Risk and Regulation: Methylmercury Exposure and Fish Consumption

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Exposure to organic mercury (methylmercury) occurs almost universally due to ingestion via contaminated fish and shellfish tissue. Sources of elemental mercury include air release by domestic industrial combustion, mining, and international mercury emissions transported via a global cycle. Deposition of inorganic mercury from air to surface waters results in methylation to organic methylmercury and bioaccumulation in the aquatic food web.

Health effects from methylmercury exposure consist mainly of neurological and neurodevelopmental effects, with fetuses particularly sensitive. Thus regulation of methylmercury exposure has concentrated on determining acceptable exposure levels and reference doses aimed toward protecting developing fetuses.

The risk of methylmercury exposure in humans is regulated largely by the federal government, especially by the Environmental Protection Agency (EPA) and the Food and Drug Administration (FDA). The EPA imposes limits on mercury emissions and seeks to research methylmercury levels in fish and humans. The EPA sets a reference dose for methylmercury exposure. The FDA uses data on methylmercury levels in fish to advise consumers on how to make informed decisions regarding fish consumption.

There are numerous shortcomings to government regulation of this issue, which has thus far been inadequately addressed. Further scientific research, improved implementation of available data and scientific conclusions, and improved public communication of risk would all lead to more effective treatment of the risk of methylmercury exposure via ingestion of fish and shellfish. This could include more effective monitoring systems of human and fish methylmercury levels, research into the process of bioaccumulation, and implementation of stricter fish labeling standards, as well as research into higher-risk subpopulations allowing for targeted standards and recommendations.
that roughly two thirds of United States emissions enter the
global cycle (i.e., are not deposited in the United States)\(^7\).

While the majority of mercury present in surface
waters of the United States derived from anthropogenic ac-
tivity is the result of air deposition (fallout of atmospheric
mercury), mercury is also released directly by municipal
wastewater treatment plants and other industrial facilities
into the water. However, the EPA describes direct release
of mercury to surface waters as “significantly smaller” than
deposition to surface waters of atmospheric mercury\(^8\). In
the western United States, historical mining methods have
left a legacy of surface water contamination, and contem-
porary mining practices pertaining to gold, silver, and
zinc extraction continue to release mercury to land, which
may eventually reach surface waters\(^9\). Indeed, the EPA
states that only three mining facilities were responsible for
more than 74 percent of all releases of mercury to land in
2004\(^10\).

In order to understand the origins of human expo-
sure to mercury via ingestion of methylmercury, it is impor-
tant to first understand the sources of mercury, and then to
comprehend the process of mercury methylation and bioac-
cumulation. Mercury deposited in water bodies is eventu-
ally incorporated into sediment, and is there methylated by
bacterial processes, creating methylmercury, an inorganic
compound. Absorbed by phytoplankton and zooplankton,
methylmercury is ingested by small fish, which are then
consumed by successively larger fish. Each level of the
food web further concentrates the levels of methylmercury
present; thus, large predatory fish may have mercury con-
centrations in their tissues that are many thousands of times
higher than the level of inorganic mercury in the water in
which they live\(^11\).

Although the general process is understood, rates of
diffusion or transport of methylmercury from sediment
sources to the water column are understudied\(^12\). In gen-
eral, methylmercury concentrations in fish vary in accord-
ance with factors such as inorganic mercury concentration
in the water, water pH and temperature, concentrations of
other chemicals such as sulfides, concentration of organic
matter in the water, and presence and abundance of various
benthic microorganisms. Thus, due to variation in local
environments, regional and local patterns of mercury de-
position and concentration, and the complexity of various
food webs, there is inherent wide variation in mercury con-
centrations among different fish species, and from place to
place. Mercury concentrations in fish tissue are
typically measured in parts per million (ppm).

**Risk Assessment**

In assessing the risk posed by ingestion of methylmercury
contained in fish and shellfish, we must consider which
individuals, groups, or populations are or may be at risk
for adverse effects. Furthermore, the actualities of health
effects of mercury exposure and scientifically determined
dose-response information are essential in order to deter-
mine what levels, if any, of mercury exposure may be con-
sidered “safe” or non-harmful, and what levels of mercury
exposure may be considered harmful, and with what spe-
cific effects. Exposure as recorded by various measures of
mercury concentration in the human body must be recon-
ciled with varying mercury concentrations in various fish
species in order to assess risk.

