

T A S K F O R C E O N

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S E T T I N G

FINAL REPORT ON  
MUNICIPAL SOLID WASTE  
I N C I N E R A T I O N

VOLUME II  
E N V I R O N M E N T A L  
A N D H E A L T H I S S U E S

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**TASK FORCE  
ON  
MERCURY EMISSIONS STANDARD SETTING**

**FINAL REPORT  
ON  
MUNICIPAL SOLID WASTE INCINERATION**

**Volume I**

**Executive Summary and Recommendations with a  
list of Task Force Members**

**Volume II**

**Environmental and Health Issues**

**Volume III**

**Technical and Regulatory Issues**

**Addendum**

**Peer Review Comments**

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**VOLUME II**  
**ENVIRONMENTAL and HEALTH**  
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## TERMS AND ABBREVIATIONS

A list of some terms used throughout this report

ADI	Acceptable Daily Intake
BD	Below Detection Limit
Hg	Mercury
LOEL	Lowest Observed Effect Level
LOAEL	Lowest Observed Adverse Effect Level
Me-Hg	Methylmercury
NOEL	No Observed Effect Level
RfC	Reference Concentration
RfD	Reference Dose
s.d.	standard deviation
kg	kilogram
g	gram
mg	milligram
$\mu$ g or ug	microgram
ng	nanogram
pg	picogram
l or L	liter
ml	milliliter
m	meter
m <sup>2</sup>	square meter
m <sup>3</sup>	cubic meter
cm	centimeter
pH	negative log of the hydrogen ion concentration
pCl	negative log of the chloride ion concentration
pM	picomolar

**PART 1**

**INTRODUCTION AND GENERAL CONSIDERATIONS**

## I. Hazard Identification

### A. Physical and Chemical Information

Mercury is ubiquitous in nature, being found in three valence states ( $\text{Hg}^0$ ,  $\text{Hg}^+$ , and  $\text{Hg}^{++}$ ) at low levels in the atmosphere, soil, and in natural waters. The most important mercury containing mineral in the earth's crust is cinnabar ( $\text{HgS}$ ), a mercury sulfide, although there are at least 30 known mercury-containing minerals on earth (Goldwater & Stopford 1977). Cinnabar volatilizes without melting at ambient temperatures and thus represents an ongoing natural emission source of mercury.

Mercury enters the geochemical cycle by simple transport in the form of metallic mercury vapors, or it may be chemically transformed into more soluble mercury salts. The atmosphere plays an important role in the mobilization of mercury, where its residence time depends upon its form. Elemental mercury ( $\text{Hg}^0$ ) has a residence time of two months to two years, while inorganic mercuric ion ( $\text{Hg}^{++}$ ) has a shorter residence time of 5-14 days due to its solubility in water and hence ability to wash out via rain and snow events. Mercurous mercury ( $\text{Hg}^+$ ) is unstable and is quickly converted to  $\text{Hg}^0$  or  $\text{Hg}^{++}$ .

There are operational definitions used among scientists to differentiate analytically among the various mercury species found in the environment. Although researchers seldom identify the actual mercury species measured, they have developed a system that adequately identifies the type of mercury present in a given sample. These are listed in Figure I-1. (For more details on these operational definitions, see Gill & Bruland (1990), Nriagu (1979), Windom & Smith (1991), Schroeder & Jackson (1985) and Lindqvist et al., (1991)).

#### 1. Volatile Forms of Mercury:

$\text{Hg}^0$ ;  $(\text{CH}_3)_2\text{Hg}$ ;  $\text{HgCl}_2$ -gaseous;  $\text{CH}_3\text{HgCl}$ -gaseous.

"Metallic" or "elemental" mercury ( $\text{Hg}^0$ ) is the only metal that exists as a liquid at room temperature. The solubility of elemental mercury in water is low, 20 ug/L (Fribert & Vostal 1972) or 56 ug/L (Wollast et al. 1975). However, contact with oxygen-containing water may increase solubility. Other factors which influence its solubility include pH, presence of complexing anions, oxygen content and the presence of organometallic complexes. Elemental mercury is lipid-soluble, has no charge, and can penetrate membranes in living systems. Depending upon conditions, it can be quickly oxidized in the environment to form mercuric salts (e.g.  $\text{HgCl}_2$ ).

"Dimethyl" mercury ( $[\text{CH}_3]_2\text{Hg}$ ), an organomercurial, is lipophilic, non-polar and almost insoluble in water. It is volatile and can occur in the atmosphere through volatilization from soil and water. This form of mercury is reported to be rarely found in the environment.

Other forms of mercury can be volatile in the environment (e.g. mercuric chloride ( $\text{HgCl}_2$ ) and methylmercuric chloride ( $\text{CH}_3\text{HgCl}$ )), though these may be subject to washout depending upon their solubility in water and are seldom found outside the perimeter of a point source; that is, these species prefer to solubilize in water but can be detected as gaseous mercury in air close to a point source. Operationally, the volatile species include only the elemental and dimethyl forms.

## 2. Nonvolatile Forms of Mercury:

Reactive:  $\text{Hg}^{++}$ ;  $\text{HgX}_2$ ;  $\text{HgX}_3$ ;  $\text{HgX}_4^{-2}$  ( $\text{X} = \text{OH}^-$ ,  $\text{Cl}^-$ ,  $\text{Br}^-$ );  $\text{Hg}^0$  on aerosol particles; Hg-organic acids.

Nonreactive:  $\text{CH}_3\text{Hg}^+$ ;  $\text{CH}_3\text{HgCl}$ ;  $\text{CH}_3\text{HgOH}$  and other organomercuric compounds;  $\text{Hg}(\text{CN})_2$ ;  $\text{HgS}$ ;  $\text{Hg}^{++}$  bound to sulfur in humic matter.

"Reactive" or acid-labile mercury is defined as an acid-soluble mercury compound; that is, species that are readily reducible to  $\text{Hg}^0$  in acidified, unfiltered water. They include: dissolved inorganic mercury species like the mercury halides ( $\text{HgX}_2$ , where  $\text{X} = \text{chlorine}$ ,  $\text{bromine}$  or  $\text{fluorine}$ ), e.g. mercuric chloride; mercuric hydroxides ( $\text{Hg}[\text{OH}]_2$ ); mercury complexes with organic acids ( $\text{Hg}^{++}$ -humic or fulvic acids); and mercury easily leached from particulates.

"Nonreactive" mercury is defined as a mercury compound which can be reduced to the elemental form ( $\text{Hg}^0$ ) by sodium borohydride. These include mercuric sulfide ( $\text{HgS}$ ), sulphur-containing organomercury complexes, and compounds containing C-Hg bonds (e.g. monomethylmercury). Alkyl mercury forms are among the most toxic, and monomethylmercury is included in this class. Monomethylmercury is referred to as Me-Hg throughout this report.

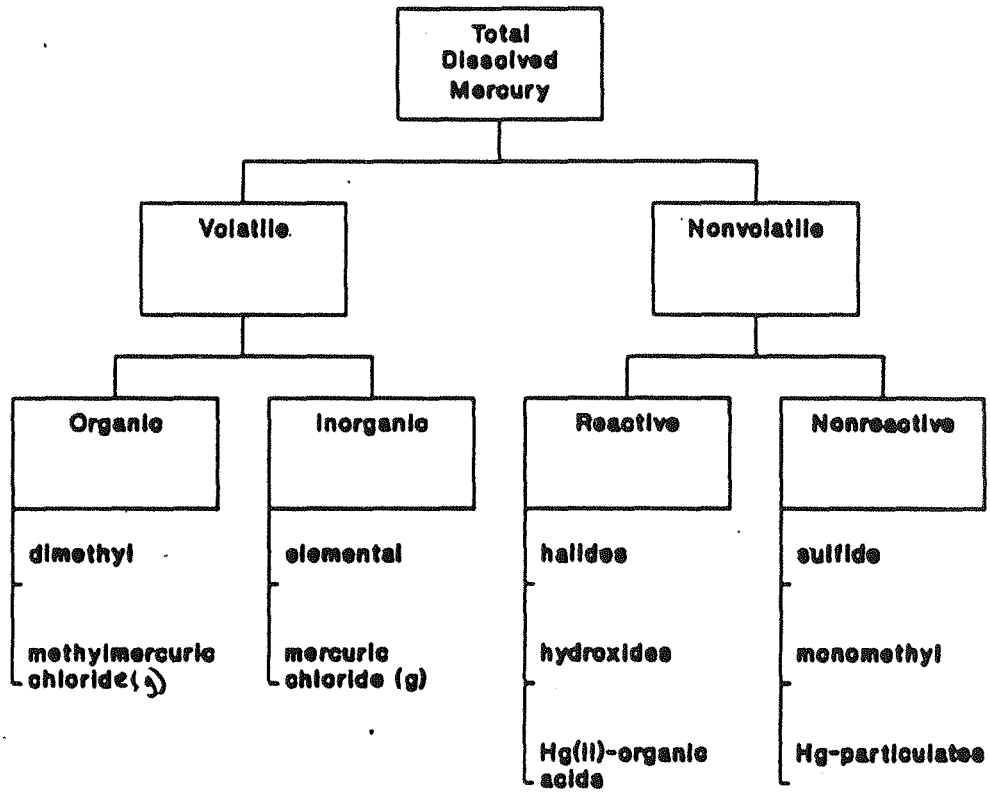
Mercury may be methylated biologically or abiotically under certain environmental conditions, but not all mercurials are converted to the methyl forms. In order for methylation to occur, there must be sufficient inorganic mercuric ions present and a methyl donor. For biologically-mediated reactions, there must be a bacterial population capable of converting mercury to methylmercury, a substrate on which the bacteria can live, acidic pH, and favorable redox conditions, suspended solid load, and sedimentation rate. High levels of methylmercury can be found in fish (where it is bioaccumulated) and in lake and deep river sediments.

## B. Reasons for Concern

Mercury in all of its chemical forms has long been known to be toxic to humans, with a specific and unique spectrum of effects on the nervous system. Recent reports in the scientific literature have provided evidence that global atmospheric mercury levels continue to rise due to anthropogenic activity. In remote North American lakes, mercury levels have been increasing since the onset of industrialization and particularly since the 1950's. In Sweden, the mercury content in lake fish has increased by about five times the historical natural level. By the end of the 1980's the Swedish public was advised not to consume fish from over 10,000 Swedish lakes, and the distribution and sale of such fish was made illegal. During the last four decades, there have been three major epidemics of methylmercury poisoning: Niigata and Minimata, Japan; and Iraq. In each of these incidents large populations were affected with severe as well as more subtle health effects. Several scientific studies (discussed in detail in Chapter III) strongly suggest that consumption by pregnant women of moderately high amounts of mercury can result in subtle developmental abnormalities in their children. Mercury is persistent in the environment in general and in the human food chain in particular. Several types of industrial and other activities, such as fossil fuel combustion, incineration of municipal solid waste, sludge incinerators, and land application of sludge, emit significant amounts of mercury to the ambient air. It is thus clearly in the public interest to place limits on future mercury emissions which are consistent with human health objectives and with the prevention of future degradation of the environment from such sources.

Chapter II of this report summarizes measured mercury values reported in the literature. It includes data for mercury in ambient air, precipitation, surface water, soils and sediments, and biota. Limited mercury deposition data are also reported. Wherever possible, comparisons are made between New Jersey data and global data in that Chapter.

Figure I-1: Mercury Species



## II. Measured Mercury Values in the Literature

### A. Mercury in Ambient Air

#### 1. Global Data

Researchers who have investigated mercury levels in ambient air concede that the volatile forms  $\text{Hg}^0$  and  $\text{Hg}^{++}$  are the most common, comprising 80-95% of the total mercury in the atmosphere; the remainder is associated with particles. Lindqvist (1985) specifies that while  $\geq 90\%$  of mercury in the atmosphere consists of elemental mercury ( $\text{Hg}^0$ ) whose residence time in the environment may range from 2 months to 2 years, it is readily oxidized in the atmosphere to more soluble forms (e.g.  $\text{HgCl}_2$ ), with residence time ranging from a few days to a few weeks and which are subject to washout via rain, and snow, and to dry deposition.

Methylmercury is rapidly converted to  $\text{Hg}^0$  or inorganic  $\text{Hg}^{++}$  in the atmosphere so significant concentrations are not expected to occur. Some types of organic mercury (e.g. dimethylmercury) may volatilize from soil or water and thus be detected in air near the earth's surface, but their levels are not considered significant.

Table A-1 of Appendix A lists the ambient air concentrations of total mercury detected globally in urban, suburban and rural/remote areas. Background mercury levels in the environment are generally thought to be 1-9  $\text{ng}/\text{m}^3$ . In pristine environments (i.e., remote ocean air) mercury levels are reported to be <1 to 3  $\text{ng}/\text{m}^3$ .

#### 2. New Jersey Data

In New Jersey, some air measurements have been taken near the municipal solid waste incinerator in Warren County (Greenberg et al. 1991). Background levels at this site (median 2.5  $\text{ng}/\text{m}^3$ ) fall within global background levels (<1 to 9  $\text{ng}/\text{m}^3$ ). Air measurements taken in areas thought to be affected by the incinerator also fell within these background levels (median 2.27  $\text{ng}/\text{m}^3$ ) and are the same order of magnitude as the modeled annual average concentrations for MSW incinerators as summarized in Table D-3 in Appendix D.

#### 3. Contribution of Anthropogenic Sources to Ambient Air Concentrations

A number of scientists believe that anthropogenic processes have contributed to increased levels of mercury in the environment since pre-industrial times (Slemr & Langer 1992; Nriagu & Pacyne 1988; Lindqvist & Rodhe 1985; and Lindberg 1987). Atmospheric increases in mercury concentrations have been reported to be  $1.46 \pm 0.17\%$  per year in the Northern Hemisphere and  $1.17 \pm 0.16\%$  per year in the Southern Hemisphere (Slemr & Langer 1992). Increases

since the nineteenth century have been reported based on peat bog cores and lake sediment records. The levels of mercury in the environment are reported to be double what they were in the nineteenth century. Nriagu (1988) and Slemr & Langer (1992) attribute 65% of European anthropogenic mercury emissions to the atmosphere to coal burning and 25% to waste incineration. Travis & Blaylock (in press) contend that in the United States municipal solid waste incinerators contribute 5% of the total anthropogenic mercury emissions. Lindqvist et al. (1991) estimate that natural mercury emissions account for up to 40% of total emissions globally; the remaining 60% are derived from anthropogenic sources.

Lindqvist et al. (1991) cites the work of Levander (1989) to demonstrate the relative importance of different anthropogenic sources of mercury. Cumulative mercury emissions from 1860-1987 in Sweden are shown in Table II-1.

Other less significant anthropogenic sources of mercury to the environment include: manufacture and use of paint; mining and smelting; waste from dentists, hospitals and labs; past use of mercurial pesticides; landfill diffusion; and other industrial processes (e.g. manufacture of electrical components).

Glass et al. (1990) attribute high mercury levels in water and fish of the St. Louis and Superior Bays to multiple sources including a sewage sludge incinerator. Sorensen et al. (1990) report that the mercury measured in water of several Minnesota lakes emanates from atmospheric deposition. Further, they attribute 60% of the mercury in sediments to atmospheric deposition, and the remaining 40% to direct inputs to the watershed. It is difficult to attribute levels of mercury in the environment to specific sources, although it is well established in the literature that anthropogenic activities have resulted in overall increases.

It is apparent that increases in mercury in the atmosphere have occurred and that the source of this mercury is anthropogenic. Mercury from waste incinerators among other anthropogenic sources has been shown to contribute to this increase, especially in Europe. Figure II-1 from Levander (1989 as cited by Lindqvist et al. 1991) shows increases in mercury graphically.

Natural processes may also contribute mercury to the environment. These include: degassing from geological mineral deposits; emissions by volcanic activity; photoreduction of  $Hg^{++}$  in natural waters, including oceans, to  $Hg^0$  or dimethylmercury; biological formation of  $Hg^0$  or dimethylmercury in soils and elsewhere; and marine degassing. Nriagu (1989) presents an inventory of metals emission to the atmosphere from some natural sources. His values for mercury are shown in Table II-2.

Table II-1: Anthropogenic mercury emissions from 1860-1987 in Sweden (Lindqvist et al. 1991)

<u>Source</u>	<u>%Hg<sup>0</sup></u>	<u>%Hg<sup>++</sup>(s)</u>	<u>%Hg<sup>++</sup>(g)</u>	<u>Range, 10<sup>3</sup>Kg</u>
Chlor-alkali	50-90	10-50	--	200-400
Metal production				200-350
Waste handling/ treatment	~20	~60	~20	80-120
Pyrite burning in pulp	100	--	--	80-120
Coal, peat and wood burning	~50	~30	~20	70-100
Other				10-20
<hr/>				
Total				600-1200

Table II-2: Global estimates of natural mercury emissions (Nriagu 1989)

<u>Source</u>	<u>Range, 10<sup>3</sup> Kg/yr</u>	<u>Median, 10<sup>3</sup> Kg/yr</u>
Wind-borne soil particles	0-100	50
Sea-salt spray	0-40	20
Volcanoes	30-2000	1000
Wild forest fires	0-50	20
Continental particulates	0-40	20
Continental volatiles	20-1200	610
Marine	40-1500	770
<hr/>		
Anthropogenic sources in 1983	910-6200	3600
Natural sources in 1983	100-4900	2500

Lindqvist (1985) reports that most mercury from a point source is dispersed regionally or globally while less than 10% is deposited locally. The conclusions of Glass et al. (1991) support this. Lindqvist further points out that mercury measurements in sediments and peat bogs show that deposition of mercury in Scandinavia over the last 100 years has increased by 5-10 times.

Lindqvist states that solid fossil fuels (e.g. coal and peat) contain mercury sulfur compounds totalling 0.1-0.3 mg/kg. The mercury content in municipal waste is 0.5-3.0 mg/kg and has a higher content of elemental or amalgamated mercury than fossil fuels.

Almost all the mercury released by the incineration process itself is in the elemental form, since heating at  $>700^{\circ}\text{C}$  leads to thermal decomposition. According to Lindqvist (1991), much of the elemental mercury in flue gases of incinerators is quickly oxidized to form mercuric chloride ( $\text{HgCl}_2$ ) when the gases cool down on heat exchangers. Lindqvist et al. (1991) estimate that of the mercury releases from waste incineration stacks, 20% is in the elemental gaseous form, 60% is in the mercuric gaseous form, and 20% is in the mercuric solid form. However, depending upon the type of cleaning system installed, the distribution may change to 50% elemental and 50% various divalent forms. Most flue gas cleaning systems remove oxidized forms of mercury. Elemental mercury may be oxidized outside the stack in ambient air where reactions with ozone and other stack-emitted constituents (i.e.,  $\text{Cl}^-$ ,  $\text{NO}_x$ ,  $\text{SO}_x$ ) lead to the formation of water-soluble species (Bergstrom 1986; Brosset & Lord 1991; Hall et al. 1990; Lindqvist 1985; Munthe 1992; Petersen & Iverfeldt 1992; and Schroeder et al. 1991).

#### B. Mercury in Precipitation

Once in the atmosphere, mercury is susceptible to washout due to rainfall or snow events. Glass et al. (1991) estimates that up to 10% of mercury in ambient air is subject to washout in any given precipitation event. Background levels are reported to be 1-60 ng/L. Table A-2 in Appendix A lists the precipitation levels detected by researchers globally.

Although there is a paucity of data regarding wet and dry deposition of mercury in the environment, some exist and are summarized in Chapter VIII.

Background mercury levels in precipitation in pristine areas are reported to be 1-60 ng/L. Precipitation measurements from the Warren County site indicate that the incinerator may influence levels of mercury in rain; precipitation samples taken downwind of the incinerator had higher mercury levels overall than samples taken upwind (see Table A-2 in Appendix A). The maximum level taken from a downwind site was 606 ng/L. However, median levels

for both background sites (13 ng/L) and downwind sites (32 ng/L) in New Jersey fall within global background levels found in pristine areas.

Other mercury determinations measured in New Jersey indicate that precipitation mercury levels in industrial areas are higher than in other areas. Rain levels in Elizabeth ranged from 14 to 59 ng/L (mean 40.7 ng/L), while levels in 3 state parks ranged from 14 to 30 ng/L (mean 22 ng/L).

### C. Mercury in Surface Water

Mercury may reach natural waters via direct deposition onto water surface, storm erosion, soil percolation, and other water induced movements. Surface water generally contains background levels from less than 1 to 11 ng/L.

Particulate matter suspended in water and bottom sediments is likely to contain relatively high levels of mercury, since this element is very strongly attracted to suspended particulates and sediments. Burghigiani & Ristori (1992) measured dissolved versus particulate mercury concentrations in an Italian river. Dissolved mercury was 1.4 - 1.48 ng/L while particulate mercury was 11.8 - 26.9 ng/L.

The solubility of metallic mercury in water is low, 20  $\mu\text{g/L}$  (Fribert & Vostal 1972). However, contact with oxygen-containing water may increase solubility. Other factors which influence its solubility include pH, presence of complexing anions, oxygen content, and the presence of other organometallic complexes.

Well-aerated waters with stable organic ligands speciate mercury among chloro and hydroxy complexes as a function of pH and pCl. Mercury in natural waters also forms very stable complexes with organic ligands.

Mercury may be methylated biologically or abiotically under certain environmental conditions, but not all mercurials are converted to the methyl forms. In order for biological methylation to occur, there must be sufficient mercuric ions present, a biotic population capable of converting mercury to methylmercury, specific pH (acidic environment), and favorable redox (aerobic) conditions, suspended solid load, and sedimentation rate. High levels of methylmercury can be found in fish (where it is bioaccumulated) and in lake and deep river sediments. Abiotic or chemical methylation may occur under anaerobic conditions when pH is low and when sufficient methyl donors are present.

Global mercury values in lakes, rivers and streams, and in lake sediments reported in the literature range from 1 to 75 ng/L, and 2-174 ng/L, respectively. Values are listed in Table A-3 of Appendix A.

In natural surface waters, two general classes of organomercurials may form: 1) the highly toxic organomercurials (mercury covalently bonded to carbon, e.g. methylmercury and dimethylmercury); and 2) divalent mercury complexed to organic ligands. Gill & Bruland (1990) found that the organomercury component in fresh surface waters can comprise a majority of the dissolved mercury (up to 89%). This is in contrast to mercury found in oceans, existing primarily in the dissolved reactive fraction. The particulate fraction can be a significant portion of total mercury present (10-92%). The particulate fraction is lowest in clear water lakes. This indicates that the transport and cycling of suspended matter must markedly influence the biogeochemical cycling of mercury in freshwater systems. Partitioning of divalent mercury complexes and organomercury forms is controlled by net methylation rates: in surface water, this is biological methylation. Very little to no organomercury has been detected in high-pH, high alkali, desert lakes or in high alpine lakes (Robertson et al. 1987).

Methylmercury is seldom detected in the water column; rather, dissolved mercury species are the dominant forms. Mercury complexes with naturally occurring humic and fulvic substances in water may account for some of the "total organic" mercury reported in the literature, according to Robertson et al. (1987).

The effects of lake conditions on methylation rates and bioaccumulation in fish are discussed in Chapter IX. Data for some of these conditions in New Jersey lakes, such as pH and conductivity, are also presented in Appendix D.

#### D. Mercury in Soils and Sediments

Total mercury levels as reported in the literature for soils and sediments are presented in Appendix A. Some researchers have analyzed lake sediments for total mercury. Mean values for uncontaminated, background sediments are reported to be within the range 0.04 to 1.3  $\mu\text{g/g}$ . Surface sediments tend to have higher concentrations of mercury than deeper cores. Some New Jersey lakes have been analyzed for sediment mercury concentrations. In Monmouth County, 9 lakes were sampled with a median of 0.07 and mean of 0.09  $\mu\text{g/g}$  (Monmouth County DOH, 1989). Recent sampling of three NJ lakes from the northern, central and southern part of the state have yielded mean total mercury levels of 0.13-0.33  $\mu\text{g/g}$  (Stevenson et al. unpubl.).

Some limited information on coastal sediments exist in the literature. In Italy, researchers report a mean of 0.23  $\mu\text{g/g}$  for sediment analyses (Giordano et al. 1992), and in Florida, muck wetland sediments have been reported at 0.15 and sandy sediments at 0.04  $\mu\text{g/g}$  (Patrick & Gambrell 1992).

Soil analyses in the literature vary widely. Many reports have examined total mercury in soil cores. Fitzgerald et al. (1992) measured 0.117  $\mu\text{g/g}$  in the organic layer of a soil core; 0.029 at 0.10 cm; and 0.008 at 10-50 cm. Some soil sampling conducted in Atlantic County by NJDEPE showed nondetectable levels of mercury ( $<0.062$  to  $<0.089$   $\mu\text{g/g}$ ).

#### E. Mercury in Biota

Data describing mercury levels in biota, including vegetation, are presented in Appendix A. Discussion in this section is limited to fish. (The ranges reported here are described in more detail in Appendix A). So many factors influence mercury levels in fish that it is difficult to make assessments on "ambient" levels. In addition to individual lake characteristics, certain features about the fish, such as age and species, may affect mercury levels. Further, fish tissue levels do not necessarily correlate with mercury levels in lakes sediments or in the water column.

Trends that can be seen among fish tissue mercury levels are: older, larger fish have higher levels than younger, smaller fish of the same species; and, mercury levels in fish living in acidic lakewater ( $\text{pH} < 6.0$ ) are higher than in fish living in non-acidic lakewater ( $\text{pH} > 6.0$ ).

Pike collected in the US have reported mean mercury concentrations of 0.33  $\mu\text{g/g}$  at less than 5 years old and 0.65  $\mu\text{g/g}$  at over 5 years old. Yellow perch values in the US and Canada reported in the literature ranges from  $<0.01$  to 1.14  $\mu\text{g/g}$ . Large-mouth bass mercury concentrations range from 0.06 to 1.00  $\mu\text{g/g}$  (measurements of large mouth bass in NJ showed a median of 0.3  $\mu\text{g/g}$ ). Mercury levels for commercial, salt water fish used in this report (Chapter IV) were: for canned tuna, 0.1-0.75  $\mu\text{g/g}$  (mean 0.17  $\mu\text{g/g}$ ), and, for non-tuna species, 0.24-0.94  $\mu\text{g/g}$  (median 0.61  $\mu\text{g/g}$ ).

Some fish analyses have been conducted in New Jersey. The work of Ellis et al. (1980) is a comprehensive study of mercury levels in New Jersey's aquatic fauna. Statewide maxima for sunfish, rainbow trout, yellow perch, eastern chain pickerel, smallmouth bass and largemouth bass ranged from 0.15  $\mu\text{g/g}$  in longear sunfish to 1.25  $\mu\text{g/g}$  in spotted sunfish. Other New Jersey fish studies include the work of Sprenger et al. (1987) who detected levels of mercury in yellow perch of 0.07-1.14  $\mu\text{g/g}$  and the work of Stevenson et al. (in progress) who detected levels of mercury of 0.135-0.397  $\mu\text{g/g}$  in largemouth bass fillets, and 0.010-0.036  $\mu\text{g/g}$  in killiefish fillets.

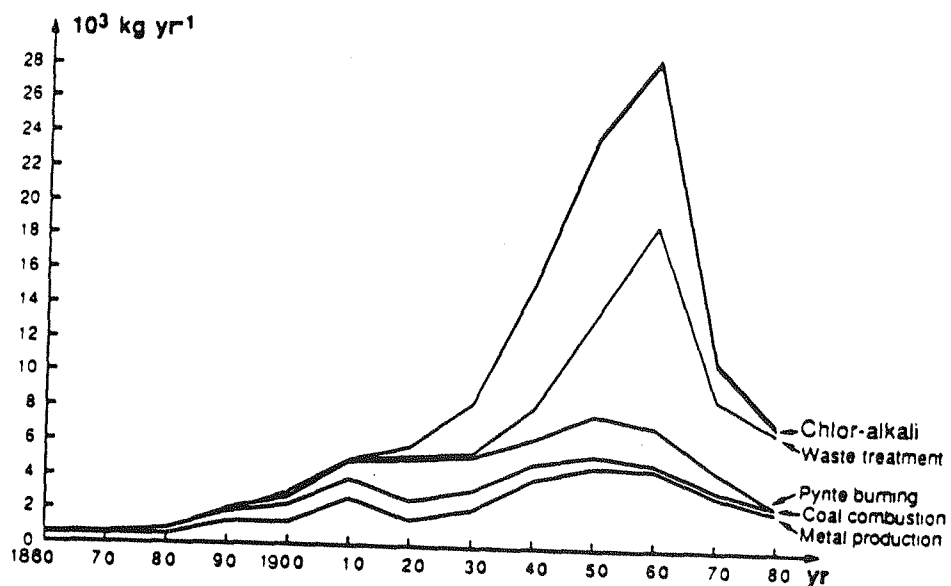
The potential threat to bird populations from existing levels of mercury in the environment has been explored by a number of researchers in New Jersey (USFWS 1991b, 1991c). In these studies, mercury was detected in piping plover eggs up to 1.07  $\mu\text{g/g}$  (USFWS

1991b) and in osprey eggs up to 0.1  $\mu\text{g/g}$  (Griffin & Steidl 1990). Analyses of the carcasses of three peregrin falcons found dead near Barnegat Bay revealed mercury levels ranging from 0.232 to 3.540  $\mu\text{g/g}$  (USFWS 1991c) as well as elevated levels of other contaminants (especially organochlorine pesticides).

#### F. Mercury Deposition Rates

There is scant information about deposition rates in the literature and much debate over the most accurate method of calculating wet and dry deposition rates. Of the deposition rate measurements and calculations conducted by researchers and reported in the literature (see Appendix A), rates for wet deposition are more common than dry deposition. Mean wet deposition rates reported in the literature range from 4.4 to 19.2  $\mu\text{g/m}^2/\text{yr}$ . One study calculated dry deposition rates for mercury as 4.4  $\mu\text{g/m}^2/\text{yr}$ .

Figure II-1: Emissions of Hg to the atmosphere from different activities in Sweden during 1860 to 1980, estimated as average values per decade (Levander, 1989 as cited by Lindqvist et al. 1991)



**PART 2**

**HUMAN HEALTH AND ECOLOGICAL EFFECTS OF MERCURY**

### III. Review of Human Methylmercury Toxicology

#### A. Organic vs. Inorganic Mercury

Except in very rare cases of contamination by industrial mercury compounds, human exposure to organic mercury consists almost entirely of exposure to monomethylmercury (Me-Hg) (WHO 1990). There is general agreement in the scientific literature that health effects from exposure to Me-Hg occur at lower levels of exposure than those related to inorganic Hg exposure. This difference results from differences in the mechanism and site of toxic action as well as from the fact that for equal ingestion exposures, methylmercury (Me-Hg) is absorbed far more efficiently than inorganic Hg (approx. 95% vs. 5% in adults) (WHO 1990). Therefore, as a first-level effort, modeling will concentrate on exposure to Me-Hg.

#### B. Possible Additivity of Toxic Effects from Exposure to Organic and Inorganic Mercury

Organic Hg is metabolized in man (and other mammals) to the mercuric ion ( $Hg^{++}$ ) inside the cell. Although there are significant differences in toxic effects between inorganic and organic forms, some of the neurologic effects of the two forms of Hg are similar. This suggests that exposure to Me-Hg and inorganic forms might be additive in producing at least some of the toxic effects of concern (WHO 1990). However, experimental approaches have failed to demonstrate such an interaction. Magos et al. (1985) found that rats dosed with either methyl-Hg or ethyl-Hg showed neurotoxicity. However, for equal doses, the methyl-Hg produced greater toxicity despite the fact that dosing with the ethyl-Hg produced greater deposition of inorganic Hg. Furthermore the sites of brain damage resulting from either methyl-Hg or ethyl-Hg exposure on histological examination were not the sites of inorganic Hg deposition. At present, there does not appear to be any direct evidence to support an assumption of additivity in the calculation of acceptable exposure to Me-Hg. Future research should address this issue.

#### C. Toxic Endpoints for Methylmercury Exposure

Renal effects in humans following Me-Hg exposure have been described as very rare (WHO 1976). Adverse renal effects in animals are seen at levels above those which are known to result in neurotoxicity (ATSDR 1989). Of the neurotoxic effects which have been observed, the most sensitive indicators of exposure are paraesthesia (numbness, or tingling, burning or itching in the skin, unrelated to dermatologic conditions) and developmental neurologic effects in-utero. Epidemiologic data are available from human exposures to Me-Hg during poisonings in Niigata and Minimata, Japan, and in Iraq (WHO 1990). Although data for causation of

paraesthesia appear to be derived largely from adults, paraesthesia presumably can occur with exposure at any age. The developmental endpoint, however, is specific to exposure of the developing fetus in-utero and results from altered formation of the brain's cellular architecture during neural development.

Comprehensive reviews of the extensive literature on the health effects and pharmacodynamics of Me-Hg are available (WHO 1990; ATSDR 1989; Choi 1989; Burbacher et al 1990). This analysis will not attempt to duplicate these efforts by considering the entire range of the dose-response curve, but will instead focus on the data relating to the lowest levels of dose consistent with public health concern.

#### 1. Current Reference Dose

The current USEPA RfD for Me-Hg is  $3 \times 10^{-4}$  mg/kg/day (0.3  $\mu$ g/kg/day) (USEPA, 1992a). This value is presently under review. The FAO/WHO Permissible Tolerable Weekly Intake (PTWI) is 3.3  $\mu$ g/kg/wk (WHO, 1976), equivalent to  $4.7 \times 10^{-4}$  mg/kg/day (0.47  $\mu$ g/kg/day). These guidelines are derived from consideration of the same data sets - poisoning episodes in Niigata, Japan (consumption of contaminated fish) and Iraq (consumption of bread made from contaminated seed grain). The data sets on which these analyses are based are primarily those of Tsubaki et al. (1978) (Niigata outbreak), Al-Mufti et al. (1976) (Iraqi outbreak), Bakir et al. (1973) (Iraqi outbreak), Clarkson et al. (1976) (Iraqi outbreak) and Nordberg and Strangert (1976) (toxicokinetic modeling of data from Iraq). These are reviewed in detail in the WHO Environmental Health Criteria Documents of 1976 (WHO 1976) and 1990 (WHO 1990). Although these studies generally address a constellation of neurological effects from Me-Hg exposure in adults and older children, paraesthesia is consistently observed in these studies at lower levels of exposure than other neurologic effects and thus corresponds to the adverse effect at the lowest-observed-effect-level (LOEL) dose.

These studies produced estimates of Me-Hg exposure from measurements of Hg in hair and blood and from estimation of actual foodborne intake of Hg. Blood samples were obtained after exposure had ceased and Hg levels in the blood were in decline. Blood levels at the time of exposure thus had to be estimated with some uncertainty. Estimates of the bio-kinetics of Me-Hg distribution among body compartments were used to extrapolate from levels in hair or blood to average daily intake. The estimates from USEPA and WHO differ primarily only in their selection of a single LOEL or threshold dose from among the values predicted by these studies. Calculated values for the LOEL and/or threshold dose range from 2.8-5.8  $\mu$ g/kg/day. Considering the different methodologies and study populations, the potential problems with self-reporting of symptoms and the uncertainties inherent in estimation of blood

levels and food intake, these values demonstrate a remarkable consistency. Following standard risk assessment practice, these values are divided by an uncertainty factor of 10 to estimate the no-observed-effect-level (NOEL) dose from the reported LOEL/threshold dose (USEPA 1992a). This yields a lowest value for the RfD of approximately  $0.3 \mu\text{g}/\text{kg}/\text{day}$  which is the current USEPA RfD value. For a generic 70 kg adult (USEPA 1992a), this is equivalent to a daily intake of  $21 \mu\text{g}/\text{day}$  and for a 62 kg woman (average of mean weights for ages 18-25 and 25-35) (USEPA 1989b), this dose is equivalent to an intake of  $19 \mu\text{g}/\text{day}$ . Although Clarkson et al. (1976) qualitatively documented severe neurologic symptoms in some children exposed in-utero, none of these studies included such exposures in the assessment of dose and response.

