice and Hammitt
Rae and Graham (84)	30%, 51%, and 100% reductions in power plant emissions (southeastern US)	IQ, nonfatal AMI, hypertension, ACM	COI VSL	Benefit: $619–2\,102 \times 10^6$ y^{-1} for four states in SE United States, only.
Rice and Hammitt (80)	26 t and 15 t US power plant emissions caps	IQ, alternatively assume with and without a threshold, nonfatal AMI, ACM	COI VSL	Benefits: IQ only (threshold): $0.075–0.120 \times 10^9 y^{-1}$ IQ (no threshold) plus AMI plus ACM: $3.8–5.7 \times 10^9 y^{-1}$
Trasande et al. (81)	Evaluate costs of current emissions from all sources	IQ	COI	9.5×10^9 per birth cohort 1.4×10^9 due to power plants
US EPA (76)	Cap of 38 t in 2010, 15 t in 2018.	IQ	COI	Benefits: 0.25 to 1.56×10^6
US EPA (85)	Hg cap under CAIR and CAMR for power plant emissions (US)	IQ	COI	Costs: 750×10^6 y^{-1} by 2020 Benefits: Less than 168×10^6 y^{-1}

Abbreviations: ACM = all cause mortality; AMI = acute myocardial infarction; COI = cost of illness; CVM = contingent valuation method; IQ = intelligence quotient change; MACT = maximum achievable control technology; TCM = travel cost method; USD = US dollars; VSL = value of a statistical life.

distribution and consumption—remains a significant human exposure pathway in some populations, and can be associated with relatively high levels of chronic MeHg exposure (66). Subsistence fisheries are often held as common property with rules of conduct tending to be informal, local, and unwritten (67). Quantitative data on production and distribution of fish are rarely available, thus complicating the evaluation of MeHg exposure. The Arctic and sub-Arctic regions present a challenge on several fronts, including the finding that Hg from lower latitudes may be deposited by so-called "Hg-depletion events" (1), even though there are few anthropogenic emission sources. In some Arctic communities, marine mammals are also a significant dietary source of MeHg. In both Arctic and sub-Arctic settings, alternatives to locally harvested fish and marine mammals may be culturally untenable, unavailable, or not affordable (68).

ECONOMIC ANALYSES OF MERCURY USES AND POLLUTION

Economists often discuss a good's "opportunity cost"; that is, what must be sacrificed to obtain a good or service. For most goods, this is reflected in the price. But when the production of a good involves the release of a pollutant, such as Hg, the price may not include the associated environmental and social costs; these costs are called externalities. A negative production externality implies that individuals are adversely affected by production in ways that did not get factored into the product price; for anthropogenic Hg releases, this results when individuals are exposed to Hg or MeHg, as described in Figure 2. Economists have developed a number of methods to measure the costs of these externalities. We briefly review those methods here, focusing on methods to quantify the benefits to human health associated with reduced Hg exposure. Relative to humans, the effects of Hg on wildlife are poorly understood, so with the exception of the effect of consumption advisories on recreational fisheries, the economic value of reducing Hg pollution has seldom been quantified for wildlife and ecosystem functioning. As our review of existing studies shows, the scientific uncertainties even for humans lead to differing assumptions about health outcomes, which are translated into substantial variation in benefit estimates.

An additional consideration is that economic valuation methods do not work well when the implied tradeoff exceeds a person's or community's ability to pay. Economic analysis becomes very challenging if such tradeoffs are not possible, as might be the case when valuing Hg contamination to subsistence fishing communities with no practical dietary substitutes for Hg-contaminated fish.

Few economic studies have been conducted to quantify the benefits of reducing Hg pollution, and all of those have been conducted in the United States (Table 3). Among those few studies, there is only one that included wildlife benefits (69). Including the effects of Hg on wildlife and ecosystems is a

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daunting task; those studies that exclude such benefits will underestimate the full benefits of Hg reduction (70).

Economists employ two general approaches in measuring the human health benefits associated with a policy (71). Benefit–cost analysis (BCA) evaluates changes in health using monetary values, such as the cost of illness (COI) and willingness to pay (WTP) or willingness to accept (WTA) approaches. Cost-effectiveness analysis (CEA) evaluates these changes using summary measures of population health [e.g., disability-adjusted life years (DALYs) and quality-adjusted life years (QALYs), more generally known as health-adjusted life-years (HALYs)]. BCA collapses all health-related benefits and costs into a single, monetary metric from which economic efficiency can be evaluated. Those policies in which the incremental benefits exceed the incremental costs are beneficial to society. In contrast, CEA does not monetize benefits, instead this approach evaluates potential policies by comparing the ratio of a policy's cost to its health outcome, in cost per HALY. Policies with low cost per HALY ratios are typically preferred to those with high ratios.

Health Benefit Assessment Using Benefit–Cost Analysis

Cost-of-illness methods are used frequently to monetize the health improvement associated with a change in morbidity (illness). These methods measure the direct costs (e.g., treatment costs) and indirect costs (e.g., foregone income) associated with illness and injury, but do not measure losses associated with pain and suffering. For MeHg, COI methods link changes in the exposure of pregnant women to modeled changes in the IQ of their offspring. IQ changes are subsequently linked to changes in future income (throughout adulthood) and supplemental educational costs. Similarly, MeHg exposures can be linked to nonfatal heart attacks, with COI used to estimate the benefits of Hg reduction policies.

WTP methods measure an individual's willingness to exchange wealth for health. Economists consider such methods to be superior to COI methods because they include factors such as impairments to quality of life. For example, the value of a statistical life (VSL) measures an average individual's willingness to pay for a small change in the probability of dying sooner (72–75). Consider a Hg-reduction policy that could reduce the probability of death in a population from 2×10^{-5} to 1×10^{-5}, a reduction of one death per 100 000 people. If people are, on average, willing to trade 60 USD in exchange for this policy, then the VSL is 6 million USD (60 USD divided by 1×10^{-5}). Currently, the most common estimates for VSL used by researchers and policy makers in industrialized countries are between 3 and 7 million USD; the range arises, in part, because of the different values people hold for different types of mortality risks. VSL is positively related to income: a VSL estimated in a high-income country is generally greater than that estimated in a low-income country.

Table 3 summarizes a number of studies that attempt to monetize the benefits (usually human health benefits) associated with reduced MeHg exposure. The BCA conducted by the US Environmental Protection Agency's (US EPA) (76) as a component of the Clean Air Mercury Rule (CAMR) examined the costs incurred by US power plants to reduce Hg emissions and the monetized benefits associated with IQ increases in the children born to pregnant women whose MeHg exposures would be reduced as a result of the rule. Other groups also conducted benefit analyses of various Hg emissions reduction proposals under consideration during the development of this regulation (77–81). While the economic valuation models used are similar, assumptions regarding the impact of decreased Hg emissions on the changes in MeHg levels in different types of fish, and the health effects considered, differ markedly. All of the analyses emphasized the numerous uncertainties in evaluating specific policies for Hg reduction, including *i)* changes in Hg deposition rates, *ii)* changes in fish MeHg levels, *iii)* changes in MeHg intake by humans and the time it takes to observe this change, *iv)* changes in IQ due to fetal exposure, and/or *v)* changes in all-cause mortality and fatal and nonfatal heart attacks in adults. Some analyses assumed that Hg emissions markedly decreased only US freshwater fish MeHg levels; others assumed that marine fish levels, the primary source of MeHg exposure in the United States, could also decline as a consequence of decreased emissions. All analyses considered the impact of reduced fetal MeHg exposures on changes in IQ and lifetime earnings, although the slope of the MeHg IQ loss dose–response functions and lifetime earnings estimates vary. Some of these analyses also examined the impact of a toxicity threshold for the fetal neurotoxicity of MeHg that would be consistent with the US EPA's reference dose. Other studies included the possible economic impacts of decreased myocardial infarctions and premature mortality in adults. (See 11, 76, 80, and 82 for descriptions of the uncertainties in the epidemiologic data regarding these health end points.) The US EPA also simulated the time required for freshwater fish levels to change following an emissions reduction. These differences led to a large range of benefit estimates across the studies.