Some adverse human health effects stemming from
methylmercury exposure are fairly well-established, while
others require further study and are largely unconfirmed.
Methylmercury exposure in humans is typically determined
by measuring the concentration of mercury in the blood or
hair. A study of a representative sample of adult women
in the United States determined that if total blood mer-
curry concentration is greater than 4 micrograms per liter
(μg/L), more than 90 percent of mercury present is organic
(methylmercury)\(^13\), and thus points to mercury exposure
from consumption of seafood. Mercury present in hair has
been found to be 80 to 98 percent methylmercury\(^14\), but
hair samples are vulnerable to external contamination by
 elemental or inorganic mercury\(^15\).

Methylmercury is well-established as a confirmed
neurotoxin at high doses, and has well-documented neuro-
logical effects at lower doses. The health effects of expo-
sure to methylmercury can be divided into neurological
and non-neurological effects, with most neurological effects
well documented, and with some purported non-neurologi-
cal effects requiring further study.

At extremely high doses, methylmercury produc-
es the dramatic symptoms of “mercury poisoning” in all
exposed, involving severe damage to the nervous system
and, with high enough doses or prolonged exposure, death.
Widely documented and studied cases of mercury poison-
ing due to methylmercury exposure include epidemics in
Japan from consumption of fish and in Iraq from consump-
tion of grain contaminated by a methylmercury fungicide.
Though the levels of methylmercury exposure associat-
ed with these events are much higher than those typically
found in the United States, in the words of Mahaffey, they
present the “strongest possible evidence linking exposure

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to methylmercury with human fatalities and neurological disease.\textsuperscript{16} Methylmercury presents a potential for neurological damage in adults, children, and developing fetuses\textsuperscript{17}.

Methylmercury is considered to have a sharp dose-response curve\textsuperscript{18} such that increases in exposure above a certain threshold dose result in rapid increases in damaging effects. In adults, the neurological symptom considered to be the most sensitive indicator of methylmercury exposure is occurrence of paresthesia (tingling of the skin)\textsuperscript{19}. Though a 5% incidence of paresthesia in adults was thought to result from blood mercury concentrations of 200 μg/L and greater and hair mercury concentrations of more than 50 parts per million (ppm), a more recent study showed that parasthesia occurred in adults with blood mercury concentrations of 34 to 97 μg/L\textsuperscript{20}. Another study showed association of decreased fine motor speed with hair mercury concentrations of 0.56 to 13.6 ppm\textsuperscript{21}. Thus these new studies suggest that the threshold dose for effects of methylmercury exposure in adults may be much lower than was previously thought.

Methylmercury exposure also has neurological effects on developing fetuses and small children. Furthermore, it is recognized that the fetus is more sensitive to mercury exposure than an adult: mothers with rather minimal methylmercury exposure and symptoms have given birth to infants with severe neurological damage\textsuperscript{22}. For this reason, establishment of safe dosage levels in recent years has been geared especially toward protecting the fetus. The EPA lists adverse effects to cognitive thinking, memory, attention, language, and fine motor and visual spatial skills in children with prenatal methylmercury exposure\textsuperscript{23}. Maternal hair mercury concentrations of 5 ppm are associated with less obvious changes in development, while concentrations of 10 to 20 ppm are associated with delayed walking and other effects\textsuperscript{24}.

While the reference dose adopted by the EPA based on recommendations from the National Research Council of the National Academy of Sciences was 5.8 μg/L concentration of mercury in mothers, corresponding to an equal concentration of mercury in umbilical cord blood, newer studies have shown that umbilical cord blood is typically higher in mercury concentration than maternal blood; thus maternal blood mercury concentration of more than 3.5 μg/L may be a more accurate estimate of an exposure level associated with elevated risk to fetal nervous systems\textsuperscript{25}. The percentage of women with blood mercury concentrations higher than the first reference dose of 5.8 μg/L, if applied to the number of total births in the United States, suggests that over 300,000 infants each year are born with prenatal exposure to mercury levels associated with neurodevelopmental damage; if the percentage of women with blood mercury levels higher than the updated reference dose of 3.5 μg/L is used, the result is more than 600,000 newborns per year.

Methylmercury exposure may also pose non-neurological risks for adults. Based on some studies, methylmercury exposure is associated with increased risk for some cardiac problems such as myocardial infarction and carotid atherosclerosis, even after controlling for the cardiac benefits of fatty acids contained in fish\textsuperscript{26}. Studies in rats have reported causation of kidney tumors by methylmercury exposure, but only at high doses, and the EPA concludes that methylmercury is not likely to cause cancer in humans\textsuperscript{27}.