## 2. Review of Human Studies of In-Utero Exposure

Developmental effects such as cerebral palsy, deafness and mental retardation were noted in the Minimata and Iraq outbreaks (WHO 1990). However, no attempt was made to measure developmental effects of less severity until the study in Iraq by Marsh et al. of 81 gestationally exposed maternal and child pairs when the children had reached a mean age of 2.5 years (Marsh et al. 1987). Me-Hg is deposited and retained in hair follicles throughout follicular growth in direct proportion to intake. This, in effect, creates a record of Me-Hg intake which can be accurately measured for the entire period corresponding to pregnancy (WHO 1990; Marsh et al. 1987; Phelps et al. 1980). The geometric mean maternal hair Hg concentration (maximum of individual pregnancy period) was  $20 \mu\text{g}/\text{g}$  hair (41% with levels  $<10 \mu\text{g}/\text{g}$ ). Marsh et al. found an increased frequency of central nervous system (CNS) abnormalities determined by clinical examination of cranial nerve signs, speech, involuntary movement, limb tone strength, deep tendon reflexes, plantar responses, coordination dexterity, primitive reflexes, sensation, posture, and the ability to sit, stand and run. The neurological findings were converted into an overall score for each child. Based on this score, an increased frequency of overall effects relative to the expected normal score of 3 was seen in both sexes. Boys however, had significantly worse scores than girls. An increased frequency of retarded onset of walking (greater than 18 months) as reported by mothers was also observed. These findings indicate subtle, but significant, developmental effects.

The overall data presented by Marsh et al. for neurological findings and onset of walking for each child are plotted relative to maternal hair concentration of Hg in Figures III-1 and III-2. For each data set, the maximum normal neurologic score considered normal by Marsh et al. and the maximum age for normal onset of walking (18 months) are noted. If the absolute occurrence of abnormal responses is considered for each endpoint, a dose-response relationship can be seen in both cases and the occurrence of abnormal responses appears to increase at a hair Hg level slightly greater than 10 ppm ( $10 \mu\text{g}/\text{g}$ ). However, the interpretation of the

dose-response relationship is complicated by the fact that for each endpoint, a substantial fraction of the children had normal responses even at the highest levels of maternal exposure. This suggests that there is a large variation in susceptibility to the developmental effects of Me-Hg in the population. Thus, as Magos (1992) has pointed out, if the occurrence of abnormal responses is evaluated as a proportion of total responses, a clear increase in abnormal responses is not reached until approximately 100  $\mu\text{g/g}$ . This is shown in Figure III-3 and III-4. The difference between the absolute and the proportional rates of response suggests the possibility of a sub-population with increased susceptibility to lower doses of Me-Hg.

Of particular importance in evaluating the significance of in-utero exposure is the observation that mothers of children born with clear developmental effects often reported no symptoms or only mild and transitory symptoms of paraesthesia (Marsh et al. 1987). A *chi*-squared analysis of the complete data set of Marsh et al. indicates no statistical association between maternal paraesthesia and the CNS effects detected in the children. There is a strong statistical association between maternal paraesthesia and delayed onset of walking. Nonetheless, only 53% of the cases of delayed onset of walking were paired with maternal paraesthesia. This analysis suggests that guidelines protective against maternal paraesthesia may not provide adequate protection against developmental effects from *in-utero* exposures.

In an analysis of Marsh et al., and recalculation of maternal exposure by single strand hair analysis of Hg, Cox et al. (1989) attempted to derive a quantitative dose-response relationship between Me-Hg exposure and developmental effects. The relationship between the maximum hair Hg levels during pregnancy of mothers of affected and unaffected children and the frequency of CNS effects or delayed onset of walking was examined by three statistical models - non-parametric kernel smoothing, logit analysis and "hockey-stick" analysis. These approaches yielded essentially equivalent fits of the data.

The "hockey-stick" model assumes the existence of an actual threshold dose below which there is no response. The data are thus fit to a function consisting of a horizontal line describing the no-response range of doses intersecting an upward sloping line describing linear dose-response. The point of intersection corresponds to the estimated (or effective) threshold dose. For delayed onset of walking, this model predicts the best estimate of the background frequency at 0% and a maternal threshold "dose" of 7.3  $\mu\text{g Hg/g hair}$ . From this analysis, Cox et al. concluded that "... Assuming the existence of a population threshold, the statistical best estimate places the value at about 10 ppm but the 95% range of uncertainty includes 0-13.6 ppm." For CNS effects, the model predicts a best estimate of background frequency of 9% and a maternal threshold "dose" of 10  $\mu\text{g Hg/g hair}$ . The kernel

smoothing and logit models make no assumptions about threshold and thus yield a continuous function predicting the probability of a response at each dose. For delayed onset of walking, the kernel smoothing and logit models both appear to give best estimates of background frequency of 0% and a 2-2.5% probability of an effect at a maternal hair Hg level of 5  $\mu\text{g/g}$ . For CNS effects, these models give a best estimate of background frequency of about 9% and a 1-2.3% probability of an effect at a maternal hair Hg level of 5  $\mu\text{g/g}$ . From the predictions of all three models, Cox et al. concluded that delayed onset of walking "...may be expected in the range of 10-20 ppm ( $\mu\text{g/g}$ )."

There are however, factors which lead to uncertainty in this analysis and make a precise interpretation of this analysis difficult. The overall data set in the Marsh study was small - 81 maternal and child pairs for CNS determinations and 78 for onset of walking. Of these, 20 children had abnormal CNS scores and 21 children had a delayed onset of walking. Only four mothers of affected children had hair Hg levels between 10 and 50  $\mu\text{g/g}$ . Mothers did not know the exact birthdates of their children resulting in an estimation of the exact age at onset of walking.

The greatest uncertainty in this analysis however, arises from the sensitivity of these statistical models to the background frequency of the abnormal responses in the Iraqi population. The "best estimates" of background frequency of abnormal responses employed by Cox et al. are determined by the value on the abscissa at the intercept of the best-fit function. These estimates are dependent on the overall fit of the various models to the complete data sets but do not necessarily reflect the actual background frequency of symptoms in the Iraqi population which is not known. Using the "hockey-stick" model, for CNS abnormalities, background frequencies of 0,2,4, and 9% result in threshold estimates of hair Hg of 7.8, 8.4 and 10 ppm with upper 95% confidence limits of the threshold of 24, 32 and 287 ppm respectively. For delayed onset of walking, the uncertainty in the estimate of the threshold is greater. Background frequencies of 0,2,4,8% result in threshold estimates of 7.3, 8, 9 and 119 ppm with 95% upper confidence limits of 13.6, 17 190 and 230 ppm respectively (WHO 1990). Thus, the uncertainty in the background frequency of these abnormalities has a relatively small effect on the best estimate of the threshold hair level, but can have a large effect on the uncertainty inherent in that estimate.

It should also be noted that this dose-response relationship is based on the maximum concentrations of Hg in the maternal hair segment formed during pregnancy. The course of the poisoning episode resulted in a sharp maximum in hair Hg levels at a well defined point in time with a steep slope on either side of the maximum (Cox et al. 1989). While the mechanism of Me-Hg effects on neurological development is not known, it is assumed that there are critical periods of vulnerability during gestation (WHO 1990).

These periods may occur during the late embryonic and late fetal stages and perhaps most particularly in the third to fourth month of pregnancy (Choi 1989; Mortensen 1992). There is no reason to assume that maximum hair levels of Hg necessarily occurred during such a critical period and hair levels corresponding to the critical period(s) could thus have been considerably lower. Furthermore, it is not clear at which point in their pregnancies each mother began consuming the contaminated bread. It is therefore possible that the children of some mothers received no in-utero exposure during the critical periods of development. This would effectively result in misclassification of some maternal-child pairs to the exposed cohort. Based on these considerations, the calculation of maternal Me-Hg exposure from peak Hg levels in hair probably overestimates the actual critical dose.

These conclusions gain some support from a study conducted in New Zealand. In the first stage of this study (Kjellstrom et al. 1986), a cohort of 10,930 pairs of mothers and newborns was established. A high Me-Hg exposure group was defined from fish consumption during pregnancy ( $n = 935$ ) and their Hg hair levels were measured. Average pregnancy period Hg levels greater than  $6 \mu\text{g Hg/g}$  hair were found in 73 of these mothers. At age 4, 31 children of these "high Me-Hg exposure" mothers were located and were each matched with one reference child on the basis of maternal ethnic group, birthdate, maternal age and birth hospital. The geometric mean maternal hair Hg concentration (average pregnancy period) was  $8.5 \mu\text{g/g}$  (90% of values  $6-12 \mu\text{g/g}$ ) for the high exposure group and  $1.6 \mu\text{g/g}$  for the reference group. Children's performance on the Denver Developmental Screening Test (DDST) was measured for both groups. This standardized test measures development in gross motor, fine motor, language and personal-social categories based on 5-10 determinations in each category. The prevalence of "abnormal" and "questionable" determinations in the high Hg exposure group was significantly greater than in the reference group (52% versus 17%,  $p < 0.005$ ) with an odds ratio of 5.3. Under the strictest of several criteria for including matched pairs in the analysis, the number of pairs decreased to 19. The odds ratio (3.5) remained elevated although the difference in test results was marginally significant ( $p < 0.1$ ) probably due to the small sample size. No evidence was found for confounding from socio-economic factors, health status, maternal smoking, or the duration of breastfeeding. A logit analysis of dose-response showed a 50% probability of decreased test performance at a maternal hair Hg concentration of about  $10 \mu\text{g/g}$  with no effect seen for hair levels below  $6 \mu\text{g/g}$ . Given the small size of the high exposure population however, quantitative dose-response conclusions can only be considered suggestive.

Several potentially important caveats to this study should be noted. Only 38 children of the 73 high Hg exposure mothers were tested. An additional 7 were eliminated due to unsuccessful matching and one refusal to participate, giving a final

participation rate of 43%. This low response rate seems mostly the result of a non-exhaustive search procedure and it is possible that there was a bias in the failure to locate children which was related to decreased test performance. Low birthweight was more prevalent in the high exposure group (5/31) than in the reference group (2/30) and within the high exposure group, low birthweight children were slightly more likely to have "abnormal" or "questionable" test performance. In addition, 5 of the high exposure children were premature (4 of these were low birthweight) compared to 2 reference children. Although none of the differences were statistically significant, low birthweight and/or premature birth in the high exposure group could have disproportionately contributed to decreased test performance. Potential confounding from socio-economic status was not examined directly. Rather, matching of pairs on the basis of birth hospital appears to have resulted in the matching of pairs from communities with similar socio-economic characteristics. The accuracy with which this methodology completely controlled for the influence of socio-economic factors is difficult to determine. Maternal alcohol consumption was not quantified, but was assumed to be closely related to smoking. Smokers were somewhat more prevalent in the high exposure group than in the reference group (48% vs. 33%), although smoking was not found to be associated with "abnormal" or "questionable" test results. While the alcohol consumption may be strongly linked with cigarette consumption, it is not clear that the influences of smoking and alcohol on DDST test results are linked in the same manner. Thus, the increased prevalence of smoking in the high exposure group could imply increased alcohol consumption whose influence on the DDST results cannot be judged. Pacific Island maternal-child pairs accounted for a disproportionately high percentage of the "abnormal" and "questionable" DDST results in both the aggregate high exposure and the reference groups. Proper matching by ethnic group should have eliminated any bias in the aggregate DDST scores resulting from ethnic-based differences in test performance. Nonetheless, high background rates in the Pacific Island group makes it difficult to distinguish between the overall and group-specific effects of Me-Hg exposure on test performance.

Finally, it should be noted that the exposure level of the high exposure group can only be considered to be "high" within the context of this study. Only 29% of the high exposure mothers had hair Hg levels  $\geq 10 \mu\text{g/g}$ . If, as suggested by Cox et al. (1989), there is a threshold for Me-Hg in-utero effects at hair Hg levels of  $10 \mu\text{g/g}$  or higher, the "exposed" group in this study contains a large proportion of children who were not effectively exposed. Thus, the association between Me-Hg exposure and DDST performance detected in this study could significantly underestimate the strength of the association for those exposed above a threshold level.

In the second stage of this study (Kjellstrom et al. 1989), 61 children of the original group of 73 high Me-Hg exposure mothers were located at age 6. The mothers had a geometric mean hair Hg level (pregnancy period average) of 8.3  $\mu\text{g/g}$ , 85% with levels of 6-12  $\mu\text{g/g}$ . The children were matched with three control children from the original cohort based on maternal ethnic group, sex, maternal age, maternal smoking and maternal residence time in New Zealand. One control group had intermediate maternal hair Hg levels (3-6  $\mu\text{g/g}$ , geometric mean = 4.5). The other two control groups both had low hair levels (0-3  $\mu\text{g/g}$ , geometric mean = 2.0), but differed in fish consumption. All children were tested with a battery of 16 scholastic, psychological and behavioral tests. Based on correlation among tests and on ability to discriminate individual performance, but blind to the influence of Me-Hg exposure, 5 of the original 16 tests were selected for further analysis. The effect of maternal hair Hg levels on performance in each of the 5 tests was determined in multiple regression analysis controlling for potential confounders including the matching variables as well as other potential confounders (e.g. social class, birthweight, maternal alcohol consumption, Apgar score, breastfeeding). In the analyses of all 5 tests by standard regression, the influence of Hg hair levels  $>6 \mu\text{g/g}$  on test performance was negative (reduction of tests scores). When robust weighting was applied to the analysis, the effect of hair Hg  $>6 \mu\text{g/g}$  was statistically significant ( $p < 0.02$ ) in three of the tests and borderline significant ( $p > 0.075$ ) in the other two. Two of the five tests (Test of Language Development - spoken language quotient and Wechsler Intelligence Scale for Children, Revised - full intelligence quotient) were significantly better at accounting for the overall variance in the test results. The influence of maternal hair Hg  $>10 \mu\text{g/g}$  in the robust regression models for these two tests containing the matching variables and 2-3 additional variables (social class,  $>6$  months of breastfeeding and number of languages spoken at home), was statistically significant and explained 2-2.5% of the overall variance in the test scores (8-10% of the variance accounted for by all the variables in the final model). Hair levels of 6-10  $\mu\text{g/g}$  accounted for about 1% of the variance, but was significant for only one of the tests.

This second stage study addressed some of the weaknesses encountered in the first stage study. A response rate of 78% of the high exposure pairs was obtained compared to 43% in the first stage allowing more definitive statistical analysis.. Matching of exposed and reference pairs appeared to be unambiguous and a larger number of potentially confounding variables was investigated. However, as pointed out by Magos (1992), as in the first stage study, Pacific Islanders were more prevalent (66%) in the high exposure group than Europeans or Maoris and appeared to perform disproportionately poorly. As discussed previously, proper matching by ethnic group should have eliminated confounding due to inherent ethnic differences in test performance and, given the apparent completeness of matching in this stage, this confounding factor

should pose even less of a potential problem than in the first stage. Nonetheless, as in the first stage the disproportionate test response of the Pacific Island group makes it difficult to separate group-specific effects of Me-Hg exposure from generalized effects. Kjellstrom et al. point out, that the number of variables addressed by the multiple regression models is likely to result in poor precision in the determination of the contribution of maternal Me-Hg exposure to test performance. However, as the purpose of this study is not to determine a dose-response relationship, but is largely to explore whether Me-Hg exposure is associated with decreased performance on these tests, the lack of precision is not necessarily a serious problem. As the authors point out, these tests are not specific for the developmental effects of Me-Hg. They are also quite different from those employed by Marsh et al. (1987) in Iraq. Thus, even if the strength of association between maternal Me-Hg exposure and test performance could be precisely estimated from these analyses, it is not clear what significance such a determination would have. Therefore, as the authors additionally note, the consistency of association across tests may be a more important indicator of the significance of the association than the quantitative estimate of the size of the effect. Finally, it should be remembered that the process of elimination of tests from the original group of 16 to the 5 was independent of the effect of Me-Hg exposure on these tests. The selection of the two final tests from among these five considered the effects of Me-Hg only as one of the independent variables in the overall models. Thus, it does not appear, as suggested (Magos 1992), that the significant associations between Me-Hg exposure and decreased test performance found in the two ultimate tests could have arisen as a chance occurrence from among the original group of 16 tests.

McKeown-Eyssen et al. (1983) measured maximum pregnancy period hair Hg levels in the mothers of 234 Cree Indian children (1-2.5 years old) in northern Quebec. The mothers were presumably exposed to Me-Hg through fish consumption. The mean hair level was 6.0  $\mu\text{g/g}$  for mothers of boys and girls (s.d. boys = 5.2, s.d. girls = 5.0) with 6%  $>20 \mu\text{g/g}$ . Clinical neurologic criteria, similar to those used by Marsh et al. as well as some portions of the DDST were employed. The association of these criteria with maternal hair Hg levels was investigated accounting for the influence of confounders (child's age, maternal age, maternal smoking, alcohol and caffeine consumption, and duration of breastfeeding). A statistically significant association was found between maternal hair Hg levels and abnormalities in muscle tone and reflexes in boys (odds ratio = 7.1), but not in girls. Although the largest incidence of these abnormalities (39%) occurred in the highest maternal exposure group (13.0-23.9  $\mu\text{g Hg/g}$  hair,  $n = 13$ ), a dose-response relationship across exposure groups was not observed. No other significant negative associations were observed. In girls however, incoordination was found to be negatively (but not significantly) associated with maternal hair Hg levels.

The authors question the significance of this finding based on lack of severity and clear clinical relevance of the abnormalities, the lack of a clear dose-response relationship and the lack of association of other abnormalities with Me-Hg exposure. However, this study, published in 1983, preceded those of Marsh et al. (1987) and Kjellström et al. (1986, 1989). The authors thus had little basis for gauging the relevant range of doses or responses. The distribution of maternal hair Hg levels in this population was considerably below that studied by Marsh or Kjellström and it is therefore not surprising that no dose-response relationship was observed across the comparatively small range of exposures or that only relatively mild effects were associated with the highest levels of Me-Hg exposure. Furthermore, the observation of an association only in boys is consistent with Marsh et al. (1987) who found that in the Iraqi cohort boys were more likely to have developmental abnormalities. Viewed in the context of these subsequent studies, this limited association appears consistent with the lower end of the dose-response scale. However, as Magos (1992) has pointed out, the negative association between maternal exposure and incoordination in girls which is likely to be without biological significance, may provide an indication of the inherent statistical uncertainty in this study. If this same level of uncertainty is assumed to apply to the positive associations, it is possible that the association between maternal Me-Hg exposure and muscle tone and reflexes seen in boys could have arisen by chance.

Each of the human studies investigating the relationship between maternal exposure to Me-Hg and developmental effects has weaknesses or uncertainties which make the determination of an absolute or effective threshold level of exposure difficult. Taken individually, none of these studies provide an unambiguous basis for such a determination. However, the statistical or methodological uncertainties and weaknesses in each study are different and do not bias them toward a similar quantitative conclusion. Viewed together, these studies suggest similar quantitative conclusions despite significant differences in geographic, cultural and ethnic characteristics of the exposed populations; different exposure scenarios; different study methodologies; and the measurement of quite different types of adverse effects. Each of the studies independently suggests an association between maternal hair levels around 10-20  $\mu\text{g/g}$  and subtle neurologic developmental effects. Such a determination does not result from a finding of scientific certainty, but is based on the assessment of the overall weight of evidence. As such, there is uncertainty inherent in the determination. However, it would be surprising if there were not some uncertainty inherent in attempting to identify a threshold exposure level for adverse effects which are not yet well defined, but potentially significant. It may well be that such uncertainty is not the result of incomplete or inexact data, but results from real variability in the toxicologic response of heterogeneous human

populations in the lower ranges of the frequency distribution of response.

Of the studies of human exposure to Me-Hg, only that of Marsh et al. (1987) and its re-analysis by Cox et al. (1989) provides a dose-response relationship which can be used to select a single exposure level, consistent with the overall weight of evidence, for the calculation of an Acceptable Daily Intake (ADI). Observation of the data of Marsh et al. in figures III-1 and III-2 suggests that on an absolute scale, the first occurrence above background of adverse responses (the Lowest-Observed-Effect-Level -LOEL) may be at a maternal hair concentration of 14-16  $\mu\text{g/g}$ . However, the analysis of Cox et al. is not directly based on or limited by an actual exposure datum corresponding to the LOEL. Rather, this analysis attempts to compensate for the relative scarcity of data at low exposure levels by employing the entire data set to extrapolate to an effective threshold exposure or to a probabilistic estimate of risk at any given exposure. This approach can lead to confusion in the application of the 'threshold' and 'LOEL' terms in discussing these data. The LOEL is usually operationally defined within a given data set by the actual measured dose at which the toxic response is first observed. The threshold dose is a theoretical construct corresponding to the dose below which no toxic effect would be observed. In practice, the threshold can only be estimated since it is always possible that a toxic response can be observed at a dose below the observed LOEL or above the observed No-Observed-Effect-Level (NOEL) dose. In a well designed dose-response study, the LOEL dose should be close to the estimated threshold dose. In the data set of Marsh et al., the inherent uncertainty in the data precludes the precise determination of a LOEL exposure from direct observation of the data and the estimated threshold level is thus, not practically distinguishable from an estimation of the LOEL. In the "hockey-stick" analyses of Cox et al., the best estimate of the threshold/LOEL is 10  $\mu\text{g/g}$ . Cox et al. (1989) appear to confirm this analysis in stating that "...motor retardation should occur in children prenatally exposed to maternal hair concentrations less, than 50 ppm and may be expected in the range of 10-20 ppm." The ADI will thus be calculated assuming an effective LOEL of 10  $\mu\text{g}$  Hg/g hair.

### 3. Biokinetic Determination of Daily Intake

WHO (1990) has reviewed the relationship between total Hg in hair and blood from 10 studies. The hair/blood ratios from these studies fall within a fairly narrow range (mean = 265 [ $\mu\text{g}$  Hg/g hair]/[ $\mu\text{g}$  Hg/ml blood], s.d. = 63.8). This ratio can be adjusted on an Me-Hg-specific basis from the observation that this ratio is about 6% smaller if organic Hg rather than total Hg is measured in both hair and blood (Phelps et al. 1980). This yields a hair/blood ratio for Me-Hg of 249 essentially identical to the generalized ratio of 250 cited by WHO (1990). Applying this relationship to a

hair concentration of 10  $\mu\text{g/g}$ , gives an estimate of a corresponding blood concentration of 0.04  $\mu\text{g/ml}$  (40  $\mu\text{g/l}$ ).

WHO (1990) has employed a basic one-compartment, first-order kinetic model of accumulation and excretion, to estimate daily intake of Me-Hg from blood concentration. In this model, the accumulation in the whole body or equivalent tissue compartment (e.g. blood) is given by:

$$A = (a/b)(1 - e^{-bt}).$$

Where A = the accumulated amount ( $\mu\text{g}$ )  
a = the daily amount taken up by the compartment ( $\mu\text{g/day}$ )  
b = the elimination rate constant ( $\text{day}^{-1}$ )  
t = time (days)

The elimination rate constant (b) is related to the half-life ( $T_{1/2}$ ) in the compartment by:

$$T_{1/2} = (\ln 2)/b.$$

The daily amount taken up by the compartment (a) is related to the daily dietary intake (d), the fraction of the ingested dose which is absorbed (F) and the fraction of the total dietary intake taken up by the compartment (f) by:

$$a = F \times f \times d.$$

By definition, under steady-state conditions, the daily amount taken up by a compartment is equal to the daily amount excreted from that compartment. This occurs after a sufficiently long period of time has passed so that  $e^{-bt}$  approaches zero and the amount accumulated in the compartment (A) is given by:

$$A = a/b.$$

The concentration in the compartment at steady-state (C) is then given by the accumulated amount divided by the volume of the compartment (v):

$$C = A/v.$$

Substituting values, yields the following relationship:

$$C = (F \times f \times d)/(b \times v).$$

Assuming 95% gastro-intestinal absorption (F), a half-life of 70 days (corresponding to an elimination rate constant of 0.01  $\text{day}^{-1}$ ), 5% of the total body burden of Me-Hg contained in the blood (f) and a blood volume (v) of 5 liters, steady-state concentration of Me-Hg in the blood ( $\mu\text{g/l}$ ) is predicted to be 95% of the daily intake ( $\mu\text{g}$ ) (WHO 1990).

WHO (1990) has compared this prediction with the relationship found in short-term controlled intake studies as well as that found in populations with long-term uncontrolled intakes. In four controlled intake studies, the ratio of Me-Hg blood concentration

( $\mu\text{g}/\text{l}$ ) to Me-Hg intake ranged from 0.8- 1.0 (mean = 0.9, s.d. = 0.12) in close agreement with the prediction of the one-compartment model. However, in five studies of populations with uncontrolled intakes whose dietary exposure to Me-Hg was presumably sufficiently long and regular to establish kinetic steady-state conditions, the ratio (calculated by linear regression of individual blood concentrations on individual daily intake) ranged from 0.3-0.8 (mean = 0.56, s.d. = 0.19). WHO (1990) speculated that the apparent divergence of these results from the prediction of the model may result from the possibility that these populations were not, in fact, at kinetic steady-state. Additionally, the fact that population-based estimates of intake are inherently uncertain may also have contributed to this difference. However, Sherlock and Quinn (1988) have pointed out that in linear regression analyses of population data, it is assumed that the intra-individual variability in the independent variable (in this case, the daily Me-Hg intake) is negligible in comparison to the intra-individual variability on the dependent variable (in this case, the blood concentration). In the uncontrolled intake studies, this was not the case. Intra-individual variability in Me-Hg intake resulted from daily variability in fish intake as well as from variability in the Me-Hg content of individual fish. Intra-individual variability in Me-Hg blood concentration, on the other hand, is damped by the relatively long half-life of Me-Hg in the blood which assures that even large increases in intake are diluted into the comparatively large existing body burden. This results in significantly underestimating the ratio of blood concentration to intake in such studies.

The one-compartment model predicts that a blood concentration of 40  $\mu\text{g}/\text{l}$  would result from an average daily Me-Hg intake of 42  $\mu\text{g}/\text{day}$ . For a 62 Kg woman, this is equivalent to a dose of 0.7  $\mu\text{g}/\text{kg}/\text{day}$  for ongoing exposure. If intake is calculated based on the prediction of the uncontrolled intake studies cited by WHO (1990) the corresponding estimate of intake could be higher by a factor of about 1.7 (0.95/0.56). The analysis of Sherlock and Quinn (1988) however, argues that the actual uncertainty in this estimate is considerably less. Furthermore, the close agreement among the controlled intake studies cited by WHO (1990), despite the wide range in exposure duration (from a single dose, to daily doses over 100 days), suggests that the assumption of kinetic steady-state in the one-compartment model employed by WHO is not critical and that violation of that assumption may lead to a relatively small error. Thus, the one-compartment kinetic model appears to give a reasonable overall average prediction of daily Me-Hg form blood concentration.

#### 4. Derivation of an Acceptable Daily Intake (ADI) From Human Exposure Studies

The ADI (as well as similar guidelines such as the RfD and the PTWI) is not necessarily a statement of scientific certainty, but

a determination of public health protection. It is intended to define the highest level of exposure which is adequate to reliably protect the most reasonably sensitive members of the population. This requires that the ADI incorporate a margin of safety adequate to compensate for those generally unquantifiable scientific uncertainties which might result in overestimating a protective level of exposure. Because of this margin of safety, the ADI does not define a strict boundary between the likelihood and absence of adverse health effects. Exposure above the ADI increases the risk of health effects, but does not necessarily imply that health effects will occur. The appropriate margin of safety is estimated by the use of uncertainty factors derived from generalized toxicologic concepts and longstanding principles of public health protection (NAS 1977, 1980). These uncertainty factors are not chemical, species or, exposure-specific. As such, the actual values taken by the factors in any given application is somewhat arbitrary, but their application follows a standardized framework.

A LOEL from human data is generally divided by an uncertainty factor of 10 to estimate a NOEL dose which is not indicated in the data (Barnes and Dourson 1988). As discussed previously, in the "hockey-stick" analysis of Cox et al. (1989) the maternal hair concentration of Hg corresponding to the best estimate of the effective threshold dose of Me-Hg is not practically distinguishable from the level corresponding to the LOEL dose. Furthermore in the probabilistic analysis of Cox et al. 10  $\mu\text{g/g}$  corresponds to a best estimate of risk of about 5-10%. These observations argue that 10  $\mu\text{g/g}$  should be considered an estimate of the LOEL rather than the NOEL. Uncertainty adjustments may also incorporate "modifying factors." The modifying factor is defined as "...an additional uncertainty factor that is greater than zero and less than 10 . The magnitude of the modifying factor depends on the professional assessment of scientific uncertainties of the study [*not otherwise addressed by uncertainty factors*], e.g. the completeness of the overall data base..." (Barnes and Dourson 1988). The likely overestimation by Cox et al. (1989) of the effective threshold dose from maximum hair levels of Hg as well as the possibility of misclassification of the exposed cohort (discussed previously) argues for the use of uncertainty modifiers. Dividing the calculated threshold maternal intake for a 62 kg woman (0.7  $\mu\text{g/kg/day}$ ) by an overall uncertainty factor of 10 yields an ADI of 0.07  $\mu\text{g/kg/day}$ . This is considerably below the current EPA RfD of 0.3  $\mu\text{g/kg/day}$ . For a 62 kg woman, 0.07  $\mu\text{g/kg/day}$  is equivalent to an intake of 4  $\mu\text{g/day}$  compared to 19  $\mu\text{g/day}$  at the current RfD.

##### 5. Derivation of an Acceptable Daily Intake (ADI) From Animal Studies

Two closely related animal studies address *in-utero* exposure to Me-Hg at doses comparable to those of concern for human exposure. Bornhausen et al. (1980), in a study of operant behavior

performance, dosed pregnant rats on days 6-9 of gestation with 0 (controls), 5, 10 or 50  $\mu\text{g}/\text{kg}$  Me-Hg/day. Male and female offspring (10 at each dose level) were tested four months after birth in a procedure requiring a minimum number of lever presses within a specified time span to obtain food. Once this task was learned, the required lever presses and the allowed time were increased in a series of three steps. For offspring of dams receiving 50 and 10  $\mu\text{g}/\text{kg}$  Me-Hg, the authors noted statistically significant decrements in performance relative to controls in each of the two highest performance demand levels. No significant decrement was seen for the offspring of dams receiving 5  $\mu\text{g}/\text{kg}$ . In an earlier study by the same group employing the same methodology (Müsch et al. 1978), performance decrements were seen in offspring of dams dosed with 2000 or 50  $\mu\text{g}/\text{kg}/\text{day}$  ( $n = 9$  for controls and each dose levels). Lower doses were not investigated. The two studies are consistent in showing an effect at maternal doses as low as 50  $\mu\text{g}/\text{kg}/\text{day}$ . It should be noted however, that the 4 daily doses of Me-Hg administered to the dams were not likely to have produced steady-state elimination kinetics. The resulting body burdens of Me-Hg were probably less than would have occurred with administration of the same dose over a longer period of time. In addition, the half-life of Me-Hg in rodents is only about 11-23% of that in primates (WHO 1976). Thus, the results reported by Bornhausen et al. are likely to overestimate the dose which would produce the same effects in rodents with chronic exposure and are also likely to overestimate the effective dose for corresponding adverse effects in humans following chronic exposure.

There are some problems in interpreting these data. While the number of tested offspring is known, the number of dams dosed with Me-Hg is not explicitly stated. It is not clear what capability of the offspring is being tested in this protocol - both learning adaptation and motor ability seem to play a role. Magos (1992) has pointed out that the presentation of the data in these studies does not permit replication or complete re-evaluation of the statistical analysis carried out by the authors. Based on partial data presented by Bornhausen (1980), Magos suggests that significant decrements in performance at the two highest demand levels may only have occurred at the 50  $\mu\text{g}/\text{kg}/\text{day}$  dose level and not, as claimed by the authors, at the 10  $\mu\text{g}/\text{kg}/\text{day}$  level. In the absence of complete data, it is not possible to judge the accuracy and significance of this claim. However, examination of the data as presented suggests that if the performance decrements which occurred at 10  $\mu\text{g}/\text{kg}/\text{day}$  are not statistically significant they are, nonetheless, consistent with an overall dose-response relationship. It therefore does not appear appropriate to equate 10  $\mu\text{g}/\text{kg}/\text{day}$  with a NOEL dose.

Among the various studies of Me-Hg developmental effects in animals, tests of operant behavior performance are not common and there does not appear to have been any attempt to independently duplicate these results. The lack of confirmation is noteworthy

because the NOEL dose found by Bornhausen et al. is considerably lower than that found in other animal studies (ATSDR 1989). Magos (1992) has noted that the total amount of delivered Me-Hg at the highest dose employed by Bornhausen et al. (1980) 4 x 50  $\mu\text{g}/\text{kg}/\text{day}$  is only 5% of the single dose in post-natal mice producing arrest of cerebellar mitosis with permanent reduction in cell number but no cytotoxicity (Sager et al. 1984). Magos concludes that the effects claimed by Bornhausen et al. (1980) occurred at a dose 1-2 orders of magnitude below those seen in other behavioral tests of the effects of in-utero exposure.

However, Gunderson et al. (1986) tested visual recognition memory in 10 infant macaque monkeys whose mothers received either 50  $\mu\text{g}/\text{kg}/\text{day}$  (8) or 70  $\mu\text{g}/\text{kg}/\text{day}$  (2) of Me-Hg throughout pregnancy as well as 10 control infants whose mothers received no Me-Hg. Visual recognition memory measures the ability of infants to distinguish between novel and previously encountered visual stimuli by measuring the duration of observation. Novel stimuli evoke longer observation. This test in macaques directly parallels that in human infants (Gunderson et al. 1986). The exposed and control infants showed no differences in birth weight, or gestation time and were clinically normal. While the control infants looked significantly longer (55%) at the novel stimulus than the familiar stimulus, the exposed infants showed no significant difference (4%) in duration of observation. The presentation of the data in this study does not permit a determination of whether the performance of infants whose mothers received 70  $\mu\text{g}/\text{kg}/\text{day}$  was different from those receiving 50  $\mu\text{g}/\text{kg}/\text{day}$ . However, the blood concentration of Me-Hg in the infants of mothers exposed to 70  $\mu\text{g}/\text{kg}/\text{day}$  was within the range seen for those whose mothers received 50  $\mu\text{g}/\text{kg}/\text{day}$ . This study tested a quite different behavioral endpoint from that of Bornhausen et al. (1980) and M $\ddot{u}$ sch et al. (1978). It is therefore difficult to say to what extent the finding of Gunderson et al. (1986) confirm those findings. Nonetheless, the study of Gunderson et al. demonstrates the plausibility of subtle neurobehavioral effects following maternal exposure to Me-Hg at or close to 50  $\mu\text{g}/\text{kg}/\text{day}$ .

Burbacher et al. (1990) have pointed out that there are wide interspecies differences in the blood/brain ratio of Me-Hg. This ratio is estimated to be 0.06 for rats and 6.00 for humans. They suggest that interspecies comparison of effective doses be made on the basis of the dose to the fetal brain rather than the administered maternal dose. Based on their calculations, the brain concentration of Me-Hg resulting from the LOEL dose reported by Bornhausen is equivalent to 0.08-0.2 of the brain concentration of Me-Hg in children of mothers in the Iraqi cohort with hair levels of 10  $\mu\text{g}/\text{g}$ . Such calculations carry uncertainties with respect to fetal blood/brain distributions and species differences in the time course of development and thus cannot currently serve as the basis for quantitative derivation of an ADI. Nonetheless, this comparison suggests that the effects seen by Bornhausen et al. may

correspond to human doses even lower than those estimated from Cox et al. (1989).