Much of the variability across the study results presented in Table 3 is a direct consequence of the differing assumptions that are made in response to uncertainties in the physical and health sciences of Hg and MeHg. For studies that have focused on nationwide programs, such as the emissions cap or the Clean Air Interstate Rule (CAIR) proposed in the United States (76–80, 83–85), we can see the impact of differing assumptions by comparing the results on a per capita basis. The EPRI study (77) and that by Gayer and Hahn (78) estimated costs of a 15-t cap-and-trade program at between 15 USD (Gayer and Hahn midpoint) and 21 USD (EPRI) per capita. Gayer and Hahn, focusing solely on IQ losses, estimate benefits of a Hg cap at less than 1 USD per capita. In contrast, the Rice and Hammitt study (80), examining a similar program but including IQ, heart attack, and all-cause mortality effects, and assuming that there will be changes in both freshwater and marine fish, estimate benefits at about 16 USD per capita. The authors noted that heart attack and all-cause mortality effects and marine fish MeHg reductions are much less certain than the other factors included in their analysis. Given the uncertainties in the timing of the benefits, they did not discount the future benefits. Gayer and Hahn firmly conclude that the benefits of the Hg cap-and-trade program are less than its costs. Palmer, Burtraw, and Shih (79), looking at Hg within the context of the CAIR, conclude that benefits (using Rice and Hammitt's estimate of 16 USD) exceed the estimated costs of CAIR (12 USD per capita). The primary factor driving the different conclusions is the decision on which health end points to include, a decision based on uncertainty in the physical and health sciences.

Economic benefits are based, fundamentally, on a person's WTP for reductions in Hg exposure. Whereas the studies in Table 3 represent a good start to a benefits literature, none of them address the theoretically correct, but difficult to estimate, WTP measures that incorporate uncertainty, such as "option price" (86). For example, consider exposure over time to a given level of Hg in commercially caught seafood. As a result of that exposure, one raises the risk of developing Hg-related health problems, such as AMI, above the baseline risk. Different people receiving the same Hg exposure will react differently: some will have a heart attack and others will not. The option price (OP) measures a person's WTP without resolution of this uncertainty, that is, without finding out if he or she is one of the

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people whose sensitivity to MeHg increases the likelihood of heart attack. The basic OP model can be augmented to incorporate uncertainty in the health risk itself (e.g., is the best estimate of MeHg-associated increase in AMI risk zero, or is it the dose-response slope 0.066 per ppm mercury in hair?); it can reflect endogeneity in the risk (e.g., risk estimates can be adjusted based an individual's actions, such as eating more or less fish), and it can include ambiguity about risks (a person may not have a good point estimate of their risk and instead consider their risk to be within a given range). An approach to economic analysis that explicitly incorporates uncertainty seems well suited for the case of Hg, in which policy choices must be made despite the presence of scientific uncertainty in environmental fate, exposures, and health effects.

The difficulty with such an approach is that different studies will produce different results. In most cases, a WTP measure such as OP would be elicited via stated preference methods, and WTP estimates will depend on the prior information that respondents have as well as that provided by the analyst. To the degree that researchers differ in the risks and uncertainties explained to respondents, study results will vary and must be evaluated within that context.

Health Benefit Assessment Using Cost-Effectiveness Analysis

The most commonly used HALY measures in CEAs are QALYs and DALYs. Different illnesses are associated with different degrees of severity; for these measures, severity is assessed by a utility weight (71, 73). The utility weights may be based on individuals' preferences for avoiding specific illnesses, or they may be based on expert opinions.

Both Ponce et al. (87) and Cohen et al. (88) examined neurocognitive deficiencies associated with fetal MeHg exposures, assuming that the deficiencies persist throughout the life of the affected individual. The benefit was assessed by multiplying the utility weight (i.e., the decrease in the quality of life that results from cognitive deficits) by the duration of the effect (persisting over the individual's lifetime). For QALYs, this calculation results in the prediction of the QALYs an affected individual experiences; they are compared with the increased number of QALYs an individual would experience if a policy to reduce MeHg exposures were put into place. The benefits are expressed as net QALYs gained by the population. For DALYs, the product of the utility weight and duration of the effect results in a prediction of disability-adjusted life years incurred. Population benefits of a policy are then evaluated by the decrease in the number of disability-adjusted life years incurred.

Both QALYS and DALYs are used as the denominators in cost-effectiveness analyses, with the numerator being the cost of the policy. The cost effectiveness of different policies can be compared; those that exhibit the largest gain in QALYs (or decrease in DALYs) for the lowest cost are preferred to those that exhibit smaller gains in QALYs for larger costs.

Benefits Assessment in Different Social Contexts

All of the BCAs and CEAs of Hg policies that we found have been conducted in developed countries, with monetary measures and HALYs used to address different economic questions associated with MeHg exposures that result from eating fish. While some of these analyses have attempted to identify subpopulations that benefit from implementation of a policy, most could be improved from additional analyses of the distribution of benefits and costs across the population. Finally, some of these analyses have examined the time frame over which anticipated MeHg exposure decreases and health-consequent benefits might occur. For many, benefits accruing soon after expenses are incurred are preferred to those same benefits accruing in the future (i.e., time value of money). While beyond the scope of this manuscript, both the manner and rate by which future health benefits are discounted is central to many policy analyses and may be particularly important for Hg, given the uncertain environmental response times implied by current research on the biogeochemistry of Hg (3).

In contrast to the number of evaluations in developed countries, we are not aware of a benefits assessment that examines the costs or benefits of reduced Hg releases and MeHg exposures in developing countries, or benefit assessments focused on those engaged in subsistence fishing or small-scale gold mining (Table 4). Such assessments could differ substantially from those in developed regions. Moreover, caution is warranted when applying these economic techniques in situations in subsistence societies where some Hg policies may greatly disrupt the social structure in a society with few alternatives to subsistence fisheries (Table 4; 89, 90), or in regions where small-scale gold mining is prevalent and there is no realistic alternative to using Hg to make a living. Table 4 also demonstrates that economic analyses of the costs and benefits of reducing mercury reductions are lacking for many of the Hg exposure pathways.

Assessments for Other Effects, Including Recreational Fishing and Environmental Degradation

Hg contamination may affect the quality of current recreational experiences, decrease future recreational use in this generation and future generations, and affect other values that one might hold irrespective of use. In a BCA, values for these impacts may be elicited directly by using stated preference techniques that rely upon choice in hypothetical situations (e.g., choice experiments or contingent valuation) or they may elicit value indirectly by using revealed preference approaches based on the observed choices of people (e.g., the travel cost model). Champ, Boyle, and Brown (91) provide an excellent introduction to these methods.

Jakus, McGuinness, and Krupnick (92) used observed human behavior to measure changes in commercial and recreational values due to fish and wildlife consumption advisories. For commercial fisheries, the cost of advisories was based on the market demand and supply for contaminated commercial species. Advice suggesting reduced consumption of striped bass means that at least some consumers will consume fewer bass at any given price. This "shift" in commercial demand is used to measure cost in terms of lost market value accruing to consumers and producers. A similar approach was used to measure impacts on recreational angling. Advice advocating restricted consumption of fish or wildlife altered the number of recreational trips or changed the species targeted. The travel cost method was used to calculate the change in the net value of fishing with and without consumption advisories. The method does not account for possible health impacts to those who do not change behavior in response to advisories.

