FROM THE ARCHIVES

Assessment of Mercury in the Environment: A Report (1978)

Pages
196

Size
5 x 9

ISBN
0309027365

Panel on Mercury; Coordinating Committee for Scientific and Technical Assessments of Environmental Pollutants; Environmental Studies Board; Commission on Natural Resources; National Research Council

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An Assessment of Mercury in the Environment

A report prepared by the
Panel on Mercury
of the Coordinating Committee for Scientific and
Technical Assessments of Environmental Pollutants

·Environmental Studies Board
·Commission on Natural Resources
National Research Council

iv

CONTENTS*

CHAPTER 6: EFFECTS ON HUMAN HEALTH   88

*The different units used for expressing concentrations
of mercury in various media are so diverse that
confusion sometimes results. The Panel on Mercury has
chosen to use the official metric system of units
uniformly throughout this report. To facilitate
comparisons, however, a table of units is presented in
Appendix A.

THE STUDY ON SCIENTIFIC AND TECHNICAL ASSESSMENTS
OF ENVIRONMENTAL POLLUTANTS

In early 1975 the U.S. Environmental Protection Agency (EPA) approached the National Research Council of the National Academy of Sciences to request a series of comprehensive scientific and technical assessment documents on selected multimedia environmental pollutants. These documents would be used by EPA as a basis for preparing Scientific and Technical Assessment Reports (STARs), which in turn would be used as the scientific and technical basis for possible regulatory action on these pollutants. EPA was anxious that these background documents use an environmental "mass balance" approach, i.e., that they attempt to account for the sources, sinks, and receptors for the pollutant as it moves through the environment. The initial two reports (Nitrates and Nonfluorinated Halomethanes) were also to explore the technology for controlling the pollutants and the costs and benefits of available means of control, whereas the subsequent two (Mercury and Kepone/Mirex/Hexachlorocyclopentadiene) were not.

Within the NRC, responsibility for the study was assigned to the Environmental Studies Board of the Commission on Natural Resources. When it agreed to undertake the study, the Environmental Studies Board identified two distinct objectives. The first was to conduct a series of assessments of specific pollutants, as required by EPA. The second was to draw upon the experience gained from conducting a limited number of such assessments, in order to address the broader methodological problem of how such assessments should be done and how best to use the limited resources of scientific expertise for environmental issues to meet EPA's expanding need for independent, critical scientific evaluations of pollutants.

A Coordinating Committee for Scientific and Technical Assessments of Environmental Pollutants (STAEP) was appointed to oversee panels, which would conduct the

assessments of specific pollutants, and to make
recommendations regarding the best methodology for
producing such assessments. The pollutants to be
studied were mutually agreed upon by the EPA and the
NRC; pollutants were chosen that posed particularly
complicated or difficult problems of assessment, given
current scientific and technical knowledge.

In September 1976, to help meet the requirements of a
consent agreement for documents on health and ecological
effects, EPA requested additional assessment reports
under the STAEP study program. The NRC agreed to
conduct two additional studies and, by mutual agreement,
mercury was chosen as the topic for one. The additional
studies differed from the initial two in that (1) the
performance period was shortened and the scope
consequently narrowed, and (2) the reports were to be
based primarily on a literature survey and review
prepared by another contractor for EPA's Review of
Environmental Effects of Pollutants (REEP) program.
Undertaking these additional studies would benefit EPA
by providing the best available expert judgment, and the
STAEP Coordinating Committee would gain the experience
of another method by which assessments could be
performed and would have a broader base upon which to
report on the methodology of assessing pollutants.

THE PANEL ON MERCURY

STAEP's Panel on Mercury was appointed in November
1976. The panel was charged to provide EPA with a
critical assessment of the available scientific and
technical knowledge on the human health and ecological
effects of mercury as an environmental pollutant. In
addition to assessing the ecological and environmental
health hazards of mercury, the study was to identify
areas in which the scientific evidence is uncertain or
too inconclusive to arrive at an assessment, and to
suggest areas where additional research is needed to
provide a sounder basis for future regulatory action by
EPA.

The panel conducted its study through a series of five
1-day working sessions from December 1976 through June
1977, including a public information-gathering session
where scientific and technical information from the
interested public was solicited and presented to the
panel. At its first meeting the panel determined that
the literature review (REEP-Mercury) provided by EPA was
not satisfactory as a primary reference for their work.
This unexpectedly expanded the panel's work to include
an extensive search and review of the general scientific
literature by the panel members and NRC staff. In the

FINDINGS AND OVERVIEW

This study responds to a request by EPA for a critical
assessment of the most up-to-date scientific and
technical knowledge on the effects of mercury as an
environmental pollutant. To base this assessment on the
most complete, recent information available, each member
of the National Research Council's Panel on Mercury
updated one area of research to include the most recent
publications in two basic categories: the impact of
mercury on the environment and on human beings. The
principal findings arising from this study are
summarized below, followed by an overview that
introduces the main themes to be found in the body of
the report. Documentation for the findings can be found
in the body of the report as noted parenthetically after
each finding.

FINDINGS

General

1. Mercury compounds have no known normal metabolic
function, and their presence in the cells of living
organisms, including human beings, represents
contamination from natural and anthropogenic sources.
In view of the toxicity of mercury and the inability of
researchers to specify the threshold levels of toxic
effects on the basis of present knowledge, all such
contamination must be regarded as undesirable and
potentially hazardous. (See Chapter 6.)

Global Cycles

2. Although these estimates are tentative, the "pre-
man" and present-day global mercury cycle models
presented in this report (see Chapter 1, section
entitled The Natural Mercury Cycle) support the
following conclusions:

1

● The atmosphere plays an important role in the mobilization of mercury. From 25 to 30 percent of the total atmospheric mercury burden is anthropogenic. The residence time for mercury in the atmosphere is estimated to be 11 days.

● The mercury burden of lakes and rivers has increased 2 to 4 times pre-man levels.

● Total increases in oceanic mercury concentrations have been negligible. The residence time for oceanic mercury is 3200 yr.

● The mercury burden of freshwater and estuarine sediments has increased 2 to 5 times pre-man levels. The residence time for mercury in oceanic sediments is 2.5×10^8 yr.

● The mercury content of soil appears to have increased by about 0.02 percent. The residence time for mercury in soils is 1000 yr.

3. Flux rates between continental, oceanic shelf, and open ocean air masses and between shelf and open ocean water masses cannot be evaluated with current information. Models do not resolve the question of whether increases in mercury levels in watercourses, air, and soil have also elevated mercury levels in freshwater and land biota. The biological cycle of mercury is delicately balanced, however, and small perturbations in input rates and the chemical form of mercury can result in increased methylation rates in sensitive systems. (See Chapter 1, sections entitled The Natural Mercury Cycle and The Global Cycle of Methylmercury.)

Transformation and Uptake in the Environment

4. Identification of "hot spots" of anthropogenic mercury emissions to air, water, and land is valuable for establishing monitoring and health evaluation programs; it is not yet clear how valuable this identification is for projecting the mercury burden of aquatic and terrestrial biota and of the human population. Since the behavior of mercury is a function of its chemical form, data on emissions can be evaluated together with prevailing physical, geological, and chemical regimes for each region to identify sensitive areas. The release of mercury to the environment from coal-fired power plants, mining, and smelting operations is of special concern since these sources are currently uncontrolled and the use of coal is expected to increase significantly. (See Chapter 1, section entitled Man-Made Mercury Emissions in the United States.)

5. Mercury that is not recycled by industrial and other users is released into the environmental cycle and

becomes available for potential methylation. If the element is not recycled, conversion to its original geochemical form (for example, mercury sulfide) and burial would be the best safeguard against transformation into methylmercury. (See Chapter 2, section entitled Mercury in the Lithosphere.)

6. Once the sources of anthropogenic mercury to aquatic ecosystems are removed, the mercury content of the sediments and aquatic organisms appears to decrease slowly. The rate is a function of (a) the degree of contamination, (b) the chemical form, (c) the physical-chemical conditions of the system, (d) the hydraulic turnover time, and (e) the rate at which the mercury is either removed (bedload transport) or buried in the sediments. (See Chapter 2, section entitled Mercury in Natural Waters.)

7. Measurements of mercury levels in water and sediments, though useful, are not sufficient to ascertain the rates of methylation or uptake in biota. Methylation rates in ecosystems are a function of the mercury burden, bacterial population, nutrient loadings, pH and redox condition, suspended sediment load, sedimentation rates, and other physiochemical conditions. Variations in methylation rates as a function of each of these parameters in situ remain unknown. However, cutting back on the point discharges of soluble inorganic mercury compounds can significantly affect methylmercury synthesis in polluted areas. (See Chapter 1, sections entitled The Global Cycle of Methylmercury and Man-Made Mercury Emissions in the United States; and Chapter 3, Summary and Conclusions.)

8. For chemically sensitive waterways such as poorly buffered lakes, the combined effects of acid precipitation and increased emissions of mercury to the atmosphere with subsequent deposition may pose a serious threat if the correct biomethylation conditions are met because more methylmercury would be available to the biota. (See Chapter 1, section entitled Man-Made Mercury Emissions in the United States.)

9. There is evidence that the bioaccumulation of methylmercury into the tissues of higher organisms (e.g., fish) is probably diffusion controlled. The rates for bioaccumulation are so rapid (20×10^{-9} s for methylmercury chloride to diffuse through cell membranes into cells) that even low concentrations of methylmercury in water lead to elevated concentrations in fish. (See Chapter 3, sections entitled Kinetics of Methylmercury Biosynthesis and Methylmercury Diffusion into Cells and Summary and Conclusions.)

10. In highly contaminated aquatic systems fish and other aquatic organisms may be at risk because they efficiently bioconcentrate mercury; fish-eating birds

and mammals may also be at risk. In areas without high natural or anthropogenic pollution, most animals do not accumulate high levels of mercury and therefore do not appear to be immediately at risk. However, the sublethal effects on wildlife of long-term exposure to low levels of methylmercury are currently undefined. (See Chapter 4.)

Measurement and Monitoring

11. Analytical methods for measuring total mercury in most biospheric and lithospheric samples appear to be adequate. Measurement of total mercury in the atmosphere and in the hydrosphere poses problems, however, because the different mercury species measured are currently defined by the collection technique and the concentrations encountered are low. Nonetheless, experienced analysts can usually determine total mercury and methylmercury adequately. Strict quality control and frequent interlaboratory comparisons must be an integral part of any mercury analysis program. (See Chapter 2 and Appendix A.)

12. It is difficult to determine the extent to which an aquatic system is polluted by mercury because the processes of methylation and uptake are so complex. At present, the most effective indicator of both the degree of mercury pollution and the potential hazards to humans and wildlife is the mercury content of fish. In using this indicator, factors that bear on the fish mercury content such as age, species, and nutritional habits must be taken into account. (See Chapter 2.)

Human Exposure and Risk: Establishing Guidelines

13. The most direct threat to human health from mercury compounds is from consumption of methylmercury. Therefore, the tolerance, guideline, or acceptable level of mercury in food established for the protection of human health should be set on the basis of the methylmercury content of the food rather than the total mercury content. (See Chapter 5.)

14. By themselves, mercury compounds in the concentrations and forms usually found in the ambient atmosphere and in drinking water do not contribute significantly to mercury intoxication in human beings. The levels of methylmercury in plants are also generally extremely low, and do not contribute significantly to the methylmercury burden in human beings, with the exception of plants grown on contaminated soil or from mercury-treated seed stock. (See Chapter 5, sections

entitled Air and Drinking Water and General Food
Surveys.)
    15.   While all foods contain minute quantities of
mercury, only fish and seafood, and to a lesser extent
meat, present a potentially serious health hazard
because of their relatively high levels of
methylmercury. (See Chapter 5, section entitled General
Food Surveys.)
    16.   The guideline for acceptable levels of mercury
in fish and other seafood is based, at least in part, on
the estimated average rates of daily fish consumption.
Although this protects the average consumer, it does not
take into account the subpopulations that consume large
amounts of fish and seafood or sport fishes from highly
contaminated waterways. (See Chapter 5, sections
entitled Mercury Levels in Fish and Human Uptake of
Mercury in Fish.)
    17.   Preliminary data from animal studies suggest
that selenium has a protective effect against
methylmercury poisoning. However, the biochemical
mechanism for selenium-methylmercury antagonism is not
presently understood. Some information exists on the
selenium-mercury ratio in ocean fish, but similar data
supporting this ratio are scarce for freshwater fish.
Bishop and Boomer (1974) have presented data that
indicate that for freshwater fish there is no selenium-
mercury ratio. Currently, no data from exposed
populations document the protective effects of selenium
in humans. Therefore, present knowledge does not
justify modification of currently accepted guidelines
for total mercury or methylmercury in food on the basis
of selenium content. Further studies are needed to
establish the biochemical role of selenium and other
compounds that may interact with mercury and increase or
decrease its toxicity to living organisms. (See Chapter
5, section entitled The Effects of Selenium on
Methylmercury Toxicity.)

                    Effects on Human Health
        (For a quantitative assessment see Chapter 6.)

    18.   In human populations exposed to toxic doses of
methylmercury, signs and symptoms are dominated by
neurological disturbances. Currently, only relatively
insensitive clinical methods are available to evaluate
the effects of chronic low-level exposure to
methylmercury. Thus, current limits for detecting the
effects of methylmercury in human populations should not
be equated with threshold levels, for other more subtle
effects such as behavioral or intellectual deficits may
not be detectable by the clinical procedures that have

been used. More sensitive objective techniques for
assessing neurotoxic effects of methylmercury need to be
developed to supplement standard clinical neurological
procedures. (See Chapter 6, section entitled Toxic
Effects of Methylmercury in Adult Populations.)

19. Behavioral effects on humans of chronic low-
level exposure to methylmercury are not fully
understood, and information about possible genetic,
reproductive, teratogenic, and carcinogenic effects of
mercury compounds is incomplete and contradictory.
Methylmercury has been reported to be weakly mutagenic
in Drosophila, to reduce fertility in some rodent
species, and to cause chromosome breakage in exposed
human beings. Better data should be developed with
which to evaluate the potential health significance of
such effects. (See Chapter 6.)

20. Human subpopulations may be at increased risk
because of consumption of very large amounts of fish or
other seafood, or sport fishes from highly contaminated
waterways; because of predisposing genetic or
environmental factors; or because of differences in
susceptibility at different developmental stages of the
life-cycle. These subpopulations should be examined
carefully. (See Chapter 6.)

OVERVIEW

The data so far collected on mercury contamination
have affirmed that anthropogenic sources are raising the
levels in air, soil, freshwater lakes and streams, and
ocean estuaries if not in the oceans themselves. These
increases are being methylated and translocated through
the food chain. While the small amounts of mercury in
most foods and larger concentrations from industrial
exposures affect a few people, the greatest hazard from
environmental exposures is to hypersensitive individuals
who consume excessive quantities of fish or other
seafood contaminated with methylmercury. The symptoms
of acute poisoning have been documented in previous
epidemics, and the precision and accuracy of analytical
techniques for measuring organic and inorganic mercury
in biological samples continue to improve. However, the
data are still incomplete and the medical and technical
proficiency are too limited to assess conclusively the
effect of chronic exposure to low levels of mercury
pollution on human beings as well as lower orders of the
food chain and the environment. The mercury problem,
therefore, clearly needs to be reassessed periodically
to take advantage of new data and of the continuing
refinement of chemical and analytical techniques. The
approach of the present reassessment has been to review

the global occurrence of mercury and the mechanisms by
which it is transported and accumulated (Chapters 1-3)
and then to discuss effects--first on the ecology
(Chapter 4) and finally on human health (Chapters 5 and
6). The authors have attempted to be consistent in
reporting concentrations for biological samples on a
wet-weight basis throughout the report, unless otherwise
noted.

Occurrence and Bioaccumulation of Mercury

Mercury Cycles

The clearest conclusion to emerge from the panel's
review of the literature on the global cycle of mercury
is that substantial evidence remains to be collected
before definitive appraisals are possible. At this
time, the transport of mercury in the global cycle and
its sinks in the environment have primarily been
described by means of mathematical models based on
assumptions that have become questionable in the light
of recent data. For example, calculations of man's
influence on the global mercury cycle, which were based
on earlier measurements of mercury concentrations in
Greenland ice cores over time, need to be reevaluated.
Based on revised assumptions, a comprehensive synthesis
of old and new information on the global mercury cycle
in a "pre-man" and a present-day framework is
presented in Chapter 1.

New data introduced in Chapters 1 and 2 indicate that
the total atmospheric mercury burden is probably less
than previously estimated, and that approximately 25 to
30 percent is the result of man-made emissions. In the
United States the annual consumer commercial and
industrial mercury consumption for 1973 is estimated to
be 1.9×10^9 g. The total environmental losses
resulting from these activities, as well as from mining
and fossil fuel burning, is estimated at 1.5×10^9 g.
The range varies widely from one state to another,
depending on the population density.

Estimates discussed in Chapter 2 of the annual natural
flux of mercury to the atmosphere range from 25,000 to
30,000 metric tons (1 ton = 10^6 g), with elemental
mercury the primary form that cycles from the
earth's surface to the atmosphere. Mercury's residence
time in the atmosphere is shorter than for any global
compartment, and is estimated at approximately 11 days
in this study. Elemental mercury is removed from the
atmosphere by rain and snow; and dry removal may also
occur. In the lithosphere, much of the mercury in

mineralized areas is bound as its highly insoluble sulfide, cinnabar (ore).

Much has been learned over the past two decades about the many kinetic factors that can change the speciation of mercury in the aquatic environment. Calculations show that ionic divalent mercury is likely to be formed in well-oxygenated waters. This mercury species can undergo several important reactions: (1) formation of the highly insoluble mercury sulfide under anaerobic conditions, (2) reduction to metallic mercury vapor (degassing) under appropriate conditions, and (3) conversion to alkylmercury (methylmercury and dimethylmercury) compounds. Formation of monomethylmercury occurs in sediments under aerobic and anaerobic conditions and is greatly favored by low pH (optimum pH 6.0). This is a key reaction since it greatly increases the ability of mercury to cross biological membranes, and methylmercury is rapidly removed from the aquatic environment through bioconcentration. Although many of the details of the described cycle are as yet conjectural, the key elements discussed are supported by current knowledge of mercury speciation.

We do not yet have enough information to evaluate flux rates for continental, oceanic shelf, and open ocean air masses, or between shelf and open ocean water masses; nor has the flux of alkylated mercury in natural conditions been determined although order-of-magnitude values are estimated in Chapter 1. We do, however, know that mercury levels in watercourses, air, and soil are increasing. Whether these increases have also elevated the levels in biological specimens cannot reliably be determined using models, but thermodynamic calculations predict that under favorable environmental conditions biological and chemical methylation of mercury will occur, and the high levels of methylmercury found in aquatic biota indicate that even extremely low concentrations bioaccumulate rapidly.

Bioaccumulation Processes

Chapter 3 assesses the most recent research on the chemical and biochemical mechanisms for methylation and demethylation. Biomethylation is facilitated by three methylating coenzymes in biological systems, but only methyl-B_{12} is capable of methylating soluble inorganic mercury salts to methylmercury and dimethylmercury under both aerobic and anaerobic conditions. The rate of methylmercury synthesis is determined by the available concentrations of soluble mercuric ion species and methyl-B_{12} compounds as well as by the nature of the

microbial community. The presence of demethylating organisms will allow a steady-state concentration of methylmercury to build up in an ecosystem, but the concentration would be lower than if they were less abundant. However, if, as the recent evidence indicates, the bioaccumulation of methylmercury into the tissues of higher organisms such as fish is diffusion controlled and very rapid, then even low concentrations in water can lead to elevated concentrations in fish.

Effects of Mercury on the Environment

Evidence presented in Chapter 4 supports the contention that, despite the often simultaneous processes of methylation and demethylation, fish and shellfish concentrate high levels of primarily methylmercury (usually 90 percent or more in that form) from the small quantities in the waterways. In contrast, non-fish-eating animals and birds usually concentrate less than 0.02 $\mu g/g$. In most unpolluted fresh waters, the top predatory fish such as bass, pike, and walleye may have natural levels of methylmercury ranging from 0.4 to 1.0 $\mu g/g$ (wet weight) although concentrations as high as 2.0 $\mu g/g$ may occur, whereas in highly contaminated fresh waters, they may average 10 $\mu g/g$, with some fish exceeding 24 $\mu g/g$. In general the degree to which the fresh water is contaminated is the principal determinant of mercury concentrations in fish. Nonetheless, age, weight, species, metabolic rate, and region of habitation also signficantly affect mercury levels.

Marine fish also concentrate primarily methylmercury. However, unlike freshwater fish, the levels are usually below 0.01 $\mu g/g$ except for large carnivorous fish such as tuna and swordfish. They may have mercury levels that range from 0.2 to 1.5 $\mu g/g$. The quantity of mercury absorbed may be influenced by the position the species occupies in the food chain, the amount of salt in the seawater, as well as by the other factors described for freshwater fish. In marine fish the presence of selenium in levels equal to or exceeding the mercury content of the fish may reduce the toxic effects of methylmercury. For freshwater fish, the selenium levels may be too low to achieve this effect. High concentrations of mercury and selenium have been found together in the livers and brains of apparently healthy sea mammals; and the preserved umbilical cords of people at Minamata who were not affected by mercury poisoning had a 1:1 ratio of mercury and selenium. The mechanism of the protective action is not clear. The presence of selenium appears to immobilize the methylmercury

compound but does not appear to speed elimination of mercury from the body. More studies of selenium should be conducted, and other compounds are also being investigated for mutual antagonism toward methylmercury.

Whether the levels of mercury in ocean fish are rising because of contamination cannot be determined because the historical data are insufficient to make accurate comparisons. However, evidence does show that fish from contaminated fresh waters may concentrate enough mercury to place local human populations at risk.

As the element concentrates up the food chain, it may affect the growth, reproduction, and behavior of organisms. The few data available indicate that most plants and other forms of life contain naturally occurring traces of mercury. Mercury poses a potential threat to the bottom of the food chain when it retards the growth of algae and zooplankton. Further up the food chain, insects and other invertebrates have a wide range of tolerances for various mercury compounds and concentrations.

Birds that prey on contaminated aquatic organisms such as fish also have high levels of methylmercury. Their food preferences and habitats, including migratory patterns, are the most important determinants of mercury concentrations. Life spans also have an effect: those that live longer have more time to concentrate mercury. In some birds the reproductive rate is affected.

Among larger vertebrates, premature births have increased among California sea lions since 1968, perhaps because of high DDT and PCB levels as well as mercury. Seals also have shown high mercury levels in other contaminated areas of the world.

Impact on Human Health

The information in the earlier chapters on occurrence of mercury in the environment and its effects on ecosystems forms the basis for the panel's assessment, in Chapters 5 and 6, of the routes by which environmental mercury reaches human beings and of the risks it poses to human health. These chapters also review the likely impact at the various stages of the life cycle, as well as possible genetic, reproductive, teratogenic, and carcinogenic effects.

Determinants of Risk

During the past few years substantial data have been gathered about mercury levels in human food, dietary patterns, and human mercury poisoning. The low levels

of mercury in drinking water pose no threat to health.
Nor do the minute quantities naturally accumulated in
all foods. However, where fish have concentrated
excessive amounts of methylmercury, sensitive
individuals who consume large quantities may be at risk
of poisoning. The groups at risk are indicated by fish
consumption patterns, level of exposure to contaminated
fish, and individual sensitivity.

A major risk is posed for fishermen and especially
native Canadian guides in northwestern Ontario, where
fish with up to 24 µg/g (wet weight) total mercury have
been taken from the heavily contaminated Wabigoon-
English-Winnipeg river system. A potential threat is
also being investigated by the New Jersey Department of
Environmental Protection in Hackensack Meadows.

Fish consumption varies with geographical location,
race, and ethnic origin; and susceptibility to
methylmercury poisoning appears to vary not only by
individual sensitivity but also through the influence of
factors such as nutritional status and concurrent
exposure to other toxicants and infectious agents.
These variables have not been evaluated yet in terms of
possible additive or potentiating effects.

Toxicity to Adults and Dose-Response Relationships

To establish acceptable human and environmental
tolerance levels for mercury, the toxicity to the
central nervous system from chronic, low-level
exposures, as well as other potential adverse genetic,
reproductive, teratogenic, and carcinogenic effects,
must be assessed. Results from study of human exposure
to methylmercury compounds in Japan and Iraq have
contributed significantly to the needed data base. A
Swedish Expert Group made a detailed risk-evaluation of
long-term human exposure to methylmercury compounds in
the heavily exposed Japanese populations. The lowest
observed blood mercury concentrations associated with
the onset of symptoms was 340 ng/ml. Subsequently, with
data from the large epidemic in Iraq, it was possible to
determine the body burdens of methylmercury at the time
of onset of various toxic symptoms and signs. The most
sensitive index of toxicity was determined to be
paraesthesia, which appeared at a mean body burden of
between 25 mg and 40 mg mercury (as methylmercury in a
50-kg individual) depending on the estimated conversion
factor. From these and other studies it appears that
the critical organ system for adult human beings is the
central nervous system.

While much has been learned from such studies,
difficulties in quantifying clinical effects have

prevented the determination of precise dose-response relationships at chronic, low-level exposure. Moreover, other more subtle effects may not be observed with current clinical techniques, and the minimal adult body burden at which methylmercury begins to cause damage to the nervous system must be assumed to be lower than can be demonstrated with the current limits of detection.

Toxic Effects on Developing Fetuses and Other Health Effects

Methylmercury poisoning has a proven toxic effect on developing fetuses; the fetal brain appears to be the most sensitive organ. In Japan prenatally exposed infants were often severely damaged, while their mothers frequently showed few or no symptoms. In Iraq it was possible to document prenatal transplacental exposure by measuring the maternal blood and hair mercury levels. There appeared to be clinically detectable fetal brain damage when the peak maternal hair mercury concentration rose above 100,000 ng/g during pregnancy (approximately equivalent to a blood mercury concentration of 400 ng/ml). Further evaluation of larger numbers of prenatally exposed individuals is likely to reveal that observable effects result from even lower maternal levels. In the exposed Iraqi population 31 percent of women who had apparent symptoms or signs attributable to methylmercury poisoning had maximum hair mercury levels less than 100,000 ng/g. In these individuals it has been suggested that there is a category of methylmercury poisoning in which symptoms such as paraesthesia, headaches, persistant pain, and weakness of the limbs predominate, with little or no evidence of neurological damage on clinical examination, and that these effects may occur with blood mercury levels well below 400 ng/ml.

Methylmercury has been shown to cause congenital malformations in mice and chromosomal abnormalities in rapidly growing plant root cells and lymphocytes cultured in vitro from methylmercury exposed humans. The significance of these observations for human health requires further evaluation. Overall, data on possible genetic, reproductive, carcinogenic, and teratogenic effects of mercury compounds are meager and somewhat contradictory.

Tolerances and Guidelines

Many important questions remain to be answered about
the effects of chronic, low-level human exposure to
mercury, particularly methylmercury. However, because
of the element's toxicity, temporary guidelines have
been established to limit the potential hazard,
particularly from consumption of mercury-contaminated
fish. In the United States the 0.5 μg/g FDA interim
guideline usually only affects such large commercial
species as tuna, halibut, and swordfish. Some states
have imposed restrictions on the quantities of sport
fishes taken from highly polluted waterways (see
Appendix C).

Many other countries have also set recommended
guidelines to limit the consumption of fish contaminated
with methylmercury. These guidelines usually recommend
that pregnant women eat none because of the greater
danger to the fetus. In 1972, the World Health
Organization set a provisional tolerable weekly intake
of 0.3 mg total mercury of which no more than 0.2 mg
should be methylmercury. No firm basis has yet been
established for determining a safe standard for mercury
in foods. (See Appendix B: A Brief Review of FAO/WHO
Deliberations on Setting Acceptable Tolerances for
Mercury Residues in Foods, 1963-1976.)

Analytical Methods

One reason why only tentative or provisional
guidelines have been established is that more refined
analytical techniques are needed to measure selectively
the minute quantities of various inorganic and organic
mercury compounds in biological samples as well as in
water, soil, and air. Earlier analyses generally
measured only total mercury. However, the greater
sensitivity, selectivity, and reliability of current
techniques permit both inorganic and organic compounds
to be measured. In the past, analyses tended to err on
the low rather than on the high side because of lack of
instrumental sensitivity, accidental volatilization of
mercury, and failure to detect organic mercury
compounds. Current analytical methods are more accurate.
Errors are introduced mainly during sample preparation
and secondly by the level of sensitivity of the
instrument.

The major disagreement among researchers is over which
methods are most effective and how best to limit error.
The most common method of mercury analysis is cold vapor
atomic absorption spectrometry, which measures total
mercury; gas chromatography is generally accepted as the

most sensitive method for organomercurial determinations at present. Controversy continues over the relative value of such methods as cold vapor atomic absorption spectroscopy and neutron activation analyses for total mercury determinations.

The use of standard methods and environmental reference samples within a laboratory reduces the likelihood of systematic errors and improves the accuracy of various analytical methods. Atomic absorption equipment is calibrated with synthetic standard samples, and some variabilities among operators and instruments can be controlled by automation. The final results can be checked by interlaboratory comparisons. Results from analyses of blind samples have been compared since the mid-1960s and the discrepancies have decreased. See Appendix A for a fuller discussion of analytical methods.

CHAPTER 1

THE GLOBAL CYCLE OF MERCURY

THE NATURAL MERCURY CYCLE

In considering sources, mechanisms of transport, and sinks of materials in the environment, it is often useful to consider their global cycle. Models can offer an initial insight into the general behavior of an element and often establish a framework for subsequent research. By considering both "pre-man" and present-day elemental global cycles, Garrels et al. (1973) have shown that these models also are helpful for identifying and evaluating anthropogenic impacts on fluxes between different reservoirs in nature.

Insights from Existing Models

Mercury has attracted more attention than many other trace elements in the past few years, and the recent literature contains a large body of information on its distribution in the environment. This information has facilitated the development of several global mercury models (Kothny 1973, Korringa and Hagel 1974, Wollast et al. 1975, Abramovskiy et al. 1975). Van Horn (U.S. EPA 1975b) has also developed a detailed mercury balance for the United States. Although details of calculations and basic assumptions vary among the different authors, several important conclusions have been drawn from these previous models about the behavior of mercury in nature. Some of the more conspicuous can be summarized as follows:

1. The atmosphere plays an important role in the mobilization of mercury. The earth surface-to-atmosphere flux is several times larger than that occurring between continents and oceans.
2. The earth surface-to-atmosphere flux seems to involve mainly elemental mercury vapor, whereas the flux between continents and oceans involves inorganic divalent mercury, much of it associated with dissolved

15

and particulate organic matter. In terms of quantity, alkylated forms of mercury do not contribute appreciably to the global mobilization of the element. A biological cycle that involves the transfer of alkylated forms of mercury does, however, exist. It is discussed in more detail below as well as in Chapters 3 and 4.

3. According to various estimates, the residence time for mercury in the atmosphere varies from 5.5 to 90 days, and the proportion of man-made mercury in the atmosphere ranges from 10 to 80 percent.

4. Use of mercury by man and subsequent emissions to land, rivers, and lakes, together with increased erosion rates, have elevated the mercury content of lakes and rivers by a factor of 2 to 4. Increases in total oceanic mercury concentrations have been negligible. The mercury content of soil appears to have increased by about 0.02 percent (Wollast et al. 1975).

Assumptions Involved in Existing Models

Estimates of the residence time of mercury as well as of the proportion of anthropogenic mercury in the atmosphere differ considerably. Therefore, it is worthwhile to consider some of the assumptions on which the published models are based.

Many calculations on the atmospheric flux of mercury are derived from measurements of dated Greenland ice cores. Recent evidence (Weiss et al. 1975) suggests that there has been no systematic change in the mercury concentration in these ice cores over the past 150 yr. In view of this evidence, previous observations of increased mercury concentrations in recently deposited snow and subsequent conclusions that this increase is related to anthropogenic inputs still appear to be unresolved. The mercury deposition at Greenland calculated from the 1975 Weiss et al. data seems, rather, to reflect polar and oceanic deposition rates not influenced by man.

Measurements of the natural mercury flux from the earth's surface to the atmosphere are mainly based on degassing rates measured in California. Degassing rates used by various authors range from 0.0014 to 10 $\mu g/m^2/day$. In addition, some investigators have assumed that degassing takes place only over the continents, whereas others consider this rate equal for continents and oceans. Further research is needed to clarify this issue, although Wollast et al. (1975) argue rather convincingly that the oceans must be a source for atmospheric mercury. The low atmospheric mercury concentrations over the oceans indicate, however, that

lower degassing rates can be expected than those that occur on the continents.

Removal of mercury from the atmosphere has been thought to occur mainly via washout by precipitation. Conflicting conclusions about changes in the atmospheric mercury concentrations before and after rain events leaves the validity of this assumption in doubt. This topic is treated in further detail in the next chapter.

The assumption that mercury concentrations in air are constant with increasing altitude must also be questioned. Recent data by Abramovskiy et al. (1975) show that the mercury concentration decreases exponentially with increasing altitude. The decrease has been shown to follow the relationship $C_z = C_0 e^{-kz}$, where C_0 = ground concentration, C_z = concentration at any altitude, z, and k = a constant, approximately equal to 10^{-3} per meter. These considerations make it likely that previous estimates of the atmospheric mercury pool have been excessive.

An Updated Model

The preceding discussion covers some of the salient features of previous publications. Figures 1.1 and 1.2 combine data from these previous models with the new information to summarize current knowledge of the global mercury cycle in a pre-man and a present-day framework. Mercury fluxes and reservoirs have been calculated from the data presented in Tables 1.1 and 1.2 and from other sources discussed elsewhere in this report.

The pre-man cycle presented in Figure 1.1 is a modification of the model presented by Wollast et al. (1975). The most important assumptions used in constructing these models can be summarized as follows:

● The mercury deposition rate over polar regions, derived from Weiss et al. (1975), is taken to be 22 × 10^{-6} g/m²/yr. A similar deposition rate is assumed for oceanic areas. If the average mercury concentration in air for these areas is 0.7 × 10^{-9} g/m³ (see Chapter 2), the deposition velocity is approximately 10^{-3} m/s (wet + dry) (Chamberlain 1960). If the deposition velocity remains constant over oceanic shelf areas and continents, the rate of deposition for these two areas is (1.5/0.7) × 22 × 10^{-6} = 47 × 10^{-6} g/m²/yr and (4.0/0.7) × 22 × 10^{-6} = 126 × 10^{-6} g/m²/yr. The assumption that the deposition velocity for polar, open ocean, and oceanic shelf areas is similar seems quite reasonable, whereas the assumption that the deposition velocity over continents is the same remains to be tested.
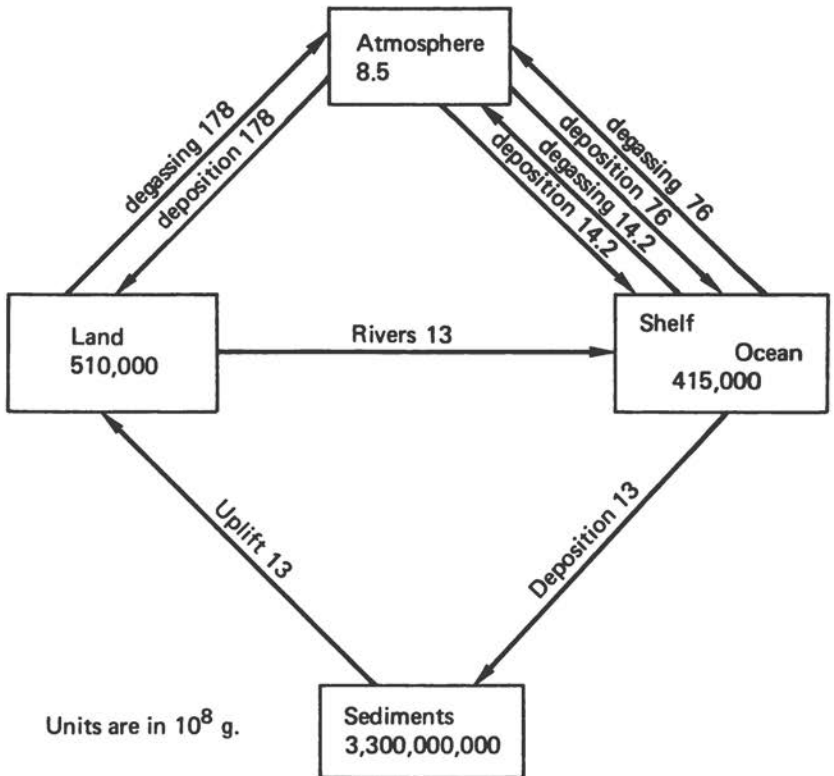
SOURCE: Modified from Wollast et al. (1975). Pages 145-166, *Ecological Toxicology Research: Effects of Heavy Metals and Organohalogen Compounds.* Reprinted with permission from Plenum Press, New York.

