

THE MADISON DECLARATION ON MERCURY POLLUTION¹

WITH NONTECHNICAL SUMMARY OF PRINCIPAL FINDINGS²

This Declaration summarizes the scientific and technical conclusions presented by four expert panels³ 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 August 6-11, 2006. The 1,150 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 the sources of atmospheric mercury, methylmercury exposure and its effects on humans and wildlife, the socioeconomic consequences of mercury pollution, and recovery of mercury contaminated fisheries.

DECLARATION	SUMMARY
<p>Source Attribution of Atmospheric Mercury Deposition</p> <p>Question to the Panel: <i>“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?”</i></p>	
<p>(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.</p>	<p>For a given location, it is possible to distinguish between local and global sources of atmospheric mercury with confidence.</p> <p>There is less certainty in assessing regional sources of atmospheric mercury.</p>
<p>(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.</p>	<p>Downwind of major industrial source areas of reactive gaseous mercury, mercury deposition tends to be dominated by anthropogenic emissions rather than natural emissions.</p>

¹ Copyright 2007, *Ambio: A Journal of the Human Environment*, Royal Swedish Academy of Sciences.

² Summaries prepared by the University of Wisconsin Sea Grant Institute in consultation with the panel chairs. The Sea Grant Institute was a cosponsor of the 8th International Conference on Mercury as a Global Pollutant.

³ A list of the names, affiliations and email addresses of panel members is appended.

THE MADISON DECLARATION ON MERCURY POLLUTION

DECLARATION	SUMMARY
<p>(A3) Long-Term Trends and Source-Receptor Relations. Since the Industrial Revolution, mercury deposition has increased globally by a factor of 2 to 4, 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 and/or contemporaneous increase in emissions from other geographic areas.</p>	<p>On average, about three times more mercury falls from the sky now than before the Industrial Revolution 200 years ago. For the last 30 years, however, emissions from developing countries have increased, offsetting decreased emissions from developed countries.</p>
<p>(A4) Atmospheric Response Times. A decade ago, the half-life of mercury in the global atmosphere was estimated to be about one 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 Hg⁰ and emitted back to the atmosphere, and a potential greater deposition rate of elemental mercury.</p>	<p>The amount of mercury in the atmosphere is apparently not changing.</p> <p>Thus, new findings of a shorter atmospheric half-life suggests greater movement of mercury to and from Earth's surface.</p>
<p>(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.</p>	<p>Over the last 10 years, scientists have learned that much more mercury falls from the sky as "dry deposition" than in rainfall or other "wet deposition."</p> <p>This supports the findings of a shorter half-life and faster mercury cycling. (see A4)</p>
<p>(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 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.</p>	<p>Computer models provide the best means of assessing the relative importance of different mercury pollution sources.</p>

THE MADISON DECLARATION ON MERCURY POLLUTION

DECLARATION	SUMMARY
<p>(A7) Other Important Non-Mercury Factors. It is possible, and perhaps likely, that the global mercury cycle has been altered substantially in recent decades by “non-mercury” 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.</p>	<p>Our understanding of the global mercury cycle is being confounded by climate change, increasing ozone levels and other non-mercury factors that may affect how long mercury stays in the atmosphere and how and where it falls.</p>
<p>Health Risks and Toxic Effects of Methylmercury</p> <p>Question to the Panel: “<i>What is the evidence that humans, fish, wildlife, and other biota are being adversely affected by exposure to methylmercury?</i>”</p>	
<p>Part 1: Human Health</p>	
<p>(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.</p>	<p>Elevated mercury exposure due to frequent consumption of fish with high levels of mercury has been observed in people throughout the world and at all levels of society.</p>
<p>(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.</p>	<p>At present levels of exposure, methylmercury constitutes a public health problem in many parts of the world.</p>
<p>(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. <i>Total fish consumption</i>—without differentiating the fish species consumed—is not necessarily a dependable metric for estimating methylmercury exposure.</p>	<p>Mercury levels in hair and blood (including umbilical cord blood) are valid measures of exposure and reflect mercury intake from consumption of fish.</p>