Hagen, Vincent, and Welle (69) used a stated preference method, contingent valuation, to value changes in human health as a result of Hg-reduction policies, as well as the effects on recreational anglers and on wildlife. In their study, people were asked to estimate their willingness to pay for a policy designed to reduce environmental Hg levels. Respondents were asked to consider benefits from changes in their health, their family's health and, possibly, the health of their neighbors and others. As such, their analysis included cultural values, as in the case of a parent who would like his or her children to enjoy eating fish that they have caught as a family, irrespective of the health implications. Values may have been expressed for future

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Table 4. Mercury exposure pathways and the extent of knowledge pertinent to cost–benefit analysis of reduction options. Benefit and cost methods that have been applied are in bold (references in parentheses); other possible approaches are listed. In many cases a necessary prerequisite is to quantify any connection between Hg releases and any endpoint.

Exposure pathway	Measurement tools (for benefits of reducing exposure)	Source of elevated Hg	Hg exposure reduction options	Measurement tools (for costs of reducing exposure)
FH—commercial fishing	**COI, VSL** (76, 78, 80, 81, 84, 85, 92)	VA—atmospheric deposition	a. Reduce Hg emissions from coal, ore refining, and small scale gold mining via: i. Incentives to develop control technology. ii. Incentives to transfer control technology globally. b. Purchase and consume low-Hg fish c. Consume nonfish source of protein	a. **COT** (77) b. **LMV (92)** c. COS
FH—recreational fishing	**COI, VSL (92)**	VA—atmospheric deposition	a. Reduce Hg emissions b. Consume low-Hg fish, release high-Hg fish. c. Fish low-Hg waters (e.g. different lake)	a. **COT (78)** b. **LRV (92)** c. **LRV (92)**
FH—subsistence fishing	COI, VSL	VA—atmospheric deposition	a. Reduce Hg emissions b. Reduce fishing & find alternate source of protein. c. Disseminate culturally appropriate fish consumption advice. d. Consume low-Hg fish, sell high-Hg fish.	a. COT b. QOL c. QOL d. QOL
FH—subsistence fishing	COI, VSL	DA—disposal of products and wastes to aquatic systems	a. Reduce Hg discharge to fishery b. Reduce fishing and find alternate source of protein c. Create incentives for switching to Hg-free products and processes for chlor-alkali and vinyl chloride monomer plants d. Disseminate culturally appropriate consumption advice. e. Consume low-Hg fish, sell high-Hg fish.	a. COT b. QOL c. COS d. QOL e. QOL
FH—subsistence fishing	COI, VSL	OA—ore refining discharges	a. Reduce Hg discharge b. Reduce fishing and find alternate source of protein d. Disseminate culturally appropriate fish consumption advice e. Consume low-Hg fish, sell high-Hg fish	a. COT b. QOL c. QOL d. QOL
FH—subsistence fishing and recreational fishing	COI VSL	Reservoir creation or operation	a. Evaluate impacts before reservoir creation b. Create and operate to minimize Hg in fish c. Reduce fishing & find alternate source of protein d. Disseminate culturally appropriate fish consumption advice e. Consume low-Hg fish, sell high-Hg fish f. Consume low-Hg fish, release high-Hg fish	a. COT b. COT COS c. QOL d. COT e. QOL f. **LRV (92)**
FH—subsistence fishing	COI VSL	SA—small-scale gold mining discharges	a. Provide incentives for efficient or reduced Hg use through: i. Reduce international supply of Hg through retirement, etc. ii. Transfer of culturally acceptable technology. iii. Build community capacity to reduce Hg-related problems. b. Economic development for opportunities other than gold mining. c. Reduce fishing & find alternate source of protein.	a. COS COT i. **COHR (62)** ii. COT iii. COT b. COED c. COS QOL
FW—wildlife consumption of fish	**SPM (69)**	VA—atmospheric deposition	a. Reduce Hg emissions.	COT
FW—wildlife consumption of fish	SPM	Reservoir creation or operation	a. Evaluate impacts before reservoir creation. b. Operate to minimize Hg in fish.	a. COT b. COT COS
FW—wildlife consumption of fish	SPM	OA—ore refining	Reduce Hg discharge	COT
FW—wildlife consumption of fish	SPM	DA—disposal of products, wastes to aquatic systems	Reduce Hg discharge to fishery	COT
FW—wildlife consumption of fish	SPM	SA—small-scale gold mining	a. Provide incentives for efficient or reduced Hg use through: i. Reduce international supply of Hg through retirement, etc. ii. Transfer of culturally acceptable technology iii. Build community capacity to reduce Hg-related problems b. Economic development for opportunities other than gold mining	a. COS COT i. COHR ii. COT iii. COT b. COED
SH—Inhalation exposure	COI, VSL	SV—inhalation while concentrating gold	a. Provide incentives for efficient or reduced Hg use through: i. Reduce international supply of Hg through retirement, etc. ii. Transfer of culturally acceptable technology iii. Build community capacity to reduce Hg-related problems b. Economic development for opportunities other than gold mining	a. COS COT i. COHR ii. COT iii. COT b. COED
MH—inhalation exposure	COI, VSL COI, VSL	MV—inhalation while manufacturing with Hg	a. Provide incentives for Hg-free processes b. Develop inexpensive Hg vapor monitoring	COS COT

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Table 4. Continued.

Exposure pathway	Measurement tools (for benefits of reducing exposure)	Source of elevated Hg	Hg exposure reduction options	Measurement tools (for costs of reducing exposure)
PH—inhalation exposure	COI	PV—Hg vapor from Hg-containing product use and breakage	a. Choose Hg-free products (e.g., dental fillings, thermometers, medical devices, pharmaceuticals, barometers, paints, thermostats, lamps, etc.)	a. COS
			b. Create incentives for switching to Hg-free product production.	b. COS

Abbreviations: COED = cost of economic development; COI = cost of illness (including both MeHg and loss of protein and PUFA); COS = cost of substitute; COHR = cost of Hg retirement; COT = cost of technology, including the costs of research, and development and dissemination of fish consumption advice; LMV = lost market value; LRV = lost recreational value; PUFA = polyunsaturated fatty acids; QOL = quality of life; SPM = stated preference method; TCM = travel cost method; VSL = value of a statistical life (including both MeHg and loss of protein and PUFA).

generations and broader ecosystem services (so-called nonuse values), and thus WTP may or may not be tied to one's direct exposure to Hg.

While all of the previous analyses have been conducted in developed countries, both revealed preference and stated preference methods have been used in the developing nations on issues other than Hg. In less-developed regions, the suite of Hg impacts may be similar to those encountered in more-developed countries, but the values that these societies (e.g., subsistence fishing communities) place on these impacts could be very different from the values of those in more-developed countries (Table 4) and should be examined in future studies.

POLICY OPTIONS TO REDUCE Hg POLLUTION

Environmental Hg releases can be reduced by policies that reduce the supply or demand of Hg, implement technological controls (or processes) that reduce mercury releases during the production of goods that result in Hg releases (e.g., gold and electricity), or reduce the quantities of produced goods that result in such releases. In general, policy options used routinely to reduce pollutant emissions from industrial processes include technology requirements, emission performance standards, emission taxes, and cap-and-trade (CAP) approaches. Other policy options such as subsidies and restrictions on the sale and disposal of Hg (and Hg-containing items) could influence Hg releases from small-scale practices such as artisanal gold mining. Application of any of these alternative policies to Hg reduction will have benefits and costs. An economic approach to evaluating different policy options is to balance, at the margin, the benefits and costs of any policy option. Such policies are deemed economically efficient.

In the case of Hg, economic analysis is complicated by the need to track benefits and costs at various geographic scales, from local to global. While the costs associated with the implementation of new processes or control technologies can be estimated in a relatively straightforward manner, the assessment of benefits is complicated by the scientific uncertainties reported in the environmental literature [i.e., the linkages between reducing environmental Hg releases and lower levels of Hg in the atmosphere and in fish (1, 3)] and health science literature [i.e., linkages between reduced levels in the environment, reduced exposures, and health improvement (3, 11)]. Ideally, economic analyses highlight these uncertainties as well as those introduced in the benefit–cost component of the analysis, and researchers will conduct additional analyses to assess the sensitivity of the results to the assumptions associated with the uncertainties (93). Resolution of uncertainty—regardless of the source—will increase the reliability of the economic analyses.

