CHAPTER 2

Group Report: Mercury

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INTRODUCTION

Among the metals and metalloids of concern for their potentially harmful effects in the environment, mercury is unique for a number of reasons. It is chemically distinctive (Carty and Malone, 1979) and because of its tendency to form strong covalent bonds its behavior in biological systems is also distinctive. The biogeochemical properties of mercury mean that elevated exposures tend to be quite natural phenomena. This suggests that relatively minor perturbations of key portions of the cycle (i.e. deposition, accumulation by fish) could result in major changes in the exposure to or uptake by sensitive human populations. The subtlety of mercury’s early toxic effects suggests the need for careful monitoring of these key parts of the cycle and of target populations.

The major pathway to man is commonly accepted to be ingestion of aquatic organisms, particularly fish. Mercury is the only metal which indisputably biomagnifies through the food chain. In addition, unlike most other metals, mercury is efficiently transformed into its most toxic form (methyl mercury) in the aquatic environment. These facts make mercury exposure to target populations potentially serious even in remote areas. All of the above observations suggest that the effects of environmental exposure to mercury may be insidious, difficult to detect, and potentially widespread.

In the context of global metal cycles, the primary form of mercury emissions to the environment further exemplify its uniqueness. Mercury emissions, both natural and anthropogenic, are dominated by losses of vapour forms to the atmosphere (Kothny, 1973). These forms have a relatively long residence, conducive to long range transport (Lindqvist et al., 1984). As a result, the bulk of the global atmospheric pool of mercury is in vapor form (Brosset, 1981). All of these facts indicate the truly global dispersion of
mercury, regardless of its source. Although emission estimates have a high degree of uncertainty, several published calculations support the idea that natural emissions are quantitatively comparable with those of man (Pacyna, this volume, Ch. 7). However, some estimates suggest that natural sources dominate (Fitzgerald et al., 1984). Even though anthropogenic emissions of total mercury have increased by two or three times between 1900 and 1970 (Andren and Nriagu, 1979), with several documented incidences of severe local effects, we feel that the total global atmospheric load of mercury is relatively insensitive to man's direct emissions. Therefore, we should focus on changes in the general chemistry of the atmosphere which could somehow enhance mercury deposition and thus have a significant effect on the global cycle. These changes could be reflected in major perturbations in both urban and remote areas.

The objective of our study group was to identify several major research questions which deserve particular attention at this time. These are addressed below. For recent reviews on the state of knowledge regarding mercury cycling the reader should consult Nriagu (1979) and Lindqvist et al. (1984).

**QUESTION 1**

Is there a human health problem related to mercury? Have any health effects been documented recently? If so, who are the populations at risk? Have any direct environmental effects ever been documented, or are the indirect 'effects' of food chain transfer and bioaccumulation the major sources for concern?

Food chain transfer with or without biomagnification seems to be confined to certain well-defined food chains with major aquatic links. For example, direct effects on fish-eating birds and mammals have been reported in isolated cases in Canada and Sweden. In addition to documented acute effects of human exposure to mercury we must remember that we have no data on the potential effects of long-term chronic exposure of man to levels of atmospheric Hg vapour which are measurably elevated above background levels, but not high enough to result in immediate toxic effects. For a review of the general human health effects of mercury the reader is directed to Piotrowski and Inskip (1981).

**Mercury and Human Health**

The primary route of exposure of methylmercury to man is through the consumption of food, especially fish. Several human populations around the world have been identified on the basis of their high consumption of fish as being in a high-risk group with respect to methylmercury poisoning.
Elevated mercury levels in many of the fish have been linked to both anthropogenic (Canada, Italy) and natural (New Guinea, New Zealand) sources (Hutton, this volume, Ch. 6). Many attempts to diagnose methylmercury intoxication have been inconclusive studies. Future clinical, epidemiological or neurological testing of humans must be statistically valid and rigorously designed.