FIGURE 1.1 Pre-man global cycle for mercury.
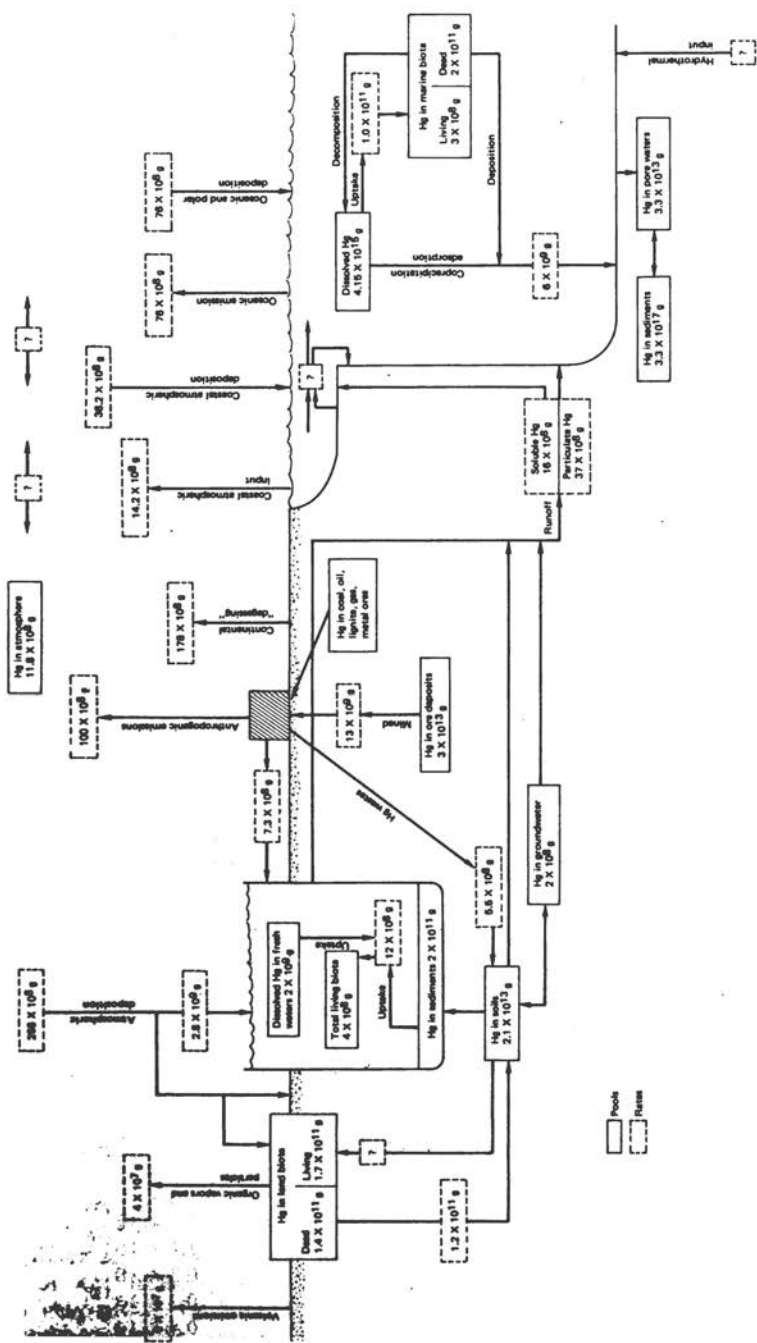
FIGURE 1.2 Present-day global cycle for mercury.

TABLE 1.1 Parameters Used in Constructing the Mercury Budgets

Area of the earth	510	$\times 10^{12} \text{ m}^2$
Area of the continents	149	$\times 10^{12} \text{ m}^2$
Area of polar regions	14.7	$\times 10^{12} \text{ m}^2$
Area of oceanic shelf regions	30	$\times 10^{12} \text{ m}^2$
Area of oceans	361	$\times 10^{12} \text{ m}^2$
Area of earth's freshwater systems	2.6	$\times 10^{12} \text{ m}^2$
Continental run-off volume	3.2	$\times 10^{16}$ ℓ/yr
Suspended sediment yield	183	$\times 10^{14}$ g/yr
Precipitation over continents	1.07×10^{17}	ℓ/yr
Precipitation over oceans	4.11×10^{17}	ℓ/yr
Atmospheric input of sea salt	1	$\times 10^{15}$ g/yr
Atmospheric input of volcanic dust	25	$\times 10^{12}$ g/yr
Atmospheric input of dust	5	$\times 10^{14}$ g/yr
Atmospheric input of vegetation	2	$\times 10^{14}$ g/yr
Open ocean primary productivity	16.3	$\times 10^{15}$ g C/yr
Coastal zone primary productivity	3.6	$\times 10^{15}$ g C/yr
Primary productivity in upwelling areas	0.1	$\times 10^{15}$ g C/yr
Open ocean fish production	1.6	$\times 10^{12}$ g/yr
Coastal fish production	1.2	$\times 10^{14}$ g/yr
Fish production in upwelling areas	1.2	$\times 10^{14}$ g/yr
Net primary production of land biota	47.6	$\times 10^{15}$ g/yr
Net primary production of lake and stream biota	0.6	$\times 10^{15}$ g/yr

C = Carbon

TABLE 1.2 Mercury Content of Global Reservoirs

Pool	Pool Mass (g)	Hg Concentration	Total Hg in Pool (g)
Atmosphere	$5 \quad \times 10^{21}$		8.5×10^8
Oceanic and polar		$z_0 = 0.7 \text{ ng/m}^3$	2.4×10^8
Oceanic shelf areas		$z_0 = 1.5 \text{ ng/m}^3$	0.5×10^8
Continental		$z_0 = 4.0 \text{ ng/m}^3$	5.6×10^8
Lithosphere			
Soils	$3 \quad \times 10^{20}$	71 ng/g	2.1×10^{13}
Freshwater sediments	$6.5 \quad \times 10^{17}$	330 ng/g	2.1×10^{11}
Oceanic sediments	$10.2 \quad \times 10^{23}$	330 ng/g	3.3×10^{17}
Hydrosphere			
Oceans	1.37×10^{24}	0.03 ng/g	41.5×10^{14}
Sediment porewaters	3.3×10^{23}	0.1 ng/g	3.3×10^{13}
Lakes and rivers	$3 \quad \times 10^{19}$	0.06 ng/g	0.2×10^8
Glaciers	$2 \quad \times 10^{22}$	0.05 ng/g	0.1×10^{11}
Groundwater	$4 \quad \times 10^{18}$	0.05 ng/g	0.2×10^7
Biosphere			
Living land biota	$8.3 \quad \times 10^{17}$	200 ng/g	1.7×10^{11}
Dead land biota	$7 \quad \times 10^{17}$	200 ng/g	1.4×10^{11}
Living marine biota	$1.5 \quad \times 10^{15}$	200 ng/g	0.3×10^7
Dead marine biota	$10 \quad \times 10^{17}$	200 ng/g	0.2×10^{10}
Living freshwater biota	$2 \quad \times 10^{15}$	200 ng/g	0.4×10^7

z_0 = surface altitude

- Using mercury degassing rates measured by McCarthy et al. (1969), Van Horn (U.S. EPA 1975b) conducted a state-by-state inventory of mineralized and nonmineralized areas. From the results, an average degassing rate of 130×10^{-6} g Hg/m^2/yr was calculated for the United States. This rate was assumed to remain the same for all continents. The degassing rates that support the atmospheric mercury concentrations for oceanic shelf, open ocean, and polar regions were then calculated to be $(1.5/4.0) \times 130 \times 10^{-6} = 49 \times 10^{-6}$ g/m^2/yr and $(0.7/4.0) \times 130 \times 10^{-6} = 23 \times 10^{-6}$ g/m^2/yr, respectively. These calculations balance the input rates quite well.

- Since no increase in mercury deposition rates has been observed in polar regions, it was assumed that emissions of mercury to the atmosphere from anthropogenic sources would result in increased deposition rates only over continents and oceanic shelf areas. As additional data become available this assumption will undoubtedly have to be revised since atmospheric constituents in the Northern Hemisphere are transported from east to west over considerable distances. From an environmental protection point of view, however, this assumption is conservative because it maximizes mercury deposition rates for continental and oceanic shelf areas.

- Since there are no data to indicate that degassing rates in oceanic shelf and open ocean areas have increased over the past century, it was assumed that these two areas realized a net increase of 22×10^8 g/yr from the atmosphere (see Figure 1.2).

- Flux rates between continental, oceanic shelf, and open ocean air masses and between shelf and open ocean water masses cannot be evaluated with current information. Evidence suggests, however, that much of the riverborne mercury is deposited initially in estuarine and continental shelf areas.

Many conclusions derived from the two models just presented are similar to those discussed earlier, but there are some differences as well. The residence times of mercury for the various reservoirs calculated here are: soils, 1000 years; atmosphere, 11 days; oceans, 3200 years; and sediments, 2.5×10^8 years. The shorter residence time for atmospheric mercury calculated in this report (see Chapter 2) is at variance with results from several previous models. The discrepancy can only be resolved with additional measurements both for different altitudes and for remote areas. Additional data on atmospheric mercury levels in remote areas would be especially helpful since the degree of variation of tropospheric gases has been shown to be inversely

proportional to their residence time in a model proposed by Junge (1974).

The model further indicates that from 25 to 30 percent of the atmospheric mercury burden is due to man-made emissions. It is still possible that a fraction of the atmospherically deposited mercury is again revolatilized. The magnitude of this deposition/volatilization phenomenon is presently unknown. Similarly, the mercury burden of rivers (water plus bottom and suspended sediments) has increased by a factor of 4 when compared with pre-man levels. This increase is presumably due to greater suspended sediment loads as well as to use of mercury by man.

Because of large variations in the mercury levels of freshwater and land biota and lack of historical data for a wide range of biological specimens, the models fail to resolve the question of whether increases have occurred in these compartments. The evidence for mercury increases in fish is discussed in Chapters 4 and 5. However, a survey taken for this study of mercury concentrations in sediment cores from freshwater lakes and estuaries indicates that these reservoirs have levels approximately 2 to 5 times higher than in precultural times (Shimp et al. 1970, Lindberg et al. 1975, U.K. Department of the Environment 1976).

THE GLOBAL CYCLE OF METHYLMERCURY

Chapter 3 reviews the mechanisms in the biological cycling of mercury. The discussion in this section, consequently, is confined to alkylated mercury flux calculations for fresh and ocean water systems.

Although many laboratory measurements of methylation and demethylation rates have appeared in the literature (Booer 1944, Kimura and Miller 1964, Wood et al. 1968, Jensen and Jernelov 1969, Landner 1971, Yamada and Tonomura 1972, Bisogni and Lawrence 1973, Spangler et al. 1973a, Billen 1973), quantifications of the flux of alkylated mercury for in situ natural conditions are lacking. Methylation rates in natural freshwater sediments spiked with inorganic mercury range from about 5 to 15 percent per year per gram of sediment. Although natural bacterial populations were maintained (i.e., there was no microbial enrichment), these experiments reflect only extremely polluted areas (100 μg Hg/g). Demethylation rates have also been measured by Spangler et al. (1973a) and by Billen (1973), and they were in the same range as the methylation rates in the mercury-contaminated samples. A delicate balance between methylation and demethylation is thus implied by these results.

These rates, however, do not reflect more commonly encountered mercury concentrations; nor do they necessarily represent the different redox, pH, and microbial variations in nature. Billen and Wollast (1973) have concluded that the most intense mercury methylation occurs in the transitional "oxidizing-anaerobic" zones of natural sediments. In sediments, pH commonly ranges between 6 and 8, while E_h ranges from approximately -0.2 to +0.4 V for these same zones. Bacterial demethylation seems to occur over a broader range of redox conditions. These physicochemical and microbiological factors have made in situ field measurements of methylation and demethylation rates difficult. In addition, extremely fast methylmercury cell diffusion rates and analytical problems make it necessary to calculate alkylmercury fluxes by indirect methods.

Based on the flux rates presented in Figures 1.1 and 1.2, it is possible to make some crude approximations of the net methylation rates in fresh and oceanic waters. The uptake rate can be calculated if the fish population is assumed to be the ultimate sink for methylmercury in these two reservoirs and that once methylmercury is produced, it is taken up by fish and other biota. According to Wetzel (1975), the annual fish production in fresh waters ranges from about 1 to 100 $g/m^2/yr$. If one takes 20 $g/m^2/yr$ as an annual average, the global fish production for these waters is 52×10^{12} g/yr. If one further assumes that the average mercury content of fish is 200 ng/g and that all of this is methylmercury, the annual uptake rate in the world's freshwater systems is 10^7 g methylmercury/yr. With the same basic assumptions and a total fish production rate of 2.4×10^{14} g/yr (Ryther 1969), the annual methylmercury uptake by oceanic fish is 4.8×10^7 g. On the assumption that the methylmercury uptake rate in fish is approximately equal to the net methylation rate of each reservoir, the rate would be 10^7 g/yr and 4.8×10^7 g/yr for fresh waters and oceans, respectively. Since open ocean fish production represents only 1 percent of the total oceanic production, it appears that most of the methylation is confined to nearshore and upwelling areas. The actual sites of methylation remain to be investigated.

If it is assumed that methylation takes place in bottom sediments in both reservoirs and is restricted to the upper 5 to 10 cm (Jernelov 1970), it is also possible to calculate the fraction of total mercury that is available for methylation. The amount of total mercury in the upper 10 cm of freshwater and oceanic sediments is approximately 2×10^{11} g and 3×10^{13} g, respectively (Kothny 1973). The average net methylation

rate for the two reservoirs is thus less than 0.005
percent per year. If the oceanic methylation site is
restricted primarily to estuarine and continental shelf
sediments, this rate is increased by approximately an
order of magnitude.

Although the calculations above contain many
simplifying assumptions (for instance, ignoring
diffusion rates of methylmercury from sediment to water
and the fact that biota other than fish are the ultimate
sink), they should nevertheless reflect order-of-
magnitude fluxes. The model explains the extremely low
alkylmercury concentrations in water and sediments (Chau
and Saitoh 1973, Andren and Harriss 1975). These
calculations also indicate that the biological cycle is
delicately balanced and that small perturbations in
input rates and the chemical form of mercury can result
in increased methylation rates in sensitive ecosystems.
It is also apparent that studies of the behavior of
methylmercury in nature must be intimately linked with
bioenergetic studies.

MAN-MADE MERCURY EMISSIONS IN THE UNITED STATES

The most recent detailed analysis of man-made versus
natural mercury emissions in the United States is that
of Van Horn (U.S. EPA 1975b). The analysis includes a
detailed summary of past, present, and future mercury
consumption patterns in the coterminous United States.
This information is presented in Table 1.3. Total
mercury losses to air, water, and land by different
activities on a national basis are presented in Table
1.4. In addition to consumption through commercial,
industrial, and consumer uses, which alone accounts for
more than 60 percent of the total man-related mercury
losses, the other major sources of anthropogenic mercury
emissions are mining and related activities, energy-
related activities, manufacturing, and processing.

Data from Table 1.4 indicate that the total
anthropogenic mercury losses in the United States for
1973 were approximately 1.5×10^9 g. Of these, 31
percent was emitted to air, 6 percent to water, 63
percent to land, and 1.5×10^8 g were recycled. The
natural degassing rate for the United States was
estimated to be about 1×10^9 g/yr. If it is assumed
that mercury emitted from the various sources has the
same atmospheric residence time, it is evident that 32
percent of the atmospheric mercury burden in the United
States is anthropogenic in origin.

A state-by-state estimate of natural and man-made
mercury emissions to air and water is presented in Table
1.5. The man-made increases in mercury discharges to

TABLE 1.3 Mercury Consumption in the United States, 1970-1973 and Projected for 1985

End Use Activity	1970	1971	1972	1973	Projected for 1985
				$(10^6$ g$)$	
Agriculture	62.3	50.8	63.2	62.9	24.1
Catalysts	77.0	34.3	27.5	23.2	17.2
Dental preparations	78.6	64.4	102.6	92.2	129.0
Electrical apparatus	548.7	572.6	535.0	619.2	662.0
Caustic chlorine	516.4	421.5	396.2	449.6	481.6
Laboratories	62.1	46.7	20.4	22.6	?
Industrial instruments	166.2	134.4	225.0	246.1	447.2
Paints	355.9	296.0	282.8	261.5	106.6
Pharmaceuticals	23.7	23.0	19.9	20.8	17.2
Other	209.3	170.0	147.2	69.1	206.4
TOTAL (rounded)	2100	1814	1820	1867	2091

SOURCE: Derived from U.S. EPA (1975b).

TABLE 1.4 Total Mercury Losses in 1973 for the Coterminous United States

Activity Description	Total Losses to			Total Hg Lost	Total Hg Recycled
	Air	Water	Land		
			$(10^6$ g$)$		
Hg mining, smelting and processing	7.85	0	0.42	8.27	0
Other mining activities	50.71	3.07	4.91	58.09	0
Energy related activities	104.5	2.18	42.07	148.73	0
Manufacturing and processing (see Table 1.3)	26	21.72	271.33	319.05	20.66
Commercial, industrial and consumer consumption	282.2	60.73	646	990	126.11
TOTAL	471.26	87.70	964.73	1524.73	146.77
Sewage	4.01	19.92	22.88	46.0	
Natural degassing	1018.7			1018.7	

SOURCE: Derived from U.S. EPA (1975b).

TABLE 1.5 Mercury Emissions in the United States, 1973

| | Man-made Losses to | | Natural Losses to | | Percent Due to Man | |
	Air	Water	Air	Water	Air	Water
			$(10^6$ g/yr)			
Alabama	9.9	2.5	9.6	4.1	51	38
Arizona	22.3	2.4	85.8	11.3	21	18
Arkansas	3.0	1.8	9.8	2.0	23	47
California	36.8	14.2	118.2	26.3	24	35
Colorado	3.5	1.6	39.2	6.0	8	21
Connecticut	6.0	2.0	1.8	0.01	77	99
Delaware	1.9	0.5	0.4	0.01	83	98
Florida	12.1	4.6	10.2	0.8	54	85
Georgia	9.5	3.2	22.0	2.7	30	54
Idaho	2.8	0.6	31.3	6.3	8	9
Illinois	22.4	8.0	10.5	1.4	68	85
Indiana	12.2	3.8	6.8	3.2	72	54
Iowa	5.6	2.0	10.6	5.9	35	25
Kansas	3.7	2.1	15.5	2.2	19	49
Kentucky	10.5	2.1	7.5	1.2	58	64
Louisiana	8.4	2.6	8.5	0.5	50	84
Maine	2.5	0.7	5.8	0.3	30	70
Maryland	7.6	3.0	3.7	0.1	67	97
Massachusetts	11.4	3.9	3.0	0.6	79	87
Michigan	18.8	5.2	10.7	0.8	64	87
Minnesota	7.0	2.7	14.9	6.0	32	31
Mississippi	5.1	1.4	8.9	4.3	34	25
Missouri	13.8	3.4	13.0	4.6	50	43
Montana	4.0	1.0	55.0	1.9	7	34
Nebraska	2.3	0.7	14.5	1.9	14	. 27
Nevada	5.6	0.3	83.1	4.2	6	7
New Hampshire	1.4	0.3	3.4	0.1	29	75
New Jersey	14.6	3.4	1.4	0.2	91	94
New Mexico	4.9	0.5	45.9	20.7	10	2
New York	35.5	7.1	18.1	0.6	66	92 `
North Carolina	12.2	2.2	18.5	1.2	40	65
North Dakota	0.9	1.0	13.1	0.9	6	53
Ohio	25.3	4.5	7.8	1.8	76	71
Oklahoma	4.6	1.3	13.0	25.5	26	5
Oregon	3.6	0.8	72.7	2.6	5	24
Pennsylvania	34.8	5.1	17.0	0.8	67	86
Rhode Island	1.8	0.4	0.2	0.02	90	95
South Carolina	5.5	1.2	5.7	1.1	50	52
South Dakota	1.1	0.4	14.4	1.3	7	24
Tennessee	10.0	2.3	7.8	5.2	56	31
Texas	25.9	4.7	49.6	18.5	34	20
Utah	7.8	0.7	31.0	3.0	20	19
Vermont	0.7	0.2	2.2	0.2	24	50
Virginia	8.1	1.8	15.0	1.1	35	62
Washington	9.0	1.5	25.2	0.9	26	63
West Virginia	5.4	0.8	9.1	0.6	37	57
Wisconsin	8.4	2.3	10.3	0.9	45	72
Wyoming	0.7	0.1	36.8	2.9	2	3

SOURCE: Derived from U.S. EPA (1975b).

air must be interpreted with care because each state has a different natural degassing rate. Thus, states with a low natural rate might show a greater increase even though the man-made emissions are lower than those in a state with high emission rates from both sources. These calculations can only be validated through a properly administered monitoring program. Calculations of man-made mercury emissions to water indicate that annual natural emissions have at least been doubled by anthropogenic discharges in Connecticut, Delaware, Florida, Illinois, Louisiana, Maine, Maryland, Massachusetts, Michigan, New Hampshire, New Jersey, New York, Ohio, Pennsylvania, Rhode Island, and Wisconsin.

Because the relation between mercury emissions and population density is an important consideration, Van Horn expressed the data in Table 1.5 on the basis of population. The mean mercury emissions in kilograms per 1000 population per year, calculated for the coterminous United States in 1973, were: air, 1.81 ± 0.53; water 0.41 ± 0.08; land, 4.55 ± 1.20. Table 1.6 identifies so called "hot spots" in terms of mercury emissions to air, water, and land as a function of population density. These "hot spots" are states with exceptionally high levels of mercury emissions per 1000 population and have been related to either chloralkali plants or copper smelting facilities. On the other hand, Van Horn's analysis showed that the presence of chloralkali plants and copper smelting facilities does not necessarily lead to elevated concentrations.

It is not yet clear how valuable these data are for projecting the mercury burden of aquatic and terrestrial biota and the human population, but their value is apparent in establishing monitoring and health evaluation programs. Since the behavior of mercury is very much a function of its chemical form, potential biomethylation "hot spots" might be predicted by evaluating the data presented in Tables 1.5 and 1.6 together with prevailing physical, geological, and chemical regimes for each region. For the aquatic system, various factors must be considered such as pH, redox conditions, alkalinity, buffering capacity, suspended sediment load, and lake or river geomorphology.

Within this context, Brouzes et al. (1977) have found that poorly buffered lakes in the Canadian shield region are particularly sensitive to the impact of acid rain. Fish collected in these lakes exhibit high mercury concentrations even when the lakes and rivers are not near industrial activities, roads, or villages. Similarly sensitive areas should be identified in the United States in addition to systems that have received direct mercury spills. The release of mercury to the

TABLE 1.6   Annual Man-Made Mercury Emissions in the Coterminous
United States, 1973

Level of Emissions	Air	Water	Land
		(g/1000 pop.)	
"Hot spots"	Arizona, Montana, Nevada, New Mexico, Utah $> 4 \times 10^3$	Arizona, Montana $> 0.8 \times 10^3$	Delaware, Kentucky, Louisiana, West Virginia $> 9 \times 10^3$
Moderately elevated	Georgia, Idaho, Kentucky, Missouri, Pennsylvania, West Virginia $(2.3\text{-}4) \times 10^3$	Delaware, Idaho, Kansas, Nevada, South Dakota, Tennessee, Utah $(0.49\text{-}0.8) \times 10^3$	Georgia, Maine, Montana, New Jersey, Tennessee, Washington $(5.7\text{-}9) \times 10^3$
Below national average	Colorado, Kansas, Nebraska, Vermont $< 1.28 \times 10^3$	Florida, Vermont $< 0.33 \times 10^3$	Arizona, Florida $< 3.4 \times 10^3$

SOURCE: Adapted from U.S. EPA (1975b).

environment from coal-fired power plants, mining, and
smelting operations is of special concern since these
sources are currently uncontrolled and the use of coal
is expected to increase significantly.

CHAPTER 2

FORMS AND OCCURRENCE OF MERCURY IN THE ENVIRONMENT

Because different chemical forms of mercury possess different reactivity and toxic characteristics, it is important to identify these in the environment. Two major classes of mercury compounds are generally distinguished, inorganic and organic. From the standpoint of risk to human health from environmental exposures, the alkylated mercurials within the class of organic mercury compounds are the most important because of their toxic properties and their tendency to bioaccumulate. Of the alkylmercurials, methylmercury presents the greatest risk. However, both the inorganic forms (such as metallic mercury and mercury sulfide, the principle mercury ore) and the organic forms of mercury are subject to conversion in the environment, and thus, their availability is important.

MERCURY IN THE ATMOSPHERE

The sources of mercury to the atmosphere have not been completely identified, quantified, or characterized. Our present understanding, however, indicates that the most important sources are rock and soil volatilization, volcanic exhalations, biological emanations, suspended continental dust, and various anthropogenic inputs. On the basis of data obtained by analyzing dated Greenland and Antarctic snow cores (Weiss et al. 1971), several authors (Korringa and Hagel 1974, Heindryckx et al. 1974, Wollast et al. 1975) have suggested that the annual natural flux of mercury to the atmosphere is from 25,000 to 30,000 metric tons per year (1 ton = 10^6 g). This includes the input from both terrestrial and oceanic sources, although the relative importance of the two is not known. By the same analogy, these authors argue that present-day input of mercury to the atmosphere from both natural and anthropogenic sources ranges from 41,000 to 50,000 metric tons, because data from the snow cores showed this increase in the more recent deposits. As shown below, recent measurements at

another station in Greenland do not show this decrease with depth in the snow cores. The atmospheric cycle of mercury must be analyzed by other means, as has already been discussed in Chapter 1.

For a firmer understanding of present knowledge about its atmospheric cycle, a compilation of recently published mercury levels discussed in this section is presented in Table 2.1. Because relatively few measurements have been made of large-scale geographically representative areas, it is prudent to use observed ranges rather than to emphasize mean or average concentrations. Since the latter values are, nevertheless, quite useful for a better understanding of the mercury cycle, they are included. Remote oceanic areas have atmospheric mercury concentrations of around 0.7 ng/m^3, almost all in the vapor form. Remote rural areas exhibit mercury concentrations that cluster around 4 ng/m^3, with less than 5 percent in particulate form. Urban areas are quite variable, but seem to exhibit concentrations of somewhat less than 10 ng/m^3 with variable particulate mercury fractions. Point sources such as volcanoes, mines, and industries can increase the atmospheric burden considerably, and Carr and Wilkniss (1973) have argued that volcanic emissions can account for any variability of the mercury burden in snow cores.

Mercury Vapor

Although by no means substantiated on a global scale, many investigators have hypothesized that elemental mercury, Hg^0, is the major form of mercury vapor (Williston 1968, McCarthy et al. 1969, Johnson and Braman 1974). This should be especially true near geothermal and volcanic areas. Because of disproportionation reactions between mercuric oxide and metallic mercury at high temperatures, it is also thought that more than 97 percent of the mercury emitted from coal-fired steam plants is in the form of elemental mercury (Billings et al. 1973, Klein et al. 1975).

Johnson and Braman (1974) recently developed a technique for distinguishing between different chemical forms of mercury vapor in air. Air is first drawn through a glass fiber filter and then through a series of selective adsorption tubes that contain HCl-treated siliconized Chromosorb W, NaOH-treated Chromosorb W, silver-coated glass beads, and gold-coated glass beads. This system collects particulate mercury, mercury(II)-type compounds, methylmercury-type compounds, metallic mercury, and dimethylmercury. The measurements substantiate previous observations that mercury in air

TABLE 2.1   Summary of Current Data on Atmospheric Mercury Levels for Various Locations

	Range	Mean
	(ng/m^3)	
A. Remote and Rural Areas		
Oceanic		
Particulate	< 0.005–0.06	<0.15
Vapor	0.6–0.7	0.7
Non-mineralized terrestrial		
Particulate	< 0.005–1.9	0.15
Vapor	1–10	4.0
Volcanic		
Particulate + vapor	20–37,000	–
Mineralized terrestrial		
Particulate + vapor	7–20,000	–
B. Urban Areas		
Particulate	< 0.01–220	2.4
Vapor	0.5–50	7.0
C. Industrial*		
Vapor	7–5,000,000	–

*These measurements include chlor-alkali plants, thermometer factories, smelters, and mercury mines.

is primarily a vapor. Concentration ranges and means
for 11 stations around Tampa, Florida, are shown in
Table 2.2 (Johnson and Braman 1974). Their data
indicate that dimethylmercury is rarely encountered in
the atmosphere, but that other forms are present in
varying amounts. The particulate fraction averaged 4
percent of the total mercury collected. The vapor
species were averaged as follows: Hg(II)-type, 25
percent; methylmercury-types, 21 percent; Hg⁰, 49
percent; and dimethylmercury, 1 percent. The authors
pointed out that Hg⁰ and methylmercury-type vapors
exhibited fairly uniform concentrations whereas the
other forms varied considerably and the particulate
mercury was strongly correlated with wind conditions.
The authors indicated that methylated mercury was
emitted from the terrestrial system as well as from a
highly polluted bay nearby.

These and all previous data must, however, await
further evaluation and validation. The different
species of mercury collected by the previously described
technique are essentially defined operationally, i.e.,
glass fiber filters pass a sizable number of particles
of less than 0.3 μm; and it is therefore possible that a
portion of the first tube collects particulate matter as
well. This problem will be present whenever filtration
techniques are used to separate particulate from vapor
phase mercury. Disproportionation reactions also
readily take place, especially when in contact with many
different collection surfaces. Extremely difficult
analytical techniques as well as limited geographical
sampling do not permit further conclusions as to the
mercury speciation in air. More emphasis must be placed
on obtaining an atmospheric mercury data base from
culturally and geographically representative areas of
the world.

Mercury in Rain and Snow

Even less information exists on mercury in rain and
snow, and it is presently difficult to predict levels
for various locations. The published data are
summarized in Table 2.3. A partial explanation for this
lack of data can be attributed to difficulties
encountered in rain sampling as well as in the
interpretation of the data. Except for areas of direct
input, most measurements show levels of from 0.01 to 0.1
$\mu g/l$. Some contradictions seem to exist in the
literature as to the rainout and washout efficiencies of
mercury. Measurements by McCarthy et al. (1969) showed
that a heavy rainstorm very efficiently removed mercury
from air whereas Johnson and Braman (1974) reported

TABLE 2.2 Levels and Chemical Forms of Mercury in Tampa, Florida[a]

	ng/m^3					
	[Hg] part.	[Hg(II)] vap.	[CH$_3$Hg]	[Hg^0]	[CH$_3$HgCH$_3$]	Σ Hg
Range	<0.03–13	<0.03–220	<0.07–119	<0.03–49	<0.03–3	1.8–298
Mean[b]						
Day	0.27	0.86	0.63	2.67	0.05	4.48
Night	0.17	1.58	1.56	5.03	0.06	8.40

[a]From 11 stations around Tampa, Florida at two different dates.
[b]Mean values collected at one station for 33 consecutive 2 hour samples.

SOURCE: Johnson and Braman (1974). Reprinted with permission from Environmental Science and Technology 8:1003-1009. Copyright by the American Chemical Society.

TABLE 2.3 Mercury Levels in Precipitation

Location and Type	Range (μg/ℓ)
A. Rural U.K.	<0.2
Germany (1934)	0.05–0.48
Oak Ridge, Tenn., U.S.A.	<0.05–0.54
B. Snow Layers	
Sweden: surface layer	0.07
sublayer	0.21
near chlor-alkali plant	<2–11
Canada: urban	3.5–4
rural	<0.01–0.52
C. Greenland and Antarctica	
Before 1900*	0.013–0.169
After 1900*	0.040–0.23

*No apparent trends with age of snow deposit (Weiss et al. 1975).

essentially the same atmospheric mercury concentration before, during, and after a thunderstorm. Therefore, it is possible that mercury can have a significant dry removal component (vapor impact) as well. Until this question has been resolved use of historical records of snow cores to deduce the mercury content of snow at the time it was deposited is in doubt. It is entirely possible that the measured mercury concentrations in snow reflect variable proportions of dry plus wet input.

The chemical form of mercury in precipitation are not known, and attempts to determine the physical form of mercury in precipitation have met with limited success. This is mainly due to extremely difficult sampling problems. Mini dust storms often precede precipitation events. Since most investigators use automatic rain-collecting devices that collect time-integrated samples, avoiding this problem is difficult. Several reports on concentrations in precipitation include mercury added to rainwater by windblown dust of a very localized nature (i.e., from the immediate vicinity of a sampler). To separate dissolved and particulate mercury requires operationally defined procedures such as filtration or centrifugation, which also have inherent problems. The sample must also be properly preserved if losses to the walls or by volatilization are to be prevented.

Many of the severe sampling and analytical problems indicated above stem from the need to use operational definitions, i.e., based on collection techniques, when assigning proper values to the chemical and physical forms of the sampled mercury. Additionally, because recent data indicate no systematic change in mercury concentrations of snow cores from Greenland, the previous calculations of man's influence on the global mercury cycle need to be reevaluated.

MERCURY IN THE LITHOSPHERE

Mercury in the lithosphere has been reviewed in two recent reports (World Health Organization 1976b, U.K. Department of the Environment 1976). In mineralized areas, in some rocks, or where anoxic conditions exist, much of the mercury is in the form of its sulfide (cinnabar). This is the most insoluble form of mercury. However, cinnabar is mined for industrial and human consumption uses and when released into the environment is transformed into other mercury compounds, making it more readily available for potential methylation. Once mercury is thus mobilized, if it is not then recycled but, rather, released into the environment, conversion to its original geochemical form (for example mercury

sulfide) and burial would be the best safeguard against transformation into methylmercury.

Statistical correlations between total sediment sulfur and mercury have been interpreted as evidence that mercury often occurs either as an insoluble sulfide or adsorbed onto the surface of sulfide minerals (mainly FeS_2). According to Andersson (1967) and Keckes and Miettinen (1970), mercury is predominantly bound to soils and sediments through association with organic matter. In addition, Vernet and Thomas (1972) and Thomas (1973) suggest that observed relationships between total mercury, iron, and phosphorus indicate that mercury is bound to an inorganic iron-phosphate complex, probably adsorbed onto hydrated oxide and manganese coatings of clay particles.

Divalent inorganic mercury can undergo reduction to elemental mercury. Certain bacteria of the genus Pseudomonas can perform the conversion (Magos et al. 1964, Furukawa et al. 1969). Processes leading to the formation of elemental mercury in soils and sediments are not well understood, but must form the basis for soil degassing of mercury. According to the WHO report (1976b:50): "Unfortunately, other than very crude generalizations, little is known of the details of kinetics of these processes in nature."