Each of the main policy options has particular strengths and weaknesses. Technology requirements mandate a particular production or control technology, and can have the advantage of offering well-understood pollution reduction. On the other hand, for any specific level of reduction in the release of a given pollutant, economic models suggest that, when compared with other policy options, technology mandates generally have a higher cost than more flexible approaches and may provide less incentive for technological innovation (94, 95). Performance standards offer some flexibility by which firms can reduce costs; for example, firms have an economic incentive to develop less costly control technologies. Ideally, performance standards provide regulators assurance about the level of a pollutant released at each regulated source, an important advantage for pollutants that can have significant local impacts.

Market-based reduction policies can take the form of emission taxes or CAP. Under an emission tax approach, a source may emit any quantity of a pollutant desired but is taxed for each unit released. In CAP, the regulatory authority sets an aggregate emissions level and issues permits (that sum to the target level) to polluters by an auction or simple distribution. Polluters are free to trade permits, with the prevailing price naturally reflecting the incremental cost of control. Economic models suggest that market-based policies spur innovation of new production technologies that are more efficient or less costly than extant technologies (34, 94, 95). If sources of emissions face differing abatement costs, the emissions tax and CAP approaches generally offer lower total costs of control (when summed over all facilities) relative to technology or performance standards. This is because choices about how much to control are made by facility management based on the prices they face rather than by the regulatory authority. A requirement of either the emissions tax or CAP approaches is that emissions be accurately monitored and enforced. For pollutants with local impacts, a key disadvantage of the emissions tax and CAP approaches is the potential for local impacts to persist or increase where facilities deem it uneconomical to reduce pollution. Some have suggested that this problem can be addressed through differential trading rates in a CAP (96), differential taxes, or combining a CAP with a performance standard. Devising a policy to avoid local impacts under a CAP would require considerable knowledge about local factors that control exposure to such a pollutant.

With respect to large point sources of Hg emission to the air, reduction policies have generally relied on performance standards; for example, in the United States, performance standards were finalized for municipal waste combustors in 1995, for medical waste incinerators in 1997, and for hazardous waste incinerators in 2002 (43). However, in the case of coal-fired electric power plants, in 2005 the US federal government promulgated a CAP (5, 76). At the time of this writing, the regulation on Hg releases from both large and small sources, and strategies to address supply and demand of Hg, are rapidly evolving. This evolution is partly a function of the limits of

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economic analysis and uncertainties over the environmental fate, exposure, and health effects associated with Hg releases. While an economic approach may assess the relative economic efficiency of any set of potential policies, the choice of any one policy is subject to a variety of factors, only one of which is efficiency. For example, two different policies may be economically efficient, yet reduce very different amounts of pollutant, have different levels of benefits and costs, or have benefits and costs that are distributed quite differently across a population. Strictly speaking, benefit–cost analysis has little to say about a choice between these two policies. Thus, acceptable policies must also satisfy political, social, and cultural criteria.

POLICIES TO LIMIT EXPOSURES TO MERCURY THROUGH RISK COMMUNICATION

In addition to reducing Hg releases, human Hg exposures can also be reduced through risk communication policies, including fish consumption advisories, improved communication of the occupational risks associated with Hg releases during artisanal gold mining, and product labeling. In this section, we focus on consumption advisories and also provide a brief description of the risk communication challenges associated with small-scale gold mining. We note that the impacts of product labeling policies need additional study.

Fish consumption advisories are considered by many policy makers to be an unfortunate and, hopefully, interim public health necessity. In general, advisories are based on an assessment of the human health risks associated with pollutant exposures, including fetal exposures that result from consuming contaminated fish (e.g., 97). The primary policy goal of a fish consumption advisory is to reduce pollutant exposure by reducing the intake of a contaminant (MeHg, in this case), while maintaining recommended fish intake; this is accomplished by recommending consumption of different fish species, smaller fish, or fish from a different fishery (i.e., a different body of water that has less contaminated fish). The risk managers who develop fish consumption advisories consider multiple issues, including what level(s) of fish contamination should trigger the issuance of an advisory, the species of fish involved, and their availability to consumers. Consideration is given to identifying groups or individuals who should follow the advisory (e.g., fishers, women of reproductive age, or those responsible for food preparation). Techniques for communicating with different audiences need to be evaluated. Last, but certainly not least, the languages and concepts used by consumers to convey information should be studied, as well as aspects of local fishing economies, including distribution and sharing practices. The practical implications of advisories on fish consumption have seldom been documented, and most likely vary a great deal depending on the nature of the advice, how it is communicated, and the alternatives available to the community (e.g., 98, 99).

For public health officials, a fundamental tension exists in the development of fish consumption advisories: how to communicate to people that they should avoid eating highly contaminated fish, but that they should continue to eat fish because of its nutritional value and the associated health benefits. Fish are high in protein and low in saturated fat. Frequent fish consumption has been shown to reduce the risk of heart disease (100). Policy makers, thus, prefer to consider such advisories as "interim" in the expectation that other measures can be implemented that will effectively reduce the fish tissue MeHg concentrations (3). Knowledge of the impacts of fish consumption advisories on diet is quite limited, and this is an area that requires further research (88).

The contamination of fish by MeHg can be an elusive concept to convey because this toxicant cannot be detected visually in the fish and the fish do not appear to be diseased. Consumers can interpret consumption advisories in various ways; accurate communication of an advisory can be especially difficult when there are linguistic or cultural differences between the risk manager and the affected population (99). Since individuals who communicate advisories may differ in their views of the risks involved and their relation to the nutritional role of fish (e.g. 101), conflicting advice may be received by a fishing community. Those who develop advisories, therefore, require appropriate knowledge of the nature of the fishery itself, as well as fish sharing, preparation, and consumption rates and practices (97, 102, 103).

The use of fish consumption advisories presents additional challenges for remote and isolated communities dependent on subsistence fisheries, including some North American indigenous populations (99). In some subsistence societies, advisories have resulted in widespread avoidance of fish rather than reduced consumption of the most contaminated fish (104). The loss or substantial disruption of local fisheries can also have significant public health implications, as well as related cultural impacts (105). Alternative dietary choices available in larger population centers may simply not be available to subsistence populations, and the communities may already face significant diet-related public health problems (such as diabetes). In such settings, health service providers face the task of balancing the message of the consumption advisories against the public health consequences of significant changes in human nutrition (106).

In 2004, the US EPA and Food and Drug Administration (FDA) released a joint statement (107) that described fish and shellfish as important components of a healthy diet. It noted that "a well-balanced diet that includes a variety of fish and shellfish can contribute to heart health and children's proper growth and development," although "nearly all fish and shellfish contain traces of Hg ...." Finally, the statement advised women who may become pregnant, pregnant women, nursing mothers, and young children to avoid some types of fish (i.e., shark, swordfish, and king mackerel) because of the high MeHg levels and to eat up to two meals each week of fish or shellfish that are low in MeHg (e.g., shrimp, canned light tuna, salmon, pollock, and catfish). The joint statement also cautioned consumers to check local advisories about the safety of consuming fish caught in local bodies of water, noting that if no advice was available, to eat up to one fish meal of average size per week, but not to consume any other fish during that week. Several investigators have compared the risks associated with MeHg exposures and the nutritional value and health benefits of fish consumption (11, 87, 88, 108). Their findings generally are consistent with the EPA and FDA statement.

In the case of small-scale gold mining using Hg amalgamation, the primary toxicological issue is the inhalation of Hg converted to the gas phase during the heating of the amalgam. Heating often takes place inside or near the home. Artisanal workers and their families can be exposed to harmful levels of Hg vapor. Risk communication in the form of advice to avoid the Hg amalgamation technique or to reduce exposure during its use must take into account the limited options available to the gold miners and the widespread poverty and hardship associated with this occupation. Field researchers (e.g., 48, 53) emphasize that effective risk communication strategies need to be intertwined with strategies targeting improved profitability through better gold recovery methods or reduced losses of Hg, thus reducing the artisinal miner's production costs. Within each country, the industry is geographically scattered, so the logistical aspects of risk communication are a major challenge. Thus, to be effective, in each region, risk communication

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strategies may involve training a cadre of small-scale gold miners who can demonstrate and discuss the advantages of improved practices to their fellow miners (53).