We have begun to see that certain subpopulations are particularly at risk for methylmercury exposure, including pregnant women (as a function of the sensitivity of the fetus), young children, and nursing mothers. However, other subpopulations are also at high risk. As previously stated, methylmercury exposure is a product of fish and shellfish consumption; thus, subpopulations with high fish consumption are more at risk for higher levels of methylmercury exposure than the general public, or the national average.

Residents of coastal and island areas have been found to have higher than average consumption of seafood. Average fish and shellfish consumption for Floridians was equivalent to the 90th percentile for consumption of seafood by women as found by a national study; average consumption of fish and shellfish for Puerto Rico was greater than the 90th percentile for consumption of seafood in the United States overall\textsuperscript{28}. Results of blood mercury screening by the Louisiana State Office of Public Health showed that individuals who reported eating fish at least once a week had blood mercury levels twice as high as individuals who ate fish only twice a month or less often\textsuperscript{29}.

The EPA estimates that, on average, about 60 percent of people in the United States eat fish at least once a week\textsuperscript{30}. However, certain other subgroups eat seafood substantially more frequently than the general population. Minority groups tend to eat fish more often, and in larger amounts; Native Americans as well as individuals of Asian or Caribbean ancestry tend to eat seafood more frequently than other subpopulations\textsuperscript{31}. Individuals who fish recreationally or for subsistence, including members of certain Native American tribes, also consume fish at higher levels than the national average\textsuperscript{32}. 

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The most potent health risks (neurological damage and neurodevelopmental effects) posed by methylmercury ingestion via fish and shellfish consumption are well-documented and scientifically accepted. Mercury originating largely from industrial sources is deposited in surface waters, transformed into organic mercury by the process of methylation by microorganisms, and is then concentrated in the tissues of fish and shellfish via bioaccumulation. Methylmercury is bound to amino acids in fish flesh, and cannot be eliminated by removing visible fat or by other preparation or cooking methods. Although methylmercury concentration in fish and shellfish ranges from less than 0.1 ppm to more than 1 ppm, and average values for each species allow some discretion in consuming low-methylmercury species, there is considerable variation within species, and at any rate, any consumption of fish leads to some methylmercury exposure. Level of methylmercury exposure is a consequence of both the amount and variety of fish consumed, and is essentially unavoidable with fish consumption.

**Regulation**

Since methylmercury exposure is an automatic consequence of fish and shellfish consumption, and since the risks of methylmercury exposure are well known and scientifically quantified, methylmercury exposure and hence methylmercury contamination of fish and shellfish are essentially public health risks. Furthermore, methylmercury contamination of surface waters and, consequently, food webs, is known to be the direct result of domestic and international industrial mercury emissions, with some more minor contributions from other sources. With this in mind, the issue of contamination of fish and shellfish with methylmercury (the source of virtually all methylmercury exposure), and the attendant issue of mercury emissions, especially atmospheric mercury emissions, merit serious government attention and regulation. The fact that a lower-end estimate of 300,000 newborns, out of an annual total of four million births, were deemed to have been exposed before birth to levels of methylmercury associated with elevated risk of neurodevelopmental problems, and the associated sharp dose-response curve for adverse effects of methylmercury exposure, should be sufficient to recruit government attention. Additionally, the government (state or federal) is likely best equipped to compile and communicate information on contamination levels of various fish and shellfish species, and the federal government is best-poised to impose regulation on mercury emissions across a broad spectrum of industries.

Currently, the federal government addresses the issues of mercury pollution and methylmercury exposure in several ways. The main agencies imposing regulations and communicating risk to the public are the EPA and the Food and Drug Administration (FDA). These efforts take the form of regulation of sources of mercury emissions, national testing of methylmercury levels, determination of safe or recommended dosage levels, communicating the risks of methylmercury exposure to the public, collecting data on methylmercury concentrations in fish, and providing advice to the public about safe fish consumption.