Within the last few years an enormous quantity of data on relative mercury concentrations in soils and sediments has appeared in the literature. Table 2.4 summarizes mercury levels in rocks, soils, and sediments from a recent review (U.K. Department of the Environment 1976). Mercury concentrations of unpolluted, nonmineralized soils can vary by 2 orders of magnitude with the lower concentrations at about 0.01 $\mu g/g$. The mean values for American and British soils were 0.071 $\mu g/g$ (Shacklette et al. 1971) and 0.060 $\mu g/g$, respectively. The former value is most often used as the overall mean crustal concentration of mercury. Although highly variable, the mercury content of soils in mineralized areas can exceed 500 $\mu g/g$. Cinnabar ore deposits usually contain from 0.5 to 1.2 percent mercury. The mercury content of nonmineralized freshwater sediments exhibits ranges similar to those of soils, although the available data indicate a mean of about 0.3 $\mu g/g$. Concentrations of 800 to 1000 $\mu g/g$ have been observed in polluted sediments, usually in close proximity to chloralkali plants. Aston et al. (1972) found a mean mercury value of 0.41 $\mu g/g$ for North Atlantic sediments, and Weiss et al. (1972) found values ranging from 0.012 to 0.173 $\mu g/g$ in sediments collected in the Pacific Ocean off the west coast of Mexico. Many more measurements of mercury in oceanic sediments are required to arrive at an average value although 0.33

TABLE 2.4   Mercury Concentrations in the Lithosphere

	Range	Mean
	(μg/g)	
Soils		
Sweden	0.01–1.0	0.070
Finland	0.02–0.2	0.060
England	0.01–15.0	0.060
Scotland	0.01–1.96	0.080
Japan	0.18–0.33	0.28
United States	0.01–4.7	0.071
Freshwater Sediments		
Sweden	0.034–26.5	0.3
Finland	0.05–170	–
England	0.01–1.026	–
United States	0.01–1200	0.3
Estuarine Sediments		
England	0.01–150	0.40
Canada	0.02–26.0	–
United States	0.01–0.5	0.33
Marine Sediments		
North Atlantic	–	0.41
Off West Mexico	0.012–0.173	–

$\mu g/g$ has been used in this and other reports for budget calculations (Wollast et al. 1975). According to Joensuu (personal communication, Rosensteil School of Marine and Atmospheric Sciences, University of Miami, 1977), his unpublished data suggest that this value should be revised to between 0.05 and 0.1 $\mu g/g$.

MERCURY IN NATURAL WATERS

Our knowledge of the chemical forms of mercury in natural waters is, at present, incomplete. The most common theoretical approach has been to predict the chemical forms of mercury by using thermodynamic calculations (Anfalt et al. 1968, Dyrssen and Wedborg 1974, Hem 1970, Wollast et al. 1975). These calculations predict that redox, pH, and ligand conditions are very important. Hem (1970) and Wollast et al. (1975) predict that in well-oxygenated waters (E_h > 0.4 V) dissolved mercury should exist as mercury(II)-type compounds, in moderately oxidizing to mildly reducing conditions (E_h -0.2 to 0.4 V) as Hg^0 or $Hg(II)$, and in reducing conditions (<-0.2) as Hg^0 or $HgS_2^=$. Anfalt et al. (1968) predicted that $Hg(II)$ would exist as $Hg(OH)_2$, $HgCl_2$, or $HgOHCl$ in fresh waters with a strong dependence on pH and pCl. Dyrssen and Wedborg (1974) also calculate that the most important species in seawater appear to be $HgCl_4^=$, $HgCl_3Br^=$, $HgCl_3^-$, $HgCl_2Br^-$, and $HgCl_2^0$ at relative concentrations of 65, 12, 12, 4, and 3 percent, respectively. Wollast et al. (1975) have shown that Hg_2^{++} can only exist at concentrations greater than 450 mg/l of total Hg, a level that is unlikely in natural waters.

There are, however, many kinetic factors that can change the speciation of mercury in the aquatic environment. These include: associations with dissolved organic matter, associations with suspended particulate matter (clays, hydrous oxides, and detrital organic matter), and methylation-demethylation processes. Fitzgerald and Lyons (1973) concluded that 50 to 60 percent of dissolved Hg in coastal waters could exist in association with organic matter of unspecified composition. Lindberg and Harriss (1973) and Andren and Harriss (1975) observed a strong association between dissolved organic matter and mercury in fresh, estuarine, and interstitial waters. The latter authors also suggested that substantial amounts of Hg are removed by flocculating organic matter in the transition zone between fresh and saltwater. Lindberg et al. (1975) have indicated that strong mercury-organic matter associations also prevent the highly insoluble mercury sulfide from precipitating in reducing sediments. This

again emphasizes the importance of organic matter in regulating the behavior of mercury. The nature of this association is not at present well understood although functionalities such as -SH, -COOH, and -N are assumed to be responsible.

Because of its reactivity, mercury is strongly bound to suspended particulate matter, so that in most fresh water (especially in contaminated areas) more than 50 percent is transported in this manner (Cranston and Buckley 1972, Lindberg et al. 1975). Wollast et al. (1975) also report that hydrous metal oxides significantly reduce and modify the form of Hg in natural waters. These and other reports indicate that the concentration of dissolved Hg in fresh water varies between 0.02 and 0.06 $\mu g/l$, whereas typical oceanic values range from 0.01 to 0.03 $\mu g/l$. Data are scarce on the partitioning between dissolved and particulate phases in sea water, although they indicate that most of the Hg exists in the dissolved form (Carr et al. 1972).

Very little information has been presented on the levels of methylated mercury in fresh and marine waters although it has been shown that methylation takes place in fresh and coastal water sediments. Foreback (1973) indicated that approximately 50 percent of the dissolved Hg in a polluted Florida bay was methyl or phenylmercury. Chau and Saitoh (1973) measured methylmercury levels in several Canadian lakes. With a detection limit of 0.24 ng/l they found no methylmercury in unfiltered Great Lakes water and a concentration of 0.5 to 0.7 ng/l in four smaller, polluted lakes. Andren and Harriss (1975) could detect no methylmercury in samples from rivers and coastal waters of the eastern Gulf of Mexico. It thus appears that where methylmercury compounds are formed they are rapidly taken up by biota, and that nonpolluted waters consequently contain less than 0.2 to 1.0 ng/l of Hg in the methylated form.

Field and laboratory measurements nevertheless suggest that, even with the extremely low concentrations of methylated forms of mercury present in the aquatic environment, the major form of mercury in the biota is still methylated. This indicates that uptake rates and subsequent bioaccumulation of mercury must be rapid. It is, therefore, imperative that the natural methylation-demethylation balance not be disturbed by the discharge of mercury (in whatever form) and excess nutrients to the environment.

CHEMICAL AND BIOCHEMICAL MECHANISMS FOR
METHYLATION AND DEMETHYLATION; KINETICS

"The case of mercury pollution has clearly
demonstrated the profound importance of
understanding biologically-mediated transformation
reactions that yield metal-organic compounds with a
high potential for bioaccumulation and toxicity."
(NRC 1977)

BIOMETHYLATION OF MERCURY

Three methylating coenzymes have been discovered that
participate in biomethylation reactions in biological
systems: (1) $\underline{S}$-adenosylmethionine, (2) $\underline{N}^5$-
methyltetrahydrofolate, and (3) methyl-B_{12}. Both from a
theoretical viewpoint and from direct experimental
evidence, it has been established that only methyl-B_{12}
is capable of methylating soluble inorganic mercury
salts to methylmercury and dimethylmercury. This
reaction occurs both under anaerobic and aerobic
conditions (Wood et al. 1968; Hill et al. 1970; Adin and
Espenson 1971; Neujahr and Bertilsson 1971; Schrauzer et
al. 1971, 1973; Wood 1971). Recent studies have
elucidated detailed mechanisms for the biosynthesis of
methylmercury and dimethylmercury from methyl-B_{12} (Imura
et al. 1971; DeSimone et al. 1973; Wood 1973, 1974,
1975a, 1975b). The second-order rate constant for the
biosynthesis of methylmercury from mercuric acetate has
been determined as $3.7 \times 10^2 \text{ s}^{-1} \text{ m}^{-1}$; and, therefore,
under optimum conditions the kinetics for methylmercury
synthesis have been shown to be extremely rapid
(DeSimone et al. 1973). The rate at which methylmercury
is formed in any environment is largely determined by
the available concentrations of soluble inorganic
mercuric ion and methyl-B_{12} (DeSimone et al. 1973, Wood
1975a).
The biosynthesis of methylmercury in sediments and by
bacteria isolated from sediments is well established
(Jensen and Jernelov 1968; Langley 1971; Bisogni and
Lawrence 1973, 1975a, 1975b; Vonk and Sijpesteijn 1973;

Spangler et al. 1973b; Jernelov 1974). It has also been confirmed that methylmercury synthesis is catalyzed by bacteria which have B_{12} dependence (Vonk and Sijpesteijn 1973). The rate of methylmercury biosynthesis can be enhanced by adding B_{12} to certain bacterial cultures (Vonk and Sijpesteijn 1973).

The biosynthesis of methylmercury in fungi is less clear, especially since it has been reported that Neurospora (a B_{12} independent mold) is capable of synthesizing methylmercury from the methionine biosynthetic pathway and mercuric salts (Landner 1971). Since many molds and photosynthetic bacteria are capable of synthesizing ethylene from methionine, it seems likely that this methylmercury is formed by the well-known reaction between ethylene and mercuric ion. This reaction was probably responsible for the chemical synthesis of contaminating methylmercury at Minamata (Kurland et al. 1960).

DEMETHYLATION OF METHYLMERCURY

The demethylation of methylmercury by sediments, microorganisms isolated from sediments, soil, and fecal organisms has been well established (Furukawa and Tonomura 1971, 1972a, 1972b; Summers and Silver 1972; Summers and Sugarman 1974; Schottel et al. 1974; Tezuka and Tonomura 1976). The enzyme system has been partially purified and was shown to consist of a hydrolase and a reductase. The hydrolase hydrolyzes the mercury-carbon bond to produce methane and mercuric ion. The reductase reduces the mercuric ion to mercury metal, which is volatilized from the aqueous culture medium into the atmosphere. Microbial resistance to methylmercury poisoning, as well as inorganic mercury poisoning, has been shown to develop through the transfer of extra-chromasomal factors (episomes) which carry the genetic information needed to produce both the hydrolase and reductase enzymes.

In purified enzyme systems, rate constants have been obtained for methylation (DeSimone et al. 1973) and demethylation (Tezuka and Tonomura 1976). Based on these initial studies, it appears that under ideal conditions demethylation is several orders of magnitude slower than methylation.

KINETICS OF METHYLMERCURY BIOSYNTHESIS
AND METHYLMERCURY DIFFUSION INTO CELLS

The rate of methylmercury biosynthesis in selected ecosystems is determined by the microbial community

CH_4 + C_2H_6 -

Hg^0

Air

$(CH_3)_2Hg$

Fish

Shellfish

Water

CH_3Hg^+

CH_3SHgCH_3

Hg^0 ← Bacteria ← CH_3Hg^+ → Bacteria → $(CH_3)_2Hg$ $CH_3S-HgCH_3$

Sediment

Bacteria

Bacteria

Hg_2^{2+} ⇌ Hg^{2+} + Hg^0

Bacteria
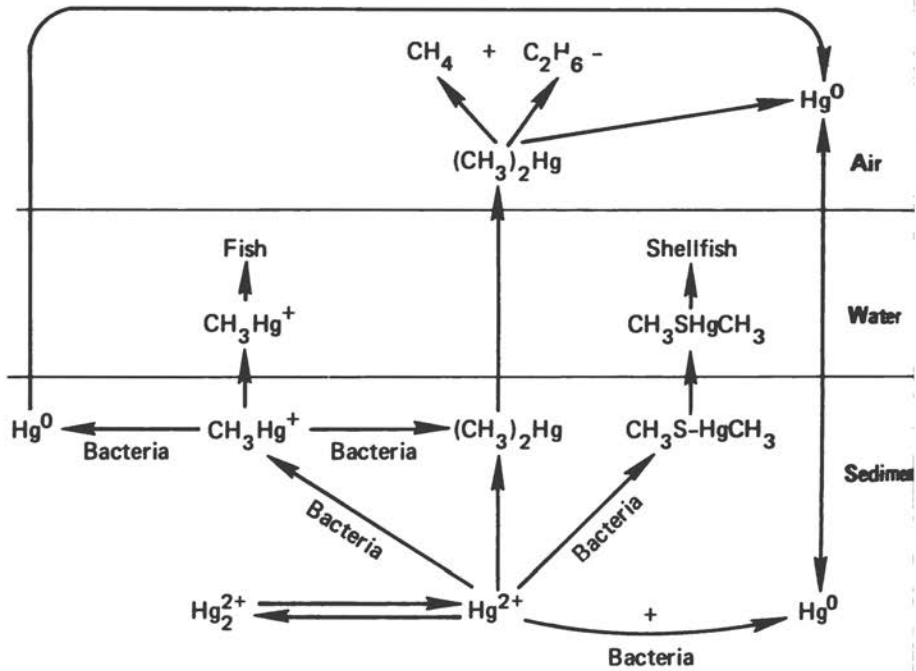
SOURCE: Modified from Wood (1974). Science 183:1049-1052. Copyright 1974 by the American Association for the Advancement of Science.

FIGURE 3.1 The mercury cycle demonstrating the bioaccumulation of mercury in fish and shellfish.

together with the available concentrations of soluble
mercuric ion species and methyl-B_{12} compounds. The
presence of demethylating microorganisms will allow a
steady-state concentration of methylmercury to build up
in any ecosystem, but the concentration would be lower
than if they were less abundant. As with other elements
on the earth's crust, mercury has a biological cycle
(see Figure 3.1). Therefore, potential environmental
problems with mercury develop only if the steady-state
concentration of methylmercury increases in a particular
environment (Wood 1976).

Evidence is now available to show that the
bioaccumulation of methylmercury into the tissues of
higher organisms (e.g., fish) is probably controlled by
diffusion (S. Rakow and J.R. Lakowicz 1977, manuscript
in preparation. Membrane permeability to methylmercuric
chloride by fluorescense quenching. University of
Minnesota, Freshwater Biological Institute). It has
been determined that methylmercury chloride diffuses
through cell membranes (40 angstroms) into cells in 20 ×
10^{-9} s (Rakow and Lakowicz cited above). Once
methylmercury diffuses through the cell membrane, it is
rapidly bound by sulfhydryl groups (see Chapter 5,
section entitled Protective Mechanism), thereby
maintaining the concentration gradient across the
membrane. This means that extremely low concentrations
of methylmercury will bioaccumulate rapidly in
ecosystems, thus explaining the bioaccumulation
phenomenon observed for methylmercury in the
environment, as well as the low steady state
concentration found in water.

SUMMARY AND CONCLUSIONS

The biological synthesis of methylmercury is a natural
phenomenon, and methylmercury is an integral part of the
mercury cycle. The rate of synthesis of methylmercury
is determined by the size of the natural microbial
populations capable of synthesizing this neurotoxin.
Microbial population sizes are determined by the extent
of nutrient supplies to lakes, rivers, and coastal areas
(i.e., the extent of eutrophication). Cleansing our
waterways can certainly have a major effect on
methylmercury synthesis in polluted areas, as could
cutting back on the point sources for soluble inorganic
mercury compounds.

Finally, once formed, methylmercury diffuses rapidly
into the cells of higher organisms. The rates for
bioaccumulation are so rapid that even low
concentrations of methylmercury in water lead to
elevated concentrations in fish.

CHAPTER 4

THE ECOLOGICAL EFFECTS OF MERCURY

Although the acute toxic effects of inorganic and organic mercury compounds on plants and animals have been determined, the effects from chronic low-level exposure have not been widely documented because these effects may not be apparent for a long time and the bioassay procedures currently available are insufficiently sensitive and selective to monitor them. Although low-level exposure to mercury has been associated with reduced growth and photosynthesis in phytoplankton and with teratogenic and mutagenic malformations in plants and animals, more precise tests and additional data are needed.

Most non-fish-eating terrestrial animals are not exposed to high levels of mercury and generally do not appear to be at risk except in regions of naturally occurring mercury deposits or in anthropogenically polluted areas. However, in most aquatic systems the organisms are constantly exposed to very low levels of methylmercury in water and food. If the watercourse receives anthropogenic mercury pollution, these low levels may increase and methylmercury may be concentrated to hazardous levels. Aquatic organisms absorb and magnify methylmercury at each trophic level of the food chain, thereby threatening not only the ecosystems but also human health, via the food chain. The results of one study of biological mercury magnification in the aquatic food chain are presented in Table 4.1.

An overview of the current information on the levels of mercury in terrestrial and aquatic ecosystems is presented here. Unless otherwise noted, the mercury concentrations are reported on a wet-weight basis. The levels in the atmosphere, soils, and water are discussed in Chapter 2 and levels in foodstuffs are discussed in Chapter 5.

44

TABLE 4.1   Biological Magnification of Mercury in the Aquatic Food Chain

	Number of Samples	Range of Values ($\mu g/g$)	Arithmetic Mean ($\mu g/g$)	More Numerous Organisms
Algae Eaters	39	0.01–0.18	0.05	Zooplankton; Snails; Mayfly nymphs
Zooplankton Eaters	9	0.01–0.07	0.04	Insect larvae; Minnows
Omnivores	9	0.14–1.16	0.45	Insect larvae and adults; Scuds
Detritus Eaters	12	0.13–0.89	0.54	Worms; Clams; Insect larvae
Predators	25	0.01–5.82	0.73	Insect larvae and adults; Frogs

SOURCE: Hamilton (1971).

MUSEUM SPECIMENS AS POLLUTION INDICATORS

Analyzing several species of preserved birds, Swedish researchers discovered that alkylmercury fungicides introduced to treat seed grain in the early 1940s had increased the mercury levels in the feathers of seed-eating birds. These results motivated other investigators to analyze additional preserved specimens. When four late Pleistocene mammals were tested, no mercury was detected in the bone marrow of a horse. Tissues from the foot of a mammoth, however, tested at 0.06 μg/g, and muscle tissue from a bison and moose showed 0.70 and 0.95 μg/g, respectively (Harris and Karcher 1972).

The Ontario Ministry of the Environment has analyzed two major sets of museum fish, one from Lake St. Clair and consisting of about 600 preserved fish covering the period 1853 to 1969, the other comprising 100 fish from Lake Simcoe covering the interval 1914 to 1968 (J.N. Bishop, personal communication, Laboratory Services Branch, Ontario Ministry of the Environment, Rexdale, Ontario, 1977). The former study indicated that fish captured after 1959 had significantly higher mercury levels than those from pre-1959 times. The Simcoe study indicated no change for the only species adequately represented (whitefish).

Isolated specimens of Pleistocene mammals without modern equivalents are only of historical interest. However, efforts have been made to compare the mercury burdens of preserved fish collected 25 to 100 yr ago for the purpose of determining whether levels of mercury are increasing over time in particular species. Most of these studies, however, are likely to be of more academic than practical importance because of insufficient data and possible contamination of the samples. The major source of error is in the preservation process. Comparisons of mercury levels in museum specimens and fresh or frozen fish must be considered unreliable until the changes that result from preservation are more fully understood. Mercury can be introduced as a trace impurity with the preservative or on metal tags or other identification materials placed in the container with the fish. Gibbs et al. (1974) found that the mercury content of eight commercial brands of formaldehyde ranged between undetectable and 9 μg/l while metal identification tags contained between 30 and 50 μg Hg/g. Therefore, to provide a minimum degree of statistical confidence in the data, a number of preserved specimens of the same species, size, and age should be collected at each sampling location at selected time intervals; and the preservation and storage histories should be properly recorded. This

might be done for future comparisons, but most current
studies fall woefully short of these conditions and can
only confirm that mercury does appear in samples of fish
taken at earlier times.

Very few preserved marine fish have been analyzed, and
arguments continue over whether any reliable conclusions
can be drawn from the limited data (Barber et al. 1972,
Miller et al. 1972, Saperstein 1973, Gibbs et al. 1974).
More freshwater fish have been analyzed, and those
caught recently average more mercury than preserved
specimens, although this depends heavily on the degree
of pollution, size of the fish, and the year and
location where they were collected (Evans et al. 1972,
Harris and Karcher 1972). The limited data presently
available indicate that a small mercury burden may be
normal and of natural origin, especially in marine fish.
The levels in marine fish appear not to have changed to
any measurable degree since the 1870s; the higher levels
in freshwater fish are usually the result of additions
from anthropogenic sources. However, care must be
exercised in extrapolating the results of a very limited
number of analyses of specimens with unknown
preservation and storage histories. They lack enough
statistical confidence to draw comparisons that indicate
the degree and extent of mercury pollution in the
present marine ecosystem.

ALGAE, PLANTS, AND INVERTEBRATES

Algae

Aquatic organisms derive most of their energy from
algae at the base of the aquatic food chain. Very low
levels of mercury can kill some species of algae and
inhibit the growth of others, thereby disrupting the
complex interdependency between phytoplankton and the
populations that feed upon them. Furthermore, from this
initial point of entry, the element is concentrated up
the food chain.

Algae accumulate and concentrate mercury from the
water primarily by surface absorption and to a lesser
degree by adsorption (Hannerz 1968, Glooschenko 1969).
The process of uptake is essentially passive.
Freshwater algae and macrophytes were shown to
accumulate radioactive mercury-203 in aquarium
experiments by Makhonina and Gileva (1968).

Organomercurials retard the growth and viability of
several species of marine algae more effectively than
inorganic mercury (Boney et al. 1959, Boney and Corner
1959, Matida et al. 1971). As little as 0.1 μg/l of
some alkylmercurial fungicides decreased the growth and

photosynthesis of laboratory cultures of the marine
diatom Nitzschia delicatissima as well as some
freshwater phytoplankton. Panogen®, MEMMI®, and
phenylmercuric acetate (PMA) were lethal to both marine
and freshwater phytoplankton at 50 $\mu g/l$ while less than
0.6 $\mu g/l$ drastically limited their growth (Harriss et
al. 1970). PMA inhibited the growth of three
phytoplankton species at concentrations as low as 0.06
$\mu g/l$ (Nuzzi 1972). Boney and Corner (1959) concluded
that algae may be highly susceptible to alkylmercurials
because of the high lipid content of their cell
membranes while Matson et al. (1972) established that
mercuric chloride and methylmercuric chloride inhibited
the biosynthesis of lipids in selected species.

Young algae spores exposed to mercury may be retarded
in growth as well as in the formation and maturation of
reproductive structures, placing them at a competitive
disadvantage with other species (Boney 1971). In
addition, the response of phytoplankton to mercury
toxicity has been related to other chemicals in the
water. Increasing concentrations of other nutrient
elements in the water can decrease the accumulation of
mercuric chloride (Matida et al. 1971, Hannan and
Patouillet 1972), and, conversely, extremely low levels
may be lethal if other nutrients are insufficient
(Harriss et al. 1970).

Plants

Aquatic Plants

Although aquatic vegetation contains much more mercury
than the surrounding water, its total mercury level is
relatively low. Plants in unpolluted waters usually
measure between 0.03 and 0.08 $\mu g/g$ total mercury while
similar plants in polluted waters have concentrations up
to 37 $\mu g/g$.

While most of the mercury in an aquatic system is
associated with the sediments, higher plants form the
largest biomass component in many watercourses, and they
are capable of removing mercury from the water. The
uptake of both inorganic and organic mercury is
proportional to the length of exposure and the
concentration (Fang 1973, Mortimer and Kudo 1975).
Mortimer and Kudo found methylmercury to be more toxic
than mercuric chloride for the aquatic plant Elodea
densa. Fang demonstrated that Ceratophyllum demersum
and Elodea canadensis metabolized PMA into mercuric
mercury and small amounts of methylmercury and
ethylmercury, and that the biological half-life of
mercury residues ranged between 43 and 58 days.

Terrestrial Plants

All plants appear to concentrate traces of mercury, but the amount depends on the plant species and locality, and the chemical form of the mercury available. The latter is most important. Rooted plants absorb elemental mercury and alkylmercurials much more easily than they do ionic inorganic mercury (Dolar et al. 1971). Generally, total mercury concentrations in most common edible plants and foods derived from plants range from less than 1.0 to 300 ng/g, with the higher levels due to natural deposits in the soil.

Regardless of the mercury concentration in the soil, plants in well-aerated soils normally contain less than 0.1 to 0.7 μg/g on a fresh-weight basis. Plants have a barrier to the uptake and circulation of inorganic mercury salts and organically complexed mercurials adsorbed on clay, as in aerated soils, but apparently many plants have no barrier against the uptake of gaseous mercury through the roots. In soils where decaying sulfides release gaseous elemental mercury, the vegetation has from 0.2 to 10 μg/g on a dry-weight basis. In reducing soils, physical and chemical forces firmly hold the mercury in insoluble sulfides or organic complexes so that, typically, only from 0.01 to 0.04 μg/g is available to concentrate in the plants (Kothny 1973).

Invertebrates

Insects and Freshwater Invertebrates

Depending on the level of pollution, many invertebrate organisms tolerate and magnify various mercurials to some degree. In unpolluted Swedish waters, the level of mercury in caddisflies (Trichoptera), stoneflies (Plecoptera), alderflies (Neuroptera), and hogslater (Asellus) ranged between 0.025 and 0.072 μg/g. In waters polluted with phenylmercuric compounds, the levels were approximately 100 times greater and ranged from 1.9 to 17 μg/g (Johnels et al. 1967, 1968).

Although Hannerz (1968) found no direct correlation between an invertebrate's mercury burden and its trophic level, he did observe that predaceous insect larvae, such as dragonflies and alderflies (Sialis), accumulated more mercury than organisms which feed on decaying plants or detritis. The extent to that organisms concentrated mercury from water varied from <100 to >12,000-fold, depending on such factors as the form of the mercury, time of exposure, and nature of the food consumed, as well as the feeding habits and metabolic

activity of the respective organisms. They accumulate
more methyl, methoxyethyl, and phenylmercury than
inorganic mercuric ion. In some cases, the insect's
trophic level was less important than the quantity of
mercury available for uptake (Hannerz 1968). Among
larger invertebrates, the accumulation of mercury also
depends on the compound as well as the species. Table
4.2 summarizes the concentrations among inhabitants of a
pond ecosystem a month after it was treated with a
single application of various mercury compounds.

Marine Invertebrates

Accumulation. In highly contaminated marine
environments, invertebrates can concentrate large
amounts of mercury. Mercury levels in crayfish in the
Wabigoon River system in Ontario and in Minamata Bay
ranged from 0.87 to 35.7 μg Hg/g compared to a range of
0.09 to 0.27 μg Hg/g for crayfish in unpolluted waters.
Oysters (undisclosed species) at Minamata contained 5.61
μg Hg/g (Kitamura 1968) while oysters from Galveston and
Chesapeake bays contained between 0.30 and 1.00 μg Hg/g
(Kurland et al. 1960). In contrast, the average mercury
content of rock oysters (Crassostrea commercialis)
cultivated in relatively unpolluted industrialized and
nonindustrial areas of Australia contained between 0.003
and 0.017 μg/g (Hussain and Bleiler 1973).
Toxicity. Much of the current information about
marine invertebrates deals with acute mercury toxicity
as it varies among species and also within a particular
species according to its life stage. Larvae are
generally most sensitive (Connor 1972).
Lethal doses of inorganic mercuric salts have been
shown to relate directly to the species among
invertebrates and range between 0.05 and 1800 mg/l
(Jones 1940, Clark 1947, Barnes and Stanbury 1948,
Pyefinch and Mott 1948, Hunter 1949, Wisely and Blick
1967, Knapik 1969). Corner and Sparrow (1957) found
that brine shrimp and barnacle larvae were more
sensitive to organomercurials than mercuric chloride and
the toxicity levels reflected the rate of uptake for
both species. Test animals absorbed fat-soluble
organomercurial compounds into fatty tissue more rapidly
than inorganic mercuric chloride. Among the various
compounds containing radioactive mercury-203, n-
amylmercuric chloride was 20 times more toxic to
barnacles than mercuric chloride and 100 times more
toxic to brine shrimp (Corner and Sparrow 1957, Corner
and Rigler 1958).
Elimination. The patterns of elimination of
radioactive mercury compounds were similar for fish,

TABLE 4.2   Concentration Factors After a Month Following a Single Dose of Various Mercury Compounds into a Pond

	Mercuric Chloride	Methoxyethyl Hydroxide	Phenylmercuric Acetate	Methylmercuric Hydroxide
Water plants (six species)				
Submerged parts[a]	4–264	68–771	40–2350	34–3200
Emergent (three species)	3–77	4–53	8–90	8–25
Algae	252	920	1220	——
Moss	393	560	3900	5900
Invertebrates (four species)	247–560	510–1990	900–4200	3290–8470
Sediment	359	743	6800	6100

[a]Much of the variation is due to interspecific variation within the plants studies. For *Iris pseudacorus*, the values for the four compounds ranged from 4 to 98, while for *Lysimachia nummularia* the range was 264 to 3200.

SOURCE: Derived from Hannerz (1968).

freshwater crabs, other crayfish, and molluscs. The biological half-lives for mercuric and phenylmercuric nitrate injected into the foot muscle of a freshwater mussel were 23 and 43 days, respectively. The corresponding half-lives for methylmercuric nitrate varied between 86 and 435 days, with the shortest half-life in the youngest animal (Miettinen et al. 1969). Although the quality of the water did not affect the animal's rate of excretion, temperature did. The half-life was estimated to be as long as 2 yr in summer and 3 yr in winter.

Crayfish appear to retain more mercury and eliminate it more slowly when it is injected into muscle instead of the mouth, apparently because there the mercury is bound to protein in the tissues and central nervous system. Only 4 to 20 percent is rapidly eliminated according to Miettinen et al. (1968, 1969, 1972b). They estimate that the half-life of mercury in the bodies of the crayfish can range from 50 to 500 days depending on the temperature of the water and route of administration. Overall, an organism's retention of mercury depends primarily on the form and means of accumulation. These data are summarized toward the bottom of Table 4.3.

FISH

Freshwater and marine organisms and their predators normally contain more mercury than terrestrial animals. In general, the uncontaminated background levels for the flesh of animals, birds, and eggs are less than 0.02 μg/g. Levels of mercury in freshwater organisms and fish taken from areas free of known mercury contamination range from 0.02 to 0.2 μg/g. Levels in top predatory fish such as bass (Micropterus dolomieui), pike (Esox lucius), and walleye (Stizostedion vitreum) range from 0.4 to 1.0 μg/g. However, mercury levels vary widely depending on the age, weight, length, species, metabolic rate, degree of pollution, locale, and in some cases sex (Forrester et al. 1972, Olsson 1976, Bishop and Neary 1977). Moreover, statistical evaluation is seldom adequate because the values are usually based on small sample sizes, composite samples from many different watercourses, or extrapolations from the literature. Data on mercury levels in aquatic organisms are difficult to compare primarily because analytical uncertainties (Westoo 1975), poor experimental designs, and biased interpretations of the results have produced many ambiguities and contradictions (see Appendix A).

The predominance of methylmercury in fish (80 to 90 percent as compared with inorganic mercury) has been demonstrated by numerous analyses (Bache et al. 1971, Kamps et al. 1972, Westoo 1973, Bishop and Neary 1976), although lower percentages of methylmercury have occasionally been reported for some fish (Bache et al. 1973, Freeman and Horne 1973). Body burdens are elevated substantially where the watercourses are contaminated with mercury from industrial and mining processes as well as organic and inorganic nutrients from anthropogenic sources.

Accumulation and Elimination

Fish accumulate mercury in excess of the 0.5 $\mu g/g$ FDA interim guideline from waters with low natural levels as well as from those that are anthropogenically contaminated. This accumulation is part of a dynamic process in which an organism's body burden strives to maintain equilibrium between intake and elimination. As discussed in Chapter 3, bioaccumulation occurs when the rate of uptake exceeds that of elimination, and this depends on a number of physical, chemical, and biological parameters that vary in each aquatic ecosystem (Burrows et al. 1974, Jernelov et al. 1975).

Uptake

The mercury accumulated in fish comes primarily through absorption from the water across the gills or through the food chain (Boetius 1960, Hannerz 1968, Hasselrot 1968, Amend et al. 1969, Backstrom 1969, Rucker and Amend 1969, Olson et al. 1973, Olson and Fromm 1973, Uthe et al. 1973, de Frietas et al, 1974, Hasselrot and Gothberg 1974), although some higher species may convert inorganic mercury into methylmercury (Westoo 1968, Imura et al. 1972). Some mercury is also taken up through the fish's mucus layer and/or skin (McKone et al. 1971, Burrows et al. 1974). The short chain alkylmercurials are absorbed much faster than alkoxyalkyl, aryl, or ionic inorganic mercurials (Hannerz 1968, Backstrom 1969).

Comprehensive data are not available on the feeding habits of fish. However, Jernelov (1972) and Jernelov and Lann (1971) estimated that fish at the higher trophic levels obtain half of their mercury burden through their food and the remainder from the water; fish at the lower trophic levels receive as much as 75 percent of their mercury burden from the water. On the other hand, Hannerz (1968) reported that animals

TABLE 4.3 Biological Half-life of the Slow and Fast Component of Excretion for Ionic and Protein-Bound Radioactive Mercurials

Aquatic Organism	Mercury Form	Method of Administration	Duration of Experiment (days)	Temp °C	Fast Excretion as Percent of Original Activity (days)*	Slow Excretion Biological 1/2 Life (days)*	Reference
Flounder *(Pleuronectes flesus)*	MMN	POP	100	13–19	33±4	780±120	a
Flounder *(Pleuronectes flesus)*	MMN	POI	100	13–19	12±4	700±50	a
Flounder *(Pleuronectes flesus)*	MMN	IMI	100	13–19	——	1200±400	a
Flounder *(Pleuronectes flesus)*	MMN	OBS	28	13–19	17±3	169±61	b
Flounder *(Pleuronectes flesus)*	MMN	OBS	77	13–19	23±2	430±115	b
Flounder *(Pleuronectes flesus)*	PMN	OBS	28	13–19	28±10	58±8	b
Flounder *(Pleuronectes flesus)*	PMN	OBS	77	3–19	40±7	164±16	b
Perch *(Perca fluviatilis)*	MMN	OBS	9–28	13–19	12±5	112±21	b
Perch *(Perca fluviatilis)*	MMN	OBS	77	3–19	8±8	470±37	b
Perch *(Perca fluviatilis)*	MMN	OBS	77	3-19	30	>470	b
Perch *(Perca fluviatilis)*	PMN	OBS	34	3–19	32	157	b
Pike *(Esox lucius)*	MMN	POP	130	13–19	~10	750±50	a
Pike *(Esox lucius)*	MMN	POI	130	13–19	~5	640±120	a
Pike *(Esox lucius)*	MMN	IMI	130	13–19	——	780±80	a
Pike *(Esox lucius)*	MMN	OBS	20	16-19	10±5	140±40	b
Pike *(Esox lucius)*	MMN	OBS	84	3–19	11±1	490±30	b
Pike *(Esox lucius)*	PMN	OBS	21–53	11–18	35–41	64±4	b
Pike *(Esox lucius)*	PMN	OBS	84	3–18	30	190	b
Roach *(Leucisus rutilus)*	MMN	OBS	8	15–17	12±4	25±9	b
Sea Bass *(Serranus scriba)*	MMN	OBS	60	17–23	4–6	267±27	c
Eel *(Anguilla vulgaris)*	MMN	POP	130	13–19	~17	910±40	a
Eel *(Anguilla vulgaris)*	MMN	POI	130	13–19	~17	1030±70	a
Eel *(Anguilla vulgaris)*	MMN	IMI	130	13–19		1030±80	a

54

Mollusc *(Tapes decussatus)*	MMN	IMI	99	17–23	20	481±40	c
Mollusc *(Tapes decussatus)*	MC	IMI	——	– –	——–	~100	d
Mollusc *(Tapes decussatus)*	MC	ASW	——–	——–	——	10 days	d
Mollusc *(Tapes decussatus)*	MC	RSF	——	——	——–	5 days	d
Mollusc *(Lymnaea stagnalis)*	MMN	OBS	14–29	18±2	12	27±13	b
Mollusc *(Mytilus galloprovincialis)*	MMN	IMI	98	17–23	20	1000	c
Mussel *(Pseudanodonta complenata)*	MMN	IMI	275	13±6	7	435	b
Mussel *(Pseudanodonta complenata)*	MMN	IMI	63–106	17±3	10±3	129±22	b
Mussel *(Pseudanodonta complenata)*	MMN	IMI	54–106	17±3	9±5	86±10	b
Mussel *(Pseudanodonta complenata)*	PMN	IMI	6–40	8±2	3±3	43±7	b
Mussel *(Pseudanodonta complenata)*	MN	IMI	12–15	8±2	3±3	23±6	b
Crayfish *(Astacus fluviatilis)*	MMN	IMI	63	18±2	5	297	b
Crayfish *(Astacus fluviatilis)*	MMN	OBS	14–63	18±2	13±4	144±37	b
Crab *(Carcinus maenas)*	MMN	IMI	35	17–23	4–6	400±50	c
Seal *(Pusa hispida)*	MMN	POP	153	——–	20	~500	e

*Error given as mean deviation from mean.

