

## 2.0 PROBLEM DESCRIPTION

### 2.1 WHY MERCURY POSES A RISK

The amount of mercury released from both natural and anthropogenic sources is difficult to quantify. Studies by Nriagu and Pacyna (1988) estimated global natural emissions at 3000 tons per year and the median for global emissions from human activities at 3560 tons per year (1983 basis). A more recent critical review by Jackson (1997) estimated that 2000 tons of mercury are emitted each year from natural sources, while 4000 tons of mercury are emitted each year from sources attributed to human activities (*e.g.*, combustion of fossil fuel and solid waste). An overview of global atmospheric emissions prepared by Schroeder and Munthe (1998) cited a number of other natural and anthropogenic source estimates along with the scientific uncertainties associated with estimates for both.

When airborne mercury is deposited on land or in water, biological transformations can occur that yield methylmercury. In its methylated form, mercury accumulates most efficiently in the aquatic food web resulting in risks to both humans and ecosystems (EPA, 1997a). Nearly all of the mercury that accumulates in fish is methylmercury. In lakes, rivers, and reservoirs, methylmercury is taken up by fish, resulting in significant increases in its concentration in fish tissue (*i.e.*, bioaccumulation). In some instances, the concentrations of methylmercury in fish may be several orders of magnitude greater than the concentrations in the surrounding water or sediment. Inorganic mercury, which is less efficiently absorbed and more readily eliminated from the body than methylmercury, tends not to bioaccumulate.

Human and wildlife exposure to methylmercury occurs almost exclusively through fish consumption. Fish eaters at the top of the aquatic food web generally exhibit higher methylmercury concentrations than those lower in the food web. The decision to focus on this exposure pathway in the *Mercury Research Strategy* is supported by modeling results from the *Mercury Study Report to Congress* (Volume IV: An Assessment of Exposure to Mercury in the United States). That modeling effort demonstrated that fish consumption poses the greatest risks to human health and wildlife. The impacts from urban and agricultural modeling were not of a comparable concern.

### 2.2 IMPACTS OF METHYLMERCURY ON HUMAN HEALTH AND WILDLIFE

Methylmercury is known to have toxic effects in humans, causing permanent damage to the brain and kidneys. The developing nervous system (*e.g.*, human fetuses, bird

embryos) is particularly sensitive to methylmercury exposures. Human epidemics of methylmercury poisoning (*e.g.*, Japan, Iraq) have established its toxicity to the nervous system (EPA, 1997a).

