United States
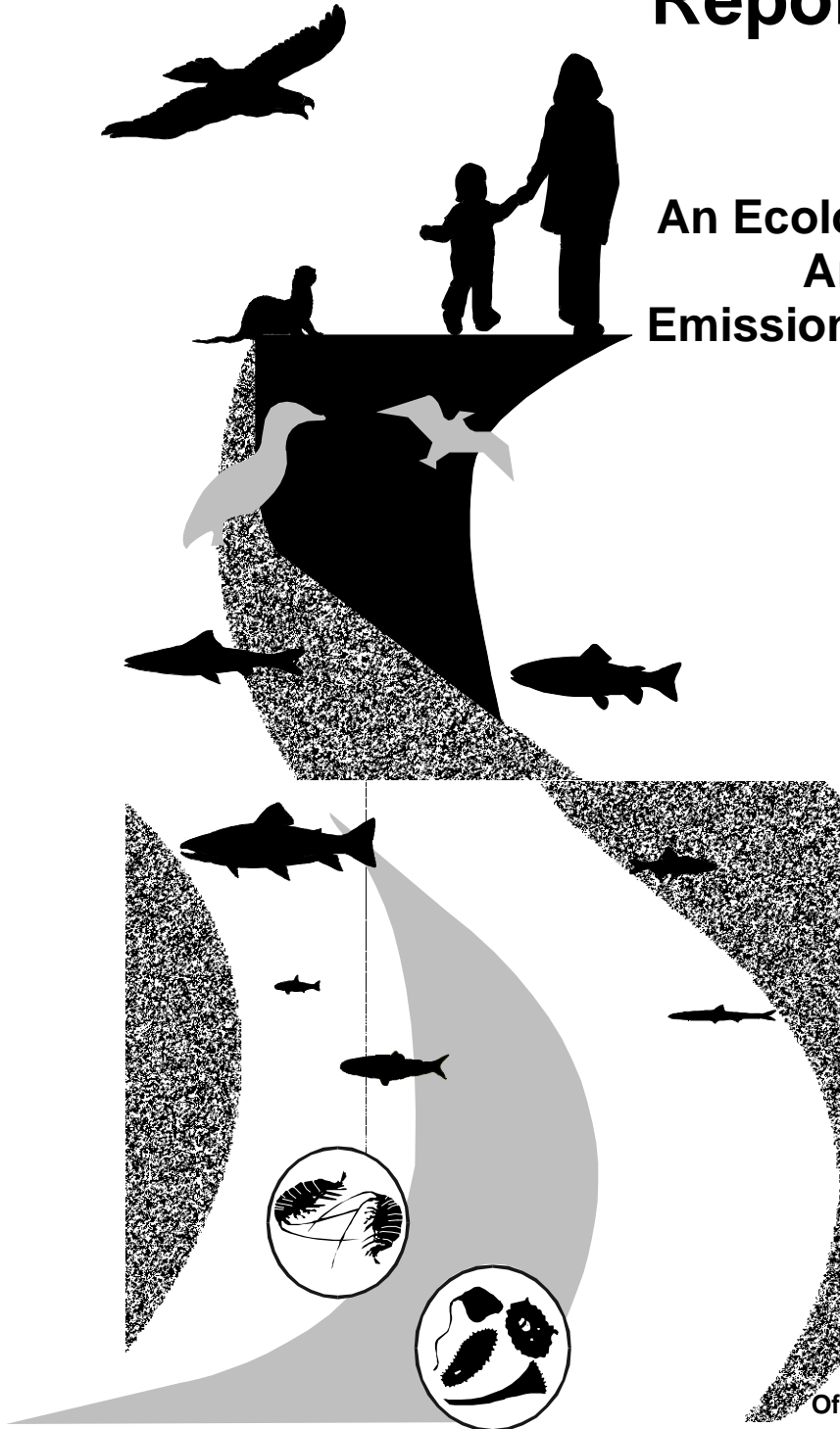Environmental Protection
Agency

Air

EPA-452/R-97-008
December 1997

Mercury Study
Report to Congress

Volume VI:
An Ecological Assessment for
Anthropogenic Mercury
Emissions in the United States

🌱EPA

Office of Air Quality Planning & Standards
and
Office of Research and Development

c7o032-1-1

MERCURY STUDY REPORT TO CONGRESS

VOLUME VI:

**AN ECOLOGICAL ASSESSMENT FOR ANTHROPOGENIC
MERCURY EMISSIONS IN THE UNITED STATES**

December 1997

**Office of Air Quality Planning and Standards
and
Office of Research and Development**

U.S. Environmental Protection Agency

TABLE OF CONTENTS

U.S. EPA AUTHORS

Principal Author:

John W. Nichols, Ph.D.
Mid-Continent Ecology Division
Office of Research and Development
Duluth, MN

Contributing Authors:

Robert B. Ambrose, Jr., P.E.
Ecosystems Research Division
National Exposure Research Laboratory
Athens, GA

Chris Cubbison, Ph.D.
National Center for Environmental Assessment-
Cincinnati
Office of Research and Development
Cincinnati, OH

Anne Fairbrother, Ph.D., D.V.M.
Environmental Research Laboratory-Corvallis
Corvallis, OR
currently with:
Ecological Planning and Toxicology, Inc.
5010 S.W. Hout St.
Corvallis, OR 97333

Martha H. Keating
Office of Air Quality Planning and Standards
Research Triangle Park, NC

Kathryn R. Mahaffey, Ph.D.
National Center for Environmental Assessment-
Cincinnati
Office of Research and Development
Cincinnati, OH

Debdas Mukerjee, Ph.D.
National Center for Environmental Assessment-
Cincinnati
Office of Research and Development
Cincinnati, OH

Glenn E. Rice
National Center for Environmental Assessment-
Cincinnati
Office of Research and Development
Cincinnati, OH

David J. Reisman
National Center for Environmental Assessment-
Cincinnati
Office of Research and Development
Cincinnati, OH

Rita Schoeny, Ph.D.
National Center for Environmental Assessment-
Cincinnati
Office of Research and Development
Cincinnati, OH

Jeff Swartout
National Center for Environmental Assessment-
Cincinnati
Office of Research and Development
Cincinnati, OH

Michael Troyer
Office of Science, Planning and Regulatory
Evaluation
Cincinnati, OH

SCIENTIFIC PEER REVIEWERS

Dr. William J. Adams*
Kennecott Utah Corporation

Dr. Brian J. Allee
Harza Northwest, Incorporated

Dr. Thomas D. Atkeson
Florida Department of Environmental Protection

Dr. Donald G. Barnes*
U.S. EPA Science Advisory Board

Dr. Steven M. Bartell
SENES Oak Ridge, Inc.

Dr. David Bellinger*
Children's Hospital, Boston

Dr. Nicolas Bloom*
Frontier Geosciences, Inc.

Dr. Mike Bolger
U.S. Food and Drug Administration

Dr. Peter Botros
U.S. Department of Energy
Federal Energy Technology Center

Thomas D. Brown
U.S. Department of Energy
Federal Energy Technology Center

Dr. Dallas Burtraw*
Resources for the Future

Dr. Thomas Burbacher*
University of Washington
Seattle

Dr. James P. Butler
University of Chicago
Argonne National Laboratory

Elizabeth Campbell
U.S. Department of Energy
Policy Office, Washington D.C.

Dr. Rick Canady
Agency for Toxic Substances and Disease
Registry

Dr. Rufus Chaney
U.S. Department of Agriculture

Dr. Joan Daisey*
Lawrence Berkeley National Laboratory

Dr. John A. Dellinger*
Medical College of Wisconsin

Dr. Kim N. Dietrich*
University of Cincinnati

Dr. Tim Eder
Great Lakes Natural Resource Center
National Wildlife Federation for the
States of Michigan and Ohio

Dr. Katherine Flegal
National Center for Health Statitistics

Dr. Lawrence J. Fischer*
Michigan State University

Dr. William F. Fitzgerald
University of Connecticut
Avery Point

A. Robert Flaak*
U.S. EPA Science Advisory Board

Dr. Bruce A. Fowler*
University of Maryland at Baltimore

Dr. Steven G. Gilbert*
Biosupport, Inc.

v

SCIENTIFIC PEER REVIEWERS (continued)

Dr. Cynthia C. Gilmour*
The Academy of Natural Sciences

Dr. Robert Goyer
National Institute of Environmental Health
Sciences

Dr. George Gray
Harvard School of Public Health

Dr. Terry Haines
National Biological Service

Dr. Gary Heinz*
Patuxent Wildlife Research Center

Joann L. Held
New Jersey Department of Environmental
Protection & Energy

Dr. Robert E. Hueter*
Mote Marine Laboratory

Dr. Harold E. B. Humphrey*
Michigan Department of Community Health

Dr. James P. Hurley*
University of Wisconsin
Madison

Dr. Joseph L. Jacobson*
Wayne State University

Dr. Gerald J. Keeler
University of Michigan
Ann Arbor

Dr. Ronald J. Kendall*
Clemson University

Dr. Lynda P. Knobeloch*
Wisconsin Division of Health

Dr. Leonard Levin
Electric Power Research Institute

Dr. Steven E. Lindberg*
Oak Ridge National Laboratory

Dr. Genevieve M. Matanoski*
The Johns Hopkins University

Dr. Thomas McKone*
University of California
Berkeley

Dr. Malcolm Meaburn
National Oceanic and Atmospheric
Administration
U.S. Department of Commerce

Dr. Michael W. Meyer*
Wisconsin Department of Natural Resources

Dr. Maria Morandi*
University of Texas Science Center at Houston

Dr. Paul Mushak
PB Associates

Harvey Ness
U.S. Department of Energy
Federal Energy Technology Center

Dr. Christopher Newland*
Auburn University

Dr. Jerome O. Nriagu*
The University of Michigan
Ann Arbor

William O'Dowd
U.S. Department of Energy
Federal Energy Technology Center

Dr. W. Steven Otwell*
University of Florida
Gainesville

Dr. Jozef M. Pacyna
Norwegian Institute for Air Research

SCIENTIFIC PEER REVIEWERS (continued)

Dr. Ruth Patterson
Cancer Prevention Research Program
Fred Gutchinson Cancer Research Center

Dr. Donald Porcella
Electric Power Research Institute

Dr. Deborah C. Rice*
Toxicology Research Center

Samuel R. Rondberg*
U.S. EPA Science Advisory Board

Charles Schmidt
U.S. Department of Energy

Dr. Pamela Shubat
Minnesota Department of Health

Dr. Ellen K. Silbergeld*
University of Maryland
Baltimore

Dr. Howard A. Simonin*
NYSDEC Aquatic Toxicant Research Unit

Dennis Smith
U.S. Department of Energy
Federal Energy Technology Center

Dr. Ann Spacie*
Purdue University

Dr. Alan H. Stern
New Jersey Department of Environmental
Protection & Energy

Dr. David G. Strimaitis*
Earth Tech

Dr. Edward B. Swain
Minnesota Pollution Control Agency

Dr. Valerie Thomas*
Princeton University

Dr. M. Anthony Verity
University of California
Los Angeles

*With EPA's Science Advisory Board, Mercury Review Subcommitte

WORK GROUP AND U.S. EPA/ORD REVIEWERS

Core Work Group Reviewers:

Dan Axelrad, U.S. EPA
Office of Policy, Planning and Evaluation

Angela Bandemehr, U.S. EPA
Region 5

Jim Darr, U.S. EPA
Office of Pollution Prevention and Toxic
Substances

Thomas Gentile, State of New York
Department of Environmental Conservation

Arnie Kuzmack, U.S. EPA
Office of Water

David Layland, U.S. EPA
Office of Solid Waste and Emergency Response

Karen Levy, U.S. EPA
Office of Policy Analysis and Review

Steve Levy, U.S. EPA
Office of Solid Waste and Emergency Response

Lorraine Randecker, U.S. EPA
Office of Pollution Prevention and Toxic
Substances

Joy Taylor, State of Michigan
Department of Natural Resources

U.S. EPA/ORD Reviewers:

Robert Beliles, Ph.D., D.A.B.T.
National Center for Environmental Assessment
Washington, DC

Eletha Brady-Roberts
National Center for Environmental Assessment
Cincinnati, OH

Annie M. Jarabek
National Center for Environmental Assessment
Research Triangle Park, NC

Matthew Lorber
National Center for Environmental Assessment
Washington, DC

Susan Braen Norton
National Center for Environmental Assessment
Washington, DC

Terry Harvey, D.V.M.
National Center for Environmental Assessment
Cincinnati, OH

LIST OF TABLES

LIST OF FIGURES

LIST OF SYMBOLS, UNITS AND ACRONYMS

BAF	Bioaccumulation factor
BAF_3	Aquatic life bioaccumulation factor for trophic level 3
BAF_4	Aquatic life bioaccumulation factor for trophic level 4
BCF	Bioconcentration factor
BSAF	Biota-sediment accumulation factor
BMF	Biomagnification factor
bw	Body weight
CAA	Clean Air Act as Amended in 1990
d	Day
DDE	p,p-Dichlorodiphenyldichloroethylene
DDT	4,4-Dichlorodiphenyltrichloroethane
DOC	Dissolved organic carbon
F_A	Average daily amount of food consumed
FCM	Food chain multiplier
FD_3	Fraction of the diet derived from trophic level 3
FD_4	Fraction of the diet derived from trophic level 4
GAS-ISC3	Short range air dispersion model for mercury
GLWQI	Great Lakes Water Quality Initiative
ha	Hectare
Hg^0	Elemental mercury
Hg_2^{2+}	Mercurous ion
Hg^{2+}	Mercury II
IEM-2M	Indirect exposure model for mercury
IJC	International Joint Commission
kg	Kilogram
L	Liter
LC_{50}	Lethal concentration (for fifty percent of population)
LD_{50}	Lethal dose (for fifty percent of population)
LCUB	Large coal-fired utility boiler
LOAEL	Lowest-observed-adverse-effect level
m	Meter
m^3	Cubic meter
MCM	Mercury cycling model
MDNR	Michigan Department of Natural Resources
mg	Milligram
MHg	Methlymercury
MWC	Municipal waste combustor
MWI	Medical waste incinerator
ng	Nanogram
nM	Nanomole
NCBP	National Contaminant Biomonitoring Program
NOAEL	No-observed-adverse-effect level
PCBs	Polychlorinated biphenyls
pg	Picogram
pH	Logarithm of the reciprocal of the hydrogen ion concentration. A measure of acidity
PPF	Predator-prey factor

LIST OF SYMBOLS, UNITS AND ACRONYMS (continued)

PPF_4	The observed ratio of the concentration at trophic level 4, divided by the concentration at trophic level 3
ppm	parts per million
RELMAP	Regional Lagrangian Model of Air Pollution
SAB	Science Advisory Board
sp.	Species
UF_A	Uncertainty factor for species extrapolation
UF_S	Uncertainty factor for use of less than lifetime study
UF_L	Uncertainty factor for use of a lowest adverse effect level
U.S. EPA	U.S. Environmental Protection Agency
μg	Microgram
μM	Micromole
W_A	Average daily volume of water consumed
WC	Wildlife criterion level
WC_f	Final wildlife criterion level
WC_i	Intermediate wildlife criterion level
WC_s	Species-specific wildlife criterion level
Wt_A	Average species weight

EXECUTIVE SUMMARY

Section 112(n)(1)(B) of the Clean Air Act (CAA), as amended in 1990, directs the U.S. Environmental Protection Agency (U.S. EPA) to submit to Congress a comprehensive study on emissions of mercury to the air. Volume VI, which addresses the ecological exposure and effects assessment for mercury and mercury compounds, is part of an eight-volume report developed by U.S. EPA in response to this directive.

Volume VI is an ecological risk assessment for anthropogenic mercury emissions. It follows the format of the U.S. EPA Framework for Ecological Risk Assessment (U.S. EPA, 1992a), with minor changes as suggested in the draft Proposed Guidelines for Ecological Risk Assessment (U.S. EPA, 1996). The first step in the Framework is the problem formulation phase, wherein the potential ecological impacts of mercury are reviewed. This is followed by the presentation of a conceptual model describing how airborne mercury accumulates in aquatic biota, biomagnifies in aquatic food chains and is consumed by wildlife that eat contaminated fish. Subsequent steps in the assessment include exposure and effects assessments. Exposure and effects information are then considered together in an effort to develop qualitative statements about the risk of airborne mercury emissions to piscivorous avian and mammalian wildlife. An outcome of this effort is a recalculation of the wildlife criterion (WC) value for mercury in aquatic systems. A characterization of the risks to wildlife from anthropogenic mercury emissions is provided in Volume VII of this Report to Congress.

Scope of the Assessment

The scope of this assessment was limited solely to anthropogenic mercury that is emitted directly to the atmosphere. The origins and extent of these emissions are reviewed in Volume II of this Report. This analysis did not address mercury originating from direct wastewater discharge to water bodies, mining waste or the application of mercurial pesticides. In a number of instances, these and other "point" sources have been related to unacceptably high mercury levels in fish, triggering site-specific fish consumption advisories. Clearly, where such point sources exist, there is a need to address the combined impacts of mercury originating from all sources, including air emissions.

Mercury in the Environment

Wet deposition is thought to be the primary mechanism by which mercury emitted to the atmosphere is transported to surface waters and land, although dry deposition may also contribute substantially. Once deposited, mercury enters aquatic and terrestrial food chains. Mercury concentrations increase at successively higher trophic levels as a result of bioconcentration, bioaccumulation and biomagnification. Of the various forms of mercury in the environment, methylmercury has the highest potential for bioaccumulation and biomagnification. Predators at the top of these food chains are potentially at risk from consumption of methylmercury in contaminated prey. Based on a review of available information, it was concluded that piscivorous (fish-eating) birds and mammals are particularly at risk from mercury emissions. This risk is likely to be greatest in areas that receive high levels of mercury deposition, although local and regional factors can substantially impact the amount of total mercury that is translocated from watersheds to waterbodies and undergoes chemical transformation to the methylated species.

The assessment endpoint for this ecological risk assessment is the maintenance of self-sustaining wildlife populations. Measurement endpoints include the growth and survival of individual animals, reproductive success, and behavior.

Exposure of Piscivorous Wildlife to Mercury

Exposure was characterized in a progressive manner, with varying reliance on computer models for mercury deposition and fate. The objective of this analysis was to characterize the extent to which piscivorous wildlife are exposed to mercury originating from airborne emissions. Details on exposure assessment inputs, methods and results can be found in Volumes III and IV of this Report. Three general approaches were used, which are described as follows.

1.   **Estimation of current average exposure to piscivorous wildlife on a nationwide basis.**

The first analysis was conducted without computer models. Estimates of current mercury exposure to selected piscivorous wildlife species were calculated as the product of the fish consumption rate and measured mercury concentrations in fish. This analysis was not intended to be a site-specific analysis, but rather to provide national exposure estimates for piscivorous wildlife. This analysis used mean total mercury measurements from two nationwide studies of fish residues and published fish consumption data for the selected wildlife species. The relative ranking of exposure in μg/kg bw/d of selected wildlife species was as follows: kingfisher > river otter > loon = osprey = mink $\geq$ bald eagle.

2.   **Estimation of mercury deposition on a regional scale (40 km grid) and comparison of these deposition data with species distribution information.**

The second type of analysis was carried out on a regional scale. A long-range atmospheric transport model (RELMAP) was used in conjunction with the mercury emissions inventory provided in Volume II of this Report to generate predictions of mercury deposition across the continental U.S. Ecosystems subject to high levels of mercury deposition will be more exposed to mercury than ecosystems with lower levels of mercury deposition. The pattern of mercury deposition nationwide, therefore, will influence which ecoregions and ecosystems might be exposed to hazardous levels of mercury. Thus, predictions of mercury deposition were compared with the locations of major lakes and rivers, national resource lands, threatened and endangered plant species and the distributions of selected piscivorous wildlife species. Additionally, mercury deposition data were superimposed onto a map of surface waters impacted by acid deposition, because it has been shown that low pH values are often correlated with high levels of mercury in fish. The extent of overlap of selected species distributions with areas receiving high rates of deposition (>5 µg/m^2) was characterized.

Avian wildlife considered in this analysis included species that are widely distributed (kingfishers) and narrowly distributed (bald eagles, ospreys, and loons). All the birds selected were piscivores that feed at or near the top of aquatic food chains and are therefore at risk from biomagnified mercury. Two of the mammals selected for this analysis (mink and river otters) are piscivorous and widely distributed. The other mammal selected, the Florida panther, is not widely distributed but is listed as an endangered species. The Florida panther lives in an environment known to be contaminated with mercury and preys upon small mammals (such as raccoons), which may contain high tissue burdens of mercury. Results for each avian and mammalian species are summarized in Table ES-1.

Approximately 29% of the kingfisher's range occurs within regions of high mercury deposition. On a nationwide basis, mercury does not appear to be a threat to this species. However, kingfishers consume more mercury on a body weight basis than any other wildlife species examined.

Table ES-1
Percent of Species Range Overlapping with Regions of High Mercury Deposition

Species	Percent of Range Impacted
Kingfisher	29%
Bald Eagle	34%
Osprey	20%
Common Loon	40%
Florida Panther	100%
Mink	35%
River Otter	38%

Although a recovery in the population of bald eagles has resulted in a status upgrade from "endangered" to "threatened" in five states (Michigan, Minnesota, Oregon, Washington and Wisconsin), bald eagle populations are still depleted throughout much of their historical range. Bald eagles can be found seasonally in large numbers in several geographic locations, but most of these individuals are transient, and the overall population is still small. Historically, eagle populations in the lower 48 states have been adversely impacted by the effects of bioaccumulative contaminants (primarily DDT and perhaps also PCBs). Approximately 34% of the bald eagle's range overlaps regions of high mercury deposition. Areas of particular concern include the Great Lakes region, the northeastern Atlantic states and south Florida.

Nationwide, approximately 20% of the osprey's total range overlaps regions of high mercury deposition; however, a much larger fraction of the osprey's eastern population occurs within these regions. The osprey diet consists almost exclusively of fish. Osprey populations underwent severe declines during the 1950s through the 1970s due to widespread use of DDT and related compounds.

Nearly 40% of the loon's range is located in regions of high mercury deposition. Limited data from a study of a mercury point source showed that loon reproductive success was negatively correlated with exposure to mercury in a significant dose-response relationship. In some cases, mercury residues in fish collected from lakes used as loon breeding areas may exceed levels that, on the basis of this point source study, are associated with reproductive impairment. Loons frequently breed in areas that have been adversely impacted by acid deposition. An assessment of mercury's effects on loon populations is complicated by the fact that decreases in surface water pH have been associated with both increased mercury residues in fish and declines in the available forage base.

All (100%) of the panther's range falls within an area of high mercury deposition. Mercury levels found in tissues obtained from dead panthers are similar to levels that have been associated with frank toxic effects in other feline species. The State of Florida has taken measures to reduce the risk to panthers posed by mercury. Existing plans include measures to increase the number of deer available as prey in order to reduce the reliance of panthers on raccoons. Raccoons frequently feed at or near the top of aquatic food webs and can accumulate substantial tissue burdens of mercury. An evaluation of the risk posed by mercury to the Florida panther is complicated by the possible impacts of other chemical stressors, habitat loss, and inbreeding.

Approximately 35% of the range of mink habitat coincides with regions of high mercury deposition nationwide. Mink occupy a large geographic area and are common throughout the U.S. Given the opportunity, mink will prey on small mammals and birds. Many subpopulations, however, prey almost exclusively on fish and other aquatic biota. Due to allometric considerations, mink may be exposed to more mercury on a body weight basis than larger piscivorous mammals feeding at higher trophic levels. In several cases, mercury residues in wild-caught mink have been shown to be equal to or greater than levels associated with toxic effects in the laboratory.

River otter habitat overlaps regions of high mercury deposition for about 14% of the range for this species. River otters occupy large areas of the U.S., but their population numbers are thought to be declining in both the midwestern and southeastern states. The river otter's diet is almost exclusively of aquatic origins and includes fish (primarily), crayfish, amphibians and aquatic insects. The consumption of large, piscivorous fish puts the river otter at risk from bioaccumulative contaminants including mercury. Like the mink, mercury residues in some wild-caught otters have been shown to be close to, and in some cases greater than, concentrations associated with frank toxic effects.

3.   Estimation of mercury exposure on a local scale in areas near emissions point sources.

A final analysis was conducted using a local-scale atmospheric fate model (GAS-ISC3), in addition to the long-range transport data and an indirect exposure methodology, to predict mercury concentrations in water and fish under a variety of hypothetical emissions scenarios. GAS-ISC3 simulated mercury deposition originating from model plants representing a range of mercury emissions source classes. The four source categories were selected based on their estimated annual mercury emissions or their potential to be localized point sources of concern. The categories selected were these: municipal waste combustors (MWCs), medical waste incinerators (MWIs), utility boilers, and chlor-alkali plants. To account for the long-range transport of emitted mercury, the 50th percentile RELMAP atmospheric concentrations and deposition rates were included in the estimates from the local air dispersion model. To account for other sources of mercury, estimates of background concentrations of mercury were also included in this exposure assessment.

These data were used to estimate the contributions of different emission source types to mercury exposure of selected wildlife species. It was concluded from this analysis that local emissions sources have the potential to increase significantly the exposure of piscivorous birds and mammals to mercury. Important factors related to local source impacts include quantity of mercury emitted by the source, species and physical form of mercury emitted, and effective stack height. The extent of this local contribution also depends upon watershed characteristics, facility type, local meteorology, and terrain. The exposure of a given wildlife species is also highly dependent upon the fish bioaccumulation factor, the trophic level(s) at which it feeds and the amount of fish consumed per day.

Although the accumulation of methylmercury in fish tissues appears to be highly variable across bodies of water, field data were determined to be sufficient to calculate representative means for different trophic levels. The variability can be seen in the distribution of the methylmercury bioaccumulation factors (BAF) for fish in trophic levels 3 and 4. These values, summarized in Table ES-2, are believed to be better estimates of mercury bioaccumulation in natural systems than values derived from laboratory studies.

Table ES-2
Percentiles of the Methylmercury Bioaccumulation Factor

Parameter	Percentile of Distribution				
	5th	25th	50th	75th	95th
Trophic 3 BAF	4.6×10^5	9.5×10^5	1.6×10^6	2.6×10^6	5.4×10^6
Trophic 4 BAF	3.3×10^6	5.0×10^6	6.8×10^6	9.2×10^6	1.4×10^7

Effects Assessment for Mercury

Due to the broad range and extent of mercury emissions throughout the United States, many potential ecological effects could have been considered. Neither the available data nor existing methodology supported evaluation of all possible effects.

The ecosystem effects of mercury are incompletely understood. No applicable studies of the effects of mercury on intact ecosystems were found. The ecological risk assessment for mercury did not, therefore, address effects of mercury on ecosystems, plant and animal communities or species diversity. Effects of methylmercury on fish and other aquatic biota were also not characterized, although there is evidence of adverse impacts on these organisms following point source releases of mercury and in aquatic environments affected by urban runoff.

Data on methylmercury effects in wildlife suitable for dose-response assessment are limited to what are termed "individual effects" in the U.S. EPA Framework for Ecological Risk Assessment (U.S. EPA, 1992a). A reference dose (RfD), defined as the chronic NOAEL, was derived for avian species from studies by Heinz (1975, 1976a,b, 1979) in which three generations of mallard ducks (*Anas platyrhychos*) were dosed with methylmercury dicyandiamide. The lowest dose, 0.5 ppm (78 µg/kg bw/d), resulted in adverse effects on reproduction and behavior and was designated as a chronic LOAEL. A chronic NOAEL was estimated by dividing the chronic LOAEL by a LOAEL-to-NOAEL uncertainty factor of 3. Calculated in this manner, the RfD for avian wildlife species is 26 µg/kg bw/d.

The RfD for mammalian species was derived from studies involving subchronic exposures with mink (Wobeser, 1973, 1976a,b), in which animals were dosed with mercury in the form of mercury-contaminated fish. The dose of 0.33 ppm (55 µg/kg bw/d) was selected as the NOAEL for subchronic exposure. As this was less than a lifetime exposure, the subchronic NOAEL was divided by a subchronic-to-chronic uncertainty factor of 3. Calculated in this manner, the RfD for mammalian wildlife species is 18 µg/kg bw/d.

Risk Assessment for Mercury

Ecological risk assessment methods relevant to chemical effects on wildlife are reviewed. The data needs of these methods vary widely and dictate, to a considerable degree, which methods can be applied to a given situation. Guidance is provided on the risk assessment methods that may be most applicable to airborne mercury emissions, given the nature and extent of currently existing information. Additional guidance is provided by reviewing published assessments for piscivorous species living in the

Great Lakes region, south Florida, central Ontario, and coastal regions of Georgia, South Carolina and North Carolina.

The scope of the present Report was intended to be national in scale. It was determined, therefore, that any effort to assess the risk of mercury to a given species living in a defined location would be inappropriate. Instead, an effort was made to compare mercury exposure and effects in a general way using data collected from throughout the country and, in so doing, to develop qualitative statements about risk.

Consistent with this broader-scale approach, an effort was made to derive a wildlife criterion (WC) value for mercury that is protective of piscivorous wildlife. This WC is defined as the concentration of mercury in water that, if not exceeded, protects avian and mammalian wildlife populations from adverse effects resulting from ingestion of surface waters and from ingestion of aquatic life taken from these surface waters. The health of wildlife populations may, therefore, be considered the assessment endpoint of concern. Although not generally derived for the purpose of ecological risk assessment, WC values incorporate the same type of exposure and effects information used in more standard approaches. Such calculations also provide for a simple assessment of risk in any given situation; that is, by determining whether the concentration of mercury in water exceeds the criterion value.

The principal factors used to select wildlife species for WC development were: (1) exposure to bioaccumulative contaminants; (2) species distributions; (3) availability of information with which to calculate criterion values; and (4) evidence for bioaccumulation and/or adverse effects. All of the species selected feed on or near the top of aquatic food webs. The avian species selected were the bald eagle (*Haliaeetus leucocephalus*), osprey (*Pandion haliaetus*), common loon (*Gavia immer*) and belted kingfisher (*Ceryle alcyon*). The mammalian species selected were the mink (*Mustela vison*) and river otter (*Lutra canadensis*).

Because this assessment depends to a large extent on the assignment of BAFs for mercury in fish at trophic levels 3 and 4, an effort was made to review published field data from which these BAFs could be estimated. A Monte Carlo analysis was then performed to characterize the variability around these estimates. The results of this effort are reported in Appendix D of Volume III and are summarized in Table ES-2.

A WC value for mercury was estimated as the ratio of an RfD, defined as the chronic NOAEL (in µg/kg bw/d), to an estimated mercury consumption rate, referenced to water concentration using a BAF. Individual wildlife criteria are provided in Table ES-3. This approach is similar to that used in non-cancer human health risk assessment and was employed previously to estimate a WC for mercury in the Water Quality Guidance for the Great Lakes System (GLWQI). The present effort differs, however, from that of the GLWQI in that the entire analysis was conducted on a methylmercury basis. Additional differences resulted from the availability of new data, including measured residue levels in fish and water, and a re-evaluation of the toxicity data from which RfD estimates were derived. In this Report, a more sensitive endpoint was selected for mammalian species, with the goal of assessing the full range of effects of mercury. These changes reflect the amount of discretion allowed under Agency Risk Assessment Guidelines.

Species-specific WC values for methylmercury were estimated for selected avian and mammalian wildlife (identified above). A final WC was then calculated as the lowest mean of WC

values for each of the two taxonomic classes (birds and mammals). The final WC for methylmercury was based on

Table ES-3
Wildlife Criteria for Methylmercury

Organism	Wildlife Criterion (pg/L)
Mink	57
River otter	42
Kingfisher	33
Loon	82
Osprey	82
Bald eagle	100

individual WC values calculated for mammalian species, and was estimated to be 50 picograms (pg) methylmercury/L water.

The WC for methylmercury can be expressed as a corresponding mercury residue in fish though the use of appropriate BAFs. Using the BAFs presented in Table ES-2 (50th percentile), a WC of 50 pg/L corresponds to methylmercury concentrations in fish of 0.077 µg/g and 0.346 µg/g for trophic levels 3 and 4, respectively. In addition, a WC for total mercury can be calculated using an estimate of methylmercury as a proportion of total mercury in water. Based upon a survey of speciation data, the best current estimate of dissolved methylmercury as a proportion of total dissolved mercury was determined to be 0.078. Using this value, a methylmercury WC of 50 pg/L corresponds to a total dissolved mercury WC of 641 pg/L. An additional correction is needed if the WC is to be expressed as the amount of total mercury in unfiltered water. The available data, although highly variable, suggest that on average total dissolved mercury comprises about 70 percent of that contained in unfiltered water. Making this final correction results in a WC of 910 pg/L (unfiltered, total mercury), which is approximately 70 percent of the value published previously in the GLWQI.

Conclusions

The following conclusions are presented in approximate order of degree of certainty in the conclusion, based on the quality of the underlying database. The conclusions progress from those with greater certainty to those with lesser certainty.

- Mercury emitted to the atmosphere deposits on watersheds and is translocated to waterbodies. A variable proportion of this mercury is transformed by abiotic and biotic chemical reactions to organic derivatives, including methylmercury. Methylmercury bioaccumulates in individual organisms, biomagnifies in aquatic food chains and is the most toxic form of mercury to which wildlife are exposed.

- The proportion of total mercury in aquatic biota that exists as methylmercury tends to increase with trophic level. Greater than 90% of the mercury contained in freshwater fish exists as methylmercury. Methylmercury accumulates in fish throughout their lifetime, although changes in concentration as a function of time may be complicated by growth dilution and changing dietary habits.

- Piscivorous avian and mammalian wildlife are exposed to mercury primarily through consumption of contaminated fish and accumulate mercury to levels above those in prey items.

- Toxic effects on piscivorous avian and mammalian wildlife due to the consumption of contaminated fish have been observed in association with point source releases of mercury to the environment.

- Concentrations of mercury in the tissues of wildlife species have been reported at levels associated with adverse health effects in laboratory studies with the same species.

- Piscivorous birds and mammals receive a greater exposure to mercury than any other known receptors.

- BAFs for mercury in fish vary widely; however, field data are sufficient to calculate representative means for different trophic levels. These means are believed to be better estimates of mercury bioaccumulation in natural systems than values derived from laboratory studies. The recommended methylmercury BAFs for tropic levels 3 and 4 are 1,600,000 and 6,800,000, respectively (dissolved basis).

- Based upon knowledge of mercury bioaccumulation in fish, and of feeding rates and the identity of prey items consumed by piscivorous wildlife, it is possible to rank the relative exposure of different piscivorous wildlife species. Of the six wildlife species selected for detailed analysis, the relative ranking of exposure to mercury is this: kingfisher > otter > loon = osprey = mink $\geq$ bald eagle. Existing data are insufficient to estimate the exposure of the Florida panther relative to that of the selected species.

- Local emissions sources (<50 km from receptors) have the potential to increase the exposure of piscivorous wildlife well above that due to sources located more than 50 km from the receptors (i.e., "remote" sources).