The most appropriate estimate of the NOEL dose of Me-Hg from the data of Bornhausen et al. (1980) appears to be 5  $\mu\text{g}/\text{kg}/\text{day}$ . If, as implied by Magos (1992), the LOEL dose from this study is taken to be 50  $\mu\text{g}/\text{kg}/\text{day}$  rather than 10  $\mu\text{g}/\text{kg}/\text{day}$  as indicated by the authors, this estimate of the LOEL dose would be consistent with that from Müsch et al. (1978) as well as that from Gunderson et al. (1986). As discussed previously, the NOEL dose is generally estimated from the LOEL dose by dividing the LOEL dose by an uncertainty factor of 10 (Barnes and Dourson 1988). Thus, if the LOEL dose from these studies is taken to be 50  $\mu\text{g}/\text{kg}/\text{day}$ , the NOEL is still estimated to be 5  $\mu\text{g}/\text{kg}/\text{day}$ . Since, as discussed, the dose-response relationship reported by Bornhausen et al. likely overestimates the corresponding effective dose with chronic human exposure, 5  $\mu\text{g}/\text{kg}/\text{day}$  does not appear to be an overly conservative estimate of the NOEL dose from these data sets. To derive an ADI from animal data, a NOEL dose is usually divided by an uncertainty factor of 100 (NAS 1977, 1980; Barnes and Dourson 1988). Adjusting the 5  $\mu\text{g}/\text{kg}/\text{day}$  NOEL estimate accordingly, yields an ADI of 0.05  $\mu\text{g}/\text{kg}/\text{day}$ . This is nearly identical to the ADI of 0.07  $\mu\text{g}/\text{kg}/\text{day}$  estimated from Cox et al.

## 6. Exposure Through Breast Milk

The children studied by Marsh (1987), Kjellström et al. (1986, 1989) and McKeown-Eyssen et al. (1983) were exposed to maternal Me-Hg through breast milk as well as in-utero. Kjellström et al. and McKeown-Eyssen et al. attempted to control for exposure through breast milk by investigating the influence of a variable for duration of breastfeeding. While no such influence was detected, duration of breastfeeding would not necessarily be predictive of post-natal developmental effects if there are specific windows of post-natal vulnerability to Me-Hg. Thus, it is possible that the effects associated with maternal exposure in these studies may, in part, reflect exposure through breast milk.

Amin-Zaki et al. (1974) investigated total Hg in the blood of infants exposed through breast milk in Iraq. The mothers were exposed to Me-Hg only post-partem. Sampling of this population began several months after cessation of exposure to the contaminated grain when maternal body burdens of Hg were declining. In four pairs followed over about seven months, the ratio of infant blood Hg concentration to maternal blood Hg concentration rose from less than 1.0 to 10-70. At least eight infants sampled only once had blood levels of total Hg greater than 200  $\mu\text{g}/\text{l}$  which, the authors note, corresponds to the estimated threshold for induction of toxic effects in adults (WHO 1976). Nonetheless, the authors report finding no clinical signs of toxicity in the infants.

Skerfving (1988) obtained blood and breast milk from 10 Swedish mothers exposed to Me-Hg through fish. Although Me-Hg accounts for about 95% of the total Hg in adult whole blood (WHO 1990), Skerfving found that it accounted for only about 20% of the total Hg in the milk. It should be noted that while the gastrointestinal absorption of inorganic Hg in adults is only about 5% (WHO 1991), it may be much greater in infants as indicated by the observation that newborn rats absorb about 38% of an oral dose of inorganic Hg (WHO 1991). Thus, the levels of total Hg in the infants' blood observed by Amin-Zaki et al. reflect an unknown contribution from inorganic Hg which may explain the absence of Me-Hg toxicity.

While Me-Hg exposure from milk appears to be less than might be predicted on the basis of maternal blood levels, there is currently no basis for the identification of a toxic endpoint appropriate to the estimation of risk from Me-Hg exposure in breast milk. There is a suggestion that developmental effects may result from post-natal exposure (Sager et al. 1984), but no dose-response data are available. The endpoint investigated by Cox et al. is specific to exposure in-utero, while the paraesthesia endpoint was not derived with reference to infants who may well have a different sensitivity than adults or older children.

#### 7. Selection of an Appropriate ADI Value

There remain significant gaps in our knowledge of the toxicology of low level in-utero exposure to Me-Hg. These gaps produce uncertainty in the determination of a threshold or "safe" dose. Clearly, additional research is needed and this research may change the current evaluation. However, as discussed, the ADI (or RfD) is not a finding of scientific certainty. As such, the derivation of an ADI value is based on the determination of the most reasonable value from the existing scientific literature which can provide an adequate level of public health protection. This criterion allows for the derivation of an ADI despite the existence of uncertainty in the scientific literature providing that the derived value is reasonably supported by the existing data.

The weight of evidence from several sources supports the conclusion that the current RfD for Me-Hg is not adequate to provide a reasonable margin of safety against effects in-utero. Both human and animal data suggest that an ADI based on developmental effects could be lower than the current RfD by a factor of 4 or more. At present, the analysis by Cox et al. (1989) of the data of Marsh et al. (1987) provides the best estimate from the human data base of an appropriate value for a ADI based on in-utero exposure. This value is calculated as 0.07  $\mu\text{g}/\text{kg}/\text{day}$ . Data from animal studies support an ADI for in-utero exposure of 0.05  $\mu\text{g}/\text{kg}/\text{day}$ . Because the human data are more directly relevant and several studies independently contribute to a consistent finding, they provide a more appropriate basis for an ADI than the animal

data. Nonetheless, there is little practical difference between the ADIs derived from these data sets and in the absence of reasonable human data, the animal data could support the derivation of an ADI.

It is important to realize that the mechanism and clinical manifestations of the adverse effects from relatively low-level in-utero exposure to Me-Hg are not well characterized. Among the human and animal studies reviewed here, several different endpoints, which, at least superficially, appear to be unrelated, have been investigated (e.g. neuro-motor development, visual recognition memory, scholastic performance). It is probably incorrect to interpret these studies as addressing isolated adverse effects, or to see the diversity among adverse effects as contradictory. Rather, the multiplicity of possible adverse neurologic symptoms should probably be interpreted as evidence of the potential for a broad impairment of brain development at an early stage prior to the separation and specialization of the affected functions.

Figure III-1: Neurologic Scores by Maternal Hair Hg

Neurologic Scores by Maternal Hair Hg

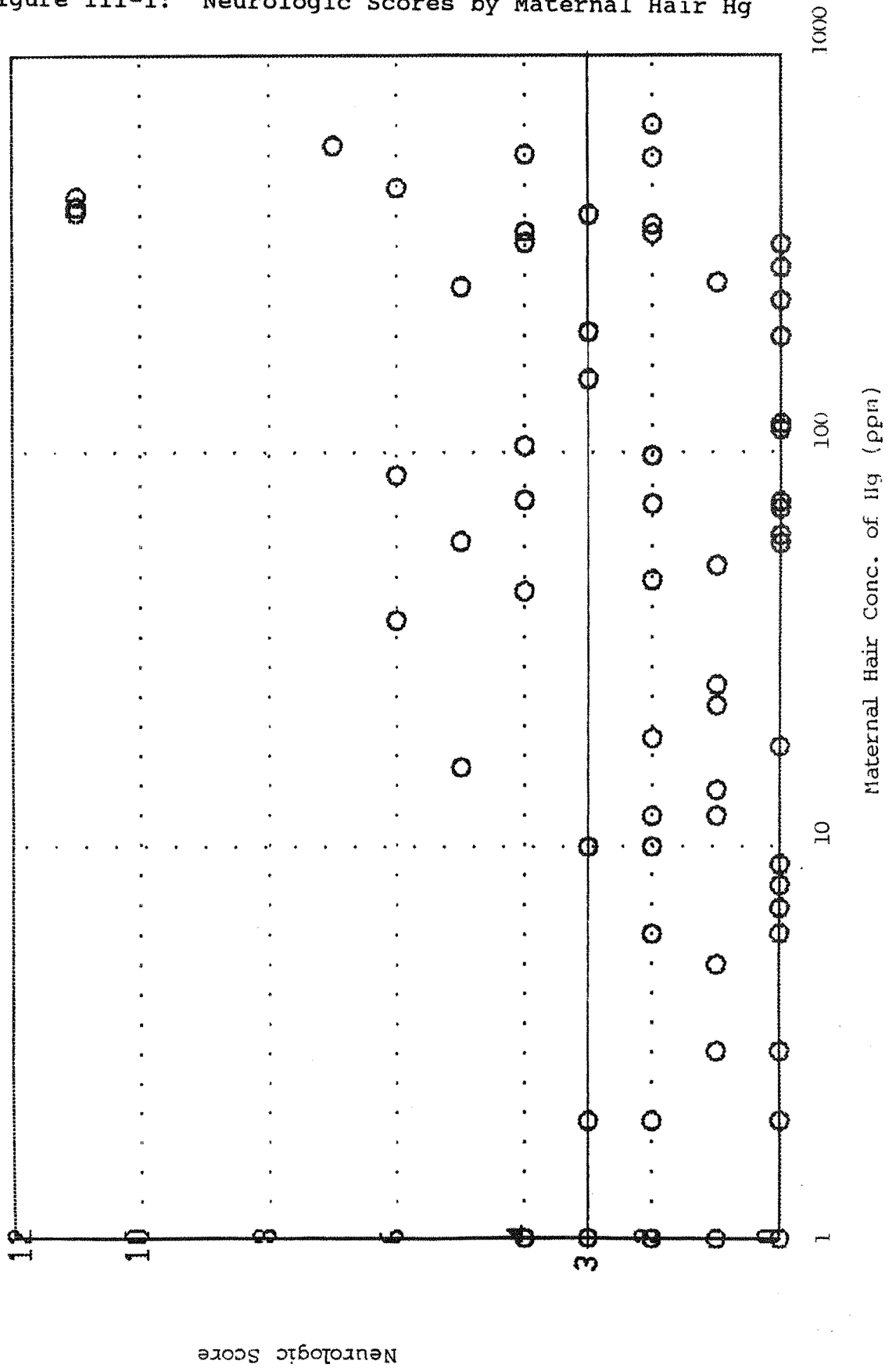
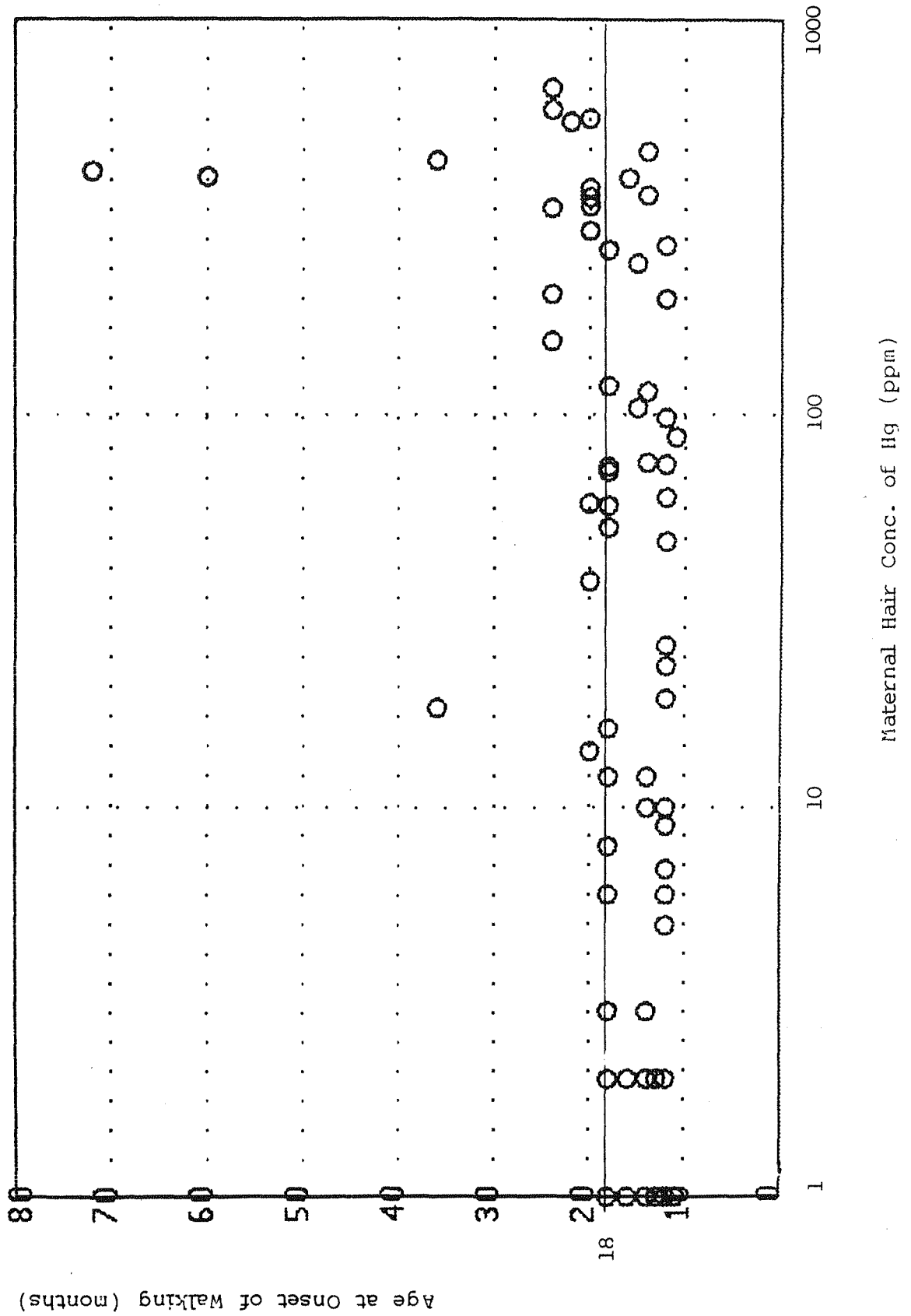


Figure III-2: Onset of Walking by Maternal Hair Hg

Onset of Walking by Maternal Hair Hg



# Neurological Scores by Maternal Hair Hg

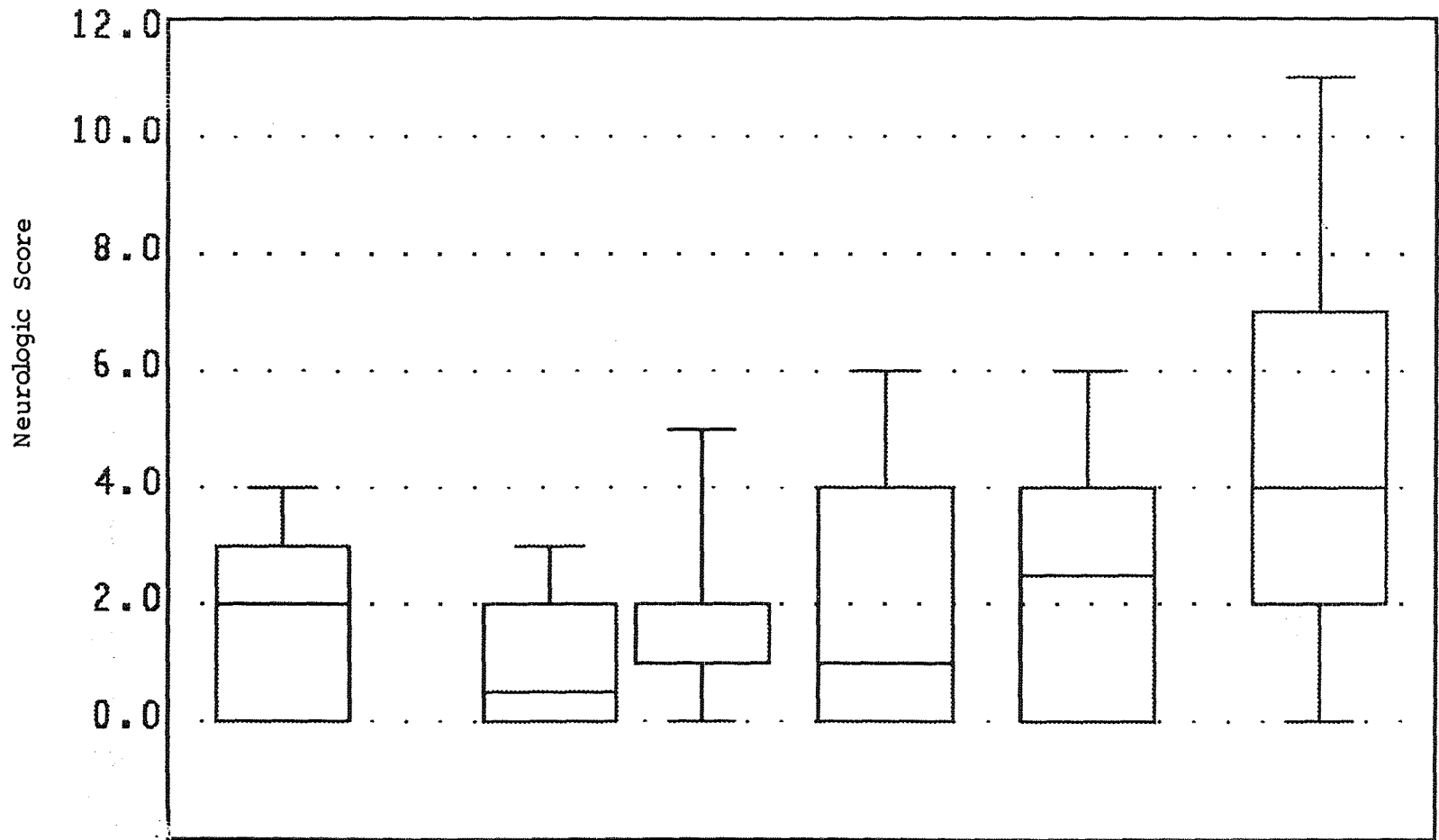
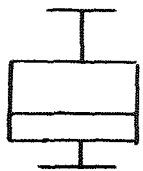


Figure III-3: Neurological Scores by Maternal Hair Hg - Grouped Results

- 42 -



95th percentile  
75th percentile  
50th percentile  
25th percentile  
5th percentile

0-2  
(25)

3-10  
(10)

11-25  
(7)

26-75  
(11)

76-250  
(10)

250-1000  
(18)

Maternal Hair Conc. of Hg (ppm)  
(number of observations)

# Onset of Walking by Maternal Hair Hg

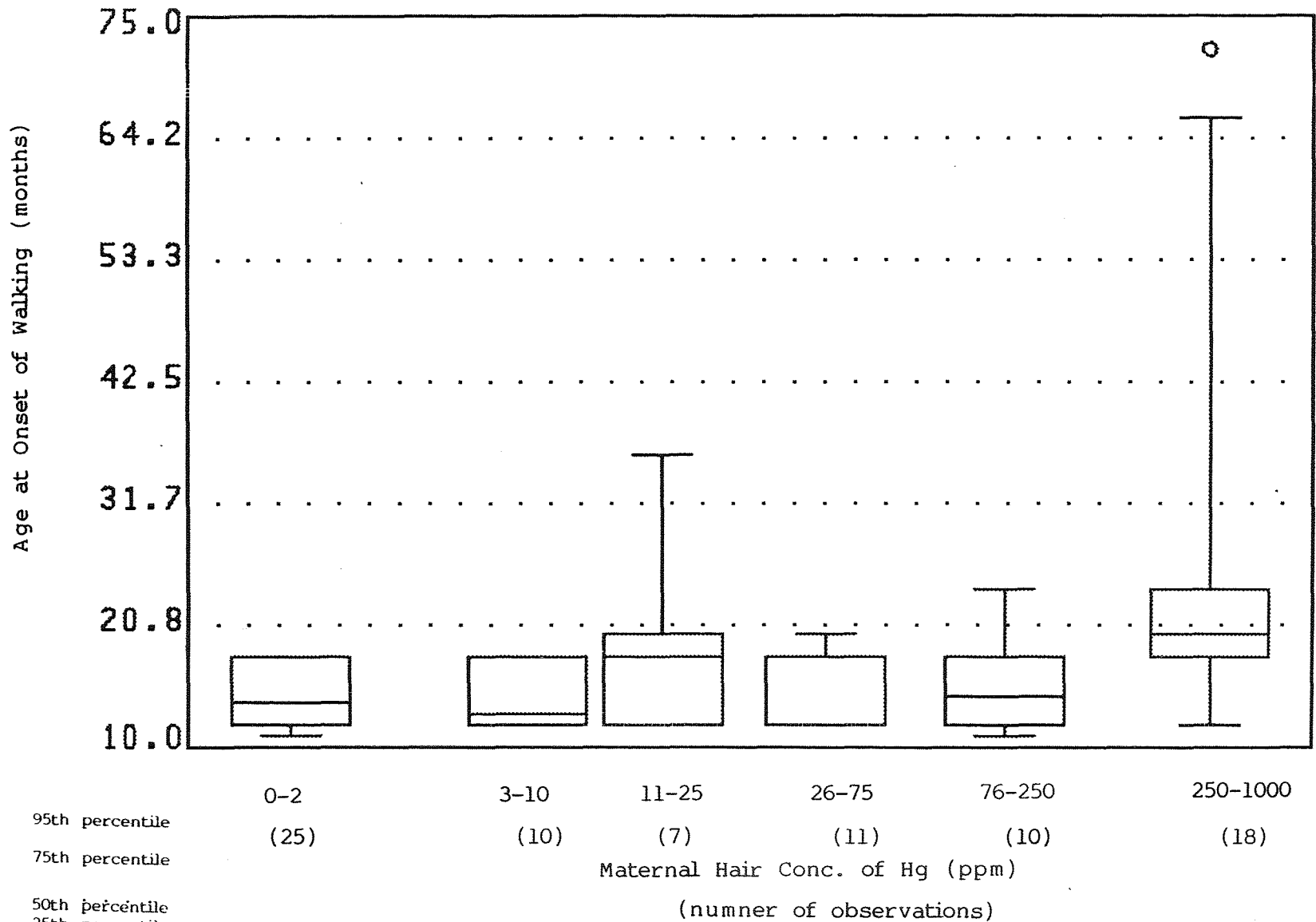
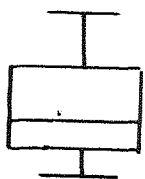


Figure III-4: Onset of Walking by Maternal Hair Hg - Grouped Results



95th percentile  
 75th percentile  
 50th percentile  
 25th percentile  
 5th percentile

#### IV. Background Exposure of Methylmercury in the Population

##### A. Estimate of Current National Background Exposure

Fish and seafood represent the only significant source of methylmercury exposure in the general population (WHO 1990) and its consumption can therefore be used to estimate Me-Hg exposure. Currently no data exist which are specific to background levels of fish/seafood in New Jersey. However, New Jersey's coastal location and the easy availability of fresh fish suggest that national data do not overestimate New Jersey-specific consumption. The Me-Hg content of fish varies by species as well as the age of the individual fish. A study by the National Marine Fisheries Service in 1978 (Hall et al. 1978) measured total Hg levels in the edible portions of 159 species of finfish, 18 species of mollusks and 16 species of crustaceans taken from U.S. coastal waters. The average Hg concentration of each species was determined and each species was assigned to a concentration category (0.1  $\mu\text{g}$  Hg/g wide). Based on the species in each category, the percentage of the total U.S. catch intended for human consumption was determined for each concentration. These values are given in Table 1. The distribution of Hg levels by percentage of the U.S. catch intended for human consumption is taken as a reasonable estimate of the actual Hg concentration in fish/seafood in the diets of fish/seafood eaters. The overall concentration average weighted by percentage of the catch is 0.11  $\mu\text{g}$  Hg/g. Recent analysis of unpublished NMFS data indicates that Hg levels in fish/seafood have remained constant since 1978 (USFDA 1992). It should be noted that these data do not address fish caught outside of US coastal waters, including imported fish. Also, these data do not provide information on the variability in consumption by individuals compared to the species distribution in the US catch.

The Market Research Corporation of America's (MRCA) 1982-87 14 Day Menu Census VI study gathered nationwide fish consumption data for the fish/seafood-eating portion of the population (USFDA 1992). The mean fish/seafood consumption among consumers is 32 g/d and the 90th percentile is 64 g/d. These data are not broken down by species or by the sex of the consumers.

These data on fish/seafood consumption are consistent with several recent estimates. The USEPA (1989b) estimated total average fish/seafood consumption at 30 g/day based on the average of two separate studies. The National Marine Fisheries Services (NMFS) reported that average per capita consumption of fish was 21 g/day in 1986, noting that this represented a 62% increase from 1960 (USEPA 1989b). These estimates are, however, lower than some based on older data. Tollefson and Cordle (1986) reported on a study conducted for the Tuna Research Foundation based on 1973-4 consumption data which found a mean fish/seafood consumption in the entire study population of 18 g/day. The USFDA 1982 revision of the Total Diet Study (Pennington 1983) using consumption data from

1976-80, estimated average per capita fish/seafood consumption for 25-30 year olds at 17 g/day. Differences among estimates may also result from whether consumption is expressed only for that portion of the population which eats fish/seafood, or for the entire population on a per capita basis. Estimates of consumption which include only fish eaters will necessarily be higher.

Cappon and Smith (1982) measured the fraction of total Hg in fish/seafood present as Me-Hg and found a mean value for marine fish of 70.1%. Bloom (1992) has recently argued that contamination in sampling handling and statistical inconsistencies led to underestimations in earlier analyses. Under ultra-clean conditions, the mean Me-Hg fraction of 15 species of saltwater and freshwater fish and crustaceans was 0.95 (s.d. = 0.063). The gastrointestinal absorption of Me-Hg is assumed to be between 90 and 100% (WHO 1990).

Daily intake of Me-Hg from fish can be calculated as follows:

$$I = M \times CT \times F \times A$$

I = Me-Hg intake ( $\mu\text{g}/\text{d}$ )

M = mass fish/seafood consumed per day (g/d)

CT = concentration of total Hg ( $\mu\text{g}/\text{g}$ )

F = fraction of total Hg present as Me-Hg

A = fractional GI absorption of Me-Hg

The data discussed above were used in a Monte Carlo probabilistic analysis of this equation. This is a computational technique which allows calculations with probability distributions rather than single numerical values. The final product is a probability distribution which describes the entire range of possible outcomes (Iman and Helton 1988; Thompson et al. 1992). The probability distributions used in this analysis are presented in Table IV-1. A Monte Carlo simulation of 20,000 iterations with Latin Hypercube sampling using the @RISK software package (release 2.01, Palisade Corp.) yielded the probability distribution of daily background Me-Hg intake in the fish eating population shown in Table IV-2 and Figure IV-1. To test the stability of this simulation, these results were compared to a 10,000 iteration simulation of the same model. For the 95th and 99th percentiles of Me-Hg intake, the difference ( $\mu\text{g}/\text{day}$ ) between the 20,000 and 10,000 iteration simulations were -1.9% and -3.8% respectively. These differences are not significant and indicate that the current simulation accurately represents the overall model.

The distribution in Table IV-2 and Figure IV-1 applies to the portion of the population which eats fish/seafood. Based on MRCA data (USFDA 1992), this includes 77% of the total U.S. population. Based on a 1973-4 study conducted for the Tuna Research Foundation (Tollefson and Cordle 1986) this includes 93% of the US population. The average of these values is 85%. Table 2 can be modified accordingly to reflect Me-Hg intake values in the total population.

Thus, the mean Me-Hg intake for the total population is approximately  $0.85 \times 3.8 \mu\text{g/day} = 3.2 \mu\text{g/day}$ .

The USEPA (1990b), employing unpublished U.S. FDA Market Basket data, estimated that the mean adult intake of total Hg from meat, fish and poultry for the entire population is  $3.27 \mu\text{g/day}$  due primarily to fish. WHO (1990) estimated a mean intake of total Hg from fish of  $2.9 \mu\text{g/day}$  based on data from Belgium. Assuming that all or most of these estimates reflect Me-Hg intake from fish/seafood, these estimates are in close agreement with the estimate of mean Me-Hg intake of  $3.2 \mu\text{g/day}$  presented here.

#### B. Comparison of Acceptable Intakes to Current Levels of Consumption

In Table IV-3 the maximum acceptable daily intake of Me-Hg for the current RfD and for the ADI for an in-utero endpoint are compared to the estimated percentiles of Me-Hg intake in the fish eating population. The fish/seafood consumption data employed in the Monte Carlo analysis did not differentiate by sex. The percentiles of Me-Hg intake in Table IV-3 for the 62 kg woman in Table IV-3 thus implicitly assume that women and men consume fish at the same rate. This is probably not entirely correct. The 1980-82 U.S. FDA Total Diet Study (Pennington 1983) indicates that 25-30 year old women consume fish/seafood at 62-95% the rate of men of the same age. Therefore, the percentile values for the 62 kg woman could reasonably be about 80% of the values given in Table IV-3.

Although fish/seafood appears to be the only significant source of Me-Hg exposure in the population, this calculation is not based on actual measurements of Me-Hg intake or body burden and is therefore only an indirect estimate of exposure and risk. Nonetheless, Table IV-3 suggests that a small percentage of the general population has Me-Hg intakes above the current RfD level and that a significant fraction of woman of childbearing age have intakes above an ADI based on the in-utero endpoint. Fitzgerald and Clarkson (1991) have also suggested that, based on an in-utero endpoint, a significant fraction of the population may be at risk from Me-Hg exposure. Instead of comparing the exposure to an RfD with its attendant uncertainty factors, exposure can be compared to the unadjusted estimate of the effective dose for in-utero developmental effects ( $43 \mu\text{g/day}$  for a 62 kg woman). Based on the results of the Monte Carlo simulation, about 0.5% of women of childbearing age in the total population have background exposures of Me-Hg which exceed this level. It should be noted however, that predictions from Monte Carlo distributions become increasingly uncertain in the extreme tails of the overall distribution.

This analysis is based on the consumption of marine fish. Some populations consume a much larger proportion of commercially or recreational caught freshwater fish. Data from such groups are

limited, but Clarkson (1990) has suggested that these populations may have a considerably higher Me-Hg exposure than the general fish eating population. Currently, it is not known to what extent there are sub-populations or individuals in New Jersey which may consume significant quantities of local freshwater fish. Anecdotal evidence, however, suggests that such individuals do exist.

### C. Acceptable Increase in Methylmercury Intake

The uncertainties in the toxicologic and exposure analyses presented here argue against viewing the foregoing as a precise estimate of health risk. However, given the apparent inadequacy of the current RfD to provide the anticipated level of protection against in-utero developmental effects and given current patterns of fish/seafood consumption, these analyses suggest that at current national levels of dietary exposure, a significant fraction of women of childbearing age may be exposed to Me-Hg at levels which pose an unacceptable risk of adverse developmental effects. Thus, basic public health principles require that nationally, there should be no significant increase in current population exposure to Me-Hg. Until New Jersey population-specific data on background levels of exposure become available, this conclusion should apply equally to New Jersey.

Table IV-1

Inputs to Monte Carlo Analysis of Me-Hg Exposure of  
Fish/Seafood Eaters

M:	minimum	= 0 g/d
	maximum	= 227 g/d (0.5 lb, 7 d/wk)
	mean	= 28 g/d
	90 %tile	= 53 g/d
CT:	minimum	= 0 $\mu\text{g/g}$
	maximum	= 1.0 $\mu\text{g/g}$
	52% of catch	= 0.05 $\mu\text{g/g}$ *
	39% of catch	= 0.15 $\mu\text{g/g}$
	5% of catch	= 0.25 $\mu\text{g/g}$
	2% of catch	= 0.35 $\mu\text{g/g}$
	0.7% of catch	= 0.55 $\mu\text{g/g}$
	0.1% of catch	= 0.75 $\mu\text{g/g}$
F:	mean	= 0.95
	s.d.	= 0.06
	(normal distribution truncated at 0.75 and 1.0)	
A:	0.9-1.0 (uniform distribution)	

\* concentration reported as  $<0.1 \mu\text{g/g}$

Table IV-2

Selected Percentiles of Estimated Me-Hg Intake  
in the Fish/Seafood Eating Population

mean = 3.8  $\mu\text{g}/\text{day}$

50% <1.7

75% <3.8

90% <8.8

95% <15.3

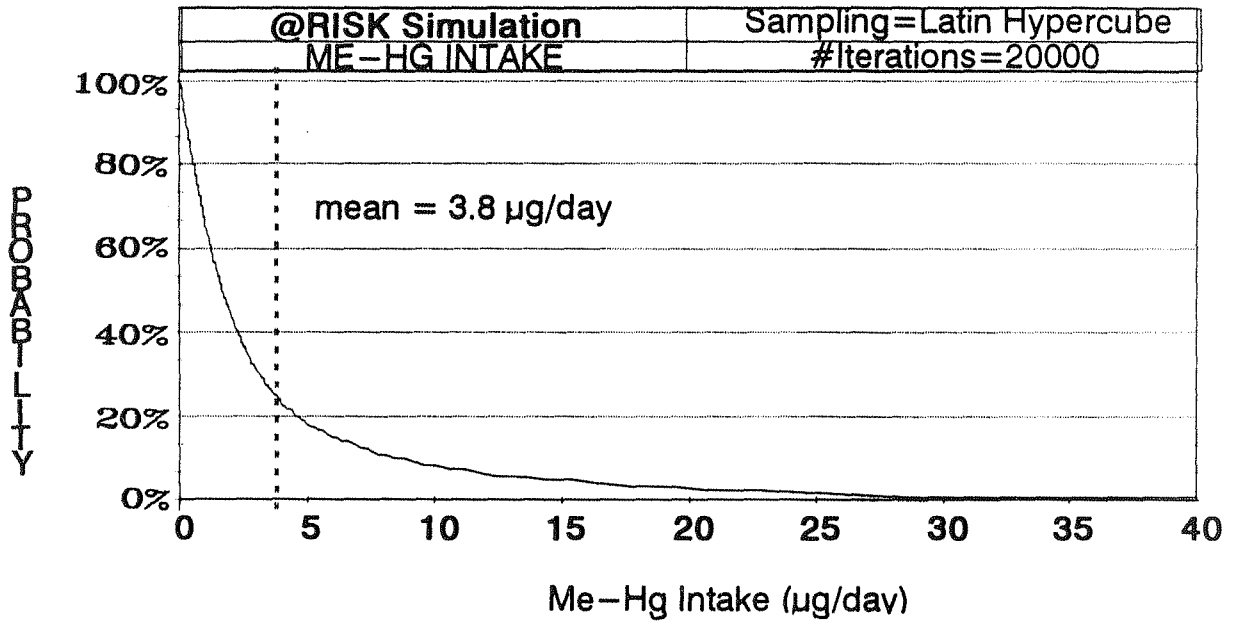
99% <33.1

Table IV-3

Maximum Intake of Me-Hg Based on Reference Dose Guidance and  
Relation to Estimated Current Intake

	<u>70 kg adult</u>		<u>62 kg woman</u>	
	Maximum Acceptable Intake ( $\mu\text{g}/\text{day}$ )	Percent of fish eating population with background intakes $\geq$ the recommended intake	Maximum Acceptable Intake ( $\mu\text{g}/\text{day}$ )	Percent of fish eating population with background intakes $\geq$ the maximum recommended intake
Current USEPA RfD based on paraesthesia: 0.3 $\mu\text{g}/\text{day}$	21	3%	19	4
ADI based on Developmental Endpoint 0.07 $\mu\text{g}/\text{kg}/\text{day}$ endpoint: 0.07 $\mu\text{g}/\text{day}$	--	--	4	23%

Figure IV-1: Me-Hg Intake from Fish/Seafood for Fish Eaters (ug/day)



## V. Inhalation Pathway

### A. USEPA Reference Concentration

USEPA has developed a reference concentration (RfC) for inhalation of elemental mercury. The RfC is defined as an estimate of a continuous inhalation exposure ( $\text{mg}/\text{m}^3$ ) to the human population (including sensitive subpopulations) that is likely to be without an appreciable risk of deleterious noncancer effects during a lifetime of exposure. USEPA's mercury RfC is  $3 \times 10^{-4}$  (0.0003)  $\text{mg}/\text{m}^3$ , or  $3 \times 10^{-1}$  (0.3)  $\mu\text{g}/\text{m}^3$  (USEPA 1992a). This is based on a Lowest Observed Adverse Effect Level (LOAEL) of  $0.009 \text{ mg}/\text{m}^3$ , which was determined from a number of human studies (Fawer et al. 1983; Piikivi 1989; Piikivi & Hanninen 1989; Piikivi & Tolonen 1989). To obtain the RfC, the LOAEL was divided by an uncertainty factor of 30: 10 to account for the most sensitive human population and the use of a LOAEL; and 3 for a lack of reproductive studies in the available database.

