Chapter 2 - OCCURRENCE OF MERCURY IN ENVIRONMENTAL MEDIA

A. Introduction

The issue of mercury in the environment has generated several important reports that reflect the evolution of our understanding of ecotoxicology and environmental science. Beginning in the 1960’s, Swedish scientists played a lead role, partly because the widespread use of mercurial fungicides to protect grain during the long Scandinavian winters had resulted in extensive poisoning of granivorous and raptorial birds. The report by Lofroth (1970) on methylmercury toxicity and the volume *The Biogeochemistry of Mercury in the Environment* (Nriagu 1979) summarized much of the early research.

Given the mobility of mercury in the environment and its ability to bioaccumulate in food chains, knowledge of the occurrence of mercury in various environmental media is critical to understanding and predicting both human and ecological exposures and risk from mercury. Figure 2.1 shows some of the complexity of mercury exposure pathways.

![Figure 2.1. The complexity of various mercury exposure pathways.](image)

Table 2-1 shows how various sources contribute to potential exposure pathways for methylmercury (MeHg), ionic mercury (Hg^{++}), and elemental mercury (Hg^0). Where data are available, the table provides estimates of daily exposure relevant to New Jersey.
Organomercurials are readily absorbed through the skin and can lead to fatal poisoning, but this is likely to occur only during occupational contact with the materials. It is not likely that handling fish muscle during preparation results in any dermal or inhalation absorption. These pathways are not included in Table 2-1.

Table 2.1. Sources and Estimates of Daily Human Exposures to Mercury. (Unless otherwise indicated, exposures are estimates of average daily intake in NJ and/or nationwide.)

<table>
<thead>
<tr>
<th>Source of Exposure</th>
<th>Methylmercury (MeHg) (µg/day)</th>
<th>Inorganic Hg Salts (Hg$^{++}$) (µg/day)</th>
<th>Elemental Hg (Hg°) (µg/day)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Foods (non-fish)</td>
<td>Negligible</td>
<td>0.9$^a$</td>
<td>Negligible</td>
</tr>
<tr>
<td>Commercial fish</td>
<td>6$^b$</td>
<td>&lt;1$^c$</td>
<td>Negligible</td>
</tr>
<tr>
<td>Sport fish</td>
<td>No population-based data available</td>
<td>No population-based data available</td>
<td>Negligible</td>
</tr>
<tr>
<td>Public supply water</td>
<td>Negligible</td>
<td>&lt;&lt;4$^d$</td>
<td>Negligible</td>
</tr>
<tr>
<td>Private wells</td>
<td>Negligible</td>
<td>0.4-4 (45% of exposed population)</td>
<td>0.006-0.03 (by inhalation in a shower)$^{e,f}$ (For consumers of water from selected wells in southern NJ$^g$)</td>
</tr>
<tr>
<td>Outdoor air</td>
<td>Negligible</td>
<td>Negligible</td>
<td>0.04 - 0.2$^g$</td>
</tr>
<tr>
<td>Indoor air</td>
<td>Negligible</td>
<td>Negligible</td>
<td>No population based data available</td>
</tr>
<tr>
<td>Soil ingestion</td>
<td>Negligible</td>
<td>&gt;3$^h$ For sites exceeding NJDEP soil cleanup criterion for Hg</td>
<td>Negligible</td>
</tr>
<tr>
<td>Dental amalgams</td>
<td>Negligible</td>
<td>Negligible</td>
<td>.3-17 µg depending on number and age of fillings$^a$</td>
</tr>
</tbody>
</table>
b. (Stern et. al. 1996) based on NJ fish consumers, general population.
c. Based on assumption that MeHg accounts for >90% of total mercury in fish.
d. Based on lack of systematic exceedance of drinking water Maximum Contaminant Level (MCL) for inorganic mercury, and assuming 2 L/day of drinking water consumption.
e. (USGS 1997). Based on 2,239 (non-randomly) selected private wells in southern NJ. [NB: Because wells were not selected at random this value cannot be extrapolated to the general population.]
f. See Volume II, Chapter 7 Occurrence and Impact of Mercury in New Jersey's Environmental Media, "Water in Private Wells" of this report for details of assumptions and modeling) based on average concentration of “volatile” mercury in wells exceeding 2 g/l total mercury. Assumes mercury identified as “volatile” mercury is elemental mercury. Assumes 10-50% volatilization of elemental mercury during a 15 minute shower. (ATSDR 1999a). Based on 1980 US EPA estimate of nationwide average ambient air mercury levels of 2-10 ng/m³, and assumed breathing rate of 20 m³/day.
g. Applies only to sites exceeding NJDEP cleanup criterion for total mercury. Assumed to be inorganic mercury (Hg²⁺), and assuming average daily soil ingestion of 200 mg/day. Assuming 100% bioavailability for total mercury by ingestion. This value cannot be extrapolated to the general population.