- Field data are insufficient to conclude whether the mink, otter or other piscivorous mammals have suffered adverse effects due to airborne mercury emissions.

- Field data are insufficient to conclude whether the loon, wood stork, great egret, or other piscivorous wading birds have suffered adverse effects due to airborne mercury emissions.

- Field data are suggestive of adverse toxicological effects in the Florida panther due to mercury. Unfortunately, the interpretation of these data is complicated by the co-occurrence of several other potentially toxic compounds, habitat degradation, and loss of genetic diversity. Field data suggest that bald eagles have not suffered adverse toxic effects due to airborne mercury emissions.

- Reference doses (RfDs) for methylmercury, defined as chronic NOAELs, were determined for avian and mammalian wildlife. Each RfD was calculated as the toxic dose (TD) from laboratory toxicity studies, divided by appropriate uncertainty factors. The RfD for avian species is 21 µg/kg bw/d (mercury basis). The RfD for mammalian wildlife is 18 µg/kg bw/d (mercury basis).

- Based upon knowledge of mercury exposure to wildlife and its toxicity in long-term feeding studies, WC values can be calculated for the protection of piscivorous avian and mammalian wildlife. A WC value is defined as the concentration of total mercury in water which, if not exceeded, protects avian and mammalian wildlife populations from adverse effects resulting from ingestion of surface waters and from ingestion of aquatic life taken from these surface waters.

- The methylmercury WC for protection of piscivorous avian wildlife is 61 pg/L (mercury basis).

- The methylmercury criterion for protection of piscivorous mammalian wildlife is 50 pg/L (mercury basis).

- The final methylmercury criterion for protection of piscivorous wildlife species is 50 pg/L. This value corresponds to a total mercury concentration in the water column of 641 pg/L, and methylmercury concentrations in fish of 0.077 ppm (trophic level 3) and 0.346 ppm (trophic level 4).

- Modeled estimates of mercury concentration in fish around hypothetical mercury emissions sources predict exposures within a factor of two of the WC. The WC, like the human RfD, is predicted to be a safe dose over a lifetime. It should be noted, however, that the wildlife effects used as the basis for the WC are gross clinical manifestations. Expression of subtle adverse effects at these doses cannot be excluded.

- The adverse effect level (population impacts on piscivorous wildlife) for methylmercury in fish that occupy trophic level 3 lies between 0.077 and 0.3 ppm. A comparison of this range of values with published residue levels in fish suggests that it is probable that individuals of some highly exposed wildlife subpopulations are experiencing adverse toxic effects due to airborne mercury emissions.

There are many uncertainties associated with this analysis, due to an incomplete understanding of the biogeochemistry and toxicity of mercury and mercury compounds. The sources of uncertainty include the following:

- Variability in the calculated BAFs is a source of uncertainty. BAFs given in this Report relate methylmercury in fish to dissolved methylmercury levels in the water column. Methods for the speciation of mercury in environmental samples are rapidly improving but remain difficult to perform. Questions also remain concerning the bioavailability of methylmercury associated with suspended particulates and dissolved organic material. Local biogeochemical factors that determine net methylation rates are not fully understood. The food webs through which mercury moves are poorly defined in many ecosystems and may not be adequately represented by a four-tiered food chain model.

- The representativeness of field data used in establishing the BAFs is a source of uncertainty. The degree to which the analysis is skewed by the existing data set is unknown. A

disproportionate amount of data is from north-central and northeastern lakes. The uncertainty associated with applying these data to a national-scale assessment is unknown.

- Limitations of the toxicity database present a source of uncertainty. Few controlled studies of quantifiable effects of mercury exposure in wildlife are available. These are characterized by limited numbers of dosage levels, making it difficult to establish NOAEL and LOAEL values. The toxic endpoints reported in most studies can be considered severe, raising questions as to the degree of protection against subtle effects offered by RfD and WC values. Use of less than lifetime studies for prediction of effects from lifetime exposure is also a source of uncertainty.

- Concerns exist regarding the possibility of toxic effects in species other than the piscivorous birds and mammals evaluated in this Report. Uncertainty is associated with mercury effects in birds and mammals that prey upon aquatic invertebrates and with possible effects on amphibians and aquatic reptiles. Uncertainty is also associated with mercury effects in fish. Toxicity to terrestrial ecosystems, in particular soil communities, is another source of uncertainty.

- Lack of knowledge of wildlife feeding habits is a source of uncertainty. Existing information frequently is anecdotal or confined to evaluations of a particular locality; the extent to which this information can be generalized is open to question. In some instances, the feeding habits are relatively well characterized (e.g., Florida panther), whereas the extent of mercury contamination of prey is poorly known (e.g., in raccoons).

- While the methods used to assess toxicity focus on individual-level effects, the stated goal of the assessment is to characterize the potential for adverse effects in wildlife populations. Factors that contribute to uncertainty in population-based assessments include: variability in the relationship between individuals and populations; lack of data on carrying capacity; and relationships of one population, of the same or different species, to another population.

- A focus on populations may not always be appropriate. This could be true for endangered species, which may be highly dependent for the survival of the species on the health of a few individuals. This may also be true for some regional or local populations of widespread species; the local population may be "endangered" and, thus, dependent on the survival of individuals.

- Multiple stressor interactions involving chemical effects are, in general, poorly known. Even less well known are the possible impacts of land and water use practices on water quality and large-scale ecosystem attributes (e.g., community structure and biodiversity).

1. INTRODUCTION

Section 112(n)(1)(B) of the Clean Air Act (CAA), as amended in 1990, requires the U.S. Environmental Protection Agency (U.S. EPA) to submit a study on atmospheric mercury emissions to Congress. The sources of emissions that must be studied include electric utility steam generating units, municipal waste combustion units and other sources, including area sources. Congress directed that the Mercury Study evaluate the rate and mass of mercury emissions, health and environmental effects, technologies to control such emissions and the costs of such controls.

In response to this mandate, U.S. EPA has prepared an eight-volume Mercury Study Report to Congress. The eight volumes are as follows:

I.   Executive Summary
II.   An Inventory of Anthropogenic Mercury Emissions in the United States
III.   Fate and Transport of Mercury in the Environment
IV.   An Assessment of Exposure to Mercury in the United States
V.   Health Effects of Mercury and Mercury Compounds
VI.   An Ecological Assessment for Anthropogenic Mercury Emissions in the United States
VII.   Characterization of Human Health and Wildlife Risks from Mercury Exposure in the United States
VIII.   An Evaluation of Mercury Control Technologies and Costs

This volume (Volume VI) is an ecological assessment of airborne mercury emissions. It provides an overview of the ecological effects of mercury, uses published data on fish residues as well as modeling predictions from Volume III to assess potential ecological exposures, and reviews available toxicity and bioaccumulation data for the purpose of developing qualitative statements about the risk of airborne mercury emissions to piscivorous avian and mammalian wildlife. In addition, these data are used to calculate a criterion value for the protection of piscivorous wildlife species, using the same general methodology employed in the Great Lakes Water Quality Initiative (U.S. EPA 1993b, 1993c, 1995b).

Volume VI is organized according to the format provided by U.S. EPA's Framework for Ecological Risk Assessment (U.S. EPA, 1992a). Chapter 2 corresponds to the problem formulation phase of the assessment and reviews the potential ecological impacts of mercury. Based upon this information, it is concluded that piscivorous avian and mammalian wildlife are potentially at risk due to airborne mercury emissions. A conceptual model is presented to describe how airborne mercury becomes concentrated in aquatic biota, which serve as the primary food source for piscivorous wildlife. An exposure analysis is presented in Chapter 3, and effects are analyzed in Chapter 4. Effects and exposure information are considered together in Chapter 5 as a means of assessing the risk of airborne mercury emissions to piscivorous avian and mammalian wildlife. Chapter 6 lists the main conclusions of this report, while Chapter 7 presents a list of critical research needs. References are provided at the end of this Volume in Chapter 8. An ecological risk characterization for mercury is presented separately in Volume VII of this Report.

The scope of this assessment is limited to consideration of only mercury that is emitted directly to the atmosphere. The origins and extent of these emissions are reviewed in Volume II of this Report. This analysis does not address mercury originating from mine leachate, the manufacturing and disposal of batteries, dental amalgam (in municipal wastewater), or the application of mercurial pesticides. In a number of instances, these and other "point" sources have been related to unacceptably high mercury

levels in fish, triggering site-specific fish consumption advisories. Clearly, where such point sources exist, there is a need to address the combined impacts of mercury originating from all sources, including air emissions.

The exposure analysis for piscivorous wildlife was designed to address the following questions:

- What is the current degree of exposure of piscivorous avian and mammalian wildlife?

- In what broad geographical areas of the continental United States is there a high probability for co-occurrence of high mercury deposition rates and wildlife species of concern?

- What is the relative increase in exposure that can be anticipated for wildlife species that live in proximity to mercury emissions sources?

The first of these questions was addressed by defining what piscivorous wildlife eat and then characterizing the mercury content of these food items. The second question was addressed by superimposing the results of a long-range transport analysis onto wildlife distribution information. The last question was addressed by using the results of a local-scale air dispersion model, combined with an indirect exposure methodology, to generate hypothetical exposure scenarios for wildlife. This short-range analysis is similar to that used in the human health exposure assessment (Volume IV). Descriptions of the long- and short-range air dispersion models and the indirect exposure methodology are provided in Volume III.

The primary goal of the effects analysis was to identify and review toxicity studies with wildlife species that could be used to estimate chronic NOAEL values for avian and mammalian wildlife. In addition, field data were reviewed as a means of comparing mercury residues in wild animals with those shown to associated with toxic effects in laboratory or other studies.

Finally, exposure and effects information are reviewed in an effort to develop qualitative statements about the risk of mercury emissions to piscivorous avian and mammalian wildlife. This assessment includes a review of previously published efforts to assess the risk of mercury to several wildlife species living in restricted geographical locals. Exposure and effects information are also used to calculate a water-based wildlife criterion value for mercury, which, if not exceeded, would be protective of piscivorous avian and mammalian wildlife. The general method used to calculate this criterion value is similar to that used previously to estimate criterion values for mercury in the Great Lakes Water Quality Initiative (U.S. EPA 1993b, 1993c, 1995b). An effort was made to calculate fish residue concentrations corresponding to this criterion value. These residue values were then compared with measured values obtained in environmental sampling efforts. Owing to its importance for both the ecological and human health assessments, published data for fish and other aquatic biota were evaluated to calculate bioaccumulation factors (BAFs) for methylmercury and to characterize the uncertainties associated with these estimates. The data and methods used to derive these BAFs are presented in Appendix D of Volume III. A summary of this material is provided in Chapter 5 of the present Volume.

2. PROBLEM FORMULATION

U.S. EPA defines ecological risk assessment as "a process that evaluates the likelihood that adverse ecological effects may occur or are occurring as a result of exposure to one or more stressors" (U.S. EPA, 1992a, 1996). A "stressor" is defined as any chemical, biological, or physical entity that can induce an adverse response of ecological components, i.e., individuals, populations, communities, or ecosystems. Although ecological risk assessment follows the same basic risk paradigm as human health risk assessment, there are three key differences between the two types.

- Ecological risk assessment can consider effects on populations, communities and ecosystems in addition to effects on individuals of a single species.

- No single set of ecological values to be protected is applicable in all cases; instead, they must be selected for each assessment based on both scientific and societal merit.

- Nonchemical stressors (e.g., physical disturbances) often need to be evaluated as well as chemical stressors.

The problem formulation phase of an environmental risk assessment consists of four main components: (1) integrating available information on the stressors, potential exposure pathways, ecosystems potentially at risk, and ecological effects; (2) selecting assessment endpoints (the ecological values to be protected); (3) developing a conceptual model of the problem; and (4) formulating an analysis plan for the exposure and effects characterization phases of the assessment.

Section 2.1 reviews the characteristics of mercury in the environment, including its various chemical forms (speciation), chemical transformations and movement within and between the air, surface water, and soil compartments of the environment (cycling). Section 2.2 identifies the pathways by which plants and animals can be exposed to mercury in both aquatic and terrestrial ecosystems. Section 2.3 provides an overview of what is known about the effects of mercury on organisms, populations, communities and ecosystems. Section 2.4 identifies ecosystems and ecosystem components that are thought to be most at risk from mercury in the environment. Section 2.5 describes the selection of assessment and measurement endpoints for the ecological risk assessment. A conceptual model of mercury fate and effects in the environment is presented in Section 2.6. An analysis plan for the exposure and effects characterizations is provided in Section 2.7.

It should be noted that this review of mercury fate and effects is limited to consideration of only terrestrial and freshwater aquatic ecosystems. It is recognized that mercury that deposits in coastal areas can be translocated to estuarine environments, and that biota which inhabit these and nearby marine systems have the potential to be adversely impacted. Presently, however, uncertainties regarding mercury deposition, cycling, and effects in such environments are so great as to preclude even a qualitative risk assessment.

2.1 Stressor Characteristics: Mercury Speciation and Cycling

Mercury in the environment can occur in various physical and chemical forms. Physically, mercury may exist as a gas or liquid, or it may be associated with solid particulates. Chemically, mercury can exist in three oxidation states:

(1) Hg^0 – **elemental mercury**, also called metallic mercury;

(2) Hg_2^{2+} - **mercurous ion** (monovalent mercury, mercury I); or

(3) Hg^{2+} - **mercury II** (mercuric ion, divalent mercury).

Mercury also reacts with other chemicals to form inorganic compounds (e.g., $HgCl_2$ - mercuric chloride) and organic compounds (e.g., CH_3Hg^+ - monomethylmercury, $(CH_3)_2Hg$ - dimethylmercury, C_6H_5HgCl - phenyl mercuric chloride). Figure 2-1 illustrates the major transformations between these different forms in the environment. Dimethylmercury is highly volatile and dissociates to monomethylmercury at neutral or acid pH (pH < 8) (Huckabee et al., 1979). In contrast, monomethylmercury is stable and tends to accumulate in living organisms (Bloom, 1992). Throughout this volume, monomethylmercury is referred to simply as methylmercury.

Figure 2-1
Cycling of Mercury in Freshwater Lakes (adapted from Winfrey and Rudd, 1990)
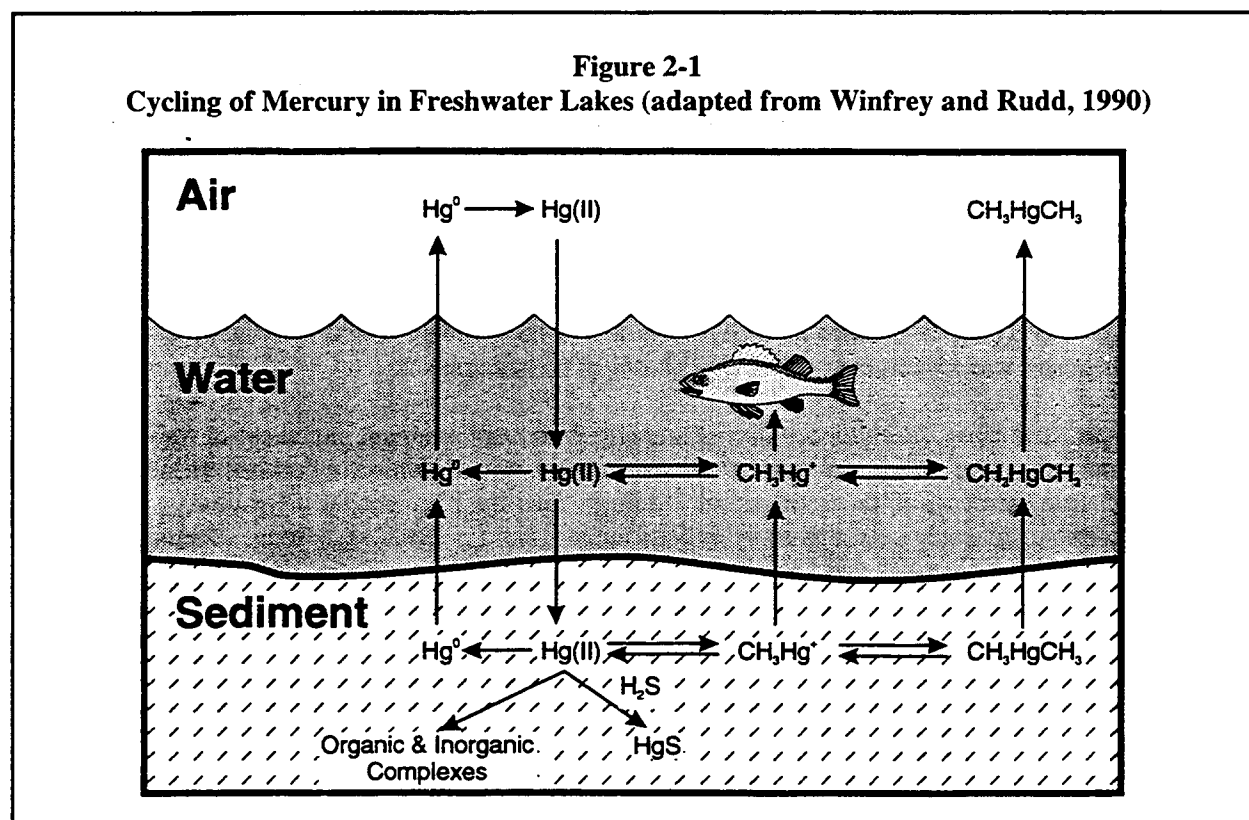
As discussed in the box below, methylation is an important step in the mercury cycle that strongly influences the ecological fate and effects of mercury. Methylmercury is readily accumulated by fish due to efficient uptake from dietary sources and to low rates of elimination (Bloom, 1992). It is also the most toxic form of mercury to wildlife (Eisler, 1987).

Mercury cycling and partitioning in the environment are complex phenomena and are influenced by numerous environmental factors. The following sections provide a brief overview of mercury speciation and partitioning in the atmosphere, surface water and soil, including information from specific case studies. For a detailed review, see Volume III of this Report.

FOCUS ON METHYLMERCURY

Methylmercury is the form of mercury of particular concern in ecosystems for three reasons.

(1)   All forms of mercury can be converted to methylmercury by natural processes in the environment.
(2)   Methylmercury bioaccumulates and biomagnifies in aquatic food webs.
(3)   Methylmercury is the most toxic form of mercury.

In the 1960s, researchers found methylmercury in fish in Swedish lakes, although no discharge of methylmercury had occurred in those lakes (Bakir et al., 1973). Later research determined that the methylation of mercury in sediments by anaerobic sulfur-reducing bacteria was a major source of methylmercury in many aquatic environments (Gilmour and Henry, 1991; Zillioux et al., 1993). Aerobic bacteria and fungi, including yeasts that grow best in acid conditions, also can methylate mercury (Eisler, 1987; Yannai et al., 1991; Fischer et al., 1995). In addition, fulvic and humic material may abiotically methylate mercury (Nagase et al., 1984; Lee et al., 1985; Weber, 1993). The major site of methylation in aquatic systems is the sediment, but methylation also occurs in the water column (Wright and Hamilton, 1982; Xun et al., 1987; Parks et al., 1989; Bloom and Effler, 1990; Winfrey and Rudd, 1990; Bloom et al., 1991; Gilmour and Henry, 1991; Miskimmin et al., 1992). Wetlands may be particularly active sites of methylation (St. Louis et al., 1994; Hurley et al., 1995). The rate of mercury methylation varies with microbial activity, mercury loadings, suspended sediment load, DOC, nutrient content, pH, redox conditions, temperature, and other variables. Demethylation occurs via biotic and abiotic mechanisms, including photodegradation (Sellers et al., 1996). The net rate of mercury methylation is determined by competing rates of methylation and demethylation.

Methylmercury bioaccumulates and biomagnifies in aquatic food webs at higher rates and to a greater extent than any other form of mercury (Watras and Bloom, 1992). "Bioaccumulation" refers to the net uptake of a contaminant from the environment into biological tissue via all pathways. It includes the accumulation that may occur by direct contact of skin or gills with mercury-contaminated water as well as ingestion of mercury-contaminated food. "Biomagnification" refers to the increase in chemical concentration in organisms at successively higher trophic levels in a food chain as a result of the ingestion of contaminated organisms at lower trophic levels. Methylmercury can comprise from 10 percent to over 90 percent of the total mercury in phytoplankton and zooplankton (trophic levels 1 and 2) (May et al., 1987; Watras and Bloom, 1992), but generally comprises over 90 percent of the total mercury in fish (trophic levels 3 and 4) (Huckabee et al., 1979; Grieb et al., 1990; Bloom, 1992; Watras and Bloom, 1992). Fish absorb methylmercury efficiently from dietary sources and store this material in organs and tissues. The biological half-life of methylmercury in fish is difficult to determine but is generally thought to range from months to years.

Methylmercury is the most toxic form of mercury to birds, mammals, and aquatic organisms due to its strong affinity for sulfur-containing organic compounds (e.g., proteins). Biological membranes, including the blood-brain barrier and the placenta, that tend to discriminate against other forms of mercury allow relatively easy passage of methylmercury and dissolved mercury vapor (Eisler, 1987). Methylmercury can cause death, neurological disorders, organ damage, impaired immune response, impaired growth and development and reduced reproductive success (Klaassen, 1986). In mammals, fetuses are particularly sensitive to mercury, experiencing deleterious developmental effects when the mothers appear to be unaffected (Clarkson, 1990).

2.1.1   Mercury in Air

In the atmosphere, most mercury (95 to over 99 percent) exists as gaseous Hg^{o}; the remainder generally is comprised of gaseous divalent (Hg^{2+}) mercury and mercury associated with particulates (Lindqvist, 1991; MDNR, 1993). Gaseous methylmercury may also may exist in air at measurable concentrations, especially near mercury emissions sources. Mercury associated with particulates in air includes Hg^{2+}, which is thought to occur primarily as mercuric chloride (MDNR, 1993).

The form of mercury in air affects both the rate and mechanism by which it deposits to earth. Oxidized and particulate mercury are more likely to be deposited than Hg^{o} because they are more soluble in water and are scavenged by precipitation more easily. They are also thought to be dry deposited more easily. As a result, oxidized and particulate forms of mercury are thought to comprise the majority of

deposited mercury, even though they comprise only a few percent of the total amount of mercury in the atmosphere (Lindqvist, 1991).

Wet deposition is thought to be the primary mechanism for transporting mercury from the atmosphere to surface waters and land (Lindqvist, 1991). In the Great Lakes area, for example, wet deposition is believed to account for 60 to 70 percent of total mercury deposition. Hg^{2+} is the predominant form in precipitation (MDNR, 1993).

2.1.2   Mercury in Surface Water

Mercury can enter surface water as Hg^{o}, Hg^{2+}, or methylmercury. Once in aquatic systems, mercury can exist in dissolved or particulate forms and can undergo the following transformations (see Figure 2-1) (Lindqvist et al., 1991; Winfrey and Rudd, 1990).

- Hg^{o} in surface waters can be oxidized to Hg^{2+} or volatilized to the atmosphere.

- Hg^{2+} can be methylated in sediments and the water column to form methylmercury.

- Methylmercury can be alkylated to form dimethylmercury.

- Hg^{2+} and methylmercury can form organic and inorganic complexes with sediment and suspended particulate matter.

Each of these reactions can also occur in the reverse direction. The net rate of production of each mercury species is determined by the balance between forward and reverse reactions.

Estimates of the percent of total mercury in surface waters that exists as methylmercury vary. Generally, methylmercury makes up less than 20 percent of the total mercury in the water column (Kudo et al., 1982; Parks et al., 1989; Bloom and Effler, 1990; Watras et al., 1995a). In lakes without point source discharges, methylmercury frequently comprises ten percent or less of total mercury in the water column (Lee and Hultberg, 1990; Lindqvist, 1991; Porcella et al., 1991; Watras and Bloom, 1992; Driscoll et al., 1994, 1995; Watras et al., 1995b). A review of speciation data collected to date suggests that methylmercury as a percent of total averages just under 8 percent (see Volume III, Appendix D of this Report).

Contaminated sediments can serve as an important mercury reservoir, with sediment-bound mercury recycling back into the aquatic ecosystem for decades or longer. Biological processes affect this recycling process. For example, sulfate-reducing bacteria may mediate mercury methylation (Gilmour and Henry, 1991). Benthic invertebrates may take up mercury from sediments, making it available to other aquatic animals through the food chain and to vertebrates that consume emergent aquatic insects (Hildebrand et al., 1980; Wren and Stephenson, 1991; Dukerschein et al., 1992; Saouter et al., 1993; Tremblay et al., 1996; Suchanek et al., 1997). Chemical factors, such as reduced pH, may stimulate methylmercury production at the sediment/water interface and thus may accelerate the rate of mercury methylation resulting in increased accumulation by aquatic organisms (Winfrey and Rudd, 1990). Attributes of the sediment, including organic carbon and sulfur content, can influence mercury bioavailability (Tremblay et al., 1995). DOC appears to be important in the transport of mercury to lake systems but, at high concentrations, may limit bioavailability (Driscoll et al., 1994, 1995).

2.1.3   Mercury in Soil

        Mercury deposited from the air forms stable complexes with soil particles of high organic or
sulfur content and with humic and fulvic acids (Andersson, 1979; WHO, 1989; Johansson et al., 1991;
Yin et al., 1996). These chemical bonds limit mercury's mobility in soils and its availability for uptake
by living organisms. In general, the distribution of mercury in soil is likely to follow the distribution of
organic matter. Mercury has a long retention time in soils. As a result, mercury that has accumulated in
soils may continue to be released to surface waters for long periods of time, possibly hundreds of years
(Johansson et al., 1991)

        Hg^{2+} in soils can be transformed to other mercury species. Bacteria and organic substances can
reduce Hg^{2+} to Hg^o, releasing volatile elemental mercury to the atmosphere. Alternatively, bacteria and
organic substances can methylate mercury, and subsequently demethylate it, depending on environmental
conditions (Allard and Arsenie, 1991; Gilmour and Henry, 1991).

        Recent measurements of volatile exchange between air and soil indicate that soil emissions could
be similar in magnitude to atmospheric deposition, suggesting that the total sink capacity of soils is less
than previously thought (Kim et al., 1995). Similarly, measurements of canopy emissions indicate that
forest ecosystems may not act as efficient sinks for atmospheric mercury (Lindberg, 1996). It is
uncertain at present how much these loss processes affect the retention of mercury in upper level soils.

2.2   Potential Exposure Pathways

        Plants and animals can be exposed to mercury by direct contact with contaminated environmental
media or ingestion of mercury-contaminated water and food (see Figure 2-2). Mercury deposited in soil
can be a source of direct exposure from physical contact (e.g., earthworms and terrestrial plants).
Animals also can ingest mercury in soil, either purposefully (e.g., earthworms) or incidentally (e.g.,
grazers). Mercury in the air can be taken up directly by terrestrial or aquatic emergent plants or inhaled
by terrestrial animals. Mercury in water can be a source of direct exposure to aquatic plants (e.g., algae
and seagrasses) and animals (e.g., zooplankton and fish) and can be ingested by terrestrial animals in
drinking water. Finally, both aquatic and terrestrial animals can be exposed to mercury in contaminated
food sources.

        Not all of these potential exposure pathways are equally important, however. The remainder of
this section evaluates the likely importance of different routes of exposure consequent to mercury release
to air. Section 2.2.1 discusses the fate and bioavailability of mercury in aquatic systems and the
pathways by which aquatic plants and animals can be exposed to mercury directly in contaminated water
or indirectly through aquatic food webs. Section 2.2.2 provides information on the fate and
bioavailability of mercury in terrestrial ecosystems and the pathways by which terrestrial plants and
animals can be exposed. Bioaccumulation of mercury in aquatic and terrestrial organisms is discussed
further in Section 2.3.1.

2.2.1   Exposure Pathways in Aquatic Systems

        Figure 2-3 illustrates the potential distribution of mercury in a water body. As shown, mercury
can be present in surface waters in various forms: (1) dissolved in the water; (2) concentrated in the
surface

2-5

microlayer (the uppermost layer of a surface water); (3) attached to seston[1]; (4) in the bottom sediments; and (5) in biota (e.g., fish and macroinvertebrates[2]).

Figure 2-2
Possible Routes of Exposure to Mercury
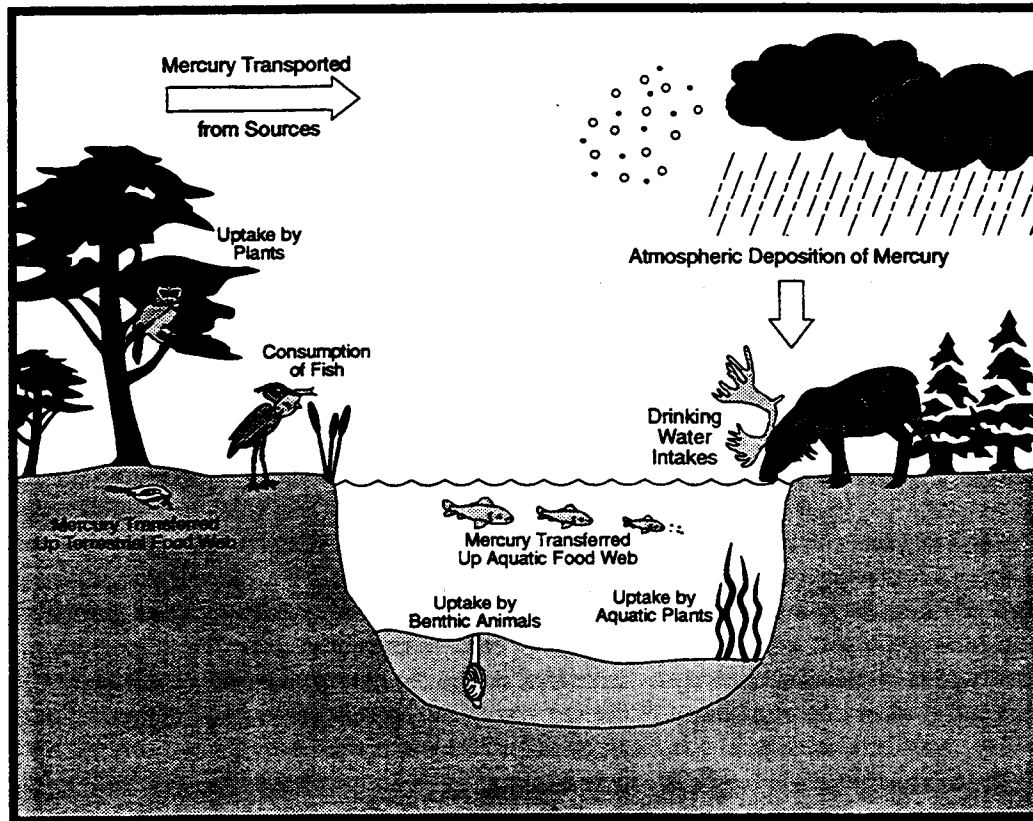
c54054-1-1

The form and location of mercury in a water body determines its bioavailability. For example, dissolved mercury is available for direct uptake by aquatic plants, fish and invertebrates. Mercury that concentrates in the surface microlayer is available to organisms that live, reproduce, or feed on the surface of water bodies (i.e., neuston). Mercury attached to seston can be ingested by aquatic animals that feed on plankton. Additionally, mercury that has accumulated in the sediments may be available to benthic plants and animals.

---

[1]Seston is suspended particulate matter, including detritus (dead organic matter) and plankton (i.e., living plants and animals that passively float or weakly swim in the water column such as algae, water fleas, and copepods).

[2]Macroinvertebrates are invertebrates (i.e., animals without backbones) that are visible to the naked eye, such as worms, clams, snails, insects and insect larvae, and crayfish.

Figure 2-3
Distribution of Mercury in a Water Body
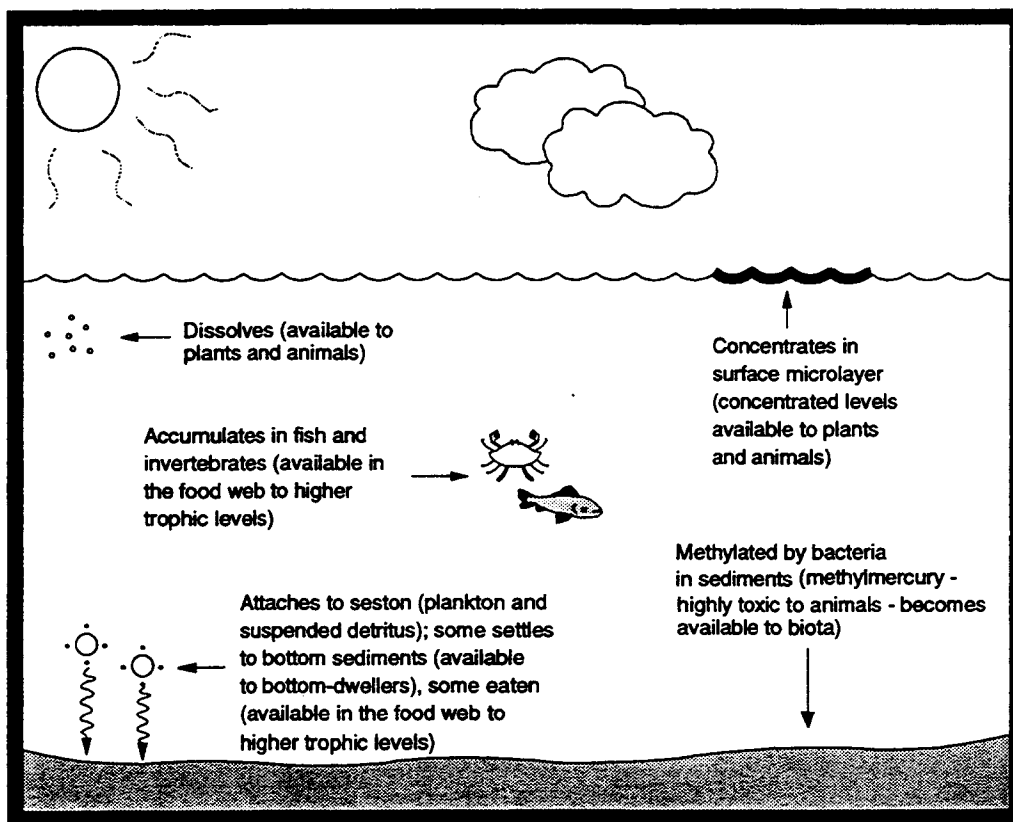
c54054-1-2

Aquatic plants may take up mercury from air, water or sediments (Crowder, 1991; Ribeyre and Boudou, 1994). Planktonic plants (i.e., phytoplankton such as algae) are not rooted; therefore, their only route of exposure is uptake from water. Both submerged aquatic vegetation and wetland emergent plants are rooted and can be exposed to mercury in sediments. In locations with mercury-contaminated sediments, mercury levels in aquatic macrophytes[3] have been measured at 0.01 $\mu g/g$, indicating that these plants do not strongly accumulate mercury from sediments (Wells et al., 1980; Crowder et al., 1988). The ratio between inorganic and organic mercury varies in plants (Crowder, 1991).