CONCLUSIONS

Mercury is a naturally occurring element that can affect the health of humans and wildlife. Humans have historically found Hg to be a useful liquid metal for a variety of social and economic purposes, and currently release some 2400 t y^{-1} of Hg into the atmosphere. The best estimate is that almost 90% of anthropogenic emissions comes from the combustion of fossil fuels in electric power generation and from the release of Hg in large- and small-scale mining operations, although emissions associated with intentional Hg use are difficult to quantify and may be substantially underestimated. Some Hg emissions to the atmosphere are deposited relatively near the source, but a large portion enters the global atmospheric reservoir.

The most important pathways of human exposure to Hg are through the consumption of MeHg-contaminated fish and inhalation of Hg vapor. Worldwide, the greatest source of inhalation exposure likely is from small-scale gold mining. The release of Hg used in restorative dentistry can be a source of exposure, but the associated risks are not well understood. Exposure to environmental Hg is believed to have a number of potential negative effects on human health, including: *i)* cognitive deficits (e.g., reduced IQ) in children due to fetal exposure and in adults exposed to high concentrations of Hg vapors, *ii)* possible increases in fatal and nonfatal heart attacks, and *iii)* increases in premature death (i.e., some studies link Hg exposures to increased risk of premature mortality regardless of cause).

While the general pathways outlined in this paper are accepted, a great deal of uncertainty remains in the linkages between emissions and human health. Resolution of these uncertainties is important for analysis of Hg reduction policies but will be difficult to achieve. The key physical and health science questions include the magnitude of the environmental response, the environmental response time (i.e., the length of time between decreased Hg emissions and consequent changes in human health risks), to what degree decreased exposures will reduce the risks of cognitive deficits, heart attack, or premature death and, if so, in which populations. While some exposure pathways are better understood than others, the full extent of health damages in many of these pathways is not known. For example, while fetal exposures are relatively well studied (although uncertainties remain), the dose–response functions for health effects resulting from chronic exposure in adults, such as cardiovascular disease, remain uncertain.

Uncertainty with respect to health effects has implications for choices among many policy alternatives. Both of the primary approaches to economic evaluation, BCA and CEA, require that one be able to link changes in Hg emissions to changes in various health outcomes; uncertainty with respect to policy benefits implies uncertainty in the benefit–cost or cost-effectiveness metrics coming out of the analysis. The few economic studies of Hg policies all express concern about the degree of uncertainties in the physical and health science of Hg exposure, suggesting that a valuation approach that explicitly deals with uncertainty and ambiguity is warranted. Three other aspects of the economic and social analysis are also important for future research. First, all of the economic studies have been conducted in the context of a developed economy, but key aspects of Hg reduction policies affect people living in developing countries or regions (e.g., small-scale mining operations or subsistence fisheries). Second, little research has been done to quantify non–health-related benefits, such as nonuse values associated with benefits to future generations. Third, costs (both economic and social) associated with structural changes in local food production systems such as subsistence fishing are not well understood and, therefore, not well quantified in cost–benefit analyses. Given uncertainty with respect to benefits and costs, it is difficult to conclude *a priori* that any one Hg-reduction policy is "better" than any other from an economic point of view, or even if the benefits of such a policy exceed the costs.

Given the variety of sources and reduction options, many approaches to reduce Hg releases or exposures are being proposed and considered at multiple levels in governments around the world. Economic and comparative risk analyses need to be conducted to examine these approaches. Because Hg emissions in one part of the planet are transported globally, the efficiencies and risks associated with these approaches need to be evaluated in light of the local, regional, and global impacts. Likewise, international trade also moves elemental Hg globally and is linked to emissions and exposure; better data on country-to-country and in-country commercial flows of Hg (indeed, most countries have a poor understanding of their domestic use of Hg) will contribute to refining existing policies and developing new ones. Decision makers also need to analyze potential interrelationships between policies so that one policy does not reduce the efficiency of another. To the extent possible, effective and acceptable policies must satisfy political, social, and cultural criteria, as well as economic criteria.

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The Madison Declaration on Mercury Pollution

This declaration summarizes the scientific and technical conclusions presented by four expert panels in their critical synthesis manuscripts and in plenary sessions at the Eighth International Conference on Mercury as a Global Pollutant, convened in Madison, Wisconsin, USA, on 6–11 August 2006. The 1150 registered participants in this conference constituted a diverse, multinational body of scientific and technical expertise on environmental mercury pollution. This declaration conveys the panels' principal findings and their consensus conclusions on key policy-relevant questions concerning atmospheric sources of mercury, methylmercury exposure and its effects on humans and wildlife, socioeconomic consequences of mercury pollution, and recovery of mercury-contaminated fisheries.

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MADISON WISCONSIN
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SOURCE ATTRIBUTION OF ATMOSPHERIC MERCURY DEPOSITION

Panelists: Steve Lindberg (Chair), Russell Bullock, Ralf Ebinghaus, Daniel Engstrom, Xinbin Feng, William Fitzgerald, Nicola Pirrone, Eric Prestbo, Christian Seigneur

Question to the Panel: For a given location, can we ascertain with confidence the relative contributions of local, regional, and global sources, and of natural versus anthropogenic emissions to mercury deposition?

(A1) Identification of Atmospheric Sources. It is possible to infer local, regional, and global atmospheric sources of mercury, depending on the level of uncertainty considered acceptable. Greater confidence can be assumed for source-receptor assessments that are very near or very far from major point sources and if the "global pool" is considered a recognizable "source." About two-thirds of the "global background mercury" or global pool is derived from human activities, and about one-third is from natural (geologic) sources. Many locations of interest or concern are affected primarily by mercury sources located between the local and global scales (i.e., at the intermediate scale), where source-attribution assessments would have the greatest uncertainty. *(93% support)*

(A2) Attribution of Natural vs. Anthropogenic Emissions. Compared to gaseous elemental mercury, oxidized forms of mercury that exist in a point-source plume can be rapidly deposited and thus may constitute a significant portion of observed mercury deposition near the source. Emissions originating as reactive gaseous mercury (i.e., not from post-emission oxidation during transport) are nearly always from anthropogenic sources, whereas emissions from undisturbed natural sources and from surfaces emitting previously deposited mercury are dominantly elemental mercury. It can, therefore, be reasonably inferred that emissions from natural surfaces have minimal effect on local atmospheric deposition near major source areas. *(87% support)*

(A3) Long-Term Trends and Source-Receptor Relations. Since the Industrial Revolution, mercury deposition has increased globally by a factor of two to four, even at remote locations. During the past 30 years, however, there has been no discernable net change in the global atmospheric pool of mercury, or total emission inventory of mercury, even though North American and European emissions have decreased substantially. In view of these emission reductions, the observed steady-state nature of the global atmospheric pool of mercury suggests a probable compensating factor, a contemporaneous increase in emissions from other geographic areas, or both. *(97% support)*

(A4) Atmospheric Response Times. Ten years ago, the half life of mercury in the global atmosphere was estimated to be about 1 year, whereas present estimates range from months to a year. Because the atmospheric global pool is relatively well constrained, a shorter half life of mercury in the atmosphere generally implies a higher deposition flux resulting from greater (than previously recognized) oxidation rates, the propensity for recently deposited oxidized mercury to be reduced to Hg^0 and emitted back to the atmosphere, and a potentially greater deposition rate of elemental mercury. *(91% support)*