In contrast to other toxic elements of concern (e.g. Cd, Pb) little is known about nutritional or other dietary factors (e.g. Ca interactions) which can influence methylmercury uptake and toxicity in humans or other mammals. Routine tests of humans in high-risk groups should be developed using the least intrusive but valid techniques (e.g. hair analysis). Also, surveillance of humans in areas subject to direct mercury discharge into the environment should be undertaken, regardless of fish consumption in these areas. While the use of mercury for agricultural purposes and direct industrial discharges of mercury into the environment have been largely curtailed in North America and much of Europe, this is not the case in developing countries around the world. Precautions must be taken to prevent future episodes of human poisoning by mercury.

**Mercury and Environmental Health**

Mercury is naturally present in low quantities in all living organisms and, under any degree of environmental pollution, may be efficiently accumulated by the biota. Mercury is unique among other toxic elements as it is the only metal which is consistently biomagnified within the aquatic food chain. However, evidence for effects in the environment have been limited to the upper trophic levels, though experimental demonstrations of effects on phytoplanktonic photosynthesis at ppb of mercury in marine waters have been reported.

Biological impacts associated with mercury in the environment have generally been associated with substantial local discharges of mercury into air or water from point sources. For example, elevated levels of mercury have been observed in a variety of aquatic benthic organisms, plants and fish living downstream of a chloralkali plant (Hildebrand et al., 1980b). While toxic effects have not been documented for the aquatic biota, organisms consuming contaminated aquatic species have suffered mercury poisoning. Species affected in Canada under these conditions include a variety of fish-eating birds (Fimreite, 1979), wild mink, and otter (Wobeser and Swift, 1976; Wren, 1984). Serious contamination of terrestrial wild birds has been documented in Canada, Sweden and other countries as a result of the use of organo-mercurial compounds as seed dressings (Borg et al., 1969; Fimreite, 1979). In Sweden, the species most affected were birds of prey and predatory animals such as fox and marten (Borg et al., 1969).
In summary, biological effects attributable to mercury have almost always been associated with recognized point sources of mercury discharge into the environment. While mercury levels in biota, especially in aquatic species, may be elevated in certain areas resulting from more diffuse sources of mercury in the environment (e.g. fossil fuel combustion, see Question 7) no direct widespread biological effects have been unequivocally linked to these elevated tissue levels. However, many animal and plant species are sensitive to mercury poisoning, and this fact warrants continued surveillance of mercury in biota from a wide range of conditions and geographical locations. In addition, because of its capacity to become biomagnified upward in natural food chains, mercury monitoring must also be directed toward predatory species at the highest trophic levels which will be exposed to the most elevated mercury levels within a particular ecosystem.

QUESTION 2

What are the natural versus the anthropogenic fluxes of mercury to the global atmosphere? Are the same species of mercury released from each source? If natural fluxes actually do exceed anthropogenic fluxes by 3–5 times, as some have suggested, does it matter where the sources are, or merely that dispersion and deposition are occurring on a global scale?

Estimates of global emissions of mercury to the atmosphere are highly variable and necessarily imprecise because of the uncertainty about natural emission rates. Even more uncertain is the actual speciation of the emitted mercury, generally assumed to be elemental mercury vapor (Hg\(^0\)) (Lindqvist et al., 1984). Recent estimates of global emissions from natural sources are on the order of 2700 (Pacyna, this volume, Ch. 7*) to 6000 (Fitzgerald et al., 1984) tons/year while those for anthropogenic emissions are 630 to 2000 tons/year (same authors, respectively). Despite this uncertainty, both estimates result in a similar ratio of natural/man-made losses to the atmosphere (about 3–4 : 1). Earlier estimates (Andren and Nriagu, 1979) were higher for both pathways, but resulted in a ratio of the same order (2 : 1). Thus, there is general agreement that natural sources are somewhat larger than those due to man’s activities. Given this and the global uniformity in atmospheric concentrations (Slemr et al., 1984), it could be argued that the location of the specific sources is not particularly important on a global scale, although it can be critical on a local scale.