For aquatic animals, the primary exposure routes of concern are direct contact with mercury-contaminated water and sediments and ingestion of mercury-contaminated food. Fish can absorb mercury through the gills, skin and gastrointestinal tract (Wiener and Spry, 1995). The proportion of mercury taken up by any given route varies with fish size, and perhaps also with seasonal factors such as water temperature, diet and prey availability (Post et al., 1996). These fish then become a source of mercury for piscivorous birds and mammals. Emergent aquatic insects represent another potential source of mercury for insectivorous birds and mammals (Dukerschein et al., 1992; Saouter et al., 1993).

---

[3]Macrophytes are aquatic plants that are large enough to be visible to the naked eye.

Mercury in aquatic biota tends to occur at higher concentrations in higher trophic levels (discussed in more detail in Section 2.3 of this Volume). An example aquatic food web is shown in Figure 2-4. At the top trophic levels are piscivores, such as humans, bald eagles, cormorants, herring gulls, loons, kingfishers, mergansers, herons, egrets, ospreys, bald eagles, river otters, mink, alligators, snapping turtles and water snakes. The largest of these species (e.g., bald eagle and otter) can prey on fish that occupy high trophic levels, such as trout and salmon, which in turn feed on smaller "forage" fish, such as smelt, alewife, minnow, chub, and sculpin. Smaller piscivorous wildlife (e.g., kingfishers, ospreys, and terns) tend to feed on the smaller forage fish, which in turn feed on zooplankton or benthic invertebrates. Zooplankton (e.g., copepods and water fleas) feed on phytoplankton (i.e., microscopic algae), and the smaller benthic

Figure 2-4
Example Aquatic Food Web
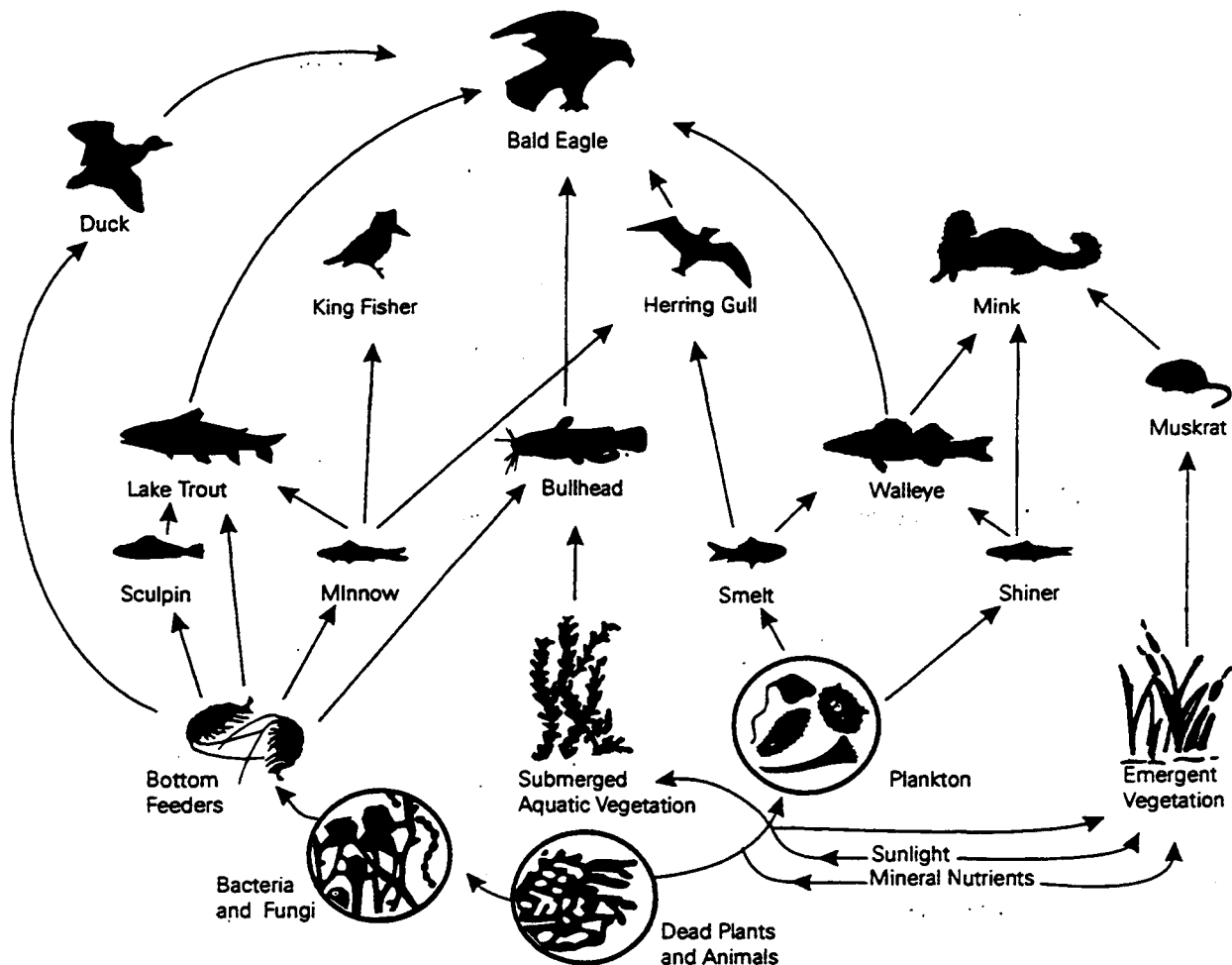
invertebrates tend to feed on algae and detritus. Thus, mercury can be transferred and accumulated through three or four trophic levels to reach the prey of piscivorous wildlife species. Studies with lake trout suggest that differences in food web structure can substantially impact mercury accumulation by large predatory fish (Cabana and Rasmussen, 1994; Cabana et al., 1994; Futter, 1994).

2.2.2   Exposure Pathways in Terrestrial Systems

Several exposure pathways are possible for both plants and animals in terrestrial systems. The two main pathways by which terrestrial plants can be exposed to mercury are uptake from soils into the roots and absorption directly from the air. Potential exposure routes for terrestrial animals include the following: (1) ingestion of mercury-contaminated food; (2) direct contact with contaminated soil; (3) ingestion of mercury-contaminated drinking water; and (4) inhalation. Food ingestion is of primary concern for vertebrate carnivores (including humans). Once mercury enters a terrestrial food web, like that shown in Figure 2-5, it can be transferred in increasing concentrations to higher trophic levels (Talmage and Walton, 1993). A special case exists when terrestrial carnivores consume prey that have accumulated mercury originating from aquatic sources. Perhaps the best known example is that of the Florida panther, which consumes raccoons that have accumulated mercury through consumption of contaminated fish and shellfish (Roelke et al., 1991).

2.2.2.1 Terrestrial Plants

Uptake by plants plays a major role in the entry of metals to terrestrial food webs. Mercury uptake by terrestrial vascular plants[4] can occur through the roots or through the leaves, by way of stomata[5] (Mosbaek et al., 1988; Crowder, 1991; Maserti and Ferrara, 1991). A vascular plant's uptake of mercury from the soil depends on soil type, with uptake decreasing as organic matter, which binds mercury, increases (WHO, 1989). Uptake of mercury through leaves is considered to be a negligible source of mercury for beech and spruce (Schmidt, 1987) but is an important route for pines and herbaceous plants (Mosbaek et al., 1988; Maserti and Ferrara, 1991). Bryophytes and lichens have no roots and take up metals only from air or water (WHO, 1989; Crowder, 1991). Some species of bryophytes and lichens can bioconcentrate mercury to relatively high levels (e.g., up to 1200 μg/g in *Sphagnum* sp.) (Siegal et al., 1985). Some woody plants (e.g., *Pinus* sp.) also bioconcentrate mercury (Siegal et al., 1987).

2.2.2.2 Terrestrial Animals

Dietary exposure is the primary route of mercury uptake for vertebrate members of terrestrial food webs. Figure 2-5 illustrates a terrestrial food web. Plants are eaten by a wide diversity of herbivorous animals (e.g., grasshoppers, caterpillars, mice, voles, rabbits, and deer). Insects, earthworms and other soil macroinvertebrates can accumulate mercury to levels well above those of the soil in which they reside (Siegel et al., 1975; Helmke et al., 1979; Beyer et al., 1985), and are themselves consumed by many species of birds, shrews, snakes, and amphibians. Small mammals, birds, reptiles and amphibians are consumed by larger predators, such as owls, hawks, eagles, mink, and wolves. Thus, mercury can be transferred and accumulated through two or three trophic levels to reach the prey of top carnivores in terrestrial systems.

---

[4]Plants with roots, stems, and leaves, such as ferns and seed plants.

[5] Stomata (plural of stoma) are the minute openings in the epidermis of leaves, stems, or other plant organs that allow gas to diffuse in and out.

Figure 2-5
Example Terrestrial Food Web
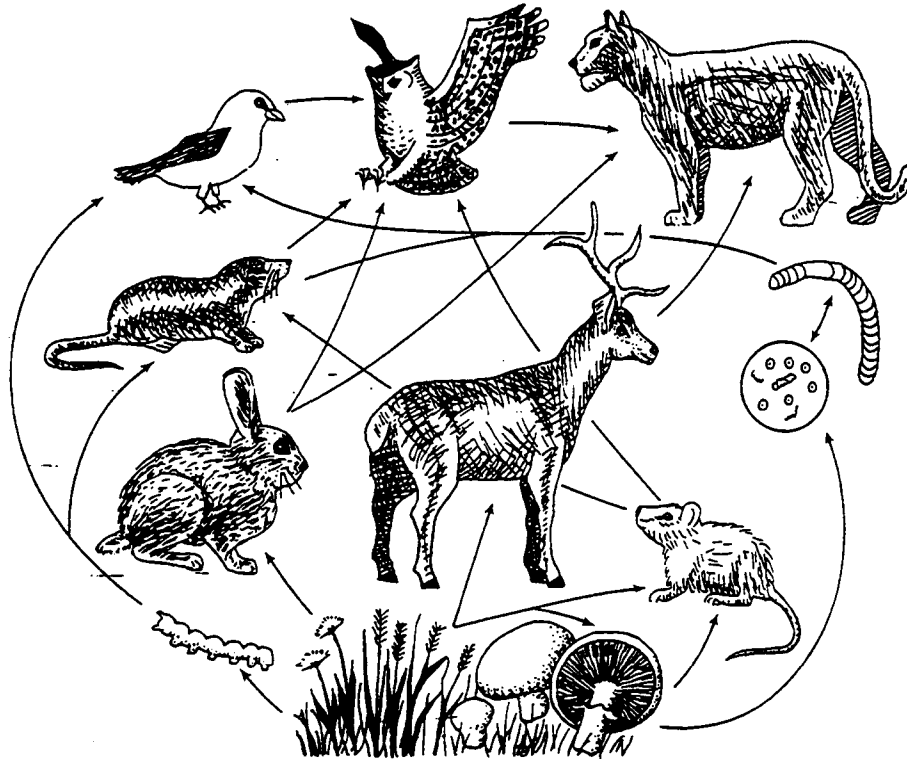
For these terrestrial animals, exposure to mercury depends largely on the animal's feeding strategy. For example, generalist herbivores (plant-eaters) may be less exposed to mercury than species that are specialized in or restricted to feeding on highly exposed plant species (e.g., reindeer foraging mostly on lichens and bryophytes).

2.2.3   Summary of Aquatic and Terrestrial Exposure Pathways

Food chain transfers of mercury are thought to be the most important exposure pathway in both aquatic and terrestrial ecosystems. Mercury, however, tends to bioaccumulate and biomagnify more strongly in aquatic than in terrestrial ecosystems. There are several possible explanations for this observation. First, the transfer of metals to higher trophic levels depends to some extent on where the metals are stored within prey organisms. Birds and mammals accumulate mercury in their feathers and fur, which are not eaten or are poorly digested. In contrast, most of the mercury in fish is contained in muscle tissue, which is consumed and digested by piscivorous wildlife. In addition, mercury in terrestrial food webs frequently exists in an inorganic form, rather than as methylmercury. Inorganic mercury accumulates to only a limited extent in plants and soil organisms and does not biomagnify in the organisms that feed on them. Finally, aquatic food chains often include more trophic levels than terrestrial food chains. A typical food chain in aquatic systems would consist of: phytoplankton/algae/detritus ⁻ zooplankton/benthic

invertebrates → small forage fish → larger piscivorous fish. Piscivorous birds and mammals would represent the fifth step in the chain. In some cases a sixth step exists, as when a bald eagle consumes a piscivorous herring gull. A typical food chain in terrestrial systems might be: plants → small herbivorous mammals → predatory birds and mammals. Another typical terrestrial food chain would be: plants → herbivorous insects → small birds → birds of prey. In these examples, the top predators represent the third and fourth step in the chain (although additional steps are possible), instead of the fifth or sixth level as can be the case for aquatic systems.

2.3   Ecological Effects

This section provides an overview of potential effects of mercury on ecosystems and components of ecosystems. Contaminants such as mercury can affect individual organisms, populations, communities, or ecosystems (see Table 2-1). Effects on individuals can be lethal or sublethal, including behavioral, reproductive and developmental effects. Additionally, effects can be immediate, due to acute (short-term) exposures or may be manifested only after chronic (long-term) exposures.

In animals, toxic effects caused by mercury exposure vary depending on a number of factors, including but not limited to these:

- delivered dose (i.e., amount and duration of exposure);

- the form of mercury to which an organism is exposed;

- physical and chemical parameters of the environment (e.g., pH, temperature, and DOC);

- the extent to which an organism is exposed to other chemical or non-chemical stressors;

- the life stage, age, sex, species, and physiological condition of the exposed organism; and

- the extent to which the organism can detoxify or eliminate absorbed mercury.

The remainder of this section provides an overview of potential adverse ecological effects of mercury. Section 2.3.1 discusses the bioaccumulation and biomagnification of mercury in food chains, Section 2.3.2 reviews individual-level effects, Section 2.3.3 reviews population-level effects, and Section 2.3.4 reviews effects on communities and ecosystems.

2.3.1   Bioaccumulation of Mercury

As discussed previously, plants and animals may absorb mercury from direct exposure to contaminated media. In addition, animals can acquire mercury through ingestion of mercury-contaminated food. These pathways determine how much mercury an organism is exposed to from outside sources. An additional factor that determines the effect of mercury on ecological systems is how much mercury is accumulated by organisms. Mercury accumulation can result in concentrations in exposed plants and animals that are higher than those in surrounding media or in ingested food. This section outlines the basic processes by which mercury accumulates and introduces the different ways that chemical accumulation in biological systems is measured and expressed.

Table 2-1
Examples of Effects of Contaminants on Ecosystem Components

Component	Examples of Effects
Individual	Change in respiration Change in behavior (e.g., migration, predator-prey interactions) Inhibition or induction of enzymes Increased susceptibility to pathogens Decreased growth Decreased reproduction Death
Population	Decreased genotypic and phenotypic diversity Decreased biomass Increased mortality rate Decreased fecundity rate Decreased recruitment of juveniles Increased frequency of disease Decreased yield Change in age/size class structure Extinction
Community	Decreased species diversity Change in species composition Decreased food web diversity Decreased productivity Increased algal blooms
Ecosystem	Decreased diversity of communities Altered nutrient cycling Decreased resilience

Three terms are commonly used to describe the mechanism by which a contaminant accumulates in living tissues. The term "bioconcentration" refers to the accumulation of a chemical that occurs as a result of direct contact of an organism with its surrounding medium (e.g., uptake by a fish from water through the gills and epithelial tissue or uptake by earthworms from soil through the skin) and does not include the ingestion of contaminated food. The term "bioaccumulation" refers to the net uptake of a contaminant from all possible pathways and includes the accumulation that may occur by direct exposure to contaminated media as well as uptake from food. The term "biomagnification" refers to the increase in chemical concentration in organisms at successively higher trophic levels as a result of the ingestion of contaminated organisms at lower trophic levels. Mercury is known to bioconcentrate, bioaccumulate and biomagnify. In fact, mercury is one of the few metals that is known to biomagnify in aquatic food webs.

Different numerical factors are used to estimate the extent to which a contaminant bioconcentrates, bioaccumulates and biomagnifies.

- The bioconcentration factor (BCF) is the ratio of a substance's concentration in tissues (generally expressed on a whole-body basis) to its concentration in the surrounding medium (e.g., water or soil) in situations where an organism is exposed through direct contact with the medium.

- The bioaccumulation factor (BAF) is the ratio of a substance's concentration in tissue to its concentration in the surrounding medium (e.g., water or soil) in situations where the organism is exposed both directly and through dietary sources.

- The biota-sediment accumulation factor (BSAF) is a specialized form of the BAF that refers to the chemical concentration in an aquatic organism divided by that in surficial (aquatic) sediments. To date it has been applied only to bioaccumulative organic compounds, but in principal it could be applied to mercury also. When applied to organic compounds, chemical concentrations in tissues and sediment are generally normalized for lipid content and organic carbon content, respectively.

- The predator-prey factor (PPF, also known as the biomagnification factor, or BMF) is the factor by which a substance's concentration in the organisms at one trophic level exceeds the concentration in the next lower trophic level. For example, the PPF for mercury at trophic level 4 equals the observed mercury concentration in trophic level 4 organisms divided by mercury concentration in trophic level 3 organisms.

- The food chain multiplier (FCM) is the factor by which the BAF of a substance at trophic level 2 or higher exceeds the BCF at trophic level 1. Implied by this definition is the assumption that organisms at trophic level 1 are at or near chemical equilibrium with their environment.

Although generally developed for individual organisms, BAF, BSAF, PPF and FCM values can also be viewed as trophic-level specific. Depending on environmental levels of mercury, sufficient mercury may accumulate in organisms at one or more trophic levels to produce adverse effects at the individual, population, community or ecosystem level.

Mercury accumulates in an organism when the rate of uptake exceeds the rate of elimination. All forms of mercury can accumulate to some degree; however, methylmercury generally accumulates to a greater extent than other forms. Methylmercury is absorbed into tissues quickly and becomes sequestered due to covalent reactions with sulfhydryl groups in proteins and other macromolecules (see Section 4 of this Volume for more detail). Inorganic mercury can also be absorbed but is usually taken up at a slower rate and with lower efficiency than methylmercury. Elimination of methylmercury takes place very slowly resulting in tissue half-lives (the time required for half of the mercury in the tissue to be eliminated) ranging from months to years (Westermark et al., 1975). Elimination of methylmercury from fish is so slow that long-term reductions of mercury concentrations in fish are often due mainly to growth of the fish. In comparison, other mercury compounds are eliminated relatively quickly, resulting in reduced levels of accumulation (Eisler, 1987).

Methylmercury and total mercury concentrations both tend to increase in aquatic organisms as the trophic level in aquatic food webs increases. In addition, the proportion of total mercury that exists as methylmercury generally increases with trophic level (May et al., 1987; Watras and Bloom, 1992; Becker and Bigham, 1995; Hill et al., 1996; Tremblay et al., 1996; Mason and Sullivan, 1997).

Accordingly, mercury exposure and accumulation is of particular concern for animals at the highest trophic levels in aquatic food webs and for animals that feed on these organisms.

2.3.1.1 Field-derived BAFs, BSAFs, and PPFs

In this section, BCFs for organisms that occupy the base of aquatic food chains are reviewed, along with BSAFs for fish and PPFs for avian and mammalian piscivores. BSAFs for earthworms and benthic invertebrates are also presented because both represent possible vectors for mobilization of sediment-associated mercury and subsequent translocation to wildlife. Median BAFs for fish occupying trophic levels 3 and 4 are derived in Volume III, Appendix D. A summary of these calculations is presented in Chapter 5 of this Volume.

Recent studies with marine phytoplankton suggest that mercury accumulation at the lowest levels of aquatic food webs is controlled largely by the availability of neutral mercury complexes (primarily $HgCl_2$ and CH_3HgCl) (Mason et al., 1996). Factors that can alter the concentration of these neutral species include pH, chloride concentration, and the amount of dissolved organic material. Additionally, it was found that most (63%) of the methylmercury that diffuses into phytoplankton becomes localized in the cytoplasm. Copepods assimilated almost all of this cytoplasmic mercury when they were fed contaminated phytoplankton. In contrast, inorganic mercury was concentrated predominantly (91%) in cell membranes and was poorly (15%) assimilated. Research on a Lake Michigan food web suggests that similar mechanisms may be responsible for controlling mercury uptake by freshwater phytoplankton (Mason and Sullivan, 1997). Such studies are extremely important, since mercury uptake at the lowest trophic levels is likely to be the single most important determinant of levels achieved by fish and piscivorous wildlife.

Data published by Becker and Bigham (1995) can be used to calculate a methylmercury BCF of 107,000 for phytoplankton in Onondaga Lake. Corrected for the (assumed) percentage of methylmercury in lake water (8%) and phytoplankton (24%), these data give a total mercury BCF of approximately 36,000. Using total mercury data reported by Mason and Sullivan (1997), and assuming that dry weight is 10% of wet weight, a BCF of about 7,000 can be calculated for phytoplankton in Lake Michigan. BCFs (total mercury basis, approximated from Hg^{2+} data) ranging from about 2,000 to 40,000 were reported for periphyton collected from two streams in eastern Tennessee (Hill et al., 1996). A total mercury BCF of approximately 20,000 was reported for phytoplankton in a northern Wisconsin lake (reference basin; Watras and Bloom, 1992). Expressed on a methylmercury basis, the BCF for phytoplankton in the same Wisconsin lake was approximately 90,000.

BAFs for zooplankton, expressed as ratios of total mercury, can be calculated from data presented by Sorenson et al. (1990), Lindqvist (1991) and Mason and Sullivan (1997). Respectively, the calculated values are 35,600, 285,000, and 3,100. A BAF of approximately 56,200 was reported for zooplankton by Watras and Bloom (1992; reference basin). Expressed on a methylmercury basis, the BAF measured by Watras and Bloom (1992) was about 1,000,000. Total mercury BAFs estimated for zooplankton in 12 northern Wisconsin lakes ranged from about 4,800 to 270,000 (Back and Watras, 1995). BAFs expressed on a methylmercury basis for the same 12 lakes ranged from about 11,000 to 12,600,000. Much of this variability appeared to be correlated (inversely) with lakewater DOC content. Work conducted by Slotten et al. (1995) and Suchanek et al. (1997) suggests that mercury bioaccumulation by zooplankton may vary seasonally, although in both of these studies data interpretation was complicated by the presence of mercury point sources. Becker and Bigham (1995) reported a methylmercury BAF of approximately 87,000 for zooplankton in Onondaga Lake, which has also received substantial mercury inputs from local point sources.

To date, BSAFs for mercury in aquatic biota have been estimated by only a few authors (e.g., Tremblay et al., 1996); however, a substantial amount of data exists that allows such calculations to be made. Hildebrand et al. (1980) observed a linear relationship between total mercury in sediment and that in benthic invertebrates. A BSAF of approximately 0.4 is obtained from the slope of this relationship (after expressing benthos data on a dry weight basis). The relationship between total mercury in fish (rock bass and hog suckers) and that in sediments was reported by Hildebrand et al. (1980) to be logarithmic. Taking as an average a fish tissue value of 4.0 μg/g (dry weight; converted from 1.0 μg/g wet weight) and solving for the sediment concentration yields a value of 2.78 μg/g. The BSAF is equal to the ratio of fish and sediment values, or approximately 1.4. Total mercury data presented by Sorenson et al. (1990) yield BSAFs (dry weight basis) of approximately 2.0 and 10.1 for zooplankton and northern pike, respectively. Data presented by Wren and MacCrimmon (1986) allow BSAFs to be calculated for two Ontario lakes that differed considerably with respect to total mercury residues in biota. In both lakes BSAFs (dry weight basis) were very similar, ranging from approximately 5.1 (clams) to 24.0 (northern pike) in the less contaminated of the two lakes, and 3.4 (clams) to 27.1 (pike) in the other system. Using the mid-range of values reported by Lindqvist (1991), BSAFs (dry weight basis) of approximately 2.2, 17.2, 17.7, and 45.7 are obtained for zooplankton, macroinvertebrates, yellow perch (small and large), and northern pike (large and small), respectively. Boyer (1982) reported total mercury concentrations in fish and sediments from several locations on the upper Mississippi River. Expressed on a dry weight basis, these data yield BSAFs ranging from 2.5 to greater than 50. Using "canal median" total mercury data from Stober et al. (1995), a BSAF (wet weight basis) of about 0.6 can be calculated for mosquitofish in the Florida Everglades region. This value would increase somewhat if expressed on a dry weight basis. Saouter et al. (1993) exposed mayflies for 10 days to methylmercury in sediment and obtained a BSAF (wet weight basis) of 4.0. A BSAF for zooplankton of about 1.4 (dry weight basis) can be calculated from mean total mercury data obtained in a survey of 73 Canadian lakes (Tremblay et al., 1995). Tremblay et al. (1996) reported the BSAF (dry weight basis) for aquatic insects to be about 3.0 when calculated using total mercury data, and from 6.0 to 22.0 when expressed on a methylmercury basis.

In summary, BSAFs calculated for total mercury in aquatic biota ranged from 0.4 to about 50 and, within a given system, appeared to increase with trophic level. In terms of both magnitude and the increase with trophic level, BSAFs for mercury are similar to BSAFs reported for hydrophobic organic compounds (lipid/carbon normalized). It could be hypothesized, therefore, that similar processes are at work. This is unlikely, however, since bioaccumulation of organic compounds is largely a partitioning process, while for mercury the chemical interactions tend to more specific, often involving the formation of covalent bonds. Because mercury does not partition into lipid, normalization for lipid content makes little sense. The existence of strong relationships between mercury and organic carbon content (see for example Wiener et al., 1982; Lindqvist, 1991) suggests, however, that some type of sediment carbon normalization may be appropriate. A single study by Tremblay et al. (1996) suggests that within a given system BSAFs expressed on a methylmercury basis will exceed values calculated using total mercury data. While likely at higher trophic levels, additional data at lower trophic levels are needed to determine the extent to which this observation may be generalized.

Limited data are available that allow calculation of BSAFs for earthworms. The concentration of mercury in earthworms collected from an uncontaminated field site was 27.1 times that of soil and 6.9 times that of decaying vegetation (dry weight basis) (Siegel et al., 1975). In a 12 week laboratory exposure, earthworms accumulated an average of 21.3 times the mercury concentration of the soil to which they were exposed (including control and treatment groups) (Beyer et al., 1985).

PPFs for piscivorous birds and mammals are difficult to determine accurately because residue data cannot be attributed with any specificity to residues in a particular prey item; feeding observations for the species in question are rarely reported in these studies. Where possible, PPFs were estimated by constructing rough averages of residue values in prey items occupying similar trophic levels. For this analysis, mink, mergansers, and loons were assumed to feed exclusively at trophic level 3. River otters were assumed to feed at trophic levels 3 (80%) and 4 (20%).

PPFs calculated for piscivorous birds from breast muscle mercury levels ranged from 1.7 for the hooded merganser (Vermeer et al., 1973) to 7.7 for the herring gull (Wren et al., 1983). Intermediate values were calculated for the common merganser (2.5) (Vermeer et al., 1973) and loon (6.8) (Wren et al., 1983). Data collected by Wren et al. (1996) from Muskota, Ontario, permit PPFs to be calculated for mink and otter. Calculated from liver residues, these data yield PPFs of 6.2 and 4.7, respectively. Muscle tissue data reported in the same study yield PPFs of 8.1 and 1.7 for mink and otter, respectively. A PPF of 3.0 (muscle tissue basis) can be calculated for otters from Tadenac Lake, Ontario (Wren et al., 1993). Averaged across sampling locations and assuming consumption of the fish species analyzed, PPFs of 2.7 (muscle basis) and 5.7 (liver basis) may be estimated for otters in Georgia (Halbrook et al., 1994).

In a study designed specifically to assess the degree of mercury biomagnification in piscivorous mammals, liver residues were paired by location with residue levels in fish (Foley et al., 1988). These data yield PPFs of 3.9 and 3.4 for mink and otter, respectively. Kucera (1983) reported that the ratio of mercury concentrations in mink and otter to that in predatory fish in the same region was about 10. A similar conclusion was reached by Francis and Bennett (1994) for otters in northern Michigan, based upon an analysis of liver tissues. Thus, it can be shown that mercury biomagnifies in piscivorous wildlife, although the extent of this biomagnification is less than that typically reported for persistent organic compounds. For example, data reported by Braune and Norstrom (1989) suggest that the PPF for PCBs in piscivorous birds can approach 100. These observations have led to the suggestion that mercury is eliminated by piscivorous wildlife in feathers and fur, and perhaps also via a demethylation pathway (Wren et al., 1986); however, extensive elimination would be expected to result in PPFs of 1 or less.

2.3.1.2 Mercury Residues in Fish

Consistent with a need to characterize the exposure of piscivorous avian and mammalian wildlife to mercury, an effort was made to estimate "national average" values for mercury in fish at trophic levels 3 and 4. The calculation of true "national average" values would require the collection of a large number of samples from randomly selected lakes and rivers. Instead, the published literature contains a number of papers in which mercury concentrations are given for relatively small numbers of fish from restricted geographical regions. Many of these studies were initiated due to known or suspected problems with mercury in the region of interest. Thus, a sample developed from a compilation of these data could be biased toward the high-end of the distribution of mercury concentrations nationwide.

A survey of the literature revealed only three nationwide fish collection efforts that used consistent sampling and mercury measurement techniques. In a study conducted by U.S. EPA, samples were obtained from 374 sites across the U.S. (U.S. EPA, 1992b; Bahnick et al., 1994). Site selection was based partly on proximity to suspected point and non-point pollution sources, and a majority of sites were located on streams and rivers. Additionally, fish were collected from 35 "remote" sites that were thought to provide background pollutant concentrations in fish. Whole-body mercury levels were determined for bottom feeders, and mercury levels in fillets were analyzed for game fish. The maximum mercury level detected was 1.80 μg/g wet weight, and the mean across all fish and sites was 0.26 μg/g (see Table 2-2).

The highest values were detected in piscivorous game fish (trophic level 4), including walleye, bass and northern pike. Lower levels were found in herbivores (e.g., carp and sucker), omnivores (e.g., catfish), and species that prey extensively on insects (e.g., trout and crappie). In general, this sampling effort did not address fish that occupy trophic level 3 (forage fish).

Table 2-2
Nationwide Average of Mercury Residues in Fish

Fish Species	Mercury Concentration Averaged Across Sampling Sites (μg/g wet weight)
Carp	0.11
Sucker (White, Redhorse and Spotted)	0.17
Catfish (Channel and Flathead)	0.16
Bass (Largemouth, Smallmouth and White)	0.38
Walleye pike	0.52
Northern pike	0.31
Crappie	0.22
Brown Trout	0.14
Mean of All Fish Sampled	0.26

Source: Bahnick et. al., 1994.

Mercury levels in fish were measured at over 100 sites as part of the National Contaminant Biomonitoring Program (NCBP) administered by the U.S. Fish and Wildlife Service. Two compilations of NCBP mercury data have been published. The first summarizes data collected from 1978-1981 (Lowe et al., 1985). The second reports on data collected from 1984-1985 and draws comparisons with the results
of the earlier study (Schmitt and Brumbaugh, 1990). As with the Bahnick et al. (1994) study, most of the sampling sites were located on streams and rivers, many of which receive municipal and other waste. In addition, similar species were collected, with an emphasis on large piscivores, herbivores and omnivores. A review of these data suggests that piscivores accumulate more mercury than other fish species. Thus, lake trout (mean concentration of 0.17 µg/g) and largemouth bass (0.14 µg/g) contained more mercury than co-collected non-piscivorous species (0.07 and 0.09 µg/g, respectively). The maximum mercury concentration reported was 1.09 µg/g, and the mean across all fish and sites was 0.11 µg/g. Of importance for calculating a "national average" mercury concentration in fish, Schmitt and Brumbaugh (1990) reported that mercury levels in fish did not change between 1976 and 1984. Attention was focused, therefore, on the Lowe et al. (1985) study because it comprised a larger number of individual samples and because fish length and weight were also reported.

An average mercury concentration in piscivorous fish analyzed by Bahnick et al. (1994) was calculated from data presented by these authors (Table 3 in their report). For this Report, the following species were classified as trophic level 4 piscivores: largemouth bass, smallmouth bass, walleye, brown trout, white bass, and northern pike. The mean ($\pm$ SD) of concentration data presented for these six species is 0.35 $\pm$ 0.13 µg/g.

An average value for piscivores analyzed by Lowe et al. (1985) was estimated using data presented by these authors (Appendix A in their report). Each value reported for a site and species represented a composite of three to five fish. The criteria established for using a reported value were: (1) the species is a recognized piscivore; (2) the average size of specimens comprising a sample was > 0.5 kg; and (3) the sampling site was located in the contiguous 48 states. For this Report, the species identified as trophic level 4 piscivores were: largemouth bass, smallmouth bass, striped bass, white bass, rock bass, northern pike, walleye, sauger, lake trout, brown trout, rainbow trout, and northern squawfish. The mean ($\pm$ SD) of all data presented for these twelve species was 0.18 $\pm$ 0.19 µg/g (N = 119), or just over one-half the concentration calculated using the Bahnick et al. (1994) data.

A "national average" mercury concentration for trophic level 4 fish was estimated as the average of mean values calculated using data from Bahnick et al. (1994) and Lowe et al. (1985). This value is 0.26 µg/g. As indicated above, neither of these nationwide sampling efforts adequately characterized mercury concentrations in fish at trophic level 3. A "national average" for trophic level 3 was therefore estimated by dividing the average mercury concentration in piscivorous fish by an appropriate predator-prey factor (PPF). A PPF for trophic level 4 (PPF_4) can be estimated from existing field data. This calculation was made in Appendix D, Volume III of this Report, resulting in a mean PPF_4 of 4.9. Dividing this value into the average residue for trophic level 4 fish yields a value for trophic level 3 of 0.052 μg/g.

The extent to which these "national average" estimates reflect the true population means at each trophic level is unknown. A comparison of these values with published residues from a large number of studies suggests, however, that they are "reasonable" and can be used in exposure assessments for piscivorous avian and mammalian wildlife.

2.3.1.3 Mercury Residues in Avian and Mammalian Wildlife

A large volume of mercury residue data exists for both avian and mammalian wildlife that cannot be directly related to mercury concentrations in water or sediment. Nevertheless, these data are of considerable value because they indicate the range of mercury concentrations that can be expected in animals inhabiting both contaminated and uncontaminated environments. A comparison of these residues with those obtained from laboratory dose-response studies provides additional information, including the extent of difference between "natural background" residues and those that are associated with toxic effects. Emphasis is placed on piscivorous birds and mammals living in association with freshwater ecosystems. Data are also provided for the tree swallow due to its link to aquatic sediments through consumption of emergent insects.