As was noted above, the RfC has been developed for elemental mercury only. An attempt was made by the RfC workgroup within USEPA to develop an RfC for mercuric chloride, but the data were found to be non-verifiable. Therefore, applying the existing RfC for elemental mercury to inorganic mercury exposures is quite tenuous. However, the comparison is often made for lack of any other benchmark. It is not appropriate to use this RfC for the evaluation of any organic forms of mercury.

### B. Comparison to Ambient Air Concentrations

To estimate the possibility of noncarcinogenic effects from exposure to toxic chemicals from a facility, the hazard index is calculated. For inhalation exposure, this is done by dividing the incremental ambient air concentration (that part contributed by the facility) by the reference concentration. If this is less than one, and the background concentration is negligible, then the ambient concentration is less than the RfC, and there should be no harmful incremental effect. If the hazard index exceeds one, there may or may not be a problem, depending on the magnitude of the hazard index, the accuracy and variability in the reported concentration, the toxic endpoints of the chemical's effect, and other factors.

As described in Chapter II, Hg concentrations measured in the ambient air have been reported to be  $<1$  to  $3 \text{ ng}/\text{m}^3$  in pristine areas and  $<1$  to  $9 \text{ ng}/\text{m}^3$  at other sites that could be considered to be background. Appendix A also summarizes mercury measurements taken in some urban areas which show concentrations ranging up to  $50 \text{ ng}/\text{m}^3$ . These concentrations, when compared to the RfC, result in

a Hazard Index  $\leq 0.01$  in pristine areas,  $\leq 0.03$  in other background areas, and  $\leq 0.2$  in urban areas.

It should be noted that most of the ambient air data that have been reported represent relatively short averaging times (one day to a few weeks). Annual average concentrations, which are more appropriate for comparison to the RfC, would be expected to be much lower than these reported values. The mercury data collected in the vicinity of the Warren County Municipal Solid Waste (MSW) Incinerator, for example, shows a maximum observed mercury concentration of 25 ng/m<sup>3</sup> although the median value of the reported concentrations at that site is 2.5 ng/m<sup>3</sup>. Using these two values results in a hazard index of 0.08 based on the maximum mercury concentration, as compared to a hazard index of 0.008 based on the median concentration.

When monitor data are not available, the RfC is often compared to ambient concentrations predicted by air dispersion models. This has been done for MSW Incinerators, for example, using the data reported in Appendix E. The hazard indices for inhalation from existing and permitted facilities are summarized in Table V-1. Since the RfC is derived on the basis of health effects resulting from long-term, chronic exposure, the calculation of a hazard index uses annual average predicted concentrations. Using modeled annual average ambient concentrations, the highest hazard index was 0.017 for the Warren County facility. The lowest hazard index 0.0023 for the Gloucester County facility.

Table V-1  
Inhalation Hazard Indices for Permitted NJ MSW Incinerators  
based on results of air quality modeling analyses

	<u>Predicted Annual Average Concentration</u> (ug/m <sup>3</sup> )	<u>Hazard Index</u>
Camden	0.005	0.0067
Essex	0.006	0.002
Ft. Dix	0.0032	0.01
Gloucester	0.007	0.0023
Union	0.0019	0.006
Warren	0.005	0.017

## VI. Ecological Effects

### A. Toxicity

Mercury is known to be acutely and chronically toxic to a wide range of wildlife. As is the case for humans, organomercury compounds, especially methylmercury, are much more toxic to wildlife than inorganic mercury compounds. This has been found for microorganisms, invertebrates, fish, birds, and mammals (Eisler 1987; WHO 1989).

Water concentrations of inorganic mercury in the 1 ppb range have adverse effects on sensitive aquatic organisms. For organic mercury, the threshold for sublethal toxic effects for the most sensitive parameters for fish embryos and larvae may be "well below 0.01" ppb (WHO 1989). Total mercury concentrations in water of 0.1 to 2 ppb are acutely toxic to sensitive aquatic species, and sublethal effects occur in the 0.03 to 0.1 ppb range (Eisler 1987).

Mercury in organic forms is acutely toxic to sensitive bird species at levels in their diet of 4 to 40 ppm; for inorganic mercury, the comparable dietary level appears to be at least 500 ppm. Organomercury compounds are acutely toxic to sensitive nonhuman mammals at dietary levels as low as 1 ppm (Eisler 1987).

Sublethal effects are seen in sensitive birds and nonhuman mammals when mercury levels in their diets reach 50 to 100 ppb and 1,100 ppb (fresh weight), respectively (Eisler 1987).

Eisler (1987) also summarized mercury levels in tissues of birds and mammals that have been associated with adverse effects. For birds, these levels were as low as 5,000 ppb in feathers, and 790 ppb in eggs. Residues of mercury in experimentally poisoned birds usually exceeded 20 ppm (fresh weight), and are usually similar to concentrations in wild birds that died of mercury poisoning. Tissue residues in kidney, blood, brain and hair in excess of 1,100 ppb in many nonhuman mammals are usually considered presumptive evidence of significant mercury contamination.

There are numerous limitations and uncertainties that affect any discussion of potential wildlife effects, including: inter-species variability in sensitivity to mercury; uncertainty and incomplete data regarding background exposure levels; the difficulty of determining adverse effects from mercury in the field due to the co-occurrence of other contaminants, such as PCBs and pesticides; and uncertainty regarding safe exposure levels for many species.

### B. Background Exposures

WHO (1989) reports the following typical water concentrations for dissolved mercury: open ocean - 0.5 to 3 parts per trillion

(ppt); coastal sea water - 2 to 15 ppt; rivers and lakes - 1 to 3 ppt. WHO (1989) also reports that mercury levels in uncontaminated sediments are similar to those in unpolluted soils (20 to a few hundred ppb).

#### C. Water Quality Criteria

The current USEPA (1985) water quality criteria for mercury are summarized in Table VI-1. Also presented are the criteria that were previously in effect (USEPA 1980).

Eisler (1987) sharply criticized the new criteria, arguing that the freshwater four-day criterion provides safety factors as low as 8 and 2 for protection against acute and sublethal effects, respectively; the freshwater one-hour criterion provides "essentially no significant protection" against either acute (safety factor as low as 0.04) or sublethal (safety factor as low as 0.01) effects. He leveled similar criticism against the 1985 saltwater criteria.

Eisler (1987) found the old freshwater criteria satisfactorily protective, but found the old, less stringent saltwater criteria less protective.

#### D. Sediment Effects-Based Guidance Levels

While there are no enforceable standards for the level of mercury in sediments, the National Oceanic and Atmospheric Administration (NOAA) (Long & Morgan 1990) has derived effects-based guidance levels for a large number of organic contaminants and metals, including mercury. The results of sediment toxicity tests were ordered (for the lowest sediment concentrations that had been shown to cause some adverse effects to the highest such concentration) and used to develop two guidance levels:

- Effects Range - Low (ER-L), defined as the lowest 10th percentile concentration and described as the sediment concentration above which adverse effects may begin or are predicted among sensitive life stages and/or species as determined in sublethal tests; and
- Effects Range - Median (ER-M), defined as the 50th percentile concentration and described as the concentration above which effects were frequently or always observed or predicted among most species.

The NOAA effects-based guidance values for levels of mercury in sediment are:

Effects Range - Low (ER-L):	0.15 ppm
Effects Range - Median (ER-M):	1.3 ppm

TABLE VI-1

USEPA Water Quality Criteria for Mercury  
(1  $\mu\text{g/L}$  = 1 ppb)Freshwater Aquatic Life

	<u>Acute</u>	<u>Chronic</u>
1985	2.4 $\mu\text{g/L}$ (hourly average)	0.012 $\mu\text{g/L}$ (four-day average)
1980*	0.0017 $\mu\text{g/L}$ (maximum permissible)	0.00057 $\mu\text{g/L}$ (24-hour average)

Saltwater Aquatic Life

	<u>Acute</u>	<u>Chronic</u>
1985	2.1 $\mu\text{g/L}$ (hourly average)	0.025 $\mu\text{g/L}$ (four-day average)
1980*	3.7 $\mu\text{g/L}$ (maximum permissible)	0.025 $\mu\text{g/L}$ (24-hour average)

\* 1980 criteria are for total recoverable mercury.

Although the NOAA values were derived primarily from data on marine organisms, some freshwater toxicity data were also incorporated, and the guidance levels may be used, albeit with caution, for freshwater sediments. In fact, the U.S. Fish and Wildlife Service (USFWS 1991a,d) has used them in assessments of contamination at several wildlife refuges in New Jersey, and has argued (USFWS 1991a) that biological effects would be expected to be more severe in freshwater environments than in marine environments at a given level of contamination.

#### E. Wildlife Effects in the Field

A number of researchers have documented instances of wildlife poisoning by mercury or mercury compounds. Eisler (1987) reviewed several of these cases. Das et al. (1982) reported the poisoning of game birds and other wildlife in Sweden, apparently by seeds treated with organomercurials, which was noticed in 1960. Mercury was also cited as a contributing factor in massive kills of grey heron (*Ardea cinerea*) in the Netherlands in 1976 (Van der Molen et al. 1982).

Ensor et al. (1992) collected 221 common loons in northern and central Minnesota between 1984 and 1990, examining mercury levels in the feathers of 93 live birds and in the feathers and livers of 128 birds found dead or dying. They found mercury residues in all feather and liver samples, with 22 percent of the livers analyzed having mercury concentrations equal to or in excess of the level associated with reproductive impairment in loons. Levels in the tissues of several loons were within the range known to be lethal or hazardous to other birds. Several necropsied loons had lesions and elevated mercury levels associated with mercury poisoning in other birds. Based on these findings, the researchers concluded that "mercury contamination may threaten loon populations in Minnesota."

**PART 3**

**FATE AND TRANSPORT MODELS**

## VII. Dispersion, Transport and Fate of Environmental Mercury

### A. Overview of the Process

Mercury emitted into the air has the ability to move through the environment and may result in population exposure through many different media. Figure VII-1 illustrates the possible dispersion, transport, fate and multiple routes of exposure for environmental pollutants in general. In the case of mercury, the process begins with emissions from a variety of sources, including incinerators, coal combustion, certain manufacturing operations, and natural processes such as volcanic eruptions. The mercury is dispersed through the air where it may be inhaled. It can also be deposited on plants, soils, and water bodies. This deposition may be either dry or wet (i.e. through precipitation). The type of deposition depends somewhat on the form of mercury that is emitted from the source and the chemical transformations that may take place in the atmosphere. Mercury deposited on soil may be available for direct ingestion by humans, may be taken up by plants, or may be washed out into neighboring water bodies (i.e. runoff).

Once mercury has entered a waterbody, either through direct deposition or through runoff, some of the mercury will be converted to methylmercury which then enters the foodchain through various aquatic biota. Eventually it works its way up to fish where it tends to accumulate since methylmercury is not readily excreted by fish. These fish may be caught and eaten, thus completing the exposure pathway to humans.

Each of these steps is described briefly below and discussed more thoroughly in Chapters VIII, IX and X. Then three possible approaches to modeling the fate and transport of mercury are described. Two of these models use a site-specific approach, while the third uses a generic approach.

Site-specific models address the impacts of an actual facility, using detailed information about the pollution sources, the layout of the facility, and the surrounding area. Where pieces of this information are lacking, assumptions are made about their value which tend to overestimate the impact of the facility. The more that is known about the source and its surroundings, the closer the estimated impact should be to reality. The two site-specific models described in this report were prepared to estimate the impact of existing MSW Incinerators - one in Minnesota and the other in New Jersey. These models provide a snapshot of the possible impact of MSW Incinerators on public exposure to mercury.

In the absence of site-specific information, a generic model may be used to estimate the possible impact of a source or group of sources. This type of model is useful for describing the impact of sources which do not exist, but have the potential to be built. Such a model could also be used to generalize impacts when existing

sources are too numerous to model or when insufficient data exist to use a site-specific model. The generic model developed in this report could be used to describe a worst-case scenario so that the Department can judge whether regulations under development would provide adequate protection in essentially all possible cases. This generic model can also be easily adapted for use with source categories other than MSW Incinerators.

## B. Basic Steps

### 1. Mercury Emissions and Dispersion

Since the process begins with emissions to the air, the first step in the analysis is to characterize those emissions. Ideally, one would know the amount of mercury that is emitted by the source of interest in any given year. Without continuous emissions monitoring, however, such actual emissions data are generally not known. As a surrogate for this information, stack test data or allowable emission rates (as described in a permit) are normally used. The species of mercury that are emitted and the amount of each species is also valuable information, although it is not generally available.

Other information about the source is also necessary in order to predict how the mercury will disperse in the air once it is emitted. Such information includes the height of the stack, temperature and volume of the stack gas, and the velocity of the gas as it is emitted. These data, combined with meteorological data from a nearby weather station, can be entered into a dispersion model and used to estimate the ambient air concentrations of mercury in the vicinity of the source.

### 2. Wet and Dry Deposition

Deposition is the process by which mercury moves from the air to the soil, and also directly and indirectly to the waterbody in which it may accumulate in fish. Deposition of mercury can take place either by precipitation (wet deposition) or by direct impact or settling on the surface (dry deposition). Both wet and dry deposition must be considered over both the waterbody of interest and its adjacent watershed.

The efficiency of deposition depends on the chemical form of mercury in the air, since solubility, volatility, and reactivity characteristics will affect the wet and dry deposition rates. The amount of precipitation in the area is also a critical parameter. The magnitude of the deposition is directly related to the concentration of mercury in ambient air. The size of the waterbody and of the watershed are also important factors in determining total deposition.

### 3. Runoff and Accumulation in Water Bodies

Mercury deposited onto a watershed can be transported to a waterbody via two processes: runoff of mercury-contaminated soil, and runoff of dissolved mercury in the rainwater. Soil loss is a function of rainfall rate, soil erodability, slope of the terrain, vegetative cover, land use, and sediment delivery. Solubility and the degree to which the mercury is bound to soil particles will influence the amount of mercury dissolved in rainwater.

Once it enters the waterbody, some mercury will accumulate in the sediments. Some mercury will be removed with water flowing out of the waterbody or may evaporate back into the air. Some mercury will be taken up by organisms in the food web. The remainder of the mercury will be dissolved in the water column.

### 4. Accumulation in Fish

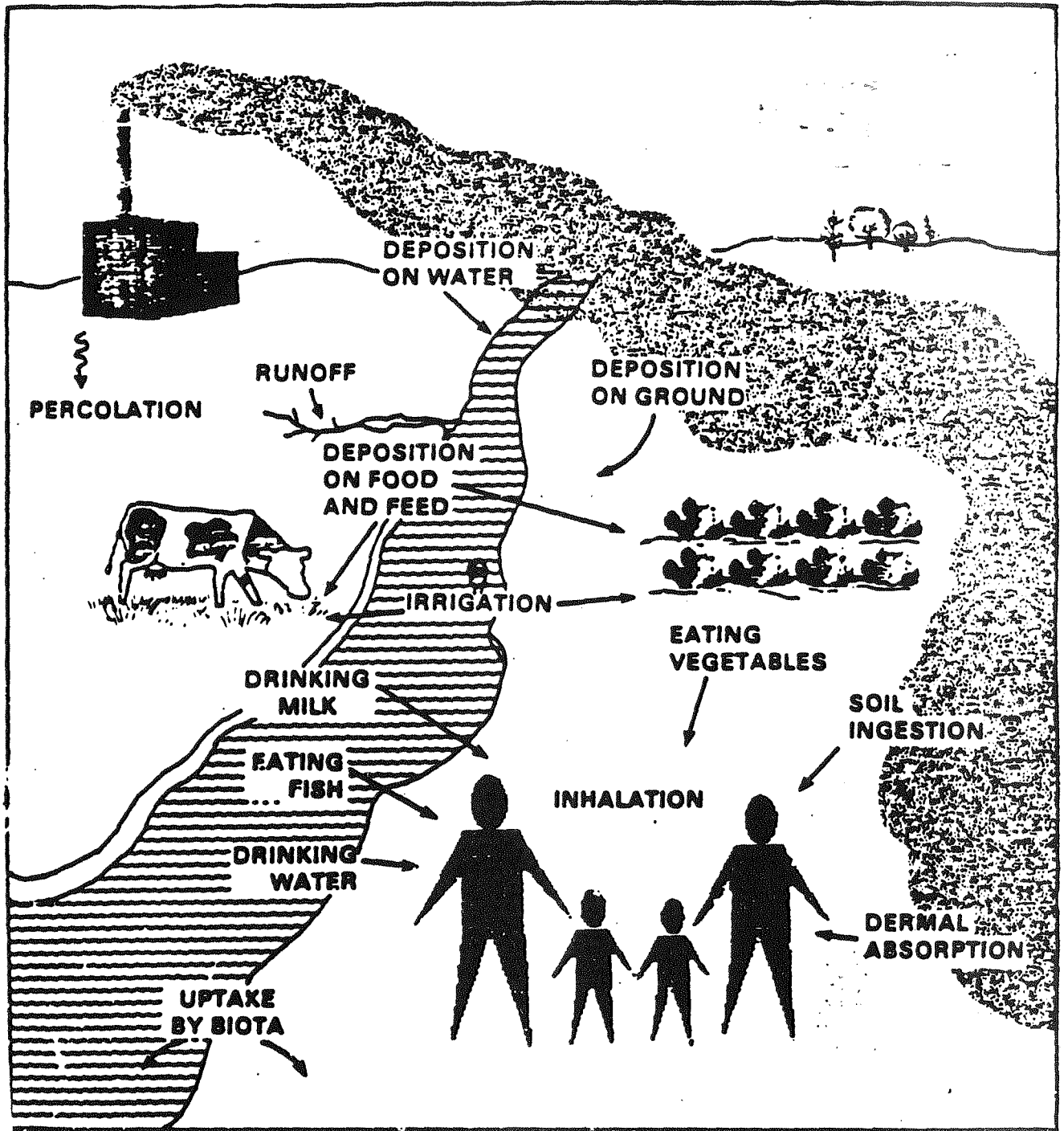
Bioconcentration factors (the ratio of mercury concentration in fish tissue and mercury concentration in water or sediments) are used to summarize all the physical processes which occur between mercury deposition in the waterbody and mercury accumulation in fish. These include methylation of mercury and mercury uptake through the food chain.

While both inorganic and organic forms of mercury can be taken up and concentrated by aquatic organisms, the bioavailability of methylmercury is much greater than that of inorganic mercury, and therefore is the focus of discussion when predicting accumulation of mercury in fish.

### 5. Fish/Mercury Consumption

Models which account for mercury deposition, runoff and build-up in waterbodies, and ultimate accumulation in fish can be used to predict an increase in the concentration of methylmercury in the tissue of fish living in those water bodies. These estimates, combined with the amount of fish that may be ingested by a sensitive individual, can be used to predict the incremental ingestion of mercury that could result from eating locally-caught fish.

Figure VII-1  
Dispersion, Transport and Fate of Environmental Pollutants



## VIII. Deposition and Runoff

### A. Deposition

Deposition of mercury from the atmosphere to land or water surfaces can take place either by precipitation (wet deposition) or by direct impact or settling on the surface (dry deposition). Atmospheric mercury can exist in a number of physical and chemical forms. Each form will have solubility, volatility, and reactivity characteristics that affect its wet and dry deposition rates. There are currently no generally accepted methods for modeling wet and dry deposition of mercury, although several approaches have been suggested; each with its own strengths and weaknesses.

Due to its high vapor pressure and elevated stack temperatures, almost all mercury at the boiler outlet is in its vapor state. Cooling the flue gas enhances the adsorption of the reactive  $\text{HgCl}_2$  onto fly ash. There is some controversy concerning how much  $\text{HgCl}_2$  is adsorbed on particles and the temperature at which this occurs. Bergstrom (1986) stated that mercury in the flue gas from waste incineration is mainly present in the vapor phase at temperatures down to  $140^\circ\text{C}$ . He found that if the flue gas is above the dew point of the system, temperature reduction does not improve the attachment of mercury to particles. Otani et al. (1986) found that at  $20^\circ\text{C}$  the total amount of mercury contained on particles generated by sludge incineration is small compared to the total mercury in the exhaust gas, and that most mercury behaves as a vapor even in the presence of particulate matter. Lindberg (1987) stated that mercury released from both high and low temperature processes enters the atmosphere primarily as vapor. Conversely, Vogg et al. (1986) claimed that adsorption of  $\text{HgCl}_2$  on fly ash is a function of temperature, and that at temperatures of  $150^\circ\text{C}$  most  $\text{HgCl}_2$  is adsorbed onto the fly ash.

#### 1. Wet Deposition of Mercury

Deposition by precipitation is a major removal process for atmospheric mercury (Lindqvist & Rodhe 1985). Soluble forms of mercury such as  $\text{HgCl}_2$ , particulate mercury and divalent inorganic mercury ( $\text{Hg}^{++}$ ) will be removed by precipitation; insoluble, volatile forms such as  $\text{Hg}^0$  and dimethylmercury are likely to remain in the atmosphere, although some recent studies (for example, Bloom & Watros 1989) have detected very small amounts of methylmercury in rain and snow.

Although there is no generally accepted model for wet deposition, one method of modeling wet removal of mercury is by using a washout ratio (WR) approach. The washout ratio represents the concentration of mercury in the precipitation divided by its concentration in air. Use of a washout ratio is an accepted and often used method of approximating wet deposition. At the 1992

USEPA/State Modelers Workshop, it was recommended that a wet deposition algorithm using washout ratios be added to the most commonly used USEPA dispersion models. Using a washout ratio, annual wet deposition (WDEP) can be estimated with the following equation:

$$WDEP = (C_{air}) (WR) (J_o) (PP_h)$$

where:

$C_{air}$  = soluble mercury concentration in air ( $\mu\text{g}/\text{m}^3$ )  
WR = washout ratio for soluble mercury  
 $J_o$  = annual average rainfall rate (m/hr)  
 $PP_h$  = hours of precipitation per year

Measured concentrations of mercury in rainwater have been used to approximate the value WR. A limited data base of such measured values exists. Using measurements principally taken in Europe, Lindqvist and Rodhe (1985) estimated WR to be approximately  $10^5$  for soluble mercury and  $10^6$  for particulate mercury. Glass et al. (1991) measured the mercury content of rainfall in Minnesota and estimated the average scavenging ratio to be  $140 \pm 80$  (at 1 mm/hr precipitation rate). This can be converted to a washout ratio for soluble mercury of  $1.17 \times 10^5$  (plus or minus  $0.67 \times 10^5$ ).

There is uncertainty in the value of the washout ratio (WR) for mercury. The Glass et al. (1991) study suggested that the actual WR value could be 57 percent higher or lower than the average found in that study. WR will vary with precipitation rate, vertical distribution of mercury, and precipitation size distribution among other factors. Slinn (1984) states that long-term average values for WR for a particular location may be accurate within a factor of 2 to 4 provided the data base for a particular pollutant is extensive. Given the small size of the wet deposition data base for mercury, it is expected that the true uncertainty is something greater than a factor of 4.

Finding an appropriate value for the air concentration ( $C_{air}$ ) in the equation can be difficult for site-specific analysis and is extremely problematic in the development of a generic model. The value selected for  $C_{air}$  should represent the average mercury concentration over the watershed during precipitation events. The dispersion models ordinarily used to predict ambient air concentrations that could result from stack emissions of a pollutant generally give values at discrete receptors, rather than average concentrations over a broad area. Getting the average concentration over the watershed would require additional programming or data analysis.

One option sometimes chosen is to use the maximum predicted air concentration over the watershed and to assume that concentrations are this high throughout the watershed. This is

indeed a conservative assumption since the actual concentrations over the watershed could vary one or more orders of magnitude, depending on its size and on the distance from the source of the emissions. In those cases (such as a generic model) where the actual location of the waterbody and its watershed is not known, the maximum ground level concentration anywhere in the source impact area may be used for  $C_{air}$ , although this approach may vastly overestimate wet deposition.

Another element of uncertainty in the estimate of  $C_{air}$  is the fact that groundlevel pollutant concentrations are generally chosen to represent it. Since wet deposition is more accurately represented by the above-ground vertical distribution of mercury within the plume, use of the ground-level concentration is a source of uncertainty in the equation. The equation is most applicable in the far field where vertical mixing has taken place. For the near field case some corrections should be made because of the limited plume cross section relative to the total mixing depth. In fact, there is empirical evidence that wet deposition of mercury near the stack is very significant (Greenberg, et al. 1992).

Values for the  $J_0$  and  $PP_h$  terms in the wet deposition equation can be obtained by examining National Weather Service data from nearby airports. Data should be taken from a site that is likely to have rainfall patterns similar to the area that is being modeled. When modeling a source which will be operating for many years, climatological precipitation data are preferred. These data provide a statistical description of precipitation events over 20 or more years.

## 2. Dry Deposition of Mercury

Little direct information is available concerning mercury dry deposition rates. Deposition velocity is a function of terrain, biotic surfaces, and canopy structure among other factors. It also varies among the different species of mercury. Insoluble forms of mercury, such as  $Hg^0$ , most likely have negligible deposition rates. Because of its volatility, some deposited mercury may be re-emitted before it is incorporated into the biosphere. It is uncertain how much this mechanism may reduce the overall deposition of mercury species.

The following equation can be used to estimate the annual amount of mercury dry deposition (DDEP):

$$DDEP = (DV) (C_{air}) (Q)$$

where:

DV = dry deposition velocity of mercury (cm/sec)

$C_{air}$  = mercury concentration in air ( $\mu g/m^3$ )

Q = fraction of year during which dry deposition is occurring

Most dry deposition estimates are based on the characteristics of particulate matter, most notably the settling velocity associated with particles of different sizes. Since the fraction of mercury in the air that is in the particulate form is expected to be negligible, this approach is not very useful in estimating dry deposition of mercury. Hanna et al. (1982) describe possible mechanisms for very small particles and gases which are more representative of the forms of mercury that are expected in the air. In this case dry deposition occurs as a result of turbulent diffusion and Brownian motion rather than by settling. Chemical absorption, impaction and other biological, chemical and physical processes then cause the material to be retained at the surface of the material. They report deposition velocities from the literature that range from 0.0001 cm/sec (for relatively inert gases such as carbon monoxide) to 0.8 cm/sec (for relatively reactive gases such as sulfur dioxide).

Global data can be examined to begin to identify the range of dry deposition values that might be expected. Dry deposition has been estimated by Lindqvist (1991) to average about 2.5 to 5 ug/m<sup>2</sup>/yr and to increase to about 10 ug/m<sup>2</sup>/yr in industrial areas. Background concentrations of total mercury are reported to be 1 to 9 ng/m<sup>3</sup> (see Section II.A), while mean concentrations in suburban and industrial areas range from 3 to 22 ng/m<sup>3</sup> (see Appendix A). Using these data points and assuming that the fraction of the year during which dry deposition can occur, Q, is about 0.90, the equation above can be used to estimate a value for dry deposition velocity, DV, which ranges from 0.002 to 0.01 cm/sec (see Table VII-1). This is within the range reported by Hanna, et al.

Another study by Lindberg et al. (1991) measured mercury deposition in a forested watershed. The deposition rate in this case was found to be 0.06 cm/sec. All of the deposition velocity discussed here must be applied to total mercury since there are no data available for particular species.

The form of the mercury as it leaves an emission point will influence the magnitude of dry deposition. The calculations made in the previous paragraph are based on total mercury air concentrations and deposition ranges. Not enough information is available to make species-specific estimates.

Another complicating factor in the calculation of dry deposition is the variation related to the spatial distribution of mercury in the air. This is particularly important when attempting to estimate deposition in the vicinity of a specific emission point. Since the average and maximum air concentration around such a source can vary be a factor of 10 to 100 or more, use of the

maximum concentration could greatly overestimate the magnitude of dry deposition at a nearby lake or watershed.

Table VIII-1  
Estimates of Dry Deposition Velocity Using Global Data

Dry Deposition Rate ( $\mu\text{g}/\text{m}^2/\text{yr}$ )	Air Concentration ( $\text{ng}/\text{m}^3$ )	Deposition Velocity ( $\text{cm}/\text{sec}$ )	Comments
2.5	1	0.009	Background Data Low Values
5	9	0.002	Background Data High Values
10	3	0.01	Urban/Industrial Data Low Concentrations
10	22	0.002	Urban/Industrial Data High Concentrations

### 3. Mercury Emissions from Municipal Solid Waste Incinerators (MSW Incinerators)

Most MSW incinerators in New Jersey use dry scrubbers, and therefore the flue gas is always above the dew point of the system. Boiler outlet temperatures will be approximately  $260^{\circ}\text{C}$ , and stack gas exit temperatures range from  $116^{\circ}\text{C}$  to  $141^{\circ}\text{C}$  at these facilities. Therefore, some of the  $\text{HgCl}_2$  may be adsorbed onto the fly ash, but it is reasonable to assume most will stay in the vapor state. Because of its solubility, this form is ideal for removal by rainfall.

The mercury in flue gas released from MSW incinerators has been reported to be predominantly oxidized mercuric chloride ( $\text{HgCl}_2$ ) and elemental mercury ( $\text{Hg}^0$ ), with  $\text{HgCl}_2$  representing approximately 70 percent and  $\text{Hg}^0$  7 percent of the total mercury (Bergstrom 1986). Vogg et al. (1986) measured similar values when analyzing flue gas from MSW incinerators. Other researchers have concluded that even in cases where elemental mercury is the predominant species from the flue gas (i.e. in cases where flue gas cleaning systems have been installed), reactions in the immediate area near the stack, of  $\text{Hg}^0$  with ozone and with other stack-emitted constituents (i.e.  $\text{Cl}^-$ ,  $\text{NO}_x$ ,  $\text{SO}_x$ ) lead to the formation of water-soluble forms (Schroeder et al. 1991; Hall et al. 1990; Munthe 1992; Petessen & Iverfeldt 1992).

Tests performed at Ogden-Martin's Stanislaus facility (USEPA 1992b) show that 34% of the total mercury is gaseous elemental Hg<sup>0</sup> on a time-weighted average basis. Due to the low water solubility of elemental mercury and the fact that it is in vapor rather than solid form, it could be estimated that about one-third of the total mercury emitted into the air by MSW incinerators will not be available for deposition in the local area.

## B. Runoff

Runoff of mercury from the watershed to an adjacent waterbody could make a significant contribution to the overall mercury content of the waterbody. Swain et al. (1992) cite their own research and the research of others who estimate that anywhere from 8% to 26% of the mercury deposited in a watershed is transported to the waterbody. Runoff of mercury from soils in the watershed is often described using the Universal Soil Loss Equation, developed by the U.S. Department of Agriculture. This equation describes the rate at which soil in the watershed is removed by erosion over a period of time.

$$X_e = (R) (K) (LS) (C) (P_s) (D)$$

X<sub>e</sub> = soil loss rate per unit area watershed over time (g/m<sup>2</sup>-yr)

R = rainfall erosion index (yr<sup>-1</sup>)

K = soil erodability factor (tons/acre)

LS = topographic or slope length factor

C = cover and management factor

P<sub>s</sub> = supporting practice factor

D = sediment delivery factor

The soil loss is a function of rainfall rate, soil erodability, slope of the terrain, vegetative cover, land use, and sediment delivery. Whether the Universal Soil Loss Equation is an appropriate method for estimating runoff is a matter of some debate within the scientific community. For example, it is not known whether mercury bound to the soil particles that runoff is in a form that will be bioavailable after it enters the waterbody. It might be preferable to focus on runoff of mercury dissolved in the water that drains from the watershed. This form of mercury may be more bioavailable than that bound to particles.

Another unknown in urban and some suburban areas is whether the runoff ever reaches the waterbody that is being studied. Storm drains and other management practices may divert runoff to a municipal treatment plant or to a totally different waterbody. Alternatively, some portion of the mercury which is deposited on impervious man-made surface, could be dissolved in rainwater and be

carried in this runoff to some waterbody either directly or through storm drains.

### C. Accumulation in Water Bodies

Knowing the runoff and the direct deposition of mercury leads to an estimate of the amount of additional mercury deposited in the waterbody. It could be crudely assumed that this is spread uniformly along the bottom of the lake and that it is mixed in the top layer of sediment. Alternatively, the mercury could be uniformly distributed in the lake water itself.

Several models are being developed which can use specific characteristics of the lake to more precisely calculate the concentration of various mercury species in sediments and in the water column (see, for example, Harris & Snodgrass 1992, Hudson et al. 1992, and Diamond & Mackey 1992). For example, mercury input to streams may be quickly flushed away compared to low flow drainage or seepage lakes. Therefore, water flow characteristics are critical to predicting mercury concentrations in the water column. If lake-specific data are available, these models would be preferable to the simple approach described in the previous paragraph. However, obtaining values for the extensive set of parameters upon which these models are based is usually quite difficult.

## IX. Trophic Uptake

Many researchers have examined mercury levels in fish and aquatic organisms. Some of this work is described in Section II.E and summarized in Table A-5. This chapter contains a brief overview of the bioavailability of mercury to fish.

### A. Bioavailability

While both inorganic and organic forms of mercury can be taken up and concentrated by aquatic organisms, the bioavailability of methylmercury is much greater than that of inorganic mercury (USEPA 1985; WHO 1989). Fish can accumulate mercury in their tissues to levels at least four orders of magnitude greater than levels found in the surrounding water. This is thought to reflect primarily food chain transfer (biomagnification), not simple partitioning between the two phases (WHO 1990). Besides accumulating methylmercury more readily, fish eliminate methylmercury more slowly from their bodies than they do inorganic mercury (WHO 1989). The relative uptake and elimination rates of the organic and inorganic forms mean that most mercury in fish tissue consists of methylmercury (80%, according to Eisler (1987); also see section IX.B.). The following twenty-two states have issued consumption advisories for fish and/or shellfish due to mercury contamination (Cunningham & Zeitlin 1990; NMDOH/NMED 1991):

California	New York
Colorado	North Carolina
Connecticut	North Dakota
Florida	Oregon
Georgia	South Carolina
Massachusetts	South Dakota
Michigan	Tennessee
Minnesota	Texas
Montana	Virginia
Nevada	Washington
New Mexico	Wisconsin

### B. Effects of Lake Parameters on Trophic Uptake

#### 1. Direct Effects on Bioavailability

Knowledge of mercury loadings, or even mercury levels in sediments in a given lake, is not sufficient to predict levels in fish. Numerous physical, chemical, and biological factors affect the availability and uptake of mercury.