THE MADISON DECLARATION ON MERCURY POLLUTION

DECLARATION	SUMMARY
<p>(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.</p>	<p>There is enough evidence showing that exposure of the fetus to methylmercury will affect children's development to justify warning children and women of child-bearing age to be careful about the species of fish they eat.</p>
<p>(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.</p>	<p>New evidence indicates methylmercury exposure may increase the risk of cardiovascular disease in humans, particularly in adult men.</p>
<p>(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 seek to maximize the intake of beneficial fatty acids while limiting exposure to methylmercury.</p>	<p>To increase the benefits and reduce the risks, consumers should choose fish with high levels of omega-3 fatty acids and low levels of methylmercury.</p>
<p>(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 is no human data demonstrating a protective role for selenium against the neurotoxicity of mercury, including developmental neurotoxicity.</p>	<p>To date, there is no evidence from human studies that selenium protects people from the toxic effects of mercury.</p>
<p>Part 2: Wildlife Health</p>	
<p>(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. Non-piscivorous 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.</p>	<p>In wildlife, methylmercury exposure poses the greatest health risk to predator fish and fish-eating birds and mammals, such as bald eagles, loons, otters, polar bears and seals.</p>

THE MADISON DECLARATION ON MERCURY POLLUTION

DECLARATION	SUMMARY
<p>(W2) Trends in Mercury Exposure in Wildlife. While available evidence indicates that reductions in mercury emissions have led to reduced concentrations of mercury in fish and piscivorous wildlife in areas impacted by local or regional point-source industrial releases, in some areas remote from industrial sources (for example, the Arctic), current temporal trends indicate increasing concentrations of mercury in some piscivorous wildlife.</p>	<p>Reductions in local and regional point-source mercury emissions have lowered mercury levels in the fish and wildlife affected by them. However, increasing mercury concentrations are now being found in a number of fish-eating wildlife in remote areas.</p>
<p>(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 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.</p>	<p>The concentrations of mercury found in fur, feathers, eggs, skeletal muscle and blood are valid measures of methylmercury exposure in wildlife.</p> <p>Toxicological assessments of wildlife should not be based on total mercury concentrations in the liver.</p>
<p>(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 production of sex hormones, 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.</p>	<p>Recent studies indicate that dietary methylmercury exposure may be impairing the reproduction of fish in aquatic food webs with high concentrations of methylmercury.</p>
<p>(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 individuals of several species.</p>	<p>Methylmercury levels in fish-eating birds and mammals in some parts of the world are reaching toxic levels, which is impairing the reproduction of fish-eating birds with high levels of methylmercury.</p>

THE MADISON DECLARATION ON MERCURY POLLUTION

DECLARATION	SUMMARY
<p>(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.</p>	<p>Methylmercury exposure may lead to population declines in birds and possibly in fish and mammals as well.</p>
<p>Socioeconomic Consequences of Mercury Use and Pollution</p> <p>Question to the Panel: “<i>What are the socioeconomic and cultural costs of mercury pollution?</i>”</p>	
<p>(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.</p>	<p>Mercury use and pollution from human activities has had, and continues to have, documented adverse social and economic consequences.</p>
<p>(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.</p>	<p>To be effective, policies intended to reduce mercury use in industrialized nations must be coupled with efforts to reduce mercury demand in developing nations.</p>
<p>(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 million 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 (~1,000 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.</p>	<p>The use of mercury in small-scale gold mining is polluting thousands of sites around the world, posing long-term health risks to as many as 50 million inhabitants of mining regions and contributing more than 10 percent of the mercury in Earth’s atmosphere resulting from human activities.</p>

THE MADISON DECLARATION ON MERCURY POLLUTION

DECLARATION	SUMMARY
<p>(S4) Multiple Effects on Subsistence-Fishing Communities. The mercury contamination of fishery resources has had a number of adverse secondary effects on the health and 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.</p>	<p>Mercury pollution is adversely affecting the health, culture and economies of some subsistence fishing communities.</p>
<p>(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 (1) rates of mercury deposition, (2) methylmercury concentrations in fish, (3) methylmercury intake and exposure in humans, and (4) 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.</p>	<p>The true total costs of mercury pollution are probably much greater than currently estimated due to the many uncertainties in these estimates, and because they don't take into account mercury's impacts on ecosystems and wildlife.</p>
<p>(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 depletion of many commercially important fish stocks, may influence future exposure of humans to methylmercury.</p>	<p>Little is known about methylmercury contamination levels in marine fishes, the ingestion of which is the main way most people are exposed to methylmercury.</p>
<p>(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.</p>	<p>It is difficult to provide advice on how to minimize mercury exposure from eating fish while noting the positive nutritional value of fish. This is particularly true when crossing cultural and linguistic boundaries.</p>