Mercury residues in tissues from birds are given in Table 2-3. The birds represented in this table include animals taken from polluted environments and individuals collected from environments for which there were no known point sources. This table is not intended to be an exhaustive compilation of measured residues, but instead illustrates the range of values encountered in environmental sampling efforts. Residues that, in the opinion of the cited author, were associated with toxic effects are noted.

Factors contributing to the accumulation of mercury in wild birds are reviewed by Scheuhammer (1987, 1991). The interpretation of residue data is complicated by the likelihood that mercury distribution in tissues varies among species, and perhaps also among individuals of a single species, depending upon age, sex, diet, and other factors. Nevertheless, several generalizations can be attempted. Mercury levels in feathers of birds experimentally dosed with methylmercury generally exceed levels in muscle, liver and kidney by a factor of four or more (Heinz, 1976a; Stickel et al., 1977; Finley and Stendell, 1978), and it has been suggested that in free-living birds greater than 50% of the total body burden of mercury may be

Table 2-3
Mercury Residues in Tissues of Piscivorous Birds

Species	Mercury (µg/g fresh weight)	Tissue	Sampling Location	Comments	Reference
Bald eagle	13.0 - 21.0	feathers	Great Lakes region	adults	1
Bald eagle	3.7 - 20.0	feathers	Great Lakes region	nestlings	1
Bald eagle	0.1 - 34.7		N. Central Florida	adults	2
Bald eagle	0.8 - 14.3	feathers	N. Central Florida	nestlings	2
Common loon	8.7	feathers	Minnesota lakes	adults	3
Common loon	2.7	feathers	Minnesota lakes	juveniles	3
Common loon	11.0 - 18.0	feathers	Wisconsin lakes	adults	4
Common loon	2.0 - 5.0	feathers	Wisconsin lakes	juveniles	4
Wood stork	1.9	feathers	South Florida	juveniles	5
Bald eagle	0.15 - 0.29	eggs	British Columbia		6
Bald eagle	0.07 - 0.41	eggs	15 States (USA)		7
Common loon	0.40 - 1.10	eggs	Wisconsin lakes		4
Common loon	2.0 - 3.0	eggs	Northwestern Ontario	polluted by point source; LOAEL - reproduction	8
Common tern	3.6	eggs	Northwestern Ontario	polluted by point source; LOAEL - reproduction	9
Herring gull	2.3 - 15.8	eggs	Clay Lake, Ontario	polluted by point source, no adverse effects	10

Table 2-3 (continued)
Mercury Residues in Tissues of Piscivorous Birds

Species	Mercury (µg/g fresh weight)	Tissue	Sampling Location	Comments	Reference
Wood stork	0.7	eggs	South Florida		11
Tree swallow	0.04 - 0.08	eggs	Lower Great Lakes	consume emergent aquatic insects	12
Common loon	1.6 - 47.7	liver	Northwestern Ontario	LOAEL - reproduction	8
Common loon	9.5 - 90.0	liver	Wisconsin lakes	adults found dead	4
Common loon	5.6	liver	Minnesota lakes	adults found injured	3
Great White Heron	0.6 - 59.4	liver	South Florida	mixed age birds found dead	13
Great Blue Heron	0.2 - 7.3	liver	South Florida	nestlings	14
Great Blue Heron	0.1 - 74.5	liver	South Florida	fledglings/young adults	14
Common loon	0.2 - 6.9	breast muscle	Northwestern Ontario	polluted by point source	8
Common goldeneye	0.9 - 19.4	breast muscle	Clay Lake, Ontario	polluted by point source	10
Common merganser	4.4 - 13.1	breast muscle	Clay Lake, Ontario	polluted by point source	10
Hooded merganser	3.9 - 17.6	breast muscle	Clay Lake, Ontario	polluted by point source	10
Herring gull	0.7 - 4.0	breast muscle	Tadenac Lake, Ontario		15

Table 2-3 (continued)
Mercury Residues in Tissues of Piscivorous Birds

Species	Mercury (µg/g fresh weight)	Tissue	Sampling Location	Comments	Reference
Common loon	1.5	breast muscle	Tadenac Lake, Ontario		15

References:
1. Bowerman et al., 1994; range of means across sampling locations.
2. Wood et al., 1996; range of contour feathers recovered at nest sites. Means for nestlings and adults were 3.2 and 6.0, respectively.
3. Ensor et al., 1992; mean of birds caught by nightlighting.
4. Belant and Anderson, 1990; range of individual values. Means for feathers (adult and juvenile), eggs and liver were 14.8, 4.0, 0.64 and 40.9, respectively.
5. Burger et al., 1993; mean value.
6. Elliott et al., 1996; range of means across sampling locations
7. Wiemeyer et al., 1993; range of means across sampling locations (collected after failure to hatch).
8. Barr, 1986; range of individual values. Means for liver and muscle were 13.0 and 2.3, respectively.
9. Fimreite, 1974.
10. Vermeer et al., 1973; range of individual values. Means for goldeneye, common merganser and hooded merganser were 7.8, 6.8 and 12.3, respectively.
11. Fleming et al., 1984; mean value.
12. Bishop et al., 1995; range of individual values, mean = 0.07.
13. Spalding et al., 1994; range of individual values. Means for birds that died of acute and chronic causes were 1.8 and 9.8, respectively.
14. Sundlof et al., 1994; range of individual values. Means for small nestlings, large nestlings and adults were 0.3, 1.5 and 6.6, respectively.
15. Wren et al., 1983; gull data are reported as the range of individual values, mean = 1.7.

present in the plumage (Braune and Gaskin, 1987). Natural background levels of mercury in feathers of non-piscivorous raptorial birds are thought to range from 1-5 μg/g (dry weight); however, this may vary within and among species depending upon the type of feather sampled, molting frequency and time to last molt. Changes in feather mercury levels that accompany growth and development suggest that in seabirds molting may be an efficient means of eliminating mercury (Becker et al., 1994; Burger et al., 1994). Comparable studies have not yet been conducted with birds that live in freshwater ecosystems.

Tissue levels of mercury associated with toxic effects in birds appear to exceed those in birds inhabiting relatively uncontaminated environments by a factor of ten or less (see Sections 2.3.2 and 2.3.3 for additional details). This observation is consistent with data for other environmental media (e.g., water, sediments, and fish), which evidence similar differences between natural "background" levels of mercury and those which cause significant environmental damage. Owing to their ease of collection, the analysis of bird feathers and eggs has been suggested as a means of identifying species that are at risk due to mercury. This suggestion has particular merit in view of the natural variation in mercury levels in the fish upon which these animals prey. Mercury residues in tissues also tend to integrate variations in mercury uptake and elimination due to changes in dietary habits, migration, egg production, etc.

The abundance of mercury residue information for mammals reflects the availability of specimens as a byproduct of commercial trapping. Thus, residue data are available for wild muskrat, beaver, fox, weasel, bobcat, marten, fisher, wolf, raccoon, opossum, mink and river otter. Data are also available for a number of game species, including squirrels, rabbits, caribou, moose, deer, elk, mountain goat and bear. An extensive compilation of these data is provided by Wren (1986), along with a review of tissue levels in both wild and laboratory animals that have been associated with toxic effects. Some of the data from this compilation are presented in Table 2-4, as well as more recent information. Emphasis was placed on piscivorous species due to the exposure of these animals from consumption of contaminated fish. Data from beaver and muskrat have also been included, both to provide a general comparison of aquatic-based species and because, in several studies, data were available for piscivores and herbivores from the same waterbody. Emphasis was also placed on residues in fur and liver. This was done for two reasons: (1) high residues have been found in the liver and kidney; however, there are more reported values for liver and (2) fur, like feathers, has been suggested as a way of non-invasively determining the residue status of wild animals and of identifying areas where animals may be at risk due to mercury intoxication. Finally, data from raccoons are included in Table 2-4 because they are suspected of contributing to mercury exposure in the Florida panther.

In general, the rank order of mercury residues in tissues from wild mink and otter is: liver = kidney > muscle > brain. Levels in fur relative to those in other tissues are variable but, in most cases, are higher than those in liver. Comparisons between residues in wild animals with those in animals experimentally dosed with mercury appear to be complicated by kinetics-based differences in disposition. Thus, Wobeser et al. (1976b) reported that mercury levels in the fur of experimentally dosed mink were low (1.5 μg/g) relative to concentrations in liver (24.3 μg/g), kidney (23.1 μg/g), muscle (16.0 μg/g) or brain (11.9 μg/g). A similar pattern of distribution was reported for mink by Aulerich et al. (1974). In contrast, mercury levels in the fur of an individual mink found dying of mercury poisoning were higher than concentrations in any other tissue (see Table 2-4) (Wobeser and Swift, 1976). Apparently, the length of time over which a dose is obtained dictates its distribution, with redistribution from well-perfused organs (liver and kidney) to storage tissues (fur and muscle) slowly taking place during lifetime exposures. These observations suggest that comparisons between mercury residues in wild and experimental animals should be limited to consideration of well-perfused tissues. More valid comparisons can be made between apparently unaffected wild animals and wild animals that have died from mercury poisoning.

Table 2-4
Mercury Residues in Tissues of Piscivorous Mammals

Species	Mercury (µg/g fresh weight)	Tissue	Sampling Location	Comments	Reference
Otter	6.5 (max. = 63.2)	fur	Wisconsin		1
Otter	47.0	fur	Clay Lake, Ontario	polluted by point source; death due to poisoning	2
Otter	15.2 - 25.6		Georgia		3
Mink	10.7 (max. = 17.3)	fur	Georgia		4
Mink	7.6 (max. = 41.2)	fur fur	Wisconsin		1
Mink	34.9	fur	Saskatchewan	polluted by point source; death due to poisoning	5
Raccoon	4.4	fur	S. Florida		6
Muskrat	0.06	fur	Wisconsin		1
Beaver	0.03	fur	Wisconsin		1
Otter	5.1 - 9.2	liver	Georgia		3
Otter	1.7 - 3.4	liver	Manitoba	males and females	7
Otter	2.4 - 4.5	liver	Winnipeg R.	males and females; polluted by point source	7
Otter	0.3 - 3.0	liver	Louisiana		8
Otter	0.9 - 3.5	liver	Ontario	residues correlated with acidity	9

Table 2-4 (continued)
Mercury Residues in Tissues of Piscivorous Mammals

Species	Mercury (µg/g fresh weight)	Tissue	Sampling Location	Comments	Reference
Otter	0.8 - 3.2	liver	N. Michigan		10
Otter	1.3 - 2.3	liver	New York		11
Otter	96.0	liver	Clay Lake, Ontario	polluted by point source; death due to poisoning	5
Otter	3.3 (max. = 23.6)	liver	Wisconsin		1
Mink	0.4 - 1.7	liver	Manitoba		7
Mink	2.1 (max. = 17.4)	liver	Wisconsin		1
Mink	0.1 - 2.6	liver	Ontario	residues correlated with acidity	9
Mink	58.2	liver	Saskatchewan	polluted by point source; death due to poisoning	5
Mink	0.9 - 2.9	liver	New York		11
Raccoon	2.0	liver	Wisconsin		1
Raccoon	1.5 - 24.0	liver	S. Florida		12
Muskrat	< 0.02	liver	Wisconsin		1
Beaver	0.04	liver	Wisconsin		1

References:
1. Sheffy and St. Amant, 1982; mean value.
2. Wren, 1985; one individual.
3. Halbrook et al., 1994; range of means across sampling locations.

4. Cumbie, 1975; mean value.
5. Wobeser and Swift, 1976; one individual.
6. Bigler et al., 1975; mean value.
7. Kucera, 1983; Manitoba data are reported as the range of means across sampling locations. Data from the Winnipeg river are reported as a mean value.
8. Beck, 1977; range of means across sampling locations.
9. Wren et al., 1986; range of means across sampling locations.
10. Francis and Bennett, 1994; range of individual values.
11. Foley et al., 1988; range of means across sampling locations.
12. Roelke et al., 1991; range of means across sampling locations.

An examination of Table 2-4 suggests that mercury residues in tissues from mink and otters from Wisconsin (Sheffy and St. Amant, 1982) approached, and in several cases even exceeded, those of the "naturally" poisoned animals. High mercury residues in fur were also reported for river otters trapped in several locations across Georgia (Halbrook, 1994). The livers of raccoons captured in South Florida are also notably high in mercury content (Roelke et al., 1991).

2.3.2   Individual Effects

Exposure to mercury can cause adverse effects in a wide variety of organisms, including plants, fish, aquatic invertebrates, birds and mammals. In this section, we review information on exposure levels that can cause adverse effects in these groups.

2.3.2.1 Individual Effects on Plants

Effects of mercury on aquatic plants include death and sublethal effects. Sublethal effects include plant senescence, growth inhibition, decreased chlorophyll content, decreased protein and RNA content, inhibited catalase and protease activities, inhibited and abnormal mitotic activity, increased free amino acid content, discoloration of floating leaves, and leaf and root necrosis (Boney, 1971; Stanley, 1974; Muramoto and Oki, 1984; Mhatre and Chaphekar, 1985; Sarkar and Jana, 1986). The level of mercury that results in toxic effects varies greatly among aquatic plants, as illustrated in Table 2-5.

Table 2-5
Toxicity Values for Aquatic Plants

Water Type	Hg^{2+} (HgCl or HgNO$_3$) (μg/L)		Methylmercury (μg/L)	
	Low End	High End	Low End	High End
Fresh Water	53 (alga)	3,400 (submerged aquatic vegetation)	0.8 (alga)	6.0 (alga)
Salt Water	10 (alga)	160 (seaweed)	Not available	Not available

Source: U.S. EPA, 1985.

Mercury can also cause death and sublethal effects in terrestrial plants. Sublethal effects on terrestrial plants include decreased growth, leaf injury, root damage, inhibited root growth and function, hampered nutrient uptake, chlorophyll decline and reduced photosynthesis (Schlegel et al., 1987; Lindqvist, 1991; Godbold, 1991).

Methylmercury is more toxic to terrestrial plants than Hg^{2+}. One to ten nM (nanomolar) mercuric chloride or methyl mercuric chloride (provided in a nutrient solution) can inhibit root elongation in spruce seedlings. However, methyl mercuric chloride has a greater effect than mercuric chloride at the same concentration (Godbold, 1991). Sublethal effects, including decreased transpiration, decreased chlorophyll concentration, partial stomatal closure, and reduced photosynthesis, occurred at nutrient solution concentrations of 10 nM methyl mercuric chloride (Schlegel et al., 1987).

2.3.2.2 Individual Effects on Fish and Aquatic Invertebrates

The toxicity of mercury to fish has been reviewed by Eisler (1987) and more recently by Wiener and Spry (1995). The highest mercury concentrations in fish generally occur in the blood, spleen, kidney and liver, and may exceed those in muscle by a factor of 2-10 (McKim et al., 1976; Olson et al., 1978; Ribeyre and Boudou, 1984; Boudou and Ribeyre, 1985; Harrison et al., 1990; Niimi and Kissoon, 1994). Owing to the size of these organs relative to that of other tissues, however, most of the mercury contained in a fish at any given time is associated with muscle tissue.

The toxicity of mercury varies, depending on the fish's characteristics (e.g., species, life stage, age, and size), environmental factors (e.g., temperature, salinity, dissolved oxygen content, hardness, and the presence of other chemicals), and the form of mercury available. In particular, early life stages (especially of salmonids) exhibit greater sensitivity to elevated metal concentrations than later life stages. The toxicity of Hg^{2+} compounds to salmonids and catfish tends to increase with temperature (see Table 2-6). Organomercury compounds, such as methylmercury, generally are much more acutely toxic than Hg^{2+} to aquatic organisms.

Table 2-6
Mercury Toxicity Increases With Temperature

Temperature (°C)	LC_{50} (μg/l)
Rainbow Trout with HgCl	
5	400
10	280
15	220
Juvenile Catfish with Phenylmercuric Acetate	
10	1,960
16.5	1,360
24	233

Source: U.S. EPA, 1985.

Effects of mercury on fish include death, reduced reproduction, impaired growth and development, behavioral abnormalities, altered blood chemistry, impaired osmoregulation, reduced feeding rates and predatory success, and effects on oxygen exchange. LC_{50} values for fish range from 30 μg/L for guppies to 1,000 μg/L for the Mozambique tilapia (U.S. EPA, 1985). Symptoms of acute mercury poisoning in fish include increased secretion of mucous, flaring of gill opercula, increased respiration rate, loss of equilibrium and sluggishness. Signs of chronic poisoning include emaciation, brain lesions, cataracts, inability to capture food, abnormal motor coordination and various erratic behaviors (e.g., altered feeding behavior) (Weis and Weis, 1989, 1995).

It is generally thought that toxic effects are unlikely to occur in fish in the environment, except in the case of point source pollution discharges. An accumulating body of evidence, however, suggests that histological changes and effects on behavior, reproduction, and development can occur at water concentrations as low as 0.1

μg/L (Wiener and Spry, 1995), or about two orders of magnitude higher than those generally associated with unpolluted systems. In a recent study, juvenile walleye were exposed to methylmercury in the diet at concentrations of 0.1 and 1.0 µg/g (Friedmann et al., 1996). Growth, development and hormonal status were impacted at the high dose level. No effects were seen at the lower dose level or in controls. The high dose level used in this study is within a factor of 10 of values reported for macroinvertebrates and forage fish from mercury-impacted "pristine" lakes (i.e., no known point source) in both Canada and the U.S. (Allard and Stokes, 1989; Sorenson et al., 1990; Watras and Bloom, 1992).

Levels of mercury that induce toxic effects in aquatic invertebrate species vary. For Hg^{2+}, acute values (LC_{50}) for invertebrates range from 2.2 μg/L for the cladoceran *Daphnia pulex* to 2,000 μg/L for the larval forms of three insects (U.S. EPA, 1985). Examples of some specific toxicity values for fish and aquatic invertebrates are provided in Table 2-7.

2.3.2.3 Individual Effects on Birds

Methylmercury has been shown to be more toxic to birds than inorganic mercury. Mercury poisoning in birds is characterized by muscular incoordination, falling, slowness, fluffed feathers, calmness, withdrawal, hyperactivity, hypoactivity and eyelid drooping (reviewed by Eisler, 1987; Fimreite, 1979; Scheuhammer, 1987, 1991). Acute oral toxicity studies using methylmercury yielded LD_{50} values ranging from 2.2 to 23.5 μg/g for mallards (*Anas platyrhynchos*), 11.0 to 27.0 μg/g for quail (*Coturnix*) and 28.3 μg/g for northern bobwhite (*Colinus virginianus*). Some bird kills have occurred, generally due to ingestion of mercury-based fungicides applied to grain. Whole-body residues of mercury in acutely poisoned birds usually exceed 20 μg/g fresh weight and have been found up to 126 μg/g. Mercury levels observed in such cases are generally highest in the brain, followed by the liver, kidney, muscle and carcass.

Sublethal effects of mercury on birds include liver damage, kidney damage, neurobehavioral effects, reduced food consumption, weight loss, spinal cord damage, effects on enzyme systems, reduced cardiovascular function, impaired immune response, reduced muscular coordination, impaired growth and development, altered blood and serum chemistry, and reproductive effects (Eisler, 1987; Scheuhammer, 1987, 1991; MDNR, 1993). Reproductive and behavioral effects are the primary concern, however, and can occur at dietary concentrations well below those that cause overt toxicity.

Scheuhammer (1991) concluded that on the basis of laboratory dose-response studies (Heinz, 1976a; Finley and Stendell, 1978), piscivorous birds consuming diets containing >1 μg/g (dry weight) methylmercury in their diet (approximately 0.25 μg/g wet weight) will accumulate >20 μg/g (dry weight) in their feathers. Similar levels in both spiked diets (Heinz, 1974, 1976a,b, 1979) and natural prey sources (Barr, 1986) have been shown to be toxic to birds. Thus, it appears that mercury levels in feathers exceeding 20 μg/g should be interpreted as evidence for possible toxic effects. Eisler (1987) recommended that 5.0 μg/g fresh weight in feathers be used as a criterion for the protection of birds.

Tissue mercury concentrations that are associated with toxicity in birds are remarkably similar despite differences in species, dietary exposure level and length of time necessary to produce the effect (Scheuhammer, 1991). Frank neurological signs are generally associated with brain mercury concentrations of 15 μg/g (wet weight) or higher and 30 μg/g or more in liver and kidney. Liver mercury concentrations of 2-12 μg/g (wet weight) were associated with reproductive impairment in adult pheasants and mallard ducks (Fimreite, 1971; Heinz, 1976a,b). Mortality was observed in newly hatched ducklings

Table 2-7
Toxicity Values for Fish and Aquatic Invertebrates

Organism	Hg^{2+} ($HgCl$ or $HgNO_3$) ($\mu g/L$)	Methylmercury ($\mu g/L$)
A C U T E (LC$_{50}$)		
Fresh water invertebrates	2.2 (cladoceran) to 2,000 (insect larvae)	Not available
Fresh water fish	30 (guppy) to 1,000 (tilapia)	Not available
Rainbow trout	155 to 420	24 to 84
Fresh water AWQC[a]	2.4 (total mercury)	
Salt water invertebrates	3.5 (mysid) to 400 (soft clam)[b]	Not available
Salt water fish	36 (juvenile spot) to 1,678 (flounder)[c]	51.1 (mummichog)
Salt water AWQC[a]	2.1 (total mercury)	
C H R O N I C		
Fresh water invertebrates	0.96 (cladoceran) to 1.287 (cladoceran)	< 0.04 (cladoceran)
Fresh water fish	< 0.23 (minnow) to < 0.26 (minnow)	0.29 (brook trout) to 0.93 (brook trout)
Fresh water AWQC[a]	0.012 (total mercury)	
Salt water invertebrates	1.131 (mysid)	Not available
Salt water AWQC[a]	0.025 (total mercury)	

[a] AWQCs are designed to be protective of the aquatic community as a whole.
[b] As cited in U.S. EPA, 1985, LC$_{50}$s of 10,000 and 8,700 $\mu g/L$ for Atlantic clams (*Rangia cuneata*) were reported by Olson and Harrell (1973), but Dillon (1977) reported LC$_{50}$ values of 58 and 122 $\mu g/L$ for the same clam species.
[c] As cited in U.S. EPA, 1985, an LC$_{50}$ of 2,000 $\mu g/L$ for mummichogs was reported by Klaunig et al. (1975), but Dorfman (1977) and Eisler and Hennekey (1977) reported LC$_{50}$ values of 800 $\mu g/L$ or less for the same fish species.

Source: U.S. EPA, 1985 except where otherwise noted.

with brain mercury concentrations of 3-7 $\mu g/g$ (wet weight), while levels four times these values are required to cause mortality in adults (Stoewsand et al., 1974; Finley et al., 1979; Scheuhammer, 1988).

Reproductive impairment has been observed in laboratory studies when mercury concentrations in eggs exceed 0.5 $\mu g/g$ (Fimreite, 1971; Heinz, 1974, 1976a,b, 1979). Field studies tend to confirm these results. Reproductive impairment in the loon was associated with mercury levels in eggs ranging from 2-3 $\mu g/g$ (Barr, 1986). Adverse effects on hatching and fledging were observed when mercury concentrations in the eggs of common terns exceeded 3.6 $\mu g/g$ (Fimreite, 1974). Mercury appeared to be a contributing factor to reduced reproductive success in raptors at some locations (Odsjö, 1982; Evans, 1986). In one study, however, hatching in herring gulls appeared to be unaffected, despite the fact that eggs contained upwards of 10 $\mu g/g$ of mercury

(Vermeer et al., 1973). Lowest-observed-adverse-effect level (LOAEL) and no-observed-adverse-effect level (NOAEL) values for effects of methylmercury on avian wildlife are derived in Section 4.2.2 of this Volume. Possible effects on populations of selected avian species are discussed in Section 2.3.3 of this Volume.

2.3.2.4 Individual Effects on Mammals

Extensive research on the toxicity of mercury to mammals indicates that effects vary depending on the form of mercury ingested or inhaled. Inorganic mercury is corrosive, and acute exposure to humans and other mammals may cause burning, irritation, salivation, vomiting, bloody diarrhea, upper gastrointestinal tract edema, abdominal pain, and hemorrhaging (Goyer, 1993). Ingestion of mercurial salts in large doses may cause kidney damage (Zalups and Lash, 1994). The main toxic effects due to ingestion of organic mercurials are neurological effects such as paresthesia, visual disturbances, mental disturbances, hallucinations, ataxia, hearing defects, and stupor (Clarkson et al., 1972).

Differences between the toxicity of different forms of mercury were demonstrated in a study by Aulerich et al.(1974) using mink (*Mustela vison*) fed either 5 ppm methylmercury or 10 ppm mercuric chloride. Mink treated with methylmercury died within 30 days, while those treated with mercuric chloride suffered no ill effects. Methylmercury attacks the central nervous systems in mammalian wildlife as well as in humans. The nervous system of the developing fetus may be particularly vulnerable (Bakir et al., 1973), and concern for these effects tends to drive human health risk assessments for mercury (Clarkson, 1990; reviewed in Volume V of this Report). Methylmercury ingestion can also cause reduced food intake, weight loss, muscular atrophy and damage to an animal's heart, lungs, liver, kidneys and stomach (Goyer, 1993; MDNR, 1993).

Levels of exposure that induce mercury poisoning in mammals vary among species. Death occurs in sensitive mammal species at 0.1-0.5 μg/g bw/d, or 1.0-5.0 μg/g in the diet. Smaller animals (e.g., minks and monkeys) are generally more susceptible to mercury poisoning than are larger animals (e.g., mule deer and harp seals), perhaps because of differences in elimination rates. Also, smaller mammals eat more per unit body weight than larger mammals and, thus, may be exposed to larger amounts of mercury on a body weight basis. LOAEL and NOAEL values for effects of methylmercury on mammalian wildlife are derived in Section 4 of this Volume.

2.3.3   Population Effects

Mercury contamination has been documented in endangered species, such as the Florida panther and the wood stork, as well as in populations of loons, eagles and furbearers such as mink and otters. These species experience high exposures because they either are piscivores or eat piscivores.

2.3.3.1 Loon Populations

It has been suggested by several researchers that loons are at risk from mercury contamination in aquatic food chains. Loons are primarily piscivorous but also consume benthic macroinvertebrates, such as crayfish (Barr, 1973). Mercury levels in crayfish approach and may even exceed those of forage fish from the same lakes (Barr, 1986; Allard and Stokes, 1989). Much of the loon's summer breeding range receives substantial mercury inputs from airborne deposition. In addition, many of these areas are known to be susceptible to acid deposition. As noted previously, a negative correlation often exists between lake water pH and mercury concentrations in fish.

A comprehensive study of mercury toxicity in wild loons was conducted by Barr (1986). Loons were collected from three habitats within the Wabigoon River watershed (Ontario, Canada) both above and below a chlor-alkali plant that discharged mercury into the river. The first habitat (designated C1) consisted of the lakes and river directly downstream of the plant. Habitat C2 did not receive mercury discharges but was accessible to mercury-contaminated fish that originated in C1. Habitat C3 was upstream from the chlor-alkali plant and received no appreciable mercury from other sources. Contaminated fish were prevented from entering C3 by a waterfall. A nearby habitat (C4), not connected to the other three habitats, received no mercury contamination and served as a control. Human disturbances in all habitats were determined to be minimal, and concentrations of organochlorine contaminants were low (less than 0.02 ppm total for all pesticides, including all DDT metabolites, and 0.04 ppm for PCBs).

Barr (1986) found a strong negative correlation between mercury levels in water and reproductive success in loons as far as 160 km downstream from the mercury source. Mercury in prey fish and invertebrates declined with increasing distance from the mercury source, but contaminated fish were able to migrate into the uncontaminated C2 habitat. Mercury levels in loon tissues (eggs, liver, muscle and brain in both adults and chicks) were highest in the C1 habitat but were also elevated in the C2 habitat, presumably because loons were feeding on contaminated prey which migrated from C1. Mercury levels in loons from habitat C3 (upstream from mercury source and inaccessible to contaminated fish) were comparable with levels from the uncontaminated control habitat, C4. Most of the mercury in loon tissues, with the exception of the liver, was in the form of methylmercury. Mercury in the liver appeared to be inorganic, suggesting the existence of a demethylation pathway. Dose-response relationships appeared to exist between mercury in prey and reproductive success as well as mercury in various tissues and reproductive success. For example, reductions in egg laying and in nest site and territorial fidelity were associated with prey containing mean mercury concentrations in the range of 0.3-0.4 μg/g. Reproductive success was also reduced in loons with brain or egg levels of 2-3 μg/g and in loons with liver residues above 13 μg/g. No loons reproduced successfully where prey species contained mercury at levels greater than 0.4 μg/g.

Ensor et al. (1992) captured 93 loons and collected 128 dead or dying loons from 18 northern and central counties in Minnesota. Feathers were collected from live loons. Feathers and liver tissue were collected from the dead loons. In 22 percent of the liver samples, mercury concentrations exceeded 13 μg/g, the level associated with impaired reproduction by Barr (1986). Adult loons contained greater concentrations of mercury than juvenile loons in feathers (8.7 vs. 2.7 μg/g wet weight) and in the liver (6.6 vs. 1.1 μg/g wet weight), as expected for a contaminant which bioaccumulates. The mercury in the juvenile loons was considered to be representative of local mercury contamination since all of their food would have been obtained from lakes within Minnesota. Mercury in adult loons was thought to represent contributions from both the summering grounds (Minnesota) and wintering grounds (Gulf of Mexico).

Ensor et al. (1992) concluded that juvenile loons that died of disease had significantly higher mercury levels in feathers than juvenile loons that died from injury or were caught alive. Emaciated loons also had significant (significance level not reported) elevations of mercury in both feathers and liver. It was not clear whether elevated mercury in emaciated loons resulted from concentration of existing mercury stores while body fat and protein were catabolized or whether mercury contributed to the emaciation. The authors concluded that the evidence of adverse impacts on the Minnesota loon population was sufficient to recommend monitoring mercury levels in loon populations.

Working in north central Wisconsin, Belant and Anderson (1990) collected both live and dead loons and addled eggs from abandoned nests. Residues of mercury and 14 organochlorine pesticides were measured in feathers (live and dead loons) and brain, muscle, and liver (dead loons). The conclusions reported in this study were similar to those reached by Ensor et al. (1992). Pesticide concentrations in dead loons were relatively low. In contrast, mercury levels in liver (mean concentration of 40.9 μg/kg wet weight) exceeded those associated with reproductive dysfunction as reported by Barr (1986).

Scheuhammer and Blancher (1994) reported on mercury levels in fish sampled from lakes throughout Ontario, Canada in areas without known point sources of mercury. Up to 30% of the lakes contained fish with mercury levels that exceeded 0.3 μg/kg (wet weight), the level associated with reproductive impairment in loons as reported by Barr (1986). The lack of any identified point source of mercury contamination was considered by the authors to be indirect evidence of airborne deposition of mercury over large portions of Ontario.

Preliminary results from an ongoing study of loons in northern Wisconsin were reported by Meyer et al. (1996). A significant negative correlation was found between mercury levels in blood from chicks and lake pH. Chick mortality was also greater on low pH lakes. It was not clear; however, whether these effects can be attributed to mercury or to a general reduction in the prey base of acidic lakes. Previously, it had been shown that mercury levels in blood and feathers of adult loons were negatively correlated with lake pH (Meyer et al., 1995).

The viability of loon populations within their traditional habitats in the United States is unclear. None of the studies reviewed was able to demonstrate clear population declines on a regional or national basis. Several studies have found that substantial numbers of loons contain mercury at or above levels associated with reduced reproductive success as reported by Barr (1986). It has also been suggested (but not clearly demonstrated) that sublethal effects of mercury exposure may produce greater susceptibility to environmental stresses, including other contaminants. Mercury also may make loons more susceptible to secondary infections, especially during stressful activities such as molting and migration. Investigations in response to a die-off of over 2,500 loons in the Gulf of Mexico in 1983 found that elevated levels of mercury were associated with abnormally high infestations of parasites (Barr, 1986).

2.3.3.2 Eagle Populations

Bald eagles are distributed throughout the United States. Many migrate into the lower forty-eight states only during the winter months; others are resident throughout the year. Bald eagles, like several other avian species, were adversely impacted by DDT and its metabolites during the 1950s, 60s, and 70s. Due to their status as a federally listed "threatened" species, the potential threat of mercury exposure to eagle survival and recovery is a concern.

Researchers have measured mercury residues in bald eagle feathers (U.S. FWS, 1993; Welch, 1994; Bowerman, 1994; Wood et al., 1996), eggs (Grier, 1974; Wiemeyer et al., 1984, 1993; Grubb et al., 1990; Anthony et al., 1993; Elliott et al., 1996) and blood (Anthony et al., 1993; U.S. FWS, 1993; Welch, 1994; Wood et al., 1996). Several of these studies have also reported on levels of other contaminants that might threaten eagle reproduction.

Wiemeyer et al. (1984) sampled bald eagle eggs that had failed to hatch from nests located in 14 states between 1969 and 1979; eggs were tested for organochlorine residues and mercury. The highest levels of mercury were detected in eggs from Maine. Eight organic contaminants were negatively correlated with eggshell

thinning, a trait often linked with reproductive failure in birds. When mercury levels were compared with the mean 5-year production rate for eagle nests, a weak negative correlation was found, suggesting an adverse effect of mercury. The analysis was confounded, however, by the co-occurrence of DDE in many of the eggs with the highest mercury levels. The authors concluded that mercury contamination appears to have the potential for adverse effects on eagle production in only a few of the breeding areas sampled, primarily in Maine.

Continuing the work begun earlier, Wiemeyer et al. (1993) collected eggs that had failed to hatch from nests in 15 states between 1980 and 1984 and analyzed them for organochlorine pesticides, polychlorinated biphenyls (PCBs) and mercury. These data were then combined with the data collected previously (Wiemeyer et al., 1984). As before, DDE was the contaminant most significantly (negatively) correlated with eggshell thinning, with DDD, DDT and PCBs significantly, but less strongly, correlated. The highest levels of DDE, PCBs and mercury occurred in eggs collected in Maine. Mercury levels in eagle eggs, at or above 0.28 μg/g (wet weight), were significantly correlated with a reduction in mean 5-year production rate for eagle nests. This value is comparable to the negative effect value of 0.5 μg/g derived earlier (Wiemeyer et al., 1984). The authors noted, however, that only three egg samples (all from Maine) contained mercury levels greater than 0.5 μg/g and that these eggs also contained levels of DDE known to reduce eagle productivity (>6 μg/g). Wiemeyer et al. (1993) concluded that recent data provide even less evidence than previously indicated (Wiemeyer et al., 1984) that contaminants other that DDE are adversely impacting bald eagle productivity. Grubb et al. (1990), Grier (1974), and Anthony et al. (1993) reached similar conclusions on the lack of evidence for an association between mercury levels and reproductive failure in bald eagles.