MMN--methylmercury nitrate
PMN--phenylmercuric nitrate
MC--mercuric chloride
MN--mercuric nitrate
POP- per os, proteinate-bound

POI—per os, free ionic form
IMI--into muscle, ionic form
ASW—absorption from sea water
RSF—resorption from food
OBS—orally administered into stomach of organism

References: a--Järvenpää et al. (1970).
b- Miettinen et al. (1969).
c--Miettinen et al. (1972b).
d--Unlü et al. (1972).
e Keckes and Miettinen (1972).

bioaccumulate mercury in direct relation to such factors as metabolic rate and food habits rather than position on the trophic scale. Since a fish's metabolic rate is regulated by the temperature of the water, more mercury is concentrated in the summer than in the winter (Hannerz 1968, Hasselrot 1968, MacLeod and Pessah 1973, Uthe et al. 1973, Blaylock and Huckabee 1974, Reinert et al. 1974). Similarly, because of its higher metabolic rate, red muscle tissue takes up mercury faster than white muscle (Backstrom 1969). The metabolic rate of the fish and the mercury concentration in the aquatic ecosystem appear to be more important factors in bioaccumulation than age or exposure rate (Johnels et al. 1967, 1968; Olsson 1976).

For inorganic mercurials such as mercuric nitrate, concentration factors of between 1 and 50 have been measured for fish whereas for methyl and methoxyethyl mercury the organs and tissues of fish had values between 1000 and 2500 (Hannerz 1968). Estimates of experimental bioconcentration factors for methylmercury range from 3000 for pike (Johnels et al. 1967) to more than 8000 for rainbow trout (Salmo gairdneri) (Reinert et al. 1974). In addition, Underdal and Hastein (1971) reported biological magnification factors ranging from 7000 to 10,000 for rainbow trout downstream from a wood pulp factory.

The amount of mercury the fish are able to tolerate depends on the rate of uptake as well as the quantity and form administered. Miettinen et al. (1969, 1972a) showed that the levels of methylmercury in brain and nerve tissues tended to remain constant or even to increase without being supplemented, whereas phenylmercury remained in the digestive organs and was excreted much more rapidly.

Elimination

Large concentrations of mercury found in fish are caused not only by high rates of absorption but also by slow rates of elimination. A two-phase elimination process was observed in laboratory studies: a relatively fast phase when the fish's mucus layer sloughs off, and a slower second phase attributed to losses from muscle tissues (Massaro and Giblin 1972, Burrows et al. 1974). Dilution by growth of new flesh also affects the half-life of mercury in living organisms.

The rate of elimination depends largely on the specific compound and the metabolic rate of the fish. Matida et al. (1971) determined that rainbow trout eliminated methylmercuric chloride more slowly than

ethylmercuric phosphate, phenylmercuric acetate, or
mercuric chloride. When mercury intake ceases, warmer
temperatures may speed the rate of elimination, while
the growth of new tissue further decreases the mercury
concentrations (MacLeod and Pessah 1973).

While the biological half-life of mercury can vary
widely depending on its chemical form and the species of
aquatic organism, in general, a half-life of at least 2
yr is normal for fish in the natural environment
(Lockhart et al. 1972, Bishop and Neary 1974, Hasselrot
and Gothberg 1974, Laarman et al. 1976). Table 4.3
presents the biological half-lives of mercury for
various aquatic species, in terms of both the slow and
fast phases of excretion.

Freshwater Fish

Analyses conducted by Stock and Cucuel (1934) and
Raeder and Snekvik (1941) established that in unpolluted
areas both marine and freshwater fish normally
concentrate in the range of 0.04 to 0.15 μg/g mercury,
concentrations being generally higher in predatory than
bottom feeding species. After Swedish researchers began
to analyze for methylmercury in the 1960s, they found
elevated mercury levels in fish from contaminated fresh
waters. Subsequently, levels in excess of 1 μg/g were
reported in fish from Norway (Underdal 1969), Denmark
(Dalgaard-Mikkelsen 1969), Canada (Wobeser et al. 1970;
Bligh 1970, 1971; Bishop and Neary 1976; MOE 1977),
Finland (Aho 1968), Italy (Ui and Kitamura 1971), and
the United States (Bails 1972, Henderson et al. 1972,
Henderson and Shanks 1973). Where waterways are highly
contaminated, average mercury levels in adult predatory
fish may approach 10 μg/g, and individual fish may
exceed 24 μg/g wet weight (Bishop and Neary 1974).

In Canada the mercury content of fish varies widely.
In 1975 Bishop and Neary (1976) determined the mercury
levels in over 11,000 fish representing 19 species from
47 contaminated and uncontaminated lakes in northwestern
Ontario. In all of the interlake comparisons, fish
sampled covered a wide range of lengths and weights, so
data were normalized to allow comparisons of specified
lengths and species from one lake to another, and from
one year to another for a given lake. In contaminated
lakes the mean normalized mercury concentrations ranged
from 0.40 to 1.21 μg/g for 25-in. pike, 0.54 to 1.32
μg/g for 20-in. walleye, and 0.04 to 0.28 μg/g for 20-
in. whitefish (Coregonus clupeaformis). The ranges were
even wider for anthropogenically contaminated lakes and
waterways: from 2.87 to 5.25 μg/g for 25-in. pike, 1.97

to 6.90 µg/g for 20-in. walleye, and 0.55 to 0.77 µg/g for 20-in. whitefish.

Two of the most seriously polluted Canadian watercourses are the Wabigoon-English-Winnipeg river system in northwestern Ontario (Fimreite and Reynolds 1973, Annett et al. 1975) and the Lebel-sur-Quevillion region of northwestern Quebec (Barbeau et al. 1976). In the lakes and rivers polluted by a chloralkali plant near Lebel-sur-Quevillion, the mercury content of predatory fish such as pike and walleye has ranged between 0.08 and 4.88 µg/g since 1971. The highest known levels of mercury in the Western Hemisphere were reported for fish taken from Clay Lake on the Wabigoon-English-Winnipeg river system. In 1970 the mean mercury levels were as follows: pike, 9.24 µg/g (range 3.79 to 14.9); walleye, 12.1 µg/g (range 1.2 to 24.0); burbot (Lota lota), 21.95 µg/g (range 19.1 to 24.8); whitefish, 3.58 µg/g (range 0.15 to 12.57); and white sucker (Catostomus commersoni), 3.83 µg/g (range 1.68 to 7.97) (Bishop and Neary 1976). Only at Minamata and Niigata, Japan, were higher mercury levels reported in fish and shellfish from waterways that received direct discharges of methylmercury (Kitamura 1968, Tsubaki 1971).

On the Wabigoon River and Clay Lake the average mercury concentrations of some species of fish exceeded 10 µg/g in 1970. The highest levels reported for walleye, pike, and burbot were 24.0, 27.8, and 24.8 µg/g, respectively (Fimreite and Reynolds 1973, Bishop and Neary 1976). For some lakes on the Wabigoon-English-Winnipeg river system no measurable decreases in the mean mercury levels have been observed for selected species of fish since 1970, while for selected species of fish from other lakes on the same system mean concentrations have decreased as much as 50 percent (Bishop and Neary 1976). More dramatic decreases were reported for fish taken from Lake St. Clair, the international waterway between Canada and the United States, after the major source of pollution, a chloralkali plant on the St. Clair River at Sarnia, Ontario, sharply reduced discharges in 1970 and totally eliminated them in 1975. While it is commonly believed that biomethylation of inorganic mercury was primarily responsible for the presence of methylmerucry in Lake St. Clair, the Ontario Water Resources Commission (Jernelov et al. 1972) established that a transalkylation of mercury with alkyl-lead compounds was an important source of both methyl- and ethylmercury to the system. Later, the Ontario Ministry of the Environment (MOE 1977) compared mercury concentrations in fish of similar size taken from Lake St. Clair between 1970 and 1976; the pattern of reduction closely approximates an exponential decline. In 1970 a 40-cm

walleye had a mean mercury level of 2.1 µg/g whereas by 1976 a similar fish contained only 0.56 µg/g, a 73 percent reduction.

The Canadian researchers observed similar declines for all of the species tested from Lake St. Clair. American scientists have also observed these decreases but report a somewhat less dramatic decline rate. This discrepancy may not be significant because of the differences in the age, length, and weight of the fish considered and the statistical interpretation of the data. The average mercury concentration in yellow perch (Perca flavescens), rock bass (Ambloplites rupestris), and channel catfish (Ictalurus punctatus) dropped below the 0.5 µg/g FDA interim guideline by 1972; but some American scientists suggest that 5 yr or more will be needed for walleye 56 cm or larger to reach this level (W.A. Willford, personal communication, U.S. Fish and Wildlife Service, Great Lakes Fishery Laboratory, Ann Arbor, Michigan, 1977). Large adult walleye decreased their average mercury concentrations approximately 51 percent, from 2.83 µg/g in 1970 to 1.38 µg/g in 1972. From 1972 to 1975 the average mercury content decreased an additional 25 percent, but at a much slower rate, and this slowed decline continues.

Such rapid decreases may not occur in areas where contamination is removed less quickly. Lake St. Clair is a very shallow lake, and the approximately 8- to 9-day turnover is unusually rapid. A strong river current and the propellers on large numbers of lake freighters quickly translocate the fine sediment to Lake Erie. When Thomas et al. (1975) compared mercury concentrations in the sediments of Lake St. Clair in 1970 and 1974, they concluded that 64 percent had been transported downstream to Lake Erie. While the levels of mercury in the Lake Erie sediments have not decreased substantially since 1970, a significant decrease in the mercury levels in the fish has been observed (James N. Bishop, personal communication, Laboratory Service Branch, Ontario Ministry of the Environment, Rexdale, Ontario, Canada, 1977). Therefore, the rapid decline in mercury levels in fish is probably due to the combined effects of the elimination of the major source of mercury pollution, the rapid turnover time in the waterway, and the translocation of mercury contaminated sediments out of the lake.

Marine Fish

As with freshwater fish, mercury levels in marine fish vary markedly depending on the species, metabolic rates, age, weight, and geographical locations. Not all of the

responsible factors have yet been explained, but average mercury levels are usually below 0.10 µg/g except for large carnivorous fish at the end of a long food chain, such as tuna and swordfish. Their mercury levels may exceed 1 µg/g (Doi and Ui 1975). However, where marine organisms were exposed to methylmercury from industrial effluent discharged directly into the ocean, fish and shellfish bioaccumulated up to 40 µg/g. These levels were reported at Minamata Bay, Japan, in the early 1950s (Kitamura 1968) and at Niigata, Japan, in 1965 (Tsubaki 1971).

Marine fishes generally have much lower total mercury levels than freshwater fish, and the ratio of inorganic mercury to methylmercury also varies more widely and may depend on the species. Almost all of the mercury contained in freshwater fish is in the form of methylmercury. The same is true of tuna and swordfish (Berglund et al. 1971, Doi and Ui 1975). However, in Pacific blue marlin (Makaira ampla) taken off the Hawaiian coast, only about 25 percent of the total mercury is in the form of methylmercury (Rivers et al. 1972).

In most cases, the mercury levels in marine fish seem to depend on where the fish were caught. In the United Kingdom, fish from the Thames estuary and eastern Irish Sea contained the highest levels, 0.45 to 0.5 µg/g and 0.55 to 0.64 µg/g, respectively. The mean concentration for fish caught within 25 miles of the coast was 0.29 µg/g but varied widely from area to area. Further offshore, the fish averaged 0.11 to 0.21 µg/g, and fish from the open sea ranged between 0.03 and 0.06 µg/g. Overall, the mean mercury concentration for fish and shellfish in the United Kingdom was estimated at 0.08 and 0.13 µg/g, respectively (U.K. Department of the Environment 1976). In 1973 cod, haddock, and plaice contained average mercury concentrations of between 0.04 and 0.13 µg/g. These three species accounted for 61 percent of the total fish caught in the United Kingdom.

The mercury levels in cod (Gadus morrhua) varied according to where they were taken. Those from the heavily contaminated waters of the western Baltic between Denmark and Sweden had values up to 1.29 µg/g. On the other hand, cod taken around Greenland had between 0.013 and 0.026 µg/g and North Sea cod ranged from 0.158 to 0.195 µg/g (Dalgaard-Mikkelsen 1969). In a similar study, Hall et al. (1976a) found that among 1227 Pacific halibut (Hippoglossus stenolepis) mercury concentrations in fish of the same size increased from the northern to the sourthern part of the species' ranges. Within each geographical area, the mercury concentration also increased with the size of the fish in a range from 0.15 to 0.45 µg/g. The element was

uniformly distributed throughout the edible muscle
tissues.

A study undertaken by the National Marine Fisheries
Service (Hall et al. 1976b) traced mercury levels in 205
species of finfish, molluscs, and crustaceans. These
species represented approximately 93 percent of the U.S.
commercial catch. The samples were taken from 198 sites
around the United States, including Alaska and Hawaii.
The mean levels of mercury exceeded 0.5 µg/g in less
than 2 percent of the 159 species caught for human
consumption. Finfish represented 63.9 percent; most of
their muscles had a mean mercury level below 0.3 µg/g.
Only 31 species of finfish (1 percent of the total 63.9
percent) contained mean mercury levels that exceeded the
0.5 µg/g FDA interim guideline.

Zook et al. (1976) surveyed 314 samples of 32 commonly
consumed fresh fish and shellfish for their total
mercury. The mean concentration was 0.12 µg/g. Only
three species--halibut, rockfish, and red snapper--
exceeded the 0.5 µg/g level prescribed in the FDA
interim guideline. The highest levels were 0.52, 0.85,
and 0.55 µg/g, respectively. The 183 samples of
shellfish (oysters, clams, scallops, shrimp, and crabs)
averaged 0.05 µg/g, with only 10 samples exceeding 0.10
µg/g. The leg meat of a king crab was highest at 0.13
µg/g. Overall, the mean mercury level for the fish and
shellfish samples was 0.13 µg/g with more than 96
percent testing below the FDA interim guideline of 0.5
µg/g. Similarly, among cod, clam, crab, flounder,
herring, lobster, and oysters taken off the Atlantic
coast in Canada, the mercury concentrations were 0.20
µg/g or less (Bligh 1971).

As with the National Marine Fisheries Service study,
tuna and swordfish taken off the Atlantic coast of
Canada had higher mercury concentrations than other
fish. Concentrations ranged between 0.33 and 0.86 µg/g
and 0.82 and 1.00 µg/g, respectively (Bligh 1971).
Additional data are needed to determine whether these
species contain more mercury because of physiological
peculiarities or generally increased levels in the
marine ecosystem. Environmental pollution of their
habitat, as well as age, weight, metabolic rate, and
feeding habits, may all be contributing factors. An
age-dependent increase has been indicated by many
analyses. Furthermore, scombriformes species (spiney-
finned, as mackerels, tunas, bonitas, albacores, and
swordfish) have a high rate of metabolism and a high
level of food intake. Muscle tissue in tuna and
swordfish generally also contains about twice as much
dry matter as muscle tissue in other fish (Lofroth
1973). Among 88 yellowfin tuna (_Thunnus_ _albacares_),
those that weighed less than 25 kg had mercury levels no

higher than 0.25 µg/g, those of 50 kg had mercury body burdens as high as 0.50 µg/g, while tuna that weighed between 60 and 100 kg had mercury levels as high as 1.0 µg/g. However, the larger fish also had a wide variation of mercury content (Doi and Ui 1975).

Various studies of the most common types of tuna have reported mercury levels that range widely and may depend on a number of factors. Among 911 samples of skipjack (Katsuwonus pelamis), yellowfin, and white tuna, mercury levels ranged from undetectable to 1.0 µg/g with most values between 0.20 and 0.30 µg/g. Most of the bluefin tuna (Thunnus thynnus)(285 samples) from the Bay of Biscayne had values near 0.5 µg/g in a range from 0.20 to 0.80 µg/g. The same species (136 samples) taken from the Mediterranean Sea ranged from 0.50 to 2.5 µg/g with most values close to 1.1 µg/g. Twenty samples of bigeye tuna (Thunnus obesus) of various origins had mercury levels ranging from 0.4 to 1.0 µg/g. Over 5200 samples of tuna (variety not specified) from Italy ranged from undetectable to 1.75 µg/g, with most mercury levels between 0.3 and 0.5 µg/g wet weight (U.K. Department of the Environment 1976).

BIRDS AND MAMMALS

Aquatic Birds

Determining the impact of contamination on aquatic birds is complicated because many of them live longer and migrate over a larger part of the hemisphere than their terrestrial counterparts. Exposure (including migratory patterns) and eating habits are the most significant factors in the concentration of mercury by aquatic birds. In seedeaters mercury levels can be reduced by eliminating mercury seed treatments. Plant eaters are not apt to concentrate much mercury from the little that is translocated. However, where fish concentrate extraordinarily high levels of methylmercury, so do birds that prey on them. The impact of mercury contamination has been assessed quite extensively; many investigators have reported on the content of liver and muscle tissues in aquatic bird species from various countries (Borg et al. 1969, Holt 1969, Koeman et al. 1969, Muto and Suzuki 1969, Environment Canada 1971, Fimreite et al. 1971, Johnson and Morris 1971, Koeman and van Genderen 1972, Vermeer and Armstrong 1972, Parslow 1973, Vermeer et al. 1973, Heath and Hill 1974, Annett et al. 1975).

Species of ducks that eat plants and seeds are least apt to concentrate mercury. The American widgeon (Anas americana) eats approximately 89 percent plant food

during its first 50 days of life, and the levels in five of them averaged only 0.5 μg/g in the breast tissues even along the highly contaminated Wabigoon-English-Winnipeg river system. On the other hand, young mallards (Anas platyrhynchos) have a 90 percent animal diet, and 16 of them averaged 6.1 μg Hg/g in their breast muscles. Blue-winged teal (Anas discors) eat approximately equal parts of plant and animal food, and their breast tissues ranged from 3.8 to 10.4 μg/g (Vermeer et al. 1973).

In the same study along the Wabigoon-English-Winnipeg river system, Vermeer et al. (1973) found that birds that ate more fish had the highest mercury levels. Among 21 common goldeneye (Bucephala clangula), the average was 7.8 μg/g. Seventeen common mergansers (Mergus merganser) averaged 6.8 μg/g, and seven hooded mergansers (Lothodytes cucullatus) averaged 12.3 μg Hg/g in their breast muscles. Crayfish in the stomachs of hooded mergansers contained an average of 7.1 μg Hg/g in 1973. Five ducks were tested for methylmercury content and had from 69 to 99 percent of the total mercury as methylmercury.

Among puffins, guillemonts, cormorants, gannets, gulls, ducks, and other fish-eating birds, investigators measured mercury levels in liver ranging between 0.05 and 175 μg/g while muscle tissues ranged between 0.02 and 23 μg/g. In highly polluted areas fish eaters such as herons, teal, mallard, and mergansers had levels in muscle tissue between 0.51 and 23.0 μg/g (Dustman et al. 1972, Vermeer et al. 1973, Annett et al. 1975).

Migratory birds may detoxify if they spend part of the year in uncontaminated areas. The osprey (Pandion haliaetus), which migrates between Europe and the Mediterranean or Africa, has a half-life of 2 to 3 months for methylmercury (Berg et al. 1966, Johnels et al. 1968). Thus, feathers grown in a contaminated part of central Sweden during June and July had 20 μg Hg/g whereas those grown in October and November while the birds wintered in the south had 5.0 to 6.3 μg/g. By March the levels were down to between 1.8 and 2.3 μg/g.

On the other hand, along the heavily used Mississippi Flyway, ducks may be exposed year-round if they summer on a highly polluted waterway such as the Wabigoon-English-Winnipeg river system and then migrate south to winter on the polluted Galveston Bay. Ducks taken from Lake St. Clair, North Dakota, and the Wabigoon-English-Winnipeg river system have exceeded the FDA interim mercury guideline of 0.5 μg/g; and in 1970, hunters were advised not to eat the common goldeneye, blue-winged teal, and mallard because of excess mercury in their breast muscles (Environment Canada 1971).

Birds, such as the white-tailed eagle (<u>Haliaetus</u> <u>albicilla</u>), that prey on fish but do not migrate are unable to detoxify by changing food supplies. Consequently, in 1965 some Swedish white-tailed eagles had from 40 to 65 μg Hg/g in their feathers. Their eggs seldom hatched after feather concentrations reached between 5 and 11 μg/g. The greater incidence of sterility corresponded with elevated mercury in the birds' feathers, whereas those from uncontaminated areas had feathers comparable to the nineteenth century museum specimens.

The total mercury content of eggs from aquatic bird species in various locales has been reported to range between 0.03 and 15.8 μg/g (Eades 1966, Fimreite et al. 1971, Vermeer 1971, Wahlberg et al. 1971, Dustman et al. 1972, Faber and Hickey 1973, Greichus et al. 1973, Holden 1973a, Blus et al. 1974). In contaminated areas of Alberta, Saskatchewan, and Manitoba provinces, eggs with from 0.9 to 22.7 μg/g hatched normally in 18 nests. One fledgling's liver, however, contained 10 μg Hg/g (Vermeer et al. 1973). In California, instead of detoxifying their own systems by concentrating mercury in their eggs, female gulls had mercury levels that ranged 5.5 times higher than the 0.1 to 0.4 μg/g in their eggs. Nonetheless, according to Wahlberg et al. (1971), in isolated cases human populations may be at risk when mercury contaminated wild duck eggs are consumed.

Terrestrial Birds

Like fish, wild birds concentrate the highest levels of mercury in the kidney and liver with less in the muscle tissues. Although it is difficult to determine the natural mercury levels for birds in uncontaminated areas, Holden (1972) suggests numerous values below 0.1 μg/g in liver, kidney, and muscle tissues which indicate that the natural levels for uncontaminated areas range between 0.01 and 0.1 μg/g.

In the late 1950s Swedish ornithologists observed the first mercury-related ecological problems. Many species of birds, especially birds of prey, declined both in numbers and breeding success. At the same time, mercury levels increased in the feathers of several species of seed-eating and predatory birds compared to preserved specimens (Berg et al. 1966). Mercury levels in the peregrine (<u>Falco</u> <u>peregrinus</u>) increased from a mean of 2.5 μg/g for the years 1834 to 1849 to more than 40 μg/g for the years 1941 to 1965. Eagle owls (<u>Bubo</u> <u>bubo</u>) and white-tailed eagles showed similar increases. For goshawk (<u>Accipiter</u> <u>gentilis</u>) the average mercury content

was 2.2 μg/g prior to 1947, but between 1948 and 1965 the mean raised to 29 μg/g with some values in excess of 80 μg/g (Edelstam et al. 1969).

Seed-eating birds and their predators were widely contaminated by eating alkylmercury treated seed grain left uncovered during the spring and fall planting seasons (Johnels and Westermark 1969). Very high levels of mercury (up to 270 μg/g) were measured in the livers and kidneys of dead Swedish birds. Live birds had lower levels (1 to 53 μg/g)(Borg et al. 1966, 1969). After 1966 when the alkylmercurial seed dressings were replaced with alkoxyalkylmercury compounds, mercury concentrations declined significantly in seed-eating birds and their predators (Johnels and Westermark 1969). The mercury content of chicken eggs sold on the open market in Sweden also declined from 29 ng/g in 1964 to 9 ng/g in 1966 (Westoo 1969b), and similar decreases were observed in other foods. The mercury content of the liver and muscle tissues of terrestrial birds have been reported by many investigators (Borg et al. 1966, 1969, 1970; Eades 1966; Wanntorp et al. 1967; Holt 1969; Koeman et al. 1969; Fimreite et al. 1970; Anderson and Stewart 1971; Belisle et al. 1972; Huckabee et al. 1972; Martin 1972; Wiemeyer et al. 1972; Brock et al. 1973; Buhler et al. 1973; Griffith 1973; King and Lauckhardt 1973; Martin and Nickerson 1973; Smith 1973; Weigand 1973; Fimreite 1974; Kreitzer 1974).

In the United States and Canada, mercury-treated seed dressings also elevated the levels in seed-eating birds and their predators. In Canada the average mercury levels were 1.63, 1.88, and 1.25 μg/g for seed-eating songbirds, upland game birds, and rodents while the corresponding levels in similar specimens collected from an untreated area were significantly lower: 0.03, 0.35, and 0.18, respectively (Gurba 1970, Fimreite et al. 1970).

In 1970 both countries banned alkylmercurial seed dressings, and the levels decreased in game birds that do not feed on aquatic organisms. However, where phenylmercuric seed dressings continue to be applied in the United States, pheasants (Phasianus colchicus) and other wild birds can still accumulate up to 0.4 μg/g in the spring when the treated seeds are sown. The levels usually fall to less than 0.05 μg/g by summer.

Marine Mammals

At the top of the food chain, some larger marine mammals are showing high mercury concentrations in some organs (Helminen et al. 1968, Heppleston and French 1972, Sergeant and Armstrong 1973, Anas 1974). A number

of these animals have been tested along the California
coast and even in the Canadian Arctic where they were
presumed to be far removed from industrial pollution.
At Hudson's Bay, a beluga whale (Delphinapterus leucas)
had significant levels of total mercury in various
organs: muscle, 0.97 μg/g; liver, 8.87 μg/g; kidney,
2.44 μg/g; and heart, 1.35 μg/g (Bligh 1971). Levels of
mercury in the livers of six pilot whales (Globicephala
scammoni) stranded off the California coast ranged from
8.5 to 23.9 μg/g (Anonymous 1971). Male and female
harbor porpoises (Phocoena phocoena) taken from the Bay
of Fundy also had significantly different mercury
levels. Among 41 muscle and 20 liver samples, the total
mercury for male muscle tissues averaged 0.75 μg/g in a
range from 0.21 to 1.92 μg/g. Female muscle tissue
averaged 1.02 μg/g in a range from 0.26 to 2.58 μg/g.
The liver concentrations ranged from 0.89 to 18.30 μg/g
and 0.55 to 91.30 μg/g for males and females,
respectively. Essentially all of the mercury associated
with the muscle tissues was methylated whereas in the
livers, methylmercury represented from 7.4 to 41 percent
of the mercury present (Gaskin et al. 1972).

California sea lions (Zalophus californianus
californianus) have elevated mercury, DDT, and PCB
levels; and premature births have increased in their
breeding rookeries since 1968. Whether the cause is one
or all of these has not been determined, but females
have mercury residues ranging from 38 to 64 μg/g in
their livers (Delong et al. 1973).

Seals taken from widely distant areas have shown
mercury concentrations that exceed background levels.
Adult harp seals (Pagophilus groenlandicus) averaged
0.34 and 3.68 μg/g in the muscle tissue and liver,
respectively (Sergeant and Armstrong 1973). Harbour
seals (Phoca vitulina) from the Bay of Fundy and Gulf of
Maine had average total mercury levels of 0.054 μg/g
(range, 0.027 to 0.106), 0.59 μg/g (range, 0.16 to
1.54), 8.75 μg/g (range, 0.52 to 50.9), and 0.27 μg/g
(range, 0.05 to 0.76) in their blubber, muscle, liver,
and cerebrum, respectively (Gaskin et al. 1973). Along
the Netherlands coast, harbor seals exposed to high
levels of mercury from industrial pollution had mean
mercury levels of 340 μg/g, 20 to 50 times more than the
values reported in other studies. These seals showed
definite pathological signs despite a strong correlation
between the mercury and selenium levels in their livers
(see Chapter 5) (Koeman et al. 1973).

In Scotland liver mercury levels in gray seals
(Halichoerus grypus) normally range from 10 to 50 μg/g,
but up to 720 μg/g were found in areas where the levels
in fish gave no indication of mercury contamination.
The highest values appeared mainly in very old seals

(Holden 1973a, 1973b). In Canada gray seals had up to
2.35 μg/g in the muscle and 387 μg/g in the liver
(Sergeant and Armstrong 1973). In British waters the
brains of both common seals and gray seals concentrated
0.3 to 0.7 μg Hg/g when they were from 12 to 18 months
old whereas the livers of older seals concentrated 4.9
to 113.0 μg/g. Seals from East Anglia and West Scotland
accumulated more mercury than those from the Outer
Hebrides, Shetland, and Farne islands (Heppleston and
French 1972).

In the Canadian Northwest Territory, ringed seals
(Phoca hispida) showed mean mercury levels of 27 μg/g in
liver and 0.53 μg/g in muscle. Bearded seals
(Erignathus barbatus) had 143 μg/g in their livers and
0.53 μg/g in muscle. The mercury content was positively
correlated with age and body weight, but methylmercury
was only a small fraction of the total, 5.6 and 0.38
percent, respectively (Heppleston and French 1972,
Sergeant and Armstrong 1973, Anas 1974, Smith and
Armstrong 1975).

One member of a relic population of ringed seals
(Phoca hispida saimensis) in Lake Saimaa, Finland, was
captured alive because of impaired coordination that
resembled the symptoms of mercury poisoning. The
animals had been feeding on fish containing 0.2 μg Hg/g,
and it had concentrated 210 μg/g in the liver and 197
μg/g in the flesh. Two other seals had 74 and 130 μg/g
in their livers (Henriksson et al. 1969). A 9-month-old
female ringed seal was fed radioactive (mercury-203)
methylmercury in herring to determine retention and
excretion rates. Some mercury was excreted as the body
levels increased within the first 3 weeks, but 40
percent redistributed in the body fat and was retained
for a biological half-life of 500 days, approximately
the same excretion rate as for other sea animals
(Tillander et al. 1972).

Terrestrial Mammals

The mercury burdens in terrestrial mammals usually are
directly related to their diets and are low compared
with marine mammals. Herbivores normally have the
lowest mercury levels while carnivores that prey on
aquatic organisms have the highest body burdens. Borg
et al. (1969) reported that herbivorous animals such as
the roe deer (Capreolus capreolus) and hares (Lepus
timidus, L. europaeus) had less than 1 μg/g in mixed
liver and kidney, while the mercury levels in
carnivorous animals such as marten (Martes martes),
polecat (Mustela putorius), and fox (Vulpes vulpes)
exceeded 30 μg/l in mixed liver and kidney. Johnels and

Westermark (1969) reported that herbivores such as the
cow, horse, moose, and deer concentrate only from 0.007
to 0.075 µg Hg/g in their tissues. However, in Alberta,
where organomercurial seed dressings were used, the
livers of ground squirrel (Spermophilus richardsonii)
averaged 1.05 µg/g. In Saskatchewan liver mercury
concentrations in the same species averaged only 0.10
µg/g (Fimreite et al. 1970).

In 1972, 48 fur bearing animals from the Bell-Nottaway
basin in northwestern Quebec had low mercury residues in
muscle tissue. For example, beaver (Castor canadensis)
ranged from 0.01 to 0.05 µg/g; otter (Lutra canadensis),
1.88 to 2.10 µg/g; wolf (Canis lupus), 0.38 µg/g;
rabbit, 0.03 µg/g in muscle and 0.44 µg/g in kidney;
muskrat (Ondatra zibethicus), 0.05 µg/g in muscle and
0.34 µg/g in kidney; and marten, 0.12 to 0.33 µg/g for
muscle and 0.56 to 1.02 µg/g in kidneys (Environment
Canada 1972). In other parts of Canada the following
average total mercury concentrations in muscle and liver
tissue were reported: moose (Alces alces), 0.04 µg/g
(range <0.01 to 0.17); mule deer (Odocoileus hemionus),
0.13 µg/g (range 0.06 to 0.18); caribou (Rangifer
tarandus), 0.017 µg/g in muscle and 0.20 µg/g in liver;
wolf, 0.051 µg/g in muscle and 0.24 µg/g in liver; and
Arctic fox (Alopex lagopus), 0.31 µg/g in muscle and
0.76 µg/g in liver (Smith and Armstrong 1975). In
addition, muskrat taken from the Lake Erie forest
district had liver mercury levels that ranged between
0.040 and 0.251 µg/g (Jervis et al. 1970). All of the
mercury in 10 specimens of polar bear (Ursus maritimus)
muscle tissue was in the form of methylmercury and
averaged about 0.13 µg/g (range 0.01 to 0.66
µg/g)(Desai-Greenway and Price 1976).

CHAPTER 5

ENVIRONMENTAL EXPOSURE AND UPTAKE OF
MERCURY BY HUMANS

The toxicity of methylmercury to humans is well known and is discussed in detail in Chapter 6. The uptake of this form of mercury presents a potentially serious hazard to health. The significance of environmental sources is increased by the fact that mercury methylation has not been shown to occur in vivo in humans. This chapter discusses ways in which people can be exposed to mercury compounds in the environment and, where the data permit, to methylmercury especially.

AIR AND DRINKING WATER

There is no indication that mercury compounds in the concentrations and forms found in either the atmosphere or drinking water supplies contribute significantly to the methylmercury burden in human beings. Most data indicate that the mercury levels in water are, with rare exceptions, less than 1 μg/l and almost always lower except in regions with anomalously high concentrations of mercury in the rocks and soils or near sources of anthropogenic pollution. For example, in 1971, EPA's Division of Water Hygiene analyzed 698 samples of raw and finished waters collected from 273 drinking water supplies throughout the United States. Only 11 samples exceeded 1 μg/l in a range from 1.0 to 4.9 μg/l, and only 1 sample exceeded 5 μg/l (Hammerstrom et al. 1972). A more recent survey of finished waters established that only 13, or 2.5 percent, of 512 mercury analyses exceeded the proposed 1975 federal drinking water standard of 2 μg/l (U.S. EPA 1973, 1975a). The World Health Organization (1971) has recommended an upper limit of 1 μg/l for the total mercury content of water for human consumption. Thus, assuming a daily intake of 2 liters of water and other beverages, a daily maximum of 4 μg can be attributed to this source. If the actual observed mercury concentration in most water supplies is considered (<0.1 μg/l), this source contributes less

than 0.2 μg/person/day and is inconsequential when compared with the intake from foods.

FOOD SUPPLIES

General Food Surveys

The available data indicate that almost all the methylmercury in the human diet comes from fish, other seafood, and possibly red meat. However, other kinds of food contribute to the total human mercury burden. Minute amounts of mercury occur naturally in nearly all foods and beverages, and also as a contaminant from the use of mercury compounds as fungicides and in industry.