THE MADISON DECLARATION ON MERCURY POLLUTION

DECLARATION	SUMMARY
<p>Recovery of Mercury-Contaminated Fisheries</p> <p>Question to the Panel: <i>“How would methylmercury levels in fish respond to reduced anthropogenic emissions of mercury?”</i></p>	
<p>(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.</p>	<p>The concentration of methylmercury in fish in freshwater and coastal ecosystems can be expected to decline with reduced mercury inputs.</p> <p>The rate of decline could vary considerably among water bodies, depending on the characteristics of the particular ecosystem.</p>
<p>(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: (1) efficient delivery of mercury to zones of methylation, (2) high net rates of mercury methylation within these zones, and (3) efficient uptake and trophic transfer of methylmercury through the aquatic food web.</p>	<p>The various factors that make ecosystems sensitive to mercury pollution have been identified.</p>
<p>(F3) Mercury Transport from Watersheds to Surface Waters. The rate of recovery 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 disturbance of soil, 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.</p>	<p>How quickly a body of water and its fishery respond to a reduction in mercury pollution depends largely on the amount of mercury accumulated in its watershed, human land use in the watershed, and the biochemical and physical characteristics of the watershed.</p>

THE MADISON DECLARATION ON MERCURY POLLUTION

DECLARATION	SUMMARY
<p>(F4) <i>Entry and Transfer of Methylmercury in Food Webs.</i> 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).</p>	<p>The amount of methylmercury that enters the aquatic food web partly depends on how close organisms at the base of the food web are to places where methylmercury is created.</p> <p>The concentration of methylmercury accumulated in fish is strongly influenced by the fish's position in the food web structure.</p>
<p>(F5) <i>Experimental Evidence from Ecosystem Manipulations.</i> 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.</p>	<p>Increases in direct mercury inputs cause methylmercury concentrations at all levels of an aquatic food web to increase rapidly.</p> <p>However, the rate of response to reduced atmospheric inputs of mercury will depend partly on the characteristics of the surrounding land that drains into a water body.</p>
<p>(F6) <i>Ecosystem Controls on Methylmercury Production.</i> 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 locations of soil drying and rewetting. Less is known about zones of demethylation. 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.</p>	<p>Ecosystems most likely to generate methylmercury have warm, shallow and organic-rich sediments, oxygen-poor and acidified (low pH) water, lots of dissolved organic matter, high bacterial activity, and certain other characteristics.</p>

THE MADISON DECLARATION ON MERCURY POLLUTION

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.

THE MADISON DECLARATION ON MERCURY POLLUTION

Panel on Source Attribution of Atmospheric Mercury

Steve Lindberg (*chair*), Emeritus Fellow, Oak Ridge National Laboratory (currently, Graeagle, California, United States of America). Telephone (530) 927-7627, email lindberg@Now2000.com

Russell Bullock, NOAA Air Resources Laboratory (in partnership with the U.S. Environmental Protection Agency), Research Triangle Park, North Carolina, United States of America.
bullock.russell@epa.gov

Ralf Ebinghaus, GKSS Research Centre, Geestacht, Germany. ralf.ebinghaus@gkss.de

Daniel Engstrom, Science Museum of Minnesota, Marine on St. Croix, Minnesota, United States of America. dre@smm.org

Xinbin Feng, State Key Laboratory of Environmental Geochemistry, Guizhou, China.
xinbin.feng@mail.gyig.ac.cn

William Fitzgerald, University of Connecticut, Groton, Connecticut, United States of America.
wfitzger@uconnvm.uconn.edu

Nicola Pirrone, CNR-Institute of Atmospheric Pollution, Rende, Italy. n.pirrone@cs.ia.cnr.it

Eric Prestbo, Frontier Geosciences, Seattle, Washington, United States of America.
ericp@frontiergeosciences.com

Christian Seigneur, Atmospheric and Environmental Research, San Ramon, California, United States of America. cseigneur@aer.com

Panel on Health Risks and Toxicological Effects of Methylmercury

Human Health

Donna Mergler (*chair*), CINBIOSE, World Health Organization-Pan-American Health Organisation Collaborating Centre for the Prevention of Occupation and Environment-Related Illness, University of Québec at Montreal, Montreal, Québec, Canada. Telephone (514) 987-3000 #3355, email mergler.donna@uqam.ca

Henry Anderson, Division of Public Health, Wisconsin Department of Health and Family Services, Madison, Wisconsin, United States of America. anderha@dhfs.state.wi.us

Laurie Chan, University of Northern British Columbia, Prince George, British Columbia, Canada.
lchan@unbc.ca

Kathryn Mahaffey, Washington, District of Columbia, United States of America.