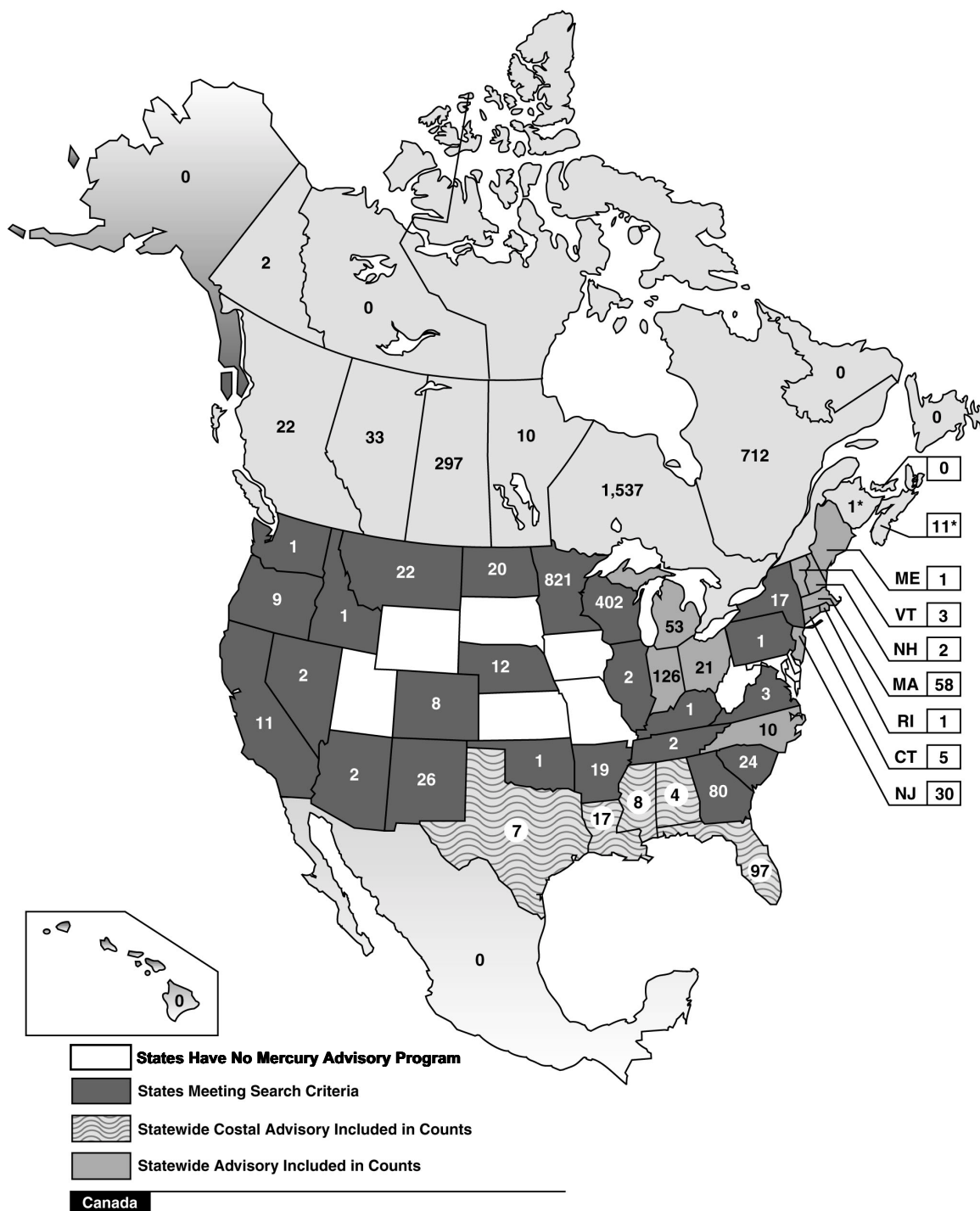
There are extensive data on the effects of methylmercury on the development of the brain (neurodevelopmental effects) in humans and animals. The most severe effects reported in humans were seen following high dose poisoning episodes in Japan and Iraq. Effects included mental retardation, cerebral palsy, deafness, blindness and dysarthria in individuals who were exposed in utero and sensory and motor impairment in exposed adults. Chronic, low-dose prenatal methylmercury exposure from maternal consumption of fish has been associated with more subtle end points of neurotoxicity in children. Those end points include poor performance on neurobehavioral tests, particularly on tests of attention, fine-motor function, language, visual-spatial abilities (*e.g.*, drawing), and verbal memory.

Excerpt from the Executive Summary of the Toxicological Effects of Methylmercury, National Research Council 2000. <http://books.nap.edu/books/0309071402/html/index.html>

#### 2.2.1 Human Health Impacts

Perhaps the most well known incident of mercury poisoning involved the consumption of methylmercury-contaminated seafood from Minamata Bay in Japan during the 1950s. In that case, mercury was used as a catalyst in an acetaldehyde production plant and was released into the Bay. The methylmercury poisoning involved the death and permanent disability of a number of individuals. The pathway of exposure being addressed in the *Mercury Research Strategy* is far more subtle. It involves the emission of low concentrations of mercury, mainly from combustion sources. These emissions lead to the build-up of methylmercury in water bodies and fish tissue over time. It is important to stress that the most likely individuals being exposed to a high level of methylmercury are consumers of large quantities of fish (*e.g.*, subsistence fishers). Pregnant women (maternal/fetal pair) and young children are particularly sensitive to exposures of high levels of mercury.

As illustrated in Figure 2, forty states have some form of mercury fish advisories for their water bodies. Statewide advisories for mercury occur consistently across the Northeastern states; Gulf Coast states have advisories in all coastal waters. In Canada, 97 percent of fish advisories are attributable to mercury. Mercury is the major reason for fish advisories, and there is an increasing trend in the



Canadian fish advisories reflect total fish advisories during 1997 (2,625).  
 More than 97% (2,572) were attributable to Mercury.

\* Provincewide advisories in effect in 1997 for Nova Scotia  
 (all rivers and lakes) and New Brunswick (all lakes).