(A5) Importance of Wet vs. Dry Deposition. In the past decade, several studies in forest settings in Europe and North America have shown that mercury fluxes in litterfall plus throughfall (as a surrogate for total wet + dry deposition) to the forest floor range from about two- to seven-fold greater than wet-deposition fluxes. Given the very low levels of reactive gaseous mercury observed in many remote forested areas, it can be reasonably inferred that a significant portion of the mercury deposited onto the forest canopy is derived from the atmospheric pool of elemental mercury. This observation has substantial implications for estimates of atmospheric deposition at local, regional, and global scales if it can be assumed that this additional flux is from dry deposition to the forest canopy. This flux of elemental mercury to the forest canopy supports the estimates of a shorter half life of mercury in the global atmosphere. *(92% support)*

(A6) Modeling and Uncertainty. Widely accepted atmospheric chemical-transport and receptor models have been applied to atmospheric mercury source assessments and are valuable tools for advancing our understanding. These models

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provide the best approach for assessing the relative importance of direct reactive mercury emissions versus post-emission formation of reactive mercury in controlling deposition patterns and rates. Although these models have advanced considerably during the past decade, more refinement of several important model parameters and inputs are needed to improve our confidence in the predicted results. These include measurement of species-specific dry deposition rates, altitudinal distributions of mercury species, identification and quantification of homogeneous and heterogeneous reaction rates, mercury speciation at emission sources, more complete global mercury emission inventories (particularly, but not solely, for developing nations), and meteorology. *(95% support)*

(A7) Other Important Nonmercury Factors. It is possible, and perhaps likely, that the global mercury cycle has been altered substantially in recent decades by nonmercury factors, most prominently physical and chemical climate change (e.g., increases in ozone concentration in the troposphere). Increases in surface temperatures and wind speeds, changes in precipitation patterns, secondary effects related to increases in ozone concentration and aerosol loading, decreases in sea-ice cover, and changes in vegetation could measurably affect the atmospheric half life of mercury, its deposition patterns, and source-receptor relations on local, regional, and global scales. Such confounding factors could influence source-attribution assessments and interpretation of long-term trends. *(84% support)*

HEALTH RISKS AND TOXIC EFFECTS OF METHYLMERCURY

Panelists: Donna Mergler (Chair), Anton Scheuhammer (Co-Chair), Henry Anderson, Laurie Chan, Kathryn Mahaffey, Michael Meyer, Michael Murray, Mineshi Sakamoto, Mark Sandheinrich, Alan Stern

Question to the Panel: What is the evidence that humans, fish, wildlife, and other biota are being adversely affected by exposure to methylmercury?

Human Health

(H1) Exposure to Methylmercury. Methylmercury is a highly toxic compound that biomagnifies through the aquatic food web, placing at risk humans who consume significant quantities of predatory fish from upper trophic levels or who rely heavily on fish as a food source. Elevated methylmercury exposure in humans is not restricted to isolated populations, because of worldwide export and availability of commercially caught fish. Rather, human exposure to methylmercury at levels exceeding those considered clearly safe and without risk of adverse effect has been observed across geographic, social, economic, and cultural boundaries. *(88% support)*

(H2) Trends in Methylmercury Exposure and Human Health. Present exposures throughout the world are lower than those that produced the historic epidemics of methylmercury poisoning in Japan and Iraq. In many populations, however, there is growing evidence that current exposures are sufficient to alter normal function of several physiological and developmental systems, indicating that methylmercury exposure still constitutes an important public health problem. Long-lasting effects of fetal methylmercury exposure have been described in children throughout the world. *(81% support)*

(H3) Biomarkers of Methylmercury Exposure in Humans. Concentrations of mercury in hair and blood (including umbilical cord blood) are both valid biomarkers of methylmercury exposure. Each measure conveys somewhat different information on exposure, and the most useful picture of exposure is obtained by data from both biomarkers, along with dietary information on the fish species consumed and other dietary data. Total fish consumption—without differentiating the fish species consumed—is not necessarily a dependable metric for estimating methylmercury exposure. *(92% support)*

(H4) Risk Assessment. Methylmercury is a developmental neurotoxin, and its developmental neurotoxicity to the fetus constitutes the current basis for risk assessments and public health policies. Uncertainties remain in the risk assessment for the neurodevelopmental effects of methylmercury. Yet there is sufficient evidence to warrant the prudent selection of fish species in the diet, particularly for pregnant women and children. *(92% support)*

(H5) Cardiovascular Effects of Methylmercury. Current studies suggest that exposure to methylmercury could increase the risk of adverse cardiovascular effects in a significant fraction of the human population. Reported effects include cardiovascular disease (coronary heart disease, myocardial infarction, ischemic heart disease), increased blood pressure and hypertension, and altered heart rate. The strongest cause–effect evidence is for cardiovascular disease, particularly myocardial infarction in adult men. *(70% support)*

(H6) Methylmercury and Omega-3 Fatty Acids. Fish can contain both methylmercury and beneficial omega-3 fatty acids. Methylmercury exerts toxicity and can also diminish the beneficial health effects of omega-3 fatty acids. As with mercury, there are large variations in the level of omega-3 fatty acids in fish. Selection of fish species for consumption should maximize the intake of beneficial fatty acids whereas limiting exposure to methylmercury. *(91% support)*

(H7) Mercury and Selenium. There is some evidence from animal studies showing that selenite protects against inorganic mercury toxicity. However, there is almost no evidence showing protection against methylmercury toxicity by organo-selenium compounds, such as selenomethione or selenocysteine, the forms of selenium commonly found in the human diet. There are no human data demonstrating a protective role for selenium against the neurotoxicity of mercury, including developmental neurotoxicity. *(69% support)*

Wildlife Health

(W1) Fish and Wildlife Species at Risk. Long-lived piscivores and other predators atop aquatic food webs are at greatest risk for elevated methylmercury exposure, accumulation, and toxicity. These species include predatory fish such as pike, walleye, and lake trout; mammals such as mink, otter, polar bears, and seals; and piscivorous birds such as common loons, bald eagles, osprey, and kingfishers. Nonpiscivorous terrestrial species (such as granivorous and insectivorous birds) generally have lower exposure to methylmercury and are generally at less risk than piscivorous wildlife for methylmercury toxicity. *(91% support)*

(W2) Trends in Mercury Exposure in Wildlife. Whereas available evidence indicates that reductions in mercury emissions have led to reduced mercury concentrations in fish and piscivorous wildlife in areas impacted by local or regional point-source industrial releases, current temporal trends indicate increasing concentrations of mercury in some piscivorous wildlife in some areas remote from industrial sources (for example, the Arctic). *(93% support)*

(W3) Biomarkers of Methylmercury Exposure in Wildlife. Concentrations of mercury in fur, feathers, eggs, skeletal muscle, and blood are valid biomarkers of methylmercury exposure in wildlife. Mercury concentrations in blood best integrate dietary methylmercury exposure at the time of sampling, whereas fur and feathers better integrate chronic

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exposure. Total mercury concentration in major organs such as liver or kidney may not be a good indicator of current mercury exposure or toxicity because a high proportion of mercury in these tissues is often present in a demethylated inorganic form typically associated with selenium and having a long biological half life. For this reason, toxicological assessments of wildlife should not be based on total mercury concentrations in liver, but should incorporate measurement of total mercury, methylmercury, and selenium, as well as analyses of total mercury in other tissues (such as muscle) where virtually all of the mercury is consistently present as methylmercury. *(96% support)*

(W4) Effects on Fish Health. Laboratory experiments have shown diminished reproduction and endocrine impairment in fish exposed to dietary methylmercury at environmentally relevant concentrations, with documented effects on sex hormone production, gonadal development, egg production, spawning behavior, and spawning success. Field surveys have found an inverse relationship between concentrations of sex hormones and methylmercury exposure. These results suggest that dietary methylmercury could adversely affect reproduction in wild populations of fish in surface waters containing food webs with high concentrations of methylmercury. *(93% support)*