The amounts of methylmercury in edible plants and plant products are generally extremely low; exceptions may occur if the plants are grown on contaminated soil or from seed stock treated with mercury. Meat and dairy products may contain low levels of total mercury, which can include a proportion of methylmercury compounds presumably derived from residues in foods that contain fishmeal and, in the past, from mercury-treated cereal grains.

Mercury levels in foods have been surveyed in several countries. In most cases the analytical data report only the total mercury content. For example, Stock (Stock and Cucuel 1934, Stock 1938) reported that traces of mercury occur in nearly all foods. Several later food surveys are summarized in Table 5.1. In general, the following average mercury concentrations were reported for uncontaminated foods: dairy products, 0.002 to 0.020 μg/g; meat, fish, and poultry, 0.010 to 0.20 μg/g; grain and cereal, 0.020 to 0.050 μg/g; potatoes, 0.006 to 0.020 μg/g; legume vegetables, 0.002 to 0.010 μg/g; and beverages, 0.002 to 0.006 μg/g.

In the United Kingdom when over 6400 samples of food were analyzed (U.K. Ministry of Agriculture, Fisheries, and Food 1971, 1973; U.K. Department of the Environment 1976), the mean level of mercury in cereals, most fresh meats, fruits and preserves, green and root vegetables was less than 0.005 μg/g on a fresh-weight basis. The natural mercury content of herbage is about 0.001 to 0.010 μg/g. In addition, animals that eat food supplements prepared from soybeans, grains, fish, milk, meat, and bone may add mercury as follows: vegetable matter <0.01 to 0.07 μg/g, fish <0.01 to 1.8 μg/g (mean value <0.3 μg/g), milk 0.01 μg/g, and 0.01 to 4 μg/g from meat and bone. Since the mean values were 0.01 to 0.04 μg/g for vegetable feeds, 0.06 to 0.23 μg/g for fish feeds, and 0.37 μg/g for meat and bone meal, it seems reasonable to conclude that natural herbage and

vegetable feeds are an insignificant source of mercury in animals. Fish meal, depending on the amount consumed, is more important because the mercury content is largely in the methyl form. Meat and bone meal supplements may also be important, but unlike the fish meal only a negligible portion of the mercury is present as methylmercury compounds (U.K. Department of the Environment 1976).

A 1970 survey of nearly 1400 Canadian foods, excluding fish, showed the mercury residues to be less than 0.060 µg/g for bread, flour, grains, and eggs, and less than 0.040 µg/g in meats and vegetables (Somers 1971). In Michigan the results of a limited survey showed that all foods except fish contain detectable traces of mercury in the range <0.01 to 0.03 µg/g (Gomez 1972). A series of market-basket studies reviewed by Berglund et al. (1971) established the average daily intake in the Scandinavian countries between 1965 and 1971. With occasional exceptions, i.e., 0.18 µg/g mercury in hog liver, the mean mercury level of foodstuffs was 0.03 µg/g or less.

In a survey of 10 food commodities by the U.S. Food and Drug Administration (FDA) in 1970, 1971, and 1972, shrimp had the highest mercury content at 0.043 µg/g and a median value of 0.014 µg/g (Simpson et al. 1974). Nonfat dry milk was next highest at 0.027 µg/g and a median of 0.010 µg/g. All other commodities (flour, sugar, potatoes, raw ground beef, chicken breast, beef liver, eggs, and fluid whole milk) had mean mercury concentrations of less than 0.003 µg/g. In the total diet fractions, only meat, fish, and poultry contained mercury as high as 0.041 µg/g (range from 0.004 to 0.041 µg/g). All other fractions (dairy products, grain and cereal products, potatoes, leafy vegetables, legume vegetables, root vegetables, garden fruits and fruits) contained average mercury levels of less than 0.002 µg/g on a dry-weight basis.

Of all food commodities analyzed for mercury, only fish appear to present a potential hazard to human beings. The 0.5 µg/g FDA interim guideline is usually only exceeded in the larger species (large tuna, halibut, and swordfish) of the commercially important marine varieties and in freshwater fish obtained from mercury polluted watercourses. Mercury levels in fish are reviewed in detail in Chapter 4 and more briefly in the next section. In all other foods, mercury either is not detectable or is present at levels approximately 2 orders of magnitude lower than the FDA interim guideline for fish.

TABLE 5.1 The Mercury Content of Selected Foods

Type of Food	Country	Mean (ng/g)	Range (ng/g)	Reference
DAIRY	United States	–	<1-3	Simpson et al. (1974)
PRODUCTS	United States	–	1-2	Kirkpatrick and Coffin (1974)
	Canada	–	2-20	Corneliussen (1969)
Milk	United States	–	5-20	Gomez (1972)
	United States	–	3-7	Stock and Cucuel (1934)
	United States	8	–	Goldwater (1964)
	United States	1	1-9	Tanner et al. (1972)
	Germany	–	6-4	Stock and Cucuel (1934)
	United Kingdom	10	ND-20	U.K. Ministry of Agriculture, Fisheries and Food (1971)
Powder	Canada	–	21-180	Jervis et al. (1970)
Nonfat-dry	United States	10	4-27	Tanner et al. (1972)
Cheese	United States	20	15-30	Gomez (1972)
	United States	80	–	Goldwater (1971)
	United Kingdom	170	ND-500	U.K. Ministry of Agriculture, Fisheries and Food (1971)
	Canada	70	–	Jervis et al. (1970)
Butter	United States	140	–	Goldwater (1971)
MEAT, FISH,	United States	–	4-41	Simpson et al. (1974)
AND POULTRY	United States	–	10-50	Corneliussen (1969)
	Canada	–	28-51	Kirkpatrick and Coffin (1974)
Meat, General	United States	–	0.8-44	Gibbs et al. (1941)
	United States	–	1.0-150	Goldwater (1964)
	United States	3	2-7	Tanner et al. (1972)
	United States	–	ND-20	Gomez (1972)
	United Kingdom	–	ND-90	U.K. Ministry of Agriculture, Fisheries and Food (1971)
	Canada	<40	–	Somers (1971)
	Germany	–	1-67	Stock and Cucuel (1934)
	Worldwide	–	3-30	Smart (1968)
Liver, Pork	United States	30	20-40	Gomez (1972)
	Canada	–	16-170	Jervis et al. (1970)
	Sweden	–	3-49	Westöö (1969a)
Liver, Beef	United States	3	<2-8	Tanner et al. (1972)
	United States	10	10-15	Gomez (1972)
	United Kingdom	40	ND-90	U.K. Ministry of Agriculture, Fisheries and Food (1971)
	Canada	–	14-199	Jervis et al. (1970)
Kidney	Germany	67	–	Stock and Cucuel (1934)
Shrimp	United States	14	5-43	Tanner et al. (1972)
Chicken	United States	20	15-30	Gomez (1972)
	Worldwide	–	5-21	Smart (1968)
Breasts	United States	3	1-7	Tanner et al. (1972)
	Canada	–	25-61	Jervis et al. (1970)
Eggs	United States	ND	–	Gibbs et al. (1941)
	United States	–	10-62	Goldwater (1971)
	United States	<2	2-5	Tanner et al. (1972)

TABLE 5.1 (continued)

Type of Food	Country	Mean (ng/g)	Range (ng/g)	Reference
	United States	30	20-40	Gomez (1972)
	United Kingdom	–	ND	U.K. Ministry of Agriculture, Fisheries and Food (1971)
	Canada	–	20-29	Jervis et al. (1970)
	Canada	<60	–	Somers (1971)
	Germany	2	–	Stock and Cucuel (1934)
	West Germany	–	5	Smart (1968)
	Sweden	–	4-21	Westöö (1969a)
	Sweden	29	15-43	Smart (1968)
	Norway	–	15-20	Smart (1968)
	Denmark	–	4	Smart (1968)
	Italy	–	5-6	Smart (1968)
	Austria	–	6-13	Smart (1968)
	Belgium	–	6-8	Smart (1968)
	Holland	–	5-7	Smart (1968)
GRAINS AND	United States	–	<2-12	Simpson et al. (1974)
CEREAL	United States	–	20-50	Corneliussen (1969)
PRODUCTS	Canada	–	2-4	Kirkpatrick and Coffin (1974)
Barley	United States	25	20-40	Gomez (1972)
Millet	United States	30	–	Bache et al. (1973)
Oats	United States	10	5-10	Gomez (1972)
Rice	United States	87	–	Gerdes et al. (1974)
	United States	10	5-10	Gomez (1972)
	United Kingdom (imported)	–	5-15	Smart (1968)
	Japan	–	80-190	Fujita (1966)
Wheat	United States	20	10-30	Gomez (1972)
	Sweden	–	8-12	Smart (1968)
	Worldwide	–	20-85	Jervis et al. (1970)
Grain	United States	–	2-6	Gibbs et al. (1941)
	United States	–	2-25	Goldwater (1964)
	Canada	<60	–	Somers (1971)
	Germany	–	20-36	Stock and Cucuel (1934)
Flour	United States	3	3-6	Tanner et al. (1972)
	United States	103	92-118	Gerdes et al. (1974)
	United Kingdom	20	ND-60	U.K. Ministry of Agriculture, Fisheries and Food (1971)
	Canada	<60	–	Somers (1971)
	Germany	–	25-35	Stock and Cucuel (1934)
Bread	United States	10	5-10	Gomez (1972)
	United Kingdom	20	ND-80	U.K. Ministry of Agriculture, Fisheries and Food (1971)
	Canada	96	5-10	Jervis et al. (1970)
	Canada	<60	–	Somers (1971)
	Germany	5	–	Stock and Cucuel (1934)

TABLE 5.1 (continued)

Type of Food	Country	Mean (ng/g)	Range (ng/g)	Reference
VEGETABLES	United States	ND	–	Gibbs et al. (1941)
	United States	–	ND-60	Goldwater (1964)
	United States	–	2-20	Goldwater (1971)
	United States	–	1-123	Gerdes et al. (1974)
	United States	–	10-35	Gomez (1972)
	United Kingdom	–	ND-100	U.K. Ministry of Agriculture, Fisheries and Food (1971)
	Canada	<40	–	Somers (1971)
	Canada	–	22-280	Jervis et al. (1970)
	Germany	–	2-44	Stock and Cucuel (1934)
Leafy	United States	–	<1-9	Simpson et al. (1974)
	Canada	1	–	Kirkpatrick and Coffin (1974)
	Worldwide	–	12-44	Smart (1968)
Root	United States	–	<1-2	Simpson et al. (1974)
	Canada	1	–	Kirkpatrick and Coffin (1974)
Legume	United States	–	<1-2	Simpson et al. (1974)
	United States	–	2-10	Corneliussen (1969)
	Canada	1	–	Kirkpatrick and Coffin (1974)
Potatoes	United States	–	<1-13	Simpson et al. (1974)
	United States	–	6-20	Corneliussen (1969)
	United States	3	1-15	Tanner et al. (1972)
	United States	6	–	Bache et al. (1973)
	United States	10	ND-15	Gomez (1972)
	United Kingdom	–	10-79	Lee and Roughan (1970)
	United Kingdom	20	ND-70	U.K. Ministry of Agriculture, Fisheries and Food (1971)
	Canada	–	200	Jervis et al. (1970)
	Canada	1	–	Kirkpatrick and Coffin (1974)
	Germany	1	–	Stock and Cucuel (1934)
	Worldwide	–	3-6	Smart (1968)
Tomatoes	United States	18	–	Bache et al. (1973)
	United States	10	ND-10	Gomez (1972)
	United Kingdom	10	ND-50	U.K. Ministry of Agriculture, Fisheries and Food (1971)
	New Zealand	–	12-144	Smart (1968)
FRUIT	United States	4	4-30	Goldwater (1964)
	United States	–	<1-3	Simpson et al. (1974)
	United States	–	ND-282	Gerdes et al. (1974)
	United States	10	ND-10	Gomez (1972)
	United Kingdom	–	ND-180	U.K. Department of Agriculture, Fisheries and Food (1971)
	Canada	–	24-270	Jervis et al. (1970)
	Canada	1	–	Kirkpatrick and Coffin (1974)
	Germany	–	4-10	Stock and Cucuel (1934)
	Worldwide	–	3-50	Smart (1968)

TABLE 5.1 (continued)

Type of Food	Country	Mean (ng/g)	Range (ng/g)	Reference
MISCELLANEOUS				
Sugar	United States	51	–	Gerdes et al. (1974)
	United States	<3	<3-10	Tanner et al (1972)
	United States	10	5-10	Gomez (1972)
Beer	United States	4	–	Goldwater (1964)
	Germany	–	0.07-1.4	Stock and Cucuel (1934)
Fat				
Vegetable	Germany	–	60-115	Stock and Cucuel (1934)
Animal	United Kingdom	140	20-260	U.K. Ministry of Agriculture, Fisheries and Food (1971)
	Germany	–	70-280	Stock and Cucuel (1934)

ND = Not Detected.

Mercury Levels in Fish

In 1970, 1971, and 1972 the FDA surveyed (1) the mercury content of a wide variety of fish samples from selected freshwater regions, commercial fish from wholesale distributors, and swordfish and canned tuna fish; (2) 10 commodities representing a high proportion of total food consumption; and (3) 12 total diet fractions collected in the FDA continuing market-basket study to determine pesticide residues in the basic 2-week diet of a 19-year-old male (Simpson et al. 1974).

The survey found that some freshwater fish species contained elevated mercury levels traceable to known sources of pollution. In 1970, over 20 percent of the fish sampled from Lake Erie exceeded 0.5 μg/g; over 50 percent of the fish from the Ontario side of Lake St. Clair were also above this level. The fish that usually contained the highest mercury levels were walleye, sheepshead, smallmouth bass, white bass, catfish, perch, and carp. Of domestic samples collected in 1971-1972 from all parts of the country, 16 of 40 samples (40 percent) had mercury levels in excess of 0.5 μg/g. Similar results were observed for imported freshwater fish, with 7 of 16 samples tested (or about 45 percent) containing more than 0.5 μg/g mercury (Simpson et al. 1974).

A number of commercial fish--halibut, bonita, mackerel, cod, and red snapper--contained slightly elevated levels but averaged below the 0.5 μg/g FDA interim guideline. For example, 500 samples of halibut showed the overall average concentration of mercury to be approximately 0.25 with about 13 samples above the 0.5 μg/g guideline. Of more than 1300 samples of 19 different species of commercial fish and seafood collected in 1971, only six species included one or more fish with mercury levels greater than the 0.5 μg/g guideline. Bonita and red snapper averaged about 0.30 μg/g wet weight. They were the only species with a significant number of samples that averaged more than 0.20 μg/g. In addition, one study of fish protein concentrates and fish meals manufactured from North American fish showed a mercury range (dry weight) from 0.3 to 0.9 μg/g (Beasley 1971).

While the mercury level of most marine and freshwater fish is less than 0.20 μg/g, some large marine fish such as tuna, swordfish, marlin, halibut, and shark usually range from 0.20 to 1.50 μg/g and can be as high as 5.0 μg/g (wet weight). Swordfish showed the highest incidence of mercury. Over 95 percent of the 853 swordfish samples analyzed in 1971 exceeded the 0.5 μg/g FDA interim guideline, with more than 50 percent exceeding 1.0 μg/g mercury (wet weight). Swordfish from

the western Atlantic (210 samples) had mean mercury levels of 1.15 $\mu g/g$ wet weight (ranging from 0.050 to 4.9 $\mu g/g$) while 49 swordfish taken near Italy had levels ranging from 0.65 to 1.75 $\mu g/g$ with most values near 1.10 $\mu g/g$ wet weight (U.K. Department of the Environment 1976). When 3000 samples of canned tuna were tested, the average total mercury content was approximately 0.25 $\mu g/g$ with only about 4 percent of the samples exceeding the 0.5 μ/g FDA interim guideline.

The overall mean value of 0.02 $\mu g/g$ was confirmed for a variety of canned fish such as salmon, herrings, pilchards, sardines, and mackerel, while the mean mercury levels in tuna ranged between 0.07 and 0.44 $\mu g/g$. For canned shellfish such as shrimp, prawns, crab, and lobster, the mean mercury levels ranged between 0.01 and 0.29 $\mu g/g$ for samples taken from various waters (U.K. Department of the Environment 1976).

Human Uptake of Mercury in Fish

Fish and shellfish are the only regular source of methylmercury in the human diet of practical importance today. Elevated mercury and methylmercury levels in human beings have been linked with fish consumption (Berglund et al. 1971, Suzuki et al. 1971, Yamaguchi et al. 1971). Where selected subpopulations and individuals eat large amounts of fish from highly contaminated local waterways, either freshwater or marine, over time a health hazard could develop (see Chapter 6). Even without anthropogenic contamination, fish and shellfish contain more mercury than other foods. Marine fish and shellfish taken from unpolluted waters typically contain 0.01 to 0.3 $\mu g/g$ mercury on a wet-weight basis. Individual fish taken from moderately polluted waters have mercury levels in the range of 0.5 to 3 $\mu g/g$. In heavily contaminated areas such as Minamata Bay and Niigata, Japan, the afflicted victims ate fish and shellfish that contained up to 35 $\mu g/g$, mostly in the form of methylmercury (Kitamura 1968). In North America, fish containing mercury concentrations in excess of 24 $\mu g/g$ (wet weight) have been taken from the heavily contaminated Wabigoon-English river system in northwestern Ontario (Bishop and Neary 1976). This situation poses a serious health hazard to tourists and especially to native Americans who continue to eat fish from these waters (Takeuchi et al. 1977).

In some areas of the United States, mercury contamination of fish has become severe enough to require health warnings or closure of sport and commercial fisheries. By November 1970, 11 states

(Alabama, Georgia, Louisiana, Michigan, Mississippi, New York, Ohio, South Carolina, Tennessee, Texas, and West Virginia) had issued health warnings and closed both or either their sport or commercial fisheries because of excessive mercury contamination. An additional 6 states (New Hampshire, New Mexico, Pennsylvania, Vermont, Virginia, and Wisconsin) issued warnings to the general public about the health hazards from eating mercury-contaminated fish (Harlan 1971). Since then, 6 states (Alabama, Georgia, Louisiana, Mississippi, Ohio, and West Virginia) have reopened their fisheries while 6 others (Michigan, New York, Tennessee, Texas, Vermont, and Virginia) currently have some waters closed to fishing because of mercury pollution. The most recent fisheries closure occurred in Virginia on June 6, 1977. In addition, 12 states (California, Idaho, Illinois, Kentucky, Massachusetts, Minnesota, Mississippi, New Mexico, North Carolina, Oregon, South Carolina, and South Dakota) have current health warnings with respect to eating mercury contaminated fish from certain state waterways. A more complete survey of the current status of individual state sport and commercial fisheries is given in Appendix C.

In the Hackensack Meadows area of New Jersey a major mercury pollution problem is being investigated by the New Jersey Department of Environmental Protection. Soil, sediments, and water contain high levels of total mercury: percents in soil, 10's of $\mu g/g$ in sediments, and ng/ml in the water. However, the levels in fish are well below the FDA interim guideline, and there is no immediate threat to human health. However, this could change if the mercury becomes mobilized and moves into the food chain, so it is a major concern (Dr. Peter W. Preuss, personal communication, Special Assistant to the Commissioner, N.J. Department of Environmental Protection, August 10, 1977).

Consumption Patterns and Estimates

A widely quoted National Marine Fisheries Service study of fish consumption patterns (Nash 1971) measured only how much fish was purchased in 1969-1970. The fish were grouped in general categories such as tuna, salmon, or halibut and such fish specialty items as tuna pie, clam chowder, and TV dinners. The study assessed the per capita fish consumption only by socioeconomic class with no consideration of extremely heavy fish eaters, but it did provide evidence that overall fish consumption had increased 13 percent by 1973. Canned tuna increased 30 percent, canned salmon decreased 33 percent, and fish and frozen fish increased 45 percent.

Among other things, prices and changing dietary
habits, ethnic or religious customs influence fish
consumption patterns. Jews eat more fish than other
religious groups, for example, while Catholics eat more
than Protestants. Blacks eat more fish than whites
(Nash 1971).

The 1973-1974 Seafood Consumption Study (National
Purchase Diary Panel, Inc. 1975) estimated sport catches
and servings eaten away from home, as well as commercial
purchases. The study assumed an average dinner portion
to be 6.0 oz (170 g), less than an average Weight
Watchers® portion (8 oz, 227 g, for women and 10 oz, 284
g, for men); and a lunch and snack portion to be 3.5 oz
(99 g) again less than the Weight Watchers® 4-oz (113-g)
portion. Thus, consumption rates were figured at less
than normal portions and minimum mercury levels.

Among the 7999 families comprising 26,848 individuals
studied in 1973-1974, 93 percent consumed seafood, with
tuna the most common fish consumed by 61.5 percent. The
average total consumption was 18.6 oz (527 g) per month
with a maximum of 247 oz (7002 g). The maximum tuna
consumption was 202 oz (5727 g). The tuna samples
showed a mean mercury concentration of 0.225 μg/g with a
standard deviation of 0.12. Pooling cans produced a
standard deviation of 0.42 μg/g for a single can of 6.5
oz (184 g). A consumption rate of 200 oz (5670 g) per
month would produce a median intake of approximately
42.5 μg/day at the rates analyzed in December 1970
(National Purchase Diary Panel, Inc. 1975). Few
individuals, however, consumed this much. While 38.5
percent of the populace ate no tuna, the average for the
remainder was 6.5 oz (184 g) per month; only 2.8 percent
ate more than 18.0 oz (510 g), and 0.5 percent more than
38 oz (1077 g) per month. Most other fish eaten by high
tuna consumers contained approximately 0.1 μg/g mercury,
according to the limited available data (Marsh et al.
1975).

The amount of mercury ingested varies from day to day
and from one individual to another because the mercury
concentrations vary in different types of foods and in
samples of the same type of food, and because people's
eating habits also vary widely. It is very unlikely
that all foods in a normally mixed diet will
consistently have high levels of mercury over long
periods of time. In Sweden, Westoo (1965) established
that the mean mercury content of 12 fish-free daily
diets in Stockholm was 10 μg mercury per day with a
range of 4 to 19 μg. More recently, Dencker and Schutz
(1971) established the mean mercury intakes for 17
individuals to be 3.6 μg (range 1.0 to 9.3 μg) while the
corresponding mean level for 58 diets containing fish

was 8.7 µg/person/day, with a range of 1.7 to 30.6 µg
mercury.

Tolerance Limits and Guidelines

   The total dietary intake of mercury in foodstuffs
varies widely from one country to another, primarily
because of differences in fish consumption rather than
other foodstuffs. Table 5.2 presents per capita seafood
consumption estimates and limits of total mercury
concentrations in food for various countries.
Guidelines or tolerance limits for acceptable levels of
mercury in fish and other seafood are usually based, at
least in part, on the average rates of daily fish
consumption.
   The average seafood intake for Japanese citizens is
109 g/day, by far the highest daily intake of the
countries listed; and the maximum mercury level declared
acceptable in fish is 0.4 µg/g. The problem of mercury
in Japanese food is exacerbated by the combination of an
exceptionally high level of fish consumption where some
fish are very highly contaminated in combination with
the low average body weight (50 kg) of the average
Japanese citizen.
   In Europe, tolerance limits range from 1 µg/g for fish
in Sweden to 0.03 µg/g for foods in general in the
Benelux countries (Smart and Hill 1968). The United
Kingdom has no statutory limits specifically for mercury
residues in foods, although the average daily
consumption of fish there varies from 12 to 34 g per
person (U.K. Department of the Environment 1976) and
contributes from 2 to 5 µg of mercury per person. The
total mercury ingested by an average individual in the
United Kingdom is about 5 to 10 µg/day, based on 1.5 kg
of food eaten daily (35 to 70 µg/week or 0.07 to 0.14
µg/kg of body weight per day for an adult). About half
of this is in the methylated form. These figures are
about 4 to 8 times lower for total mercury and 6 to 12
times lower for methylmercury than the World Health
Organization's provisional tolerable weekly intake (U.K.
Department of the Environment 1976).
   These values from the United Kingdom agree quite well
with the daily intake of less than 14 µg reported by
Abbott and Tatton (1970) for their total diet studies,
as well as the daily intakes estimated by Stock and
Cucuel (1934) of about 5.0 µg/person; by Gibbs et al.
(1941) of about 20 µg/person; and by Monier-Williams
(1950) of between 5 and 20 µg/person. The same range of
values appears to be valid for the great majority of
individuals in the United States. Peyton et al. (1975)
estimated that for a standard diet the low and high

daily intakes range between 5.4 and 14.6 μg total mercury of which 2.5 to 7.1 μg is methylmercury.

While food, and primarily fish, is the major source of mercury to humans, the average intake of methylmercury from this source is well below the WHO provisional tolerable limit. Other sources, such as skin preparations, dental fillings, and inhalation also contribute to the total mercury ingestion. The per capita total sorbed dose has been estimated to range from 17 to 41 μg mercury/person/day (Peyton et al. 1975).

First, a Swedish expert group (Berglund et al. 1971) and then the WHO recommended maximum intake levels to protect humans from risk through long-term ingestion of methylmercury. The Swedish authorities established 20 ng/g as an acceptable level of methylmercury in whole human blood with 30 μg as the acceptable daily intake (ADI). The World Health Organization (1972) accepted the findings of the Swedish authorities and set a provisional tolerable weekly intake of 300 μg of total mercury of which not more than 200 μg should be methylmercury.

Even though the recommended weekly intake of methylmercury for a 70-kg adult is essentially the same under both directives, the Swedish ADI and the WHO provisional tolerable intake should be clearly distinguished. The WHO defines an acceptable daily intake as the intake of a chemical (expressed in mg of the chemical per kg of body weight) which, during an entire lifetime, appears to be without appreciable risk on the basis of all the known facts at the time. The phrase "without appreciable risk" is taken to mean the practical certainty that injury will not result even after a lifetime of exposure. The ADI's set by WHO and FAO of the UN are intended to allocate the acceptable amounts of an additive where it will serve specific, necessary purposes in accordance with good manufacturing practice. Since such concepts are not applicable for metallic contaminants such as mercury or methylmercury in food, the Joint FAO/WHO Expert Committee on Food Additives (WHO 1972) adopted a new approach by allocating a provisional tolerable weekly intake of food contaminants. The term "tolerable" signifies permissibility rather than acceptability since the intake of mercury or methylmercury is unavoidably associated with the consumption of otherwise wholesome and nutritious food, and the term "provisional" indicates that this evaluation is tentative. (See Appendix B: A Brief Review of the FAO/WHO Deliberations on Setting Acceptable Tolerances for Mercury Residues in Foods, 1963-1976.)

TABLE 5.2 National Per Capita Seafood Consumption Estimates and Limits for Total Mercury Concentrations in Foods for Various Countries[a]

Country	Seafood Consumption (g/person/day)[b]	Food Regulated	Total Mercury Limit (μg/g)	Reference
Australia				
South Australia	17	Not specified	0.1	FAO/WHO (1968)
Victoria	17	Not specified	0.1	FAO/WHO (1968)
Western Australia	17	Not specified	0.01	FAO/WHO (1968)
Benelux	37	Not specified	0.03	FAO/WHO (1968)
Brazil	9	Not specified	0.05	FAO/WHO (1968)
Canada[c]	16	Fish	0.5	Morrison (1971)
Denmark	53	Not specified	0.05[c]	FAO/WHO (1968)
West Germany	30	Not specified	Zero[d]	FAO/WHO (1968)
		Fish (dogfish, tuna and swordfish)	0.5	
Japan[c]	109[f]	Fish	0.4[e]	Takeuchi and Eto (1975)
New Zealand	19	Not specified	Zero	FAO/WHO (1968)
		Fruits and vegetables	0.05	FAO/WHO (1968)

Sweden	56	Not specified	0.05[c]	FAO/WHO (1968)
		Fish	1.0	Berglund et al. (1971)
United Kingdom	26	All foods	None[g]	Portmann (1977)[g]
United States[c]	17	Fruits and vegetables	Zero	FAO/WHO (1968)
		Fish	0.5	Kolbye (1970)
		Water	0.002	U.S. EPA (1975b)
WHO/FAO		All foods	h	WHO (1972)

[a]Unless noted, the residues are measured as total mercury.

[b]FAO Food Balance Sheets, 1946-1966; Rome, 1971.

[c]Guideline only (maximum acceptable levels).

[d]Where derived from pesticide treatment.

[e]No more than 0.3 μg/g as methylmercury.

[f]From Takeuchi and Eto (1975).

[g]The United Kingdom Ministry of Agriculture, Fisheries and Food recommends that local enforcement officers and Public Analysts "exercise vigilance in ensuring that food containing levels of mercury unacceptable in their country of origin do not find their way on to the United Kingdom market" (J. E. Portmann, Ministry of Agriculture, Fisheries and Food, Fisheries and Food Laboratory, Burnham-on-Crouch, Essex, England, personal communication, 1977). The recommendation is aimed primarily at fish and shellfish and is designed to preclude dumping of condemned fish or shellfish by other countries.

[h]A "provisional tolerable intake" of 0.3 mg/person/week of which no more than 0.2 mg/person/week can be in the methylmercury form.

THE EFFECTS OF SELENIUM ON METHYLMERCURY TOXICITY

Nishigaki et al. (1974) analyzed 279 samples of 24
species of marine fish and found that the fish with
higher levels of methylmercury generally also contained
even higher levels of selenium (0.5 to 1.0 $\mu g/g$).
Methylmercury in tuna, swordfish, and other large ocean
fish appears to be less toxic than methylmercury
ingested under other circumstances. This hypothesis has
been proven experimentally in animals and attributed to
the antagonistic effects of selenium, which is usually
present in levels equal to or exceeding the mercury
content of the marine organism.
The protective effect of dietary selenium against
mercury toxicity was originally shown by Parizek and co-
workers (Parizek and Ostadalova 1967, Parizek et al.
1969). It was subsequently confirmed by several other
investigators (Ganther et al. 1972, 1973; Levander and
Argett 1969; Stillings et al. 1972; Iwata et al. 1973;
El-Begearmi et al. 1973, 1975; Groth et al. 1973; Potter
and Matrone 1973, Welsh et al. 1973; Froseth et al.
1974; Ganther and Sunde 1974; Hill 1974; Johnson and
Pond 1974; Stoewsand et al. 1974, 1977; Welsh and Soares
1974; Ohi et al. 1975; Ueda et al. 1975a, 1975b; Sell
and Horani 1976).

Observed Protective Effects

The most consistent beneficial influence of selenium
has been a reduction of the lethal and neurotoxic
effects of methylmercury compounds. This was noted when
the two were administered simultaneously in the diets of
rats and Japanese quail. In addition, the depression of
growth by methylmercury was partially alleviated.
Ganther et al. (1972) found that Japanese quail
(Coturnix coturnix japonica) fed 20 $\mu g/g$ mercury in a
corn-soya diet were intoxicated at 4 weeks and had a 52
percent mortality after 4 to 6 weeks. However, those
fed the same concentration of mercury in a diet
containing 17 percent tuna were free of symptoms of
intoxication for a longer time, and only 7 percent died.
Tuna contain enough selenium (normally up to 2 $\mu g/g$) to
reduce the toxic level of methylmercury. Evidence that
selenium protected against methylmercury intoxication in
quail has also been reported by Stoewsand et al. (1974).
Ohi et al. (1976) reported that naturally occurring
selenium in tuna increased the growth rate of test
animals intoxicated with methylmercury, but was only
about half as effective in preventing the neurological
symptoms of intoxication as was selenite selenium added
to their diet. In studies of chronic dietary exposure

to methylmercury and selenite, control rats died with
symptoms of neurotoxicity and concentrations of
methylmercury in the brain well below those in rats
protected by selenite (Ueda et al. 1975a, 1975b). Given
parenterally, selenite decreased the toxicity of dietary
methylmercury and temporarily increased the
methylmercury concentration in the rat brain (Iwata et
al. 1973).

Protective Mechanism

The reason for the protective action of selenium is
unclear. Sumino et al. (1977) present evidence that
selenite releases methylmercury from its linkage with
proteins and thereby influences its tissue distribution.
In a person whose methylmercury body burden is high
enough to cause clinical symptoms, the presence of
selenium appears to immobilize the methylmercury
compound, but does not appear to speed its elimination
from the body (Stillings et al. 1974).

The natural biological sink for methylmercury is in
its interaction with sulfhydryl groups. In fact,
approximately 95 percent of the methylmercury bound to
fish protein is part of the methylmercury-cysteinyl
coordination complex. This complex can be broken up
with an excess of thiols or strong acids. Recently,
Japanese workers (Sugiura et al. 1976) have shown that
the selenohydryl group binds methylmercury 100 times
more tightly than the sulfhydryl group.

The important role of selenium as an essential trace
element in biological systems is also now well
documented (Stadtman 1974). Selenium forms the active
site of the enzyme glutathione peroxidase (Stadtman
1974) and readily replaces sulfur in the sulfur-
containing amino acids. In view of the fact that the
selenohydryl group binds methylmercury 100 times more
tightly than the sulfhydryl group, it is clear that
organisms with a diet supplemented by selenium or with
high natural levels achieve an added degree of
protection against methylmercury poisoning.

Some livers and brains of apparently healthy sea
mammals have high concentrations of mercury and
selenium. Koeman et al. (1973) found a strong 1:1
correlation between the concentrations of mercury and
selenium in the livers of seals and dolphins. Martin et
al. (1976) have shown a similar correlation among the
mercury, selenium, and bromine levels of female
California sea lions. In healthy animals the ratios
among all three elements are similar, but those animals
with an imbalance have a strong tendency to produce pups
prematurely. Since Tamura et al. (1975) established

that the selenium-to-mercury molar ratio in tuna and
bonito muscle tissue was 5.8:1, it appears that the 1:1
ratio is established internally whenever sufficient
selenium is present in the mammal's diet. These
findings are in very good agreement with the results of
experimental work on the binding of mercury and selenium
to the blood plasma proteins of animals given mercuric
chloride and selenite (Burk et al. 1974) and the
correlation between mercury and selenium reported for
human subjects exposed to inorganic mercury (Kosta et
al. 1975). Recently, Nishigaki and Harada (1975)
investigated the preserved umbilical cords of
individuals affected by Minamata disease and showed that
the mercury content was much greater than the selenium
content. For people not affected by the disease, the
mercury:selenium ratio was 1:1. Moreover, Birke et al.
(1972) reported on individuals with elevated blood
mercury levels who remained free of neurological
symptoms. This suggests that agents such as selenium
may modify the toxic effects of methylmercury and may
account for some of the variations in individual dose
response. However, the protective effect from selenium
has not been documented in humans. For this reason and
because the protective mechanism is unclear, it would be
premature to modify current guidelines for acceptable
levels of mercury in food on the basis of selenium
content.

Recent studies of human populations that consume large
quantities of tuna have revealed no definitive sign of
mercury poisoning, although some individuals had
elevated mercury levels in blood and hair. Whereas
marine fish such as tuna tend to accumulate both mercury
and selenium at approximately the same level in the
oceans, fish from polluted fresh waters concentrate much
more mercury because of the greater supply in relation
to selenium. Further studies should be initiated to
establish the role of selenium in the freshwater
ecosystem.