Because of the importance of the natural emission and cycling of mercury on a global scale, it is possible that man’s major impact on the biogeochem-

* See Pacyna, Figure 7.1 for estimates of Hg fluxes for natural versus anthropogenic sources for the USA, Europe and the USSR (Editors).
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The chemical cycle is of an indirect nature. That is, barring any major changes in anthropogenic emission trends, it is not the direct release of mercury which is critical, but the way in which man's other emissions may alter the global cycle. For example, our greatest influence on the cycle could be the increased emissions of oxidants and oxidant precursors which are so important in the cycle of atmospheric S and N (NAS, 1983). The existence of various theorized soluble vapor phase species is dependent on oxidation of HgO in the atmosphere, possibly by ozone or peroxides (Lindqvist et al., 1984). One recent report suggests that the lifetime of mercury in the presence of elevated levels of O₃ is on the order of hours (Stevens et al., 1982), instead of the commonly accepted period of years (Lindqvist et al., 1984).

**QUESTION 3**

What are the current trends in mercury use on a global scale?

We need to document changes in both utilization and emission resulting from reductions in the use of mercury slimicides, Hg cell chloralkali processes, and Hg seed dressings. We may be biased in our view of the current global 'situation' by looking only at the technologically developed nations and not by also considering mercury use in developing countries. It is possible that there has been a shift in its industrial and agricultural use from the northern to the southern hemisphere. The technologies that caused problems in the developed world, such as the chloralkali and polyvinyl chloride industries, and the use of organic mercurials in biocides and slimicides are apparently being transferred to the developing countries in the southern hemisphere and tropics. However, there is only anecdotal evidence of such changes which would suggest a shift in environmental releases from the northern to the southern hemisphere. Global data to confirm these shifts are needed.

Since we suspect that man's releases of mercury to the atmosphere actually dwarf his releases directly to aquatic environments, we need to determine if a recent emission inventory exists. It appears that the newest data available is about 1975 for the USA and much of the rest of the world, although Swedish data exists for 1982. It is entirely possible that future trends will result in new problems of marine mercury pollution in the southern hemisphere. Perhaps a monitoring activity or other surveillance program of the mussel watch type (Goldberg et al., 1978) might provide answers to these questions.

Research Proposal: The UNEP should be requested to initiate a program to obtain and publish statistics on mercury production, utilization and environmental disposal for the world scientific community.
QUESTION 4

What is the speciation of mercury in the atmosphere and how critical is it to the geochemical cycle?

Unlike the other metals considered in this SCOPE report, mercury exists in both industrial and natural emissions and in ambient air primarily as a vapor species, which is conducive to long range transport and deposition on a global scale. Common wisdom suggests that the major vapor species in air is elemental mercury (Hg⁰). However, most measurements rely on various techniques which result in an operational definition of the real nature of the speciation of this vapor (Schroeder, 1982).

Knowledge of this speciation is critical to our understanding of removal processes, and perhaps more importantly, to any predictions of the influence of changing atmospheric chemistry on mercury deposition (see Question 2 above). Wet deposition is considered to be the major removal process of atmospheric mercury on a global scale (Lindqvist et al., 1984), although the global rates are highly speculative. However, since Hg⁰ is sparingly soluble in water, typical atmospheric levels can in no way account for measured concentrations in rainwater (Fogg and Fitzgerald, 1979). Some have theorized the existence of highly soluble vapor phase compounds of mercury which are formed in ambient air but which are rapidly scavenged by precipitation (Lindqvist et al., 1984). However, none of these compounds has yet been positively identified and it has been suggested that there is sufficient particle-borne soluble mercury to account for mercury in precipitation.

Analytical sampling trains need to be developed to accurately determine the true speciation of mercury vapor not only in ambient air and precipitation but in actively precipitating systems (in-cloud air) to determine the forms actually being scavenged by the raindrops. These speciation studies need to concern both chemical and physical speciation using methods validated under controlled laboratory conditions. For a review of such methods see Schroeder and Jackson (1983) and Brosset (1981, 1983, 1984).