(W5) Effects on Health of Wild Birds and Mammals. Field-based studies of wild piscivorous birds have corroborated results from controlled dietary dosing studies, demonstrating significant relationships between methylmercury exposure and various indicators of methylmercury toxicity, especially impaired reproduction, at environmentally realistic levels of dietary methylmercury intake. It is plausible that population level effects occur regionally, particularly in the most exposed cohorts of some piscivorous avian species. Similarly, for mammals, significant neurochemical effects have been documented in wild mink and in captive mink fed environmentally realistic levels of methylmercury. Higher methylmercury exposures in wild birds and mammals have caused overt neurotoxicity and death in several species. *(89% support)*

(W6) Risk Assessment. Reproduction is the demographic parameter most likely to be negatively affected by exposure to methylmercury in birds (and plausibly in fish and mammals as well). Population modeling of common loons indicates that reductions in mercury emissions could have substantial benefits for some regional common loon populations that are currently experiencing elevated methylmercury exposure. Predicted benefits would be mediated primarily through improved hatching success and development of hatchlings to maturity as mercury concentrations in prey fish decline. *(90% support)*

SOCIOECONOMIC CONSEQUENCES OF MERCURY USE AND POLLUTION

Panelists: Edward Swain (Chair), Paul Jakus, Frank Lupi, Peter Maxson, Jozef Pacyna, Alan Penn, Glenn Rice, Samuel Spiegel, Marcello Veiga

Question to the Panel: What are the socioeconomic and cultural costs of mercury pollution?

(S1) Socioeconomic Consequences. The anthropogenic mobilization of mercury from geologic materials into the biosphere has had adverse social and economic consequences. Mercury is released from geologic materials both intentionally for use in products and manufacturing, and unintentionally as an incidental emission from mineral processing and fossil-fuel combustion. The evaluation of policies for reducing mercury exposures requires a global perspective that examines the complete life cycle of mercury and accounts for social, cultural, and economic impacts. *(91% support)*

(S2) Products and Manufacturing Processes. Mercury is a global commodity with net flow from industrialized countries to developing countries, where its uses are generally less constrained. Policies in any industrialized countries that are intended to alter regional supplies or demand also affect global markets, and if not reasonably coordinated with other policies on a global scale, may have unintended effects. For example, any policies that have the indirect effect of putting downward pressure on global mercury prices could encourage greater use of mercury in small-scale gold mining, thereby increasing mercury vapor exposure of populations in developing countries. The consequences in this example could be addressed by coupling such policies with efforts to reduce mercury demand in developing countries, either through the adoption of mercury-free mining technologies or general economic development. *(92% support)*

(S3) Small-Scale Gold Mining. Persistent poverty and high gold prices have stimulated the proliferation of small-scale (artisanal) mining operations that use mercury to amalgamate gold. Miners (10 to 15 million people) and members of mining communities (up to 50 million people) often inhale air with elemental mercury concentrations exceeding 50 micrograms per cubic meter, 50 times the World Health Organization maximum public exposure guideline. Many miners and others—particularly amalgam burners, who are often women—exhibit tremors and other symptoms of elemental mercury poisoning. The mercury used in small-scale mining (~1000 tonnes per year, much supplied by developed countries) pollutes thousands of sites, poses long-term health risks to the inhabitants of mining regions, and contributes more than 10 percent of the modern anthropogenic loading of mercury to the Earth's atmosphere. *(99% support)*

(S4) Multiple Effects on Subsistence-Fishing Communities. The mercury contamination of fishery resources has had a number of adverse secondary effects on the health, societal, and economic well being in some subsistence-fishing communities, for whom the harvesting and consumption of fish is an integral component of culture and economy. These effects include conversion to less nutritious food and unhealthy diets, more sedentary lifestyles, loss of economic viability, and reduced social cohesion. *(85% support)*

(S5) General Costs of Mercury Use and Pollution. For industrialized regions, estimated health benefits of reduced methylmercury exposure, derived from benefit-cost analysis to monetize health benefits, have varied widely because of differences in the assumptions applied among studies. Depending on the health endpoints selected, the published studies have emphasized the many uncertainties in evaluating specific policies for mercury reduction, including corresponding responses in *i)* rates of mercury deposition; *ii)* methylmercury concentrations in fish; *iii)* methylmercury intake and exposure in humans; and *iv)* health effects, such as IQ reduction caused by fetal exposure. Because of our limited understanding of the impacts of mercury on ecosystems and wildlife, these impacts have not been included in the existing economic analyses, which can lead to an underestimation of the benefits of mercury reductions. *(84% support)*

(S6) Global Significance of Marine Fisheries. On a global scale, consumption of marine fish is the dominant pathway of human exposure to methylmercury. Relative to the situation for many freshwater fishes and ecosystems, there are comparatively few monitoring data for mercury in marine fishes and ecosystems. The biogeochemistry of mercury has also been more intensively studied in freshwater than in marine systems. Changes in the structure of marine ecosystems and in international trade of commercial fish, triggered by the

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depletion of many commercially important fish stocks, may influence future exposure of humans to methylmercury. *(91% support)*

(S7) Risk Communication. Effective risk communication is a significant challenge, particularly when attempting to communicate across cultural and linguistic boundaries. Confusion and misunderstanding can result when local languages and concepts are used to describe mercury in pertinent forms (e.g., methylmercury in fish and elemental mercury in air) and to explain the health risks of human exposure to these forms. Risk communication for mercury (and other contaminants) is further complicated by the importance of communicating the nutritional value of fish in the diet while providing reasonable advice to avoid levels of exposure that would place the individual at risk, with emphasis on protecting the fetus and children. *(98% support)*

RECOVERY OF MERCURY-CONTAMINATED FISHERIES

Panelists: John Munthe (Chair), Drew Bodaly, Brian Branfireun, Charles Driscoll, Cynthia Gilmour, Reed Harris, Milena Horvat, Marc Lucotte, Olaf Malm

Question to the Panel: How would methylmercury levels in fish respond to reduced anthropogenic emissions of mercury?

(F1) Response to Decreased Mercury Loadings. The concentration of methylmercury in fish from freshwater and coastal marine ecosystems will decrease in response to mercury-load reductions, although data are more definitive for aquatic systems affected by point sources than nonpoint sources. The magnitude, rate, and lag time of this decrease will vary significantly with the type of mercury contamination and with environmental factors affecting the net supply of methylmercury. *(91% support)*

(F2) Ecosystem Sensitivity to Mercury Load. Ecosystem sensitivity—the relative ability of an ecosystem to transform inorganic mercury load into methylmercury that accumulates in biota—is an important factor affecting rates of recovery. The most mercury-sensitive ecosystems have three characteristics in common: *i)* efficient delivery of mercury to zones of methylation; *ii)* high net rates of mercury methylation within these zones; and *iii)* efficient uptake and trophic transfer of methylmercury through the aquatic food web. *(96% support)*

(F3) Mercury Transport from Watersheds to Surface Waters. The recovery rate of a water body and its fishery resources to reduced atmospheric loadings of total mercury depends in part on the transport of mercury that has accumulated in the surrounding catchment. Increased transport of mercury from the catchment is associated with soil disturbance, erosion, strong hydrologic connectivity, shallow surficial deposits, high organic matter content in soil, and decomposition in soils and of plants. Available evidence indicates that human-associated disturbances and land-use change strongly influence the delivery of mercury from the catchment to receiving waters, which affects the timing and magnitude of fishery recovery. *(96% support)*

(F4) Entry and Transfer of Methylmercury in Food Webs. The efficiencies of biological uptake and trophic transfer of methylmercury through the food web are influenced mainly by site-specific physical, chemical, and biological controls. The dominant controls on uptake at the base of the aquatic food web include the physical proximity to sites of methylmercury production and the effects of partitioning and complexation on the bioavailability of methylmercury to lower trophic levels. Fish obtain most of their methylmercury via the diet. Factors affecting trophic transfer from the base of the food web to fish include the structure of the food web, the productivity of the ecosystem, and the growth efficiency of fish (fraction of energy intake devoted to growth). *(98% support)*