Figure 2. Mercury-based Fish Consumption Advisories for North America (EPA, 1999a; EPA, 1999c).

number of advisories due to its presence in the nation's waters. Based on an analysis of dietary surveys, the *Mercury Study Report to Congress* (EPA, 1997a) risk assessment concluded that typical fish consumers in the United States were not in danger of ingesting harmful levels of methylmercury. This is a reflection of the relatively low amounts of fish consumed by the typical U.S. citizen.

Based on the same analysis of United States dietary survey data, the risk assessment estimated the percentage of people from different populations who consume methylmercury in excess of the Reference Dose (RfD)(EPA, 1997a)<sup>1</sup>. Among white/non-Hispanic populations, the fraction above the RfD was 9.0 percent, among black/non-Hispanics, 12.7 percent, and among persons of Asian/Pacific Islander ethnicity, Native American tribal members, and non-Mexican Hispanics (*e.g.*, persons from Puerto Rico and other Caribbean islands), 16.6 percent. Among women of childbearing age (*i.e.*, 15 through 44 years), 7 percent of the more than 58 million women in the group (*i.e.*, more than 4 million women) are exposed to methylmercury from fish at levels in excess of the RfD, using month-long exposures as the basis for calculation.

Depending on the methylmercury concentration in the fish, women may be putting their fetuses at risk to the subtle neurological and developmental effects associated with methylmercury exposure. In addition to women of childbearing age and their fetuses, populations of concern include young children (whose nervous systems continue to develop after birth). Young children exposed to methylmercury are of particular concern (EPA, 1997a), especially when they are members of a group who depend heavily on fish and fish-eating mammals as part of their diets (*e.g.*, some native groups that are subsistence fishers).

### 2.2.2 Wildlife Impacts

Concentrations of mercury in the tissues of wildlife species have been reported at levels associated with adverse effects in laboratory studies of the same species. However, field data are insufficient to conclude whether piscivorous wading birds or mammals have suffered adverse effects due to airborne mercury emissions. Modeling analyses suggest that it is probable that individuals of some highly exposed wildlife sub-population are experiencing adverse effects due to airborne mercury.

Excerpt from the Executive Summary of the *Mercury Study Report to Congress*, Volume I, December 1997.  
<http://www.epa.gov/oar/mercury.htm>

The impacts on wildlife from exposures to methylmercury are described in detail in the *Mercury Study Report to Congress* (EPA, 1997a). For purposes of this discussion, wildlife includes fish, birds (*e.g.*, loons, ducks, eagles), and

fur-bearing mammals (*e.g.*, otters, mink, panthers). All are susceptible to adverse methylmercury health effects. Marine mammals such as seals, walruses, dolphins and whales are also susceptible to methylmercury. Trace levels of mercury have been found in the liver of seals, porpoises and dolphins (Law, et al., 1991). The exposure pathway in aquatic systems indicates that birds and small mammals that feed primarily on fish and those that prey on these fish eaters will be at the greatest risk of toxic effects from methylmercury. An important aspect of these effects are the bioaccumulation of methylmercury by less complex organisms (*e.g.*, plankton, clams, crayfish,) and then their consumption by fish and small mammals. Direct uptake of methylmercury in the water column is also a pathway of exposure. These species can actually provide an early warning of mercury contamination via indications of neurological damage and reduced reproductive levels.

Mercury toxicity in fish is variable depending on a number of factors. These include fish characteristics (*e.g.*, species, life stage, age, size), environmental factors (*e.g.*, temperature, salinity, dissolved oxygen content, water hardness, other chemicals), and the form of mercury available (EPA, 1997a). The effects of methylmercury on early life stages of fish present more acute problems such as death, reduced reproduction, impaired growth and development, behavioral abnormalities, altered blood chemistry, reduced feeding rates and predatory success, and effects on oxygen exchange. Some signs of acute mercury poisoning include increased mucous secretion and respiration rate, loss of equilibrium, and sluggishness. Chronic poisoning is represented by emaciation, brain lesions, cataracts, and an inability to capture food. Evidence suggests that effects can be detected in water concentrations between 0.1 and 1.0 micrograms per liter for some species.