Mineshi Sakamoto, National Institute for Minamata Disease, Minamata City, Kumamoto, Japan.
sakamoto@nimd.go.jp

Alan Stern, Division of Science, Research and Technology, New Jersey Department of Environmental Protection, Trenton, and School of Public Health, University of Medicine and Dentistry of New Jersey, Piscataway, New Jersey, United States of America. alan.stern@dep.state.nj.us

Wildlife Health

Anton Scheuhammer (*chair*), Environment Canada National Wildlife Research Centre, Carleton University, Ottawa, Ontario, Canada. Telephone (613) 998-6695, email tony.scheuhammer@ec.gc.ca

Michael Meyer, Wildlife Biosentinel Program, Wisconsin Department of Natural Resources, Rhinelander, Wisconsin, United States of America. michael.meyer@dnr.state.wi.us

THE MADISON DECLARATION ON MERCURY POLLUTION

Michael Murray, National Wildlife Federation, Great Lakes Natural Resource Center, Ann Arbor, Michigan, United States of America. murray@nwf.org

Mark Sandheinrich, River Studies Center, Department of Biology, University of Wisconsin-La Crosse, La Crosse, Wisconsin, United States of America. sandhein.mark@uwlax.edu

Panel on Socioeconomic Consequences of Mercury Use and Pollution

Edward B. Swain (*chair*), Minnesota Pollution Control Agency, Saint Paul, Minnesota, United States of America. Telephone (651) 296-7800, email edward.swain@state.mn.us

Paul Jakus, Department of Economics, Utah State University, Logan, Utah, United States of America. paul.jakus@usu.edu

Frank Lupi, Department of Agricultural Economics and Fisheries & Wildlife Department, Michigan State University, East Lansing, Michigan, United States of America. lupi@msu.edu

Peter Maxson, Concorde East/West Sprl, Brussels, Belgium. concorde.ew@tele2allin.be

Jozef Pacyna, Center for Ecological Economics, Norwegian Institute for Air Research, Kjeller, Norway, and Chemistry Faculty, Gdansk University of Technology, Gdansk, Poland. jp@nilu.no

Alan Penn, Cree Regional Authority, Montreal, Québec, Canada. apenn@gcc.ca

Glenn Rice, Fort Mitchell, Kentucky, United States of America. grice@hsph.harvard.edu

Samuel Spiegel, Interdisciplinary Studies, University of British Columbia, Vancouver, British Columbia, Canada. samspiegel@gmail.com

Marcello Veiga, Department of Mining Engineering, University of British Columbia, Vancouver, British Columbia, Canada. veiga@mining.ubc.ca

Panel on Recovery of Mercury-Contaminated Fisheries

John Munthe (*chair*), IVL Swedish Environmental Research Institute, Gothenburg, Sweden. Telephone +46-31-7256200, email john.munthe@ivl.se

R.A. “Drew” Bodaly, Penobscot River Mercury Study (Maine), Salt Spring Island, British Columbia, Canada. drewbodaly@yahoo.ca

Brian A. Branfireun, Department of Geography, University of Toronto at Mississauga, Mississauga, Ontario, Canada. brian.branfireun@utoronto.ca

Charles T. Driscoll, Department of Civil and Environmental Engineering, Syracuse University, Syracuse, New York, United States of America. ctdrisco@mailbox.syr.edu

Cynthia C. Gilmour, Smithsonian Environmental Research Center, Edgewater, Maryland, United States of America. gilmour@si.edu

Reed Harris, Tetra Tech, Inc., Oakville, Ontario, Canada. rharris6@cogeco.ca

Milena Horvat, Department of Environmental Sciences, Josef Stefan Institute, Ljubljana, Slovenia. milena.horvat@ijs.si

Marc Lucotte, GEOTOP, Université du Québec à Montréal, Montreal, Québec, Canada. lucotte.marc_michel@uqam.ca

Olaf Malm, Lab. Radioisotopos EPF, Inst. Biofisica CCF, Universidade Federal do Rio de Janeiro, Rio de Janeiro, Brazil. olaf@biof.ufrj.br, olaf@ibest.com.br