Other Protective Agents

While the current evidence suggests that selenium is
the main protective factor in tuna, other dietary
factors may also modify the expression of methylmercury
toxicity and must be taken into account by toxicologists
and regulatory agencies that set tolerance standards.
Vitamin E has been shown to have a similar protective
effect against methylmercury poisoning (Welsh 1974,
Harada et al. 1975, Welsh and Soares 1976, El-Begearmi
et al. 1976). However, a much higher concentration is
required to provide the same level of protection as with

selenium. The mechanism by which Vitamin E protects against methylmercury toxicity is not understood at the present time either. However, both glutathione peroxidase and Vitamin E remove activated oxygen species and may prevent radical attacks on methylmercury, which would be expected to give toxic free radicals as products (Ganther [In press]). Moreover, by adding selenium the protective effects of Vitamin E against methylmercury poisoning in quail can be enhanced. Vitamin E was most effective in diets containing 0 to 0.1 $\mu g/g$ selenium. At these levels, supplemental Vitamin E significantly decreased the clinical symptoms of toxicity and improved the survival rate of the birds (Welsh and Soares 1976). In another series of experiments, El-Begearmi and co-workers (1974) showed that a combination of sodium arsenite and sodium selenite is more effective than either one separately. Other compounds actively being investigated for mutual antagonism toward methylmercury include: cystine (Stillings et al. 1974), methionine (El-Begearmi et al. 1974), Vitamin E (Welsh and Soares 1976), ascorbic acid (C.L. Farakas, personal communication, University of Waterloo, Waterloo, Ontario, Canada, 1977), and selenocysteamine (Sugiura et al. 1976).

CHAPTER 6

EFFECTS ON HUMAN HEALTH[1]

Methylmercury compounds have no known normal metabolic function; their presence in the tissues of living organisms, including man, represents contamination from environmental sources. Ultimately, limits for the release of any potentially toxic substance, including mercury, into the general biosphere must be assessed in terms of risk both to human populations that might be exposed to it, and to the ecology. A useful cost-benefit analysis of the use of mercury compounds and their release into the environment would require accurate and specific delineation of risk. Risk must be defined in terms of the toxic effects of acute, as well as chronic or lifetime, human exposure, including all life-cycle stages. In addition to evaluating toxic effects on the central nervous system, the potential risks of adverse genetic, reproductive, teratogenic, and carcinogenic effects of these nonessential compounds must be determined before definitive human and environmental tolerance levels can be estimated with confidence.

DEFINITIONS

The critical concentration of a toxic substance in a cell or organ is that level at which an adverse or critical effect can be detected by clinical observation, functional tests, or morphological or biochemical techniques. Critical organ concentration is defined as the mean concentration in an organ at the time any of its cells reaches critical concentration. The critical organ is the organ that first attains critical organ concentration (Task Group on Metal Accumulation 1973).

Existing evidence strongly indicates that the critical organ system in methylmercury (MeHg[+]) poisoning of man is the central nervous system (Berglund et al. 1971, Bakir et al. 1973, Task Group on Metal Accumulation 1973, WHO 1976a, Tsubaki and Irukayama 1977). Symptoms and signs observed in human populations after exposure

88

to toxic doses of methylmercury are dominated by
neurological disturbance. These effects may appear
weeks to months after acute exposure to toxic doses. A
progression of symptoms and signs after short- and long-
term exposure includes: paraesthesia (numbness and
tingling of the lips, mouth, hands, and feet),
dysarthria, ataxia, concentric constriction of the
visual fields, blurred vision, blindness, deafness, and
ultimately death.

Relationships between dose and degree of an effect
have not been quantitatively established for
methylmercury because of difficulties in quantitating
clinical effects. It has, however, been possible to
relate dose and appearance of various effects of
methylmercury. Doses have been estimated from ingested
amounts as well as index media such as blood or hair
concentrations. An indication of the primitiveness of
current ability to measure effect is that the critical
effect has to be defined as broadly as paraesthesia, a
symptom response reported by the exposed individual. No
other effect has yet been recognized at lower doses.

An effect is a defined biological reaction that may be
quantifiable and graded (degree of effect), or quantal
(all or none). Response is the frequency of an effect
in a population resulting from exposure to a specific
dose, i.e., an enumeration of reactors versus
nonreactors. Effect may be thought of as a change, due
to a dose, within an individual; response, the number of
different individuals of a total population showing a
specific effect (Task Group on Metal Accumulation 1973).
Thus, it follows that there is a population dose-
response relationship for each observable effect.

TOXIC EFFECTS OF METHYLMERCURY IN ADULT POPULATIONS

Hazard Estimates

Studies of human populations in Japan and, more
recently, in Iraq after large-scale, high-dose exposure
have documented the toxicity of methylmercury compounds
for human beings (Berglund et al. 1971; Bakir et al.
1973; WHO 1976a, 1976b; Tsubaki and Irukayama 1977).
Observations on these and other exposed populations have
been used to estimate the hazard of methylmercury
exposure for human populations.

Japanese Exposures: Contaminated Fish

The two major Japanese outbreaks of methylmercury
poisoning in Minamata Bay and in Niigata were caused by
industrial release of methylmercury and other mercury
compounds into Minamata Bay and into the Agano River,
followed by accumulation of methylmercury in edible fish
and shellfish. The median total mercury level in fish
caught in Minamata Bay at the time of the epidemic has
been estimated as 11 $\mu g/g$ fresh weight (Berglund et al.
1971). More recent reports of follow-up studies on
these exposed populations have revealed many cases that
were not originally diagnosed. By 1974, more than 700
cases of methylmercury poisoning had been identified in
Minamata and more than 500 in Niigata (Tsubaki 1971, WHO
1975b, Tsubaki and Irukayama 1977).

The detailed risk-evaluation for long-term human
exposure to methylmercury compounds performed in 1971 by
a Swedish expert group (Berglund et al. 1971) was based
on these Japanese exposures and used two independent
approaches: a metabolic method and an epidemiological
method. The metabolic evaluative approach was based on
the lowest concentrations of mercury observed in the
brains of fatal cases, absorption and distribution data,
and assumption of a biological elimination half-time of
70 days. A critical daily intake was estimated to be
750 μg Hg as methylmercury for an average 70-kg person.
Assuming daily ingestion, this is the level at which
observable clinical effects would be expected to occur
in sensitive adult individuals. The epidemiological
assessment was based on the lowest blood concentrations
in Japanese individuals observed to have clinical
symptoms and signs, and on data relating levels of
mercury in nonpoisoned individuals to the estimated
daily intake of methylmercury consumed in fish. A
critical daily intake was calculated to be 300 μg Hg as
methylmercury for an average 70-kg person. A recent
reassessment of hair and blood data from Minamata and
Niigata discusses some of the difficulties of using
these observations to estimate the minimal clinical
effect level of methylmercury (Marsh et al. 1975).

In order to safeguard the health of the population,
the lower of the two estimates of critical daily intake
(i.e., 300 μg of methylmercury) was reduced by a safety
factor of 10 to arrive at an acceptable daily intake
(ADI). The Swedish expert group (Berglund et al. 1971)
concluded that this safety factor was adequate to
protect even the most sensitive members of a population
such as children and pregnant women, that is, that
clinical symptoms of mercury poisoning would not occur
until a level of 200 ng/g was reached in the blood--
equivalent to 60 $\mu g/g$ in the hair and a body burden of

20 to 30 mg for a 70-kg person. They suggested that 30 μg Hg as methylmercury be considered an acceptable daily intake for an average 70-kg person and 20 ng/g as an acceptable level of mercury as methylmercury in whole human blood.

Since the completion of the Swedish evaluation, significant new information is becoming available from additional studies of exposed human populations (Bakir et al. 1973, Clarkson and Marsh 1976, WHO 1976a). Ongoing studies of exposed Iraqi and Canadian native populations will provide additional important data for estimating human health hazard during the next few years.

Iraqi Exposure: Contaminated Bread

During the winter of 1971-1972, the largest outbreak of methylmercury poisoning ever recorded occurred in Iraq as a result of consumption of homemade bread prepared from seed wheat treated with a methylmercury fungicide. More than 6000 poisoned children and adults were admitted to hospitals throughout Iraq with nearly 500 reported hospital deaths (Bakir et al. 1973, WHO 1976a). It is probable that more people were poisoned and died without contacting the hospital system.

In the exposed Iraqi population, it has been possible for the first time to determine separate dose-response curves relating population frequencies of paraesthesia, ataxia, dysarthria, deafness, or death to body burdens of methylmercury at the time of appearance of the effect, estimated from quantity of contaminated bread ingested. Abrupt change in the slope of the observed frequencies of a specific effect with increasing body burdens has allowed an extrapolated estimate of the minimal mean body burden at the time the specific effect (the detection limit) could first be detected above mean background frequencies of the observed effect (Bakir et al. 1973). The limit of detection of this type of dose-response relationship is determined by the background frequency of the effect being measured in unexposed individuals in the population being examined. The most sensitive current index of human methylmercury toxicity is paraesthesia, which appears above background frequency in the population samples in Iraq at a mean body burden of 25 mg or 40 mg mercury as methylmercury (50-kg individual), depending on the factor used to convert intake to estimated body burden (Bakir et al. 1973; see Figures 6.1 and 6.2). Estimates of mean body burdens for appearance of other signs (ataxia, dysarthria, death) and symptoms (deafness) of methylmercury poisoning were similarly extrapolated
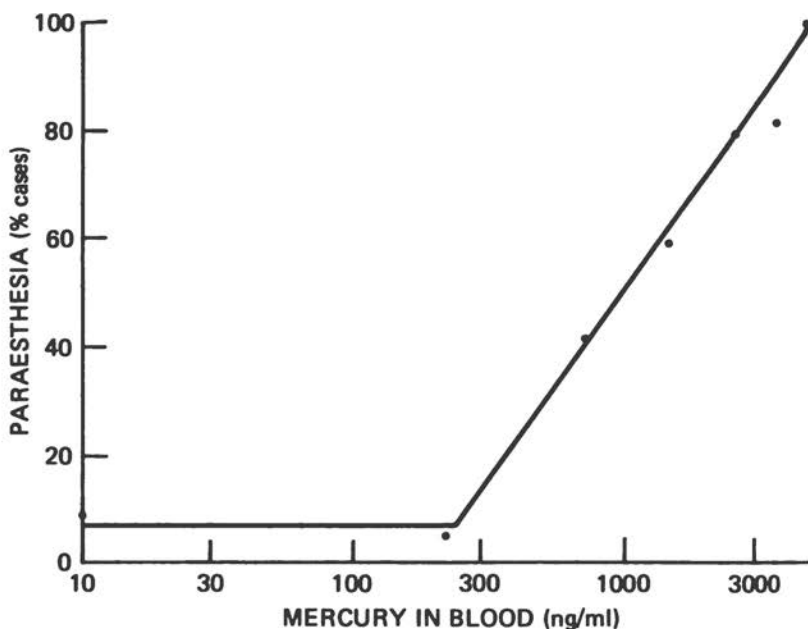
The mean blood concentrations are computed as the logarithmic means for each cohort in their table. The line connecting the first two points was assumed to be horizontal. The line connecting the other points was computed by least squares linear regression analysis.

SOURCE: Adapted from Bakir et al. (1973). Science 181:230-241. Copyright 1973 by the American Association for the Advancement of Science.

FIGURE 6.1 The frequency of paraesthesia as a function of the concentration of mercury in blood, 65 days after cessation of exposure.
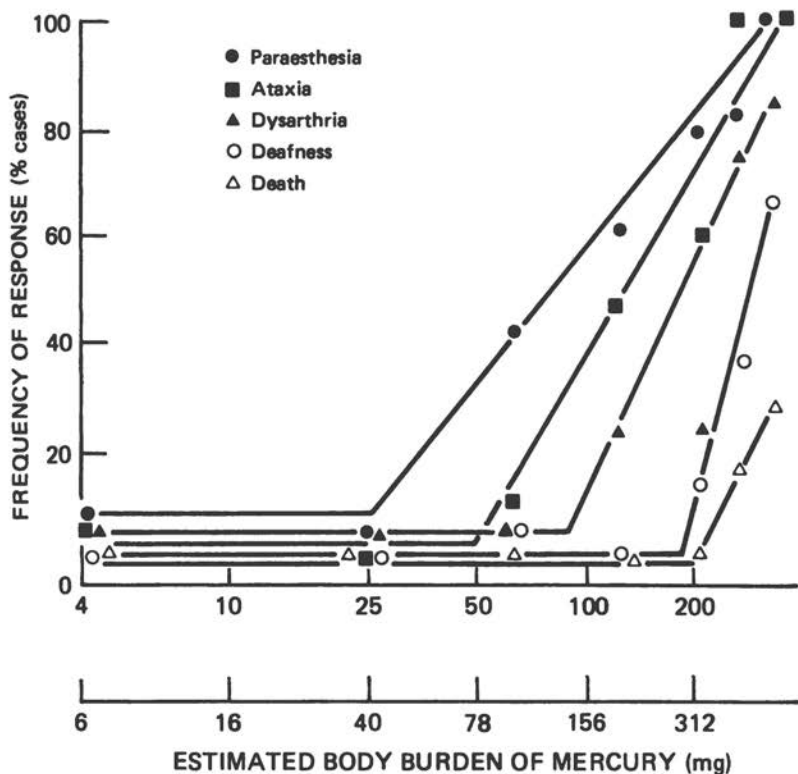
Both scales of the abscissa refer to body burdens of methylmercury (mg) at the cessation of exposure. The two scales represented different methods of calculating the body burden as discussed in the text.

SOURCE: Adapted from Bakir et al. (1973). Science 181:230-241. Copyright 1973 by the American Association for the Advancement of Science.

FIGURE 6.2 The relationship between frequency of signs and symptoms and the estimated body burden of methylmercury.

(Figure 6.2). Additional Iraqi data reported by
Kazantzis (1976) agree with these dose-response
estimates. In the exposed Iraqi population 31 percent
of women who had apparent symptoms and/or signs
attributable to methylmercury poisoning had maximum hair
mercury levels less than 100,000 ng/g. The authors
suggest that there is a category of methylmercury
poisoning in which symptoms such as paraesthesia,
headaches, persistant pain, and weakness of the limbs
predominate, with little or no evidence of neurological
damage on clinical examination, and that these effects
may occur with blood mercury levels well below 400
ng/ml. The estimate of the mean body burden that
results in the appearance of the first observable effect
(paraesthesia) in the exposed Iraqi population agrees
surprisingly well with the findings reported earlier by
the Swedish expert committee from analysis of data from
the Japanese outbreaks. They estimated a minimum body
burden of approximately 30 mg of mercury in
methylmercury form (70-kg person) to be associated with
the onset of the earliest signs and symptoms of
methylmercury poisoning (Berglund et al. 1971).

Assumptions and Reliability of the Estimates

These limits for detecting the effects of
methylmercury should not be equated with threshold
levels, since other more subtle effects may not be
observed with current techniques. The mean adult body
burden at which methylmercury begins to cause damage to
the nervous system can be assumed to be lower, perhaps
considerably lower, than current limits of detection of
minimal effect (paraesthesia). Limits of individual
variability in the population are approximately
reflected by the slope of the relationship between
response and body burden. For paraesthesia, individuals
in the Iraqi population vary by approximately a factor
of 10 (Bakir et al. 1973). The different slopes of the
other dose-response relationships shown in Figure 6.2
suggest that the extent of variation among individuals
differs for different signs or symptoms.
The reliability of these estimates of dose-response
and allowable daily intake depend upon the following
assumptions:

● nearly 100 percent absorption of methylmercury
compounds among individuals in the exposed population,
● turnover of methylmercury among various bodily
compartments, including the nervous system, that is
considerably faster than the elimination of
methylmercury from the body as a whole (implying a

constant ratio between the concentration of
methylmercury in the critical organ and in other
tissues); and
● elimination of methylmercury from the body
following a single exponential function (i.e., with
long-term exposure the steady state body burden in mg[B] is:
$B = T \times a/\ln 2$, where T = total body elimination
half-time, and a = daily dose of methylmercury in mg Hg
to a 50-kg person (Task Group on Metal Accumulation
1973).

Recent observations of the exposed Iraqi population
have documented variation in biological half-times among
exposed individuals. Using sequential hair analyses,
Al-Shahristani and Shibab (1974) have reported that
though most adults have half-times of approximately 70
days, a significant number of individuals may have
elimination half-times as long as 120 days. Since
elimination half-time has been shown to be directly
related to the cumulative body burden as a function of
the duration of exposure, individual variations in
excretion are important in estimating exposure risks.
Recently, Nordberg and Strangert (1976) incorporated
these data into a more complex metabolic formulation for
population risk evaluation. Their model estimates
directly the probability of observable toxicity among
individuals in an exposed population and does not
require the use of a "safety factor." Risk estimates
that use this method support the adequacy of the
earlier, Swedish expert group estimate of an acceptable
daily intake (ADI) of 30 μg of mercury as methylmercury
per day for a 70-kg person (Berglund et al. 1971). The
model specifically predicts that for this ADI there
would be fewer than 1 in 5000 exposed adult individuals
who would have observable toxicity.

Human Populations with
Large Intake of Freshwater Fish

Sweden

Birke et al. (1972) and Skerfving (1974) reported
observations on a total of nearly 200 individuals with
levels of methylmercury in the blood elevated by regular
consumption of fish taken from various freshwater and
coastal areas of Sweden. The fish contained mercury
levels of 0.3 to 7.0 μg/g, and human blood mercury
levels as high as 600 ng/ml were recorded. Thirty
percent of the subjects were examined in detail for
signs of mercury poisoning, and the 3 percent with the
highest fish consumption were intensively examined in

hospitals. No definitive signs or symptoms of methylmercury poisoning were observed.

Canada

Elevated levels of mercury in fish from Lake St. Clair and lakes in northwest Ontario were first detected in 1969 (Fimreite 1970). Since then, methylmercury concentrations significantly above the Canadian guideline (0.5 μg/g) have been documented widely in fish taken from lakes and waterways throughout northwest Ontario and northwest Quebec. Some of these waterways are known to have been polluted with mercury released from industrial sources, but others are not known to have been directly contaminated.

During 1975 residents of the White Dog and Grassy Narrows Reserves (Ontario) were surveyed to determine the extent of exposure to mercury through ingestion of large quantities of freshwater fish and to assess whether such exposure may pose a health hazard. Levels of mercury in the blood were observed to range widely, from less than 5 to 330 ng/ml (Clarkson 1975). Adult male guides at fishing camps had the highest levels. Their wives also tended to have higher blood levels than other women on the reserves. A newborn infant with a hair concentration of 30,000 ng/g, equivalent to a blood concentration of approximately 120 ng/ml, was also described.

During the summer of 1975, a Japanese team invited by the Canadian National Indian Brotherhood visited Grassy Narrows and White Dog Reserves and reported that 37 of 89 people examined had signs and symptoms of methylmercury poisoning (Harada et al. 1976).

Similar comprehensive surveys in northwest Quebec are under way and have documented significantly elevated blood and hair mercury levels in native Canadians known to consume large quantities of fish from freshwater lakes.

A Canadian clinical medical team headed by A. Barbeau examined native Canadian populations in northwest Quebec with significantly elevated blood mercury concentrations and reported that at least 6, and possibly 25, of 49 people examined had signs and symptoms of methylmercury intoxication (Barbeau et al. 1976). Approximately 80 percent of the individuals examined were reported to have had blood mercury levels above 50 ng/ml at the time of neurological examination.

The validity of these clinical evaluations by the Canadian and Japanese clinical teams has been questioned. Possible nutritional deficiencies and high alcohol intake by some of the individuals reported to be

poisoned by methylmercury complicate the interpretation of clinical observations. A general consensus as to whether methylmercury intake from fish ingestion has resulted in neurological damage in Canadian native populations must await further studies, particularly histopathologic examinations of brain tissues from people documented to have significantly increased methylmercury body burdens, and properly designed epidemiological population surveys.

Human Populations with Large Intake of Ocean Fish

Eighty-eight tuna fishermen in American Samoa have been found to have an average blood methylmercury level of 64 ng/ml (range, 5 to 265 ng/ml) through eating large quantities of ocean fish (Marsh et al. 1974, Clarkson and Marsh 1976). In two villages in northern Peru, 186 inhabitants who had a high dietary intake of marine fish were observed to have a mean blood methylmercury concentration of 82 ng/ml (range, 11 to 275 ng/ml) (Turner et. al. 1974). Careful clinical neurological examination of these individuals revealed no evidence of clinical methylmercury intoxication in either the Peruvian or the Samoan populations. Astier-Dumas and Cumont (1975) and Kaku et al. (1975) have reported similar results in France and Japan, respectively. It should be noted that there were no reported observations of prenatally exposed individuals in these populations.

TOXIC EFFECTS OF METHYLMERCURY ON DEVELOPING FETUSES AND INFANTS

It has become increasingly obvious that a life-cycle perspective is necessary for appropriate evaluation of potential adverse effects of environmental agents. All stages of the life cycle must be examined for sensitivity to exposure, and all stages subsequent to the exposure must be assessed to determine acute as well as possible delayed responses. Certain effects may become observable only after a latent period when the organism's adaptive capacities are stressed by other adverse environmental factors, or when aging processes reduce particular systems to smaller safety margins in terms of ranges of adaptive response capacities.

The critical organ concentration may differ for different stages of the human life cycle. The developing fetal (and newborn) brain may be the most sensitive organ (i.e., critical organ) in terms of human methylmercury toxicity (Berglund et al. 1971). Studies

in animal models, as well as comparative qualitative observations of effects on prenatally exposed infants and mothers, support the notion that methylmercury is more toxic to the fetus than to the adult animal (Berglund et al. 1971, Spyker et al. 1972, Su and Okita 1976a).

During the Japanese Minamata outbreak, 23 infants with severe psychomotor signs of brain damage were described. They were born to mothers who had consumed fish taken from waters known to be heavily contaminated with effluent containing methylmercury from a nearby factory. In contrast to the apparent brain damage of their prenatally exposed infants, these mothers were reported to lack symptoms or signs of methylmercury poisoning other than mild paraesthesia. Thus, it was concluded that methylmercury crossed the placenta and that the fetal brain was much more sensitive than the adult brain (Harada 1968). However, no documentation of dose, fetal or maternal blood, or brain levels of methylmercury was possible in the Japanese cases. A similar observation of apparent fetal brain sensitivity was more recently reported following maternal ingestion of pork contaminated with methylmercury during the third to sixth months of pregnancy (Curley et al. 1971, Snyder 1971, Pierce et al. 1972).

In the large Iraqi outbreak of methylmercury poisoning, it has been possible to document prenatal transplacental exposure by direct measurement of maternal and fetal index media. Thus, for the first time it is possible to determine prenatal and early postnatal dose-response relationships. In all but one of the infant-mother pairs studied by Amin-Zaki et al. (1974, 1976), the infant's blood mercury level was higher than the mother's during the first few months of life. Ongoing sequential analyses of mercury in hair samples obtained from a larger group of women exposed during pregnancy should recapitulate exposure and permit estimation of the maternal and fetal dose.

Preliminary results on 29 children exposed in utero and examined at 3 and 4 1/2 years of age strongly suggest that the fetal brain is the critical organ in the exposed pregnant female. A clear difference was demonstrated by the group of children who were exposed in utero to maternal body burdens of methylmercury reflected by peak maternal hair concentrations of 112,000 to 384,000 ng/g (Figure 6.3; Marsh et al. [In press]). Clinical evidence of detectable fetal brain damage was observed when the peak maternal hair mercury concentration rose above 100,000 ng/g during pregnancy. A hair mercury concentration of 100,000 ng/g is estimated to be equivalent to a blood mercury concentration of approximately 400 ng/ml, assuming a
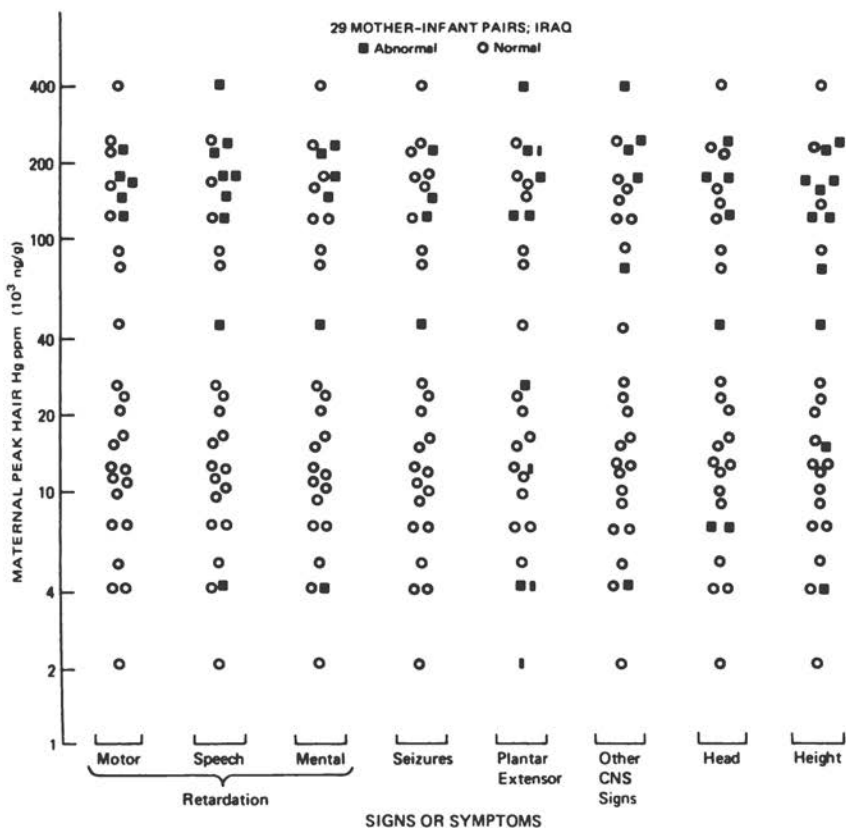
FIGURE 6.3 Signs or symptoms of mercury poisoning in children exposed *in utero* as a function of maternal hair mercury concentration (children were examined at ages 3 and 4.5 years).

hair:blood ratio of 250. Since only 29 infants were examined in this series, it is to be expected that the distribution of infants in a larger exposed population would contain individuals who would be affected at considerably lower maternal hair and blood concentrations. The conclusions of the international group of experts who authored the WHO (1977) report, Environmental Health Criteria 1, Mercury, concerning the concentrations of total mercury in indicator media associated with the earliest effects in the most sensitive group in the population are listed below.

Blood (ng/ml)	Hair (μg/g)	Equivalent long-term daily intake of mercury as methylmercury[a] (μg/kg body weight)
200-500	50-125	3-7

[a] A Japanese group has recently concluded that a daily intake of mercury of 5μg/kg is the "minimal toxic dose," following a 10-yr follow-up of the study of the Minamata outbreak (Research Committee on Minamata Disease 1975).

The WHO Task Group also urged that their conclusions not be considered independently of the section of their report entitled "Effects of Mercury on Man-- Epidemiological and Clinical Studies."

EFFECTS OF METHYLMERCURY ON GENETIC AND REPRODUCTIVE PROCESSES[2]

In rapidly dividing Alium cepa (onion) root cells, methylmercury has been shown to interfere with normal chromosome segregation by disrupting the mitotic spindle function (chromosome segregation) and causing c-mitosis at an exceedingly low concentration, 50 ng/ml (2.5×10^{-7} M), 1000 times lower than the colchicine concentration required to produce similar mitotic arrest of cell division (Ramel 1967). Significant c-mitotic effects were also observed in Vicia faba exposed to 20 ng/ml methylmercury (1×10^{-7} M) (Ramel 1972), and in cultured human leukocytes exposed to 200 to 400 ng/ml (1 to 2×10^{-6} M) (Fiskesjo 1970). These findings have recently been confirmed in methylmercury exposed Vicia faba and Tradescantia root tips (Ahmed and Grant 1972).
C-Meiotic effects of methylmercury reported in Tradescantia and sex chromosome nondisjunction in female Drosophila melanogaster fed methylmercury document the

potential for interference with the meiotic cell cycle
as well, resulting in viable aneuploid offspring (Ramel
1972). Meiotic ova from random-bred Swiss/Webster mice
cultured in vitro for 5 to 14 hours in the presence of
mercuric acetate (MA) or dimethylmercury (DM) were
compared with control ova (not exposed to mercury) in
terms of effects on first and second metaphase meiotic
figures. Dose-related effects were seen for both
compounds. Fifty thousand ng MA/ml and 25,000 ng DM/ml
completely prevented cell divisions. Twenty-five
thousand ng MA/ml and 10,000 ng DM/ml severely disrupted
meiotic progression and resulted in second meiotic
metaphase abnormalities (Jagiello and Lin 1973).

Frolen and Ramel (Ramel 1972) reported that male CBA
mice treated with a single intraperitoneal dose of 3
mg/kg methylmercury dicyandiamide showed no detectable
dominant lethal effects when mated weekly during the
ensuing 6 weeks, as indicated by incidences of living
embryos and dead implantations. However, they did
observe a significant reduction in numbers of fertile
matings compared with controls. Khera (1973) treated
rats and mice orally with methylmercury chloride, 0,
1.0, 2.5, and 5.0 mg/kg/day for 7 days. In rats he
found a significant dose-related reduction in mean
litter size at all doses during days 5 to 20 post-
treatment, which was attributed to preimplantation
losses. Male Swiss/Webster mice were dosed orally each
day for 5 or 7 consecutive days with doses up to 5
mg/kg. With serial matings a slight, but not
statistically significant, reduction in the average
number of viable embryos was observed as a result of
preimplantation losses. Lee and Dixon (1975) reported a
reduction in male fertility of mice given a single
intraperitoneal dose of 1 mg/kg methylmercury hydroxide.
Fertility profiles from serial matings suggested an
effect on spermatogonial cells and premeiotic
spermatocytes.

Recently, Suter (1975) has also reported small but
significant fertility effects in mouse dominant lethal
and reproduction capacity studies. Male mice were
injected intraperitoneally with 10 mg/kg methylmercuric
hydroxide. No fertility effects were seen in serial
matings of treated (101 $\times$ C_3H)F_1 male mice during a 48-
day period. However, (SEC $\times$ $C_{57}Bl$)F_1 males showed
significant small reductions in the average number of
total and living implants during the early post-
treatment interval (~7.5 days). A significant small
reduction in numbers of progeny produced and in numbers
of living embryos was also seen in similarly treated
females mated within the first 4.5 days after treatment.

Ramel (1972) observed that methylmercury causes
chromosome breaks in plant root cells. Fiskesjo (1970)

found no chromosome breakage in human leukocytes treated in vitro with methylmercury or dimethylmercury. However, Skerfving et al. (1974) reported a statistically significant correlation between erythrocyte total mercury concentration and percentage of cells with chromosome breaks or aneuploidy in lymphocytes cultured from persons with elevated erythrocyte methylmercury levels (range 13 to 1,100 ng/g) due to high intake of fish containing methylmercury.

Using cultured mouse leukemic cells L5178Y exposed to methylmercury in the range of 10^{-7} to 10^{-5} M, Nakazawa et al. (1975) found retardation of cell multiplication, depression of [3H] thymidine and [3H] uridine uptake, as well as induction of single strand scissions of DNA.

Dimethylmercury has also been observed to cause single strand DNA breaks in vivo in the slime mold Physarum polycephalum. Interestingly, strains of Physarum differing in geographical origin were found to have widely different sensitivities to dimethylmercury damage (Yatscoff and Cummins 1975).

A slight increase in the frequency of X-linked recessive lethals ("point mutations") in Drosophila after exposure to methylmercury in food was reported by Ramel (1972); and recently, more significant increases in X-linked recessive lethals following exposure of Drosophila to ethyl mercury-p-toluene sulphanilamide have been observed (Mathew and Al-Doori 1976).

Nakai and Machida (1973) were unable to detect nuclear nonsense and frameshift reversion mutations in Saccharomyces cerevisiae exposed to methylmercury, but they did observe induction of cytoplasmic petite mutations.

No assessment of mutagenicity in prokaryote systems such as the Ames salmonella assay has yet been made owing to technical difficulties of working with metals.

In summary, data concerning possible genetic and reproductive effects of mercury compounds is meager and somewhat contradictory. Methylmercury has been shown to be a relatively weak mutagen in Drosophila. It can interfere with mitotic and meiotic chromosome segregation in plants and animals, and in some rodent species it has been shown to cause reduced fertility. Gonadal exposure has not been assessed in exposed human individuals, and there are very few data from other mammals. Exposure to methylmercury causes chromosomal abnormalities in rapidly growing plant root cells, and has been reported to cause chromosomal damage in lymphocytes cultured in vitro from methylmercury exposed human individuals. The significance of these observations for human health remains unclear.

While methylmercury appears not to be a strong
mutagen, its effects on human reproduction and
chromosomes require further observations before the
hazards can be more accurately evaluated.

TERATOGENIC EFFECTS OF METHYLMERCURY

Though significantly increased frequencies of
congenital malformations such as cleft palate have been
observed in different strains of mice exposed prenatally
to methylmercury, no existing data on human exposure in
utero implicate methylmercury as a teratogen (Spyker and
Smithberg 1972, Su and Okita 1976b). It must, however,
be emphasized that no adequate epidemiologic study of
exposed human populations has been undertaken to assess
possible teratogenic effects. The relatively small
numbers of prenatally exposed individuals and logistical
difficulties arising from geographic location make such
studies extremely difficult to carry out. The degree of
teratogenic risk from human prenatal exposure to
methylmercury may thus remain undefined for the
indefinite future.

CARCINOGENIC EFFECTS OF METHYLMERCURY

Little information has been published on animals and
none on man with which to assess the potential
carcinogenic hazard of exposure to mercury compounds.
The carcinogenic potential of exposure to methylmercury
appears to be low, but the available data are inadequate
to make a reliable assessment.

In rats, sarcomas developed in areas in direct contact
with metallic mercury injected intraperitoneally, but no
metastases were observed (Druckery et al. 1957).
Schroeder and Mitchener (1975) reported no significant
difference in tumor frequencies between control,
unexposed, random-bred white Swiss mice and mice with
lifetime exposures to methylmercury in drinking water
(5000 ng/ml × 70 days, then 1000 ng/ml subsequently).
It has also been shown that prolonged exposure of mice
to 1000 or 10,000 ng/g methylmercury in their feed did
not alter the course of neoplasia following innoculation
with Rauscher leukemia virus (Koller 1975).

Lifetime exposure studies have been done in both sexes
of only one species, the mouse. Current recommendations
suggest the use of two rodent species for
carcinogenicity testing. Since transplacental exposure
can occur, exposure should begin before conception, and
the offspring should continue to be exposed for life
(NRC 1975).

SUMMARY AND CONCLUSIONS

Though data about the methylmercury content of marine and freshwater fish and populations have accumulated rapidly during the past few years, several gaps remain in the information required to assess accurately the hazards of human dietary exposure to methylmercury in fish.

With the possible exception of certain Canadian native populations exposed to methylmercury through eating large quantities of freshwater fish, no human poisoning has yet been documented to result from eating ocean or freshwater fish from waters not directly contaminated by the anthropogenic release of mercury. In studies of acutely and chronically exposed adult human populations in Iraq, Samoa, Peru, and Sweden, no clinically affected adults were observed who had blood methylmercury levels below approximately 400 ng/ml. However, in the exposed Iraqi population approximately one-third of the women who had symptoms or signs attributable to methylmercury poisoning had maximum hair mercury levels less than 100,000 ng/g (estimated to be equivalent to blood levels less than 400 ng/ml).

However, it should be emphasized that the current clinical limits for detecting the effects of methylmercury in human populations should not be equated with threshold levels because other more subtle effects, such as behavioral or intellectual deficits, may not be detectable by the clinical procedures that were used.

It should also be noted that other environmental factors such as nutritional status and concurrent exposure to other intoxicants and infectious agents have not yet been evaluated for their additive or potentiating effects. The possible protective role of dietary selenium, particularly in ocean fish, remains to be determined (see discussion in Chapter 5).

The question of whether appropriate dose estimation should be based on peak concentrations in index media or concentrations integrated throughout the duration of exposure remains to be answered.