As summarized in the *Mercury Study Report to Congress* (EPA, 1997a), symptoms of mercury poisoning in birds include: muscular incoordination, falling, slowness, fluffed feathers, calmness, withdrawal, hyperactivity, hypoactivity, and drooping eyelids. Liver and kidney damage, neurobehavioral effects, reduced food consumption, weight loss, spinal cord damage, enzyme system effects, reduced cardiovascular function, and impaired growth and development are several of the indicators of sublethal effects of mercury in birds. Tissue mercury concentrations that are associated with toxicity in birds are similar despite differences in species, dietary exposure level, and length of time necessary to produce the effect. Neurological signs are generally associated with brain mercury concentrations of 15 micrograms per gram (wet weight) and 30 micrograms per gram in the liver and kidneys. With respect to hatchlings, mortality was observed in ducklings at 3 to 7 micrograms per gram (wet weight), at 2 to 3 micrograms per gram in loon eggs, and at 3.6 micrograms per gram in tern eggs. No effects were seen in herring gull hatchlings although the eggs contained approximately 10 micrograms per gram of mercury.

The *Mercury Study Report to Congress* identified the mink and otter as examples of fur-bearing mammals with increased risk from methylmercury exposure (EPA, 1997a). This was for exposures related to direct discharges of mercury to water bodies. The impacts of mercury on these mammals are less clear than for either fish or birds. This may be a direct reflection of the limited number of studies conducted on fur-bearing mammals and, in some cases, the confounding effects of other stressors. These stressors, cited for the endangered Florida panther, include habitat fragmentation, inbreeding, and feminization by endocrine disrupting compounds. With respect to the Florida panther, relatively high levels of mercury (0.005 to 20.0 micrograms per gram) have been measured in archived liver samples of dead animals. In another case, one death was attributed to mercury poisoning with mercury measured at 100 micrograms per gram in the liver and 130 micrograms per gram in the hair.

Based on the investigations reported in the *Mercury Study Report to Congress* (EPA, 1997a), causal links with airborne mercury deposition have not been established, but may contribute to population effects in some birds and fur-bearing animals, including the Florida Panther. The effect of mercury from point sources on limited wildlife populations, however, has been demonstrated. Tissue residues from these studies provide a basis for evaluating risks to other wildlife populations. Overall, wildlife (*e.g.*, fish, birds, fur-bearing mammals) appear to be more susceptible to mercury effects when they are located in ecosystems that experience the following: (1) high levels of atmospheric deposition, (2) surface waters already impacted by acid deposition, (3) characteristics other than low pH that result in high levels of mercury bioaccumulation in aquatic biota, and (4) species that experience high levels of exposure.

## 2.3 MERCURY USES AND RELEASES

### 2.3.1 Uses

Mercury has been widely used in industrial applications because of its unique properties. It conducts electricity, responds to temperature and pressure changes, and forms alloys with almost all metals. In the electrical industry, mercury is used in fluorescent lamps, as part of wiring devices, and with instruments that measure temperature and pressure. It is also a component of dental amalgams used in restoring teeth. In addition to its use in specific products, mercury is used in numerous industrial processes. The largest manufacturing use of mercury in the United States is associated with the production of chlorine and caustic soda by mercury-cell chlor-alkali plants. Mercury is also used in amalgamation with other metals (*e.g.*, gold) and as an antifungal agent in wood preserving (EPA, 1997a).

### 2.3.2 Releases

The most significant releases of mercury to the environment in the United States are emissions to the atmosphere. These emissions can be characterized as releases by human activities (*i.e.*, anthropogenic), releases from geologically bound mercury through natural processes, and releases through mass transfer to the atmosphere by biologic and geologic processes from previously deposited mercury (*i.e.*, re-emitted) (EPA, 1997a)<sup>2</sup>. The *Mercury Study Report to Congress* presents an inventory (based on 1994/1995 data) of anthropogenic mercury air emissions in the United States (See Table 1). This table presents the percentage of anthropogenic emissions attributable to each major source.

Table 1. Summary of Major Sources of Anthropogenic Mercury Air Emissions (EPA, 1997a).

Source	Percent
Coal-fired electric utility boilers	32
Municipal waste combustors	18
Coal- and oil-fired commercial/industrial boilers	18
Medical waste incinerators	10
Chlor-alkali plants	4
Portland cement plants	3
Oil-fired residential boilers	2
Other sources of mercury	13

Anthropogenic mercury sources within the United States emit approximately 158 tons of mercury per year (EPA, 1997a)<sup>3</sup>. The source categories presented in the table each constitute more than one percent of the total amount of mercury emitted to the atmosphere from human activities. The greatest emissions of anthropogenic mercury to the environment are from combustion of fuel that contains trace amounts of mercury. Emissions also come from industrial processes that use mercury, and disposal (especially by incineration) of products that contain mercury either as an intentional constituent or as an impurity.