The EPA’s “Roadmap for Mercury” asserts that the agency has focused mercury reduction efforts over the last fifteen years on large point sources of emissions to the atmosphere, and as of 2005 had enacted standards for “most major known industrial sources in the U.S.” The EPA’s stated goal for the next decade is to focus on smaller industrial sources accounting for at least 20 percent of national air releases, reducing mining releases in the West, and working to address international emissions. Data show that implementation of regulations accounted for an overall 45 percent reduction in national air emissions of mercury, including over 90 percent reduction of emissions from medical and municipal waste incineration. Additionally, the Clean Air Mercury Rule of 2004 regulates coal-fired power plant mercury emissions, putting in place a nationwide cap of 38 tons per year by 2010, and 15 tons per year by 2018, which is purported to reduce mercury emissions from coal-fired power plants by 70 percent from 1999 levels. The EPA states that the concentration of methylmercury in the blood is “the best available indicator of human exposure to methylmercury through fish consumption.” The EPA also acknowledges that monitoring of methylmercury levels in human tissue is necessary in order to deduce geographic and temporal trends in United States mercury exposure, and in order to assess the effectiveness of various programs intended to reduce associated risks, addressing both mercury releases and human exposure. However, the only national source of data on methylmercury concentrations in humans is the National Health and Nutrition Examination Survey (NHANES), a survey carried out by the United States Centers for Disease Control and Prevention (CDC). The EPA states that investigating and tracking methylmercury blood concentrations in women of childbearing age is important, as it denotes both exposure of adult women and potential exposure of fetuses.
The NHANES study is conducted using roughly 8,000 individuals in twenty-five to thirty communities calculated to be representative of the United States, on women between 16 and 49 years of age, and on young children aged one to six years. Mercury measurements for adult men, children over age six, and women over age 49 were added beginning in 2003. From the 1999 and 2000 survey years, of women aged 16 to 49 years, 15.7 percent were found to have total blood mercury concentrations at or above 3.5 μg/L, and 7.8 percent were found to have blood mercury concentrations at or above 5.8 μg/L.

The Clean Water Act has established that each state and tribal authority must define acceptable levels of pollutants for each water body in its jurisdiction. The EPA published a Reference Dose in 2001 for methylmercury. A reference dose estimates a level of daily exposure unlikely to cause adverse effects over a lifetime, and takes into account higher-risk subpopulations. The EPA Reference Dose for methylmercury is currently 0.1 μg/kg body weight/day. Utilizing this reference dose, the EPA has also issued a “national methylmercury ambient water quality criterion,” set at 0.3 ppm methylmercury in fish tissues, based on estimated rates of consumption of fish. While an older reference dose of 0.3 μg/kg body weight/day was developed using occurrence of paresthesia in adults during the methylmercury poisoning epidemic in Iraq, the new reference dose concentrated on fish as the source of methylmercury and took into account what proportion of ingested methylmercury is absorbed into the human body, as well as the half-life of mercury in human tissues.

Mahaffey makes several important points about the reference dose as a mechanism for limiting daily exposure to “safe” levels. To paraphrase, the reference dose is not a strict boundary above which lies immediate danger and below which lies safety, but there is concern that adverse health effects can occur with multiple exposures. Because methylmercury has a relatively long half-life in the human body (up to 70 days), daily intake of methylmercury adds to bioaccumulation of mercury, increasing the concentration of methylmercury in the body. What is more, although the reference dose considers a lifetime of exposure to methylmercury, short-lived peak exposures can have adverse impacts on fetal development if they occur during critical periods of neurological development. There is debate over the proper period of exposure to be used in order to assess developmental neurotoxins. Additionally, although the reference dose for methylmercury is formulated to prevent adverse effects on the developing fetus, it is unclear whether young children are more similar to adults or fetuses in their susceptibility to adverse effects from exposure; studies suggest that young children are exposed to methylmercury levels two or three times typical adult exposures on the basis of μg/kg body weight.

An important part of current and future risk management carried out by federal and state governments is monitoring of methylmercury concentrations in fish tissue. In this way, trends in methylmercury exposure can be tracked. As the current strategy for reducing risk of adverse effects of methylmercury exposure is to inform the public about the associated risks and how to make intelligent choices about fish and shellfish consumption, it is essential that this data is communicated to the public in a relevant and useful manner. The FDA maintains databases of average tissue methylmercury concentrations by species, although some data is rather old.

The FDA and EPA also published a joint consumer advisory, “What You need to Know About Mercury in Fish and Shellfish” in 2004. The advisory is the main source of practical recommendations in the FDA’s online resources on methylmercury in fish. This consumer advisory makes three central recommendations for “women who might become pregnant, women who are pregnant, nursing mothers, [and] young children”: refrain from eating shark, swordfish, king mackerel, or tilefish as they contain high mercury levels; eat up to twelve ounces (two meals) of low-mercury fish and shellfish per week, and check local fish advisories to assess the safety of fish caught in local water bodies, but if no information is available, eat up to six ounces a week of locally-caught fish, eating no other fish that week. The advisory lists five common fish low in mercury: shrimp, canned light tuna, salmon, pollock, and catfish; the advisory also states that while albacore tuna contains more mercury than canned light tuna, up to six ounces may be eaten per week. This advisory also points out the health benefits of fish and shellfish consumption, including protein, low saturated fat, and omega-3 fatty acids; it states that a diet including a variety of seafood contributes to “heart health and children’s proper growth and development...women and young children in particular should include fish or shellfish in their diets.” The EPA additionally provides a National Listing of Fish Advisories database, listing information from all fish advisories as provided by the states, tribes, and Canada.