As uncertain are our estimates of wet deposition, those for dry deposition are far worse, ranging over orders of magnitude (Lindberg, this volume, Ch. 8). Particle dry deposition can be theoretically estimated and perhaps measured in some cases; however, very little data exist with which to assess dry deposition of the largest atmospheric mercury pool, the vapor species. Deposition velocities with which to estimate global fluxes do not exist. However, some crop plants are known to absorb Hg⁰ directly from the air in a stomatal process (Browne and Fang, 1978), and even the limited solubility of Hg⁰ in seawater suggests some vapor dry deposition may occur to the sea surface. Plant physiologists have long had the ability to measure uptake of various vapors by foliage using chamber experiments (e.g. Hill, 1971). This
expertise needs to be applied to mercury vapor uptake studies to determine the potential importance of this pathway. These data should then be used to estimate the extent to which direct vapour uptake by forests could be increasing the mercury burden in remote watersheds (see Question 7). If trees scavenge mercury vapor to the same extent as \( \text{SO}_2 \), \( \text{HNO}_3 \) or other major vapor species, it is possible that the commonly used bulk deposition methods of estimating mercury deposition to forests could significantly underestimate the true total input to potentially sensitive environments. One could effectively argue that atmospheric speciation is unimportant to the at-risk population which, in every case of which we are aware, is exposed directly to methylmercury in fish. However, because of the potential for increased deposition if atmospheric mercury were transformed to more soluble species, we recommend that major physical and chemical speciation studies of atmospheric Hg be instituted as soon as possible.

**QUESTION 5**

Can we trust the published data on mercury in the environment? If so, how far back in time?

The scientific community needs to develop and implement quality assurance plans and protocols for sampling, preservation, and analysis of mercury samples to assure accurate data for establishment of spatial and temporal trends and for input to new models.

The assemblage of the data base about mercury in the environment, about the natural and anthropogenic source terms, and about the reaction with the living system must be carried out with quality control procedures. The protocols for sampling, preservation, analysis and interpretation should be developed to satisfy totally the needs of the designated problem. Primary standards and interlaboratory comparison exercises are an essential part of the analytical procedures. Appropriate statistical analysis should be used to validate the data.

We recognize that recent intercalibration exercises of mercury analyses in sea water have demonstrated inadequacy in sampling and/or analytical techniques (Olaffson, 1982). We propose the formulation of primary reference standards for water, sediments and biological materials and extended intercalibration exercises involving these media.

**Research Proposal:** A workshop should be convened to develop and publish protocols for the sampling, storage, analysis and interpretation strategies to assess mercury contamination in the environment. Further, means to produce and distribute primary reference materials for mercury should be developed at the workshop.
Has the question of where methylation occurs ever been satisfactorily answered? It has been reported to occur in sediments, soils (by biotic and abiotic processes), and in fish (gut and skin). Demethylation has been reported for marine and terrestrial mammals. Has the puzzle of the lack of methylmercury in surface water been laid to rest? While these are interesting academic questions, do they matter to the ultimate receptor or next host organism, or is the only important point that the predator is ingesting methylmercury?

The first demonstrations of biological methylation of mercury were for sediments (Jensen and Jernelov, 1969; Olson and Cooper, 1976). The methylation process was shown by Wood et al. (1968) to be bacteriologically mediated, and the process of methylation is dependent upon not only the metabolic activity of bacteria and the concentration of inorganic mercury, but also on pH, redox potential, organic substrate and temperature (Beijer and Jernelov, 1979). Some experiments showed that anaerobic conditions promoted methylmercury production (Olson and Cooper, 1976) while others, for example, Bisogni and Lawrence, (1975), found that aerobic conditions were more favorable. Chemical methylation is also possible (Jewett and Brinkman, 1974; Carty and Malone, 1979), and it is probable that both chemical and biological processes are involved in the methylation of mercury.

More recent studies have indicated that methylation can also occur in the water column in freshwater (Furutani and Rudd, 1980) as well as marine systems (Topping and Davis, 1981). Methylation has also been shown to occur in fish intestines (Rudd et al., 1980) and on fish skin (Rudd, personal communication, 1984). In general the source of the mercury is the sediment or eroding material entering the water body. The relative importance of methylation in the abiotic and biotic components, respectively, is difficult to determine. Levels of total mercury in water are extremely low (see Question 7), and the percentage of methylmercury to total mercury is about 30 in freshwaters (Kudo et al., 1982; Brosset, 1981).