Mercury-bearing wastes are generated from manufacturing processes and the disposal of consumer products. In 1995 an estimated 245 tons of mercury were discarded in municipal waste streams (EPA, 1997a). Most of this waste was either incinerated or placed in landfills. Industrial hazardous wastes with high mercury concentrations are currently incinerated or retorted. Retorting involves the heating of mercury-containing wastes with the mercury converting to a vapor. The mercury vapor is then captured and condensed back to its metallic form. The intentional use of mercury in commercial products in the United States has declined by more than 75 percent from 1988 to 1996 (EPA, 1997a). This reduction is largely due to the private sector's efforts to eliminate the use of mercury in products

and processes when replacements can be found. Along with this commercial use reduction, an increase in the recycling and recovery of mercury has resulted in a supply of the metal that now exceeds domestic demand.

In addition to air emissions and land disposal, mercury is released in other ways, including discharges from industrial sources and waste sites and releases of methylmercury from sediments to water bodies. Release of mercury in water discharges is believed small when compared to atmospheric emissions, but it can have significant local effects. Mercury discharges to surface waters from abandoned gold and mercury mines in the western United States may well be the cause of fish advisories for methylmercury in a number of streams and lakes. An example is the contamination of Clear Lake in California by the Sulphur Bank Mercury Mine Superfund Site. An international example of mercury pollution from an industrial source exists in Natal, South Africa, where the Thor Chemical Plant houses large quantities of mercury wastes that have leaked/leached to the nearby environment and groundwater. Releases of methylmercury from sediments have not been well quantified, but high concentrations of methylmercury in sediments often coincide with high concentrations of methylmercury in fish tissue (EPA, 1999a).

Modeling conducted as part of the *Mercury Study Report to Congress* (EPA, 1997a) estimated that approximately one-third of the United States anthropogenic mercury emissions (about 52 tons) are deposited through wet and dry deposition within the contiguous 48 States. The remaining two-thirds is transported outside the continental U.S. and enters the global mercury cycle. It is estimated that an additional 35 tons per year are deposited in the United States from the global cycle (*i.e.*, anthropogenic, natural, and re-emitted sources) (EPA, 1997a). As a consequence of mercury emission controls on a number of sources, anthropogenic mercury emissions in the United States will most likely decline over the next several years. According to Pirrone, et al., (1996), releases from human activities globally will increase mercury deposition in the United States unless reductions also occur in other countries. The role that emissions from natural and re-emitted sources play in assessing reductions to mercury is a complicating factor. These emissions must be taken into consideration in any estimates or documentation of total mercury reductions to the environment over the longer term.

## **2.4 MERCURY TRANSPORT, TRANSFORMATION, AND FATE**

### **2.4.1 Transport**

The air transport and deposition patterns in the United States for mercury emissions depend on various factors,

including the form of mercury emitted, the location of the emissions source, the stack height of the source, the topography near the source, and the prevailing air circulation patterns. For example, anthropogenic point sources (*e.g.*, coal-fired electric utility boilers, municipal waste combustors) emit primarily elemental mercury vapor ( $Hg^0$ ), gas-phase ionic mercury ( $Hg^{+2}$ ), and lesser amounts of particulate-bound mercury ( $Hg_p$ ). The chemical and physical properties of these different mercury forms influence their behavior in the environment and their significance as contaminants that have local, regional and global scale impacts.

*Local scale impacts* result from deposition within a 30-mile radius of an emissions source. For example, a source emitting primarily  $Hg^{+2}$  can be expected to have a relatively high percentage of mercury deposited within the 30-mile radius via wet deposition (EPA, 1997a).

*Regional scale impacts* result from either wet or dry deposition associated with long-range transport of emissions over hundreds of miles dispersed across wide areas. The highest deposition rates in the United States are predicted to occur in the southern Great Lakes and Ohio River Valley, the Northeast and scattered areas in the Southeastern United States (EPA, 1997a).