Room for Improvement

Several potential problems may be found with the FDA-
EPA approach to communicating to the public the risks of ingesting methylmercury when consuming fish and shellfish. Firstly, the EPA’s “Roadmap for Mercury” states that the 2004 joint consumer advisory addresses “the risks of consumption to certain sub-populations (e.g., groups with routinely high consumption),”\textsuperscript{50} but in actuality the document makes no mention whatsoever of groups with higher risk due to higher levels of fish and shellfish consumption; rather, the entire advisory is addressed only to women who are or may become pregnant, mothers who are nursing, and young children. Although the document does acknowledge that risk from methylmercury in fish and shellfish is dependent upon the quantity and frequency of fish and shellfish consumption, specific treatment of higher-risk subpopulations would provide additional valuable information to the public.

In addition, some issue may be taken with the wording of the advisories. While the document makes clear the risk that elevated levels of methylmercury may damage a fetus or young child’s developing nervous system, it mildly states that “as a matter of prudence, women might wish to modify”\textsuperscript{51} the amount and varieties of fish that they consume if they are with child, planning to become pregnant, nursing, or feeding a young child. While the document gives specific recommendations on amount and types of fish to be safely eaten by an adult woman in the first three categories, the only advice for feeding fish to a young child is to “serve smaller portions”\textsuperscript{52}.

There is some question as to the accuracy of the FDA’s database of average methylmercury concentration by species. Furthermore, though the EPA-FDA advisory points consumers towards state fish advisories for information on fish caught locally, these advisories are geared toward individual anglers and are organized by water body. State advisories largely provide no information on consumption of commercially purchased fish, whether in terms of fish caught and/or sold within the state, or commercially available fish from elsewhere that may be purchased at supermarkets or fish markets within the state\textsuperscript{53}. National methylmercury concentration data may not necessarily be accurate in terms of being representative of locally available fish. A study conducted on fish available in markets in New Jersey found higher mean mercury levels than stated in FDA data\textsuperscript{54}. Additionally, fish of the same species available in supermarkets, such as tuna, may have come from sources in different parts of the world, each with different levels of methylmercury exposure, and certain individuals may have markedly higher concentrations of methylmercury than others\textsuperscript{55}.

While the EPA methylmercury Reference Dose and the joint consumer advisory are geared toward preventing adverse effects from chronic exposure, there is concern over the possibility of effects of one-time, peak exposure to methylmercury. Due to wide variation in methylmercury levels in some species, there is the possibility for one-time or repeated single-meal exposures to levels of methylmercury well above the stated average for a given species. One study found that risk for the developing fetus may be compounded by single-meal exposures to fish with methylmercury levels greater than 2 ppm\textsuperscript{56}.

Issues with the EPA-FDA recommendations as far as species with lower methylmercury levels may have real implications for effectiveness of risk communication with the public. Though five commonly eaten low-methylmercury species are listed, these are not necessarily fish that some individuals, especially those with higher risk due to higher levels of consumption, may consume all that often. Market labeling for fish is not necessarily reflective of the reality of the type of fish being consumed. For instance, one study found that only 45 percent of fish sold as red snapper was actually red snapper\textsuperscript{57}, and many different species, each with different typical rates of bioaccumulation and average levels of methylmercury, may all be sold under the same category (e.g., tuna)\textsuperscript{58}. There is a lack of accurate labeling as to fish species and fish origin, and a lack of regional analysis of methylmercury levels in fish available commercially, leading to a breakdown in the ability of the public to make informed decisions. With all this in mind, we may assess whether public (and scientific / public health) concerns over the risks of methylmercury ingestion from fish and shellfish consumption are being adequately addressed. The answer is, perhaps, rather mixed. While there is acknowledgement of the risk posed by methylmercury exposure, and some effort to educate the public so that informed consumer decisions regarding fish consumption, especially for women of child-bearing age and young children, may be made, there are many shortcomings, some of which have been addressed above.