B. Absorption and Bioavailability

Bioavailability refers to the ability of mercury to be transferred from one matrix to biological tissue, i.e. from water or sediment to biota, or from air, soil or food into an organism. Bioavailability depends on the properties of the matrix and the form of the mercury. The term external bioavailability is sometimes used to distinguish the transfer of mercury from environmental media into an organism, while internal bioavailability refers to the ability of mercury to be transferred from one compartment to another within an organism.

Environmental mercury in soil or sediment is not always available for methylation by bacteria. Using chemical extraction procedures, Martin-Doimeadios et al. (2000) isolated a sulfide form and found no organic mercury being formed. How long such a situation would last is not known. Benoit et al. (2001a) have quantified the impact of adding sulfide to bacterial cultures, showing a fourfold decrease in methylation as sulfide concentration was increased from micromolar to millimolar concentration. They postulate that the concentration of a neutral dissolved Hg-sulfide species is the critical factor (Benoit et al. 2001b).

Substances that enter the intestinal tract or the lungs do not necessarily gain access to the blood stream or reach critical target organs. The amount that is transferred depends on two related phenomena: absorption and bioavailability. The intestinal tract and the lungs differ in their absorptive properties for each mercury species, and absorption may vary by age, frequency of meals and other dietary factors. It is generally recognized that elemental mercury vapor is readily absorbed through the lungs (50-100%), but that absorption of liquid elemental mercury from the intestinal tract is negligible (much less than 1%). On the other hand, MeHg is readily absorbed from the intestinal tract (close to 100%) and from the lungs.

Whereas absorption is a property of the body, bioavailability reflects the nature of the medium or matrix. Certain substrates will bind mercury with greater strength or affinity, making it more difficult for the intestine to extract the mercury so it can be absorbed. Unfortunately, there are relatively few studies of the bioavailability of MeHg in different materials, so for risk assessment purposes it is assumed to be 100% in both lungs and intestinal tract. MeHg is also absorbed through skin.
C. Methylmercury (MeHg) in Environmental Media

Inorganic mercury (Hg++) falls on the water surfaces or runs off from the surrounding land and settles to the bottom sediment where bacteria transform it to methylmercury (MeHg) through the process of biomethylation. A typical pattern of biomagnification is shown in the conceptual illustration in Figure 2.2. It begins with a hypothetical water concentration of 1 ng/kg (or 1 part per trillion, 1ppt). After methylation, the MeHg is readily absorbed and retained by any organism in the food chain. Each organism eventually bioaccumulates mercury to a concentration about 10 times greater than in its food. Hence bacteria and phytoplankton would have 10 ng/kg (or 10 part per trillion, 10 ppt). The next trophic levels, protozoa and zooplankton, would accumulate 100 ng/g and so on up the food chain until human or other predators (illustrated by a kingfisher) consume fish with 1 million ng/kg or a 1 ppm concentration. The predators would then achieve concentrations of 10 ppm in their tissue. The entire process is referred to as food chain biomagnification.

**Figure 2.2. Typical Pattern of Mercury Biomagnification.**

1. Methylmercury in Food

Because MeHg is created in aquatic systems, human exposure to MeHg is almost entirely confined to consumption of aquatic organisms. In theory, human exposure could occur through consumption of fish-consuming birds and terrestrial animals such as osprey, eagles, pelicans, and bears. In practice, however, such animals are highly uncommon sources of food for humans in most places, although human populations of oceanic islands often consume fish-eating seabirds. In a survey of food analyses from 10 state food laboratories conducted in 1988-1989, MeHg was found above detection levels in only 0.09% of 13,980 samples (summarized in ATSDR 1999a). Thus, fish consumption poses the only significant source of dietary exposure to MeHg for most Americans. Details on MeHg exposure through
fish consumption are provided in Chapter 4 of this Volume. People who consume wild
game frequently may also be at increased risk. For example, some studies of duck muscle
showed levels ranging from 0.5 ppm in vegetarian ducks up to 12.3 ppm in fish-eating ducks
(Vermeer et al. 1973). Fortunately, most consumers of wild duck meat avoid the fishy-
tasting fish-eating species.