*Global scale impacts* result from  $Hg^0$  emissions that become part of the global emissions pool, where they can remain for a year or more before wet or dry deposition, on land or water. For example, recent studies indicate that in Arctic air, elemental mercury vapor may be oxidized resulting in increased mercury deposition (Schroeder and Munthe, 1998).

### **2.4.2 Transformation and Fate**

Anthropogenic mercury that is released directly to land or water bodies, or is deposited on them from the atmosphere, undergoes transformations that are not fully understood. These transformations convert some of the mercury to methylmercury. Not only is methylmercury much more toxic to humans and wildlife than inorganic mercury, but it is also more likely to bioaccumulate in fish tissue. This ability to bioaccumulate results in food chain impacts yielding higher concentrations of methylmercury in both humans and wildlife. The amount of mercury transformed to methylmercury varies greatly from one water body to another. According to Krabbenhoft, et al., (1999), there are a number of factors that influence methylmercury production beyond mercury loading. These are environmental setting (*e.g.*, climate, geology, land use, land cover), water chemistry, and wetland density with the latter being the most important basin-scale factor controlling methylmercury production.

## **2.5 MERCURY AND METHYLMERCURY RISK MANAGEMENT**

### **2.5.1 Risk Management**

Reducing risks from methylmercury is difficult because of the wide variety of sources that contribute mercury to the environment. Managing emissions and other releases of mercury requires a variety of approaches ranging from product substitution to end-of-pipe treatment. Some actions, such as eliminating mercury used in paints and batteries and controlling flue gas emissions from municipal waste combustion units and medical waste incinerators, are part of the technological options already used to reduce releases and emissions. Other options, such as removing mercury-containing products from waste streams (separation), coal cleaning, fuel switching, advanced mercury sorbents, sediment remediation methods, substitutes for mercury used in electronic switches and thermometers, and conversion of chlor-alkali plants from a mercury electrolytic cell to a membrane cell process, are available or under development.

Cooperative research between the public and private sectors is underway (*e.g.*, coal-fired utilities, mercury chlor-alkali production) to further develop management options, test and evaluate innovative solutions, refine or develop new data on their costs, and determine the benefits of combining various risk management approaches. Life-cycle tools are in various stages of development to evaluate how a mix of options can best be deployed to maximize reduction of risks to humans and wildlife at minimal cost. Development and evaluation of process changes, product substitutions, and innovative technologies will provide additional ways to address mercury. Finally, as the demand for mercury continues to decrease, issues involving mercury retirement will also come to the fore.

### **2.5.2 Risk Communication**

Communicating human health and ecological risks is an important component of any regulatory or voluntary Agency action and a vital part of effective risk management. Research can contribute to a methylmercury risk communication program in several ways that will assist EPA, state and local officials, and the public. There is a need to synchronize and standardize the fish consumption advisory messages for the numerous states in which they are issued. The criteria or standards each state uses to make fish advisory decisions are an essential element of any such effort. This research can be facilitated with a concerted and collaborative effort on the part of the federal agencies (*i.e.*, EPA, ATSDR, Food and Drug Administration [FDA]) that set various action levels for methylmercury in fish.

An improved understanding of exposure patterns (*e.g.*, amount of fish consumed, types of fish consumed, frequency of consumption) will assist in targeting both populations and the messages those populations receive. Research is needed on ways that people, in particular the populations most exposed to mercury and methylmercury risks, use information to make informed decisions. This will be particularly challenging since the most-exposed populations (*i.e.*, fetuses and young children) are not able to understand risk messages. The groups to reach will be their parents and other responsible adults.

1. A reference dose (RfD) is defined as an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive populations) that is likely to be without an appreciable risk of deleterious effects during a lifetime. (EPA, 1997a). At the RfD or below, exposures are expected to be safe. The risk associated with exposures above the RfD is uncertain, but risk increases as exposures to methylmercury increase.

2. With respect to this last category, a large portion of the deposited mercury is the result of past anthropogenic releases as well as releases from natural sources that heretofore have been sequestered (*e.g.*, arctic tundra, ice sheets, oceans and wetlands) (Lindberg, et al., 1998).

3. According to the Mercury Study Report to Congress, “[t]he current state of knowledge of mercury emissions . . . does not allow for an accurate assessment of either natural or re-emitted mercury emissions.” It is altogether likely that natural and re-emitted mercury emissions associated with contaminated soils and water bodies within the United States could add significantly to this value (EPA, 1997a).