(F5) Experimental Evidence from Ecosystem Manipulations. Experimental increases in mercury loadings directly to aquatic ecosystems (within the range relevant to atmospheric deposition) have shown that methylmercury concentrations respond rapidly to increased loading, increasing in all levels of the food web, including fish. These data suggest that the response is initially proportional, but the long-term trend is not yet known. Experimental studies also indicate that terrestrial ecosystems strongly delay the delivery of mercury in atmospheric deposition to water bodies. If the ecosystem response to decreases in mercury loading mimics the experimental increase, the response in fish-mercury concentrations will be a function of catchment characteristics that control ecosystem sensitivity. *(93% support)*

(F6) Ecosystem Controls on Methylmercury Production. Net rates of mercury methylation can vary spatially among and temporally within aquatic systems, complicating the assessment of fish-mercury responses to changes in mercury loadings. High net methylation rates in aquatic ecosystems are mainly influenced by the areal extent and the connectivity of methylating and demethylating zones within the ecosystem, the bioavailability of mercury and methylmercury to methylating and demethylating bacteria, and the relative activity of those organisms. The dominant methylating zones are warm, shallow, organic-rich sediments in lakes and wetlands, anoxic waters, and soil drying and rewetting locations. Less is known about demethylation zones. The dominant controls on the bioavailability of Hg(II) are concentration and character of dissolved organic matter, sulfur cycling (sulfate reduction, sulfide production), bacterial community structure and activity, pH, and iron redox chemistry. Land-use changes affecting hydrology and soil structure may also create conditions that alter the production of methylmercury. *(94% support)*

APPLICATION OF SCIENCE TO POLICY

The expert panels have applied the best available science to the complex topics and policy-relevant questions addressed in their synthesis papers. This Declaration summarizes some of their principal findings, as reported therein and presented by the panels at the Eighth International Conference in Madison, Wisconsin. It is hoped that this Declaration will facilitate the application of the state-of-the-science to policy on environmental mercury pollution. Although direct policy recommendations are not presented in the Declaration, this summary and the detailed supporting analyses in the synthesis papers are directly pertinent to policy discussions concerning this geographically widespread and persistent environmental problem.

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Preface to Medical Geology Papers

The following articles in this issue of* Ambio *deal with the subject of Medical Geology. Specific topics are referred to by author's name within parentheses.

"Medical Geology" is the science dealing with the relationship between natural geological factors and health in man and animals, and understanding the influence of such relationships on the geographical distribution of health problems. Medical Geology is a broad and complicated subject, which requires interdisciplinary contributions if the problems are to be understood, mitigated and/or resolved (Selinus, Weinstein).

Our environment is the entire web of geological and biological interactions that characterize the relationship between life and planet Earth, and as such has an important bearing on health and well-being. Many of us take for granted that nature will provide a benign environment. It may even be that we do not think of our natural environment as important for our health. However, some naturally occurring elements are necessary for health and well-being while others may have detrimental effects. Geological processes along with human activities have redistributed metals from sites where they are fairly harmless to new sites where they have negative effects on humans and animals (Lindh, Munthe). Essential and toxic elements in bedrock or soils are ingested via food and water, and inhaled via the air we breathe, and may become the underlying cause of endemic diseases (Appleton).

It has been recognized for many years that large areas of our planet contain endemic areas that are related to trace element excess, deficiency, and/or chronic poisoning. Thus, the health of billions of people around the globe can be affected by their geological environment. Many of these health-related problems have been associated with geological sources, e.g. contaminated drinking water, coal mining, volcanic eruptions, dust, etc., but also anthropogenic sources. In recent years, considerable interest has arisen in the assessment of the health risks posed by ambient geology. For example, the occurrence of endemic goiter and cretinism are both associated with iodine deficiency (Fuge). Selenium deficiency is related to cases of muscular dystrophy as well as the induction of endemic cardiomyopathy (Changshen Li, Fordyce). Excess fluorine in drinking water has also been associated with endemic dental and skeletal fluorosis in several geographical areas, e.g. India and Sri Lanka. Well-documented cases of chronic arsenic poisoning caused by arsenic contaminated drinking water are known in southern Taiwan, Chile, Argentina, Mexico, and China, and recently in West Bengal and Bangladesh where 70 million people are exposed to this problem (Centeno, Chin-Hsiao Tseng). The ash from volcanic eruptions can travel many times around the world, and recent satellite images have shown how dust from Sahara, Gobi and the Australian deserts has been carried by winds halfway around the world. Exposure to these dusts can cause a wide range of respiratory problems (Derbyshire).

Other topics addressed in the papers in this Ambio issue include the environmental and health problems associated with mining processes of e.g. coal (Finkelman, Orem).

Understanding the potential of Medical Geology for dealing with a wide variety of environmental health problems is of critical importance in order to bring together geoscientists and medical/public health scientists to address the problems caused by or exacerbated by geological materials such as trace elements, rocks, minerals, and water and geological processes such as volcanic eruptions and earthquakes. Before resources are committed to protect or clean-up the environment from man-made contamination, it would seem prudent to determine how much of the contamination merely reflects the preexisting natural background levels or is related to anthropogenic sources.

Hopefully, the following papers will shed further light on the complex issues involved.

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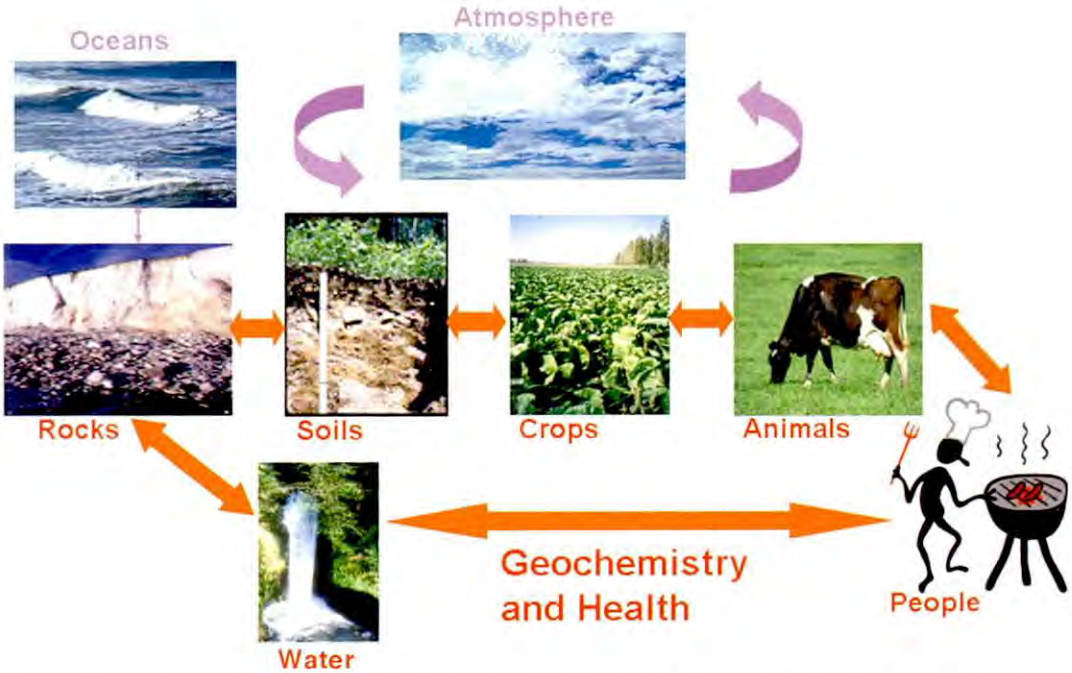

Figure 1. Simplified schematic diagram of the cycling of chemical elements from the environment to man. The main geochemistry and health pathways are shown in red. Reprinted from F. Fordce, Selenium Deficiency and Toxicity in the Environment. In: Essentials of Medical Geology: Impacts of the Natural Environment on Public Health, 2005, (ISBN 0126363412), Selinus et al., eds., with permission from Elsevier.

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