Fish are widely recognized to be a valuable source of protein with lower cholesterol than red
meat, and some species also are rich in omega-3 fatty acids which are believed to be
particularly healthful. Yet the consumption of fish varies greatly from country to country and
within countries by location, ethnic group, socioeconomic class and dietary preference. But
it has long been recognized that people who consume large quantities of fish can have
excessive exposure to bioaccumulative pollutants such as organochlorines and
methylmercury. In the United States and in New Jersey, most people eat fish occasionally,
but some eat fish frequently. Those who eat fish daily may accumulate sufficient quantities
of MeHg to become symptomatic. This is examined in more detail in Chapter 9 Section B.

2. Methylmercury in Soil

In most soil studies where mercury has been included as an analyte, the mercury is not
speciated. Therefore, there is little direct information on MeHg levels in background soils.
Some information exists on MeHg levels in soils at hazardous waste sites and in soils after
sewage sludge application.

There is recent evidence that a small, but potentially significant fraction of the total mercury
in municipal sludge and sludge-derived compost is MeHg. Carpi et al. (1998) found that
routine application of municipal sewage sludge to soil increased the concentration of MeHg
in the soil from 0.3 µg/kg to 8.3 µg/kg (8.3 ppb).

In a recent study of the speciation of mercury in the soil at a NJ hazardous waste site with
extensive mercury contamination, organic mercury appeared to constitute up to 0.2% of the
total mercury with a maximum concentration of 500 ppb. The mercury contamination at this
site apparently originated as Hg⁰ and, since this site is not a wetland, the organic mercury
presumably resulted from methylation of inorganic mercury in situ (PTI 1997). In theory, the
occurrence of MeHg in the soil resulting from natural wetlands processes, sludge application,
or disposal of inorganic mercury hazardous waste, poses the potential for MeHg exposure
through ingestion of soil (ATSDR 1999a). Since a 15 kg child is assumed to ingest
approximately 200 mg of soil per day through normal hand-to-mouth activities (EPA 1992),
and the current USEPA Reference Dose for methylmercury is 0.1 µg /kg-body wt/day, soil
would have to be contaminated with 7.5 µg MeHg/g soil in order for soil ingestion to result
in exceedance of the Reference Dose. Although much higher levels occur at certain
hazardous waste and former industrial sites, most soil samples have much lower levels than
this. The presence of MeHg in soil could also result in MeHg uptake into edible plants. There
is substantial literature on mercury concentrations in plants, but very little specifically
measures MeHg. MeHg is taken up by salt marsh grasses and freshwater plants (Ribeyre and
Bouduo 1994), hence this little studied pathway could be important for MeHg under some
circumstances. Although mercury concentrations have been measured in a wide variety of
foods, with concentrations mainly below 100 ppb, there is virtually no information on MeHg
in terrestrial food crops. Since food crops are known to be an important route of exposure to
cadmium (McLaughlin and Hamon (2001)) it is prudent to study mercury accumulation in
crops. The occurrence of MeHg in soil is of potential significance when runoff from the soil results in transport of even small amounts of MeHg to waterbodies.

3. Methylmercury in Air

Only a small amount of airborne mercury is MeHg. A Reference Concentration (specific to the inhalation route of exposure) for MeHg has not been derived. However, if the standard inhalation rate of 20 m³/day is assumed for a 70 kg adult, and it is conservatively assumed that 100% of inhaled MeHg is taken up by the circulating blood, the current USEPA Reference Dose can be calculated to be equivalent to an air concentration of 0.35 µg/m³. This estimate is intended strictly for purposes of comparison since it does not address potential differences in metabolism of ingested and inhaled mercury. There are few reports of ambient air levels of MeHg (Brosset and Lord 1995; Fitzgerald et al. 1991; Prestbo and Bloom, 1994). Available data indicate levels of 3 to 38 pg/m³ (MeHg), which is about 0.01% of the air concentration calculated above. Since MeHg has a low vapor pressure, and tends to bind tightly to organic and biochemical molecules, release of MeHg from aquatic systems would not be expected to be significant from the standpoint of inhalation exposures on or near waterbodies. Carpi et al. (1998) reported on the release of MeHg from sludge amended soil, but the concentration of MeHg in the soil was low and the amount of MeHg released to the atmosphere was not significant from the standpoint of local exposure.