Bowerman and co-workers (Bowerman, 1993; Bowerman et al. 1994) examined the productivity of bald eagles in six geographic regions, including Lakes Superior, Michigan, Huron, and Erie and the states of Michigan and Minnesota. Significant negative correlations existed between plasma levels of PCB and p,p'-DDE and reproductive success. Mercury levels in feathers ranged from 9.0 to 23.4 μg/g but were not correlated with reproductive success.

Welch (1994) sampled eggs, blood and feathers from Maine bald eagles and analyzed them for organochlorine pesticides, PCBs, TCDD equivalents (TCDD-eq), and mercury. Mercury levels in inland eagles were higher than concentrations in eagles inhabiting the coastline. In general, these elevated mercury levels appeared to be related to mercury residues in fish from the two areas. Productivity was also lower for inland eagle nests; however, the correlation of mercury levels in blood and feathers with mean productivity (5 and 15 years) was not significant.

Mercury concentrations in eagle eggs from British Columbia approached and in some instances exceeded the level (0.28 μg/g) associated with long-term declines in eagle populations as reported by Wiemeyer et al. (1993). However, populations in this region appeared at the time of the study to be increasing. Mercury residues in feathers, blood and livers from eagles in central Florida were lower than those determined for most other wild eagle populations (Wood et al., 1996).

One of the difficulties in evaluating the effect of mercury on the bald eagle is the co-occurrence of organochlorine compounds such as PCBs, DDE and TCDDs at levels that may have adverse effects on reproduction. Bowerman (1993) hypothesized that the effect of the organochlorine contaminants may be masking the effect of mercury. The U.S. Fish and Wildlife Service (1993) also suggested that, while mercury was not found in Florida bald eagles at lethal levels, sublethal levels may be adversely affecting eagle reproduction. Historical data suggest that eagle populations in the Great Lakes Basin are still well below the

region's carrying capacity. In contrast, eagle populations on many inland waters appear to be doing well (Colborn, 1991; Bowerman, 1993; Bowerman et al., 1994).

2.3.3.3 Wood Stork Populations

Mercury has been detected in feathers of the endangered wood stork, although the levels found apparently have not caused toxic effects. Young wood storks in Florida had mercury levels of 1.87 μg/g dry weight; higher mercury levels would be expected for adults from the same area (Burger et al., 1993). Fleming et al. (1984) reported mercury levels of 0.66 μg/g wet weight in wood stork eggs, which is somewhat less than Eisler's (1987) recommended criterion of <0.90-2.0 μg/g wet weight in eggs.

2.3.3.4 Other Wading Birds

The wading bird population in Florida has declined substantially since the 1940's (Ogden, 1994). While a variety of factors have been implicated, cause-and-effect relationships remain difficult to establish. The possible effect of mercury on wading birds was investigated by Spalding et al. (1994) and Sundlof et al. (1994). In general, there is a positive relationship between mercury residues in wading birds and the trophic level at which they feed (Sundlof et al., 1994). Mercury levels in livers of birds that feed on fish (e.g., Great Blue Heron, Great White Heron, and Great Egret) exceeded, in several instances, those associated by other authors with neurologic signs in birds (30 μg/g wet weight) (Scheuhammer, 1991).

2.3.3.5 Furbearer Populations

In one Ontario incident, an eagle was found scavenging on a mercury-poisoned dead otter at Clay Lake (Wren, 1985). Mercury levels in the otter (liver - 96 μg/g; kidneys - 58 μg/g; brain - 30 μg/g) were well above those known to be toxic to otters in laboratory exposures. The primary source of the mercury was a chlor-alkali plant that discharged mercury directly into the river.

In a separate incident, a mink exhibiting unnatural behavior was collected near the mercury-contaminated Saskatchewan River (Wobeser and Swift, 1976). Subsequent determination of mercury levels in the liver (58 μg/g), kidney (32.9 μg/g), muscle (15.2 μg/g), brain (13.4 μg/g) and fur (34.9 μg/g), combined with clinical and pathologic findings, were deemed sufficient by the authors to conclude that the animal had been poisoned by mercury. Residue levels found in this animal exceeded those determined in laboratory studies to be associated with toxicity. The origins of mercury in this case could not be determined; however, it was observed that fish from the Saskatchewan River contain mercury at concentrations higher than those known to cause toxicity to mink in laboratory studies.

In a study of furbearers obtained from trappers in the Wisconsin River watershed (1972-1975), otters contained the highest tissue mercury levels, followed by minks, raccoons, foxes, muskrats and beavers (Sheffy and St. Amant, 1982). Liver mercury concentrations reported by Halbrook et al. (1994) for otters collected from the coastal plain of Georgia (5.1-9.2 μg/g) were approximately one-third the levels reported for otters and mink that died in experimental dosing studies (Aulerich et al., 1974; Wobeser et al., 1976; O'Conner and Nielson, 1981), and it was speculated by these authors that sublethal behavioral and reproductive impacts could result in population level effects.

Mink populations, like those of the otter, have declined substantially in the Southeastern coastal states, particularly in the coastal plain. Mercury concentrations in mink from the coastal plain were found to be higher than those in mink from inland areas, and were in the range (3.5 μg/g in kidney) of those known to be associated with reproductive and behavioral effects in laboratory studies (Osowski et al., 1995). Liver PCB levels were also found to be significantly elevated. In this regard, it is of interest to note studies with mink which suggest that mercury and PCBs can act synergistically to reduce reproductive success (Wren et al., 1987). Giesy et al. (1994) determined that PCBs and mercury do not pose a threat to mink on three Michigan rivers. As with most assessments of this type, however, combined impacts were not considered.

2.3.3.6 Florida Panther Populations

Mercury is suspected of contributing to the death of one and possibly more endangered Florida panthers. The Florida Panther Interagency Committee (FPIC) reported that approximately 100 ppm of mercury was detected in the liver and 130 ppm in the hair of a 4-year-old female panther (FPIC, 1989). The panther, No. 27, had been radio-instrumented since 1988 and was found dead in the eastern part of the Florida Everglades National Park (FPIC, 1989). Relatively high levels of mercury (0.005-20.0 ppm) were detected in archived liver samples from six dead panthers, and levels ranging from 0.02-130.0 ppm have been measured in the hair samples from ten live individuals. The FPIC concluded that panther No. 27 died of mercury poisoning; however, the cause of death of the six archived animals was not mentioned in their report.

The most probable source of mercury contamination in Florida panthers is via the food chain. The diet of the Florida panther includes both raccoons and white-tailed deer but varies greatly depending on prey availability. Mercury contamination in raccoons has been found to occur in a distributional pattern that coincides with the species range of Florida panthers (Roelke et al., 1991). The accumulation of mercury in raccoons is due to consumption of contaminated aquatic life, including invertebrates, fish and amphibians. The panthers most at risk, therefore, appear to be those that consume mercury-contaminated raccoons. Panthers that prey on deer are less exposed to mercury because deer are herbivores and accumulate less mercury. Based upon the findings of the FPIC, habitat modifications have been implemented in the Florida Everglades to increase local deer herds.

In addition to mortality, mercury contamination could decrease reproductive success in the Florida panther. Methylmercury ingested by a pregnant mammal passes through the placenta to the developing fetus, potentially causing abortions, stillbirths, congenital defects and behavioral modifications that result in the death of neonates. Roelke et al. (1991) found a significant inverse correlation between mercury concentrations in mother panthers and survivorship of the young. Because so few Florida panthers remain (only 30 to 50 in the wild) (Jordan, 1990), the possibility exists that mercury contamination could contribute to the extinction of this endangered species (Roelke et al., 1991). However, mercury is but one of several stressors that may be affecting the panther. Habitat fragmentation, inbreeding (Roelke et al., 1993), and feminization by endocrine disrupting compounds (Facemire et al., 1995) have all been implicated as causative factors in the decline of this species.

2.3.4   Communities and Ecosystems

2.3.4.1 Aquatic Communities and Ecosystems

Effects of contaminants on aquatic communities have been investigated by examining functional and structural responses of natural assemblages in laboratory settings to toxic substances added singly or in combination. The species diversity of freshwater and brackish-water microbial communities was reduced by exposure to 40 μg/L of mercuric chloride (Singleton and Guthrie, 1977). Carbon fixation was reduced by 50 percent in freshwater phytoplankton communities exposed to 0.4 μg/L of mercuric chloride, but this effect was mitigated by the presence of humus or sediment (Hongve et al., 1980). Mercuric chloride (0.5 μg/L) administered to a marine aquatic community inhibited phytoplankton growth, killed or retarded development in copepods, and increased the number of viable bacteria (Kuiper, 1981). The species composition of the phytoplankton also changed, possibly due to selective reduction of predation by the copepods. Bacterial populations may have increased due to increased food supply in the form of dead phytoplankton (Kuiper, 1981).

In general, mercury concentrations (as Hg^{+2}) required to elicit toxic effects on natural aquatic communities exceed those commonly measured in surface waters by two or more orders of magnitude (low ng/L in waters not impacted by point source discharge) (Spry and Wiener, 1991; Wiener and Spry, 1995). Studies of the effects of methylmercury on aquatic assemblages were not found, however, and it can be reasonably anticipated that the toxicity of methylmercury to these communities would exceed that of mercuric chloride. Effects of mercury or any other substance at this level of biological organization could potentially have far-reaching impacts on the entire food chain by changing both nutrient and energy fluxes.

Field studies of mercury occurrence and effects at the community level are not available. Moreover, interpreting field studies can be difficult because more than one stressor is often present. Elevated concentrations of mercury have been found in several species of piscivorous wildlife that have experienced reproductive failure in the Great Lakes region (e.g., Caspian terns, herring gulls, double-crested cormorants, and mink) (Peakall, 1988; Colborn, 1991; Environment Canada, 1991; Gilbertson et al., 1991). However, other bioaccumulative contaminants, such as PCBs, dioxins and DDT/DDE, have been implicated as the most likely causative agents (Colborn, 1991; Gilbertson et al., 1991).

2.3.4.2 Terrestrial Communities and Ecosystems

As noted previously, atmospherically deposited heavy metals such as mercury tend to accumulate in top soils. This results in particularly high exposures in decomposer communities, which play a crucial role within the natural nutrient cycles of terrestrial ecosystems. Mercury forms stable complexes with organic substances of high molecular weight (humic acids) and exhibits limited mobility within soils. Processes that may be affected by heavy metals in top soil include litter decomposition, carbon mineralization, nitrogen transformation and enzyme activity. Mercury effects on soil microorganisms vary depending on soil type (Zelles et al., 1986). Mercury generally inhibits heat production, respiration and iron reduction by soil microorganisms in sandy soils and, to a lesser extent, in clay. At some intermediate concentrations, however, mercury may stimulate microbial activity in peat (Zelles et al., 1986).

It is difficult to estimate specific toxic levels for microbial-mediated processes in decomposer communities due to widely differing soil properties and methodological discrepancies in the literature. In a report on mercury in the Swedish environment, Lindqvist (1991) cites a study in which soil microbial activity was significantly reduced at mercury concentrations ranging from 0.06-0.08 μg/g dry weight of humus. The

concentration of mercury in forest soils in Sweden is in the range 0.01-0.09 μg/g. In a second cited study, however, the mercury concentration in soil required to reduce soil microbial activity was 50 μg/g. A common effect of metal contamination on soil animal groups is a decrease in species diversity. In some species, susceptibility to metals may be a secondary effect due to differences in food availability rather than metal toxicity per se.

2.3.5   Conclusions

Of the pathways by which ecosystems and components of ecosystems might be exposed to atmospheric mercury, exposure of high trophic level wildlife to mercury in food is particularly important. The trophic level and feeding habits of an animal influence the degree to which it is exposed to mercury. Mercury biomagnifies in aquatic food chains resulting in increasing tissue concentrations of mercury as trophic level increases. Predatory animals primarily associated with aquatic food chains accumulate more mercury than those associated with terrestrial food chains. Thus, piscivores and other carnivores that prey on piscivores generally have the highest exposure to mercury. In a study of furbearing mammals in Wisconsin, the species with the highest tissue levels of mercury were otter and mink, which are top mammalian predators on aquatic food chains (Sheffy and St. Amant, 1982). Top avian predators of aquatic-based food chains include raptors, such as the osprey and bald eagle. Smaller birds feeding at lower levels in aquatic food chains also may be exposed to substantial amounts of mercury due to their high food consumption rate (consumption/kg bw/d) relative to larger birds.

Although clear causal links have not been established, mercury originating from airborne deposition may be a contributing factor to population effects on several wading birds, loons, river otters, mink, and the Florida panther. Effects of mercury originating from point sources on restricted wildlife populations have been conclusively demonstrated and provide a tissue residue basis for evaluation of risk to other populations. Based upon reviews of both laboratory and field data, mercury levels that exceed the following values (in μg/g fresh weight) have been suggested as evidence for possible adverse impacts on avian populations: feathers - 20 μg/g (Scheuhammer, 1991); eggs - 2.0 μg/g (after conversion from dry weight) (Scheuhammer, 1991); liver - 5 μg/g (Zillioux et al., 1993). Such criteria must be used with caution, however, as residue thresholds both above and below these values have been reported. Field data for mammals are not as extensive as those for birds. Mercury residues in mink and otter that were thought to have been poisoned by mercury originating from a point source exceeded those seen in dead laboratory animals by a factor of two or more (see Section 2.3.2.4) (Wren, 1991). The reason for this variation is presently unknown. Additional information is needed before tissue-residue-based criteria for piscivorous mammals can be developed. Criterion values for fish and water that are designed to be protective of piscivorous wildlife are calculated in Section 5 of this Volume.

2.4   Ecosystems Potentially at Risk

The information presented in Sections 2.1 through 2.3 suggests that the ecosystems most at risk from mercury releases to air exhibit one or more of the following characteristics:

- they are located in areas that experience high levels of atmospheric deposition;

- they include surface waters already impacted by acid deposition;

- they possess characteristics other than low pH that result in high levels of mercury bioaccumulation in aquatic biota;

- they include species that experience high levels of exposure (e.g., piscivorous birds and mammals).

2.4.1   Highly Exposed Areas

Ecosystems subjected to high levels of mercury deposition (e.g., near sources of mercury emissions or in areas with high deposition rates) will be more exposed to mercury than ecosystems with lower levels of mercury deposition. The pattern of mercury deposition nationwide, therefore, will influence which ecoregions and ecosystems might be exposed to hazardous levels of mercury.

2.4.2   Lakes and Streams Impacted by Acid Deposition

In many aquatic systems, the tendency for mercury to bioaccumulate in fish is inversely correlated with pH and alkalinity (or acid neutralizing capacity) (reviewed by Spry and Wiener, 1991). Thus, fish in acidic lakes (pH 6.0 to 6.5 or less) often have higher body or tissue burdens of mercury than fish in nearby lakes with higher pH. This relationship has been found for a variety of fish species and water bodies, including the following:

- various fish species in 14 lakes and 31 streams in Florida (FDER, 1990);

- yellow perch from lakes in the Upper Michigan peninsula (Grieb et al., 1990);

- yellow perch from seepage lakes in Northern Wisconsin (Cope et al., 1990);

- yellow perch from an experimentally acidified lake in Northern Wisconsin (Wiener et al., 1990);

- yellow perch from Southern Ontario lakes (Suns and Hitchin, 1990);

- yellow perch from 12 Adirondack lakes (Simonin et al., 1994);

- walleyes from Wisconsin lakes (Lathrop et al., 1991);

- largemouth bass from 53 lakes in Florida (Lange et al., 1993);

- northern pike from 80 Minnesota lakes (Sorensen et al., 1990); and

- smallmouth bass from Ontario lakes (McMurtry et al., 1989).

The increased accumulation of mercury in low pH lakes appears to be due largely to increased microbial production of methylmercury (Xun et al, 1987; Bloom et al., 1991; Miskimmin et al., 1992), although biogeochemical processes that release mercury from sediments have also been implicated (Rada et al., 1993). The bioavailability of methylmercury is probably also enhanced by decreased levels of calcium, as is typical of such lakes. There are, however, exceptions to the general relationship between pH and bioaccumulation of mercury (Fjeld and Rognerud, 1993), and it has been suggested that clear correlations between pH and mercury bioaccumulation are likely to occur only when mercury deposits onto seepage lakes (Richardson et al., 1995).

2.4.3   Dissolved Organic Carbon

DOC appears to be an important determinant of mercury translocation from watersheds to waterbodies and, in many systems, may be a better predictor of fish mercury residues than pH (McMurtry et al., 1989; Nilsson and Hakanson, 1992; Fjeld and Rognerud, 1993; Driscoll et al., 1994,1995; Watras et al., 1995b,c). However, high concentrations of DOC may also complex methylmercury, diminishing its bioavailability (Driscoll et al., 1994,1995; Hintelmann et al., 1995). Methylmercury uptake across the gills of the Sacramento blackfish was measured directly by Choi et al. (1997). The addition of moderate amounts of DOC to the exposure water dramatically reduced this uptake. DOC has been shown to reduce the bioavailability of neutral organic compounds to freshwater invertebrates (Landrum et al., 1985). Studies of this type have not yet been conducted with mercury.

2.4.4   Factors in Addition to pH and DOC that Contribute to Increased Bioaccumulation of Mercury in Aquatic Biota

Numerous factors in addition to pH and DOC can influence the bioaccumulation of mercury in aquatic biota. These include the length of the aquatic food chain (Cabana and Rasmussen, 1994; Cabana et al., 1994; Futter, 1994) and water temperature (Bodaly et al., 1993). Physical and chemical characteristics of a watershed affect the amount of mercury that is translocated from soils to water bodies (McMurtry et al., 1989, Johnston et al., 1991; St. Louis et al., 1994; Joslin, 1994; Hurley et al., 1995). Interrelationships between these factors are poorly understood, however, and there is no single factor that has been correlated with mercury bioaccumulation in all cases examined.

2.4.5   Sensitive Species

For the purposes of this discussion, sensitive species are defined as those species that are more likely than others to experience adverse effects due to mercury contamination. Such species may or may not be inherently more sensitive on an absorbed dose basis. Sensitive species also may be at risk because they receive high methylmercury exposures due to their position in the food chain or because their populations are already stressed. In the first category are top-level predators of aquatic-based food webs exposed to high concentrations of mercury in their prey. Examples include piscivorous raptors (e.g., bald eagles and ospreys), waterbirds (e.g., herons, gulls, kingfishers, and cormorants), and mammals (e.g., mink and otter). The second category includes threatened and endangered species, which are species that have already experienced severe population declines and are at risk of further population declines or extinction (e.g., Florida panther).

2.5   Endpoint Selection

U.S. EPA distinguishes two types of endpoints for ecological risk assessment purposes: assessment endpoints and measurement endpoints (see text box). Assessment endpoints are explicit expressions of the actual environmental value that is to be protected. Often, the assessment endpoint cannot be measured directly, so a risk assessor selects one or more measurement endpoints that can be related, either quantitatively or qualitatively, to the assessment endpoint (U.S. EPA, 1992a). In its draft guidance on risk assessment procedures, U.S. EPA (1996) suggested that the term "measurement endpoint" be replaced by the term "measure of effect." It was deemed prudent for this Report, however, to utilize established terminology until the draft guidelines are finalized.

A goal of the problem formulation phase in an assessment is to select assessment endpoints that are relevant to decisions to be made. Factors relevant to the selection of these endpoints include: (1) ecological relevance; (2) susceptibility to known or potential stressors; and (3) representation of management goals (U.S. EPA, 1992a, 1996).

> **Endpoints for Ecological Risk Assessment**
>
> **Assessment endpoint** - an explicit expression of the environmental value that is to be protected (U.S. EPA, 1992a).
>
> **Measurement endpoint** - a measurable ecological characteristic that is related to the valued characteristic chosen as the assessment endpoint. (U.S. EPA, 1992a).

Table 2-8 provides examples of ecological assessment and measurement endpoints at various levels of biological organization. In current practice, the most tractable endpoints are at the individual or population level and include mortality, growth, development and reproduction.

Based on the information provided in Sections 2.1 through 2.4, the ecological components that appear to be most at risk from atmospheric mercury are piscivorous mammals and birds that feed at or near the top of aquatic food chains. This is particularly true of threatened or endangered species that already have suffered population declines due to one or more causes. An appropriate assessment endpoint, therefore, would be maintenance of self-sustaining populations of these species. Appropriate measurement endpoints for exposed wildlife species would include growth and survival of adults or other life-stages, reproductive success, and behavioral impacts. Alternatively, when such data are difficult to collect, it may be necessary to infer adverse effects on wildlife from laboratory toxicity studies.

2.6   Conceptual Model for Mercury Fate and Effects in the Environment

An important product of the problem formulation phase in ecological risk assessment is a conceptual model of how the stressor may affect ecological components of the natural environment (U.S. EPA, 1992a,1996). The conceptual model identifies the ecosystem(s) potentially at risk, exposure pathways between sources and receptors, and the relationship(s) between measurement and assessment endpoints. A preliminary analysis of the ecosystem, stressor characteristics, and ecological effects helps to define possible exposure scenarios (i.e., qualitative descriptions of how the stressors co-occur with or contact the various ecological components).

A conceptual model of the ecological effects of airborne mercury emissions can be visualized using Figures 2-1 through 2-5. Mercury is emitted to the atmosphere primarily as the elemental form or as an inorganic ion. Inorganic mercury returns to earth in wet deposition due to its relatively high solubility in water and because it adsorbs to airborne particulates. Elemental mercury has a long half-life in the atmosphere and tends to stay aloft but may react with other chemicals to form inorganic mercury species. Wet deposition containing mercury falls onto watersheds or directly on water bodies. Mercury deposited onto watersheds is rapidly bound to organic matter and tends to accumulate over time. A portion of this mercury is released, however, and is transported in runoff and groundwater to receiving waters such as lakes, streams and wetlands. Biotic and abiotic chemical reactions transform mercury in water and associated sediments to organic derivatives (primarily methylmercury). Organomercurial compounds then accumulate in aquatic food chains due both to their tendency to become sequestered in tissues and to the efficiency with which they are transferred from one trophic level to another. Eventually, mercury in fish is consumed by piscivorous wildlife, with the resulting potential for adverse toxicological effects. Uptake

Table 2-8
Examples of Assessment and Measurement Endpoints

Level of Organization	Assessment Endpoints	Measurement Endpoints
Ecoregion[a]	Biodiversity Regional production Landscape aesthetics	Habitat area Regional production Other landscape descriptors
Ecosystem	Productive capability Nutrient balance Soil balance	Habitat area Biomass Productivity Nutrient export
Community	Recreational quality Change to less useful/desired type Market/sport value	Species number Species evenness Species diversity Market/sport value Saprobic index
Population	Extinction Abundance Yield/production Frequent gross morbidity Massive mortality Range	Occurrence Numbers/density Age structure Fecundity Yield/production Frequency of gross morbidity Mortality rate
Individual	Survival Growth and development Reproduction Good physical condition	Longevity Growth and development Fecundity Overt symptomology Biomarkers
Abiotic	Habitat quality	Temperature Water flow Soil characteristics Sediment characteristics

[a] An ecoregion is an area (region) of relative homogeneity in ecological systems (based on elevation, soils, latitude, precipitation).

Source: Adapted from U.S. EPA, 1989.

pathways other than consumption of contaminated prey (e.g., inhalation and drinking of contaminated water) are considered to be of little consequence for piscivorous birds and mammals.

2.7   Analysis Plan

The final goal of the problem formulation phase of an assessment is to develop a plan for subsequent analyses of exposure and effect (U.S. EPA, 1996). In Chapter 3 of this Volume, an attempt is made to

characterize the exposure of piscivorous avian and mammalian wildlife to airborne mercury and to link these exposures with information pertaining to specific emissions categories. A stepwise approach was taken, with each step representing an increased level of complexity and uncertainty. Field residue data were used to the maximum extent possible for characterization of mercury bioaccumulation and biomagnification in fish. These data are believed to be better suited for this purpose than laboratory bioconcentration and bioaccumulation data. Using a previously derived "national average" mercury concentration in fish, exposures to selected wildlife species were estimated using published exposure factors. Air dispersion models were employed in this analysis, progressing from the use of a long-range transport model to estimate mercury deposition on a regional basis to the combined use of both local-scale and long-range models. Mercury deposition estimates on a regional scale were compared with the distributions of sensitive wildlife species. Finally, an effort was made to determine whether wildlife living in close proximity to a mercury emissions source experience particularly high exposures leading potentially to adverse impacts within relatively small geographical regions.

An effects assessment is conducted in Chapter 4 of this Volume by reviewing pertinent toxicology testing data, with priority given to long-term dietary exposures with wildlife species. A review of data on mercury elimination suggested the need to evaluate species differences in mercury toxicokinetics and the ameliorative effects of selenium supplementation. The primary goals of this assessment were: (1) to estimate toxic dose levels for piscivorous wildlife and (2) to provide guidance on the rational use of uncertainty factors for subsequent analyses of risk and the development of protective exposure criteria.

3. EXPOSURE OF PISCIVOROUS AVIAN AND MAMMALIAN WILDLIFE TO AIRBORNE MERCURY

3.1 Objectives and Approach

The objective of this analysis was to characterize the extent to which piscivorous wildlife are exposed to mercury originating from airborne emissions. Three general approaches were used, which may be described as follows.

1. *Estimation of current average exposure to piscivorous wildlife on a nationwide basis (Section 3.2).*

Estimates of current mercury exposure to selected piscivorous wildlife species were calculated as the product of the fish consumption rate and measured mercury concentrations in fish. This was not intended to be a site-specific analysis, but was instead intended to provide national exposure estimates for piscivorous wildlife based on typical mercury concentrations in fish. This analysis utilized mean total mercury measurements from two nationwide studies of fish residues and published fish consumption data for the selected wildlife species.

2. *Estimation of mercury deposition on a regional scale (40 km grid) and comparison of these data with species distribution information (Section 3.3).*

A long-range atmospheric transport model (RELMAP) was used in conjunction with a mercury emissions inventory to generate predictions of mercury deposition across the continental U.S. This information was then compared with wildlife species distributions to characterize the potential for co-occurrence of high mercury deposition rates and the presence of wildlife species of concern.

3. *Estimation of mercury deposition on a local scale in areas near emissions point sources (Section 3.4).*

A local-scale atmospheric transport model (GAS-ISC3) was used to simulate mercury deposition originating from four different mercury emissions source classes. The analysis was conducted for two hypothetical lakes located in the western and eastern U.S. The proximity of these lakes to the source was varied to examine the effect of this parameter on model predictions. To account for the long-range transport of emitted mercury, the 50th percentile RELMAP atmospheric concentrations and deposition rates were included in the estimates from the local air dispersion model. To account for other sources of mercury, estimates of background concentrations of mercury were also included in this exposure assessment.

3.2 Description of Computer Models

The models used for the wildlife exposure assessment are identical to those used for the human exposure assessment (see Volume IV of this Report) and are described in detail in Volume III of this Report. Atmospheric transport models were used to simulate the deposition of mercury at two different geographical scales (see Table 3-1). A regional-scale analysis was conducted using the Regional Lagrangian Model of Air Pollution (RELMAP). RELMAP calculates annual mean air concentrations and annual mean deposition rates for each cell in a 40 km grid. This analysis covered the 48 contiguous states and was based upon a recent inventory of mercury emissions sources (see Volume II of this Report).

3-1

Table 3-1
Models Used to Predict Mercury Air Concentrations,
Deposition Fluxes and Environmental Concentrations

Model	Description
RELMAP	Predicts average annual atmospheric mercury concentration and wet and dry deposition flux for each 40 km^2 grid in the U.S. due to all anthropocentric sources of mercury in the U.S. and a natural background atmospheric mercury concentration.
GAS-ISC3	Predicts average concentration and deposition fluxes within 50 km of emission source.
IEM-2M	Predicts environmental concentrations based on air concentrations and deposition rates to watershed and water body.

The local-scale exposure analysis was conducted using both RELMAP and a local air transport model, GAS-ISC3, to generate hypothetical exposure scenarios for four mercury emission source classes. GAS-ISC3 uses hourly meteorological data to estimate hourly air concentrations and deposition fluxes within 50 km of a point source. For each hour, general plume characteristics are estimated based on the source parameters (gas exit velocity, temperature, stack diameter, stack height, wind speed at stack top, and atmospheric stability conditions) for that hour. GAS-ISC3 was run using one year of actual meteorological data (1989, the same meteorologic year as was utilized in the RELMAP modeling). The average annual predicted values for air concentration and deposition rates were then used as inputs to the IEM-2M model. Finally, the IEM-2M model was used to simulate the result of deposition over a 30 year period, which is the assumed typical lifetime of a facility.

The IEM-2M model was used to translate both regional and local-scale mercury deposition estimates into mercury levels in soil, water and biota. Mercury levels in fish were calculated from average water concentrations using estimated BAFs for fish occupying trophic levels 3 and 4. It was assumed throughout the wildlife exposure analysis that 100% of mercury contained in fish exists as methylmercury.

IEM-2M is composed of two integrated modules that simulate mercury fate using mass balance equations describing watershed soils and a shallow lake. IEM-2M simulates three chemical components -- elemental mercury (Hg^0), divalent mercury (Hg^{2+}), and methylmercury (MHg). The mass balances are performed for each mercury component, with internal transformation rates linking Hg^0, Hg^{2+}, and MHg. Sources include wetfall and dryfall loadings of each component to watershed soils and to the water body. An additional source is diffusion of atmospheric Hg^0 vapor to watershed soils and the water body. Sinks include leaching of each component from watershed soils, burial of each component in lake sediments, volatilization of Hg^0 and MHg from the soil and water column, and advection of each component out of the lake.

At the core of IEM-2M are nine differential equations describing the mass balance of each mercury component in the surficial soil layer, in the water column, and in the surficial benthic sediments. The equations are solved for a specified interval of time, and predicted concentrations output at fixed intervals. For each calculational time step, IEM-2M first performs a terrestrial mass balance to obtain mercury concentrations in watershed soils. Soil concentrations are used along with vapor concentrations and deposition rates to calculate concentrations in various food plants. These are used, in turn, to calculate concentrations in animals. IEM-2M simultaneously performs an aquatic mass balance driven by direct atmospheric deposition along with runoff and

erosion loads from watershed soils. MHg concentrations in fish are derived from dissolved MHg water concentrations using bioaccumulation factors (BAFs).

Mercury residues in fish were estimated by making the simplifying assumption that aquatic food chains can be adequately represented using four trophic levels. Respectively, these trophic levels are the following: level 1 - phytoplankton (algal producers); level 2 - zooplankton (primary herbivorous consumers); level 3 - small forage fish (secondary consumers); and level 4 - larger, piscivorous fish (tertiary consumers). This type of food chain typifies the pelagic assemblages found in large freshwater lakes and has been used extensively to model bioaccumulation of hydrophobic organic compounds (see for example Thomann, 1989; Clark, 1990; and Gobas, 1993). It is recognized, however, that food chain structure can vary considerably among aquatic systems resulting in large differences in bioaccumulation in a given species of fish (Futter, 1994; Cabana et al., 1994a,b). In addition, this simplified structure ignores several important groupings of organisms, including benthic detritivores, macroinvertebrates, and herbivorous fishes. The second simplifying assumption utilized in this effort was that methylmercury concentrations in fish are directly proportional to dissolved methylmercury concentrations in the water column. It is recognized that this relationship can vary widely among both physically similar and dissimilar water bodies.

Methylmercury concentrations in fish were derived from predicted water column concentrations of dissolved methylmercury by using BAFs for trophic levels 3 and 4 (see Table 3-2). The BAFs selected for these calculations were estimated from existing field data. Respectively, these BAFs (dissolved methylmercury basis) are 6.8×10^6 and 1.6×10^6. Methylmercury was estimated to constitute 7.8% of the total dissolved mercury in the water column. The technical basis for these estimates is presented in Volume III, Appendix D.

The variability around these predicted fish residue values is highlighted in Table 3-2. Percentile information for the BAF estimates developed in Appendix D of Volume III are presented. This table demonstrates the large variability in fish residues that may occur at a given methylmercury water concentration. This variability is largely due to the variability in field-derived BAF values.

Table 3-2
Percentiles of the Methylmercury Bioaccumulation Factor

Parameter	Percentile of Distribution				
	5th	25th	50th	75th	95th
Trophic 3 BAF	4.6×10^5	9.5×10^5	1.6×10^6	2.6×10^6	5.4×10^6
Trophic 4 BAF	3.3×10^6	5.0×10^6	6.8×10^6	9.2×10^6	1.4×10^7

3.3   Current Exposure of Piscivorous Wildlife to Mercury

Four avian species (eagle, common loon, kingfisher and osprey) and two mammalian species (otter and mink) were assumed to be exposed to methylmercury through the ingestion of contaminated fish. Fish consumption is thought to be the dominant mercury exposure pathway for piscivores (see Chapter 2 of this Volume). Consequently, an analysis of these ecological receptors' methylmercury contact rate based on the daily ingestion rate of fish is reasonable and appropriate.

The piscivorous bird's or mammal's methylmercury contact rate from fish consumption can be estimated as the product of methylmercury levels in the fish and the daily amount of fish eaten. The trophic level at which piscivores feed significantly impacts their exposure to methylmercury. Those piscivores consuming a diet primarily consisting of trophic level 3 fish are expected to ingest approximately five times less methylmercury per gram of fish eaten than those eating trophic level 4 fish from the same site. Animals consuming a mixture of trophic level 3 and 4 fish would experience (on a per gram of fish basis) an intermediate level of exposure. Finally, many top level predators consume a mixture of both aquatic and terrestrially-derived prey. In general, mercury levels in the tissues of terrestrial animals are much lower than those of fish. A special case exists, however, when a terrestrial animal (e.g., a raccoon) feeds on aquatic biota and is itself preyed upon by a larger terrestrial animal (e.g., the Florida panther). A similar situation exists when a piscivorous bird (e.g., the herring gull) is consumed by a larger bird (e.g., the bald eagle). In these situations, the potential exists for the top predator to obtain a higher mercury dose than it would otherwise receive from a strictly fish-based diet. The extent of this increase depends, in turn, upon the proportion of the diet composed of these mammalian and avian prey items and the extent to which the prey items accumulate mercury in excess of levels found at trophic levels 3 and 4.

Exposure factors for the present analysis were obtained from two recent compilations of wildlife dietary habits (U.S. EPA, 1993a, 1995a) and are shown in Table 3-3. Bald eagles were assumed to eat fish derived from trophic levels 3 and 4, as well as prey derived from other sources. Expressed as percentages, these prey items were assumed to contribute 74, 18 and 8% of the daily dietary intake. For this Report, dietary items other than fish were assumed to contain no mercury. Eagles are, therefore, expected to experience a greater methylmercury exposure per gram of fish consumed than ospreys, loons, and kingfishers, which were assumed to consume only trophic level 3 fish. Part of this increase, however, is offset by the contribution of uncontaminated prey consumed by eagles. Among the mammals, otters, which were assumed to consume an 80/20 mix of trophic level 3 and 4 fish, are expected to have a greater methylmercury exposure per gram of fish consumed than mink, which were assumed to eat only trophic level 3 fish. In addition, 10% of the mink diet was assumed to consist of uncontaminated prey items.