USEPA sometimes establishes water quality criteria that vary with some other parameter, such as hardness. However, with respect to mercury, USEPA (1985) concluded "Numerous [physical and chemical] factors . . . probably affect the acute and chronic toxicity and bioaccumulation of various forms of mercury. . . .

Available data do not, however, show that quantitative relationships are consistent enough for a variety of aquatic species to enable relating water quality criteria to any of these variables." (emphasis added; references omitted) Nevertheless, the effects of a number of individual parameters on mercury bioavailability are well known.

The position in the food web and age of fish strongly affect mercury levels in tissues, with top predators and larger (and presumably older) fish exhibiting higher levels. This reflects the gradual uptake and slow elimination of mercury from the fishes' bodies.

Uptake and accumulation of methylmercury by fish is also enhanced by low pH, which favors both direct uptake through the gills and dietary uptake due to increased accumulation by organisms at lower trophic levels (WHO 1990). The availability of mercury for uptake and/or methylation by aquatic organisms is reduced by increasing salinity ( $Hg^{++}$  complexes with chloride ions), sulfide ions (which combine with  $Hg^{++}$  to form highly insoluble mercuric sulfide), and selenium (which forms even less soluble mercury selenide) (Moore & Ramamoorthy 1984; WHO 1990). Lindqvist et al. (1991) noted negative correlations between both pH and conductivity and mercury levels in pike in Swedish lakes.

Håkanson (1980) noted that mercury levels in fish (pike) are lower in lakes with high levels of bioproduction (i.e. more eutrophic lakes) than in less productive lakes. According to Håkanson, the large amounts of particulate organic matter in the water columns of these lakes reduce the availability of mercury for uptake by scavenging it from the water column. The binding constant between mercury and a given sediment is strongly related to the organic content of the sediment (Moore & Ramamoorthy 1984). Håkanson also found that lower pH levels increased mercury levels in pike.

The other factors that affect uptake and accumulation of mercury do so indirectly by affecting rates at which inorganic mercury is methylated.

## 2. Effects on Methylation Rates

Because of its greater toxicity (Chapter III) and bioavailability in comparison with inorganic mercury, greater attention has been paid to the behavior of methylmercury in aquatic systems, and to the effects of water quality conditions on methylmercury and on the processes that produce it.

Most if not all methylmercury in the environment arises from the methylation of inorganic mercury. Methylation can take place via biological (microbial, both enzymatic and non-enzymatic) or

non-biological processes. It occurs mainly in sediments, although it has also been shown to occur in the water column and in bacteria associated with the gills, outer slime, intestinal contents, and perhaps livers of fish. Once released into the water column, methylmercury enters the food chain via rapid diffusion and tight binding to proteins (Moore & Ramamoorthy 1984; WHO 1989, 1990).

Chemical and physical conditions greatly affect methylation rates in a given aquatic system. WHO (1989) noted that biological methylation is enhanced by oxidizing conditions (as opposed to anaerobic conditions); by higher temperatures (which increase microbial activity); and by higher microbial growth rates. The addition of sulfide to the system inhibits methylation by reducing the bioavailability of inorganic mercury. High levels of organic or humic material in sediments also enhance methylation by enhancing microbial activity.

Methylation rates (and, by extension, mercury levels in fish) are often higher in recently created waterbodies, such as reservoirs or impoundments. The relatively high levels of organic matter in the sediments (which had been soils) are thought to enhance microbial activity and hence methylation in the newly flooded areas. This enhancement of methylation can last several decades. The effect is especially pronounced in new lakes that have low pH and are oligotrophic (i.e. have low levels of bioproduction) (Eisler 1987; WHO 1989). Older reservoirs may also develop reducing conditions in the sediments, which would enhance binding of mercury to sulfur compounds in the sediments and thus reduce its bioavailability (Moore & Ramamoorthy 1984).

### C. Water-to-Fish Bioconcentration Factors

The aquatic chemistry of mercury, including the role of methylation processes in enhancing its bioavailability and the importance of food chain transfer, is complex. Thus bioconcentration factors (BCFs: tissue concentration divided by water concentration) determined through traditional laboratory studies must be used with caution. Still, bioconcentration factors provide a quantitative indication of the relative bioavailability of different forms of mercury. The following discussion focuses on freshwater species and systems.

#### 1. Laboratory Studies

A World Health Organization (WHO 1989) review showed that laboratory studies have yielded bioconcentration factors for inorganic mercury for various aquatic biota as high as 1,000 to 5,000 (WHO 1989). USEPA (1985), in its water quality criteria document, considered only studies in which water concentrations were adequately measured and tissue levels reached steady-state, or the study lasted more than 27 days. Only two studies on freshwater species met these criteria: a study by Snarski and Olson (1982)

which yielded a BCF (whole body) of 4994 for the fathead minnow, and a study by Boudou & Ribeyre (1984) which yielded a BCF (whole body) of 1,800 for rainbow trout. The compilation by USEPA and the Army Corps of Engineers (USEPA/ACOE 1990) in their manual on evaluating dredged material gives a lower value for mercury (BCF = 100) and a value in the same range for mercuric acetate (BCF = 3100). However, because these values are to be used in evaluations for ocean disposal of dredged material, and are adapted from a guidance document concerning ocean outfalls (Tetra Tech, Inc. 1986), they may cover only marine species. For reasons explained in Section IX.B.1, the bioavailability of inorganic mercury decreases with increasing salinity.

Laboratory-derived BCFs for organic mercury are much higher. Studies cited by USEPA (1985) reported BCFs in the following ranges: 11,000 to 33,000 (muscle) and 10,000 to 23,000 (whole body) for brook trout (McKim et al. 1976); 44,130 to 81,670 (whole body) for fathead minnow (Olson et al. 1975); 11,000 (whole body) for rainbow trout (Boudou & Ribeyre 1984); and up to 85,700 (whole body) for rainbow trout exposed via both water and food (Niimi & Lowe-Jinde 1984). Values listed by USEPA and the Army Corps of Engineers (USEPA/ACOE 1990), which may be for marine species, are in the same range (BCF = 3100 for phenylmercury and for methylmercury).

The study by Olson et al. (1975) is particularly interesting. Unlike the trout in the study of McKim et al. (1976), the fathead minnows studied by Olson et al. are browsers and probably fed on plants growing in the aquaria as well as introduced food. Thus, the higher BCFs observed in that study may more closely reflect field conditions. In general, BCFs for fish seem to increase with increasing temperature and with decreasing water concentrations, and do not differ systematically between whole-body and muscle tissue (USEPA 1980, 1985).

## 2. Field Studies

As a check on the applicability of laboratory-derived values to field situations, the results of Sorenson et al. (1989), who studied mercury concentrations in 80 Minnesota lakes, were examined. While the published results did not include concentrations for individual lakes, the earlier report did include such data, as well as an equation relating water column concentrations to concentrations found in standard-size (55 cm) northern pike:

$$\frac{\text{ng Hg}}{\text{g Pike (wet wt.)}} = 118 \left( \frac{\text{ng Hg}}{\text{l water}} \right) + 161$$

Evaluating the equation for the range of water column concentrations found in the study provides a basis for rough

calculations of "real-world" bioconcentration factors, as illustrated in Table IX-1.

Table IX-1: Bioconcentration Factors as a Function of Mercury Concentration in Water and Fish

<u>Water Concentration</u> (ng/l)	<u>Fish Concentration</u> (ng/g, wet weight)*	<u>Bioconcentration</u> <u>Factor</u>
1	279	279,000
2	397	198,500
3	515	171,667
4	633	158,250
5	751	150,200
6	869	144,833
7	987	141,000

\* Based on empirical equation derived by Sorensen et al. (1989).

## D. Sediment-to-Fish Bioconcentration Factors

### 1. Rationale

The multiple interactions of biota and physical and chemical conditions in the fate, transport, and transformation of mercury in aquatic environments make water column concentrations of dissolved mercury a somewhat unreliable predictor of concentrations in fish. At least in water bodies that have reached steady-state conditions with respect to mercury inputs and physical/chemical conditions, concentrations of mercury in the surface layer of a lake's bottom sediments may be a better predictor of equilibrium values in fish. This is due to what Håkanson (1980) called the "time-stability" provided by sediment values, in that sediments integrate levels of mercury in the waterbody over time.

### 2. Swedish Lake Studies

Regression equations derived by Håkanson (1980) and Anderson et al. (1987) (the latter are also described by Lindqvist et al. (1991)), relating sediment mercury levels and various water quality parameters to levels in fish (pike), provide a basis for calculating a range of possible sediment-to-fish bioconcentration factors. Håkanson (1980) used data from Swedish lakes to derive the following equation:

$$F(Hg) = \frac{4.8 \log(1 + \frac{Hg_{50}}{200})}{(pH-2) \log BPI}$$

where

F(Hg) = mercury concentration in a 1-kg pike (mg/kg, wet weight);  
Hg<sub>50</sub> = area-weighted mean mercury content of surface (0-1 cm) sediments (ng/g, dry weight);  
pH = mean pH of the system; and  
BPI = bioproduction index, a measure of the lake's trophic status that ranges from 2 (oligotrophic) to 10 (highly eutrophic).

Appendix B presents values for F(Hg) calculated for ranges of sediment mercury levels, pH, and BPI. Upon review of the ranges of values from which the equation was derived, a range of fish-to-sediment bioconcentration factors becomes evident. (Apparently, on the basis of the published paper: Hg<sub>50</sub> equals approximately 200 to 900 ng/g (Appendix B expands the range slightly to 1000 ng/g); pH = 4 to 8; and BPI = 2 to 8.) These bioconcentration factors increase with decreasing sediment mercury levels; with decreasing pH; and with decreasing BPI.

Lindqvist et al. (1991) described a series of equations (from Anderson et al. 1987), also derived from data on Swedish lakes, that use four parameters - sediment mercury levels, lake conductivity, lake pH, and lake surface area - to predict mercury levels in pike. Lindqvist et al. (1991) provide information regarding the significance of these four parameters to the power of the equation. Of these four, lake surface area contributes the least to the variability of mercury levels in pike. Since lake surface area will vary over a wide range of values for New Jersey lakes and, as stated, add only incrementally to the predictive power of the equations ( $r^2 = 0.68$  versus  $0.64$ ), the equation can be reduced to incorporate only sediment mercury levels, conductivity, and pH:

$$Hg-\pi = 4.2 + (0.0061 Hg_{sed}) - (0.38 \ln(cond)) - (0.44 pH)$$

where

Hg-pi = mercury concentration in pike (mg/kg, wet weight);  
 Hg<sub>sed</sub> = mercury concentration in sediment ( $\mu$ g/kg, dry weight);  
 cond = conductivity (mS/m); and  
 pH = lake pH.

Appendix C presents the values calculated from this equation for ranges of the three independent variables. Descriptive statistics on the data used in the derivation of the equation are shown below.

<u>Parameter</u>	<u>Mean (standard deviation)</u>
Hg <sub>sed</sub>	587 (1117)
conductivity	5.88 (5.14)
pH	6.26 (0.57)

This equation is evaluated for the following ranges in Appendix D: Hg<sub>sed</sub> = 50 to 2000; conductivity = 1 to 10; and pH = 5.5 to 7. The tables in Appendix D demonstrate that the sediment-to-fish bioconcentration factors increase with decreasing sediment mercury levels; decreasing conductivity; and decreasing pH. Despite the wide range in values reported for Hg<sub>sed</sub>, as indicated by the standard deviation, Hg-pi values differ by, at most, a factor of two over this range (see Appendix D).

### 3. Minnesota Lakes

As for water-to-fish bioconcentration factors, the data of Sorenson et al. (1989) for Minnesota lakes provide an additional empirical basis for a rough calculation of sediment-to-fish bioconcentration factors. As the preceding discussion suggested, sediment concentration alone is not a good predictor of

concentration in fish (Figure IX-1). Calculated sediment-to-fish bioconcentration factors for the Minnesota data vary widely, but over a similar range as that for the equations generated by the Swedish researchers; the extreme values are 0.28 and 13.85 (Figure IX-2). Considering only those sites at which more than two fish were analyzed eliminates the low value, and changes the range to 0.64 to 13.85. The second-highest value was 11.89. As with the findings based on the equations from the Swedish studies, the highest calculated BCFs are associated with the lowest sediment concentrations.

#### E. Range of Possible Bioconcentration Factors

##### 1. Water-to-Fish

The discussions in Sections IX.B and IX.C suggest a range of values for water-to-fish bioconcentration factors. Laboratory studies indicate a range of 2,000 to 5,000 for inorganic mercury and a range of 10,000 to perhaps as high as 100,000 for organic mercury. Selected field data, for levels of total mercury, indicate a range of 100,000 to 200,000.

##### 2. Sediment-to-Fish

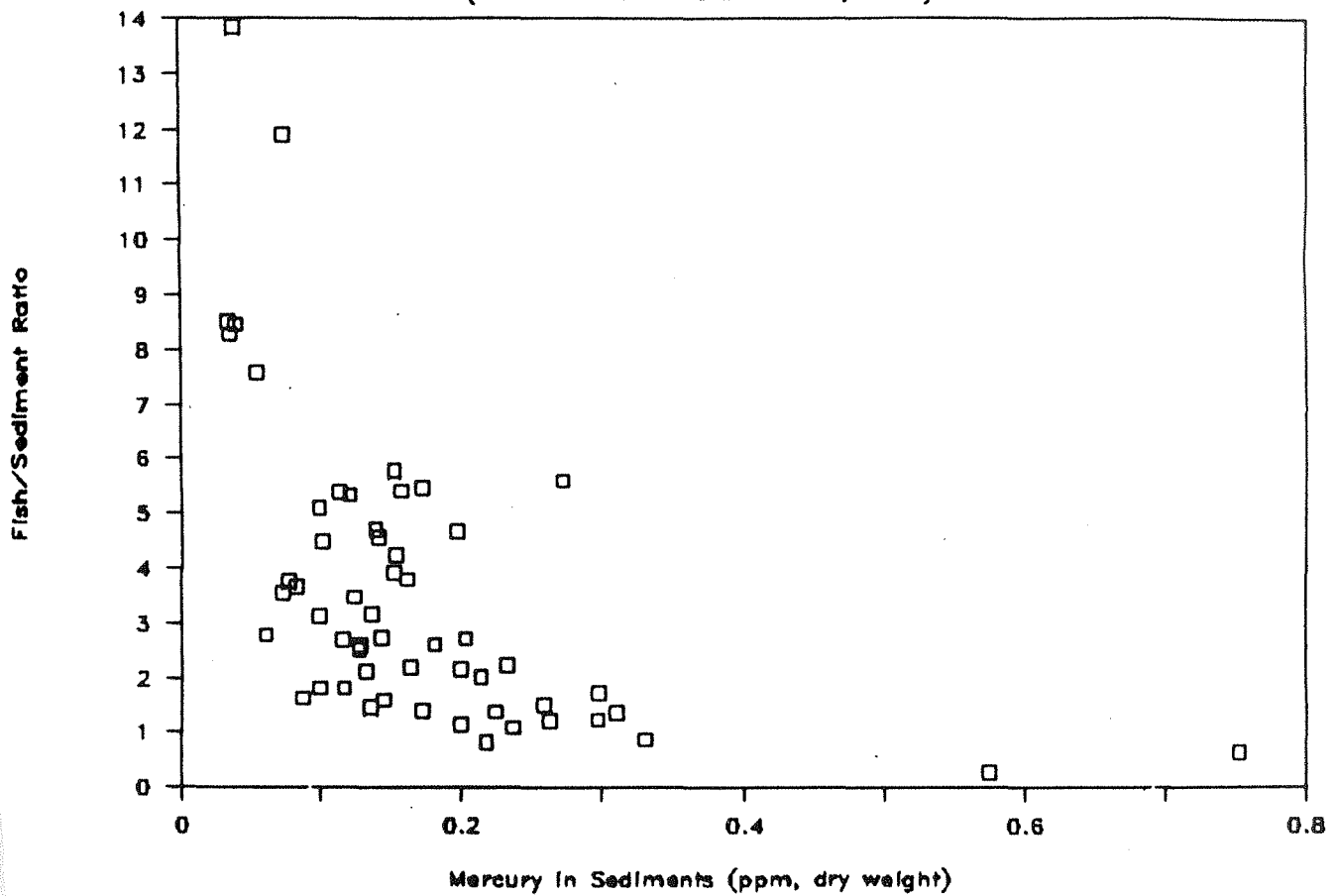
Both evaluations of the equations presented by Håkanson (1980) and Lindqvist et al. (1991), and the data of Sorensen et al. (1989) indicate similar ranges for sediment-to-fish bioconcentrations: approximately 1 to approximately 12. These factors are for total mercury levels in both sediment (dry weight basis) and fish tissue (wet weight basis).



Figure IX-2: Sediment-to-Fish Bioconcentration Factors

### Sediment-to-Fish BCFs

(from data of Sorensen et al., 1989)



## X. Fish Consumption

The fish consumption portion of the Hg F & T model must estimate the amount of fish that could be caught and eaten from nearby ponds and other waterbodies. Local consumption data are, of course, most desirable, but in its absence national data can be used. The USEPA recommended average consumption rate of 6.5 g/day is not appropriate in this model since it is recognized that fishermen are likely to eat far more fish than the average American.

### A. National Data

There are several national food consumption surveys that have attempted to quantify the amount of fish that is eaten by the average American on a daily basis. The U.S. National Marine Fisheries Service, for example, estimated that average per capita consumption of fish and shellfish was about 21 g/day in 1986 (USDA 1987). This reflects consumption of all types of fish, with an emphasis on purchased fish rather than fish that is caught by individuals. Most estimates combine consumption of freshwater fish, saltwater fish and shellfish.

Studies of consumption of recreationally caught fish generally do not distinguish between freshwater fish, saltwater fish and shellfish, either. However, a 1991 study done in Maine focused on consumption of freshwater fish caught in that state (ChemRisk et al. 1991). The results of a statewide mail survey indicated that the median consumption rate for anglers and their families was 2.0 g/day. This consumption rate in Maine may be low relative to other states given the commercial and recreational availability of saltwater fish and shellfish. This is far lower than the 6.5 g/day general consumption rate recommended for use in the Exposure Factors Handbook (USEPA 1989b).

Two other studies of recreational fishing are reviewed by USEPA in the Exposure Factors Handbook (1989). Puffer (1981) conducted over 1000 interviews with sport fishermen in the Los Angeles harbor area; and Pierce and his colleagues (1981) surveyed sport fishermen in Commencement Bay at Tacoma, Washington. USEPA concludes that the consumption rate data from these two studies should be considered representative of annual consumption rates for recreational fishermen. When local data are lacking, they recommend that the average results of the two studies be used. This average 50th percentile is 30 g/day and the average 90th percentile is 140 g/day.

### B. New Jersey Data

Data on New Jersey freshwater fish consumption are rare. However, preliminary data are available from a 1991 fish consumption survey taken at the Wanaque and Monksville Reservoirs

(Welsh 1992). Data were collected on the number of fish caught per day and the number kept by species, but totals for individuals are not given. The number of days that individuals spent fishing at the Wanaque and Monksville Reservoirs was reported to average 16.2, 15.4, and 14.2 days in 1989, 1990, and 1991, respectively. The average number of days spent fishing at other freshwater locations was reported to be 32 in 1991. This is based on a sample size of 257 anglers. This would indicate that the average angler in that part of the state goes fishing on about 45 to 50 days of the year, or about once per week.

## XI. USEPA Model

The US EPA has been developing a model which can be used to evaluate mercury exposures. This model is described in Methodology for Assessing Health Risks Associated with Indirect Exposure to Combustor Emissions (US EPA 1990b). This methodology provides a set of procedures that can be drawn up to set up a model that can be used to carry out risk assessment for a specific facility. A case study which uses this methodology is presented by Cleverly, Travis and Rice (1992). More details are also provided in the External Review Draft: Demonstration of USEPA Methodology For Assessing Health Risks From Indirect Exposure to Municipal Waste Combustor Emissions (USEPA 1991). In this paper Cleverly et al. apply the method to ten toxic air pollutants including Hg emitted by a MSW incinerator operating in Minnesota. This site-specific model evaluates five exposure pathways (inhalation, ingestion via the terrestrial food chain, ingestion of contaminated fish, ingestion of contaminated soil, and ingestion of contaminated drinking water) and reports cumulative results. The model and results are briefly described here.

### A. Source Characteristics and Location of Waterbody

The source that is modeled by Cleverly et al. is a heat recovery, fluidized-bed facility consisting of two incineration units. It is located at a sewage treatment plant along the shore of Lake Superior. The facility burns municipal solid waste which has been processed into refuse-derived fuel (RDF). This material is co-combusted with sewage sludge. The plant burns 89 tons of RDF and 317 wet metric tons of sludge per day. The incinerator is equipped with a wet scrubber system. Since the engineering expected life for the incinerators is 30 years, it is assumed that exposures will be for this duration.

The study area is a circle with 50 km radius centered on the plant. The facility is located on a plain, but there is some higher land at the edge of the study area. It contains 5 rivers and their tributaries, as well as Lake Superior. Sport fishing is common in the lake, rivers and streams.

### B. Ambient Impacts

The modeled emission rates for all of the pollutants in the study is based on the average of two sets of stack test data collected in 1987 and 1990. For mercury this rate is  $4.93 \text{ E-}04$  g/sec. It is assumed that all of the mercury is emitted as a vapor (based on the observations of Junge (1977)).

Several dispersion models are suggested for consideration in the 1990 Methodology document. The authors of the Cleverly paper chose to use COMPDEP to predict the ambient concentrations and deposition resulting from stack emissions. COMPDEP is a Gaussian

plume dispersion model developed to estimate atmospheric pollutant concentrations and wet and dry deposition in complex and rolling terrain. The model requires hourly, preprocessed meteorological data to estimate dispersion and deposition of the contaminants.

The model results are reported by Cleverly et al. in two ways - as the maximum and as the weighted average. This average represents impacts predicted over the entire study area. It is weighted to account for the fact that these receptors represent different areas within the circle.

The maximum predicted ambient air concentration (assuming that all of the mercury emitted is  $\text{Hg Cl}_2$ ) is  $5.16 \text{ E-}04 \text{ } \mu\text{g}/\text{m}^3$ . The weighted average concentration is  $4.80 \text{ E-}06 \text{ } \mu\text{g}/\text{m}^3$ . Note that the average is about 1% of the maximum. The same calculation was also done assuming that all of the mercury was emitted as  $\text{Hg}^0$  and remained in that form. Under this scenario the maximum wet deposition flux was predicted to be  $2.98 \text{ E-}09 \text{ g}/\text{m}^2/\text{yr}$  while the weighted average was  $5.70 \text{ E-}10 \text{ g}/\text{m}^2/\text{yr}$ .

### C. Deposition & Runoff

#### 1. Wet Deposition

Wet deposition was calculated using scavenging coefficients for both  $\text{Hg}^0$  and  $\text{HgCl}_2$ . Washout ratios were from Lindqvist and Rodhe (1985). The maximum wet deposition rate for  $\text{Hg Cl}_2$  is predicted to be  $7.62 \text{ E-}03 \text{ g}/\text{m}^2/\text{yr}$ , while the weighted average is much smaller at  $1.13 \text{ E-}07 \text{ g}/\text{m}^2/\text{yr}$ .

#### 2. Dry Deposition

Dry deposition was calculated using a dry settling velocity of  $0.06 \text{ cm}/\text{sec}$  after a study by Lindberg et al. (1991). The maximum dry deposition flux for either form of mercury was predicted to be  $9.72 \text{ E-}06 \text{ g}/\text{m}^2/\text{yr}$ . The weighted average dry deposition flux was  $9.06 \text{ E-}08 \text{ g}/\text{m}^2/\text{yr}$ .

#### 3. Soil Erosion & Runoff

The rate of sediment loss to the receiving water is calculated with the Universal Soil Loss Equation. This equation allows for loss of contaminant from soil due to infiltration and erosion. This model is described more thoroughly by Arnold et al. (1990).

### D. Waterbody Concentrations

The USEPA Surface Water Concentration Model consists of 3 tiers. Tier I is a conservative screening method to calculate daily intake. It assumes that all contaminant deposited onto a watershed/waterbody is transported to a receiving waterbody during

the loading period (1 year for long-term loading). Tier 2 uses input from the literature to calculate movement of contaminant through the watershed and waterbody. Tier 3 uses site-specific input to do the same calculation. Tier 2 is used in the Cleverly example.

The predicted waterbody concentrations are a result of soil erosion and runoff, and direct deposition.

#### E. Bioconcentration

The USEPA model uses a fish-water bioconcentration factor taken from the work of Strenge and Peterson (1989). The value used in the analysis is 4.625 l/g.

#### F. Incremental Mercury Ingestion

The USEPA methodology recommends that fish ingestion be based on population statistics, adjusted by 50% to account for the fraction of total consumption that can be attributed to fresh water or estuarine sources, and further adjusted to rule out the non-fish eating population. In the example calculation, a maximum of 0.0405 g/kg/day is used. This translates to 2.5 g/day for a 62 kg adult or 2.8 g/day for a 70 kg adult. Ingestion of mercury is then calculated to be 1.05 E-08 mg/kg/day (1.05 E-05  $\mu$ g/kg/day). This is about 0.01% of the ADI derived in this report.

#### G. Discussion

Cleverly and his colleagues point out that "this is the first time the EPA has modeled the wet and dry deposition flux of Hg. The analytical results are highly uncertain at this point since the existing microscale air dispersion models have yet to incorporate an algorithm to closely mimic the scavenging of soluble gases from the atmosphere through the passage of rainwater droplets....the assessment of Hg impacts is best regarded as an approximation of the ground-level contribution from the operation of the incinerator."

## XIII. Camden County MSW Incinerator Assessment

As part of the Camden County Waste-to-energy (WTE) Incinerator permit renewal application, a mercury risk assessment was carried out. This risk assessment is described in "Health Risk Assessment of Mercury Emissions from the Camden county WTE Facility" (Jones 1992) and the addendum to that report (Jones 1993). This site-specific risk assessment examined multiple exposure pathways including inhalation, plus ingestion of fruit and vegetables, mother's milk, drinking water and recreationally-caught fish. The addendum focuses on the fish ingestion pathway. This addendum incorporates mercury fate and effect algorithms which the author derived from the available literature. A sensitivity analysis was included to encompass upper and lower bound estimates for several of the more important input parameters. The results reported in this chapter are from the addendum (Jones 1993).

### A. Source Characteristics & location of Waterbody

The Camden County incinerator is a 1050 ton per day mass burn facility. Although the current contract to operate the facility only extends for 20 years, the model is based on 70 years of operation. The total Hg emission rate is assumed to be the median emission rate of 0.0342 lb/hr/unit reported from two stack tests (with three runs each) conducted in 1991. There are three units operating at Camden; therefore, there is a total emission rate of 0.103 lb/hr. The permitted emission rate for total mercury for the three units combined is 0.24 lb/hr.

It is assumed that essentially all of the mercury emissions are in the vapor phase. This assumption is based on test results from the Camden facility and from the Ogden Martin facility in Stanislaus, California, which found particulate emissions to be 1% or less. It is also assumed that 70% of the mercury emissions are in the form of  $Hg^{++}$ , based on test results from the Ogden Martin facility in Stanislaus, California.

In Jones 1992 three waterbodies (Cooper River, Newton Creek, and Big Timber Creek) are included in the risk assessment. The addendum, however, focuses on Newton Creek because of its closer proximity to the WTE facility (hence highest wet deposition flux) and its low stream flow. The watershed is divided into two portions for the purposes of calculating deposition and runoff: Upper Newton Creek and Lower Newton Creek.

### B. Ambient Impacts

The dispersion modeling for this analysis was done using the USEPA Industrial Source Complex Short Term (ISCST) model with five years of meteorological data from the Philadelphia Airport. Using this model, the maximum ambient air concentration was predicted to

be  $7.9 \text{ E-}04 \text{ } \mu\text{g}/\text{m}^3$ . This is about a factor of 400 lower than the USEPA inhalation reference concentration for elemental mercury.

### C. Deposition & Runoff

#### 1. Wet Deposition

In this analysis wet deposition is assumed to be the principle atmospheric mercury removal mechanism. Measurements from Warren County collected by Greenberg et al. (1992) are used to derive washout flux coefficients as a function of distance from the stack. These same data also confirmed the empirical washout coefficients derived by other researchers. Deposition flux is then predicted at the centroids of the Upper and Lower portions of the Newton Creek watershed. The wet deposition flux at these two locations is  $7.7\text{E-}6$  and  $19.4\text{E-}6 \text{ g}/\text{m}^2/\text{yr}$ , respectively.

#### 2. Dry Deposition

Dry deposition was calculated only for the particle fraction of the mercury emissions using the algorithm developed by the California Air Resource Board (CARB). Since the particulate emissions are a very small fraction of total emissions (less than 1%), the dry deposition of mercury was found to be negligible compared to the wet deposition (Jones 1992, 1993).

#### 3. Runoff

It is assumed that essentially all of the  $\text{Hg}^{++}$  reaching Newton Creek is from direct run-off of rainwater from impervious surfaces (determined to be about 26% of the watershed area).

### D. Waterbody Concentrations

Several steps are taken to calculate the concentration of methylmercury in Newton Creek. First, the initial water column concentration of total mercury is computed based on runoff and the Newton Creek water flow rate. This produces total mercury water column concentrations which range from 5 to 63 ng/l. Next sedimentation of particle bound mercury and other removal mechanisms are subtracted from the initial concentration estimate using field data reported by Sorensen et al. (1990). The resulting equilibrium concentration for total mercury ranges from 0.38 to 0.48 ng/l. Finally dissolved monomethylmercury (MMHg) concentrations are calculated using data from Rudd et al. (1992) which indicate that the ratio of MMHg to total mercury ranges from 0.2 to 7.1% depending on characteristics of the surrounding watershed (in this case a stream/lake setting with a small amount of wetlands). For the Newton Creek watershed, a range of 0.2 to 5% ratio is assumed with a most likely value of 1.5%. The resulting MMHg concentration is 0.8 to 24 pg/l.

## E. Bioconcentration

A bioconcentration factor from Gill and Bruland (1990) which relates water column MMHg concentration to fish tissue concentration is used in this assessment. This factor is 950,000 l/kg. The resulting upper and lower bound fish tissue concentrations range from 0.76 to 22.8  $\mu\text{g}/\text{kg}$ .

## F. Incremental Mercury Ingestion

The fish ingestion rate used in this analysis is 24 g/day. This rate represents the amount of all types of fish eaten by the average American (32 g/day) adjusted for a nine-month fishing season. The estimated daily intake is then calculated to range from 0.003 to 0.008  $\mu\text{g}/\text{kg}/\text{day}$ . This is 0.4 to 11% of the ADI derived in this report.

## G. Discussion

The Jones (1993) report draws the following conclusions from the risk assessment described above.

- \* "Based on an alternative fate and effects modeling approach which is based on currently available information and data the most likely increase in the daily intake of MMHg is equal to or less than 3% of the NJDEP guideline of 0.07  $\mu\text{g}/\text{kg}/\text{day}$ ."
- \* "The MMHg daily intake risks associated with other WTE facilities existing or planned will be less than this incremental impact unless the following conditions are encountered:
  1. Higher stack emissions per unit time.
  2. A watershed which has deposition and runoff characteristics more conducive to higher stream HgT loadings than those exhibited by the Newton Creek watershed.
  3. Receiving waterbody chemical and physical properties which are more conducive to HgT methylation than those assumed for the Newton Creek watershed.

If the latter consideration became the critical factor in determining the most likely estimate of risk, the EPRI Mercury Cycling Model or equivalent should be employed as appropriate."

### XIII. Generic Model Developed by NJDEPE

A mercury fate and transport model was developed by NJDEPE to evaluate the worst-case scenario for a generic MSW incinerator. In the absence of site-specific information, a generic model may be used to estimate the possible impact of a source or group of sources. This type of model is useful for describing the impact of sources which do not exist, but have the potential to be built. Such a model could also be used to generalize impacts when existing sources are too numerous to model or when insufficient data exist to use a site-specific model. The generic model developed in this report could be used to describe a worst-case scenario so that the Department can judge whether regulations under development would provide adequate protection in essentially all possible cases. This generic model can also be easily adapted for use with source categories other than MSW Incinerators.

This generic model is described in detail in this chapter. It is limited to two exposure pathways - inhalation and fish ingestion - since review of more comprehensive risk assessments prepared for specific incinerators indicated that these are the pathways of greatest concern.

The exposure model developed for this risk assessment describes the incremental increase of methylmercury intake from the ingestion of locally-caught fish which may be associated with total mercury emissions from MSW incinerators and other similar facilities. The model accounts for the following processes:

- dispersion of stack emissions;
- wet and dry deposition to water and land;
- runoff from the watershed to the waterbody;
- bioconcentration of mercury in freshwater fish; and
- ingestion of locally-caught fish.

Monte Carlo simulations are used to evaluate the probability distribution of the model results. (A Monte Carlo simulation is a computational technique which allows probability distributions rather than single numerical values to be input in an equation. The final product is a probability distribution which describes the entire range of possible outcomes.) The Monte Carlo analysis provides the possibility of examining a range of values for individual parameters when the actual value is not known.

#### A. Source Characteristics & Location of Waterbody

For this generic model, a hypothetical MSW incinerator is used. It is a facility of the same general size as the six MSW

incinerators which have already been permitted in the state of New Jersey (five operational, one under construction). This hypothetical facility could be located anywhere in the state. It is assumed that it is near a body of water which supports at least a limited amount of recreational fishing.

This model is intended to examine a worst-case scenario which will answer the question: "What if a large MSW incinerator were sited in a location with ideal conditions for delivering mercury to a waterbody in which fishing takes place?" In the model described in this chapter, such a location is one in a rural area with fairly high hills surrounding the facility. This combination of complex terrain in a rural area could occur almost anywhere along the Palisades or the Watchungs, in the Highlands, or along the Delaware River in the northwestern counties. Rural areas tend to have more runoff which could move mercury from the soil of a watershed into the adjacent waterbody. Complex terrain tends to result in higher air concentrations of emitted pollutants and, since the wet and dry deposition in this model is directly proportional to air concentrations of mercury, will result in higher predicted deposition rates than would occur in flat terrain.

Most MSW incinerators have a projected lifetime of 35 years. However, there is the potential for extending the lifetime to 40 or 50 years. Therefore, this model assumes a maximum lifetime of 50 years.

For the purpose of this model, it is assumed that the hypothetical facility is burning waste with the current composition typical of municipal waste and that no steps have yet been taken to remove mercury-containing materials from the waste stream. It is assumed that mercury emissions are always at the permitted level; that any exceedances would be corrected quickly and would not continue for an appreciable portion of the model period.

As discussed in Section VIII.A.3, it seems reasonable to assume that most of the Hg emitted from the MSW incinerator is in the vapor state. The mercuric chloride emissions from MSW incinerators have been reported to be around 66 to 70% of total emissions. Therefore, for this model, it is assumed that 70% of the mercury is soluble mercuric chloride, that 30% is elemental mercury, and that there are negligible amounts of particulate mercury and methylmercury. Transformations between the different mercury species is expected to be negligible in the short distances between the stack and the nearby watershed where deposition will occur.

The waterbody is assumed to be a lake (rather than a creek or river) within five kilometers of the incinerator, and to have negligible waterflow. The lake surface area is set at 50,000 square meters and the watershed is two to ten times greater, i.e. 100,000 to 500,000 square meters. These values were chosen after

review of GIS maps which showed waterbodies and their associated watersheds in the vicinity of some existing MSW incinerators. The lake is assumed to contain species of fish which are likely to bioaccumulate mercury. The fish population is naturally occurring (not stocked) and of a type that is likely to be eaten if caught.