Microbial demethylation has also been demonstrated in mercury polluted sediments (Jernelov et al., 1975) and is considered to be of fairly widespread and frequent occurrence. In fact, mercury methylation rates measured in complex systems are net methylation rates, resulting from a combination of both processes. Indirect evidence also exists for the occurrence of a demethylation in animals, as shown by a lower ratio of methyl to total mercury in liver and kidney compared with muscle. This type of evidence has been shown for marine mammals (Jones et al., 1979; Gaskin et al., 1976), for birds (Norheim and Froslie, 1978; Stoneborner and Harrison, 1981), mink (Jernelov et al., 1976), otter (Wren et al., 1980) and fish (Burrows and Krenkel, 1973).
In the opinion of the Working Group it is unlikely that a satisfactory elucidation of all the sites and mechanisms of methylation and demethylation, particularly the quantitative aspect, will be made for natural systems for a considerable period of time. Mercury speciation in water is particularly difficult to determine. At this time, it seems more pertinent to concentrate efforts on factors which predispose the carnivorous fish to accumulate methylmercury. These factors may include deposition from the atmosphere, and physiochemical variables which are known to be related to fish uptake and loss rate of mercury, both organic and inorganic. A mathematical modelling approach is likely to be promising, following a rigorously controlled collection of information in fish mercury by a standardized protocol for collection and measurement.

**QUESTION 7**

What is the mercury/rain/fish link? Is there direct evidence to support any of the hypotheses put forward to explain the trend of increased mercury levels in fish from remote lakes with low alkalinities? This relationship has been reported in several studies in Scandinavia and North America, but is it a recent phenomenon, or merely a recently discovered natural relationship? Do we have the historical data to test these hypotheses? If not, how can we design experiments or surveys to test them?

Within a given geographic area, and for surface waters which have no known point sources of mercury, the concentrations of the metal in fish muscle (of specified species and standard age) often show a statistically significant relationship with chemical and physical properties of the lake or its watershed. Most frequently repeated is the negative correlation between surface water pH and mercury in muscle (Suns et al., 1980; Håkansson, 1980; Wiener, 1983; Wren and McCrimmon, 1983). Fish mercury has also been shown to be negatively correlated with alkalinity (Suns et al., 1980) and negatively correlated with calcium concentration (Wren and McCrimmon, 1983). A positive correlation was shown by Suns et al. (1980) between mercury in perch and the watershed area/lake volume ratio.

Fish muscle containing > 1.0 μg Hg/gm (wet weight) is considered unfit for human consumption in the USA and Sweden (Piotrowski and Inskip, 1981). In Canada the recommended limit is 0.5 μg/gm. Mercury in fish, although measured as total Hg for routine determinations, is generally > 80% methyl mercury (e.g. Huckabee et al., 1979), whose concentrations increase with body size (Phillips and Buhler, 1978; Huckabee et al., 1979). Although the site of methylation is unclear, the process can be bacterially or chemically mediated. While fish cannot methylate in vivo, gut and skin bacteria can methylate mercury.
Total (and inferred methyl) mercury concentrations in edible flesh of piscivorous fish inhabiting waters of low alkalinity and pH < 6.0 frequently exceeds 1.0 μg/gm wet weight, thus creating a potential health risk. Furthermore, in Sweden and Canada a number of lakes have been ‘blacklisted’ with the issue of a warning not to consume fish. This has identified a problem in areas which have tourism and recreational fishing as an economic base.

Without historical data, we have no means to determine whether these elevations of mercury in fish from remote areas represent a recent phenomenon, or whether this is an entirely natural relationship. This means that we cannot determine from existing data whether the depression of lake pH from acidic deposition, with or without increased deposition of mercury, has increased the occurrence of mercury contamination in the areas of concern.