Table 3-3
Exposure Parameters for Mink, Otter, Kingfisher, Loon, Osprey, and Eagle

Species	Body Wt. (Wt_A) kg	Ingestion Rate (F_A) kg/d	Drinking Rate (W_A) L/d	Trophic Level of Wildlife Food Source	% Diet at Each Trophic Level
Mink	0.80	0.178	0.081	3	90
Otter	7.40	1.220	0.600	3,4	80,20
Kingfisher	0.15	0.075	0.017	3	100
Loon	4.0	0.8	0.14	3	100
Osprey	1.50	0.300	0.077	3	100
Eagle	4.60	0.500	0.160	3,4	74,18,8

The ratio of grams fish consumed per day to piscivore body weight is also significant in estimating mercury exposure on a µg/kg bw/d basis. The greater this ratio, the higher the resulting mercury exposure, assuming that methylmercury concentrations in fish remain constant. For example, osprey, loons, and kingfishers each consume trophic level 3 fish only. Kingfishers consume an amount of fish equivalent to about 50% of their body weight each day, while osprey and loons consume roughly 20% of their body weights in fish per day. The resulting average daily intake of methylmercury in μg/kg body weight will, therefore, be higher in kingfishers. Residue data used to calculate national averages for mercury concentration in fish were obtained from two studies. The first, entitled "A National Study of Chemical Residues in Fish," was conducted by U.S. EPA (1992b) and also reported in Bahnick et al. (1994). The second study, entitled "National Contaminant Biomonitoring Program: Concentrations of Seven Elements in Freshwater Fish, 1978-1981," was published by Lowe et al. (1985). These data are described in Section 2.3.1.2 of this Volume. Based upon these values, national average methylmercury concentrations in fish tissue were determined to be 0.052 µg/g and 0.26 µg/g for fish occupying trophic levels 3 and 4, respectively. Eagles consume approximately 500 g of food per day (U.S. EPA, 1993a, 1995a), 74% of which (370 g/d) consists of trophic level 3 fish, and 18% of which (90 g/d) consists of trophic level 4 fish. Multiplying these consumption rates by the methylmercury concentrations at trophic levels 3 and 4 and dividing by the average weight of an adult eagle (4.6 kg) (U.S. EPA, 1993a, 1995a) yields an average daily exposure of approximately 14 μg methylmercury/kg bw/d. Similar calculations were made for other piscivores in this hypothetical exposure scenario allowing comparisons to be made among species (see Table 3-4).

From a modeling standpoint, methylmercury levels in trophic level 3 fish and the mercury concentration in water are irrelevant to a ranking of predator exposure; only the relationship between the methylmercury concentrations in trophic levels 3 and 4 is critical. As noted previously, fish consumption rate expressed per gram of body weight has a large effect on these exposure calculations. Thus, despite consuming a comparatively small amount of the trophic level 3 fish, the kingfisher ranks well above any other birds (or mammals) in this estimated amount of mercury ingested per kg/bw.

3.4   Regional-Scale Exposure Estimates

There are many stationary, anthropogenic mercury sources in the U.S., and the impact of these emissions may not be limited to the local area around the facility. To account for impacts of mercury emitted from these non-local sources, the long-range transport of mercury was simulated using the RELMAP model. The RELMAP model was used to predict the average annual atmospheric mercury concentration and the wet and dry deposition flux for each 40 km^2 grid in the continental U.S. The emission, transport and fate of airborne mercury over the continental U.S. were modeled using meteorologic data for the year of 1989. This year was assumed to be a typical year from an atmospheric dispersion perspective. Inputs to the RELMAP model were obtained from the mercury emissions inventory presented as Volume II of this Report. In all, over 10,000 mercury emitting cells within the U.S. were addressed. A detailed description of the RELMAP model is provided in Section 4 of Volume III.

Table 3-4
Summary of Sample Calculations of
Wildlife Species Methylmercury Exposure
From Fish Ingestion, Based
on Average Fish Residue Values

Species	Sample Estimated Methylmercury Exposure from Fish Ingestion (μg/kg bw/d)
Kingfisher	25
Otter	15
Loon	10
Osprey	10
Mink	10
Eagle	9

3.4.1 Predicted Current Mercury Exposure Across the Continental U.S.

In the first stage of analysis, estimated total mercury deposition data were used with ARC/INFO cartography software to generate U.S. map overlays. The overlays can be applied to similar scale maps of natural resources and species distributions or combined with additional data, such as acid deposition or pH of surface waters. Figure 3-1 shows RELMAP projections for total (including wet and dry) anthropogenic mercury deposition. Nearly all the land area east of the Mississippi River is projected to receive mercury deposition greater than 5 $\mu g/m^2$. Highly industrialized northeastern states and south Florida are projected to receive more than 20 $\mu g/m^2$. RELMAP results are projections that may differ quantitatively from actual sampling data for a given locale. It is anticipated, however, that additional sampling data will confirm the prediction that mercury is deposited in significant quantities over large geographic areas.

Limitations on data precluded a quantitative, nation-wide analysis of the exposure of piscivorous wildlife to mercury. Existing data are sufficient, however, to permit a qualitative analysis. In the case of plant life, the analysis was limited to plotting the location of federally threatened or endangered species, thereby indicating where threatened populations coincide with estimated high mercury deposition.

Avian wildlife selected for this analysis included species that are widely distributed (kingfishers) and narrowly distributed (bald eagles, ospreys, and loons). All the birds selected were piscivores that feed at or near the top of aquatic food chains and are therefore at risk from biomagnified mercury.

Two of the mammals selected for this analysis (mink and river otters) are piscivorous and widely distributed. The other mammal selected, the Florida panther, is not widely distributed but is listed as an endangered species. The Florida panther lives in an environment known to be contaminated with mercury and preys upon small mammals (e.g., raccoons) that may contain high tissue burdens of mercury.

The maps and map overlays that follow were used to examine in a qualitative fashion the potential for anthropogenic mercury to impact representative piscivorous species in a variety of ecosystems. Animal distribution information was obtained from the Nature Conservancy (1994).

3.4.2 Locations of Socially Valued Environmental Resources

Major freshwater lakes and river systems potentially affected by high levels of atmospheric mercury deposition are illustrated in Figure 3-2. Most of the freshwater located in the lower 48 states occurs in areas where mercury deposition is predicted to be high. Because mercury accumulates in sediments, it is anticipated that significant mercury inputs to surface waters will continue for a long period of time even if atmospheric deposition is substantially reduced. The Great Lakes are particularly vulnerable due to the length of time necessary to replenish contaminated freshwater with clean freshwater.

Figure 3-3 shows the location of national resource lands, which include national parks and monuments, national forests, wildlife refuges and Native American reservation lands. The area of national resource lands that are predicted to have high mercury deposition is relatively small when compared with the total area of national resource lands, most of which are located in the western states. The small size of eastern resources makes them especially vulnerable to the effects of mercury because depleted wildlife populations cannot easily be repopulated from less-impacted adjoining regions. Increasingly, natural areas

Figure 3-1
Total Anthropogenic Mercury Deposition

Deposition 1-5 ug/m2
Deposition 5-10 ug/m2
Deposition > 10 ug/m2

3-7
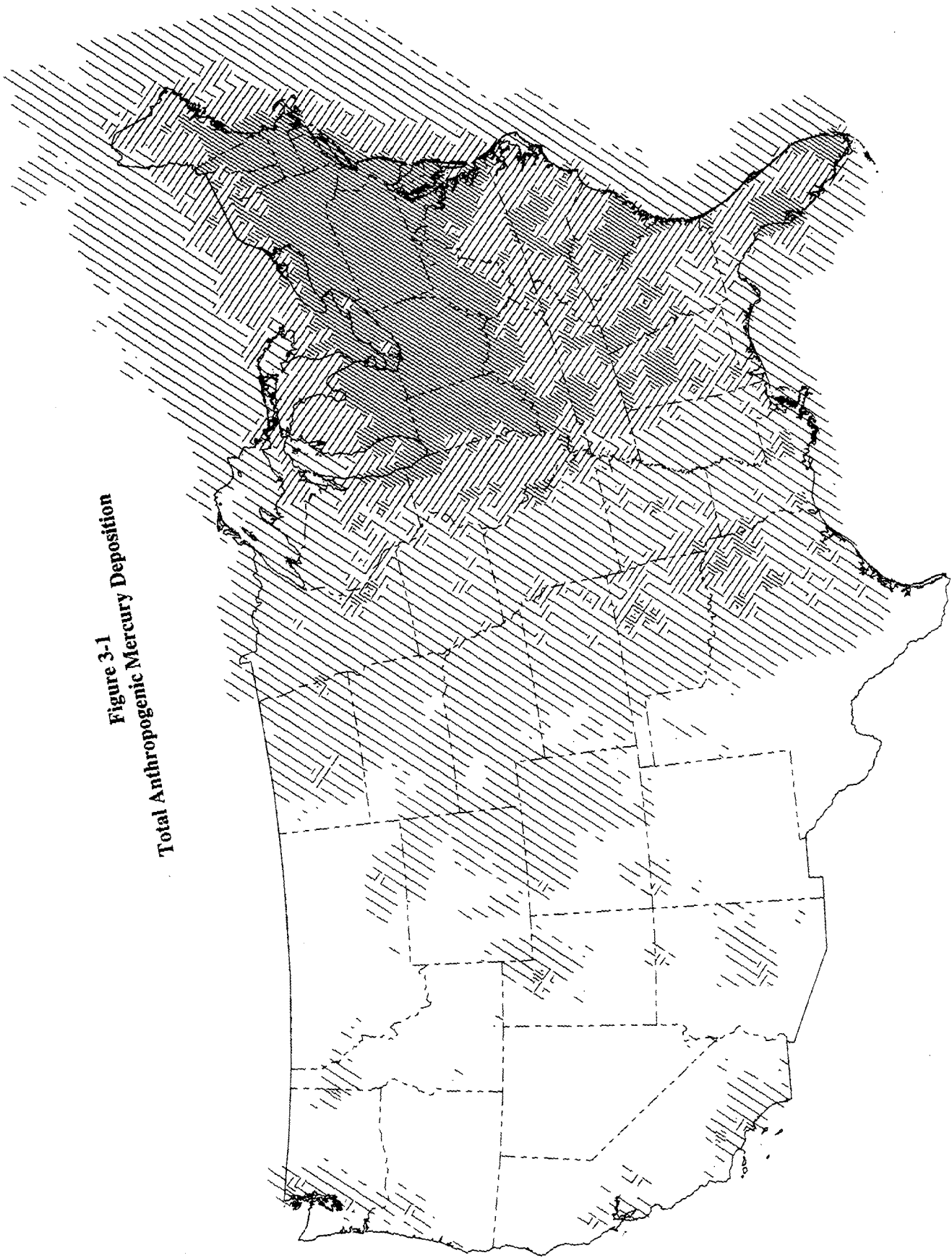
Figure 3-2
Major Rivers and Lakes
(Detail: Eastern U.S.)
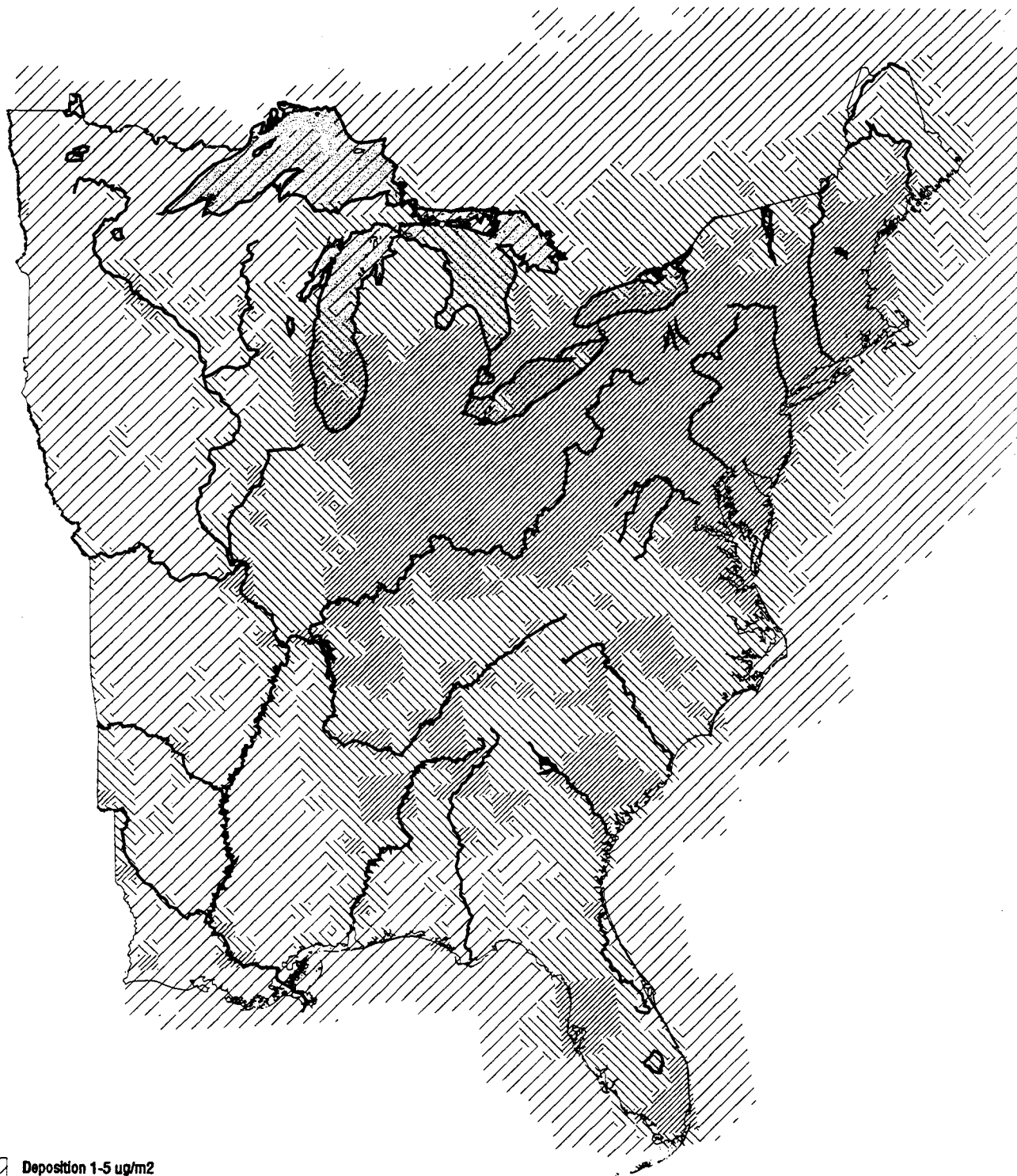
Deposition 1-5 ug/m2

Deposition 5-10 ug/m2

Deposition > 10 ug/m2

Figure 3-3
National Resource Lands
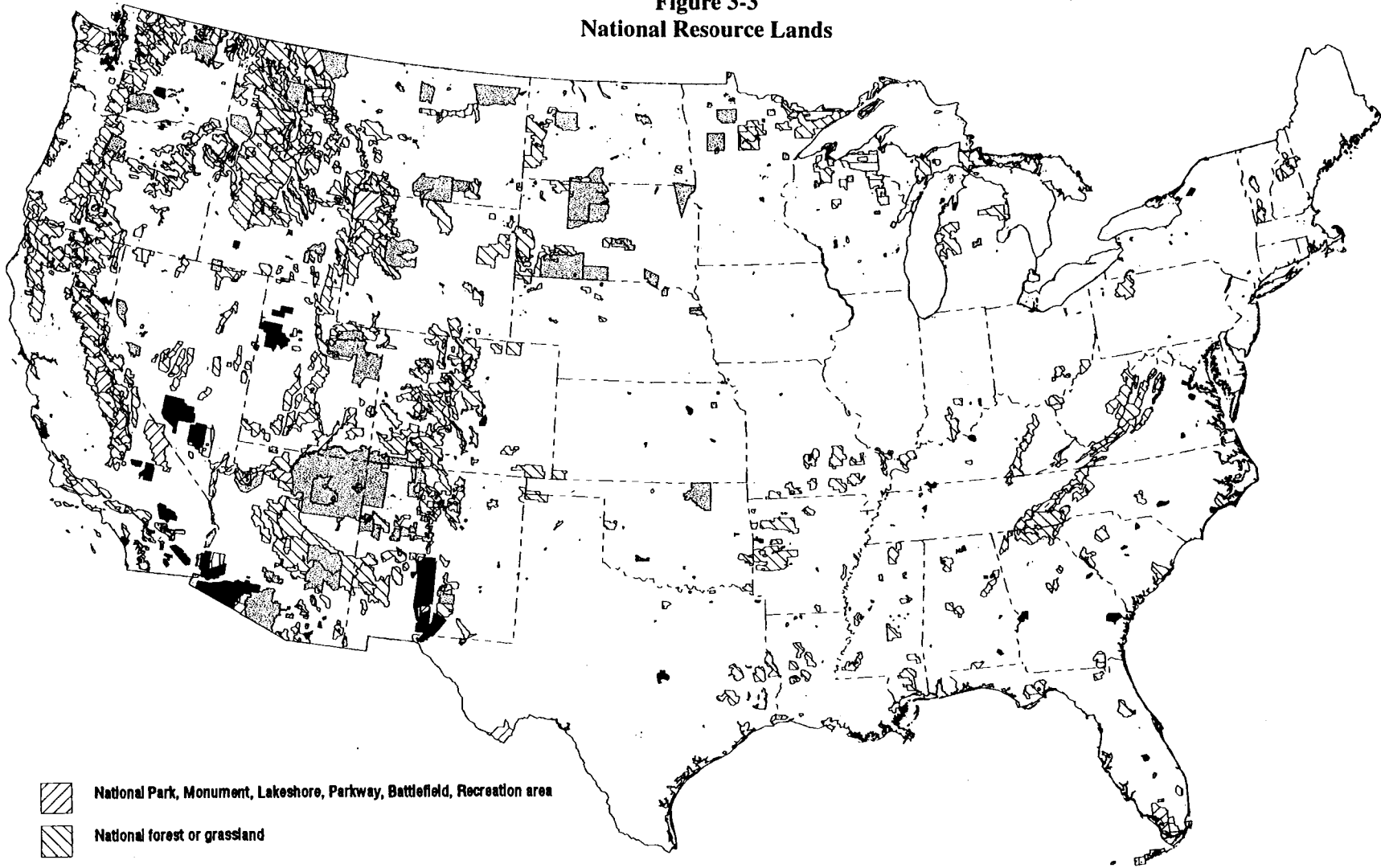
National Park, Monument, Lakeshore, Parkway, Battlefield, Recreation area

National forest or grassland

National wildlife refuge, game preserve, fish hatchery

National scenic waterways or wilderness area

Native American reservation lands

Military reservation

3-9

may become "islands" surrounded by development. The loss of biodiversity is an important problem that could be exacerbated by the added stress of mercury toxicity.

3.4.3   Airborne Deposition Overlay with Threatened and Endangered Plants

Figure 3-4 shows the geographic locations of populations of threatened and endangered plant species overlaid with RELMAP's predicted mercury deposition. Large concentrations of endangered plant populations exposed to high levels of deposition occur in central and southern Florida, along the northeastern coastal region, and scattered throughout the midwest.

3.4.4   Regions of High Mercury Deposition

Predicted mercury deposition rates in excess of 5 µg/m2 are shown in Figure 3-5. These data are used below to estimate the extent of overlap of wildlife species ranges with regions receiving high levels of mercury deposition. It should not be inferred from this analysis that wildlife living in areas that receive relatively low levels of mercury deposition are not at risk. For example, much of northern Wisconsin receives only moderate amounts of mercury, yet the occurrence of high mercury levels in fish is a well-documented problem. Nevertheless, it is of interest to define deposition patterns on a broad geographical scale. These data can then be interpreted in the context of regional and watershed-specific factors that contribute to mercury translocation, methylation, and bioaccumulation.

3.4.5   Regions of High Mercury Deposition Overlay with the Distribution of Acid Surface Waters

Figure 3-6 shows the co-occurrence of acidified surface waters (NAPAP, 1990) and regions receiving high levels of mercury deposition. While it is recognized that a variety of factors impact the methylation of mercury and its subsequent accumulation in aquatic biota (see Chapter 2 of this Volume), mercury residues in fish have been positively correlated with low pH in ecosystems of widely varying type, including both northern oligotrophic lakes and the lakes and wetlands of central Florida. Poorly buffered surface waters receiving high levels of mercury deposition are located in central Florida, throughout the Chesapeake Bay region, and in the northeastern U.S., including the Adirondack region of New York.

3.4.6   Regions of High Mercury Deposition Overlays with Wildlife Species Distribution Maps

Figure 3-7 shows the range of kingfisher habitat and areas where this habitat overlaps with regions of high mercury deposition. Kingfishers consume fish primarily from trophic level 3. Approximately 29% of the kingfisher's range overlaps with areas of high mercury deposition. On a nationwide basis, mercury does not appear to be a threat to the species. However, as indicated by the exposure assessment in Section 3.3, kingfishers consume more mercury on a body weight basis than any of the other wildlife species examined.

Figure 3-8 overlays the range of bald eagle habitat onto regions that receive high levels of mercury deposition. Although a recovery in the population of bald eagles in the lower 48 states has resulted in a status upgrade from "endangered" to "threatened," bald eagle populations are still depleted throughout much of their historical range. Bald eagles can be found seasonally in large numbers in several geographic locations, but most of these individuals are transient, and the overall population is still small. Historically, eagle populations in the lower 48 states have been adversely impacted by the effects of bioaccumulative contaminants (primarily DDT and perhaps also PCBs). Approximately 34% of the bald eagle's range overlaps with regions of high mercury deposition. Areas of particular concern include the Great Lakes region, the northeastern Atlantic states, and south Florida.

Figure 3-4
Threatened and Endangered Plant Species and Anthropogenic Mercury Deposition

● Threatened or Endangered Plant

Deposition 1-5 ug/m2

Deposition 5-10 ug/m2
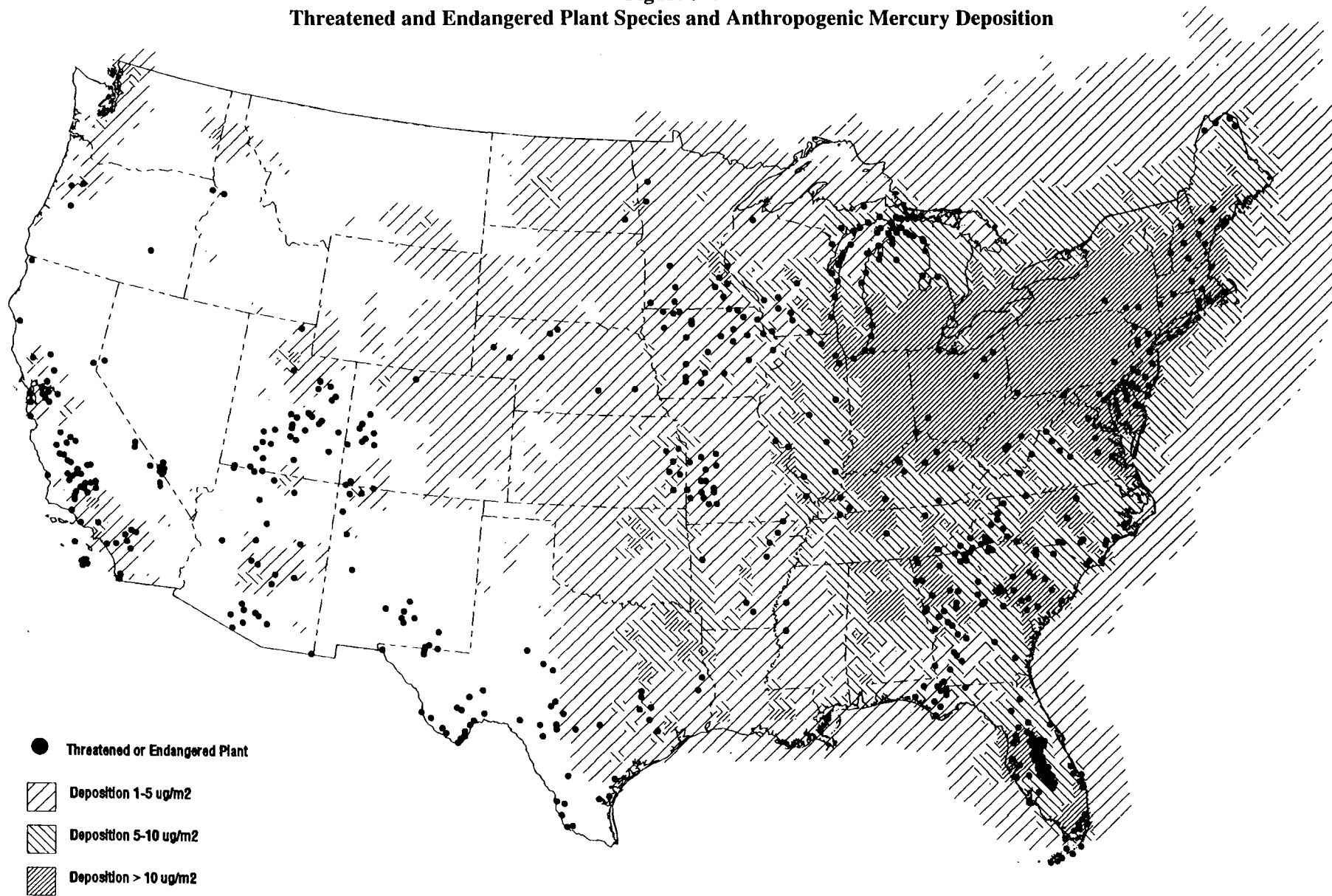
Deposition > 10 ug/m2

Figure 3-5
Regions of High Mercury Deposition
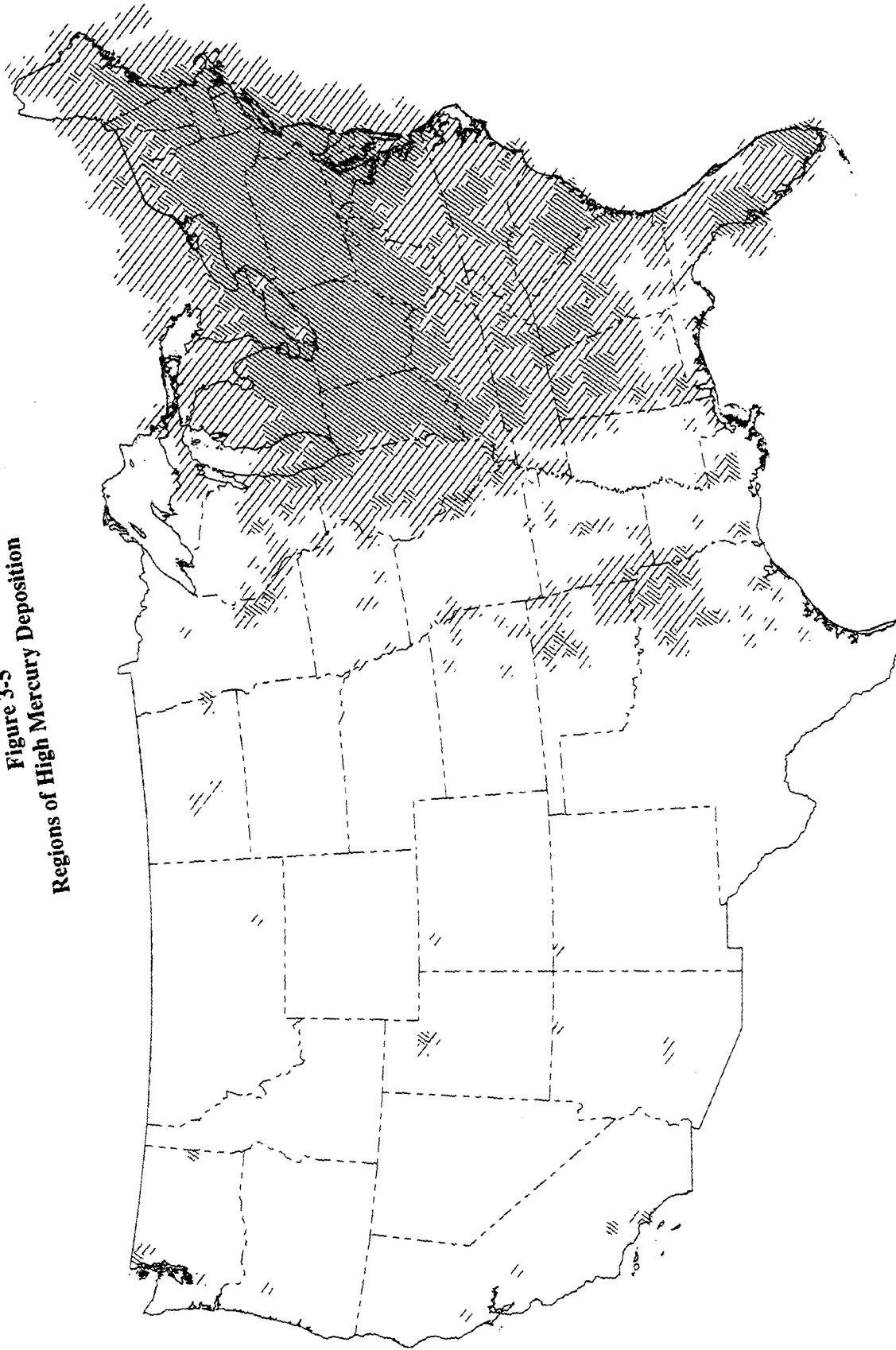
Deposition 5-10 ug/m2

Deposition > 10 ug/m2

Figure 3-6
Regions of High Mercury Deposition and the Distribution of Acid Surface Waters

NORTHEAST

UPPER MIDWEST

WEST

MID-APPALACHIANS

MID-ATLANTIC
COASTAL PLAIN

INTERIOR SOUTHEAST

FLORIDA

5-20% pH < = 5.5, Deposition 5-10 ug/m2

5-20% pH < = 5.5, Deposition > 10 ug/m2

> 20% pH < = 5.5, Deposition 5-10 ug/m2

> 20% pH < = 5.5, Deposition > 10 ug/m2
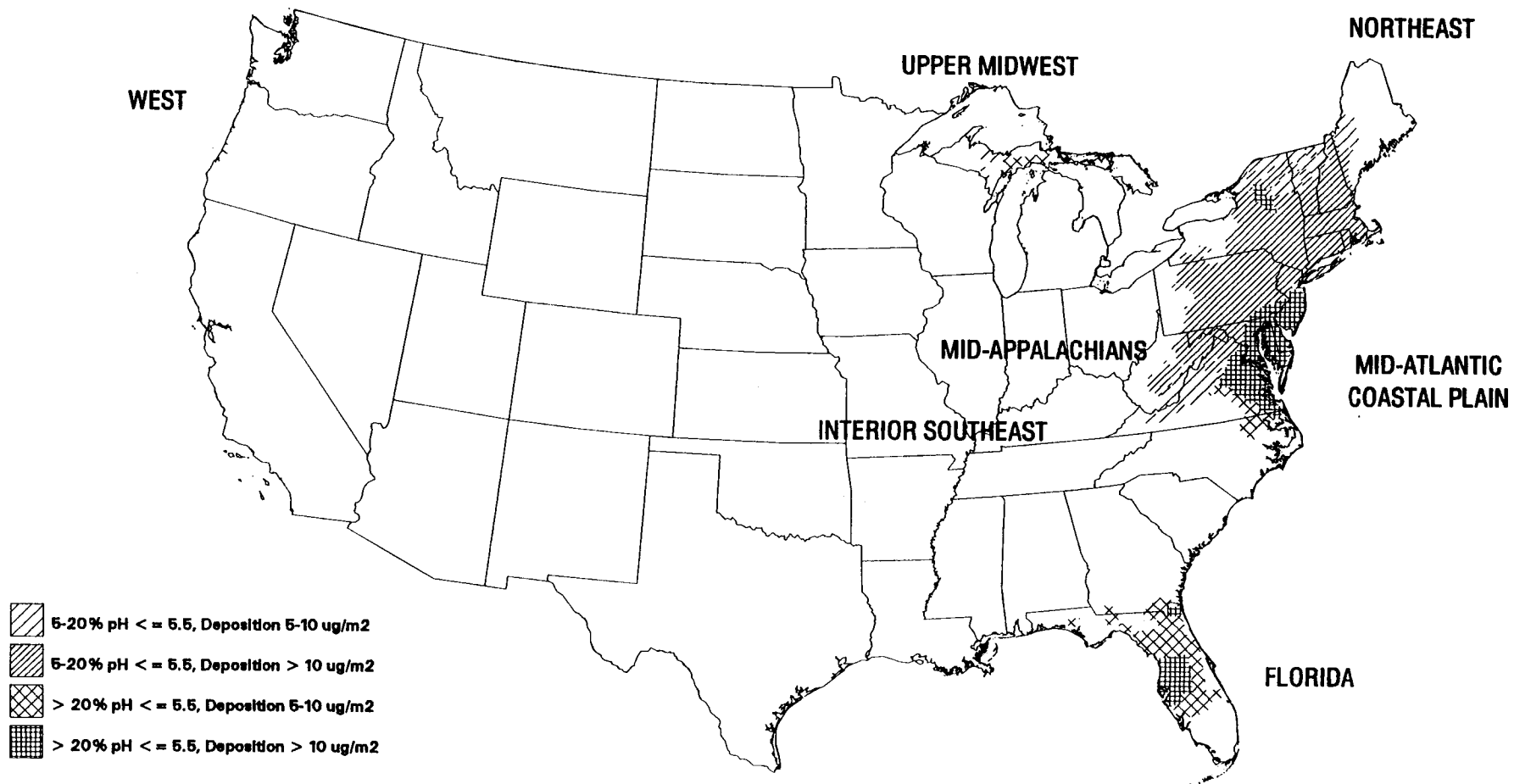
Figure 3-7
Kingfisher Range and Regions of High Mercury Deposition