## B. Ambient Impacts

Rather than selecting stack parameters and emission rates in order to model the hypothetical incinerator, existing dispersion modeling analyses for the six permitted incinerators were reviewed and used as a surrogate for ambient impacts. This information is summarized in Appendix E.

The air quality analysis and health risk assessment required by the NJDEPE Air Quality Regulation Program prior to permitting a MSW incinerator must be based on a number of worst-case assumptions. These include the following: that the facility will be operating at its maximum allowable capacity; that the facility will be emitting the maximum allowable amount of pollutants (maximum emission rates) at all times; that an exposed individual will be located at the point of maximum ground level concentration; and that the individual will remain there for his entire lifetime, breathing only the air contaminated by the facility, ingesting regular amounts of soil contaminated with emissions deposited from the facility, and so on for other exposure routes. The purpose of this approach is to determine whether the facility could cause a problem under those circumstances that represent the worst case, which could result in maximum exposure to the contaminants from the facility. If no or a minimal chance of detrimental effects is indicated, this assures that under more realistic conditions, there is even less chance of some detrimental effect occurring.

This approach, while generally conservative does not take into account the possibility of overlapping plumes from two or more adjacent MSW incinerators, such as the Camden County and Gloucester County facilities which are less than five kilometers apart. Not addressing this overlap could lead to an underestimate of air concentrations by less than a factor of two.

### 1. Maximum Concentration & Comparison to the RfC

The range of maximum annual average concentrations predicted by these modeling analyses for total mercury was 0.0007 to 0.005  $\mu\text{g}/\text{m}^3$ . The upper end of this range represents an incinerator with relatively low allowable emissions which is located in complex terrain. This upper value - 0.005  $\mu\text{g}/\text{m}^3$  - was selected to represent the most likely ambient impact for the hypothetical facility. If this same facility had the maximum mercury emission rate that is currently permitted, the annual average air concentration would be

0.012  $\mu\text{g}/\text{m}^3$ . If the facility had the lowest permitted emission rate, then the concentration would be 0.001  $\mu\text{g}/\text{m}^3$ .

Having identified this range of ambient air concentrations a comparison can be made to the USEPA reference concentration (RfC) of 0.3  $\mu\text{g}/\text{m}^3$  developed for elemental mercury (USEPA 1992a). The elemental mercury emissions are assumed to be 30% of the total. Since ambient concentrations are directly proportional to emissions, it is expected that the elemental mercury concentration would be 0.0003 to 0.0036  $\mu\text{g}/\text{m}^3$ , which is only 0.1 to 1.2% of the reference concentration. Even if the total mercury concentration were used in this analysis, the predicted impact of 0.001 to 0.012  $\mu\text{g}/\text{m}^3$  is well below the RfC. Thus it would seem that the inhalation route of exposure is not of particular concern in this case.

## 2. Ambient Concentrations for Dry and Wet Deposition

The maximum ambient concentration is not a good representation of the air concentrations in the area where deposition will be calculated. In complex terrain it is unlikely that the lake will be located at the point where this maximum occurs since it is near the top of a hill and the nearby lakes are located in valleys. Also, the ambient air concentration is likely to vary significantly over the watershed which covers an area of 100,000 to 500,000 square meters. So, for the purpose of estimating deposition, an average concentration would be preferable.

In the USEPA model described in Chapter XI, the COMPDEP model (a modified version of the USEPA COMPLEX I model) was used to predict the impact of a hypothetical MSW incinerator in an area that extended 50 km in all directions from the plant (USEPA 1990b). Using this model, it was found that the average ambient concentration within a 5 km circle surrounding the facility was about ten percent of the concentration at the point of maximum impact. The average over a 50 km circle was about three percent of the maximum concentration. Since the generic model developed here is focused on the 5 km area surrounding the source of interest, it is assumed that the average concentration over the watershed and the waterbody is 10% of the maximum. Thus, the ambient air concentration used in the dry deposition calculation is a range of 0.0001 to 0.0012  $\mu\text{g}/\text{m}^3$ , with a most likely value of 0.0005  $\mu\text{g}/\text{m}^3$ . The ambient concentrations used in the wet deposition calculation is 70% of this value to account for the proportion of the total mercury that is soluble and therefore available to be washed out in the rain. The soluble mercury concentrations used in the generic model ranged from 0.00007 to 0.00084  $\mu\text{g}/\text{m}^3$ , with a most likely value of 0.00035  $\mu\text{g}/\text{m}^3$ .

## C. Deposition and Runoff

### 1. Wet deposition rate

Wet deposition is discussed in detail in Chapter VII. The washout ratio approach described there was used in this generic model. Wet deposition is calculated using the following equation:

$$WDEP = (C_{air}) (WR) (J_o) (PP_h) (Y)$$

where

WDEP = wet deposition rate (g/m<sup>2</sup>-yr)

C<sub>air</sub> = incremental annual ambient concentration of soluble mercury  
μg/m<sup>3</sup>

WR = washout ratio for soluble mercury (unitless)

J<sub>o</sub> = average annual rainfall rate (m/hr)

PP<sub>h</sub> = annual hours of precipitation (hr/yr)

Y = conversion factor (0.000001 g/μg)

In this model the ambient concentration of soluble mercury is 70% of the total mercury concentration, as described above. The washout ratio is that reported by Glass et al. (1991) with a mean of 117,000 and a standard deviation of 67,000. Newark airport data were used to calculate an average annual rainfall rate of 0.0018 m/hr, and data from three airports - Newark, Philadelphia and Allentown - were used to describe the annual hours of precipitation. This represented by a minimum, most likely, and maximum value of 431, 637 and 806 hrs/yr, respectively.

Using the most likely values, a wet deposition rate of 0.47E-4 g/m<sup>2</sup>-yr, is predicted. Wet deposition rates, as measured and reported in the literature, include the following values.

Sweden: 0.109E-4 g/m<sup>2</sup>-yr (Lindqvist, et al. 1991)

Canada: 0.30E-4 to 0.60E-4 g/m<sup>2</sup>-yr (Johnson, 1987)

U.S. (MN): 0.065E-4 to 0.42E-4 g/m<sup>2</sup>-yr (Glass, 1991)

### 2. Dry deposition rate

Several dry deposition mechanisms are discussed in Chapter VIII. The generic model uses the following equation which is discussed there.

$$DDEP = (C_{air}) (DV) (Q) (Y) (Z)$$

DDEP = dry deposition rate (g/m<sup>2</sup>-yr)

C<sub>air</sub> = incremental annual ambient concentration of mercury (μg/m<sup>3</sup>)

DV = deposition velocity (m/sec)

Q = fraction of year with no precipitation (unitless)  
Y = conversion factor (0.000001 g/ $\mu$ g)  
Z = conversion factor (31536000 sec/yr)

In this model, the ambient concentration for mercury is the average for total mercury over the 5 km circle surrounding the facility. These values are reported above. The fraction of the year without precipitations calculated from the hours of precipitation used in the wet deposition equation.

The most difficult parameter to estimate is the deposition velocity. The value of 0.06 cm/sec reported by Lindberg et al. (1991) is the highest plausible value found in the literature, so this was used as the maximum for the Monte Carlo analysis. The deposition values calculated from general literature values and reported in Chapter VIII also seem to be in the expected range for dry deposition of total mercury. From these values, 0.002 cm/sec was selected as the minimum deposition velocity and 0.01 cm/sec was selected as the most likely value. Using the most likely value for all of the parameters in the equation above, a dry deposition rate of 0.14 E-5 g/m<sup>2</sup>-yr is predicted. This is a little more than a factor of ten lower than the wet deposition rate and the same order of magnitude as the dry deposition rate reported in the Appendix A, Table A-6 (Fitzgerald et al. 1992).

### 3. Runoff

For this generic model, runoff is described using the Universal Loss Equation. The parameters necessary to solve this equation and the assumptions made regarding their value are described below. Where the range of values for a given parameter could be estimated, the distribution was specified and entered into the Monte Carlo analysis.

When calculating soil loss to a waterbody, a slope length factor must be determined. The exponent  $\xi$  varies with slope, and is computed with the following equation from Arnold et al. (1990):

$$\xi = 0.6 [1 - \exp(-35.835 S)]$$

$\xi$  = exponent to determine slope length LS (see below)  
S = land surface slope (m/m)

The land surface slope is given a minimum of 0.02 m/m, a maximum of 0.05 m/m, and a most likely value of 0.0443 m/m.

The equation for slope length factor for determining rate of soil loss is also from Arnold et al. (1990).

$$LS = (\lambda/22.1)^{\frac{1}{6}}(65.41 S^2 + 4.565 S + 0.065)$$

LS = slope length factor to determine rate of soil loss  $X_e$  (see below) 6

$\lambda$  = land surface slope length (m)

The land surface slope length is set at 2000m for complex terrain.

The annual rate of soil loss to receiving water is based on the Universal Soil Loss Equation developed by the U.S. Department of Agriculture. Values for the various factors are from Bonazountas & Wagner (1984) and USEPA (1989b), with advice from Sanders (1992).

$$X_e = (R) (K) (LS) (C) (P_s) (D) (U) (V)$$

$X_e$  = soil loss rate per unit area watershed over time ( $g/m^2$ -yr)

R = rainfall erosion index ( $yr^{-1}$ )

K = soil erodability factor (tons/acre)

LS = topographic or slope length factor (unitless)

C = cover and management factor (unitless)

$P_s$  = supporting practice factor (unitless)

D = sediment delivery factor (unitless)

U = conversion factor (907180 g/ton)

V = conversion factor ( $2.46E-04$  acre/ $m^2$ )

The rainfall erosion index (R) for New Jersey ranges from 150 to 250/yr (Bonazountas & Wagner 1984; USEPA 1989b). The area of the state representing the complex terrain scenario was given an R value of 175/yr. For this calculation, a minimum R value of 150/yr was used.

The soil erodibility factor (K) is based on type of soil and organic matter content. For this model, the primary soil type was assumed to be loam or silty loam. Based on Sanders (1992) and USEPA (1989c), a K value of 0.35 tons/acre was used.

The cover and management factor (C) is based on type of land use and percent ground cover (Bonazountas & Wagner 1984). For this model, the area was assumed to consist of 20 to 70% undisturbed forest land with greater than 40% litter cover, resulting in a most likely C factor of 0.003, with a minimum of 0.001 and a maximum of 0.005.

The supporting practice factor ( $P_s$ ) was given a default of 0.5 (Sanders 1992). The sediment delivery factor (D) was taken from Bonazountas & Wagner (1984), and given a value of 0.4.

Using the most likely value of the parameters described above, the resulting  $X_c$  is calculated to be 27.9 g/m<sup>2</sup>-yr.

In this model, the first-order loss rate of mercury in soil due to soil erosion is also included. This equation is adapted from USEPA (1990b). It is assumed that the only loss of mercury from soil is from erosion, or runoff, of soil. There is assumed to be no degradation.

$$k_1 = \frac{X_c}{(BD_{soil})(SD_{soil})(1000)}$$

$k_1$  = first-order loss rate of mercury in soil due to soil erosion (yr<sup>-1</sup>)

$X_c$  = soil loss rate per unit area watershed over time (g/m<sup>2</sup>-yr)

$SD_{soil}$  = soil mixing depth (m)

$BD_{soil}$  = soil bulk density (kg/m<sup>3</sup>)

The soil mixing depth is set at 0.01m, as standard assumption in this type of analysis. The soil bulk density ranges from a minimum of 1100 kg/m<sup>3</sup> to a maximum of 1600 kg/m<sup>3</sup>, with a most likely value of 1300 kg/m<sup>3</sup>. These soil bulk density estimates are based on Stern (1992).

Next the soil concentration resulting from wet and dry deposition are calculated. The accumulation time, AT, is given a range of 1 to 50 years, modified by the first-order loss rate,  $k_1$ . Soil mixing depth is again set at 1 cm (0.01m).  $BD_{soil}$  has the same value used in the equation for  $k_1$ .

$$C_{soil} = \frac{(TDEP) \left[ \frac{1 - e^{(-k_1)(AT)}}{k_1} \right] (X)}{(SD_{soil})(BD_{soil})}$$

$C_{soil}$  = annual concentration of mercury in soil due to wet and dry deposition (mg/kg)

TDEP = total deposition rate of mercury to soil (sum of WDEP and DDEP, g/m<sup>2</sup>-yr)

$k_1$  = first-order loss rate of mercury in soil due to soil erosion (yr<sup>-1</sup>)

AT = accumulation time (yr)

X = conversion factor (1000 mg/g)

$SD_{soil}$  = soil mixing depth (m)

$BD_{soil}$  = soil bulk density (kg/m<sup>3</sup>)

Using the most likely values, the resulting 20-year incremental soil concentration is predicted to be 0.072 mg/kg. Note that D'Itri (1972) reported mercury soil concentrations of 10-150 mg/kg. This is much higher than the incremental contribution from this hypothetical source.

Finally, the concentration of mercury in sediment due to soil runoff is calculated using the following equation.

$$C_{sed(run)} = \frac{(X_e)(WA_L)(C_{soil})(AT)(B)}{(SD_{sed})(BD_{sed})(WA_w)}$$

$C_{sed(run)}$  = mercury concentration in sediment due to runoff (mg/kg)  
 $X_e$  = sediment loss rate per unit area watershed over time (kg/km<sup>2</sup>-yr)  
 $WA_L$  = watershed area contributing to runoff (m<sup>2</sup>)  
 $C_{soil}$  = annual concentration of mercury in soil due to wet and dry deposition (mg/kg)  
 $AT$  = accumulation time (yr)  
 $B$  = conversion factor (0.001 kg/g)  
 $SD_{sed}$  = sediment mixing depth (m)  
 $BD_{sed}$  = sediment bulk density (kg/m<sup>3</sup>)  
 $WA_w$  = area of waterbody receiving runoff (m<sup>2</sup>)

To estimate the size of the hypothetical waterbody and its associated watershed, GIS maps of the areas surrounding existing MSW incinerators were examined. A hypothetical lake with an area of 50,000 square meters was selected. The watershed was assumed to be two to ten times larger, with an area of 100,000 to 500,000 square meters.

Sediment mixing depth ( $SD_{sed}$ ) and sediment bulk density ( $BD_{sed}$ ) were obtained from Moser (1992).  $SD_{sed}$  is given a most likely value of 0.05m, with a range of 0.03 to 0.07m. It should be noted that  $SD_{sed}$  is greater than  $SD_{soil}$  because of the influence of factors such as lake depth and benthic and other activity.  $BD_{sed}$  is given a most likely value of 1500 kg/m<sup>3</sup>, with a range of 1000 to 2000 kg/m<sup>3</sup>.

The accumulation time (AT) in sediment is described by a triangular distribution. This distribution assigns a 10% probability to an AT of 10 years, a 50% probability to an AT of 25 years, and a 10% probability to an AT of 50 years. When the most likely values of the parameters described above are used, the concentration of sediment mercury due to soil runoff is predicted to be 4.02 E-3 mg/kg.

#### D. Lake Sediment Concentrations

Since there are no data to describe the depth and flow characteristics of the hypothetical lake described in this model, calculating water column concentrations of mercury is problematic. An alternative approach is to calculate the concentration of mercury in the lake sediments and then use sediment-to-fish bioconcentration factors. This alternative was chosen for use in the generic model.

The sediment mercury concentration resulting from soil runoff was described in the previous section. To this must be added the contribution from direct deposition in order to predict the incremental sediment concentration. The direct deposition is calculated with the following equation.

$$C_{sed(dep)} = \frac{(WDEP)(AT)(X)}{(SD_{sed})(BD_{sed})}$$

$C_{sed(dep)}$  = concentration of mercury in sediment due to wet deposition (mg/kg)

TDEP = total deposition rate of mercury to waterbody (sum of WDEP and DDEP, g/m<sup>2</sup>-yr)

AT = accumulation time (yr)

X = conversion factor (1000 mg/g)

$SD_{sed}$  = sediment mixing depth (m)

$BD_{sed}$  = sediment bulk density (kg/m<sup>3</sup>)

All of the parameters in this equation have values as described in previous equations. If the most likely values are used, then the sediment mercury concentration from direct deposition is predicted to be 1.6 E-2 mg/kg.

The total sediment mercury concentration is then:

$$C_{sed} = C_{sed(run)} + C_{sed(dep)}$$

$C_{sed}$  = total mercury concentration in sediments from runoff and deposition (mg/kg)

$C_{sed(run)}$  = mercury concentration in sediment due to runoff (mg/kg)

$C_{sed(dep)}$  = mercury concentration in sediment due to deposition (mg/kg)

The resulting total sediment concentration is 2.0 E-2 mg/kg. Sediment concentrations found in the literature include 0.01 to 0.05 mg/kg in a Canadian study (Parks 1991) and 0.01 to 0.753 mg/kg in US studies (Sorenson et al. 1990; Wiener et al. 1990).

#### E. Bioconcentration

For a description of the development of a sediment-based bioconcentration factor (BCF), see Chapter IX. A BCF of 12 was at the high end of the range appropriate to New Jersey waterbodies. The lower end BCF is about 1.2 with a mean of 2.3. This factor can be multiplied by sediment concentrations to predict the concentration of mercury in fish.

$$C_{fish} = (BCF)(C_{sed})$$

$C_{fish}$  = mercury concentration in fish tissue (mg/kg)

BCF = sediment-based bioconcentration factor (unitless)  
 $C_{sed}$  = total mercury concentration in sediments from runoff and deposition (mg/kg)

The resulting fish concentration using the mean BCF is 0.044 mg/kg.

#### F. Incremental Mercury Ingestion

The intake of freshwater fish is predicted using the following equation.

$$I_{Hg} = (C_{fish}) (I_{fish}) (A) (B)$$

$I_{Hg}$  = mercury intake from ingestion of local freshwater fish ( $\mu\text{g/day}$ )

$C_{fish}$  = mercury concentration in fish tissue (mg/kg)

$I_{fish}$  = local freshwater fish ingestion rate (g/day)

A = conversion factor (1000  $\mu\text{g/mg}$ )

B = conversion factor (0.001 kg/g)

As discussed in Chapter XI, data on consumption of recreationally-caught fish are scarce. The generic model follows the recommendation of USEPA (1989b) and uses as a basis the average of the 50th percentile and 90th percentile data reported by Puffer (1981) and Pierce et al. (1981). However, in the generic model these values are adjusted over a year to exclude the three winter months (December, January, February) when it is assumed that local fishing will not occur. This gives an adjusted fish ingestion distribution with a 50th percentile value of 23 g/day and a 90th percentile value of 105 g/day. When the 50th percentile value is inserted in the equation, the predicted incremental mercury ingestion rate is 1.0  $\mu\text{g/day}$ .

#### G. Monte Carlo Calculation of Me-Hg Ingestion Resulting From Complex Rural Terrain Model

Monte Carlo analysis allows calculations using input variables which have a probabilistic distribution of values (i.e. which have an inherent uncertainty) rather than a single fixed value. The result of such a calculation is itself a probability distribution of values rather than a single value. Two different types of uncertainty can be modeled using Monte Carlo techniques. The first type of uncertainty occurs when actual measured values for a parameter show a natural variability. For example: for parameter A, 5% of the measured values are less than x, 50% of the measured values are less than y, and 90% of the measured value are less than z. In such cases, the Monte Carlo calculation employs the distribution directly to model the variability of the parameter in the real world. If several such parameters are used in a Monte

Carlo calculation of a model of population exposure, the result gives the probability of exposure for the entire exposed population. For example: 5% of the population has an exposure less than  $x \mu\text{g}/\text{day}$ , 50% of the population has an exposure less than  $y \mu\text{g}/\text{day}$  etc.

The second type of uncertainty occurs when the actual measured values for a parameter are incomplete or imprecisely known and must be estimated. This is the case for the generic model described in this chapter. In such cases, the probability distribution employed in a Monte Carlo calculation does not attempt to model the real world variability of the parameter. Rather, the probability distribution is intended to give an estimate of values which are inclusive of all actual values of the parameter. While such estimates may include information as to the most likely value of the parameter, they are still estimates rather than descriptions of the parameter. If several such parameters are used in a Monte Carlo calculation of population exposure, the result gives the probability of finding all the exposures within a range of values. It may also be possible to estimate the most likely level of exposure. For example: there is a 5% probability that all exposures are less than  $x \mu\text{g}/\text{day}$ , there is a 95% probability that all exposures are less than  $y \mu\text{g}/\text{day}$  and the most likely estimate of exposure is  $z \mu\text{g}/\text{day}$ . However, these percentiles do not refer to percentiles of the exposed population and cannot be used directly to estimate population risk.

A Monte Carlo simulation of this model of Me-Hg exposure resulting from point source emissions of mercury and subsequent fish ingestion was carried out using the @ RISK software package (release 2.01, Palisade Corp.). The simulation was run with 10,000 iterations using Latin Hypercube sampling to ensure representative sampling of extreme values. This yielded the following distribution of values:

5% <  $0.13 \mu\text{g Me-Hg ingested/day}$   
95% <  $28.1 \mu\text{g Me-Hg ingested/day}$ .

Since the overall distribution was log-normal in character (skewed toward large values), a reasonable estimate of the most likely value of Me-Hg ingestion is given by the geometric mean of the distribution. The geometric mean =  $2.4 \mu\text{g}/\text{day}$ .

#### H. Discussion

If the methylmercury ingestion rates predicted by the Monte Carlo analysis are adjusted for a 62 kg woman, then the 5th percentile estimate of methylmercury ingestion is  $0.002 \mu\text{g}/\text{kg}/\text{day}$ , the geometric mean is  $0.039 \mu\text{g}/\text{kg}/\text{day}$ , and the 95th percentile is  $0.45 \mu\text{g}/\text{kg}/\text{day}$ . These values can be compared to the ADI of  $0.07 \mu\text{g}/\text{kg}/\text{day}$ . The 5th percentile is a small fraction of the ADI

(about 3%). However, the geometric mean is a little more than half of the ADI and the 95th percentile is more than six times greater.

#### XIV. Exposure Routes Not Considered in this Study

##### A. New Jersey Water Supplies

Mercury is rarely detected in finished water supplies. In 1971, USEPA analyzed 698 raw and finished water samples collected from 273 drinking water supplies throughout the United States. Eleven samples exceeded 1000 ng/L (1000-4900 ng/L) and 1 sample exceeded 5000 ng/L. All other samples were below 1000 ng/L (NRC 1978). The drinking water standard, or maximum contaminant level (MCL), for mercury is 2000 ng/L (2000 ng/L is equivalent to 2.0 ug/L or parts per billion). Reported mercury levels in drinking water are listed in Table A-3. All values reported for drinking water are for total mercury.

Few investigators have examined the occurrence of mercury in groundwater. Bloom (1989) reports that laboratory tap water from an on-site deep well in Washington State contains an extremely low and constant concentration of 0.3 ng/L. Total mercury levels in the laboratory water at Skidaway Institute of Oceanography, which also gets its water from a deep well, were found to be 0.5 ng/L. There are no reports in the available literature describing species of mercury in groundwater.

In the last few years, groundwater contamination by mercury has been detected in over 200 potable wells in southern New Jersey. The origin of the contamination is unknown but is suspected to be anthropogenic. A detailed investigation of the literature including mineralogy concludes that the levels detected in these wells are too high to have occurred naturally (Dooley 1992). Researchers (Windom & Smith 1992) have detected total mercury at levels from <1.0 to 36,400 ng/L in wells in this area. While most of this was determined to be inorganic mercury salts, volatile mercury comprised up to 10% of the total mercury in contaminated water samples. Trace levels of methylmercury were also detected, comprising 1 to 3% of the total mercury.

Groundwater is not being considered a normal route of exposure because, in each case where mercury is detected exceeding the MCL of 2000 ng/L, remediation occurs; that is, the home is either hooked up to city water or is given a treatment unit to remove the mercury. For the purposes of evaluating risk to an individual from incinerator emissions, groundwater ingestion was not considered as a background exposure.

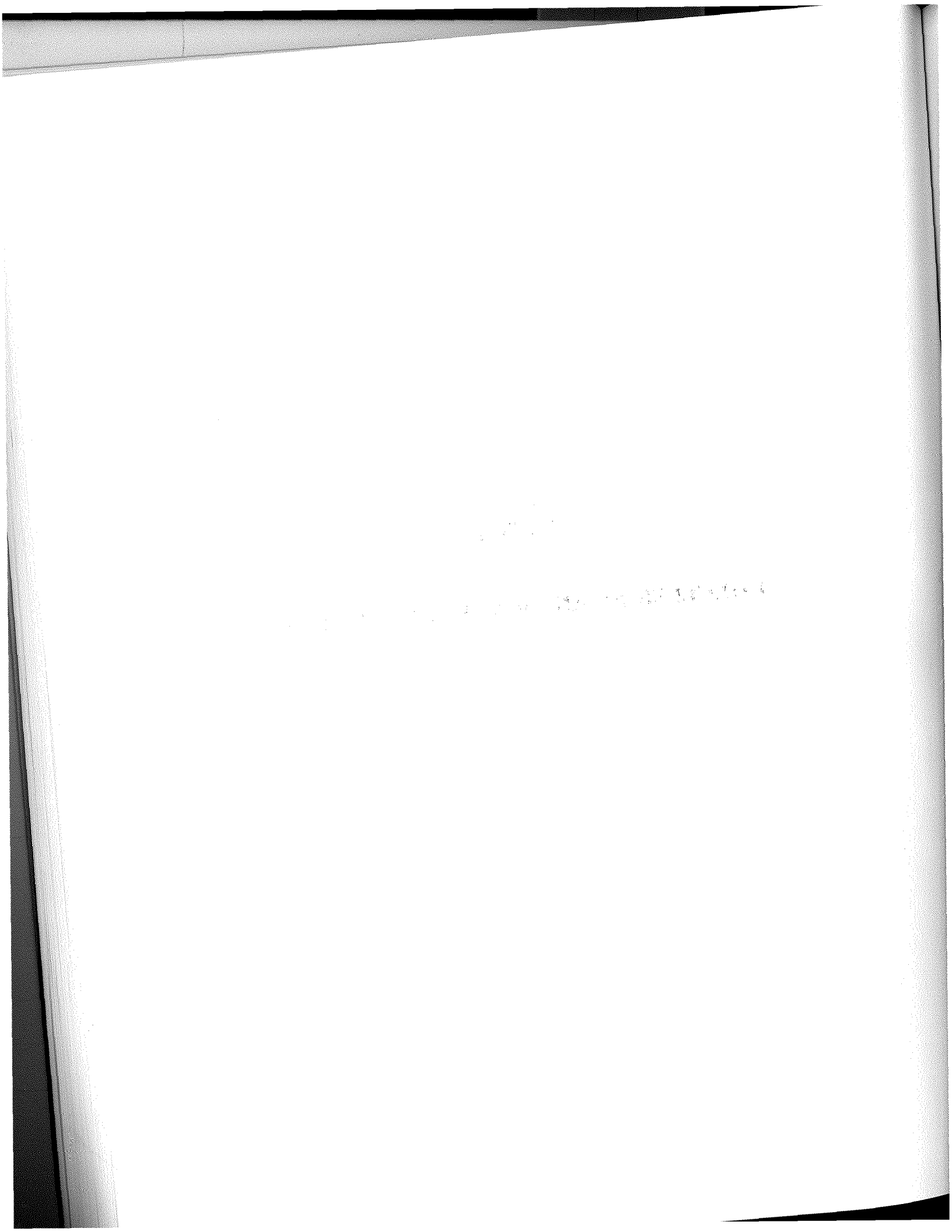
##### B. Dental Amalgams

Mercury exposure resulting from the use of dental amalgams is reported by the World Health Organization (1990) to be the most significant exposure pathway for elemental mercury in the general population. (Populations living in the general vicinity of a

stationary source of mercury were not considered in their analysis.) The possible health effects related to this exposure are currently the topic of significant scientific debate. Also unknown is the possible interaction between mercury dose received as a result of dental amalgams and the dose received through other routes of exposure, such as food. The lack of information on this subject makes it virtually impossible to evaluate this route of exposure in this risk assessment and has been recommended for future evaluation.

**PART 4**

**CONCLUSIONS AND RECOMMENDATIONS**



## XV. Conclusions and Recommendations

### A. Review of Background Data

Global mercury emissions, deposition and bioaccumulation are important aspects of the overall mercury problem. Ambient air concentrations of mercury in remote locations appear to be increasing. Existing levels of mercury in tuna, swordfish, shark and other fish that are commonly eaten can lead to mercury ingestion in excess of safe levels. Although the existing literature was reviewed extensively and discussed within the subcommittee, there remains a substantial amount of uncertainty regarding the level of global mercury contamination, its effect on the residents of New Jersey and the contribution of New Jersey sources to this contamination. Additional data, not currently available, are needed to explore more thoroughly this issue.

### B. Comparison to USEPA Reference Dose and the Acceptable Daily Intake Derived in This Report

The USEPA has established a reference dose (RfD) of 0.3  $\mu\text{g}/\text{kg}/\text{day}$  for ingestion of methylmercury. This value is presently under review. It is based on a variety of health endpoints, including paresthesia (that is, numbness, tingling, burning, or itching in the skin unrelated to dermatological conditions) in adults and older children. This RfD was derived by dividing the lowest observed effect level (LOEL) or threshold dose by an uncertainty factor of 10.

\* There is evidence to indicate neurologic development damage to infants who are exposed "in utero" through maternal ingestion. The current EPA RfD may not be protective against the occurrence of these effects. The best estimate, derived in this report, of an ADI that appears to address this endpoint is 0.07  $\mu\text{g}/\text{kg}/\text{day}$ . As with any guideline for exposure to toxic substances, this ADI is an estimate of a level which provides adequate public health protection by use of safety factors and does not necessarily directly predict the occurrence of adverse health effects.

\* Based on probabilistic calculations using national population data (i.e. fish consumption and methylmercury levels in fish) a small fraction of the New Jersey population may already be ingesting mercury in amounts which exceed the RfD established by EPA due to consumption of saltwater fish.

\* If current background exposure is compared to the more stringent ADI for developmental effects derived in this report, a larger fraction of women of childbearing age may be exposed to methylmercury at levels that exceed this ADI. Due to the margin of protectiveness inherent in the ADI, exposures exceeding the ADI will not necessarily result in adverse effects. There does not

appear to be a margin of safety, however, for significant increases in methylmercury exposure.

\* Further research is needed to define more precisely the relationship between maternal exposure to methylmercury and developmental effects. In addition, a more direct estimate of background maternal Me-Hg exposure based on direct measurement of Me-Hg in blood is also needed.

### C. Results of the Predictive Models

No generally accepted model for the simulation of mercury contamination of freshwater fish exists at this time. While this field is developing rapidly, this report was limited to the review of two site-specific and one generic model. Site-specific models address the impacts of an actual facility, using detailed information about the pollution sources, the layout of the facility, and the surrounding area. Where pieces of this information are lacking, assumptions are made about their value which tend to overestimate the impact of the facility. The more that is known about the source and its surroundings, the closer the estimated impact should be to reality. These models provide a snapshot of the possible impact of MSW Incinerators on public exposure to mercury.

In the absence of site-specific information, a generic model may be used to estimate the possible impact of a source or group of sources. The generic model developed in this report could be used to describe a worst-case scenario so that the Department can judge whether regulations under development would provide adequate protection in essentially all possible cases. This generic model can also be easily adapted for use with source categories other than MSW Incinerators.

\* Based on the generic model results, through deposition to water and soil existing emissions from MSW incinerators operating at permitted levels may result in increased mercury levels in New Jersey freshwater fish and an increase in the ingestion of methylmercury. This increased ingestion constitutes a significant fraction of the ADI for a portion of the state's population eating locally caught freshwater fish.

\* The two site-specific models, which analyze the impact of incinerators in Minnesota and Camden County, NJ, predict methylmercury ingestion rates that are a small fraction of the ADI using actual emission test data.

\* Modeling of inorganic mercury exposure through inhalation in all three models indicates a large margin of safety between predicted exposure and the current EPA Reference Concentration (RfC) of 0.3 ug/m<sup>3</sup>.

\* Refinement of the predictive models and additional validation of the assumptions used, where necessary, will significantly decrease the uncertainty in future calculations.

## XVI. Ongoing Research in NJDEPE

### A. Analysis of Mercury Species in Groundwater Using Inductively Coupled Plasma - Mass Spectrometry and Gas Chromatography

Researchers at Skidaway Institute of Oceanography in Georgia conducted the analytical portion of this NJDEPE-funded study. Water samples from 78 private potable wells in southern New Jersey were collected for mercury analysis in 1991 - 1992. The wells were located in Atlantic, Burlington, Camden, Cape May and Ocean Counties. Total mercury concentrations varied from 1 ng/L to over 36,000 ng/L. The dominant forms of mercury in the wells were  $\text{Hg}^0$  and  $\text{HgCl}_2^0$ . Monomethylmercury was present in some wells comprising up to 8% of the total mercury in one groundwater sample. This project is complete. A research summary is available by contacting the Division of Science and Research, CN 409, Trenton, NJ 08625.

### B. Environmental Occurrence and Properties of Mercury and its Compounds, A Critical Review of the Literature

Researchers at Rutgers University are working on this NJDEPE-funded study. The goal of this study is to provide a comprehensive review of mercury in the environment which will be of use to nonspecialists as well as experts in the field. The study will produce as output a monograph on mercury that will include critical discussion of research studies. In brief, the monograph will discuss the chemical and physical properties of both inorganic and organic mercury species, man-made sources of mercury, emission control technology, long-range transport including ambient reactivity, bioaccumulation, analytical techniques, occurrence and speciation of mercury in soil, sediment, air and water, ambient data in these media in a variety of uncontaminated and contaminated locales, survey of permissible levels, description of toxicology or mercury, and a complete reference list. This project began in September, 1992 and is scheduled for completion by the end of the summer, 1993.

### C. Mercury in Air and Rainwater in the Vicinity of a Municipal Resource Recovery Facility in Northwestern New Jersey

Rutgers University was also contracted to conduct this NJDEPE-funded study. The goal of this project was to determine an effective monitoring strategy for point sources of mercury. The Warren County incinerator was selected for study. The investigators found no significant difference in atmospheric mercury levels near the point source as compared with "background" samples. Rainwater samples collected near the point source, however, were higher than samples collected from unaffected sites. This project is complete. Information on the study can be obtained

by contacting the Division of Science and Research, CN 409, Trenton, NJ 08625.

D. Total Mercury in Freshwater Fish

The Philadelphia Academy of Sciences has been contracted to perform the work for this NJDEPE-funded project. Fish samples from lakes and rivers throughout the state are being collected and analyzed for total mercury. In addition to the fish samples, water samples are being collected and analyzed for water quality parameters and for mercury. The project began in September, 1992 and is scheduled to be completed in December, 1993.

E. Evaluation of Mercury Distribution and Mobility in the Kirkwood-Cohansey Aquifer System, New Jersey Coastal Plain

Researchers at the US Geological Survey are conducting this NJDEPE-sponsored study. The overall objective of the proposed research is to determine the cause or causes of elevated concentrations of mercury in water from the Kirkwood-Cohansey aquifer system. Individual objectives to be addressed include: 1) determination of mercury distribution in groundwater in the Kirkwood-Cohansey aquifer system of the NJ Coastal Plain; 2) identification of the sources of mercury that may contribute to the occurrence of mercury in groundwater and assessment of the relative importance of the sources; and 3) determination of the possible mechanisms responsible for mobilization of mercury in soils and/or coastal plain sediments, and corroboration of viable pathways for mercury from identified sources to groundwater. This study began in September, 1992 and is scheduled for completion December, 1993.

F. Inventory of Sources of Mercury in the Environment, a Multi-Media Investigation and Evaluation of Residual Contamination and Background Levels of Mercury

This research project is being conducted by researchers within NJDEPE. Samples of air, rainwater, sediment, soil, water, and biota in selected contaminated and background New Jersey sites are being collected and analyzed for total mercury. This multi-year project began in July, 1992 and the first phase will be complete by August, 1993.