Prenatally exposed children in the Iraqi population are currently being observed but final conclusions will not be possible until these individuals have been observed through their reproductive years and aging. Observations made so far suggest that there is a high risk of clinically detectable fetal brain damage when maternal hair mercury concentrations rise above 100,000 ng/g (equivalent to a blood mercury concentration of approximately 400 ng/ml). Because only a small number of prenatally exposed infants has been observed, it is likely that studies of larger exposed populations would reveal a distribution with some infants affected at

considerably lower maternal hair and blood
concentrations. Continued careful evaluation of this
very important cohort of prenatally exposed individuals
will probably provide the most sensitive assessment of
human methylmercury toxicity.

The human population is outbred, and genetic
uniqueness of each individual (except for identical
twins) can be assumed. Thus, variability among
individuals is the rule, and though most individuals in
a population will fall within a normal distribution in
terms of dose response, there is a yet undetermined
number who may have exceptional susceptibility to
adverse effects of methylmercury owing to genetic and/or
environmental factors. These individuals probably make
up a small fraction of the human population, but we have
as yet no means of estimating the numbers of people at
special risk.

Relative susceptibility at different stages of the
human life cycle, especially the developing organism
(organogenesis through puberty), and sensitivity of aged
or disabled individuals have not yet been adequately
assessed. It is prudent to retain a considerable margin
of safety in the exposure limits recommended for
pregnant women as well as other potentially susceptible
individuals. Until more definitive evaluations of the
exposed native Canadian population and the prenatally
and perinatally exposed Iraqi populations have been
completed, the guidelines concerning human exposure to
methylmercury suggested in the WHO document (1976b),
Environmental Health Criteria 1, Mercury, should be
adhered to.

NOTES

1 For more detailed information concerning toxic human
  health effects due to methylmercury exposure the
  reader is referred to the extensive reviews and
  summaries to be found in Berglund et al. (1971), U.K.
  Department of the Environment (1976), WHO (1976a,
  1976b), and Tsubaki and Irukayama (1977).

2 The literature describing possible genetic,
  reproductive, teratogenic, and carcinogenic effects
  has been collated and described in greater detail than
  the toxic effects literature since there is no recent
  summary description available in the literature.

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APPENDIX A

MERCURY ANALYTICAL METHODS[1]

CONSIDERATIONS IN ANALYSIS

Analysis is implicit in every document or article citing mercury concentration data, regardless of whether the source of mercury is experimental, environmental, animal, or human. In the event of contradictory data, it may be important to judge the reliability of the trace level analysis. Hume (1973) has examined the present state of the art of trace metal analysis and its implications; and this discussion follows his general comments.

Hume (1973) points out the general lack of interlaboratory agreement in analyses of blind samples. An example is given from an unpublished report dated 1970 of 13 laboratories that participated in analyzing the trace metal content (not mercury) of three subsamples, each taken from two large, primary samples of seawater. Conventional atomic absorption spectroscopy, neutron activation analysis, or colorimetric procedures were used. The reported values ranged from 3.7 to 47 µg/kg (ppb), with a range of relative standard deviations per set of analyses between 3 and 70 percent. Such results do not indicate that water chemists are particularly inept. Rather, Hume (1973) hastens to mention that the same lack of consistency is, at least to some degree, characteristic of all experimental measurements. However, the discrepancies are not obvious except in demanding procedures such as trace metal analyses where the "signal to noise" ratio is unfavorable and the pitfalls are numerous. Nonetheless, some sources of discrepancies can be isolated, examined, and possibly eliminated.

Sources of Discrepancies in Analytical Results

The operator and instrument variabilities are understandable sources of disagreement and can be

controlled somewhat beyond automated procedures and
instrumental techniques as are now largely implemented
at government agencies like the U.S. Environmental
Protection Agency (U.S. EPA 1974). Other sources
require an appreciation of the minute quantities of
metal measured (Table A.1). A natural mercury
concentration of 1 ng/g, or 1 ppb, usually corresponds
to less than one atom of mercury for every 10^9 (billion)
atoms of other substances. Detection of this tiny but
commonly encountered concentration is a result of a
multistage process, each step of which provides a means
for the introduction of errors. Before the actual
instrumental measurement, the sample is usually
collected, stored, pretreated for quantitative
subsampling, then chemically treated for separation or
concentration. A typical chemical treatment for a
biological sample is as follows (Smith and Windom 1972):
(1) weigh 0.5 g of frozen tissue into a 100-ml beaker,
(2) add 4.0 ml of concentrated sulfuric acid and 2.5 ml
of concentrated nitric acid, (3) cover beaker with a
watch glass and place in a water bath at 58°C overnight,
(4) carefully transfer the sample solution to a BOD
bottle, washing the original sample beaker with
redistilled water, (5) dilute sample solution to 100 ml
and add 1 ml of potassium permanganate solution, (6)
shake and add additional portions of potassium
permanganate solution until the purple color persists at
least 15 min, (8) add 2 ml of potassium persulfate
solution in 2-ml increments until a clear solution is
observed, (9) add 5 ml of stannous sulfate and
immediately attach to aeration assembly, and (10) from
recorder peak height, find the μg of mercury from a
calibration curve.

At each step, metal can be lost or gained. Reagents
are notorious for possessing background metal ion
concentrations, often far in excess of the sample (U.S.
EPA 1972). Possible loss of mercury compounds by
volatilization during chemical processing has long been
recognized as another potential source of error.
Likewise, during storage, mercury can be leached into
solution from the laboratory or container materials or
be lost to container walls by sorption (Campbell et al.
1972). Thus, one reason for promotion of neutron
activation analysis is the accessibility of a blank
container count before sample collection (Thatcher and
Johnson 1971). A recent publication (Weiss and Chew
1973) presents data indicating a 30 percent loss of
induced activity (12 percent to the walls of the vial)
in irradiation of unacidified aqueous mercury solutions.
The loss is thought to occur by two routes: adsorption
and volatilization during irradiation.

TABLE A.1   Units of Weight and Concentration[a]

Description and Unit	Equivalent
Weight:	
1 kilogram (kg)	1,000 grams (g)
1 milligram (mg)	10^{-3} g
1 microgram (μg)	10^{-6} g
1 nanogram (ng)	10^{-9} g
1 picogram (pg)	10^{-12} g
Concentration:	
Weight: weight basis (for foods, body organs, and other solids)	
1 part per million (ppm)	1 mg/kg or 1 μg/g
1 part per billion (ppb)	1 μg/kg or 1 ng/g
1 part per trillion (ppt)	1 ng/kg or 1 pg/g
Weight: volume basis (for water)	
1 mg/liter	approximately 1 μg/g on weight: weight basis
1 μg/liter	approximately 1 ng/g on weight: weight basis
1 ng/liter	approximately 1 pg/g on weight: weight basis
Weight: volume basis (for air)	
1 mg/cubic meter	10^{3} μg/cubic meter or 10^{6} ng/cubic meter

[a]The conversion of a weight:volume relationship to a volume:volume basis depends on the molecular weight of the dispersed substance (200.6 g/mole for elemental mercury). At 25 C and 760 mm Hg pressure, the conversion formula is: ppm by volume = mg/cubic meter $\times$ 24.45/molecular weight where mg/cubic meter is the measured concentration of mercury.

In addition to the trace metal, environmental samples
contain an unpredictable assortment of inorganic and
organic substances that can contribute to analytical
interference. Natural chelating agents derived from
decomposition of plant and animal matter can prevent
water-organic liquid extraction of the metal. Algal or
bacterial action on mercury may continue in stored
natural fluids while mercury in tissue samples continues
to change during decomposition. Both processes can be
controlled by proper treatment at the time of
collection.

In conclusion, trace mercury analysis in the ng/g
range is a demanding procedure, successfully
accomplished only by considering and avoiding or
compensating for all the known pitfalls. Only analyses
that use proven or standardized procedures and report
standard deviations as well as calibrations with a known
standard or spiked sample should be accepted. Trace
metal analysis is presently an extremely active area of
research directed at establishing procedures leading to
increased sensitivity and accuracy.

Evaluation of Early Analytical Results

Techniques for trace metal analyses have undergone
considerable improvement in the past 10 yr, leading to a
need for assessment of earlier analyses. The change
itself is not as important to recognize as the direction
of change. Before the mid-1960s, for instance, many
analyses of biological tissue determined total mercury
without distinguishing between inorganic and organic
forms. In identifying methylmercury as the prevalent
form in fish, Westoo (1966) focused attention on the
importance of analysis for organomercury compounds.
Today, analyses of environmental or biological samples
for mercury are more likely to involve detection of the
form as well as the quantity. Likewise, as improvements
in analytical techniques have been instituted, greater
sensitivity, selectivity, and reliability have been
attained. These factors lead to improved detection of
the mercury in a sample even as the concentrations
diminish.

Since more pathways for error lead to apparent low
rather than apparent high determinations, it is probably
safe to assume that, if earlier analyses were in error,
they were more likely to have reported concentrations
lower than the true value. Mercury was reported absent
because of poor instrument detectability. Also,
volatile mercury compounds were lost in the hot
digestion of samples, or mercury was not totally
recovered because of inefficient oxidizing reagents.

Processing for total mercury analyses often neglected organic mercury compounds entirely.

On the other hand, the commonly cited mechanism for higher-than-true mercury determinations, contamination of reagents and laboratory ware, can contribute parts per billion, or at most parts per million, in particularly aggravated cases. Blank determinations along with purification of reagents and treatment of labware help eliminate this source of error. Another important route that leads to apparent high values involves the loss of mercury from synthetic standard solutions. They are almost invariably prepared with distilled or deionized water. As noted in Table A.2, unless the sample is carefully treated, the half-life of mercury in distilled water is as little as half that of mercury in natural water; the loss involves adsorption onto or diffusion into container walls. Natural water contains an abundance of soluble ions thought to prolong the half-life of mercury (Rosain and Wai 1973). Feldman (1974) noted an apparent increase with time in the mercury concentration in natural water samples. The increase was attributable to a decrease with time in the strength of his synthetic standard solution. His recommendations for the preservation of standard solutions are discussed later in the section on chemical treatment of samples.

A fair portion of the systematic errors in mercury analysis are now likely to have been detected and documented. Although there is always a lag between documentation and implementation, the improved agreement in interlaboratory analyses (discussed later in this paper) may be indicative of improved analyses in general. Likewise, the use of standard methods and environmental samples lends added confidence to recent analyses.

ANALYTICAL PROCEDURES

Storage and Preservation

A sample that cannot be analyzed immediately must be stored. Contrary to expectation, this step is not a static phase of the analytical procedure; in fact, it presents another route for the loss of mercury. In this case "loss" indicates mercury originally present that is undetectable. Rosain and Wai (1973) studied the rate of mercury loss from solutions of natural and distilled water spiked with 25 μg/l mercury ion when stored in stoppered polyethylene, polyvinyl chloride, and soft glass containers. Severe losses observed at pH 2 and 7 were considerably curtailed by acidification with nitric

TABLE A.2 Half-Life in Days of 26 μg/1 Mercury in Aqueous Solution

| Container | pH 7 | | pH 2 |
	Distilled Water	Creek Water	Creek Water
Polyethylene	1.58	3.95	4.0
Polyvinyl chloride	1.98	4.38	1.7
Soft glass	2.98	4.44	3.5

SOURCE: Rosain and Wai (1973).

acid to pH 0.5. The half-lives of stored mercury are
given in Table A.2. The loss is exponential, with only
half the original quantity remaining after as little as
1.5 days. At pH 0.5, the loss was not detected after
4.4 days and was barely detected (2 percent loss) after
15 days. Feldman (1974), working at the 0.1 to 10.0
µg/l range, found losses of mercury from both glass and
polyethylene containers in spite of acidification and
preservation with potassium permanganate. Addition of
0.01 percent dichromate ion in place of permanganate
stabilized the acidified solutions for as long as five
months.

Solid biological samples are adequately stored without
loss of mercury by freezing immediately after collection
(Kopp et al. 1972). Preservation and storage of urine
samples have been discussed by Trujillo et al. (1974).
Potassium persulfate, added at the time of collection,
preserves the sample for several days.

Concentration and Separation

In principle, any mercury concentration, no matter how
small, can be determined if a sufficiently large sample
is collected and the total mercury separated into a
small volume. However, any process, no matter how
judiciously applied, represents a route for the loss of
mercury.

Evaporation

Concentration and separation are essentially the same
process. The first removes the matrix from the metal;
the second removes metal from the matrix. The simplest
concentration technique is evaporation, a valuable but
frequently undesirable method, considering the
volatility of many mercury compounds, particularly
organomercurials. Nonetheless, it has been used
advantageously to concentrate mercurials in benzene
solution by evaporation of a portion of the benzene
after liquid-liquid extraction of organomercury salts
preparatory to gas chromatographic injection (Longbottom
et al. 1973).

Solvent Extraction

This technique is widely used in aqueous analyses for
separating and concentrating organic mercury compounds
and depends upon the differential solubility of mercury
compounds in mutually immiscible liquids, for example,

benzene and water. Specifically, benzene or toluene is added to a mercury-containing aqueous solution, and the mixture is agitated sufficiently to allow optimum transfer of the solute between the two phases. Then the mixture is allowed to separate, and the organic phase containing mercury is drawn off. This procedure may be repeated with fresh solvent as often as necessary to effect as complete a recovery as possible. Inorganic mercury may be extracted into an organic solvent by adding an organic chelating agent to form an organic mercury complex. Dithizone, widely used to extract inorganic mercury, not only complexes quantitatively but it forms a colored complex that is detected spectrophotometrically (Sandell 1959). The principles and applications of solvent extraction to water analysis are discussed by Andelman (1971).

Amalgamation

Like solvent extraction, amalgamation depends on the differential solubility of a solute, in this case elemental mercury, in each of two different phases. Traces of metallic mercury are immediately soluble in gold and silver. Grids of gold or silver are placed in an airstream to collect airborne elemental mercury; a gold-coated, fritted glass disk has been used to capture mercury by passing air through it at 1 l/min. Similarly, mercury has been collected from acidified water samples by amalgamation on a silver wire (Fishman 1970) or by electrodeposition onto a copper cathode (Doherty and Dorsett 1971), although the presence of sulfide ion in the water causes low results (Fishman 1970). Amalgamated mercury is released (usually into an atomic absorption cell) by heating to drive off the volatile metal.

Carbon Adsorption

Atmospheric mercury can be concentrated on activated carbon. Moffitt and Kupel (1970) used a commercially available impregnated charcoal to trap mercury in industrial atmospheres. Air is pumped through a sampling tube containing two tandem charcoal sections separated from each other and from the atmosphere by glass wood plugs. The glass wood plug at the inlet end of the tube is analyzed for particulate-bound mercury, and adsorbed volatile mercury is determined by atomic adsorption spectroscopy. The effectiveness of the charcoal filter is determined by the absence of mercury on the second section.

Ion Exchange

Ion exchange separation is a well-established analytical technique, although applications to mercury analysis have been few. Selectivity occurs as a result of the relative affinity of a given ion exchange resin for particular ions (Andelman 1971). An anion exchange, resin-loaded filter paper has been used to remove mercury from natural water containing from 0.03 to 6.5 μg/l mercury (Becknell et al. 1971). Both the inorganic and organic forms of mercury are converted to $HgCl_4^{2-}$ before filtering. The mercury-loaded papers are air dried and then sealed in mylar film for irradiation before neutron activation analysis. In another study, mercury collected directly from seawater on a chelating resin was so strongly retained that no reagent was able to elute it completely for analysis by atomic absorption spectroscopy (Riley and Taylor 1968).

Chemical Treatment of Samples

Aqueous Mercury

Mercury in aquatic samples appears as inorganic or organic species either dissolved, sorbed onto particulate matter, or entrained within a particulate matrix. Total mercury determination measures all of these. Thus, samples require pretreatment to release mercury from all the forms in which it is collected and to transform it into a state that is compatible with the measuring technique. Kopp et al. (1972) have described some of the obstacles inherent in the pretreatment procedure for lake and river waters and have also proposed methods for overcoming them. The addition of acids to aqueous samples, for example, generates heat, which can lead to losses of mercury compounds by volatilization. Aquatic materials also require an oxidizing agent strong enough to decompose the organomercury compounds completely. Kopp et al. (1972) demonstrated (1) the ineffectiveness of commonly used potassium permanganate only to release all organically bound mercury, and (2) the success of added potassium persulfate in conjunction with potassium permanganate in total recovery of mercury from solutions containing dissolved phenyl and methylmercuric salts. The procedures now incorporated into the standard methodology for water and wastes (U.S. EPA 1974) are outlined briefly: (1) an aliquot is acidified with sulfuric and nitric acids, (2) potassium permanganate solution is added until the purple color persists for at least 15 min, (3) potassium persulfate is added, and

heat is applied for 2 h at 95°C, (4) sodium chloride-
hydroxylamine sulfate solution is added to reduce excess
permanganate, and (5) stannous sulfate solution is added
just before aeration into the atomic absorption cell.

Biological Mercury

To analyze for mercury in biological tissue, it is
important to determine the total mercury content and
also the fraction of organomercurial compounds,
methylmercury in particular. Uthe (1971) describes a
thoroughly tested total mercury procedure essentially
like that outlined in the section above of sources of
discrepancy of analytical results except for semi-
automation of the method. The procedure consists of
cold, wet, acid oxidation to decompose the organic
matter and release mercuric mercury, which is then
reduced to elemental mercury, the form aerated from
solution for atomic absorption measurement.

Alkylmercury determinations require a completely
different approach to preserve the organic-bond formed
during extraction from the matrix. Most techniques are
variations of the original Westoo method (Westoo 1966,
1967, 1968), and they consist basically of organic
solvent extraction and re-extraction. Chemical
treatment precedes extraction to remove interfering
substances such as sulfides and to convert the
organomercury compound to the halide salt (chloride or
bromide), the form most accessible to electron capture
detection after chromatographic separation.
Dialkylmercury compounds such as dimethylmercury are not
as strongly bound to the matrix and are easily extracted
into an organic medium (benzene or toluene) as a first
step before chemical treatment of the remaining sample.
Care is required, however, to prevent loss of the highly
volatile dimethylmercury. This extracted mercury is
then subjected to halide treatment: the
monomethylmercury halide thus produced can then be
analyzed by gas chromatography (Mushak 1973).

The U.S. Environmental Protection Agency has proposed
for adoption a procedure for methylmercury determination
in biological media as described by Longbottom et al.
(1973). Preservation, storage, chemical treatment,
extraction, and cleanup are included. Copper sulfate is
introduced at the time of collection to preserve the
integrity of mercury compounds until analysis. Fish and
sediment samples are frozen upon collection, thawed
before use, then treated with copper sulfate to displace
methylmercury from its strong, natural inorganic and
organic sulfur bonds. The free methylmercury is
converted to the bromide salt by addition of excess

potassium bromide to a solution strongly acidified with sulfuric acid (pH below 0.5); then it is extracted into a toluene layer. The extract cannot be injected into the gas chromatograph until sulfur compounds are completely removed. This is the purpose of the cleanup step, accomplished by high-efficiency extraction of methylmercury into an aqueous thiosulfate phase, addition of potassium iodide, then re-extraction into benzene or toluene for injection into the gas chromatograph. Any of the extraction or cleanup steps may be repeated for improved recovery.

Airborne Mercury

Mercury appears in air in several forms: as elemental free mercury atoms, as inorganic or organic mercury molecules, and as any of these adsorbed on particulate matter in the air. All forms present a hazard that necessitates their detection and monitoring.

Elemental mercury vapor, the form usually monitored, requires no pretreatment and is detected directly by drawing air through a high sensitivity atomic absorption spectrometer (Jepsen 1973). Alternatively, mercury vapor can be collected before analysis (1) by metering airflow through flasks containing potassium permanganate-sulfuric acid or iodine-potassium iodide solution, (2) by adsorption onto impregnated charcoal, or (3) by deposition on filter materials (Lindstedt and Skerfving 1972, Bogen 1973). Airborne mercury trapped in solution is recovered for analysis by any of the standard pretreatment methods, whereas that deposited on filters, including particulate matter, may be determined directly by neutron activation analysis (Bogen 1973).

These collection systems are inadequate to detect organic mercurials. Linch et al. (1968) found less than 20 percent of dimethylmercury and less than 50 percent of diethylmercury in air retained by an aqueous iodine-potassium iodide reagent, and less than 5 percent of dimethylmercury is retained by potassium permanganate-sulfuric acid solution. Recoveries averaging 98 percent for dimethylmercury in the 2 to 60 μg range, however, were attained with an iodine monochloride reagent. Near quantitative collection of diethylmercury, methylmercuric chloride, and ethylmercuric chloride also was established. Iodine monochloride is considered to be the only known absorbent that is effective for collecting both inorganic and organic mercury (Anonymous 1973a). The final mercury determination, however, requires a method free of interference from iodine. In other words, atomic absorption spectroscopy cannot be used.

Methods of Analysis

The reliability of any analytical procedure can be no
better than its least reliable step. Even assuming no
systematic errors, the uncertainty of an analysis is the
sum of the uncertainties contributed by the individual
analytical processes. This uncertainty arises from
random error associated with any experimental procedure.
For this reason, an analysis is a continual checking
process, first to eliminate any systematic error, and
second to minimize random error.

The crucial instrumental operation often is the
limiting factor in an analysis, and most of the
preliminaries are preparatory to the instrumental step.
In fact, an analytical method is named for the
instrument step even though it oftens requires the least
time and effort. Table A.3 shows the common
instrumental methods, including a very brief description
of sample preparations and measurement techniques.

The sensitivity (column 6) is largely instrument
dependent, experimentally determined, and variously
defined by different authors. Essentially, the
threshold of sensitivity is the smallest quantity of
substance that can be determined reliably by the
instrumental technique. In atomic absorption
spectroscopy, for instance, the sensitivity has been
defined as that concentration of analyte that yields 1
percent of a full-scale reading, or that produces an
absorption of 1 percent (Minear and Murray 1974), or
that is 3 times the standard deviation of blank readings
(Anonymous 1974).

The precision of the method is given in column 7 as
the relative standard deviation (that is, the standard
deviation of a set of sample measurements divided by the
concentration of the sample). The sample concentration
is also shown since the precision is strongly dependent
on it.

Column 8, accuracy, is measured by a procedural
calibration technique. Kaiser (1973) has discussed the
three main calibration methods for trace determination:
with synthetic standard samples, with analyzed standard
samples, or by differential additions. Ideally,
synthetic standards are prepared and processed with the
sample. With mercury, however, it is exceedingly
difficult, if not impossible, to prepare synthetic trace
samples from truly pure substances and to transfer them
into a state that duplicates the natural analytical
sample. Use of pre-analyzed natural samples solves this
problem but presupposes that the problem of analyzing a
natural trace sample with adequate calibration
procedures has already been solved (Alvarez 1974).
Nonetheless, the National Bureau of Standards (NBS)

biological pre-analyzed samples are available, and their use is increasing (Anonymous 1973b). The most prevalent calibration method, by differential additions, is the one listed here. Small, known quantities of a mercury compound are added to the unknown samples before processing. Recovery of this quantity measures the accuracy of the procedure. The main problem is to insure that the spike is in the same physical and chemical condition as the natural mercury component.

Other than the columns discussed, Table A.3 is intended to be self-explanatory. However, it is not intended to be a complete description of analytical methods. For instance, although total mercury is determined in atomic absorption spectroscopy, this does not preclude determination of organomercury concentrations. First, an intact sample is processed; then a sample in which the organomercurials have been separated by organic liquid extraction is processed. The difference between the two determinations gives the concentration of organomercury compounds. However, the species of the organomercurial cannot be determined directly by atomic absorption. A brief description is given in the following sections of the listed instrumental methods, preceded by a few well-known chemical methods. Some newer exploratory techniques are discussed in the later section on newer methods of analysis.

Gravimetric Analysis

All gravimetric procedures are essentially alike. The sample is digested with a reducing solution to convert all mercury to the elemental form. The mercury is driven off as a vapor by heating and is collected by amalgamation on a metal foil or screen. Gold or silver is usually used, and the mercury is determined by the difference in weight before and after the amalgamation. With routine analytical balances, a few mg of mercury can be determined with reasonable accuracy; with the newer microbalances, this level can be extended to a few μg. A variation of this method involves the deposition of the mercury on a platinum cathode. This is the method recommended by the American Society for Testing and Materials to measure mercury in paint (Anonymous 1972).

TABLE A.3 Methods of Analysis

(1) Analytical Method	(2) Important Application	(3) State of Measured Mercury	(4) Sample Preparation	(5) Methodology or Technique
Gas chromatography using electron detector	Biological material; Waste sludge: Bottom mud	Organomercury Components	Sample is homogenized; treated with acid, organic solvents, etc. Components are extracted with benzene.	Sample components are separated by chromatographic technique and analyzed qualitatively and quantitatively.
Atomic absorption spectroscopy cold vapor method	Effluents, wastewaters, surface and saline waters; soils and sediments after dissolution in aqua regia. Also for air and biological materials.	Elemental mercury vapor	Acidify sample with HNO_3 at time of collection. Filter if necessary, then chemically convert all mercury to mercuric ion.	Mercury is reduced to elemental state then aerated into a cell placed in the path of light of 253.7 nm wavelength. The measured absorbance is a function of mercury concentration.
Neutron activation analysis	All media and materials, biologic and inorganic.	As is	Water samples must be concentrated or extracted onto an ion exchange bed. Other materials are dried or freeze dried.	Sample is irradiated for 1-24 hours with a stream of neutrons. 197 Hg and 203 Hg are counted using a multichannel analyzer.
Colorimetric spectrophotometry	Aqueous samples, biological materials; soils and sediments.	Mercury dithizone complex	Sample is shaken with excess dithizone in acidic solution then extracted with organic solvents (organomercurials require oxidative digestion).	Absorbance of the complex at 490 nm or reduction in absorbance at 610 nm is measured.

(6) Reported Sensitivity	(7) Precision (relative standard deviation/sample size)	(8) Accuracy (recovery from spiked sample)	(9) Some Interfering Substances	(10) Selectivity	(11) Comments
0.01 μg/g (fish) 0.001 μg/g (sediment)	±10% (0.3 μg/g) ±10% (0.01 μg/g)	95.5% (0.20 μg/g) 96.3% (0.01 μg/g)	Sulfide in sample requires refined pretreatment to free organomercury.	The concentration and species of organo-mercury compound is determined.	Loss of volatile mercury compounds during pretreatment must be avoided. Injection system should be checked to prevent alteration of the organomercury compound in the injection chamber.
0.20 μg/g	±46% (0.55 μg/ℓ)	89%	Sulfide, free chlorine, iodine, CO_2, SO_2, and volatile organics interfere. Copper may interfere.	Total mercury concentration is determined.	Mercury vapor must be dried before passing through the absorption cell. Water vapor absorbs at 253.7 nm.
0.0005 μg/g (involves post-irradiation separation)	±5% (0.1 μg/g)	98%	Sodium, bromine, phosphorus may interfere. Separation techniques avoid the interference.	Total mercury is determined.	Care must be taken to prevent mercury loss by volatilization during irradiation.
0.05-0.5 μg/g	4-5%		Copper, silver, gold, palladium and platinum in trace amounts.	Total mercury is determined.	Good, but lengthy method for routine analysis of samples containing 0.5 - 50 μg/g Hg and only traces of Pb, Zn, Ni or Co.

SOURCES: U.S. EPA (1973, 1974), Anon. (1973a), Friberg and Vostal (1972), Wallace et al. (1971).

Volumetric Analysis

Aqueous volumetric procedures depend upon the conversion of mercury to Hg^{2+} ion, complexation of the ion, then titration to a readable end point. In one widely used method, the Volhard titration, potassium thiocyanate is added in excess to form the sparingly soluble compound, mercuric thiocyanate. The excess thiocyanate is titrated with ferric ion to produce a brilliant red color. The advantage of the Volhard titration is that very few metals interfere; the disadvantage is that the reaction is not entirely stoichiometric. Thus, corrections are needed that may introduce uncertainties beyond the accepted tolerances for some trace analyses (Coetzee 1961). There are also other titrimetric reactions that involve oxidation reduction reactions.

Micrometric Determination

With this method, mercury is electrodeposited from solution onto copper wire; the mercury is then distilled from the copper wire in a capillary tube. The volatilized mercury condenses in a cooled section of the tube, the condensate is united into a globule, and the diameter of the globule is measured with a microscope. Under favorable conditions, amounts exceeding 0.5 μg can be determined with an accuracy of ±2 percent, and as little as 0.01 μg with an accuracy of ±10 percent (Coetzee 1961).

Colorimetric Spectrophotometry

Until the development of more sensitive instrumental methods, dithizone extraction followed by photometric analysis was the method of choice for trace environmental mercury samples. Dithizone (diphenylthiocarbazone) complexes quantitatively with mercuric ion in acidic aqueous solution. The complex is extracted with chloroform as orange mercury dithizonate, which exhibits an absorption maximum at 490 nm. Various techniques are required to eliminate interfering metals (Sandell 1959). The sensitivity and procedures of the method are outlined in Table A.3.

Cold Vapor Atomic Absorption Spectrometry

The use of a cold vapor atomic absorption method for trace mercury analysis is so direct and accessible under

ordinary laboratory conditions that it has been detailed as an experiment for instruction in an undergraduate course in analytical chemistry (Lieu et al. 1974). The equipment is shown in Figure A.1. A hollow cathode mercury discharge lamp supplies radiation at 253.7 nm for absorption by atomic mercury vapor aerated from solution into the absorption cell. Mercury ions in solution are reduced to elemental mercury by stannous chloride, then swept with helium into the cell where radiation is absorbed by the mercury atoms. The concentration of atoms is determined from the absorbance by comparison with a standard curve. Atomic absorption is highly specific for the particular metal under investigation. Most interfering agents are readily removed, and it is suitable for measurement of most environmental materials after pretreatment. Elemental mercury in air, for instance, is monitored directly by the passage of air through an atomic absorption cell (Jepsen 1973). Unfortunately, atomic absorption determines only the total mercury content and gives no information about the species or form of the mercury.

The cold vapor method has been readily adopted and will likely become the favored method eventually. It is fast, specific for mercury, suitable for all sorts of pretreatment variations, applicable to extremely low concentrations, and above all, the equipment is readily available and reasonable in cost. The Bureau International Technique Du Chlor has selected cold vapor atomic absorption spectrometry as their standard (Anonymous 1974). The sensitivity and accuracy are well within the 0.1 μg/g ±10 percent cited as desirable for regulatory purposes (Alvarez 1974).

Gas Chromatography with an Electron Capture Detector

Gas chromatography actually refers to a separation technique. Coupling this with a detector for sensing the separated components constitutes an instrumental method, the only one in wide use that determines organic mercury compounds directly. These compounds are converted to organomercuric halides to make the mercury volatile and chromatographable. After extraction into an organic solvent, the organic mercury compounds are injected into an inlet where they are vaporized and the gases passed through a chromatographic column. The absorbent in the column is often a high-boiling liquid adsorbed on a solid substrate; hence, the term gas-liquid chromatography. The volatile organics, partitioned between the stationary phase and the mobile carrier gas, emerge from the column at different times and are determined by one of several detection devices.
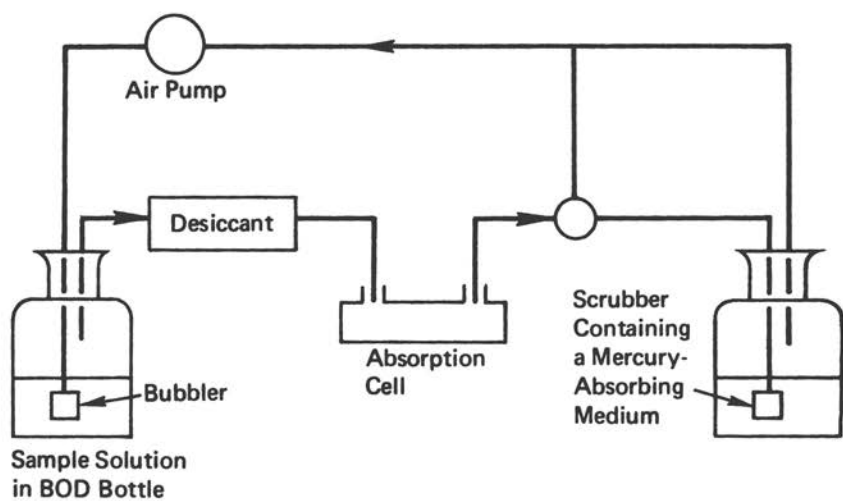
SOURCE: U.S. EPA (1974).

FIGURE A.1 Apparatus for flameless mercury determination.

The electron capture detector, used almost exclusively
in organomercury determination by gas chromatography,
employs a tritium or [63]Ni radioactive source. Its beta
radiation produces electrons in the carrier gas that are
captured by sample molecules, thus reducing the current
of electrons to an anode under fixed voltage. The
decrease in current is a measure of the amount and
electron affinity of the component (Andelman 1971).

Dressman (1972) found that phenylmercuric salts,
except phenylmercuric chloride, are converted in the gas
chromatographic injection block to diphenylmercury as
the major product, which is overlooked by electron
capture detection. Baughman et al. (1973) found a
similar conversion of methylmercury salts to
dimethylmercury, and they suggest the use of specially
treated columns for reliable analyses.

Neutron Activation Analysis

The use of neutron activation analysis of mercury in
bioenvironmental materials is now well established.
Usually, the sample is sealed in quartz vials and
irradiated with neutrons to convert [196]Hg to [197]Hg, a
radioactive isotope with a 65 h half-life. The [197]Hg is
identified with a multichannel analyzer and Ge(Li)
detector (Westermark 1972). The most important and
useful aspect of activation analysis is that no
treatment of the sample is needed before neutron
irradiation, although postirradiation separation can be
performed to increase sensitivity by removal of
interfering substances.

The major limitation on activation analysis is its
inability to distinguish the chemical state of mercury
in the sample. In addition, the excessive equipment
cost relative to other methods and the need for a nearby
neutron source (reactor) has, to some extent, reserved
this method primarily for referee analyses.

Newer Methods of Analysis

Atomic Fluorescence Spectrometry

Atomic fluorescence depends on excitation of mercury
atoms with a mercury lamp followed by detection of the
emitted radiation when the atoms return to the ground
state. This method is reported to have a sensitivity
advantage of at least a factor of 10 over atomic
absorption (Subber et al. 1974). Atomic fluorescence
spectrometry is useful to detect mercury levels between

0.1 ng and 1 μg. Above 1 μg the calibration curve becomes nonlinear (Subber et al. 1974).

The techniques for pretreatment and for reduction and release of mercury from solution are exactly the same as for atomic absorption. Here, however, the 253.7 nm radiation from a mercury line source is focused on the vapor, and the resulting 253.7 nm fluorescence emission is monitored. The added sensitivity is derived, in part, from the fact that the absorption is a very small quantity determined as the difference between two large quantities, whereas emission is this same quantity determined directly (Minear and Murray 1974). In addition, the emission is a function of the intensity of the impinging radiation, which can be varied to some extent to increase the signal. Muscat et al. (1972) used atomic fluorescence to determine mercury in a number of environmental reference materials and sediments. For a water sample from the Analytical Quality Control Laboratory, Cincinnati, containing 4.2 ng/ml, a value of 4.2±0.4 ng/ml was obtained. Wheat flour samples (International Atomic Energy Agency, Code 66/10) yielded 5.1±0.5 μg/g compared with a pooled analysis of 4.59±1.32 μg/g by neutron activation, or 4.92±0.45 μg/g by atomic absorption. Atomic fluorescence appears to be a promising technique in terms of sensitivity, availability, and ease of operation.