4. Methylmercury in Water

Mercury occurs in both surface and ground waters from both natural and anthropogenic sources. The cycling of mercury in surface waters is the basis for the accumulation of methylmercury in fish and will be discussed in several sections of this report. Mercury in ground water has likewise emerged as a public health concern in certain sections of New Jersey.

In lakes, mercury is partitioned between organic particle-bound and dissolved forms. The creation of new water bodies by dams results in the flooding of soil containing natural quantities of mercury and thereby increases the amount of mercury available for biomethylation. Reservoir creation also results in decomposition of flooded organic matter which enhances the rates of methylation. Studies of mercury in NJ lakes show higher mercury levels in fish from newly created than from old or natural lakes (see Chapter 8/Section C). Tree Swallow nestlings living near a reservoir showed a doubling in MeHg body burdens after flooding (Gerrard and St. Louis 2001). However, the toxic effects were to some extent offset by the greater abundance of food in the flooding period.

A drinking water Maximum Contaminant Level (MCL) for MeHg has not been derived (the MCL for total mercury is 2 µg/L). However, if a 2 L/day consumption of drinking water for a 70 kg adult is assumed, the current USEPA Reference Dose for MeHg corresponds to a water concentration of 3.5 µg/l (3.5 ppb). No data are available on the occurrence of MeHg in community drinking water. Recent speciation studies of the mercury contamination in ground water used as domestic drinking water in southern NJ found that up to 8% of the total mercury could be organic mercury. The maximum concentration of organic mercury was 137 ng/l (0.14 ppb) (Murphy et al. 1994). Applying the assumptions described above, this is 4% of the intake corresponding to the Reference Dose for MeHg, or the Hazard Quotient is 0.04 where a hazard quotient of 1 or greater is unacceptable. Drinking water or showering are generally negligible exposure pathways for MeHg.
5. Summary: Methylmercury in Environmental Media

Fish consumption is the only significant pathway of environmental human exposure to MeHg. The potential exists for significant exposure through soil ingestion if MeHg per se (or other forms of organic mercury) is discharged directly to the soil. MeHg in soil can also be a significant source of MeHg in aquatic systems. Little attempt has been made to identify MeHg in plants grown on mercury contaminated soil. To date, only trace levels of MeHg have been found in air. Few investigations of the presence of MeHg in drinking water have been undertaken. Data from wells with largely inorganic mercury contamination in NJ show only trace quantities of MeHg.

D. Inorganic Mercury in the Environmental Media

This section covers elemental and ionic forms of mercury. Most studies of mercury in environmental media did not speciate mercury, but report total mercury. In some cases it is possible to infer whether the mercury is organic or inorganic. Most mercury in soil, air, and water is inorganic or elemental, while most mercury in biota is organic.

1. Inorganic Mercury in Food

Food chain exposure is mainly important for MeHg, and almost all of the mercury in finfish tissue is MeHg; however, this is not necessarily true for mercury in cereals and other food sources. Although inorganic mercury is present in finfish tissue (as Hg\(^{++}\)), inorganic mercury is not significantly bioaccumulated in fish and generally constitutes less than 10% of the total mercury in fish. Inorganic mercury accounts for a higher proportion of the total mercury in crustaceans and mollusks. However, these species tend to have lower levels of total mercury than do finfish. While levels of MeHg in fish are often in the range of 0.1-1.0 ppm for commercial ocean fish and often greater than 1.0 ppm for large freshwater fish and predatory marine fish, the total mercury concentration in mollusks rarely exceeds 0.1 ppm (Stern et al. 1996). The typical levels of total mercury in lobster is reported to be 0.25 ppm (Hall et al.1978), but it is not clear whether this largely represents inorganic mercury, MeHg, or both. In a USFDA Total Diet Study conducted from 1982-1984, 23% of the total mercury was found in the non-seafood portion of the typical diet of adult males 25-30 years old (ATSDR 1999a). This component was most likely inorganic mercury.

Inorganic mercury is taken up to some extent by edible plants (Kabata-Pendias and Pendias 1984). Raw produce in Germany was found to contain total mercury concentrations of 0.005-0.05 µg/g (ppm), but raw mushrooms contained up to 8.8 ppm total mercury (ATSDR 1999a). Based on 1980-1988 UNEP/FAO/WHO data, Galae-Gorchev (1993) estimated that foods other than fish and seafood had average mercury concentrations of 0.01 µg/g, presumably as inorganic mercury.