NORTHEAST

UPPER MIDWEST

WEST

MID-APPALACHIANS

MID-ATLANTIC
COASTAL PLAIN

INTERIOR SOUTHEAST

Range for this species

Deposition 5-10 ug/m2

Deposition > 10 ug/m2

FLORIDA
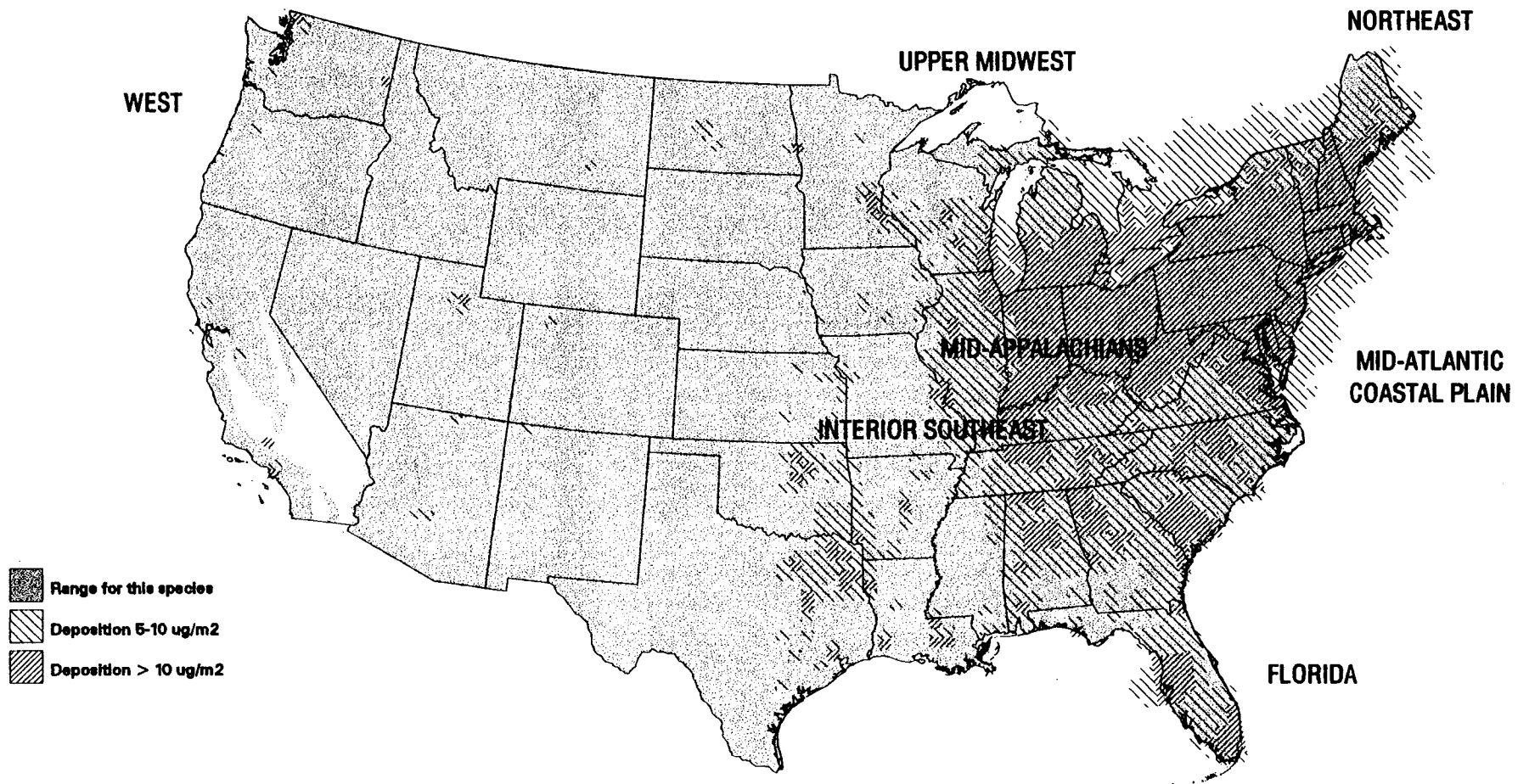
Figure 3-8
Bald Eagle Range and Regions of High Mercury Deposition

NORTHEAST

UPPER MIDWEST

WEST

MID-APPALACHIANS

MID-ATLANTIC
COASTAL PLAIN

INTERIOR SOUTHEAST

Range for this species

Deposition 5-10 ug/m2

Deposition > 10 ug/m2

FLORIDA

3-15
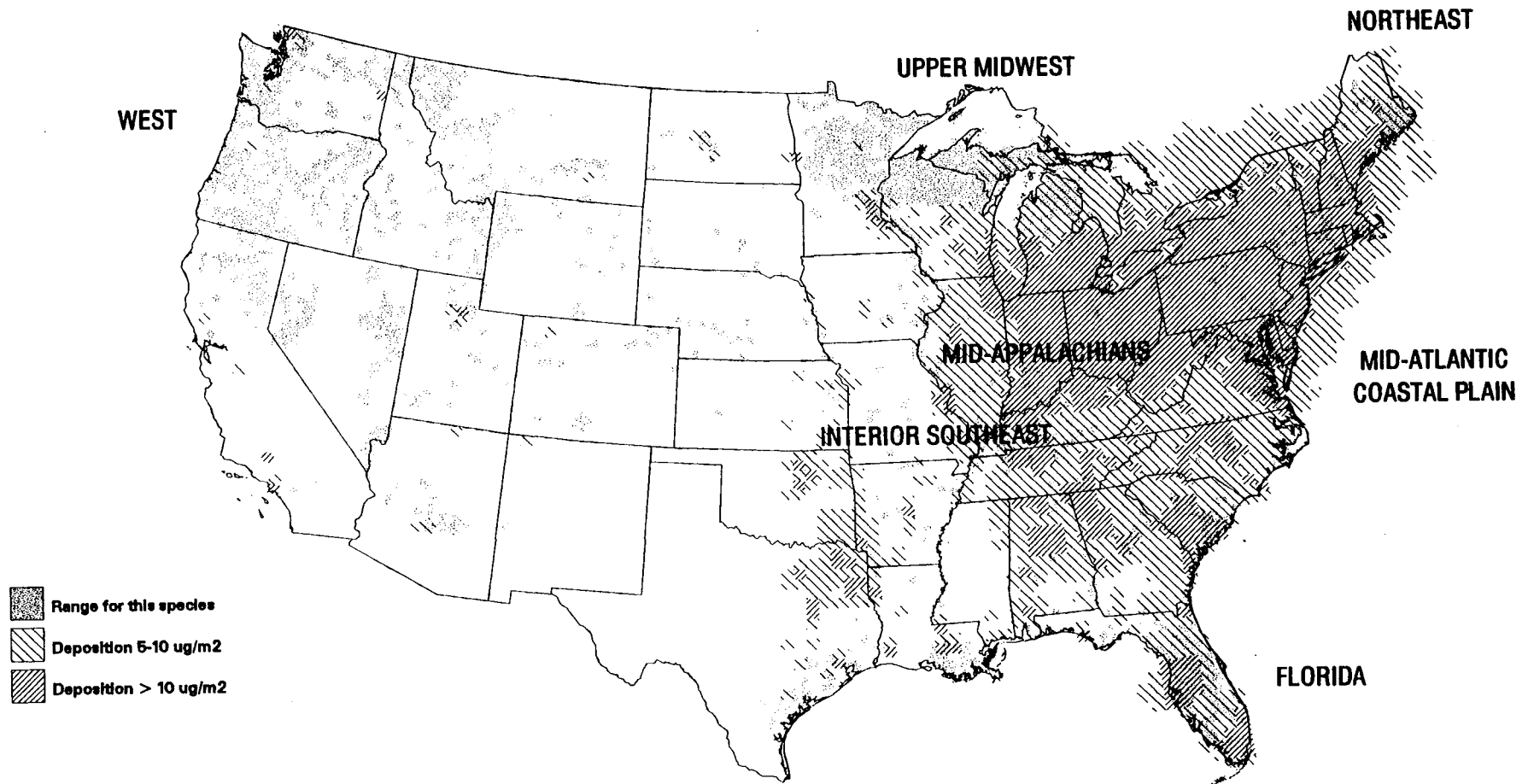
Figure 3-9 indicates where the range of osprey coincides with regions of high mercury deposition. Nationwide, approximately 20% of the osprey's range overlaps these regions; however, a much larger fraction of the osprey's eastern population occurs within these regions. The osprey diet consists almost exclusively of fish. Osprey populations underwent severe declines during the 1950s through the 1970s due to widespread use of DDT and related compounds.

Figure 3-10 depicts areas where the range of the common loon coincides with regions of concern. Nearly 40% of the loon's range is located in regions of high mercury deposition. Limited data from a study of a mercury point source showed that the reproductive success of loons was negatively correlated with exposure to mercury in a significant dose-response relationship (see Section 2.3.3 of this Volume). Mercury residues in fish collected from lakes used as loon breeding areas may, in some cases, exceed levels that, on the basis of the point source study, are associated with reproductive impairment. Loons frequently breed in areas that have been adversely impacted by acid deposition. An assessment of mercury's effects on loon populations is complicated by the fact that decreases in surface water pH have been associated with both increased mercury residues in fish and a decline in the available forage base.

Figure 3-11 shows the Florida panther's range. All (100%) of the panther's range falls within an area of high mercury deposition. Mercury levels found in tissues obtained from dead panthers are similar to levels that have been associated with frank toxic in other feline species. The State of Florida has taken measures to reduce the risk to panthers posed by mercury. Existing plans include measures to increase the number of deer available as prey in order to reduce the reliance of panthers on raccoons. As indicated previously, raccoons frequently feed at or near the top of aquatic food webs and can accumulate substantial tissue burdens of mercury. An evaluation of the risk posed by mercury to the Florida panther is complicated by the possible impacts of other chemical stressors, habitat loss and inbreeding.

Figure 3-12 shows where mink habitat coincides with regions of high mercury deposition (approximately 35% nationwide). Mink occupy a large geographic area and are common throughout this range, although rarely observed due to their nocturnal habits. Mink are extremely aggressive carnivores and, given the opportunity, will prey on small mammals and birds. Many subpopulations, however, prey almost exclusively on fish and other aquatic biota. Due to allometric considerations, the mink may be exposed to more mercury on a body weight basis than larger piscivorous mammals feeding at higher trophic levels. In several cases, mercury residues in wild-caught mink have been shown to be equal to or greater than levels associated with toxic effects in the laboratory.

Figure 3-13 shows where the range of the river otter coincides with areas of high mercury deposition (approximately 38% nationwide). River otters occupy large areas of the United States, but their population numbers are thought to be declining in both the midwestern and southeastern states. The river otter's diet is almost exclusively of aquatic origins and includes fish (primarily), crayfish, amphibians and aquatic insects. The consumption of large, piscivorous fish puts the river otter at risk from bioaccumulative contaminants such as mercury. Like the mink, mercury residues in some wild-caught otters have been shown to be close to, and in some cases greater than, concentrations associated with frank toxic effects.

3.5   Modeling Exposures Near Mercury Emissions Sources

In this section, computer models are used to predict exposures of piscivorous wildlife to mercury resulting from hypothetical local source emissions. Modeling assumptions related to the presence of "background" mercury as well as mercury transported from other regions of the U.S. are also discussed.

Figure 3-9
Osprey Range and Regions of High Mercury Deposition
(Detail: Eastern U.S.)

Range for this species

Deposition 5-10 ug/m2

Deposition > 10 ug/m2
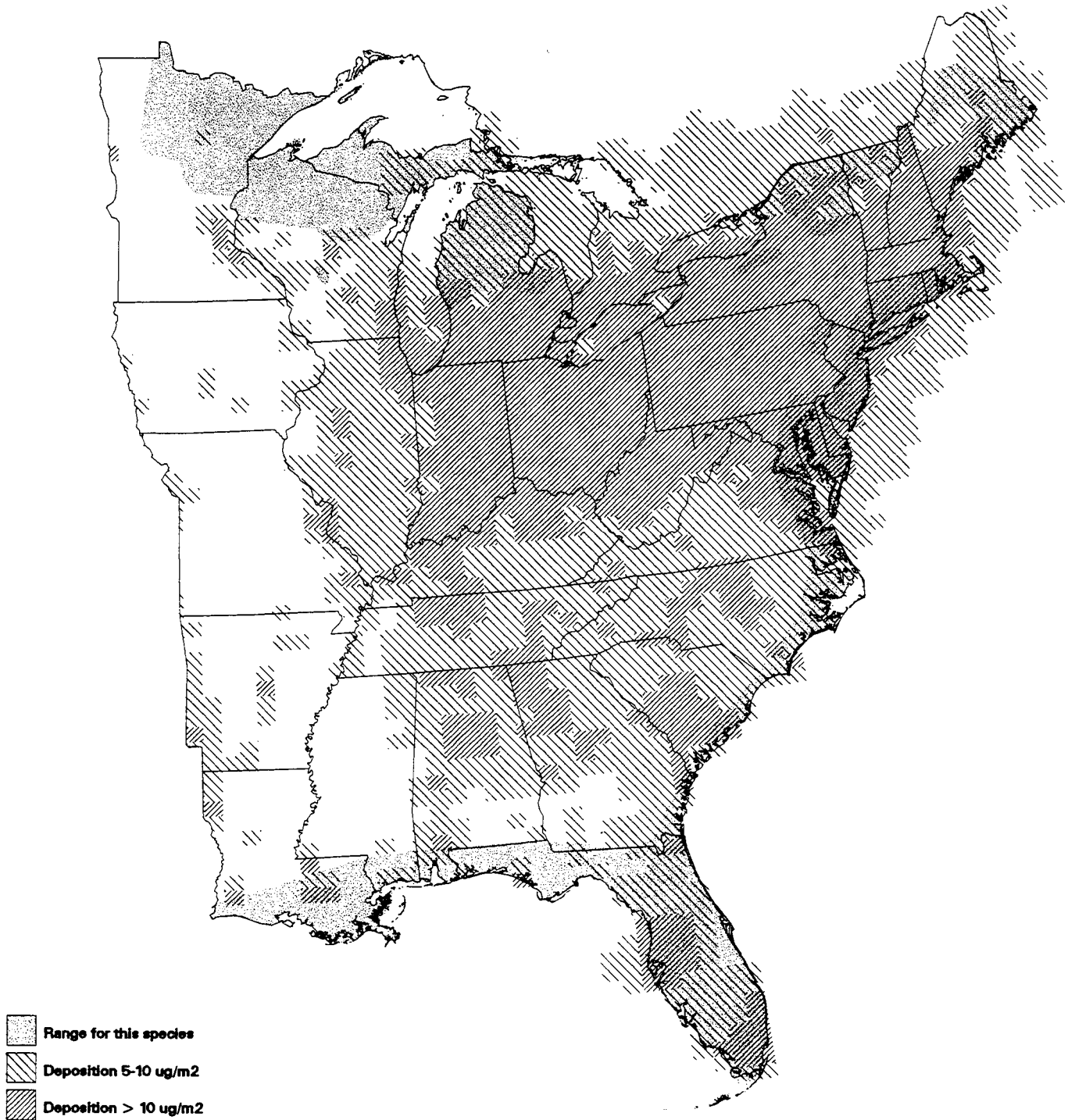
Figure 3-10
Common Loon Range and Regions of High Mercury Deposition

NORTHEAST

UPPER MIDWEST

WEST

MID-APPALACHIANS

MID-ATLANTIC
COASTAL PLAIN

INTERIOR SOUTHEAST

Range for this species

Deposition 5-10 ug/m2

Deposition > 10 ug/m2

FLORIDA

3-18
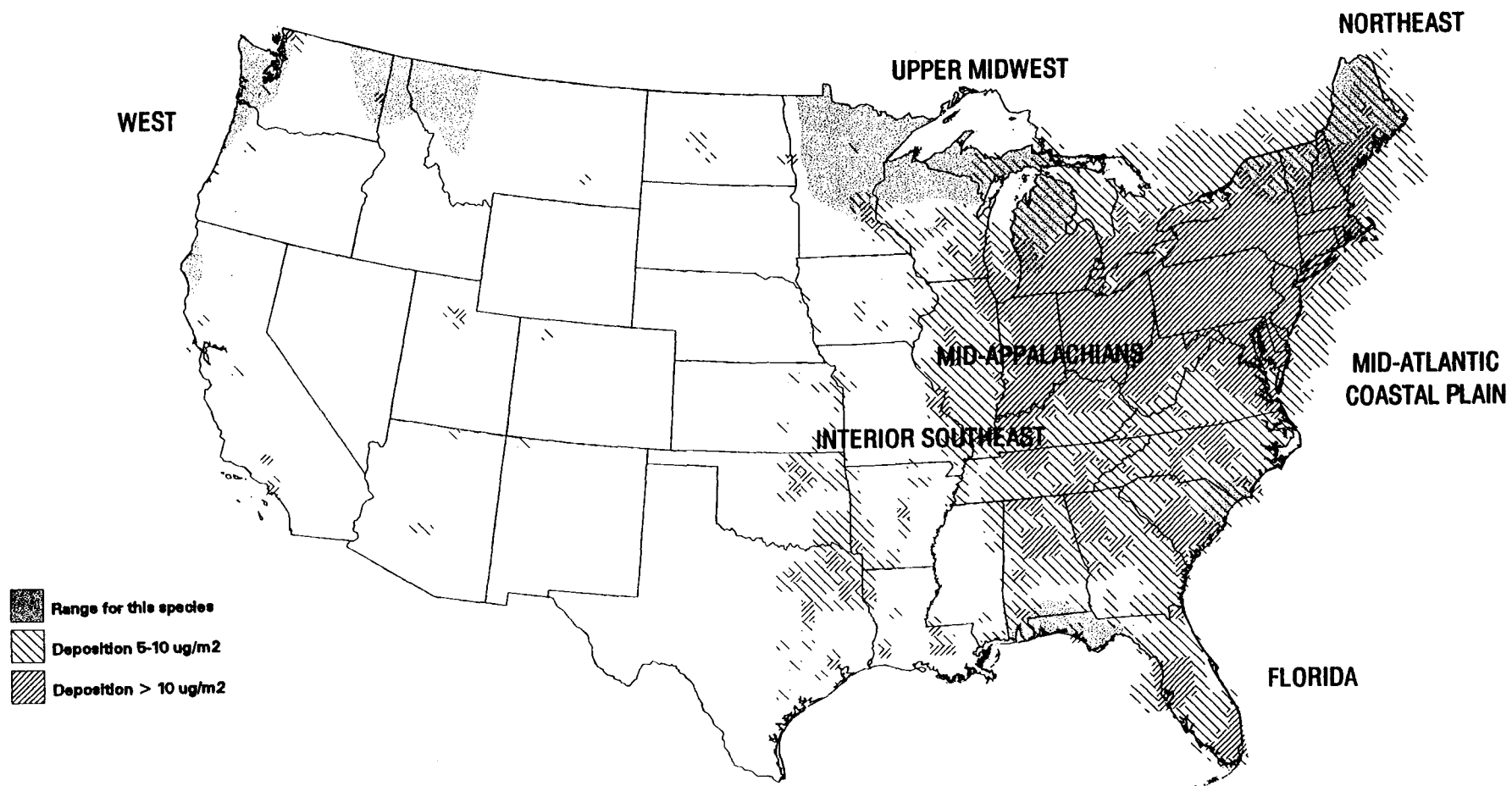
Figure 3-11
Florida Panther Range and Regions of High Mercury Deposition
(Detail: Eastern U.S.)

Range for this species

Deposition 5-10 ug/m2

Deposition > 10 ug/m2
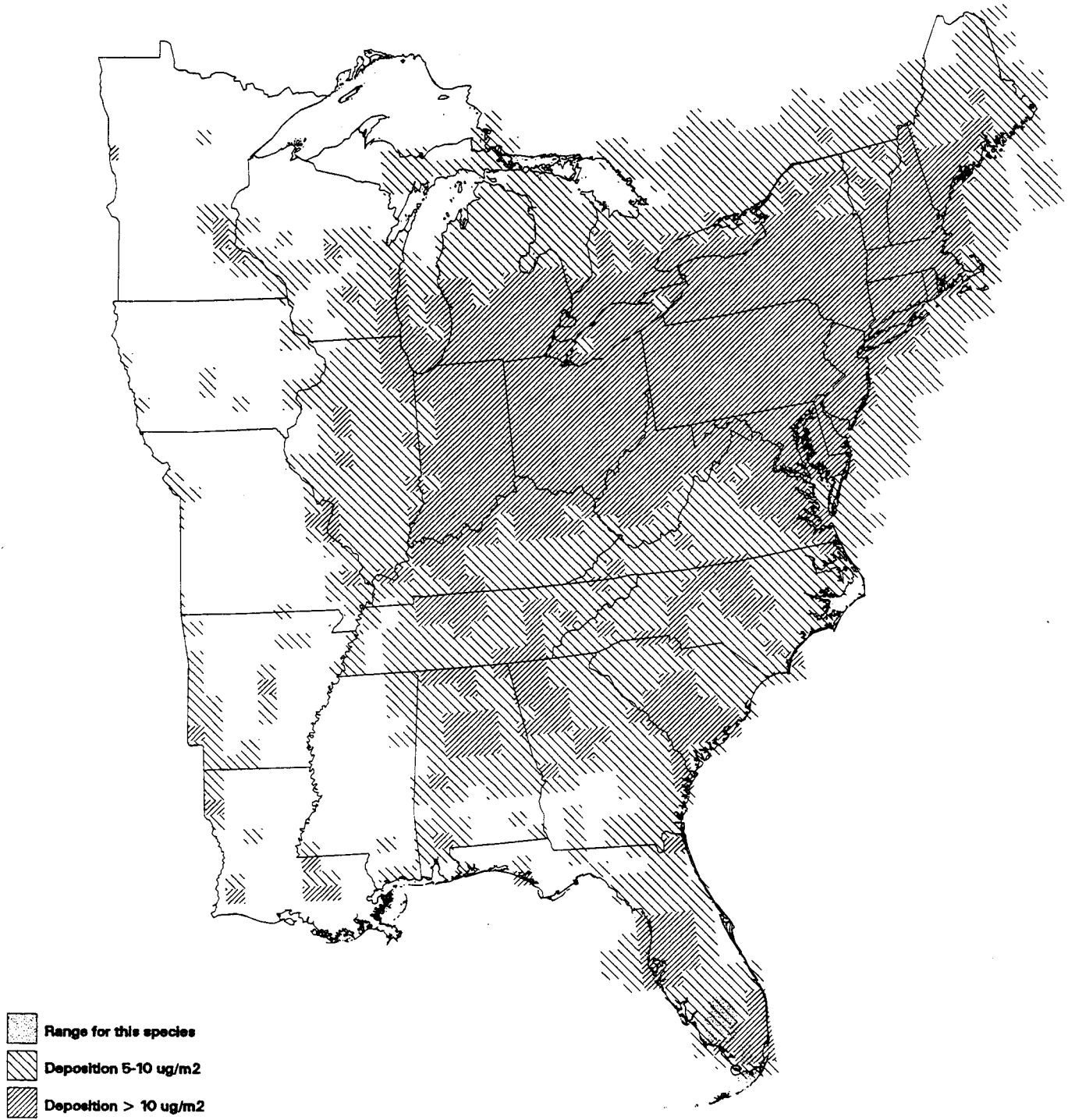
Figure 3-12
Mink Range and Regions of High Mercury Deposition

NORTHEAST

UPPER MIDWEST

WEST

MID-APPALACHIANS

MID-ATLANTIC
COASTAL PLAIN

INTERIOR SOUTHEAST

Range for this species

Deposition 5-10 ug/m2

Deposition > 10 ug/m2

FLORIDA
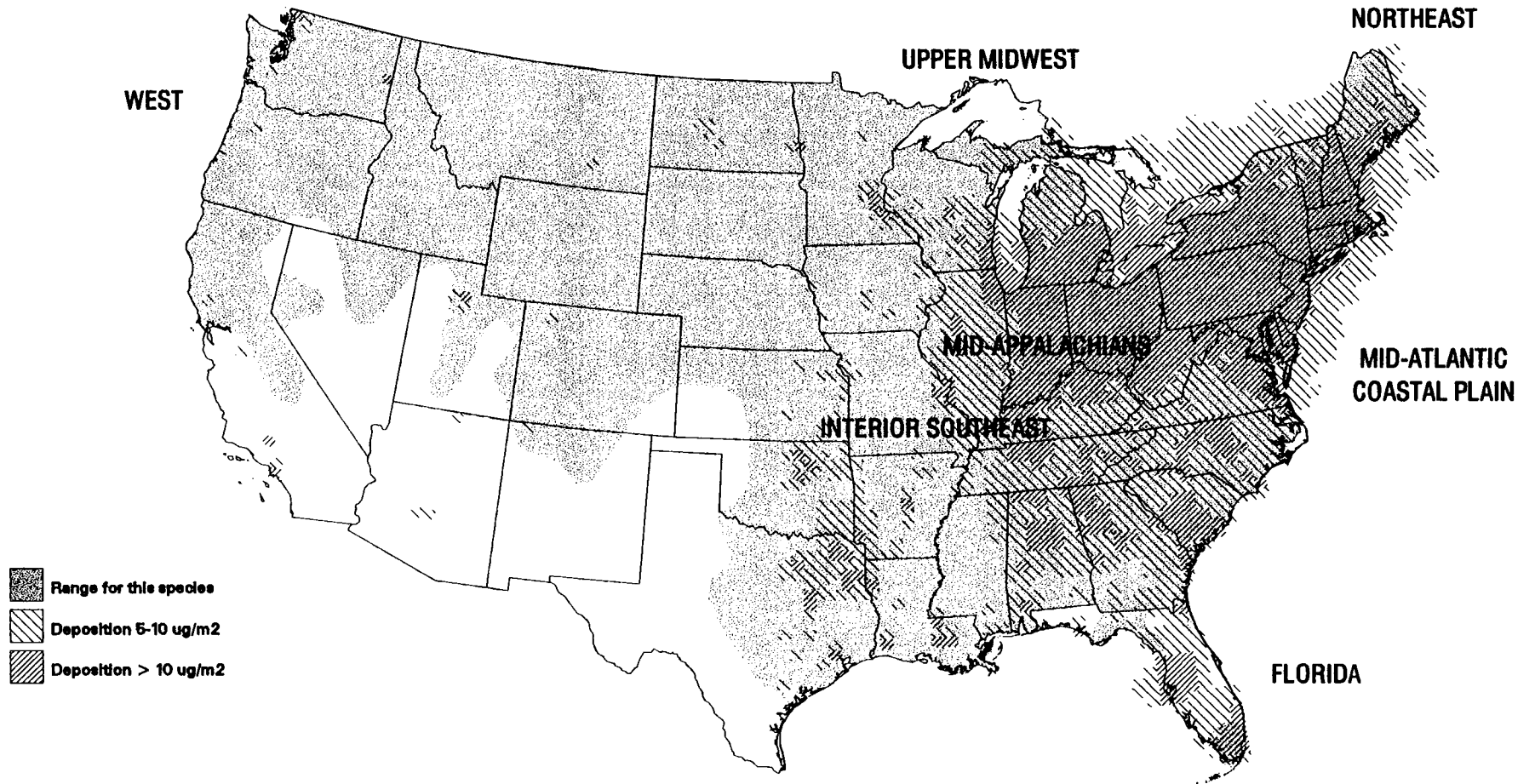
Figure 3-13
River Otter Range and Regions of High Mercury Deposition

WEST

UPPER MIDWEST

NORTHEAST

MID-APPALACHIANS

MID-ATLANTIC COASTAL PLAIN

INTERIOR SOUTHEAST

FLORIDA

Range for this species

Deposition 5-10 ug/m2

Deposition > 10 ug/m2
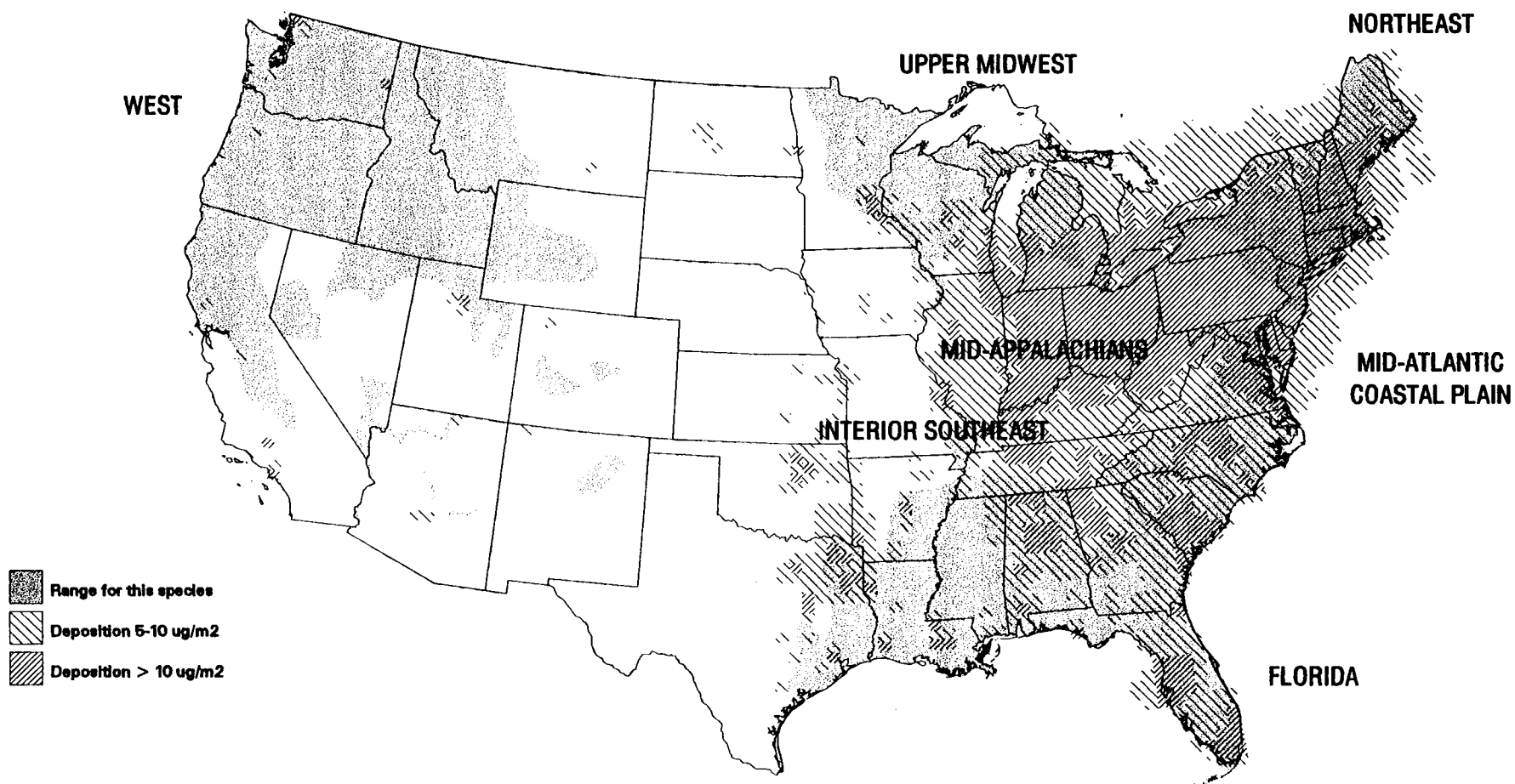
3.5.1   Estimates of Background Mercury

In Volume III of this Report, it was noted that mercury is a constituent of the environment and has always been present on the planet. Estimates of atmospheric mercury concentrations and deposition rates from periods pre-dating large-scale anthropogenic emissions ("pre-anthropogenic"), as well as levels due to current sources, were determined for hypothetical eastern and western sites. These estimates were used as inputs to the IEM-2M model. The IEM-2M model was run until equilibrium was achieved for both the eastern and western sites and for both the pre-anthropogenic and current time periods. Chemical equilibrium is defined here as "a steady state, in which opposing chemical reactions occur at equal rates" (Pauling, 1963). When modeling the pre-anthropogenic period, the initial conditions of all model compartments, except the atmosphere, were set to a mercury concentration of 0. The results of running the pre-anthropogenic conditions to equilibrium in IEM-2M were used as the initial conditions for estimating the current mercury concentrations. Table 3-5 lists the estimated mercury air concentrations and deposition rates used at both hypothetical sites and for both time periods.

Table 3-5
Inputs to IEM-2M Model for the Two Time Periods Modeled

Time Period	Eastern Site		Western Site	
	Air Concentration ng/m^3	Annual Deposition Rate $\mu g/m^2/yr$	Air Concentration ng/m^3	Annual Deposition Rate $\mu g/m^2/yr$
Pre-Anthropogenic	0.5	3	0.5	1
Current	1.6	10	1.6	2

3.5.2   Hypothetical Wildlife Exposure Scenarios

The exposure of piscivorous wildlife to mercury originating from hypothetical point sources was characterized using the same approach as that used to characterize human exposure to mercury from consumption of contaminated fish (see Volumes III and IV). A benefit of this approach is that it facilitates comparisons between exposure levels to human and wildlife receptors.

Mercury exposure was assessed for piscivorous wildlife hypothetically located at two generic lacustrine sites: (1) a humid site east of 90 degrees west longitude and (2) a more arid site west of 90 degrees west longitude (see Volume III for site descriptions). Both sites were assumed to be located in relatively flat terrain. Exposure at each site was assessed for piscivorous wildlife living around one of three lakes located at 2.5, 10, or 25 km from the emissions source, as shown in Figure 3-14. The primary physical differences between the two hypothetical sites as parameterized included the assumed average annual precipitation rate, the assumed erosion

Figure 3-14
Configuration of Hypothetical Water Body and Watershed Relative to Local Source
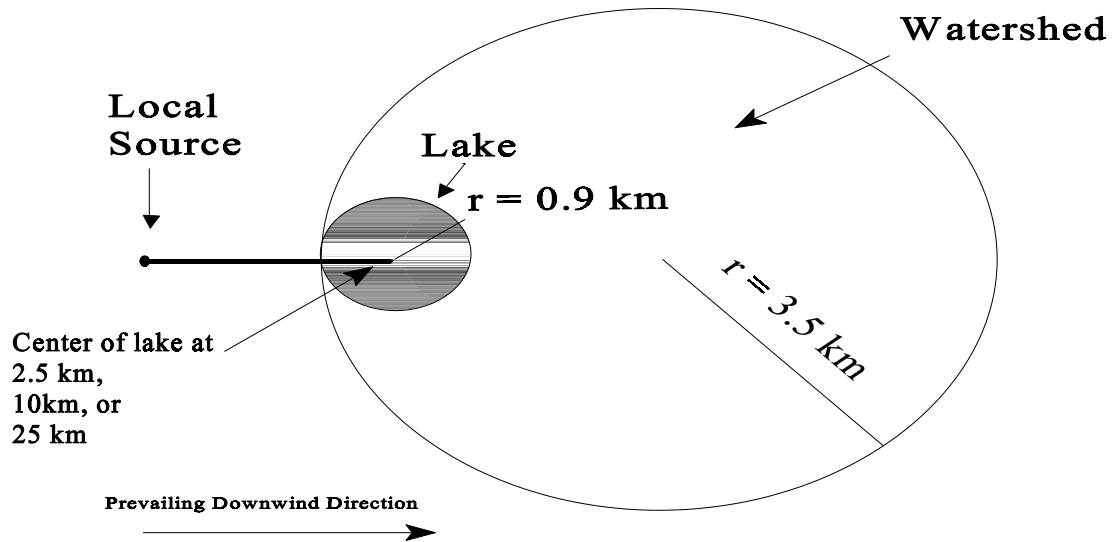
characteristics for the watershed, and the amount of dilution flow from the water body. The eastern site had generally steeper terrain in the watershed than was assumed for the western site. The drainage lakes were assumed to be circular with a diameter of 1.78 km and average depth of 5 m, with a 2 cm benthic sediment depth. The watershed area was 37.3 km^2. In each case, deposition information was used to estimate mercury concentrations in water, averaged over the entire lake.