The major hypotheses concerning the fish mercury-lake pH relationship include the following:

(i) Bacterial methylation is enhanced by low pH. Recent studies on methylation in sediments by Ramlal et al. (1985) and by Steffan and Winfrey (1984) show clearly that the reverse is true, that is that low pH significantly decreases bacterial methylation rates in sediments.

(ii) Given a specific concentration of methyl mercury, the mono-methyl mercury-dimethyl mercury equilibrium is pH dependent, with mono-methyl mercury formation being favored at low pH (Jensen and Jernelov, 1969; Atmosphere-Biosphere Report 1981).

This has not been verified experimentally, but still remains as a viable hypothesis. Determination of mercury species in waters where total Hg does not exceed 10 ng/litre cannot be made directly. Miller and Akagi (1979) offer an alternative suggestion: the partitioning of mercury between sediment and water is pH dependent. They show that a decrease of 1 pH unit (for sandy sediment systems) or of 2 pH units (for organic sediments) doubled the amount of methyl mercury in the water column while the total amount of methyl mercury in the system remained unchanged.

(iii) Increased deposition of mercury from long range transport has occurred in conjunction with increased acid deposition, resulting in increased mercury in fish.

Total atmospheric deposition of mercury to a lake and its watershed is difficult to estimate for technical reasons (see Question 4). Dry deposition is particularly hard to measure, while the proportion of mercury re-emitted to the atmosphere is unknown. Mercury deposited onto land is likely to be bound in the organic layers of forest soils, and a considerable amount of this organic material will eventually find its way
to the lake via runoff and leaching. Acidic precipitation could enhance the transport from watershed to lake, and possibly from atmosphere to watershed/lake system as well. Lindqvist et al. (1984) discuss this source of mercury to lakes, and give the range of 20–25 gm/year/km for mercury in runoff. Lindberg (1980) has also hypothesized that rain acidification could directly enhance the wet deposition of mercury to such lakes. This is because solubility of atmospheric mercury may be influenced by the acidity of the scavenging raindrop (see also Lindberg, this volume, Ch. 8).

It is possible to make rough calculations of a mercury budget for a lake, as done by Lindqvist et al. (1984) for southern Sweden. These authors suggest that increased mercury loading is at least partially responsible for increased fish mercury observed in remote lakes in south and central Sweden. Total mercury in water is consistently low (0.010–0.040 µg/litre) for the types of lakes under discussion; sediment mercury, while quite low (100–500 µg/kg), has increased in south and central Sweden over the last century.

Methylation of mercury (Question 6) can occur in water, sediment or in the watershed. Recent investigations (Hultberg, personal communication) suggest that non-biological methylation of mercury in the watershed stimulated by metals such as iron and manganese may result in a major contribution of the mercury into the lake in a bioavailable form. Acidic deposition would enhance this process.

(iv) Nutrients, organic content or general lake trophic status influence fish mercury indirectly.

Other factors related to fish mercury include size of fish, which may be related to lake pH in that stressed fish may grow more slowly, and trophic status of the water, the effect of which is probably indirect and is still controversial. Studies by Beijer and Jernelov (1979) suggested that fish from lakes of higher trophic status had less mercury, while recent evidence (discussed in Wren, 1983) tends to show the reverse effect. Although lake trophic status, alkalinity and pH tend to be correlated, this variable may nevertheless introduce a confounding effect in some data sets.

**QUESTION 8**

Do we have a significant mercury legacy in stored solid wastes? If all active emissions were stopped today, how much anthropogenic mercury would continue to be released from orphan wastes by volatilization or leaching?