G. Fish Consumption Patterns by New Jersey Anglers

Scientists at the New Jersey Marine Sciences Consortium, in conjunction with NJDEPE's Division of Science and Research, will obtain pertinent fish consumption data from anglers throughout the state. Information on the species of fish caught, frequency of consumption, waterbody location will be collected through field surveys and interviews from 1000 NJ households, contact with

fishing organizations, and analysis of sales and landings data.  
The project is anticipated to start in the Fall of 1993.

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**APPENDIX A**

**SUMMARY OF MEASURED MERCURY VALUES**

**IN THE LITERATURE**

**APPENDIX A. Summary of Measured Mercury Values in the Literature**

The values presented below are minimum and maximum ranges reported in the literature for the parameters indicated. Attached are tables which delineate the actual ranges, means and/or median reported by individual researchers for the following:

- Table 1: Mercury concentrations in the atmosphere
- Table 2: Mercury concentrations in precipitation
- Table 3: Mercury concentrations in water
- Table 4: Mercury concentrations in soils and sediments
- Table 5: Mercury concentrations in biota
- Table 6: Mercury deposition rates
- Table 7: Speciation of mercury in environmental media

References cited in this Appendix can be found in the REFERENCE section of this report.

	<u>Data Ranges</u>	<u>Mean Ranges</u>	<u>Median</u>
<b><u>ATMOSPHERIC</u></b>			
Developed areas from suburban to industrial:	BD-550 ng/m <sup>3</sup>	3-22 ng/m <sup>3</sup>	
"Pristine" land or remote ocean:	1-14 ng/m <sup>3</sup>	0.4-9 ng/m <sup>3</sup>	
US: Warren, NJ (2 km from RRF)	0.08-20 ng/m <sup>3</sup>	3.4 ng/m <sup>3</sup>	2.27 ng/m <sup>3</sup>
US: Warren, NJ	0.53-25 ng/m <sup>3</sup>	5.0 ng/m <sup>3</sup>	2.5 ng/m <sup>3</sup>
<b><u>PRECIPITATION</u></b>			
Rain and snow: influenced by emission source:	4-3400 ng/L	19-240 ng/L	
" : no known influence	BD-100 ng/L	1-60 ng/L	
US: Warren, NJ (2 km from RRF)	5-606 ng/L	87 ng/L	32 ng/L
US: Warren, NJ	<2-63 ng/L	33 ng/L	13 ng/L
<b><u>WATER</u></b>			
Lakes:	0.3-245 ng/L	0.04-74 ng/L	
Rivers & Streams	1-27 ng/L	1-7 ng/L	
Groundwater in NJ: contaminated	2700-36400	8424 ng/L	4600 ng/L
" : pristine	<1-1650 ng/L	5 ng/L	
Drinking/Tap water in US:	BD-4900 ng/L	0.3-25 ng/L	
Open Ocean (seawater):	0.19-3 ng/L		
Coastal (seawater):	1.6-7 ng/L		
Ice:	<0.001-17	<0.001-10 ng/L	
Runoff:	0.19-10 ng/L		
<b><u>SOIL &amp; SEDIMENTS</u></b>			
Lake sediments:	10-753 ng/g	70-310 ng/g	
U.S. soils:	12-200 ng/g	8-117 ng/g	
max. in U.S. contaminated soils:	3 x 10 <sup>6</sup> ng/g		

Appendix A (continued)

BIOTA (see attached)

OTHER (see attached)

Table A-1: Mercury Concentrations in the Atmosphere

Location	Sample Type	Mercury Units	Range	Mean	Median	Reference
<b>Urban, industrial areas</b>						
General	air	ng/m <sup>3</sup>	0.5-7.5	4.0		Nriagu, 1990
urban	air	ng/m <sup>3</sup>	2-30			Lindberg, 1987
urban	air	ng/m <sup>3</sup>		10		WHO, 1990
urban	air (Hg <sup>0</sup> )	ng/m <sup>3</sup>	0.5-50	7.0		Schroeder, 1982
large cities	air	ng/m <sup>3</sup>	5-50			Lindberg, 1987
power plant plume	air	ng/m <sup>3</sup>	200-1700			Lindberg, 1987
Sweden	air	ng/m <sup>3</sup>	0.8-13.2	4.0		Lindqvist & Rodhe, 1989
Italy	air	ng/m <sup>3</sup>	2.2-31.5	10.1		Lindqvist & Rodhe, 1989
Italy	air	ng/m <sup>3</sup>	12.1-35.3	22.2		Lindqvist & Rodhe, 1989
Slovenia	air	ng/m <sup>3</sup>	4-10			Lupsina & Horvat, 1992*
Slovenia - mine	air	ng/m <sup>3</sup>	11-550			"
Canada	air	ng/m <sup>3</sup>		11		WHO, 1990
US: Duluth, MN (10 km from MWC)	air	ng/m <sup>3</sup>		3		Glass et al. 1991
US: Warren, NJ (2 km from RRF)	air (Hg <sup>0</sup> )	ng/m <sup>3</sup>	0.08-20	3.4	2.27	Greenberg et al. unpubl.
US: Warren, NJ	air (Hg <sup>0</sup> )	ng/m <sup>3</sup>	0.53-25	5.0	2.5	Greenberg et al. unpubl.
<b>Suburban</b>						
Sweden (7 sites)	yearly air	ng/m <sup>3</sup>	1.5-6.3 monthly means 2.7-4.0 yearly means			Brosset, 1982
Canada	air	ng/m <sup>3</sup>		8		Lindqvist & Rodhe, 1989
US: Tampa, FL	air (Hg <sup>0</sup> )	ng/m <sup>3</sup>	BD-49		4	Johnson & Braman, 1974
	air (Hg <sup>2+</sup> )	ng/m <sup>3</sup>	BD-220		1.5	"
	air (methyl Hg)	ng/m <sup>3</sup>	BD-119		1.0	"
<b>Rural, Remote (terrestrial)</b>						
"background"	air	ng/m <sup>3</sup>	2.4-7.0			Panel, 1978
rural	air	ng/m <sup>3</sup>	3-10			Lindberg, 1987
rural	air	ng/m <sup>3</sup>	0.02-3.0	1.6		Nriagu, 1990
remote	air	ng/m <sup>3</sup>	0.004-0.75	0.38		Nriagu, 1990
Northern Hemisphere	air	ng/m <sup>3</sup>	2.0			Volland, 1991
Northern Hemisphere	air	ng/m <sup>3</sup>	2.0			WHO, 1990
Sweden, rural	air	ng/m <sup>3</sup>	2-6	3.3		Lindqvist & Rodhe, 1989
Italy, rural	air	ng/m <sup>3</sup>	1.2-4.1	3.2		Lindqvist & Rodhe, 1989
Italy	air	ng/m <sup>3</sup>	6.4-13.7			Barghigiani & Ristori, 1992*
Slovenia	air	ng/m <sup>3</sup>	2-5			Lupsina & Horvat, 1992*
Canada, rural	air	ng/m <sup>3</sup>		3		Lindqvist & Rodhe, 1989
Canada, rural	air	ng/m <sup>3</sup>		9		WHO, 1990

Table A-1 (continued)

US: Tampa, FL	air	ng/m <sup>3</sup>	3-300			Johnson & Braman, 1974
US: Little Rock Lake, WI	air	ng/m <sup>3</sup>		2.41		Fitzgerald & Watras, 1989
US: Little Rock Lake, WI	air	ng/m <sup>3</sup>	1.9-2.0 (mean ranges)			Wiener et al. 1990
<u>Remote- ocean air</u>						
Remote marine	air	ng/m <sup>3</sup>	1-3			Lindberg, 1987
US: oceanic	air	ng/m <sup>3</sup>	0.6-0.7	0.7		Schroeder, 1982
Italy - sea	air	ng/m <sup>3</sup>	0.9-3.6	2.1		Lindqvist & Rodhe, 1989
Atlantic	air	ng/m <sup>3</sup>	1-11	2.8		Lindqvist & Rodhe, 1989
"	"	"			1-3	"
North Atlantic	air	ng/m <sup>3</sup>	1-1.9	1.6		"
North Atlantic	air (gaseous)	"		1.96		Slemr, 1992
South Atlantic	air (gaseous)	"		1.33		"
Long Island Sound	air	ng/m <sup>3</sup>	1.6-7.2	2.9		Lindqvist & Rodhe, 1989
Tropical Pacific	air	ng/m <sup>3</sup>	0.8-3.2	1.7		"

\* data is from an abstract from work presented at International Conference on Mercury as a Global Pollutant in Monterey, CA, 1992.

Table A-2. Mercury Concentrations in Precipitation

Location	Sample Type	Mercury Units	Range	Mean	Median	Reference
<b>Rain</b>						
"background"	rain	ng/L	5-100			WHO, 1990
general	rain	ng/L	2-10			Fogg & Fitzgerald, 1989 & Brosset, 1987
global - land	rain	ng/L	5-30			Lindqvist & Rodhe, 1985
global - oceans	rain	ng/L	1-10			Lindqvist & Rodhe, 1985
N. Pacific Ocean	rain	ng/L	10-50			Lindqvist & Rodhe, 1985
Sweden	rain	ng/L	10-75			Lindqvist & Rodhe, 1985
Sweden	rain	ng/L	50-100			USEPA, 1991 & Glass et al. 1986
Sweden	rain	ng/L	<0.1-0.3			Lee & Hultberg, 1990
Denmark	rain	ng/L	5-300	80 (1979 study cited)		Lindqvist & Rodhe, 1985
UK-Liverpool	rain	ng/L	6-30	17		Lindqvist & Rodhe, 1985
Japan	rain	ng/L	0.7-1.5	1.0		Lindqvist & Rodhe, 1985
Japan	rain	ng/L	1-2			Glass et al. 1986
Italy (polluted)	rain	ng/L	10-85			Lindqvist & Rodhe, 1985
Canada	rain & snow	ng/L	20-100 (1980 study cited)			Lindqvist & Rodhe, 1985
Canada (Forbes, Ontario)	rain & snow	ng/L	10-1220	240		Glass et al. 1986
Canada (Dorset, Ontario)	rain & snow	ng/L	<10-40	21		Glass et al. 1986
US: MN	rain	ng/L	16.8-24.2	18.7	18.6	Sorenson et al. 1990
US: MN (3 sites)	rain	ng/L		19		Swain, 1989
US: MN (Duluth)	rain & snow	ng/L	4-3400	133	37	Glass et al. 1986
US: MN (Duluth)	rain	ng/L		10.5 (1989)		Glass et al. 1991
US: MN (Duluth)	rain	ng/L		22.6 (1988)		Glass et al. 1991
US: " (10 km from MWC)	wet deposition	ug/m <sup>2</sup> /year		14		Glass et al. 1991
US: MN (Marcell)	rain & snow	ng/L	5-100	23	17	Glass et al. 1986
US: MN (Marcell)	rain	ng/L		17.7; 18.0		Glass et al. 1991
US: MN (Ely)	rain	ng/L		19.7; 81.4		Glass et al. 1991
US: MN	rain	ng/L	8.6-19.9			Sorensen et al. 1992*
US: Southern New England Coastal	rain	ng/L	2-21	10		Lindqvist & Rodhe, 1985
US: Northern New England Mountain	rain	ng/L		60		Lindqvist & Rodhe, 1985
US: Avery Point, CT	rain/snow	ng/L	3-17	12		USEPA, 1991 & Glass et al. 1986
US: Cape Cod, MA	rain/snow	ng/L	6-18	11		"
US: WA	rain	ng/L	2.8-15.13			Bloom & Watras, 1989
US: Warren, NJ (2 km from RRF)	rain	ng/L	5-606	87	32	Greenberg et al. unpubl.
US: Warren, NJ	rain	ng/L	<2-63	33	13	Greenberg et al. unpubl.
US: NJ (industrial site)	rain	ng/L	14-59	41		Stevenson et al. unpubl.
US: NJ (3 suburban sites)	rain	ng/L	14-30	22		"

Table A-2 (continued)

<u>Snow</u>					
Japan	snow	ng/L	0.6-3.4	1.0	Lindqvist & Rodhe, 1985
Canada: Lake Superior	snow	ng/L	12-100	26	Glass et al. 1986
Canada: Ontario	snow	ng/L		14	Glass et al. 1986
US: Alaska	snow	ng/L	< 5-26		Lindqvist & Rodhe, 1985
US: MN (3 sites)	snow	ng/L		15-100 (mean ranges)	Glass et al. 1986
US: WI (2 sites)	snow	ng/L		12	Glass et al. 1986
US: WA	snow	ng/L	4.05		Bloom & Watras, 1989
<u>Ice</u>					
Greenland	ice cores	ng/L	2-17	10	Lindqvist & Rodhe, 1985
Antarctic	ice cores	ng/L		<0.001-<0.002	Vandal, et al. 1992*
Antarctic	surface snow blocks			<0.006	"

\* data is from an abstract from work presented at International Conference on Mercury as a Global Pollutant in Monterey, CA, 1992.

Table A-3: Mercury Concentrations in Water

Location	Sample Type	Mercury Units	Range	Mean	Median	Reference
<b>Lakes</b>						
General: rivers & lakes	rivers & lakes	ng/L	1-5			Lindqvist & Rodhe, 1985
Sweden (25 lakes)	lake water	ug/m <sup>3</sup>	2-12	0.04-0.76	3.1-6.2	Lindqvist et al. 1991
Sweden	lake water	ng/L methyl	0.09-0.32			Lee & Hultberg, 1990
Japan, unpolluted	lake water	ng/L	0.3-2.3			Nojiri, 1986
China	lake water	ng/L	3.42-14.52			Bloom & Liang, 1992*
Canada: Lake Superior	lake intake	ng/L	<4-10	7		Glass et al. 1986
Canada: Black Lake	lake water	ng/L	3.4			Schintu & Kudo, 1989
Canada: Ontario	wetland pond	ng/L	0.05-0.4-methyl			Rudd et al. 1992*
US: WI (4 lakes)	lake water	ng/L	0.9-1.85			Fitzgerald & Watras, 1989
US: WI surface	lake water	ng/L	18-245	74		Glass et al. 1986
US: WI bottom	lake water	ng/L	13-134	50		Glass et al. 1986
US: WI	lake water	ng/L	0.9-1.0	1.0		Weiner et al. 1990
US: MN (80 surface)	lake water	ng/L	0.9-7.0	2.47	2.30	Sorenson et al. 1990
US: MN (3 sites)	lake water	ng/L		3		Swain, 1989
US: Silver Lake, CA	lake water	ng/L		0.6		Gill & Bruland, 1990
US: WA	lake water	ng/L		0.163-10.68		Bloom & Watras, 1989
US: NJ (3 lakes)	lake water	ng/L	1-26	1.2-18		Stevenson et al. unpubl
<b>Rivers, Streams</b>						
Sweden (13 streams)	streams	ng/L		1.0-6.5		Iverfeldt & Johansson, 1988
Italy	rivers	ng/L	1.4-1.48 dissolved 11.8-26.9 particulate			Barghigiani & Ristori, 1992*
Canadian Arctic	river	ng/L-dissolved		4		Lockart et al. 1992
"	river parti.	ng/g-particulate		70-120		"
Canada: Ottawa River	river	ng/L	7.7-9.0			Schintu et al. 1989
Canada: Gatineau River	river	ng/L	11.4			
Canada: into Harp Lake	streams	ng/L	1.6-3.2	2.0	2.0	Mierle, 1990
Canada: before wetland	stream	ng/L	10-16			Rudd et al. 1992*
Canada: after wetland	stream	ng/L	1-7			"
US: CA (near cinnabar deposit)	up creek	ng/L	2.8			Gill & Bruland, 1990
"	down creek	ng/L	34			Gill & Bruland, 1990
US: New York	streams	ng/L	< 400			Rohman et al. 1987
US: Duluth, MN (MWC)	upstream	ng/L	4.5			Glass et al. 1986
	downstream	ng/L	12.0			Glass et al. 1986
<b>Ground Water</b>						
US: all states	groundwater	ng/L	< 10-100			D'Itri, 1972
US: NJ (above MCL of 2000)	groundwater	ng/L	2700-36400	8424	4600	Windom et al. 1992
US - NJ (below MCL of 2000)	groundwater	ng/L	<1-1650	101	5	"

Table A-3 (continued)

Drinking/Tap Water

Canada	tap water	ng/L	3.9		Schintu & Kudo, 1989
US: all states	tap water	ng/L	<1,000-4,900	<1,000	NRC, 1978
US: summary	tap water	ng/L		25	WHO, 1990
US: WA	tap water	ng/L		0.3	Bloom & Watras, 1989

Ocean Water

open	seawater	ng/L	0.19-2.10		Gill & Fitzgerald, 1988
open	seawater	ng/L	0.5-3		Lindqvist & Rodhe, 1985
					WHO, 1990
coastal	seawater	ng/L	2-15		"
coastal-estuary	seawater	ng/L	2.6-6.7		Leermakers & Bueyans, 1992*
coastal-estuary	seawater	pM	8.5-158 total		Glass & Flegal, 1992*
		pM	1.6-8.7 dissolved		"

Runoff

Sweden	into lake	ng/L	1-10		Lindqvist et al. 1991
Finland	runoff	ng/L methyl	0.19-1.99	0.70	Verta et al. 1992*

\* data is from an abstract from work presented at International Conference on Mercury as a Global Pollutant in Monterey, CA, 1992.

Table A-4: Mercury Concentrations in Soils and Sediments

Location	Sample Type	Mercury Units	Range	Mean	Median	Reference
<b>Lake Sediments</b>						
Italy	sed. contam	µg/g-dry		0.61		Giordano et al. 1992
	sed. uncontam	µg/g-dry		0.07		"
Canada: background	sediments	µg/g	0.01-0.05			Parks, 1991
Canada: contaminated	surface sed	µg/g		2.69-9.22		Harrison & Klaverkamp, 1990
Canada: uncontaminated	surface sed	µg/g		0.10-1.30		"
US: MN	sediments	µg/g	0.034-0.753	0.174	0.154	Sorenson et al. 1990
US: WI	sediments	µg/g-dry	0.01-0.170			Wiener et al. 1990
US: WI	sed, 0-15 cm	µg/g-dry	0.09-0.19			Rada et al. 1989
	sed, >15 cm	µg/g-dry	0.04-0.07			"
US: MA & CA	sediments	µg/g-wet	0.001-0.075			Gilmour, 1992*
US: MS	sed, contamin	µg/g-dry		0.31		Wiener et al. 1992*
	sed, background		0.041		"	"
US: NJ (Monmouth 9 Lakes)	sediments	µg/g	<0.02-0.25	0.09	0.07	Monmouth, 1989
US: NJ (3 lakes)	sediments	µg/g	0.065-0.384	0.127-0.326		Stevensen et al. unpubl
<b>River Sediments</b>						
Germany (Elbe, contam)	susp. part.	µg/g-dry	0.05-2.7 methyl			Wilken & Hintelmann, 1991
		µg/g-dry	2-59 total	30		"
Italy	sed, mouth	µg/g	1-2			Barghigiani & Ristori, 1992*
	sed, offshore	µg/g	0.1-0.2			"
<b>Coastal Sediments</b>						
Italy	sediment	µg/g-dry	>0.02-2.03	0.23		Giordano et al. 1992
US: FL (Everglades)	muck soil	µg/g		0.154		Patrick & Gambrell, 1992*
	sandy soil	µg/g		0.036		"
<b>Soil</b>						
Sweden	soil (humus)	g/m <sup>2</sup>		1.6		Lindqvist et al. 1991
Sweden	soil (B hor.)	g/m <sup>2</sup>		5.6		Lindqvist et al. 1991
Sweden	peat 0-4 cm	µg/g-dry	0.033-0.139	0.076	0.072	Jensen & Jensen, 1991
	peat >4 cm	µg/g-dry	0.012-0.170	0.075	0.065	"
US: ?	soil	µg/g	10-150			D'Itri, 1972
US: ?	soil	µg/g	BD-0.20			Trost & Bisque, 1970
US: MN, forest	soil, organic	µg/g		0.117		Fitzgerald et al. 1992*
	soil, 0-10 cm	µg/g		0.029		"
	soil, 10-50 cm	µg/g		0.008		"
US: TN (contaminated)	floodplain soil	µg/g	0.50-3 x 10 <sup>3</sup> (insoluble Hg)			Revis et al. 1990
US: Atlantic, NJ	soils	µg/g	<0.062-<0.089			NJDEPE, unpubl.

\* data is from an abstract from work presented at International Conference on Mercury as a Global Pollutant in Monterey, CA, 1992.

Table A-5: Ambient Mercury Concentrations in Biota

Location	Sample Type	Mercury Units	Range	Mean	Median	Reference
<b>Plankton</b>						
Sweden (25 lakes)	plankton	µg/g dry	0.1-0.7			Lindqvist et al. 1991
Finland	zooplankton	µg/g-dry	0.06-0.19			Rask & Metsala, 1992*
US: MN	zooplankton	µg/g	0.009-0.209	0.088	0.086	Sorenson et al. 1990
US: MN	plankton	µg/g	50-200			Glass et al. 1992
<b>Crayfish</b>						
Canada	contaminated	µg/g	0.8-1.5			Parks, 1991
	uncont.	µg/g	0.05-0.2			Parks, 1991
Finland	macroinvert.	µg/g-dry	0.1-1.4			Rask & Metsala, 1992*
<b>Insects</b>						
US: MS	mayflies ♀	µg/g-dry	0.044-0.102	0.074		Dukerschein et al. 1992
"	mayflies ♂	"	0.060-0.177	0.100		"
<b>Eels</b>						
US: Warren, NJ	muscle	µg/g	0.021-0.280	0.10	0.085	Greenberg et al. unpubl.
US: Warren, NJ	liver	µg/g	0.01-0.181	0.072	0.072	Greenberg et al. unpubl.
<b>Saltwater Fish</b>						
North Sea	porpoise-musc.	µg/g-dry	0.21-24.3	5.03	3.06	Joiris et al. 1990
	" - liver	µg/g-dry	1.15-504	75.45	7.33	"
	" - kidney	µg/g-dry	0.56-25.6	8.41	5.04	"
US: PR	Humacao-fillet	µg/g-wet	0.092-0.460	0.113-0.176		Burger et al. 1992a
"	" - liver	"		0.08-0.133		"
US: PR	assorted spp.	µg/g	0.025-1.29			"
US: MA (contaminated)	flounder eggs	µg/g-dry	0.051-0.226	0.093		Vitaliano & Zolanowicz, 1992
Italy	mar. organisms	µg/g	0.8-17			Barghigiani & Ristori, 1992*
<b>Freshwater Fish</b>						
US: MN, upstream of MWC	"fish"	µg/g		0.36		Glass et al. 1991
US: MN, downstream of MWC	"fish"	µg/g		0.36		Glass et al. 1991
US: MN	Pike	µg/g	0.140-1.52	0.439	0.390	Sorenson et al. 1990
US: MN & WI	Pike (0-5)	µg/g-wet	0.07-1.64	0.33	0.43	Grieb et al. 1990
"	Pike (6-8+)	ug/g-wet	0.15-1.12	0.65	0.69	Grieb et al. 1990
Finland	Northern pike	µg/g-dry		3.6		Rask & Metsala, 1992*
Canada (near smelter)	Nor.pike-liver	µg/g-dry	0.03-0.88	0.03-0.57	0.05-0.62	Harrison & Kleverkamp, 1990
"	" - muscle	"	0.05-0.69	0.09-0.47	0.09-0.49	"
Canada (pristine)	" - muscle	"	0.26-1.90	1.01		"
Canada (near smelter)	White sucker, liver"	"	0.003-0.05	0.02-0.05	0.02-0.05	Harrison & Kleverkamp, 1990
"	"- muscle	"	0.02-0.10	0.02-0.06	0.02-0.06	"
Canada (pristine)	White sucker-liver	"			0.003-0.06	"

Table A-5 (continued)

"	"-muscle	"		0.08-0.17		"
US: MN	Walleye	µg/g	0.120-1.75			Sorenson et al. 1990
US: PA (2-5 years old)	Walleye-whole	µg/g-wet	0.19-0.94	0.45	0.23	Spotts & Rice, 1992
"	" - fillet	"	0.21-1.33	0.59	0.30	"
Russia (pH > 6)	Perch	µg/g		0.2		Haines et al. 1992*
" (pH < 6)	"	"		0.1-3.0		"
US: MN	Yellow perch	µg/g	0.0190.046	0.030		Glass et al. 1992
Canada (contaminated)	Yellow perch	µg/g	0.35-0.42			Parks, 1991
Canada (uncontaminated)	Yellow perch	µg/g	0.02-0.04			Parks, 1991
US: MN & WI	Y. perch (0-5)	µg/g-wet	<0.01-0.84	0.23	0.23	Grieb et al. 1990
"	Y. perch (6-10+)	"	<0.01-2.36	0.53	0.49	Grieb et al. 1990
US: WI	Y. perch-whole	µg/g-wet	0.03-0.29	0.08-0.15		Cope et al. 1990
US: NJ	Yellow perch	µg/g-wet	0.07-1.14			Sprenger et al. 1987
US: PA (2-5 years old)	Y. Perch-fillet	µg/g-wet	0.21-0.28	0.25	0.26	Spotts & Rice, 1992
Finland	European perch	µg/g-dry		1.8		Rask & Metsala, 1992*
US: MN	Log perch	µg/g	0.024-0.075	0.040		Glass et al. 1992
"	Spottail shiner	µg/g	0.038-0.134	0.060		Glass et al. 1992
"	Emerald shiner	µg/g	0.036-0.191	0.080		Glass et al. 1992
"	Johnny darter	µg/g	0.026-0.047	0.034		Glass et al. 1992
US: MN & WI	White sucker (0-5)	µg/g wet	<0.01-0.49	0.09	0.07	Grieb et al. 1990
"	" (6-9)	µg/g wet	<0.01-0.59	0.25	0.16	Grieb et al. 1990
"	Largemouth bass (0-5)	"				
"	" (6-10)	µg/g-wet	0.06-0.81	0.26	0.23	Grieb et al. 1990
"	"	µg/g-wet	0.29-1.00	0.63	0.64	Grieb et al. 1990
US: PA (2-5 years old)	L. bass-whole	µg/g-wet	0.06-0.22	0.13	0.15	Spotts & Rice, 1992
"	" - fillet	"	0.12-0.49	0.24	0.22	"
US: NJ	L. bass-fillet	µg/g-wet	0.135-0.397	0.230-0.303	0.277	Stevenson et al. unpubl.
US: CA	L. bass-fillet	µg/g		0.19-0.92		Stratton et al. 1987
US: CA	S. bass-fillet	µg/g		0.23		Stratton et al. 1987
US: PA (2-5 years old)	S. bass-whole	µg/g-wet		0.20		Spotts & Rice, 1992
"	" - fillet	"		0.33		"
US: PA (2-5 years old)	Chain pickerel	µg/g-wet	0.30-0.39	0.35	0.35	Spotts & Rice, 1992
"	" - fillet	"	0.25-0.58	0.45	0.52	"
US: NY	Lake trout(1-12)	µg/g-dry		0.19-0.75		Gutenmann et al. 1992
US: CA (fillet)	Rainbow trout	µg/g		0.17		Stratton et al. 1987
US: CA	White croaker	µg/g-wet	<0.05-0.724			CAEPA, 1991
US: NJ	Killie-fillet	µg/g-wet	0.010-0.036	0.013-0.032	0.022	Stevenson et al. unpubl.
US: CA (fillet)	Channel catfish	µg/g		0.36-0.57		Stratton et al. 1987
US: CA (fillet)	White catfish	"		0.53-0.75		Stratton et al. 1987
US: CA (fillet)	B&W crappie	µg/g		0.38		Stratton et al. 1987
US: CA (fillet)	Brown bullhead		0.25-0.26			Stratton et al. 1987

Table A-5 (continued)

**Birds & Feathers**

US: MN	loon-liver	µg/g-wet	0.17-83	3.60	4.70	Ensor et al. 1992
"	"-feather	"	0.05-29	3.21	3.10	"
US: NY	tern-feather	µg/g	1.68-2.61			Burger & Gochfeld, 1992
US: HA (breast feathers)	sooty tern	µg/g-dry		0.769		Burger et al. 1992b
"	brown noddy	"		0.460		"
"	wedgetailed shearwater			3.851		"
US: HA Atoll (breast feathers)	sooty tern	µg/g-dry		0.984		"
	brown noddy			1.296		"
	wedgetailed shearwater			1.908		"
Portugal (breast feathers)	?	µg/g		1.1-19.3		Monteiro et al. 1992*

**Aquatic Vegetation**

US: WI-spring/summer	aufwuchs (algae)	ng/m <sup>2</sup>	60-940			Cope & Rada, 1992
US: WI-summer/fall	"	ng/m <sup>2</sup>	250-2130			"

**Terrestrial Vegetation**

Czechoslovakia (contaminated)	mushrooms	µg/g-dry		0.3-6.5		Kalavc et al. 1991
" (uncontaminated)	"	"		0.2-11		"
Italy	pine needles	µg/g	0.038-0.213			Barghigiani & Ristori, 1992*
Italy	broom twigs	µg/g	0.82-1.17			Barghigiani & Ristori, 1992*
Italy	lichens	µg/g	45-49.5			Barghigiani & Ristori, 1992*
Slovenia (contaminated)	lichens	µg/g-dry	2-48			Lupsina & Horvat, 1992*
Slovenia (pristine)	lichens	µg/g-dry	0.2-0.4			Lupsina & Horvat, 1992*
Finland	moss	µg/g-dry	0.06-0.09			Rask & Metsala, 1992*
US: NY (upwind MSW)	grasses	µg/g-dry	<0.01-0.11	0.04	0.30	Bache et al. 1991
" (downwind MSW)	"	"	0.01-0.20	0.07	0.09	"
US: FL (leaves)	sugarcane	µg/g-dry		0.068 - dead		Patrick & Gambrell, 1992*
	"	"		0.085 - live		"

**Other**

Czechoslovakia	feeding yeast	µg/g	0.008-0.187	0.025-0.067	0.021-0.064	Cibulta et al. 1992
China	fillet (sp ?)	µg/g-wet		0.012		Bloom & Liang, 1992*

\* data is from an abstract from work presented at International Conference on Mercury as a Global Pollutant in Monterey, CA, 1992.

Table A-6: Mercury Deposition Rates

Location	wet or dry	Mercury Units	Range	Mean	Median	Reference
Sweden	wet	$\mu\text{g}/\text{m}^2/\text{yr}$	2.5-35.1	14.2	10.9	Lindqvist et al. 1991
"	in runoff	$\mu\text{g}/\text{m}^2/\text{yr}$		144 $\pm$ 70 (methyl)		Lee & Hultberg, 1990
Canada: Ontario	wet	$\mu\text{g}/\text{m}^2/\text{yr}$	30-60			Johnson, 1987
Canada: Harp Lake	wet	$\mu\text{g}/\text{m}^2/\text{yr}$		4.79		Mierle, 1990
US: MN (80 lakes)	wet	$\mu\text{g}/\text{m}^2/\text{yr}$	10.4-15.4	12.6	12.7	Sorensen et al. 1990
US: MN (3 sites)	wet	$\mu\text{g}/\text{m}^2/\text{yr}$	6.5-41.9	19.2	16.7	Glass et al. 1991
US: AK	?	$\mu\text{g}/\text{m}^2/\text{yr}$		5		Engstrom et al. 1992*
US: MN	?	$\mu\text{g}/\text{m}^2/\text{yr}$		15		"
US: WI	wet	$\mu\text{g}/\text{m}^2/\text{yr}$		6.6		Fitzgerald et al. 1992*
US: WI	dry	$\mu\text{g}/\text{m}^2/\text{yr}$		4.4		"
US: MN	wet	$\mu\text{g}/\text{m}^2/\text{yr}$	5.1-10.0			Sorensen et al. 1992*

\* data is from an abstract from work presented at International Conference on Mercury as a Global Pollutant in Monterey, CA, 1992.

Table A-7: Speciation of Mercury in Environmental Media

Media	Mercury Units	Total Mercury	Hg <sup>+2</sup>	Organic Mercury	% Organic	Reference
rain	ng/L	4-3400			54-80 (73)	Lindqvist et al. 1991
rain	ng/L	2.8-15.13	0.17-10.8	0.035-0.350	2-5	Bloom & Watras, 1989
snow	ng/L	4.05	0.52-1.9	0.015-0.043	2-5	"
lake water	ng/L			0.09-0.32		Lee & Hultberg, 1990
lake water	ng/L	1.9-5.83		1.18-2.53	5.7-13	Lee & Hultberg, 1990
lake water	ng/L	3.4	2.1	1.3	38	Schintu & Kudo, 1989
lakes (10)	% total				27-87	Gill & Bruland, 1990
	% dissolved			17-89	"	"
lakes (25): Spring	ug/m <sup>3</sup>	3.7	3.4			Lindqvist et al. 1991
" : Summer	"	3.1	2.1			"
" : Fall	"	6.2	3.4			"
lakes (25)	ug/m <sup>3</sup>	2-12		0.04-0.76	<1-38	Lindqvist et al. 1991
lake	ng/L	0.16-10.7	0.07-0.52	<0.004-0.636	1-9	Bloom & Watras, 1989
lake	ug/g	3.42-14.5		0.1-0.021	1-3	Bloom & Liang, 1992*
suspended part.	ug/g-dry	2-59		0.05-2.7	2-8	Wilken & Hint., 1991
runoff water	ng/L			0.19-1.99	5-27	Verta et al. 1992*
rivers/lakes	ng/L			1.3-4.1	22-37	Schintu et al. 1989
river water	ng/L	7.7-9.0	6-6.7	1.7-2.3	19-30	Schintu & Kudo, 1989
river water	ng/L	11.4	7.3	4.1	36	Schintu & Kudo, 1989
freshwater	%			pH 4-6.....	90-98	Lindqvist & Rodhe, 1985
"	%			pH 7-8.....	2-34	"
surface water	%				92	"
ground water (pristine)	ng/L	2-27	2-27	BD	0	Windom et al. 1992
ground water (all)	ng/L	2-36,400	2-36,100	BD-2.4	<1-3	"
tapwater	ng/L	3.9	2.9	1.0	26	Schintu et al. 1989
bed sediments					0.1-10	Schintu et al. 1989
lake sediments	ng/g-wet	1-75		0-1.3	0-7,37	Gilmour, 1992*
wetlands	ng/L	1-7		2	30	Rudd, et al. 1992*
lichens, polluted	ug/g-dry	17-188		23-106		Lupsina & Horv, 1992*
aquatic plants					28	Schintu et al. 1989
invertebrates					40	"
zooplankton					45	"
fish					70-90	"
zooplankton	ug/L dry	0.1-0.7			30-90	Lindqvist et al. 1991

Table A-7 (continued)

porpoises- muscle	μg/g-dry	2.62-24.3	2.27-10.75	30-100	Joiris, 1990
" - liver	μg/g-dry	1.15-504	0.85-8.92	2-100	"

\* data is from an abstract from work presented at International Conference on Mercury as a Global Pollutant in Monterey, CA, 1992.