Spark Source Mass Spectrometry

In an analytical program undertaken by the NBS to provide a reliable value for the mercury content of ground orchard leaves, a standard environmental reference material (SRM 1571), three analytical methods were used: atomic absorption spectrometry, neutron activation, and stable-isotope dilution with spark source mass spectrometry (Alvarez 1974). The first two are well tested, reliable methods. Spark source mass spectrometry has not been previously applied to environmental mercury problems, although it has been used at NBS for standardization needs. The apparatus consists of a spark source mass spectrometer with Mattauch-Herzog geometry (double-focusing). The environmental standards were processed routinely to oxidize the organic matter; then the mercury-containing solution was spiked with 0.370 μg ^{201}Hg and 50 μg ^{198}Hg as a carrier. The mercury was electrodeposited onto high-purity gold wires for sparking in the mass spectrograph. The concentration was calculated from the altered isotope ratio, ^{201}Hg/^{202}Hg. The results are given in Table A.4. The average concentration,

0.141±0.009 µg/g, is in good agreement with averages
determined not only by other methods but by other
laboratories.

Gas Chromatography with a Microwave
Emission Spectrometric Detector

A gas/chromatography microwave-excited-spectrometric
detector (GC-MES) has been built and applied to detect
dimethylmercury, diethylmercury and methylmercuric
chloride at the 6-, 7-, and 4-pg levels, respectively
(Talmi 1974). The relative sensitivity is at the 5 ng/g
level. The technique is reported to be substantially
faster and simpler than most that are presently
available. In this detector, the intensity of the
253.7-nm line emitted from the microwave generated
plasma used as a GC detector is monitored to
characterize the molecule and to determine its
concentration. A comparison with a conventional
electron capture detector (ECD) is given in Table A.5.
The GC-MES appears to be equal to the GC-ECD in
sensitivity, yet superior in all other respects.

Gas Chromatography with a Mass-Spectrometric Detector

Although gas chromatography/mass spectrometry has been
applied for at least 5 yr to study various organic
pollutants (Webb et al. 1973), it has been used only
sparingly to detect organomercural compounds (Johansson
et al. 1970). Baughman et al. (1973) used a gas-liquid-
chromatography/mass-spectrometry (GLC-MS) apparatus to
demonstrate that ionic methylmercury compounds undergo
decomposition during GLC. Johansson et al. (1970)
analyzed eight samples of fish flesh containing from
0.14 to 3.2 µg/g mercury as methylmercury. Excellent
agreement was obtained in a comparison with the results
from using gas chromatography/mass spectrometry, gas
chromatography/electron capture detection, and neutron
activation analysis. The three methods deviated less
than ±10 percent from the average value. The mass
spectrometer provides a positive identification of
organomercury compounds, including organomercuric
iodide.

Stripping Voltammetry

Anodic stripping voltammetry is a convenient,
sensitive method for metals analyses in aqueous media,
particularly seawater (Smith and Windom 1972). The

TABLE A.4 Comparison of Mercury Results by Isotope Dilution with Other Analytical Methods for Orchard Leaves, SRM 1571

Average Concentration and 95% Confidence Limits ($\mu g/g$)	Number of Determinations	Analytical Method	Lab
0.141 ± 0.009	6	Isotopic dilution	NBS
0.160 ± 0.012	15	Atomic absorption	NBS
0.155 ± 0.006	11	Neutron activation	NBS
0.145 ± 0.014	5	Neutron activation	A
0.148 ± 0.010	4	Neutron activation	B

SOURCE: Alvarez (1974).

TABLE A.5 Comparison Between Electron Capture and Plasma Emission Spectroscopy Systems

Item	Electron Capture	Plasma Emission Spectroscopy
Selectivity	Response to electron-absorbing compounds, especially halogens, nitrates, and conjugated carbonyls	Spectroscopical selectivity. Sensitive to practically all elements. Can be used both as selective and general-purpose detector
Sensitivity	Picogram level	Picogram level
Linear range[a]	50 to 100	10^3 to 10^4
Stability	Fair	Good to excellent
Temperature limit	250°C (^{3}H), 350°C (^{63}Ni)	Practically unlimited
Carrier gas	N_2 or Ar– 10% CH_4	Ar and He
Repairs	By producer only	Very simple procedure; usually involves a replacement of the quartz capillary
Life expectancy	Limited by "poisoning" compounds	Practically unlimited; unaffected by any compounds
Remarks	Easily contaminated, easily cleaned, sensitive to water, carrier gas must be dried	Not contaminated, very easy to clean, insensitive to water, sensitive to nitrogen tracers

[a]Linear range is defined as the ratio of the highest to the lowest concentration values that lie on a linear calibration curve.

SOURCE: Talmi (1974).

customary electrode, a mercury drop or mercury film, however, is obviously unsuitable for mercury determination; and since mercury amalgamates with platinum or gold electrodes, another electrode material is required. Allen and Johnson (1973) used a rotating ring-disk electrode that had a glassy carbon disk plated with a thin film of gold. Mercurous ion in 1.0 M sulfuric acid solution was determined in the 0.10- to 4.00-μg/l range, with a relative standard deviation of 7.5 percent. The limit of detection for the technique is approximately 0.01 μg/l. This method is academic and will probably never be used in actual analyses for mercury.

COMPARISON OF ANALYTICAL METHODS

Gas chromatography is generally accepted as the most sensitive, accurate method for organomercury determinations. Controversy continues, however, over the relative merits of atomic absorption spectroscopy and neutron activation analysis for total mercury determinations. Wood (1972) claims the latter technique is superior, asserting that consistently lower results are obtained with atomic absorption analyses. Hume (1973), however, cites a comparison of analytical results for cobalt in two samples of seawater from eight laboratories, four that use atomic absorption with chelation-extraction and four that use neutron activation. The absorption averages were 5 to 6 times larger than those for activation.

Interlaboratory comparisons of different instrumental methods are necessary to elucidate the advantages and pitfalls of various techniques and to display the uncertainty inherent in some trace analyses.

Standardization

Analyses for trace levels of mercury are performed in laboratories throughout the world. Comparisons of these results are difficult, however, because of a lack of uniformity. Kaiser (1973) has addressed this problem in terms of trace analyses in general, presenting an excellent treatment of expressions of accuracy as mentioned above in the section on analytical procedure.

All values measured are subject to errors, e.g., systematic and random. The random error is usually evaluated by the individual investigator or laboratory from a statistical analysis of the data. Systematic errors, on the other hand, are not readily accessible to evaluation individually, although blank readings are

intended to eliminate them. Moreover, this type of error acquires additional importance in trace element determinations because it may contribute disproportionately to the "analytical signal." Wilson (1974) has reviewed the role of systematic and random errors in standardization procedures. An acceptable test for systematic errors includes the use of biological and environmental standard reference materials. They enable the analyst to verify the accuracy (absence of systematic error) in his analytical procedure. The NBS supplies certified biological and environmental samples tested by many techniques, the results of which are shown in Table A.4 for Orchard Leaves, SRM 1571 (Alvarez 1974).

Interlaboratory Comparisons

Interlaboratory comparisons have been conducted since the mid 1960s. These have involved not only different laboratories but also different instrumental methods. An examination of the results of a few of these studies indicates a trend toward increased accuracy, although it is not always immediately apparent, since comparisons have shifted from the $\mu g/g$ to the ng/g range.

A comparative analysis of a standard plant material submitted to laboratories around the world before 1967 resulted in an inconsistent average mercury content of 0.150 ± 0.008 $\mu g/g$ by activation analysis and 0.0122 ± 0.0024 $\mu g/g$ by colorimetric analysis (Bowen 1967). Rottschafer et al. (1971) likewise reported the following inconsistent results when replicate fish tissue samples were analyzed for mercury in 1970 or earlier by different laboratories and various techniques:

Analytical Method	Results ($\mu g/g$)
Atomic absorption, laboratory 1	0.06
Atomic absorption, laboratory 2	1.00
Atomic absorption, laboratory 3	0.85
X-Ray fluorescence	0.40
Destructive neutron activation	0.87

A study reported in 1974 is shown in Table A.4. Here, not only was excellent agreement obtained between different laboratories with the same technique, but also by one laboratory with three different methods. Note that the 100-$\mu g/g$-concentration range was chosen as

desirable for regulatory purposes (Alvarez 1974).
Another recent study (Anonymous 1974) involved one
method, atomic absorption spectroscopy, 37 laboratories,
and samples containing mercury that varied over 3 orders
of magnitude of concentration from 20 µg/g to 20 ng/g.
The results are shown in Table A.6. The relative
standard deviation from the mean is plotted against the
concentration in Figure A.2 to show the strong
functional dependence of the reproducibility on
concentration. High relative standard deviations
occurred at low concentrations, but such results are not
unexpected insofar as "analytical noise" is concerned
(Kaiser 1973).

The studies cited here are not intended to be a
comprehensive treatment of interlaboratory comparisons
with regard to analytical capability. Rather, they are
meant to demonstrate a definite trend toward improved
precision and accuracy in procedures for analyzing
mercury in the environment. Currently, mercury and
organomercurials can be determined about as well as any
trace element or trace organometallic of environmental
significance (Shults 1975).

NOTE

1 This appendix is adapted from pages 26-59, Review of
the Environmental Effects of Mercury, prepared by the
Biomedical Sciences Section, Information Center Complex,
Information Division, Oak Ridge National Laboratory for
the U.S. Environmental Protection Agency, EPA-IAG-D4-
0403. January 27, 1975.

TABLE A.6 Results of Statistical Evaluation of Interlaboratory Analysis for Mercury

| Item | Standard Samples | | Waste Water | Caustic Soda | |
	Ctg. 5.0 mg Hg/kg	Ctg. 20.0 mg Hg/kg		Sample 1	Sample 2
Arithmetic mean	5.03 mg/kg	20.3 mg/kg	3.18 mg/kg	21.4 μg/kg	201 μg/kg
Repeatability[a]:					
Standard deviation	0.18 mg/kg	0.8 mg/kg	0.10 mg/kg	1.7 μg/kg	11 μg/kg
Relative standard					
deviation	3.5%	4.0%	3.1%	8.0%	5.4%
Reproducibility[b]:					
Standard deviation	0.30 mg/kg	1.1 mg/kg	0.34 mg/kg	4.8 μg/kg	38 μg/kg
Relative standard					
deviation	5.9%	5.4%	11%	22%	19%

[a]Repeatability means single laboratory, single operator, and single apparatus precision;
[b]Reproducibility means multi-laboratory, multi-operator, and multi-apparatus precision.
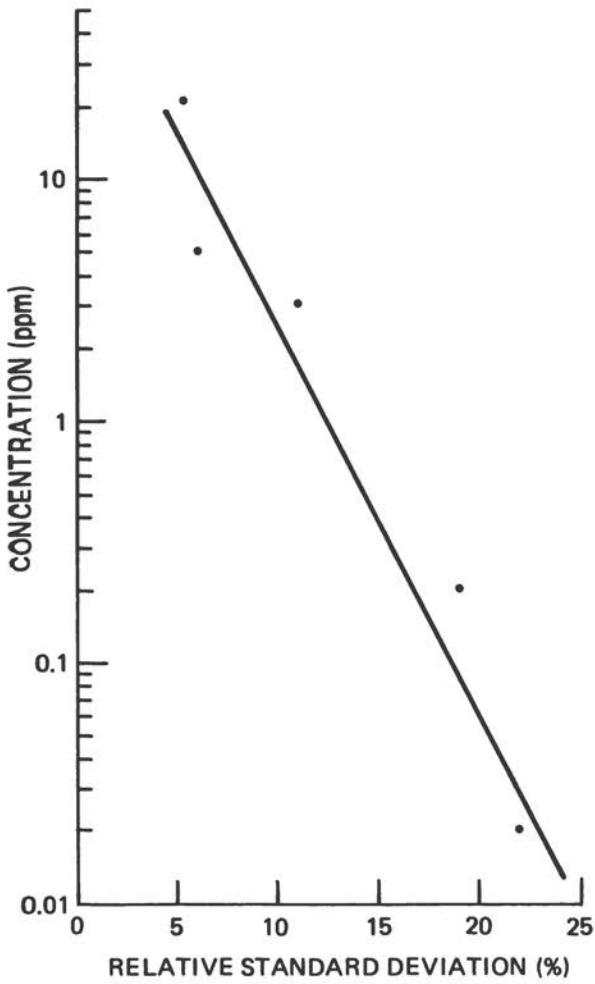
SOURCE: Anon. (1974).

FIGURE A.2   Concentration of mercury vs. standard deviations.

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A BRIEF REVIEW OF FAO/WHO DELIBERATIONS
ON SETTING ACCEPTABLE TOLERANCES
FOR MERCURY RESIDUES IN FOODS, 1963-1976[1]

In a series of joint meetings between 1963 and
1976 the FAO Working Party on Pesticide Residues
and the WHO Expert Committee on Pesticide Residues
have consistently taken the position that no firm
basis has yet been established for determining
what would be a safe level of mercury in food to
protect the health of human beings. Therefore, no
acceptable daily intake (ADI) or tolerance levels
have yet been established despite suggested
recommendations for provisional standards, the
most recent in 1972.

Nevertheless, a great deal of confusion continues with
respect to an ADI and/or food tolerance for total
mercury allegedly set in 1963 when the First Session of
the FAO/WHO Codex Alimentarius Commission convened to
implement the Joint FAO/WHO Food Standards Program.
They were provided with an Extract from the Draft Latin-
American Food Code, which is appended to their report as
Appendix E.2 (FAO/WHO 1963). Article 10 of the Draft
reads, in part, as follows:

The presence of the metals and metalloids
(incidental or residual additives) listed
hereinafter shall be tolerated in foods (with the
exception of drinking water, fish and shellfish),
provided that they are kept within the following
limits: Mercury...Maximum: 0.05 parts per million.

While this was given a "first reading'' by the First
Session of the Codex Alimentarius Commission, it was not
adopted and should not be interpreted to mean that the
commission accepted a level of 0.05 μg Hg/g in
foodstuffs (D.G. Chapman, personal communication, Food
Safety Unit, World Health Organization, Geneva, 1977).
Nevertheless, erroneous reports of its adoption continue
(Goldwater 1974). Later in 1963, on the basis of animal
studies, the FAO/WHO Joint Committee on Pesticide
Residues estimated but did not set or recommend a

maximum acceptable daily intake of 0.05 µg per kg of
body weight phenylmercuric acetate for humans (FAO/WHO
1964).

The 1966 FAO Working Party and WHO Expert Committee on
Pesticide Residues again considered the toxicology of a
number of organomercurial fungicides used in agriculture
and once more concluded that sufficient data were not
available to arrive at an acceptable daily intake (ADI)
or even a temporary tolerance. By way of guidance,
however, they suggested a practical residue limit of
from 0.02 to 0.05 µg Hg/g of food according to local
conditions (FAO/WHO 1967a). This practical residue
limit was also suggested in 1967 but has not been
included in any of the subsequent Joint FAO/WHO reports
on pesticide residues (FAO/WHO 1968a). The 1967 report
also noted that small natural concentrations of mercury
are widespread, but the levels vary from one area to
another. Moreover, where crops or foodstuffs are
treated with organomercurials in accordance with good
agricultural practices, the following maximum levels of
mercury residues were found: rice, 0.3 µg/g
(provisional); apples and tomatoes, 0.1 µg/g; eggs and
meat (except liver and kidney), 0.1 µg/g; potatoes, 0.05
µg/g; and wheat and barley, 0.03 µg/g (FAO/WHO 1968a).

The 1966 FAO/WHO Expert Committee on Pesticide
Residues and Food Additives also attempted to compute
the possible daily intakes of pesticide residues on
foods based on high estimates of food consumption in the
United States (FAO/WHO 1967b, 1967c). To do this, they
assumed that all foodstuffs within the same category
were treated with the pesticide. Thus, the high
consumption value for fish and shellfish was estimated
at 60 g per person per day. The average daily dietary
intakes of mercury were estimated to range from 0.3 to
1.0 µg per kg body weight (21 to 70 µg per 70-kg person
per day, 0.15 to 0.49 mg per 70-kg person per week),
with little accumulation in the tissues. These
committees recommended that every effort be made to
control and reduce mercury contamination of the
environment and consequently the levels in food. They
also affirmed the urgent need for further studies of the
distribution of mercury in foods and beverages and in
human tissues of various ages in different environments.

In 1967 the Joint Meeting of the FAO Working Party and
the WHO Expert Committee on Pesticide Residues again set
no ADI, tolerance or practical residue limits because of
a lack of data. However, they strongly discouraged
agricultural uses that would increase the level of
mercury in food. Nonetheless, the committee contended
that organomercurial seed dressings applied up to the
time of petal fall would not result in residues on
apples in excess of normal background levels (FAO/WHO

1968a, 1968b). This position was continued until 1969 (FAO/WHO 1969, 1970). After 1967 the succeeding FAO/WHO Committees on Pesticide Residues did not directly recommend a practical residue limit but until 1972 did refer to the suggested practical residue limit of 0.02 to 0.05 μg Hg per g originally published in 1967 (FAO/WHO 1968a).

The 1969 FAO/WHO report on pesticide residues in food noted the wide use of organomercurials to protect seed and seed grain and the high toxicity of these compounds to human beings, as well as the fact that unintentional residues were appearing in food crops, animals, and animal products. Therefore, the report stressed the need for safer substitutes and urged governments and other concerned organizations to give this research a high priority (FAO/WHO 1970).

In April 1972, the Joint FAO/WHO Expert Committee on Food Additives (FAO/WHO 1972b, 1972c) finally proposed a provisional tolerable intake (PTI) of 0.3 mg mercury per person per week, of which no more than 0.2 mg should be in the methylated form. For a 70-kg adult, this is equivalent to a daily intake of about 0.6 μg per kg of body weight, with no more than 0.4 μg per kg per day as methylmercury. The 1972 Joint FAO/WHO Expert Committee on Food Additives also stated that the use of alkyl and aryl mercurial fungicides as seed dressings should be discouraged. They noted that aryl and inorganic mercury compounds can be biologically converted into alkylmercury compounds by organisms in nature and recommended research to replace the organomercurial fungicides with compounds that are less likely to poison human beings accidentally. This recommendation was based on the committee's acknowledgement that a number of fatal human poisonings had occurred because seeds treated with alkylmercury compounds had accidentally or improperly been diverted from sowing to human consumption. Nevertheless, the 1972 committee again confirmed the opinion of the 1967 committee (FAO/WHO 1968a) that the uptake of mercury into crops from seed dressing was insignificant as a potential source of food contamination. They recommended that the uses of mercury compounds in agriculture be reviewed by the FAO Working Party of Experts on Pesticide Residues and the WHO Expert Group on Pesticide Residues (FAO/WHO 1973).

In 1973 the FAO/WHO Codex Alimentarius Commission (1974a) noted that no limits were set for mercury in food, nor was a Codex referee method of analysis established. However, for human beings a provisional tolerable weekly intake (PTWI) was set at 5 μg per kg body weight for total mercury intake and 3.3 μg per kg body weight for methylmercury expressed as mercury.

While all mention of mercury fungicides has been deleted from the FAO/WHO Joint Report of Pesticide Residues in Food since 1973 (FAO/WHO 1974c, 1975, 1976), special publications have continued to deal with the subject. The FAO/WHO (1974b) Joint Report on The Use of Mercury and Alternative Compounds as Seed Dressings contended that until effective and broad spectrum alternative fungicides are available, the use of alkoxyalkylmercury and arylmercury compounds will have to continue under safeguards decided upon by individual countries. In addition, in light of the serious poisoning outbreaks in various countries, the committee recommended that alkylmercurials be limited to the treatment of nuclear stocks in the first few generations of seed multiplication and that the handling of the treated seed be strickly controlled. Because of the known high toxicity of alkylmercury compounds, it was also recommended that their use never be permitted for the treatment of seed to be exported for the production of food. The following year the World Health Organization sponsored a conference on human intoxication resulting from alkylmercury-treated seeds and dealt mainly with the Iraqi methylmercury poisoning outbreak of 1971-1972 (WHO 1976a). As new data were added about this condition, the resolve was reinforced to prevent similar outbreaks at all cost in the future. Finally, in 1976 the World Health Organization published Environmental Health Criteria 1, Mercury (WHO 1976b) assessing not only the existing information on the relationship between mercury exposure and man's health but also providing guidelines for setting exposure limits consistent with health protection.

NOTE

1 For definitions of terms accepted by the FAO/WHO Joint Meetings on Pesticide Residues in Food see:

- FAO/WHO (1976) for definitions of pesticide, pesticide residue, good agricultural practice in the use of pesticides, acceptable daily intake, temporary acceptable daily intake, conditional acceptable daily intake, potential daily intake, maximum residue limit, temporary maximum residue limit, extraneous residue limit, further work required;
- FAO/WHO (1970) for definitions of practical residue limit, tolerance, and temporary tolerance; and
- FAO/WHO (1972b) for definition of provisional tolerable weekly intake.

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CURRENT STATUS OF STATE SPORT AND COMMERCIAL
FISHERIES WITH RESPECT TO MERCURY POLLUTION
JUNE 1977

The following states have reported no closures of
sport and/or commercial fisheries and have not issued
any restrictions or health warnings regarding the
consequences of eating mercury-contaminated fish and
other seafood:

Alaska	Indiana	New Jersey
Arizona	Iowa	North Dakota
Arkansas	Kansas	Oklahoma
Colorado	Maine	Puerto Rico
Connecticut	Maryland	Rhode Island
Delaware	Missouri	Utah
Florida	Montana	Washington
Guam	Nebraska	Wyoming
Hawaii	Nevada	

The listing below describes the current status of the
states that have, since 1970, closed sport or commercial
fisheries and/or issued health warnings about the
consequences of eating fish or other seafood
contaminated with mercury. The status of mercury in the
United States as a whole is illustrated in the
attachment at the end of this Appendix.

STATE	CURRENT STATUS
Alabama*	The 1970 restriction on commercial fishing in the Tombigbee, Tensaw, and Mobile rivers and their respective tributaries as well as the waters of upper Mobile Bay was lifted on July 7, 1972. However, all of the Pickwick Reservoir in Alabama still remained closed. The Pickwick impoundment closed to commercial fisheries on July 7, 1970,

STATE	CURRENT STATUS
	was reopened on May 21, 1975. This closure was not mentioned in the 1970 FWQA Survey.[1] Alabama has not issued a health warning with respect to the dangers of eating mercury-contaminated fish.
California**	The warning to eat only one meal per week of striped bass and catfish from the Sacramento-San Joaquin Delta and San Francisco Bay area were issued by the State Department of Health and are still in effect. In addition, in 1972, warnings were issued by the Santa Clara County Park and Recreation District that fish (largemouth bass, sunfish, catfish, and rainbow trout) taken from Calero, Almaden, and Guadalupe reservoirs may contain high levels of mercury and should not be eaten.
Georgia*	In 1970 the Savannah River and New Savannah Dam on Highway 17 as well as the Brunswick Estuary were closed to sport fishing. The Brunswick Estuary was also closed to commercial fishing. All restrictions and closures were removed from the Brunswick Estuary on October 19, 1970, and the Savannah River in September, 1972.
Idaho**	No state restrictions or fishery closure. Conditional warnings (no person should eat more than 1/2 lb of fish per week; and pregnant women, infants, and children should not eat any fish taken from American Falls Reservoir) were issued by the State Health Department for selected species of fish in the American Falls Reservoir (January, 1971 and 1972), Hells Canyon Dam, and other reservoirs on the Snake River (January 1971).
Illinois**	The 1970 policy of no state sport or commercial fishery closures and no health warning advice to fishermen or the public about the consequences of eating mercury-contaminated fish was revised because certain species of fish taken from three reservoir lakes (Rend Lake, Cedar Lake, and Lake Shelbyville) exceeded the FDA mercury action

[1]Harlan, J.R. (1971) Mercury Pollution Survey. Sport Fishing Institute Bulletin No. 221. Washington, D.C.: 719 13 Street, N.W.

STATE	CURRENT STATUS
	level of 0.5 μg/g. As a result, the public was warned to limit consumption to no more than 1/2 lb per week of largemouth bass, shorthead redhorse, black buffalo, bullhead, and yellow bullhead from these lakes. No mercury contamination problems have been identified that affect the commercial fisheries.
Kentucky**	The 1970 health warning and restrictions issued for fish taken from the Tennessee River at Calvert, Kentucky, have been relaxed. At present, a warning pertains only to local residents who eat 20 meals per week of fish from the watercourse over a long period of time (years). Nonresidents are not affected by this warning.
Louisiana*	In 1970 Louisiana issued a health warning for fish taken from the Calcasieu River and stopped the interstate shipment of these fish. All state restrictions were removed in 1975 because the mercury concentrations in fish were within the FDA tolerance level of 0.5 μg/g.
Massachusetts**	In 1970, minor fishery closures and a health warning were issued for three specific areas and fisheries. As a result of mercury contaminations above the FDA action level of 0.5 μg/g, two shellfish areas were closed in December 1970. The areas were Sippican Harbor in Marion and Quisset Harbor in Falmouth. In 1975, the total closures were modified and portions of the harbors were reopened to shellfishing because mercury levels had declined. Neither area was heavily industrialized, and the source of mercury was marinas that had utilized mercury-based paint in boat yard work. Also in 1970, a health warning was issued for persons who were engaged in recreational finfishing in the Taunton River. Fish could be taken, but people were advised not to consume those from the northern boundary of the town of Fall River north to the northern boundary of the town of Dighton. This warning was a result of an industrial discharge, and the advisory is still in effect. The industrial discharge has since been terminated.

180

STATE	CURRENT STATUS
Michigan***	On April 15, 1970, sport fishing was banned and health warnings posted on the St. Clair River and Lake St. Clair. The commercial fishing for walleye in Lake Erie was banned on April 29, 1970. On May 20, 1970, the sport fishing restrictions were reduced to "catch and release" status in the St. Clair and Detroit rivers and Lake St. Clair. In Lake Erie and Lake Huron (south of Port Sanilac) sport fishermen could keep all fish except walleye, white bass, and freshwater drum while commercial fishermen could keep all species except walleye. During the summer of 1970 the courts set aside the sport fishing ban and allowed fishermen to keep their catches. This ruling is still in effect as is the health warning originally imposed in the spring of 1970. Subsequently, walleye were given sport status, which protected them from commercial exploitation.
Minnesota**	There have been no closures of sport or commercial fisheries in the state. On December 11, 1970, the Department of Health advised that anglers restrict intake of fish from certain water to once a week due to high mercury levels. Subsequent analyses of fish for mercury resulted in several modifications of the warning between 1970 and 1976. The following four watercourses were found to contain some fish exceeding the FDA action level of 0.5 μg/g: (1) the St. Louis River below Coloquet, (2) the Upper Mississippi River between Grand Rapids and Brainerd, (3) the Red River along the Dakota border, and (4) Crane Lake near the Canadian border. The latest modification was on May 14, 1976, when it was advised that fish from Crane Lake be eaten no more than once a week.
Mississippi**	On August 1, 1975, the Mississippi portions of Pickwick Lake were reopened to commercial fishing. In addition, all fish containing more than 0.5 μg/g mercury would be subjected to seizure by the Food and Drug Administration. A warning was also issued by the Mississippi State Board of Health that eating fish from Pickwick Lake by pregnant women should be kept to a minimum and that all other persons should restrict their

STATE	CURRENT STATUS

normal intake of fish from this lake to not
more than two meals per week.

New Hampshire*   No state restrictions. The 1970
danger warnings for pickerel, yellow perch,
and smallmouth bass from the Merrimack and
Connecticut rivers have been removed because
public health officials contend that the
current creel limits preclude anyone from
eating sufficient quantities of fish to be
harmful to health.

New Mexico**   Sport fishery health cautions for the
Navajo and Ute Lakes were issued by the
Health and Social Services Department (HSSD)
in 1970 and are still in effect. The public
was advised not to eat more than 2 lb per
week of any species of fish taken from Navajo
Lake. If walleye and largemouth bass larger
than 1.5 lb were taken from Ute Lake, the
recommended consumption was to be limited to
less than 1 lb per week per adult person, and
the recommended consumption of catfish larger
than 5 lb was limited to 2 lb per week per
person. Warnings against eating large
amounts of fish taken from Summer, Elephant
Butte, and Caballo lakes were also issued.
The HSSD stressed that it was safe to eat
fish from any New Mexico lake provided that
the recommended consumption limits were
observed. No action was taken by the State
Game and Fish Department pursuant to the HSSD
warnings. Very little public concern has
been evident since 1971.

New York**   With the exception of three bodies of
water, officials have proclaimed that it is
safe to eat fish once a week without fear of
mercury contamination. Onondaga Lake is
closed to fishing. People are advised not to
eat lake trout from Lake George or
muskellunge from the St. Lawrence River,
although all other species of fish from these
waters may be eaten once a week. Pregnant
women and infants are advised not to eat any
freshwater fish. There are no commercial
fishing restrictions.

North Carolina** For the inland fisheries, the 1970
general danger warnings to fishermen are
still in effect. No closures or health
warnings have been issued for the marine
fisheries. However, the FDA ban on swordfish

STATE	CURRENT STATUS

ended a small fishery for this species on the northern coast of this state.

Ohio*   In 1970 the Lake Erie commercial fishery was closed for all fish except perch. An embargo was placed on white bass and a sport fishery health warning announced. Since then the 1970 restrictions were ruled unconstitutional by the Ohio Supreme Court because "The Division of Wildlife is not and was not responsible for consumer protection." No state restrictions or health warnings are presently in effect.

Oregon*   The 1970 policy of no state or commercial fishery closures continues. However, in 1970 health warnings were issued for rainbow trout, black crappie, suckers, and largemouth bass taken from the Antelope and Owyhee reservoirs and parts of the Wallametta River. A curtailed intake of any fish taken from these waters was recommended particularly for infants and pregnant women. In 1975 a similar warning was issued for striped bass.

Pennsylvania*   In 1970 the Department of Environmental Resources issued an advisory that large predator game fish such as walleye, drum, smallmouth bass, and white bass may exceed the FDA action level of 0.5 μg/g for mercury, and therefore some restriction on the human consumption of these fishes may be advisable. At present, Pennsylvania officials do not consider mercury pollution a serious problem in their state and have not placed any official restrictions on catching game fish. Nor have any health warnings been issued with respect to eating the species.

South Carolina**   In 1970 the sport and commercial fisheries were closed on the Savannah River from Augusta, Georgia, to the coast. These restrictions were removed in 1972. In 1972 an advisory was issued that recommended limiting the consumption of fish taken from Lake Jocassee to 1.5 lb of dressed fish per week and no intake by pregnant women. The elevated levels of mercury in Lake Jocassee fish were the result of natural conditions. They included the slightly higher soil mercury levels in the lake area and, more

| STATE	CURRENT STATUS
	significantly, the oligotrophic condition of the lake. The advisory is currently in effect, and the mercury levels are being monitored.
South Dakota**	In 1970 the state reported no closures or advice to fishermen about health hazards associated with eating fish taken from South Dakota waters. Since then, only the Cheyenne Arm of Oahe Reservoir has been posted by the state's health officer. In June 1973, commercial and sport fishermen were warned not to eat more than 1 1/2 lb of fish from this water per week. This health warning is still in effect.
Tennessee***	In September 1970, the the Tennessee River and Pickwick Lake commercial fisheries were closed and a health warning along with a catch and release policy instituted for their sport fisheries. Both the commercial and sport fisheries restrictions were removed from Pickwick Lake and the Tennessee River in August 1971. The catch and release restrictions and health warning imposed on sport fishing in the North Fork Holston River in September 1970 are still in effect and commercial fishing is not allowed.
Texas***	In 1970 approximately 19,900 acres of Lavaca Bay was closed to commercial oyster harvest. As of September 1, 1971, the number of acres of Lavaca Bay that are closed to the commercial harvest of shellfish has been reduced from 19,900 to 11,000 acres. Additionally, the acreage closed to the harvest of oysters was not closed in its entirety because of mercury pollution. Prior to its reclassification in 1970, Lavaca Bay had approximately 8500 acres which were closed because of sanitary and bacteriological reasons.
Vermont***	In 1970 Lake Champlain and its tributaries were closed to the commercial harvest of walleye. In addition, an embargo on commercial sales of walleye from Lake Champlain, its tributaries, and Lake Memphremagog is still in effect. On April 25, 1973, the sport fishery danger warnings imposed in 1970 were continued for the consumption of walleye from Lakes Champlain, its tributaries, and Lake Memphremagog.

STATE	CURRENT STATUS
Virginia***	In 1970 the sport fishery on the North Fork of the Holston River below Saltville was closed by the Virginia Department of Health. In 1975 this restriction was relaxed to permit fishing under a catch and release regulation. The health warning issued in 1970 and again in 1975 concerning the danger of eating fish taken from these waters is still in effect. On June 6, 1977, the Virginia Department of Health closed the sport fishery on the South River, the south fork of the Shenandoah River. Sport fishing is allowed under a "catch and release" policy, but citizens are warned that fish from these waters are unfit for human consumption.
West Virginia*	Sport and commercial fisheries in West Virginia are presently not restricted due to mercury pollution. The Ohio River commercial fishery which was closed on August 20, 1970, was reopened on July 1, 1973. Currently, West Virginia has no health warnings about the dangers of eating mercury-contaminated fish.
Wisconsin*	In 1970 a catch and release policy was recommended for the Wisconsin River along with a health warning not to consume more than one meal per week of fish taken from this river. At present there are no state restrictions because mercury levels in the Wisconsin River system have recently dropped below the FDA action level of 0.5 $\mu g/g$. Contracts for commercial fishing are now being granted for the Wisconsin River and its impoundments, and warnings on fish consumption limits are no longer being issued.

* States that have rescinded closures of sport and/or commercial fisheries or health warnings issued since 1970.

** States where health warnings are in effect about the consequences of eating mercury-contaminated fish or other seafood from selected watercourses in the state.

*** States where sport or commercial fisheries are currently closed and health warnings are in effect.

STATUS OF MERCURY IN THE UNITED STATES: 1977
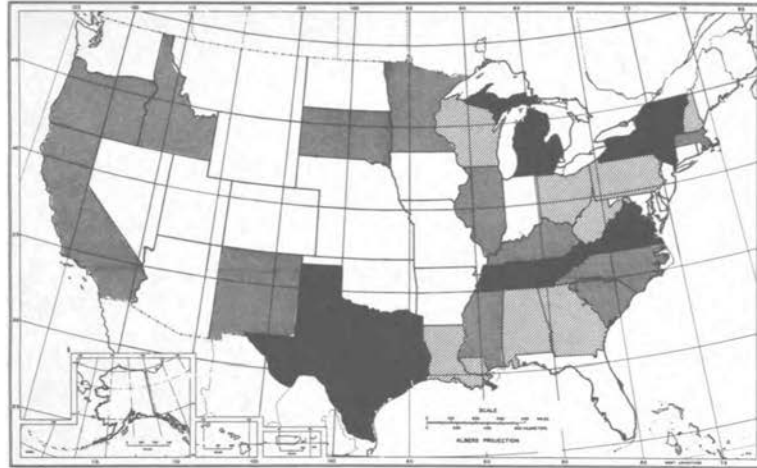
STATES WHERE CLOSURES OF SPORT OR
COMMERCIAL FISHERIES AND HEALTH
WARNINGS ARE NOW IN EFFECT.

STATES WITH CURRENT HEALTH WARNINGS
ABOUT THE CONSEQUENCES OF EATING
MERCURY-CONTAMINATED FISH OR OTHER
SEAFOOD FROM SELECTED WATERCOURSES
IN THE STATE.

STATES THAT HAVE REOPENED SPORT OR
COMMERCIAL FISHERIES OR RESCINDED
HEALTH WARNINGS ISSUED SINCE 1970.

STATES THAT REPORT NO CLOSURES OF
SPORT OR COMMERCIAL FISHERIES, AND
NO HEALTH WARNINGS ABOUT MERCURY
POLLUTION SINCE 1970.