In individuals who do not consume fish, and who are therefore presumably exposed only to inorganic mercury, the typically low concentrations of mercury in blood and hair indicate that very few are likely to exceed the current USEPA Reference Dose for inorganic mercury (based on mercuric chloride) of 0.3 µg/kg/day.

2. Inorganic Mercury in Soil
Inorganic mercury salts (i.e., \( \text{Hg}^{++} \)) are generally stable in terrestrial soils, and methylation is generally negligible. Interconversion among the various anions or ligands which associate with \( \text{Hg}^{++} \) however, is possible, with conversion to the sulfide being the most thermodynamically favored conversion, yielding the most stable form (HgS). Given the US EPA Reference Dose for inorganic mercury (as \( \text{HgCl}_2 \)) of 0.3 \( \mu \text{g/kg/day} \) and assuming that a 15 kg child ingests 200 mg of soil daily, the concentration of inorganic mercury salts in the soil would have to be 22.5 \( \mu \text{g/g} \) (22.5 ppm) to exceed the TfD. Background levels of mercury in NJ soils are generally less than 1 ppm, although they may typically be in the range of 1-2 \( \mu \text{g/g} \) (ppm) in some urban soils and have been observed as high as 7.7 ppm on golf courses (Fields et al. 1999; see also Chapter 7 of this volume). \( \text{Hg}^{++} \) concentrations in NJ soils are sometimes found to approach or exceed this level at sites where mercury-containing waste has been discharged. The oral bioavailability of the various \( \text{Hg}^{++} \) compounds (i.e., the extent to which they are taken up through the gastrointestinal tract and are available for distribution to target sites in the body) varies roughly in proportion to their solubility. Although bioavailability data for inorganic mercury compounds is sparse, it appears that only about 2% of an oral dose of HgS can be absorbed (Stern 1997a). By comparison, for \( \text{HgCl}_2 \), estimates of absorption ranges from less than 7% to about 25% (Stern 1997a). Thus, the levels of \( \text{Hg}^{++} \) in soil which may actually pose a significant health risk depend to some extent on the specific compound. These values apply to the pure form of the compound. It is likely that when ingested in a soil matrix, the bioavailability is decreased, but few quantitative data on bioavailability in soil are available.

\( \text{Hg}^0 \) in soil has the potential to volatilize to the surrounding air. \( \text{Hg}^0 \) vapor released from soil to the outdoor air will tend to dissipate rapidly. However, if released into confined spaces such as into buildings built over \( \text{Hg}^0 \) contaminated soil, indoor air levels could reach levels of health concern. Exposure to \( \text{Hg}^0 \) in soil can also occur through ingestion of soil. Because of the tendency of \( \text{Hg}^0 \) liquid to form globules, it is generally not uniformly distributed among soil particles and determinations of average concentration may differ significantly among samples. \( \text{Hg}^0 \) is poorly absorbed through the gastrointestinal tract with bioavailability of about 0.01% reported (WHO 1991). Ingestion of \( \text{Hg}^0 \) in soil is therefore not considered to be a pathway leading to potentially significant exposures.

3. Inorganic Mercury in Air

\( \text{Hg}^{++} \) is released from combustion sources due to the liberation of existing \( \text{Hg}^{++} \) in the combusted material, or due to oxidation of \( \text{Hg}^0 \). Additionally, once airborne, some \( \text{Hg}^{++} \) is formed by atmospheric oxidation of \( \text{Hg}^0 \) through oxidation by ozone. Inhalation of inorganic mercury is generally not a significant pathway of exposure. \( \text{Hg}^{++} \) is subject to removal from the atmosphere by washout during precipitation events and mercury adsorbed to airborne particulates falls out as dry deposition. Deposition on watershed lands or directly to waterbodies by these processes is a major source of mercury transport into aquatic systems where it can become methylated and undergo biomagnification in biota.