As a result of legislated regulations the industrial emissions of mercury both to surface waters and to the atmosphere have been identified and consider-
ably reduced. However, the processes of reduction in most cases resulted in a shift from direct emissions of Hg vapor or divalent Hg into air and water to emissions into landfills in the form of solid or slurried wastes containing mercury in various forms. For example, nearly 90% of the current mercury use by the chloralkali industry in the United States is deposited in waste ponds (> $10^5$ kg/yr) (Harriss and Hohenemser, 1978). Conversion of existing Hg-cell plants to non-Hg processes suggests that existing waste ponds will be the major source of mercury release from this industry in the future unless these wastes are stored such that volatilization and leaching losses do not occur. The loss of mercury from one such orphan waste site in the United States was estimated to be 75 kg/yr, 50% to water and 50% to air, which is comparable with losses from active Swedish plants (Lindberg and Turner, 1977). In addition, air concentrations of mercury vapor near this site were close to the USA guidelines for ambient air, and concentrations of methyl mercury in downstream fish exceeded WHO guidelines (Hildebrand et al., 1980a).

Because of the massive amounts of these wastes produced (> $10^9$ kg/yr in the USA alone), the volatility and solubility of mercury in such wastes, and the potential residence time of mercury in large waste deposits (hundreds of years), these areas must be located and monitored. An additional but similar problem involves often extensive areas of sediment in lakes and rivers which were at one time contaminated by local industrial activities. Examples exist in many countries (Nriagu, 1979). Although direct emissions to these water bodies have ceased, the sediments represent a potential long term source of mercury to the aquatic system, particularly if the sediments are dredged or otherwise resuspended (e.g., Lindberg and Harriss, 1977).

Mercury bioconcentration and methylation in aquatic food chains (see Questions 1 and 6) indicates that fish consumption is a major mercury pathway to man. This suggests that any potential large scale or long term releases of mercury to surface waters from contaminated waste storage areas or sediments must be closely monitored. As a first step we recommend that all nations inventory the locations of major mercury users, both past and present, to prevent the loss of such critical information upon cessation of active production at these sites. All too often hazardous waste sites are forgotten until years later when it is too late or too costly to remedy the problem. Once these sites have been identified and located, simple but routine monitoring programs should be instituted, concentrating on key indicator organisms such as top carnivore fish or mammals.

**QUESTION 9**

What is the true nature of the Hg-Se relationship recently described? Selenium is said to decrease uptake and toxicity of mercury to certain organisms.
What are the mechanisms and are we simply trading one toxic metal problem for another?

Several investigators have demonstrated that the administration of selenium together with methylmercury reduces the toxicity of the mercury (NRCC 1979). These effects have been demonstrated experimentally both in vitro and in vivo for a number of laboratory animals. There is also some suggestion that selenium may provide protection against methylmercury toxicity in marine mammals which accumulate high levels of mercury. A 1:1 Hg : Se molar ratio in tissues has previously been reported (Koeman et al., 1975). However, other studies show that the ratio of Hg : Se is not always 1:1 (molar) in these organisms (Koeman et al., 1975) and that an efficient demethylation process occurs in the target organs of many marine mammals and birds. This also affords protection against MeHg toxicity (Norheim and Froslie, 1978; Gaskin et al., 1979).

Despite the amount of research and discussion of the subject, the mechanisms through which selenium provides protection against methylmercury poisoning are far from clear (Skerfving, 1978). The mechanism of action of selenium does not appear to involve a decrease in tissue Hg levels, but may involve free radicals formed by the breakdown of MeHg molecules (Ganther, 1978).

No recent advances have contributed to unraveling the interactions of selenium and mercury. The status quo can best be expressed from the review of Piotrowski and Inskip (1981): 'the present-day recognition of selenium's influence on the metabolism of MeHg is of limited use in the understanding of mechanisms involved in the Se: MeHg interactions'.

Other factors have also been shown experimentally to modify the toxic effects of Hg. For example, Vitamin E delayed the onset of methylmercury poisoning in animals (Welsh and Soares, 1976), while Gilbert et al. (1983) demonstrated in vitro that Vitamin E has protective potential against the genotoxicity of methylmercury. Yamane et al. (1976) observed that Zn acetate modified the toxicity of methylmercury to rats, although other researchers have not observed similar effects (Johnson and Pond, 1974). Despite these studies using culture tissues or laboratory animals, the interactive effects of diet, nutrition, disease and natural stresses on the uptake and toxicity of methylmercury to populations of wild organisms and to humans remains unknown.

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