**APPENDIX B**

**FISH-TO-SEDIMENT RATIOS  
FROM THE EQUATION OF HÅKANSON**

APPENDIX B.  
Fish-to-Sediment Ratios from the Equation of Håkanson (1980)

Table B-1  
Levels of Mercury in Fish and Fish-to-Sediment Ratios for pH = 4 (Håkanson)

Hg in sediments: (ng/g, dry wt)	50	100	200	300	500	1000
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MERCURY LEVEL IN FISH (mg/kg, wet wt.):

BPI	50	100	200	300	500	1000
2	0.77	1.40	2.40	3.17	4.34	6.20
3	0.49	0.89	1.51	2.00	2.74	3.91
4	0.39	0.70	1.20	1.59	2.17	3.10
5	0.33	0.60	1.03	1.37	1.87	2.67
6	0.30	0.54	0.93	1.23	1.68	2.40
7	0.28	0.50	0.85	1.13	1.55	2.21
8	0.26	0.47	0.80	1.06	1.45	2.07
9	0.24	0.44	0.76	1.00	1.37	1.96
10	0.23	0.42	0.72	0.96	1.31	1.87

FISH/SEDIMENT RATIO:

BPI	50	100	200	300	500	1000
2	15.5	14.0	12.0	10.6	8.7	6.2
3	9.7	8.9	7.6	6.7	5.5	3.9
4	7.7	7.0	6.0	5.3	4.3	3.1
5	6.7	6.0	5.2	4.6	3.7	2.7
6	6.0	5.4	4.6	4.1	3.4	2.4
7	5.5	5.0	4.3	3.8	3.1	2.2
8	5.2	4.7	4.0	3.5	2.9	2.1
9	4.9	4.4	3.8	3.3	2.7	2.0
10	4.7	4.2	3.6	3.2	2.6	1.9

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BPI = bioproduction index.

Table B-2  
 Levels of Mercury in Fish and Fish-to-Sediment Ratios for pH = 5 (Håkanson)

Hg in sediments:            50            100            200            300            500            1000  
 (ng/g, dry wt)

MERCURY LEVEL IN FISH (mg/kg, wet wt.):

BPI	50	100	200	300	500	1000
2	0.52	0.94	1.60	2.12	2.89	4.14
3	0.32	0.59	1.01	1.33	1.82	2.61
4	0.26	0.47	0.80	1.06	1.45	2.07
5	0.22	0.40	0.69	0.91	1.25	1.78
6	0.20	0.36	0.62	0.82	1.12	1.60
7	0.18	0.33	0.57	0.75	1.03	1.47
8	0.17	0.31	0.53	0.71	0.96	1.38
9	0.16	0.30	0.50	0.67	0.91	1.30
10	0.16	0.28	0.48	0.64	0.87	1.25

FISH/SEDIMENT RATIO:

BPI	50	100	200	300	500	1000
2	10.3	9.4	8.0	7.1	5.8	4.1
3	6.5	5.9	5.0	4.4	3.6	2.6
4	5.2	4.7	4.0	3.5	2.9	2.1
5	4.4	4.0	3.4	3.0	2.5	1.8
6	4.0	3.6	3.1	2.7	2.2	1.6
7	3.7	3.3	2.8	2.5	2.1	1.5
8	3.4	3.1	2.7	2.4	1.9	1.4
9	3.2	3.0	2.5	2.2	1.8	1.3
10	3.1	2.8	2.4	2.1	1.7	1.2

Table B-3  
Levels of Mercury in Fish and Fish-to-Sediment Ratios for pH = 6 (Håkanson)

Hg in sediments:                    50            100            200            300            500            1000  
(ng/g, dry wt)

MERCURY LEVEL IN FISH (mg/kg, wet wt.):

BPI	50	100	200	300	500	1000
2	0.39	0.70	1.20	1.59	2.17	3.10
3	0.24	0.44	0.76	1.00	1.37	1.96
4	0.19	0.35	0.60	0.79	1.08	1.55
5	0.17	0.30	0.52	0.68	0.93	1.34
6	0.15	0.27	0.46	0.61	0.84	1.20
7	0.14	0.25	0.43	0.57	0.77	1.10
8	0.13	0.23	0.40	0.53	0.72	1.03
9	0.12	0.22	0.38	0.50	0.68	0.98
10	0.12	0.21	0.36	0.48	0.65	0.93

FISH/SEDIMENT RATIO:

BPI	50	100	200	300	500	1000
2	7.7	7.0	6.0	5.3	4.3	3.1
3	4.9	4.4	3.8	3.3	2.7	2.0
4	3.9	3.5	3.0	2.6	2.2	1.6
5	3.3	3.0	2.6	2.3	1.9	1.3
6	3.0	2.7	2.3	2.0	1.7	1.2
7	2.8	2.5	2.1	1.9	1.5	1.1
8	2.6	2.3	2.0	1.8	1.4	1.0
9	2.4	2.2	1.9	1.7	1.4	1.0
10	2.3	2.1	1.8	1.6	1.3	0.9

Table B-4  
 Levels of Mercury in Fish and Fish-to-Sediment Ratios for pH = 7 (Håkanson)

Hg in sediments:            50            100            200            300            500            1000  
 (ng/g, dry wt)

MERCURY LEVEL IN FISH (mg/kg, wet wt.):

BPI	50	100	200	300	500	1000
2	0.31	0.56	0.96	1.27	1.74	2.48
3	0.19	0.35	0.61	0.80	1.09	1.57
4	0.15	0.28	0.48	0.63	0.87	1.24
5	0.13	0.24	0.41	0.55	0.75	1.07
6	0.12	0.22	0.37	0.49	0.67	0.96
7	0.11	0.20	0.34	0.45	0.62	0.88
8	0.10	0.19	0.32	0.42	0.58	0.83
9	0.10	0.18	0.30	0.40	0.55	0.78
10	0.09	0.17	0.29	0.38	0.52	0.75

FISH/SEDIMENT RATIO:

BPI	50	100	200	300	500	1000
2	6.2	5.6	4.8	4.2	3.5	2.5
3	3.9	3.5	3.0	2.7	2.2	1.6
4	3.1	2.8	2.4	2.1	1.7	1.2
5	2.7	2.4	2.1	1.8	1.5	1.1
6	2.4	2.2	1.9	1.6	1.3	1.0
7	2.2	2.0	1.7	1.5	1.2	0.9
8	2.1	1.9	1.6	1.4	1.2	0.8
9	1.9	1.8	1.5	1.3	1.1	0.8
10	1.9	1.7	1.4	1.3	1.0	0.7

Table B-5  
 Levels of Mercury in Fish and Fish-to-Sediment Ratios for pH = 8 (Håkanson)

Hg in sediments: (ng/g, dry wt)	50	100	200	300	500	1000
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MERCURY LEVEL IN FISH (mg/kg, wet wt.):

BPI						
2	0.26	0.47	0.80	1.06	1.45	2.07
3	0.16	0.30	0.50	0.67	0.91	1.30
4	0.13	0.23	0.40	0.53	0.72	1.03
5	0.11	0.20	0.34	0.46	0.62	0.89
6	0.10	0.18	0.31	0.41	0.56	0.80
7	0.09	0.17	0.28	0.38	0.52	0.74
8	0.09	0.16	0.27	0.35	0.48	0.69
9	0.08	0.15	0.25	0.33	0.46	0.65
10	0.08	0.14	0.24	0.32	0.44	0.62

FISH/SEDIMENT RATIO:

BPI						
2	5.2	4.7	4.0	3.5	2.9	2.1
3	3.2	3.0	2.5	2.2	1.8	1.3
4	2.6	2.3	2.0	1.8	1.4	1.0
5	2.2	2.0	1.7	1.5	1.2	0.9
6	2.0	1.8	1.5	1.4	1.1	0.8
7	1.8	1.7	1.4	1.3	1.0	0.7
8	1.7	1.6	1.3	1.2	1.0	0.7
9	1.6	1.5	1.3	1.1	0.9	0.7
10	1.6	1.4	1.2	1.1	0.9	0.6

**APPENDIX C**

**FISH-TO-SEDIMENT RATIOS  
FROM THE EQUATION OF LINDQVIST**

APPENDIX C.  
 Fish-to-Sediment Ratios From the Equation of Lindqvist et al. (1991)  
 (From Anderson et al. 1987)

Table C-1  
 Levels of Mercury in Fish and Fish-to-Sediment Ratios for pH = 4 (Lindqvist)

Hg in sediments ( $\mu\text{g}/\text{kg}$ , dry wt.)	50	100	200	300	500	1000	2000
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MERCURY LEVEL IN FISH (mg/kg, wet wt.):

Conductivity (mS/meter)							
1	2.47	2.50	2.56	2.62	2.75	3.05	3.66
3	2.05	2.08	2.14	2.21	2.33	2.63	3.24
5	1.86	1.89	1.95	2.01	2.13	2.44	3.05
10	1.60	1.63	1.69	1.75	1.87	2.18	2.79
25	1.25	1.28	1.34	1.40	1.52	1.83	2.44
50	0.98	1.01	1.08	1.14	1.26	1.56	2.17
100	0.72	0.75	0.81	0.87	1.00	1.30	1.91

FISH/SEDIMENT RATIO:

Conductivity (mS/meter)							
1	49.4	25.0	12.8	8.7	5.5	3.1	1.8
3	41.1	20.8	10.7	7.4	4.7	2.6	1.6
5	37.2	18.9	9.8	6.7	4.3	2.4	1.5
10	31.9	16.3	8.4	5.8	3.7	2.2	1.4
25	24.9	12.8	6.7	4.7	3.0	1.8	1.2
50	19.7	10.1	5.4	3.8	2.5	1.6	1.1
100	14.4	7.5	4.1	2.9	2.0	1.3	1.0

Table C-2  
 Levels of Mercury in Fish and Fish-to-Sediment Ratios for pH = 5 (Lindqvist)

Hg in sediments  
 (µg/kg, dry wt.)      50      100      200      300      500      1000      2000

MERCURY LEVEL IN FISH (mg/kg, wet wt.):

Conductivity (mS/meter)	50	100	200	300	500	1000	2000
1	2.03	2.06	2.12	2.18	2.31	2.61	3.22
3	1.61	1.64	1.70	1.77	1.89	2.19	2.80
5	1.42	1.45	1.51	1.57	1.69	2.00	2.61
10	1.16	1.19	1.25	1.31	1.43	1.74	2.35
25	0.81	0.84	0.90	0.96	1.08	1.39	2.00
50	0.54	0.57	0.64	0.70	0.82	1.12	1.73
100	0.28	0.31	0.37	0.43	0.56	0.86	1.47

FISH/SEDIMENT RATIO:

Conductivity (mS/meter)	50	100	200	300	500	1000	2000
1	40.6	20.6	10.6	7.3	4.6	2.6	1.6
3	32.3	16.4	8.5	5.9	3.8	2.2	1.4
5	28.4	14.5	7.6	5.2	3.4	2.0	1.3
10	23.1	11.9	6.2	4.4	2.9	1.7	1.2
25	16.1	8.4	4.5	3.2	2.2	1.4	1.0
50	10.9	5.7	3.2	2.3	1.6	1.1	0.9
100	5.6	3.1	1.9	1.4	1.1	0.9	0.7

Table C-3

Levels of Mercury in Fish and Fish-to-Sediment Ratios for pH = 5.5 (Lindqvist)

Hg in sediments ( $\mu\text{g}/\text{kg}$ , dry wt.)	50	100	200	300	500	1000	2000
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MERCURY LEVEL IN FISH (mg/kg, wet wt.):

Conductivity  
(mS/meter)

1	1.81	1.84	1.90	1.96	2.09	2.39	3.00
3	1.39	1.42	1.48	1.55	1.67	1.97	2.58
5	1.20	1.23	1.29	1.35	1.47	1.78	2.39
10	0.94	0.97	1.03	1.09	1.21	1.52	2.13
25	0.59	0.62	0.68	0.74	0.86	1.17	1.78
50	0.32	0.35	0.42	0.48	0.60	0.90	1.51
100	0.06	0.09	0.15	0.21	0.34	0.64	1.25

FISH/SEDIMENT RATIO:

Conductivity  
(mS/meter)

1	36.2	18.4	9.5	6.5	4.2	2.4	1.5
3	27.9	14.2	7.4	5.2	3.3	2.0	1.3
5	24.0	12.3	6.5	4.5	2.9	1.8	1.2
10	18.7	9.7	5.1	3.6	2.4	1.5	1.1
25	11.7	6.2	3.4	2.5	1.7	1.2	0.9
50	6.5	3.5	2.1	1.6	1.2	0.9	0.8
100	1.2	0.9	0.8	0.7	0.7	0.6	0.6

Table C-4  
Levels of Mercury in Fish and Fish-to-Sediment Ratios for pH = 6 (Lindqvist)

Hg in sediments ( $\mu\text{g}/\text{kg}$ , dry wt.)	50	100	200	300	500	1000	2000
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MERCURY LEVEL IN FISH (mg/kg, wet wt.):

Conductivity  
(mS/meter)

1	1.59	1.62	1.68	1.74	1.87	2.17	2.78
3	1.17	1.20	1.26	1.33	1.45	1.75	2.36
5	0.98	1.01	1.07	1.13	1.25	1.56	2.17
10	0.72	0.75	0.81	0.87	0.99	1.30	1.91
25	0.37	0.40	0.46	0.52	0.64	0.95	1.56
50	0.10	0.13	0.20	0.26	0.38	0.68	1.29
100	-0.16	-0.13	-0.07	-0.01	0.12	0.42	1.03

FISH/SEDIMENT RATIO:

Conductivity  
(mS/meter)

1	31.8	16.2	8.4	5.8	3.7	2.2	1.4
3	23.5	12.0	6.3	4.4	2.9	1.8	1.2
5	19.6	10.1	5.4	3.8	2.5	1.6	1.1
10	14.3	7.5	4.0	2.9	2.0	1.3	1.0
25	7.3	4.0	2.3	1.7	1.3	0.9	0.8
50	2.1	1.3	1.0	0.9	0.8	0.7	0.6
100	-3.2	-1.3	-0.3	0.0	0.2	0.4	0.5

Table C-5  
Levels of Mercury in Fish and Fish-to-Sediment Ratios for pH = 6.5 (Lindqvist)

Hg in sediments ( $\mu\text{g}/\text{kg}$ , dry wt.)	50	100	200	300	500	1000	2000
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MERCURY LEVEL IN FISH (mg/kg, wet wt.):

Conductivity  
(mS/meter)

1	1.37	1.40	1.46	1.52	1.65	1.95	2.56
3	0.95	0.98	1.04	1.11	1.23	1.53	2.14
5	0.76	0.79	0.85	0.91	1.03	1.34	1.95
10	0.50	0.53	0.59	0.65	0.77	1.08	1.69
25	0.15	0.18	0.24	0.30	0.42	0.73	1.34
50	-0.12	-0.09	-0.02	0.04	0.16	0.46	1.07
100	-0.38	-0.35	-0.29	-0.23	-0.10	0.20	0.81

FISH/SEDIMENT RATIO:

Conductivity  
(mS/meter)

1	27.4	14.0	7.3	5.1	3.3	2.0	1.3
3	19.1	9.8	5.2	3.7	2.5	1.5	1.1
5	15.2	7.9	4.3	3.0	2.1	1.3	1.0
10	9.9	5.3	2.9	2.2	1.5	1.1	0.8
25	2.9	1.8	1.2	1.0	0.8	0.7	0.7
50	-2.3	-0.9	-0.1	0.1	0.3	0.5	0.5
100	-7.6	-3.5	-1.4	-0.8	-0.2	0.2	0.4

Table C-6

Levels of Mercury in Fish and Fish-to-Sediment Ratios for pH = 7 (Lindqvist)

Hg in sediments ( $\mu\text{g}/\text{kg}$ , dry wt.)	50	100	200	300	500	1000	2000
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MERCURY LEVEL IN FISH (mg/kg, wet wt.):

Conductivity (mS/meter)							
----------------------------	--	--	--	--	--	--	--

1	1.15	1.18	1.24	1.30	1.43	1.73	2.34
3	0.73	0.76	0.82	0.89	1.01	1.31	1.92
5	0.54	0.57	0.63	0.69	0.81	1.12	1.73
10	0.28	0.31	0.37	0.43	0.55	0.86	1.47
25	-0.07	-0.04	0.02	0.08	0.20	0.51	1.12
50	-0.34	-0.31	-0.24	-0.18	-0.06	0.24	0.85
100	-0.60	-0.57	-0.51	-0.45	-0.32	-0.02	0.59

FISH/SEDIMENT RATIO:

Conductivity (mS/meter)							
----------------------------	--	--	--	--	--	--	--

1	23.0	11.8	6.2	4.3	2.9	1.7	1.2
3	14.7	7.6	4.1	3.0	2.0	1.3	1.0
5	10.8	5.7	3.2	2.3	1.6	1.1	0.9
10	5.5	3.1	1.8	1.4	1.1	0.9	0.7
25	-1.5	-0.4	0.1	0.3	0.4	0.5	0.6
50	-6.7	-3.1	-1.2	-0.6	-0.1	0.2	0.4
100	-12.0	-5.7	-2.5	-1.5	-0.6	0.0	0.3

Table C-7

Levels of Mercury in Fish and Fish-to-Sediment Ratios for pH = 8 (Lindqvist)

Hg in sediments ( $\mu\text{g}/\text{kg}$ , dry wt.)	50	100	200	300	500	1000	2000
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MERCURY LEVEL IN FISH (mg/kg, wet wt.):

Conductivity (mS/meter)							
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1	0.71	0.74	0.80	0.86	0.99	1.29	1.90
3	0.29	0.32	0.38	0.45	0.57	0.87	1.48
5	0.10	0.13	0.19	0.25	0.37	0.68	1.29
10	-0.16	-0.13	-0.07	-0.01	0.11	0.42	1.03
25	-0.51	-0.48	-0.42	-0.36	-0.24	0.07	0.68
50	-0.78	-0.75	-0.68	-0.62	-0.50	-0.20	0.41
100	-1.04	-1.01	-0.95	-0.89	-0.76	-0.46	0.15

FISH/SEDIMENT RATIO:

Conductivity (mS/meter)							
----------------------------	--	--	--	--	--	--	--

1	14.2	7.4	4.0	2.9	2.0	1.3	1.0
3	5.9	3.2	1.9	1.5	1.1	0.9	0.7
5	2.0	1.3	1.0	0.8	0.7	0.7	0.6
10	-3.3	-1.3	-0.4	0.0	0.2	0.4	0.5
25	-10.3	-4.8	-2.1	-1.2	-0.5	0.1	0.3
50	-15.5	-7.5	-3.4	-2.1	-1.0	-0.2	0.2
100	-20.8	-10.1	-4.7	-3.0	-1.5	-0.5	0.1

**APPENDIX D**

**SEDIMENT-TO-FISH BIOCONCENTRATION FACTORS**

APPENDIX D.  
Sediment-to-Fish Bioconcentration Factors  
Ranges for Conditions in New Jersey Lakes

For this report, bioconcentration factors for fish were derived using parameters selected to represent New Jersey lakes. Parameters used in the model in Chapter XIII represent ranges of values reported in this state. The results described in this report incorporate conditions that exist in New Jersey, but not necessarily the most conservative situation.

We reviewed data collected by NJDEPE (undated), USGS (undated), USEPA (1986), and Sprenger et al. (1988) to establish ranges for pH, conductivity, and bioproduction in New Jersey lakes. These ranges of values can then be used to generate ranges of sediment-to-fish bioconcentration factors from the two equations described above.

In examining the data for pH, we chose to focus on a value in the middle of the range of values presented for each waterbody. This is consistent with the use of the mean pH of the system by Håkanson (1980). The mid-range values for the forty-three lakes monitored by NJDEPE (undated) ranged from approximately 4 (at several lakes in Burlington and Cape May counties) to approximately 8. USGS (undated) data for twenty stream stations just below lakes and reservoirs showed a similar pattern, with mid-range values from approximately 4.5 (in the Batsto, Mullica, and Bass Rivers) to approximately 8. USEPA's (1986) study of lakes throughout the eastern United States included nineteen lakes in northwestern New Jersey, where mid-range pH values were between 7 and 8.5. However, the data of Sprenger et al. (1988) for six lakes on the Kittatinny Ridge in northwestern New Jersey show that low pH values are not restricted to the southern part of the state: pH values in that study ranged from 3.7 to 5.8. On the basis of these data, we chose 4 to 8 as a reasonable range of pH values for New Jersey lakes.

Conductivity readings at the twenty stations monitored by USGS (undated) ranged widely, from approximately 30  $\mu\text{mhos/cm}$  (3 mS/m) for some readings in the Mullica, Batsto, Oswego, Bass, and Rockaway Rivers to approximately 2000  $\mu\text{mhos/cm}$  (200 mS/m). Mid-range values ranged from approximately 50  $\mu\text{mhos/cm}$  (5 mS/m) in the Batsto, Oswego, and Bass Rivers to approximately 1000  $\mu\text{mhos/cm}$  (100 mS/m) in the Elizabeth River. Measured conductivity in the nineteen northwestern New Jersey lakes studied by USEPA (1986) ranged from 3 to approximately 30 mS/m. We chose 3 to 100 mS/m as a reasonable range for lakes in New Jersey.

Bioproduction index (BPI) is much more difficult to determine, since it requires more extensive data collected over several seasons. Håkanson (1980) provides a table showing characteristic

features of lakes with varying trophic status. Parts of that table are reproduced below in Table D-1.

Table D-1  
Characteristic Features of Lakes with Varying Trophic Status\*

<u>Trophic Level</u>	<u>Secchi Depth (Meters)</u>	<u>Chlorophyll a (mg/m<sup>3</sup>)</u>	<u>Total-P (mg/m<sup>3</sup>)</u>	<u>BPI</u>
Oligotrophic	>5	<2	<5	<3.1
Mesotrophic	3-6	2-8	5-20	2.8-4.5
Eutrophic	1-4	6-35	20-100	4.3-6.8
Very eutrophic	0-2	30-400	>100	6.5-10.0

<u>Trophic Level</u>	<u>Summer Secchi Depth (Meters)</u>	<u>Summer Chlorophyll a (mg/m<sup>3</sup>)</u>	<u>Winter Total-P (mg/m<sup>3</sup>)</u>
Oligotrophic	>3-5	≤2-4	≤10-15
Eutrophic	<1.5-2	≥6-10	≤20-50

<u>Parameter</u>	<u>Håkanson(1980)</u>	<u>Lindqvist et al.(1991)</u>	<u>New Jersey</u>
pH	4-8	5.5-7	4-8
Conductivity (mS/m)	--	1-10	3-100
BPI	2-8	--	2-8
Sediment Hg (ng/g)	200-1000	50-2000	<100-800

\* from Håkanson (1980)

Håkanson (1980) also provides a figure that depicts the relationship between BPI and total-P (total phosphorus) for fifteen Swedish lakes. The BPI increases sharply from about 3 for total-P values around 5  $\mu\text{g/l}$  ( $= \text{mg/m}^3$ ), increases more slowly to around 5 for total-P values between 20 and 40  $\mu\text{g/l}$ , and around 6 for a total-P value of 70  $\mu\text{g/l}$ . USEPA's definitions of oligotrophic and eutrophic conditions (B. Cann, NJDEPE Bureau of Water Monitoring, pers. comm.) closely match those of Håkanson (1980).

Total-P levels measured by USEPA (1986) in the nineteen lakes in northwestern New Jersey ranged from 4 to 29  $\text{mg/m}^3$ . The samples were collected in November, and thus may be taken to reflect winter conditions, at least to a greater extent than they do summer conditions. Most of the data for total-P collected by NJDEPE (undated) were for summer conditions, but values for winter (through March) ranged from  $<20 \text{ mg/m}^3$  to as high as 70  $\text{mg/m}^3$ ; values for April were between 20 and 80  $\text{mg/m}^3$ . On the basis of total-P values only, the BPI for New Jersey lakes would appear to range from less than 3 to as high as 6 or 7.

Late fall Secchi depths measured in the USEPA (1986) study ranged from 1.5 to 5 meters, but the EPA definitions require summer measurements. Typical values measured by NJDEPE (undated) ranged from less than 1 meter to 2.1 meters. This latter range suggests that most of the lakes studied were eutrophic according to USEPA's definition, and eutrophic to very eutrophic under the definition of Håkanson (1980), with a BPI from around 4 to greater than 7.

We were able to obtain summertime values for chlorophyll A, the final parameter that we considered in determining the range of BPIs for New Jersey lakes, for only a handful of lakes monitored by NJDEPE (undated). Typical summertime values ranged from around 10  $\text{mg/m}^3$  to over 100  $\text{mg/m}^3$ . Values for other seasons were as low as 3  $\text{mg/m}^3$ . The summertime values put these lakes easily into the eutrophic range defined by USEPA; the full range of values (all seasons) extends over the ranges used by Håkanson (1980) to define mesotrophic, eutrophic, and very eutrophic lakes, with BPIs from around 4 to greater than 7.

Our limited review of data for New Jersey lakes suggests that most lakes in New Jersey are at least moderately eutrophic, but that some lakes may be mesotrophic or perhaps even oligotrophic. On this basis, we chose a range of 2 to 8 for the BPI of New Jersey lakes.

We did not perform a comprehensive review of available data on mercury levels in New Jersey sediments. However, examination of a few unpublished data sources revealed less than 100 ppb mercury in sediment (dry weight) for stations on unpolluted streams just downstream of lakes or reservoirs; levels from 200 ppb to 800 ppb on the Wanaque River, near a known contamination source (USGS,

undated); and from less than 100 ppb to as high as 480 ppb in lakes in Monmouth County (Monmouth County Board of Health 1989, 1990).

The ranges of values used by Håkanson (1980) and Lindqvist et al. (1991) for the various parameters in their equations, and the corresponding ranges for New Jersey lakes, are presented in Table D-2.

We see that there is considerable overlap between the ranges used to derive the predictive equations and the ranges for New Jersey lakes, except for conductivity, one of the independent variables in the equation of Lindqvist et al. (1991).

The bioconcentration factors that result from the two equations are summarized in Table IX-3.

Table D-2

Ranges of Sediment-to-Fish Bioconcentration Factors  
 Derived from the Equations of  
 Håkanson (1980) and Lindqvist et al., (1991)

1) Håkanson (1980) (For BPI = 2 (highest BCFs) to BPI = 8)

Sediment Mercury Concentration (ng/g, dry weight)

<u>pH</u>	50	100	200	300	500	1000	2000
4	NC	NC	4.0-12.0	3.5-10.6	2.9-8.7	2.1-6.2	NC
5	NC	NC	2.7-8.0	2.4-7.1	1.9-5.8	1.4-4.1	NC
5.5	NC	NC	NC	NC	NC	NC	NC
6	NC	NC	2.0-6.0	1.8-5.3	1.4-4.3	1.0-3.1	NC
6.5	NC	NC	NC	NC	NC	NC	NC
7	NC	NC	1.6-4.8	1.4-4.2	1.2-3.5	0.8-2.5	NC
8	NC	NC	1.3-4.0	1.2-3.5	1.0-2.9	0.7-2.1	NC

NC = not calculated.

Values for pH and sediment mercury concentration are the same for both tables to allow easy comparison.

Table D-2  
(continued)

2) Lindqvist et al. (1991) (For conductivity = 1 mS/meter (highest BCFs) to 10 mS/meter)

pH	Sediment Mercury Concentration (ng/g, dry weight)						
	50	100	200	300	500	1000	2000
4	NC	NC	NC	NC	NC		NC NC
5	NC	NC	NC	NC	NC		NC NC
5.5	18.7-36.2	9.7-18.4	5.1-9.5	3.6-6.5	2.4-4.2	1.5-2.4	1.1-1.5
6	14.3-31.8	7.5-16.2	4.0-8.4	2.9-5.8	2.0-3.7	1.3-2.2	1.0-1.4
6.5	9.9-27.4	5.3-14.0	2.9-7.3	2.2-5.1	1.5-3.3	1.1-2.0	0.8-1.3
7	5.5-23.0	3.1-11.8	1.8-6.2	1.4-4.3	1.1-2.9	0.9-1.7	0.7-1.2
8	NC	NC	NC	NC	NC	NC	NC

NC = not calculated.

Values for pH and sediment mercury concentration are the same for both tables to allow easy comparison.

**APPENDIX E**

**MUNICIPAL SOLID WASTE INCINERATOR EMISSIONS**

APPENDIX E.  
Municipal Solid Waste Incinerator Emissions

New Jersey Municipal Solid Waste (MSW) Incinerator Data

NJDEPE has approved the construction and operation of six MSW incinerators within the past six years. They are listed in Table D-1. The Union County MSW incinerator is still under construction; the rest have been built and are operating. It should be noted that the Fort Dix incinerator is much smaller than those built to handle the municipal solid waste for entire counties.

Table E-1  
Permitted New Jersey MSW Incinerators

<u>Facility</u>	<u>Location</u>	<u>Operating Since</u>
Camden County	City of Camden	1991
Essex County	City of Newark	1990
Fort Dix	Fort Dix Military Reservation	1986
Gloucester County	West Deptford Twp.	1990
Union County	City of Rahway	--
Warren County	Oxford Twp.	1988

For more information on the burn rate, air pollution control equipment, stack test data, and other technical aspects of these facilities, see Chapters 2 and 6 of the report of the Technical and Regulatory Issues Subcommittee (Volume III).

Permitted mercury emission rates for the MSW incinerators listed above range from 0.0036 lb/hr (0.0004 g/sec) to 0.24 lb/hr (0.03 g/sec) (see Table E-2). After facility start-up, stack tests are carried out to check the emissions for compliance with the conditions of the facility permits. Results of recent stack tests are included in Table E-2. The Fort Dix MSW incinerator has never been able to meet its permitted emission rates for mercury, even after implementing waste separation. It has been operating under an Administrative Consent Order while additional controls are being evaluated. The Warren County incinerator was initially found to be exceeding its permitted emission rate also, but recent stack tests have shown mercury emissions to be within permitted levels. For detailed stack test histories for each operating incinerator, see Chapter 6 of Volume III of this Task Force Report.

Table E-2  
New Jersey MSW Incinerators - Emission Rates

	Permit (lb/hr)	Permit (g/sec)	Stack Test		Modeled (lb/hr)	Modeled (g/sec)
			Actual Emissions (lb/hr)	(g/sec)		
Camden	0.24	0.03	0.098	0.012	0.31	0.0391
Essex	0.16	0.02	0.064	0.008	0.16	0.0202
Ft. Dix	0.0036	0.0004	0.008	0.001	0.01	0.0020
Gloucester	0.24	0.03	0.127	0.016	0.24	0.0302
Union	---	---	---	---	0.23	0.0290
Warren	0.10	0.13	0.048	0.006	0.10	0.0126

#### Range of Modeled Ambient Air Impact of NJ MSW Incinerators

As part of the permitting process for these MSW incinerators, a dispersion modeling analysis was prepared by each applicant and reviewed by NJDEPE. The emission rates used in the modeling analysis in support of the air pollution control permit were always greater than or equal to the rates that were finally permitted (see Table E-2). In addition to emission rates, the model uses information about the stack and characteristics of the effluent gas which is summarized in Table E-3. Based on this information the modeling predicted that these incinerator emissions would result in maximum 24-hour concentrations of mercury ranging from 0.008 to 0.158  $\mu\text{g}/\text{m}^3$  and maximum annual average emissions of 0.0006 to 0.005  $\mu\text{g}/\text{m}^3$  (see Table E-4). (Twenty-four hour average ambient concentrations are typically based on less variation in meteorological conditions than those used to model annual average concentrations, resulting in 24-hour averages that are greater than the annual averages.) As a validation step, these modeled values are compared against measured ambient air concentrations as reported in the scientific literature. Mercury levels in ambient air in pristine areas is reported to be 0.001-0.003  $\text{ug}/\text{m}^3$  and in developed or urban areas to be 0.003-0.022  $\text{ug}/\text{m}^3$  (see Appendix A). The modeled values for ambient concentrations of mercury in the vicinity of a MSW incinerator fall within these ranges.

These values were used as the starting point for the exposure assessment calculations. Although some of these incinerators were ultimately permitted at emission rates that are significantly lower than the modeled emission rates (i.e., the Camden and Ft. Dix incinerators), these model predictions are still a useful starting point for estimating the ambient air impacts of MSW incinerators. The model results show that, depending on emission rate, stack height, and other variables, the impacts all fall within an order of magnitude of each other. In addition, since ambient concentrations are directly proportional to the emission rate for a given facility, new concentrations can be calculated to reflect

any reductions in emission rate that may be required by a new mercury standard.

Table E-3  
Model Inputs for Permitted NJ MSW Incinerators

	<u>Stack Height (feet)</u>	<u>Effective Stack Diameter (feet)</u>	<u>Exit Velocity (ft/sec)</u>	<u>Exit Temperature (°F)</u>
Camden	365	9.32	66.4	240
Essex	279	13.20	61.35	285
Fort Dix	80	2.67	30.3	146
Gloucester	260	5.66	81.4	243
Union	280	10.80	65	275
Warren	250	4.07	60	240

Table E-4  
Modeling Results for Permitted NJ MSW Incinerators

Modeled Maximum Ambient Concentration

	<u>24-hr (<math>\mu\text{g}/\text{m}^3</math>)</u>	<u>Annual Average (<math>\mu\text{g}/\text{m}^3</math>)</u>
Camden	0.0158	0.002
Essex	0.008	0.0006
Ft. Dix	*	0.0032
Gloucester	0.01	0.0007
Union	0.018	0.0019
Warren	0.1	0.005

\* Not available

## Discussion of Air Quality Modeling Limitations and Uncertainties

Dispersion modeling is a technique used for calculating airborne concentrations of pollutants that are the result of emissions. Computer models can be designed that use a series of equations to estimate air pollutant concentrations resulting from a single source. The models used to determine mercury concentrations assume that a non-reactive material is continuously released from a point source, transported in a downwind direction and dispersed over time. The degree of spread (or dispersion) of a pollutant is dependent on atmospheric stability and local surface features. In addition to dispersion, ground-level pollutant concentrations are dependent on stack gas parameters, such as gas temperature and exit velocity. The USEPA-approved models used in the modeling analysis are classified as Gaussian models. Gaussian models assume that atmospheric dispersion results in cross-sections of pollutant concentrations in both the horizontal and vertical directions which have normal (bell-shaped or Gaussian) distributions.

To account for varying meteorological conditions at each facility, dispersion modeling was carried out using representative meteorological data collected at a nearby National Weather Service station. Meteorological parameters entered into the dispersion models include wind speed and direction, temperature, atmospheric stability, and mixing height, all of which are constantly changing and affecting the dispersion of pollutants.

Pollutant dispersion can also be affected by surface features, such as roughness and terrain elevation. To determine local dispersion characteristics, land use within a 3 kilometer radius of the facility is classified as either urban or rural. Typically, urban areas have a greater surface roughness as well as surfaces that absorb heat. Terrain features are also considered in the model selection process.

Stack parameters considered in the modeling analysis include stack gas exit temperature, exit velocity and stack height. These parameters determine the height that the plume will rise above the stack top, which, combined with meteorological conditions and local dispersion characteristics, determines the ground-level pollutant concentrations.

Theoretical receptors are used by the dispersion models to determine the magnitude and location of the facility's impacts. Receptors are points where pollutant concentrations are calculated. Initially, a coarse receptor grid is used, with receptors placed at regular intervals from the property fenceline out to a distance of 1 kilometer or more, depending on the modeling parameters. Areas within the grid that are calculated to have high impacts are then remodeled with a more dense receptor grid so that the point of

maximum predicted concentration can be determined. The maximum concentration is then used to determine the health-related impacts associated with mercury emission.

The Camden County MSW incinerator was originally modeled with four incinerators, each having its own flue. The four flues were combined, for modeling purposes, into one effective stack with a diameter of 9.3 feet. Only three of these incinerators were actually built in Camden. The final permitted emissions are only about 75% of that which was modeled. However, the effective stack diameter of the facility will be reduced down to 8.1 feet because only three flues exist. This will cause a smaller plume rise and a higher ground-level concentration (for constant emissions). These two effects, lower mercury emissions and a lower plume height, will tend to cancel each other out so that the predicted ground-level concentration of mercury from the three incinerators will be approximately equal to that predicted for four incinerators.