\( \text{Hg}^0 \) in ambient air circulates as part of the global atmospheric mercury budget and is enhanced by localized sources. Ambient air concentrations of \( \text{Hg}^0 \) are reported to range from about 2 ng/m\(^3\) to about 10 ng/m\(^3\), with the higher end of this range reflecting contributions from specific local sources (ATSDR 1999a). These levels should be contrasted with the current US EPA Reference Concentration for \( \text{Hg}^0 \) of 3 \( \times 10^4 \) mg/m\(^3\) (0.3 \( \mu \text{g/m}^3 \)). Thus, ambient air exposures to \( \text{Hg}^0 \) are unlikely to pose a significant potential for health risk. In contrast, exposure to \( \text{Hg}^0 \) vapor indoors as a result of spills or intentional application of liquid
Hg$^0$ (such as in certain cultural practices) can be significant with respect to health effects. As little as one drop (0.05 ml) of liquid Hg$^0$ in a sealed bedroom-sized room (assuming a room volume of about 33 m$^3$ and no air exchange) can result in an air concentration equal to the US EPA Reference Concentration.

Another source of indoor Hg$^0$ exposure is residential occupancy of buildings in which mercury was previously used in manufacturing. In a former factory in Hoboken, NJ, converted to residential occupancy, air concentrations of Hg$^0$ ranged from 5-888 µg/m$^3$. Two thirds of the residents had elevated mercury in urine (< 20 µg/L). Subtle neurological effects possibly related to this exposure were observed among some residents (Fiedler et al. 1999).

4. Inorganic Mercury in Water

Due to the moderate solubility in water of some of the salts of Hg$^{++}$ (e.g., 1 g/35 ml for HgCl$_2$; ATSDR 1999a), Hg$^{++}$ can occur as a significant contaminant in drinking water either through direct discharge to surface water sources, or through leaching to ground water from contaminated soil. A survey of 6,856 samples of ground water drinking water sources in California found 27 exceedances of the Maximum Contaminant Level of 2µg/l (ATSDR 1999a). In southern New Jersey, compilation of well sampling data from 2,239 private wells in seven counties showed detectable levels of mercury in 59% of the wells (detection limit 0.2-0.5 µg/l), and exceedance of the Maximum Contaminant Level in 306 wells (13.7%) (USGS 1997). Speciation of the mercury in these samples, revealed that HgCl$_2$ accounted for a median fraction of about 94% of the total Hg. Volatile mercury (assumed to be Hg$^0$) accounted for a median fraction of about 6% of the total Hg. Hg$^0$ in water is very poorly absorbed through the gastrointestinal tract, but can potentially be inhaled if volatilized from water to indoor air particularly when the water is heated and/or agitated such as in a shower. Estimates of inhalation exposure to Hg$^0$ volatilized from shower water indicate that if the Hg$^0$ concentration in water were at the Maximum Contaminant Level and the conditions of use result in 50-100% volatilization of the Hg$^0$, the inhaled dose from a shower would approach the RfD (Stern 1997b).

5. Summary: Inorganic Mercury in Environmental Media

Although it is difficult to identify dietary intake data specific to inorganic Hg, it does not appear that dietary intake approaches the Reference Dose for inorganic mercury in any identifiable group of people in NJ except for those occupationally exposed. Soil highly contaminated with mercury salts may result in exposure above the Reference Dose due to soil ingestion particularly for small children. However, the bioavailability of the various mercury salts varies widely. Ambient air concentrations of inorganic mercury are unlikely to approach levels of health concern. However, very little Hg$^0$ is required to pose a health hazard under indoor conditions. Inorganic mercury in drinking water has been observed to exceed the Maximum Contaminant Level in some locations. While such contamination is largely due to mercury salts, some Hg$^0$ has been observed in such cases. Hg$^0$ volatilized from water during showering may approach levels of health concern under some circumstances.

E. Hair Mercury as a Biomarker of Exposure

Hair has proven to be useful for biomonitoring methylmercury exposure. Hair is a better indicator of methylmercury than of inorganic mercury exposure, and about 80% of the mercury in hair is MeHg (Cernichiari et al. 1995). The more or less constant growth rate of
hair (1.1-1.3 cm/month, March et al. 1995), allows the profiling of temporal exposure patterns. For example, in the case of the death of a researcher, Dr. Karen Wetterhahn had accidental exposure to MeHg and the profile of mercury in her hair reached a maximum greater than 900 ppm and declined steadily thereafter, confirming her reported one-time exposure (Nierenberg et al 1998).

Hair thus allows a retrospective approach to estimating the time and magnitude of exposure. People who eat fish less than once a week and have no other mercury exposure generally have hair levels less than 1 ppm. A level of 10 ppm is considered a threshold indication of risk. Women from a fishing community on the coast of northern Peru have hair mercury levels from 1.2-30 ppm (geometric mean 8.3), which was presumably derived from the preponderance of marine fish in the diet (March et al. 1995).