To that end, public concerns are not being well-served by the total effort by regulatory agencies to assess, regulate, and educate about the risk. The FDA-EPA joint consumer advisory on fish consumption and methylmercury sends mixed messages with regard to the health benefits and risks of fish consumption. Even efforts by the FDA to assess risks versus benefits of fish consumption were
criticized at length by the EPA in sixty-three pages of comments largely suggesting that the scientific effort was insufficient, inaccurate, unclear, and obtuse. The language of FDA advisories could be more attuned to the realities of the risk from methylmercury consumption, and there is no attention to the risks faced by specific higher-risk subpopulations. The EPA Reference Dose and the consumer advisory focus on chronic low-level exposure to methylmercury, and do not address medical concerns over the dangers posed by rare events of high dose exposure, especially with respect to critical windows of fetal neurodevelopment. The EPA has listed several research questions which show some attention to these issues, including extent and time-frame of decrease of methylmercury levels in fish with decrease of domestic mercury emissions, the need for further research into methylmercury exposure levels in higher-risk subpopulations, and investigating effective means of public risk communication.

Recommendations

Thus several recommendations may be made for future efforts at regulation and risk communication by the EPA and FDA, as well as state governments. Efforts on the federal level to reduce total domestic emissions of mercury are on the right track, and should continue to be implemented and enforced. Efforts to reduce international emissions, especially from the largest sources, should be pursued.

Continued and improved monitoring of methylmercury levels in humans should be carried out. Such improved monitoring might present a national profile in addition to specific information related to higher-risk subgroups. This information could prove extremely useful to targeting specific geographic locations or population groups for methylmercury exposure reduction, and would help provide specific strategies for each group.

There is a need for a widened and comprehensive national monitoring system of methylmercury concentrations in fish tissues. Such a system could utilize cooperation between federal and state governments, tribal governments, and local governments in order to provide a comprehensive geographical and temporal tracking system for methylmercury concentrations in fish and shellfish, and for targeting and limiting human methylmercury exposure. Such a system should take into account both local fisheries and sources of commercially available fish, with monitoring of domestic and imported fish and shellfish. Current state fish consumption advisories are inadequate and often only applicable to fish caught by anglers in local waterways. Commercially available fish constitutes the vast majority of fish consumed in the United States.

Implementation of a stricter system of fish labeling would allow the public to make more informed consumer decisions with regard to fish consumption and methylmercury exposure. Labeling and selling fish as simply tuna is largely useless when the fish sold may be any one of several species, all with differences in average levels and patterns of methylmercury concentration. Studies should concentrate on differences in fish preference by consumers of different socioeconomic and ethnic backgrounds, and assess these on the basis of differences in fish availability by region. For instance, fish such as cobia, king mackerel, and sea trout are unlikely to be widely commercially available in inland areas, whereas mass-marketed fish, such as tuna and salmon, will be. Burger et al further recommend that communities (fish consumers themselves) should be involved in a process of prioritizing fish to be studied for methylmercury concentrations, and in developing effective means of risk communication.

Further research into a variety of questions surrounding methylmercury bioaccumulation of fish, ingestion by humans, and adverse health effects with respect to dosage levels will be helpful in providing a better scientific basis for policy. Factors influencing rates of mercury methylation and bioaccumulation are poorly understood. Ecosystems are highly variable and may have differing effects on these processes, and hence on human exposure. Further research could ascertain a more scientifically acceptable and reliable risk-benefit analysis of fish consumption, comparing health benefits with risks of methylmercury exposure (and exposure to other toxins present in fish); this research could be combined with better human and fish methylmercury monitoring to provide a real picture of which fish are beneficial to consume, and which fish would be better to avoid. Some fish are high in beneficial nutrients and fatty acids, while others are not; largely independently, some fish are high in methylmercury levels, and some are not. This issue is by its nature very complex, and national averages based on limited data are insufficient to treat the issue.

A crucial emerging area for study will involve investigation of patterns of methylmercury exposure with regard to ethnic differences, dietary preferences, genetic differences, and variability in mercury uptake and excretion. Likewise, further study of and use of existing knowledge of public responses to various risk communication techniques will improve communication and implementation of im-
proved understanding of the risk.

The combination of these means for treatment of the issue of methylmercury exposure via ingestion of fish and shellfish should improve scientific understanding of the risk, and the consequent improved ability of regulatory agencies to regulate mercury emissions and inform the public should improve the ability of the public to make informed decisions with respect to fish and shellfish consumption. Thus exposure to methylmercury, a significant public health threat, can be efficiently reduced with efficacy by the consumer.

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Endnotes


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