3.5.3   Predicted Mercury Exposure Around Emissions Sources

The goal of the local scale analysis was to evaluate the extent to which mercury emissions sources have the potential to create locally elevated mercury exposures for piscivorous wildlife receptors. Air concentrations and deposition rates due to a single local source were predicted using the GAS-ISC3 atmospheric dispersion and deposition model. For the purposes of this study, hypothetical sources were assumed to contribute mercury in addition to that simulated by RELMAP. Details of the local-scale modeling exercise are presented in Volume III of this Report. Additionally, current background concentrations of mercury in various media were estimated and used as inputs to the modeling (see Volume III for description).

Model plants (hypothetical anthropogenic mercury emissions sources) representing four source classes were developed to represent a range of mercury emissions sources. The source categories were selected for the indirect exposure analysis based on their estimated annual mercury emissions or their potential to be localized point sources of concern. The categories selected were: municipal waste combustors (MWCs), medical waste incinerators (MWIs), utility boilers, and chlor-alkali plants. Table 3-6 shows the process parameters assumed for each of these facilities. The characteristics of the facilities were derived based on typical rather than extreme representations; the facilities are known as model plants (see Volume II).

Table 3-6
Process Parameters for the Model Plants Considered in the Local Impact Analysis

Model Plant	Plant Size	Capacity (% of year)	Stack Height (ft)	Stack Diameter (ft)	Hg Emission Rate (kg/yr)	Speciation Percent $(Hg^0/Hg^{2+}/Hg_P)$	Exit Velocity (m/sec)	Exit Temperature (°F)
Large Municipal Waste Combustors	2,250 tons/day	90%	230	9.5	220	60/30/10	21.9	285
Small Municipal Waste Combustors	200 tons/day	90%	140	5	20	60/30/10	21.9	375
Large Commercial HMI Waste Incinerator (Wetscrubber)	1500 lb/hr capacity (1000 lb/hr actual)	88%	40	2.7	4.58	33/50/17	9.4	175
Large Hospital HMI Waste Incinerators (Good Combustion)	1000 lb/hr capacity (667 lb/hr actual)	39%	40	2.3	23.9	2/73/25	16	1500
Small Hospital HMI Waste Incinerators (1/4 sec Combustion)	100 lb/hr capacity (67 lb/hr actual)	27%	40	0.9	1.34	2/73/27	10.4	1500
Large Hospital HMI Waste Incinerators (Wet Scrubber)	1000 lb/hr capacity (667 lb/hr actual)	39%	40	2.3	0.84	33/50/17	9.0	175
Small Hospital HMI Waste Incinerators (Wet Scrubber)	100 lb/hr capacity (67 lb/hr actual)	27%	40	0.9	0.05	33/50/17	5.6	175
Large Coal-fired Utility Boiler	975 Megawatts	65%	732	27	230	50/30/20	31.1	273
Medium Coal-fired Utility Boiler	375 Megawatts	65%	465	18	90	50/30/20	26.7	275
Small Coal-fired Utility Boiler	100 Megawatts	65%	266	12	10	50/30/20	6.6	295
Medium Oil-fired Utility Boiler	285 Megawatts	65%	290	14	2	50/30/20	20.7	322
Chlor-alkali plant	300 tons chlorine/day	90%	10	0.5	380	70/30/0	0.1	Ambient

[a] Hg^0 = Elemental Mercury
[b] Hg^{2+} = Divalent Vapor Phase Mercury
[c] Hg_P = Particle-Bound Mercury

GAS-ISC3 was employed to estimate deposition originating from local point sources (<50 km from the receptor). The IEM-2M model was then utilized to estimate the fate of mercury in the watershed and water body. The estimated concentrations of dissolved methylmercury in the water column were used to predict methylmercury concentrations in fish that occupy trophic levels 3 and 4. This was accomplished by multiplying the predicted methylmercury dissolved water concentration by the BAF at each trophic level. Wildlife receptors were assumed to ingest the fish at rates given previously (Table 3-3).

3.5.4   Results of Hypothetical Exposure Scenarios

High rates of mercury deposition were associated with proximity to industrial sources emitting substantial levels of divalent mercury (see Tables 3-7 and 3-8). Additional factors that contributed to high local deposition rates include low stack height and slow stack exit gas velocities. In general, predicted dissolved methylmercury concentrations in lake waters located 2.5 km from the source were higher than levels predicted at 10 or 25 km. This was due primarily to the dilution of the mercury emissions in the atmosphere. Mercury concentrations in fish (hence the mercury exposure to piscivores) were proportional to dissolved methylmercury levels in the local waters. When the two hypothetical locations were compared (western and eastern), higher mercury concentrations were predicted to occur in the environmental media at the eastern location. This was due primarily to higher levels of precipitation at the eastern site, which tends to remove mercury from the atmosphere. Also, the assumptions of background mercury are higher for the eastern than the western site. On a per kilogram of body weight per day basis, the species predicted to be most exposed were the kingfisher and the otter.

3.5.5   Issues Related to Combining Models to Assess Environmental Fate of Mercury and Exposures to Wildlife

In modeling the environmental fate and subsequent exposure of piscivorous wildlife to mercury emitted from a number of different sources, many simplifying assumptions have been made. Each simplifying assumption is associated with some degree of uncertainty; the accumulation of these uncertainties results in uncertainty in the exposure levels predicted by the models. Many of the input parameters to the models may also be quite variable across time and location. This variability leads to uncertainty in the modeling results. While no effort is made here to quantify these variabilities and uncertainties, this section will attempt to describe those deemed most significant to this element of the assessment.

There is no consensus approach for developing exposure scenarios for pollutants such as mercury, which have always been environmental constituents (i.e., how to incorporate background concentrations into environmental fate modeling). The approach developed for this document is clearly not the only approach that could have been taken to account for environmental background concentrations; however, each potential alternative approach evaluated also presented associated uncertainty. If the error in estimate of background results in an overestimation of concentrations in environmental media from these sources, the presented impacts of anthropogenic sources will be underestimated, and vice versa.

Combining the outputs of the different environmental fate models, while deemed necessary for this pollutant, clearly compounds the uncertainty relating to individual model assumptions and input parameter uncertainties. The chemical properties associated with elemental mercury and divalent mercury species in the atmosphere are assumed to be very dissimilar. This necessitates an atmospheric modeling approach that can account for long range atmospheric transport of anthropogenic emissions as well as local transport from a given source. The primary impacts of environmental mercury result from bioaccumulation and biomagnification in the aquatic food chain. This necessitates the use of a model such as IEM-2M that

Table 3-7
Predicted MHg Exposure to Ecological Receptors for Eastern Site (Local + RELMAP 50th Percentile)

		MHg Concentration (µg/g)						Predicted MHg Exposure from Ingestion of Fish (mg/kg/day)					
		MHg Dissolved Concentration (ng/L)	Tier3	Tier4	Background	RELMAP	ISC	Bald Eagle	Osprey	Kingfisher	River Otter	Mink	Loon
Variant b:Large Municipal Waste Combustor	2.5 km	1.7E-01	2.7E-01	1.2E+00	38%	7%	54%	4.4E-02	5.4E-02	1.4E-01	7.4E-02	5.4E-02	5.4E-02
	10 km	1.1E-01	1.8E-01	7.6E-01	58%	11%	31%	2.9E-02	3.6E-02	8.9E-02	4.8E-02	3.6E-02	3.6E-02
	25 km	8.9E-02	1.4E-01	6.0E-01	73%	14%	13%	2.3E-02	2.8E-02	7.1E-02	3.9E-02	2.8E-02	2.8E-02
Variant b:Small Municipal Waste Combustor	2.5 km	9.5E-02	1.5E-01	6.4E-01	68%	13%	18%	2.5E-02	3.0E-02	7.6E-02	4.1E-02	3.0E-02	3.0E-02
	10 km	8.2E-02	1.3E-01	5.6E-01	79%	15%	6%	2.2E-02	2.6E-02	6.6E-02	3.6E-02	2.6E-02	2.6E-02
	25 km	7.9E-02	1.3E-01	5.3E-01	83%	16%	2%	2.1E-02	2.5E-02	6.3E-02	3.4E-02	2.5E-02	2.5E-02
Large Commercial HMI	2.5 km	9.6E-02	1.5E-01	6.5E-01	68%	13%	19%	2.5E-02	3.1E-02	7.7E-02	4.2E-02	3.1E-02	3.1E-02
	10 km	8.0E-02	1.3E-01	5.4E-01	82%	16%	3%	2.1E-02	2.5E-02	6.4E-02	3.5E-02	2.5E-02	2.5E-02
	25 km	7.8E-02	1.2E-01	5.3E-01	83%	16%	1%	2.0E-02	2.5E-02	6.2E-02	3.4E-02	2.5E-02	2.5E-02
Large Hospital HMI	2.5 km	1.9E-01	3.1E-01	1.3E+00	34%	6%	60%	5.0E-02	6.2E-02	1.5E-01	8.4E-02	6.2E-02	6.2E-02
	10 km	9.4E-02	1.5E-01	6.4E-01	69%	13%	18%	2.5E-02	3.0E-02	7.5E-02	4.1E-02	3.0E-02	3.0E-02
	25 km	8.1E-02	1.3E-01	5.5E-01	80%	15%	5%	2.1E-02	2.6E-02	6.5E-02	3.5E-02	2.6E-02	2.6E-02
Small Hospital HMI	2.5 km	8.5E-02	1.4E-01	5.8E-01	76%	15%	9%	2.2E-02	2.7E-02	6.8E-02	3.7E-02	2.7E-02	2.7E-02
	10 km	7.8E-02	1.3E-01	5.3E-01	83%	16%	1%	2.0E-02	2.5E-02	6.3E-02	3.4E-02	2.5E-02	2.5E-02
	25 km	7.8E-02	1.2E-01	5.3E-01	84%	16%	0%	2.0E-02	2.5E-02	6.2E-02	3.4E-02	2.5E-02	2.5E-02
Large Hospital HMI (wet scrubber)	2.5 km	8.1E-02	1.3E-01	5.5E-01	80%	15%	4%	2.1E-02	2.6E-02	6.5E-02	3.5E-02	2.6E-02	2.6E-02
	10 km	7.8E-02	1.2E-01	5.3E-01	84%	16%	1%	2.0E-02	2.5E-02	6.2E-02	3.4E-02	2.5E-02	2.5E-02
	25 km	7.7E-02	1.2E-01	5.3E-01	84%	16%	0%	2.0E-02	2.5E-02	6.2E-02	3.4E-02	2.5E-02	2.5E-02
Small Hospital HMI (wet scrubber)	2.5 km	7.8E-02	1.2E-01	5.3E-01	84%	16%	0%	2.0E-02	2.5E-02	6.2E-02	3.4E-02	2.5E-02	2.5E-02
	10 km	7.7E-02	1.2E-01	5.3E-01	84%	16%	0%	2.0E-02	2.5E-02	6.2E-02	3.4E-02	2.5E-02	2.5E-02
	25 km	7.7E-02	1.2E-01	5.3E-01	84%	16%	0%	2.0E-02	2.5E-02	6.2E-02	3.4E-02	2.5E-02	2.5E-02
Large Coal-fired Utility Boiler	2.5 km	1.3E-01	2.1E-01	9.1E-01	48%	9%	42%	3.5E-02	4.3E-02	1.1E-01	5.8E-02	4.3E-02	4.3E-02
	10 km	8.6E-02	1.4E-01	5.9E-01	75%	14%	10%	2.3E-02	2.8E-02	6.9E-02	3.8E-02	2.8E-02	2.8E-02
	25 km	8.0E-02	1.3E-01	5.5E-01	81%	15%	4%	2.1E-02	2.6E-02	6.4E-02	3.5E-02	2.6E-02	2.6E-02
Medium Coal-fired Utility Boiler	2.5 km	1.0E-01	1.6E-01	6.9E-01	64%	12%	24%	2.7E-02	3.2E-02	8.1E-02	4.4E-02	3.2E-02	3.2E-02
	10 km	8.3E-02	1.3E-01	5.6E-01	78%	15%	7%	2.2E-02	2.7E-02	6.6E-02	3.6E-02	2.7E-02	2.7E-02
	25 km	8.0E-02	1.3E-01	5.4E-01	81%	16%	3%	2.1E-02	2.6E-02	6.4E-02	3.5E-02	2.6E-02	2.6E-02
Small Coal-fired Utility Boiler	2.5 km	8.3E-02	1.3E-01	5.6E-01	79%	15%	6%	2.2E-02	2.6E-02	6.6E-02	3.6E-02	2.6E-02	2.6E-02
	10 km	7.9E-02	1.3E-01	5.4E-01	82%	16%	2%	2.1E-02	2.5E-02	6.3E-02	3.4E-02	2.5E-02	2.5E-02
	25 km	7.8E-02	1.2E-01	5.3E-01	83%	16%	1%	2.0E-02	2.5E-02	6.2E-02	3.4E-02	2.5E-02	2.5E-02

		MHg Concentration (µg/g)					Predicted MHg Exposure from Ingestion of Fish (mg/kg/day)					
		MHg Dissolved Concentration (ng/L)	Tier3	Tier4	Background	RELMAP	ISC	Bald Eagle	Osprey	Kingfisher	River Otter	Mink	Loon
Medium Oil-fired Utility Boiler	2.5 km	7.8E-02	1.2E-01	5.3E-01	83%	16%	1%	2.0E-02	2.5E-02	6.2E-02	3.4E-02	2.5E-02	2.5E-02
	10 km	7.8E-02	1.2E-01	5.3E-01	84%	16%	0%	2.0E-02	2.5E-02	6.2E-02	3.4E-02	2.5E-02	2.5E-02
	25 km	7.7E-02	1.2E-01	5.3E-01	84%	16%	0%	2.0E-02	2.5E-02	6.2E-02	3.4E-02	2.5E-02	2.5E-02
Chlor-alkali plant	2.5 km	1.0E+00	1.6E+00	6.8E+00	6%	1%	92%	2.6E-01	3.2E-01	8.0E-01	4.4E-01	3.2E-01	3.2E-01
	10 km	1.8E-01	2.8E-01	1.2E+00	37%	7%	56%	4.6E-02	5.7E-02	1.4E-01	7.7E-02	5.7E-02	5.7E-02
	25 km	1.0E-01	1.6E-01	6.8E-01	65%	12%	23%	2.6E-02	3.2E-02	8.0E-02	4.4E-02	3.2E-02	3.2E-02

Table 3-8
Predicted MHg Exposure to Ecological Receptors for Western Site (Local + RELMAP 50th percentile)

		MHg Concentration (µg/g)						Predicted MHg Exposure from Ingestion of Fish (mg/kg/day)					
		MHg Dissolved Concentration (ng/L)	Tier3	Tier4	Background	RELMAP	IS	Bald Eagle	Osprey	Kin gfisher	River Otter	Mink	Loon
Variant b:Large Municipal Waste Combustor	2.5 km	8.8E-02	1.4E-01	6.0E-01	15%	1%	84%	2.3E-02	2.8E-02	7.1E-02	3.8E-02	2.8E-02	2.8E-02
	10 km	5.5E-02	8.8E-02	3.7E-01	24%	2%	74%	1.4E-02	1.8E-02	4.4E-02	2.4E-02	1.8E-02	1.8E-02
	25 km	2.7E-02	4.4E-02	1.9E-01	48%	4%	48%	7.1E-03	8.7E-03	2.2E-02	1.2E-02	8.7E-03	8.7E-03
Variant b:Small Municipal Waste Combustor	2.5 km	3.3E-02	5.3E-02	2.3E-01	40%	3%	57%	8.7E-03	1.1E-02	2.7E-02	1.5E-02	1.1E-02	1.1E-02
	10 km	1.9E-02	3.1E-02	1.3E-01	68%	6%	26%	5.1E-03	6.2E-03	1.5E-02	8.4E-03	6.2E-03	6.2E-03
	25 km	1.6E-02	2.5E-02	1.1E-01	84%	7%	9%	4.1E-03	5.0E-03	1.3E-02	6.8E-03	5.0E-03	5.0E-03
Large Commercial HMI	2.5 km	3.4E-02	5.4E-02	2.3E-01	39%	3%	58%	8.8E-03	1.1E-02	2.7E-02	1.5E-02	1.1E-02	1.1E-02
	10 km	1.7E-02	2.7E-02	1.1E-01	80%	7%	14%	4.3E-03	5.3E-03	1.3E-02	7.2E-03	5.3E-03	5.3E-03
	25 km	1.5E-02	2.4E-02	1.0E-01	89%	8%	3%	3.9E-03	4.7E-03	1.2E-02	6.4E-03	4.7E-03	4.7E-03
Large Hospital HMI	2.5 km	1.4E-01	2.3E-01	9.6E-01	9%	1%	90%	3.7E-02	4.5E-02	1.1E-01	6.1E-02	4.5E-02	4.5E-02
	10 km	3.1E-02	5.0E-02	2.1E-01	42%	4%	54%	8.2E-03	1.0E-02	2.5E-02	1.4E-02	1.0E-02	1.0E-02
	25 km	1.8E-02	2.9E-02	1.2E-01	73%	6%	20%	4.7E-03	5.8E-03	1.4E-02	7.8E-03	5.8E-03	5.8E-03
Small Hospital HMI	2.5 km	2.3E-02	3.6E-02	1.5E-01	58%	5%	37%	6.0E-03	7.3E-03	1.8E-02	9.9E-03	7.3E-03	7.3E-03
	10 km	1.5E-02	2.4E-02	1.0E-01	87%	7%	6%	4.0E-03	4.9E-03	1.2E-02	6.6E-03	4.9E-03	4.9E-03
	25 km	1.4E-02	2.3E-02	9.9E-02	91%	8%	1%	3.8E-03	4.6E-03	1.2E-02	6.3E-03	4.6E-03	4.6E-03
Large Hospital HMI (wet scrubber)	2.5 km	1.8E-02	2.9E-02	1.2E-01	74%	6%	20%	4.7E-03	5.7E-03	1.4E-02	7.8E-03	5.7E-03	5.7E-03
	10 km	1.5E-02	2.4E-02	1.0E-01	90%	8%	3%	3.8E-03	4.7E-03	1.2E-02	6.4E-03	4.7E-03	4.7E-03
	25 km	1.4E-02	2.3E-02	9.8E-02	92%	8%	1%	3.8E-03	4.6E-03	1.2E-02	6.3E-03	4.6E-03	4.6E-03
Small Hospital HMI (wet scrubber)	2.5 km	1.5E-02	2.3E-02	9.9E-02	91%	8%	2%	3.8E-03	4.6E-03	1.2E-02	6.3E-03	4.7E-03	4.6E-03
	10 km	1.4E-02	2.3E-02	9.7E-02	92%	8%	0%	3.7E-03	4.6E-03	1.1E-02	6.2E-03	4.6E-03	4.6E-03
	25 km	1.4E-02	2.3E-02	9.7E-02	92%	8%	0%	3.7E-03	4.6E-03	1.1E-02	6.2E-03	4.6E-03	4.6E-03
Large Coal-fired Utility Boiler	2.5 km	3.1E-02	4.9E-02	2.1E-01	43%	4%	53%	8.0E-03	9.8E-03	2.4E-02	1.3E-02	9.8E-03	9.8E-03
	10 km	1.9E-02	3.0E-02	1.3E-01	70%	6%	24%	4.9E-03	6.0E-03	1.5E-02	8.2E-03	6.1E-03	6.0E-03
	25 km	1.8E-02	2.9E-02	1.2E-01	73%	6%	21%	4.8E-03	5.8E-03	1.5E-02	7.9E-03	5.8E-03	5.8E-03
Medium Coal-fired Utility Boiler	2.5 km	2.3E-02	3.6E-02	1.5E-01	58%	5%	37%	5.9E-03	7.3E-03	1.8E-02	9.9E-03	7.3E-03	7.3E-03
	10 km	2.0E-02	3.2E-02	1.4E-01	66%	6%	28%	5.2E-03	6.4E-03	1.6E-02	8.7E-03	6.4E-03	6.4E-03
	25 km	1.8E-02	2.8E-02	1.2E-01	74%	6%	19%	4.6E-03	5.7E-03	1.4E-02	7.7E-03	5.7E-03	5.7E-03
Small Coal-fired Utility Boiler	2.5 km	1.9E-02	3.0E-02	1.3E-01	70%	6%	24%	4.9E-03	6.0E-03	1.5E-02	8.2E-03	6.1E-03	6.0E-03
	10 km	1.6E-02	2.6E-02	1.1E-01	81%	7%	13%	4.3E-03	5.2E-03	1.3E-02	7.1E-03	5.2E-03	5.2E-03
	25 km	1.5E-02	2.4E-02	1.0E-01	88%	7%	4%	3.9E-03	4.8E-03	1.2E-02	6.5E-03	4.8E-03	4.8E-03

Table 3-8 (continued)
Predicted MHg Exposure to Ecological Receptors for Western Site (Local + RELMAP 50th percentile)

		MHg Concentration (µg/g)						Predicted MHg Exposure from Ingestion of Fish (mg/kg/day)					
		MHg Dissolved Concentration (ng/L)	Tier3	Tier4	Background	RELMAP	IS	Bald Eagle	Osprey	Kin gfisher	River Otter	Mink	Loon
Medium Oil-fired Utility Boiler	2.5 km	1.5E-02	2.3E-02	1.0E-01	90%	8%	2%	3.8E-03	4.7E-03	1.2E-02	6.4E-03	4.7E-03	4.7E-03
	10 km	1.5E-02	2.3E-02	9.9E-02	91%	8%	2%	3.8E-03	4.7E-03	1.2E-02	6.3E-03	4.7E-03	4.7E-03
	25 km	1.4E-02	2.3E-02	9.8E-02	92%	8%	1%	3.8E-03	4.6E-03	1.2E-02	6.3E-03	4.6E-03	4.6E-03
Chlor-alkali plant	2.5 km	1.0E+00	1.6E+00	6.9E+00	1%	0%	99%	2.7E-01	3.3E-01	8.1E-01	4.4E-01	3.3E-01	3.3E-01
	10 km	1.2E-01	1.9E-01	8.0E-01	11%	1%	88%	3.1E-02	3.8E-02	9.5E-02	5.2E-02	3.8E-02	3.8E-02
	25 km	3.7E-02	5.9E-02	2.5E-01	36%	3%	61%	9.7E-03	1.2E-02	3.0E-02	1.6E-02	1.2E-02	1.2E-02

estimates intercompartmental fluxes and resulting concentrations in abiotic and biotic components of the watershed and waterbody. Finally, exposure predictions are modeled as simplified daily average estimates. Seasonal variability among other important exposure factors are not taken into account. Each of these models has parameter inputs that are variable and uncertain. Collectively, these result in uncertainty in the quantitative predictions of the models.

The current scientific understanding of the environmental cycling of mercury (regardless of source) is incomplete. As described in Volume III, areas of uncertainty include emissions speciation, the atmospheric chemistry of emitted mercury, canopy interactions, factors that affect the aquatic mercury cycle (including both the magnitude of effect exhibited by a given factor as well as potential interactions among different factors), and the metabolism of mercury in different piscivorous species.

4. EFFECTS OF MERCURY ON AVIAN AND MAMMALIAN WILDLIFE

Perhaps better than any other metal, mercury illustrates the point that toxicity depends on the chemical species in question. As indicated previously, mercury can exist in an elemental form, as divalent inorganic mercury, or as any one of several organic forms. Of the possible organic forms that may be present in natural systems, methylmercury generally predominates. Both inorganic and methylmercury can accumulate in aquatic biota. However, the proportion of total mercury that exists as the methylated form generally increases with trophic level, often approaching 100% at trophic levels 3 and 4. It is appropriate, therefore, to focus attention on the toxicity of methylmercury to piscivorous avian and mammalian wildlife. A review of mercury toxicity to mammalian systems is provided by Goyer (1993). The toxicity of mercury to birds is reviewed by Scheuhammer (1987). It is not our intention to duplicate these efforts. Instead, a brief summary of methylmercury toxicity to vertebrate systems is presented, with the goal of providing guidance on selection of appropriate toxicological endpoints. This general discussion is followed by brief reviews of several toxicity studies involving avian and mammalian wildlife species (Sections 4.1 and 4.2). Information relating mercury residues in tissues to observed toxic effects is summarized in Section 4.4. Research on selenium/mercury interactions and the activity of endogenous demethylating systems is described in Section 4.5. A single study on the interactive effects of mercury and PCBs on reproduction in mink is reviewed in Section 4.6, emphasizing the point that wild animals are often exposed to multiple chemical stressors.

4.1   Mechanism of Toxicity

Methylmercury in the diet is absorbed with high efficiency in the vertebrate digestive tract and associates rapidly with sulfhydryl-containing molecules in blood, including both free amino acids (primarily cysteine) and glutathione (Carty and Malone, 1979). These mobile complexes transport methylmercury to tissues and organs and may facilitate its movement across cell membranes. In particular, there is good evidence for saturable transport of methylmercury-cysteine complexes across both the blood-brain and placental barriers (Kerper et al., 1992; Kajiwara et al., 1996). Although it exhibits a range of toxic effects in several target tissues, the primary effects of methylmercury are on the central nervous system. Neurotoxicity occurs in both adults and developing animals. In the latter case, this effect appears to be linked to a disturbance of microtubule function in dividing cells, resulting in anti-mitotic activity (Rodier, 1995). The mode-of-action of methylmercury in the differentiated nervous system is less well known, but may involve selective effects on astrocytes and other neuroglial cells (Cranmer et al., 1996).

In chronic toxicity evaluations with mammals, including humans, the most sensitive indicator of toxic effect is cognitive impairment of animals exposed during development (see Volume V of this Report). In general, the sophisticated methods employed in such studies have not been used in toxicological evaluations with wildlife. Instead, less "subtle" endpoints are generally employed, including reduced hatching success and diminished mobility. The work of Heinz with mallard ducklings (Heinz 1976a,b, 1979) represents a notable exception to this general rule (see Section 4.2). For wildlife, therefore, it is difficult to establish whether reproductive or behavioral endpoints are most "sensitive" to methylmercury exposure. Efforts to distinguish between these endpoints are complicated further by the fact that reproductive impacts can occur as a result of direct effects on the developing nervous system, impaired behavior of adults (e.g., unsuccessful matings or diminished quality of parental caregiving), or a combination of both.

4.2   Toxicity Tests with Avian Wildlife Species

Most studies of chronic exposure to birds have been conducted using mercury-contaminated grain. Fimreite (1970) identified a LOAEL of 1.1 μg/g/d for growth inhibition in leghorn cockerel chicks (*Gallus*) based upon 6 μg/g methylmercury dicyandiamide in the feed. Fimreite (1971) also identified a LOAEL of 0.18 μg/g/d for reproductive effects (reduced survival, reduced egg production, and defective shells) in ring-necked pheasant (*Phasianus colchicus*) fed seed treated with methylmercury dicyandiamide. Scott (1977) identified a LOAEL of 4.9 μg/g/d for reproductive effects (reduced fertility, reduced egg number, reduced survival, defective shells) in domestic chickens.

The most comprehensive studies of the effect of mercury on birds were conducted by Heinz (1974, 1975, 1976a,b, 1979). Heinz assessed the effects of dietary methylmercury dicyandiamide (0, 0.5 and 3.0 μg/g as elemental mercury) over three generations of mallard ducks. In the first generation, treatment began in adult ducks. Subsequent generations received treatment beginning at nine days of age. Initially, Heinz (1974) identified a NOAEL of 0.5μg/g based upon reproductive effects in a 21 week study. In a later study (Heinz, 1976a,b), reproduction in first and second generation ducks was evaluated, and the NOAEL for the first generation was again determined to be 0.5μg/g. The second generation, however, suffered adverse reproductive effects including eggs laid outside the nest box ($p<0.05$), reduced number of ducklings surviving to one week of age ($p<0.05$), and reduced growth of ducklings ($p<0.05$) at the 0.5μg/g dose. Consequently, the LOAEL was 0.5μg/g for reproductive effects for the second generation; no NOAEL was identified. A third generation of mallards also demonstrated adverse reproductive effects at 0.5μg/g mercury in the diet. Effects observed included reduced number of viable eggs laid per day ($p<0.01$) and thinner egg shells ($p<0.05$).

Heinz (1975, 1979) also examined behavioral effects of mercury exposure on the approach response of chicks to maternal calls and avoidance of frightening stimuli. In third generation ducklings there was a reduction in response rate and speed of response to maternal calls ($p<0.01$). When data were pooled from all studies and subject to analysis of variance (ANOVA) with multiple comparisons, alterations of behavior were observed in the lowest dose groups in all generations (0.5μg/g). These alterations included reduction in the number of ducklings which approached maternal calls ($p<0.01$) and an increase in the distance traveled to avoid a threatening stimulus ($p<0.05$). In summary, no NOAEL could be determined for behavioral effects, and the NOAEL for reproductive effects could only be demonstrated for the first generation.

For the determination of an appropriate LOAEL in this Report, it was concluded that effects observed in second and third generation ducks at 0.5μg/g should not be discounted. It seems likely that the effects observed in the second and third generations were a result of the earlier onset of dosing. For this reason, 0.5μg/g was selected as a LOAEL for mallard ducks. Assuming a feeding rate of 156 g/kg bw/d for adult mallards, the LOAEL is 78 μg Hg/kg bw/d for reproduction and behavior.

4.3   Toxicity Tests with Mammalian Wildlife Species

River otters (*Lutra canadensis*) fed 2μg/g methylmercury for six months suffered from anorexia and ataxia (O'Connor and Nielson, 1981). In mink, 27μg/g of dietary phenylmercuric chloride caused lethality in 40% of the males and 31% of the females within six weeks of exposure (Borst and Lieshout, 1977).

Wobeser et al. (1976a,b) studied the effects of dietary consumption of methylmercury on ranch mink. There were two parts to this study, which together formed the basis of Wobeser's dissertation research (Wobeser, 1973). In the first part (Wobeser et al., 1976a), 25 adult female mink and their litters were divided into three

groups: Group I contained five females and 19 kits (control); Group II contained 10 females and 34 kits (50% fish diet); and Group III contained 10 females and 29 kits (75% fish diet). The ration was prepared using mercury-contaminated freshwater drum from Lake Winnipeg, Manitoba; mercury in fish tissue was assumed for the purposes of the present analysis to consist primarily of methylmercury. The fish was supplied in a ground, frozen form and was then mixed with cereal and uncontaminated chow to a desired composition of 50 or 75 kg fish/100 kg of food. All mink were fed once daily in slight excess of consumption. The three exposure groups were observed for 145 days. Assuming a food consumption rate of 160 g/kg bw/d (appropriate to captive animals) (Bleavins and Aulerich, 1981) and an average weight of 0.8 kg for the mink, these treatments corresponded to dosing levels of approximately 35 and 55 μg Hg/kg bw/d. One female and 3-6 kits were euthanized every 15 (treatment) or 30 (control) days. Complete necropsies were then performed. No clinical signs of disease were observed in any of the mink within the experimental period, and no mortality or growth impairment occurred which could be attributed to the feeding of mercury-contaminated fish.

In a second experiment (Wobeser et al., 1976b), 30 adult female mink were assigned to one of six groups of five animals each. The animals were fed chow spiked with methylmercuric chloride at 0.0 (control), 1.1, 1.8, 4.8, 8.3, or 15.0 μg/g (by analysis), corresponding to dosing levels of 180, 290, 770, 1330, and 2400 μg/kg bw/d. Two mink from each group were allowed to die of intoxication or were euthanized after 93 days (the end of the experiment). Animals were necropsied and the tissues analyzed for mercury content. All animals in the control group remained clinically normal, and the only clinical sign in the 1.1 μg/g dose group was a slight tendency for two of the animals to move more slowly than the others during the last few days of the experiment. Anorexia, posterior ataxia, and lateral recumbency were observed in the other four dose groups. Death occurred within 26-36 days at 4.8 μg/g and within 19-26 days at 8.3 μg/g. Histopathological abnormalities were seen at 1.1 μg/g, including pale, yellow livers, lesions in the central nervous system, and axonal degeneration.

Based upon a review of the Wobeser studies (Wobeser, 1973; Wobeser et al., 1976a,b), it can be concluded that the LOAEL for subchronic exposure of mink to methylmercury is 180 μg/kg bw/d (1.1 μg/g dose group), using nerve tissue lesions as an effects endpoint. The NOAEL derived from these studies is 55 μg/kg bw/d. Importantly, it was Wobeser's opinion that had the studies been carried out for a longer duration, nervous tissue damage observed in the 1.1 μg/g dose group would have become manifested as impaired motor function.

Charbonneau et al. (1974) fed random-bred domestic cats (*Felis domesticus*) 3, 8.4, 20, 46, 74 or 176 μg/kg/d of mercury, either as methylmercuric chloride in food or as methylmercury-contaminated fish, 7 d/week for 2 years. Clinical examinations of the animals were conducted periodically. Neurological examinations, using a modification of the method of McGrath (1960), were conducted prior to the test, monthly throughout the test, and more frequently as clinical signs of methylmercury toxicosis became apparent. Neurological impairment, including hindrance of the hopping reaction and hypalgesia, was observed in animals exposed to 46, 74, or 176 μg/kg/d, regardless of whether casts were fed contaminated fish or spiked food. No treatment-related effects were observed in three lower dosage groups. Overt signs of toxicity, including ataxia, loss of balance, and motor incoordination, were observed in animals fed 74 or 176 μg/kg/d. These findings suggest that 20 μg/kg/d is the NOAEL and 46 μg/kg/d is the LOAEL for chronic dietary exposure to methylmercury in domestic cats. Charbonneau et al. (1974) also concluded that there was no difference in toxicity or bioavailability between naturally contaminated fish and fish spiked with methylmercuric chloride.

4.4   Tissue Mercury Residues Corresponding to Adverse Effects

Mercury residues associated with toxic effects in birds are reviewed by Scheuhammer (1987). Adult pheasants fed a methylmercury-spiked diet for 12 weeks accumulated liver residues of 2 µg/g but exhibited no discernable adverse effects. However, there was a decrease in hatchability of fertilized eggs due to embryonic mortality and an increase in the number of unfertilized eggs. Unhatched eggs contained 0.5 to 1.5 µg/g as mercury. In a multigenerational study, hen mallards fed methylmercury in the diet accumulated liver residues of approximately 1.5 µg/g without apparent adverse effect (Heinz, 1979). Ducklings born to these hens exhibited behavioral effects including reduced response to maternal calls and hyper-responsiveness to a frightening stimuli. Mercury residues in the eggs from which these ducklings hatched were approximately 0.8 µg/g. Kidney residues considerably higher (>20 µg/g) than those just reviewed were measured at death in mercury-dosed birds of several species (Finley et al., 1979).

Wobeser et al. (1976b) reported that mercury residues in the liver and kidney of mink that died during a 93-day feeding study were 24.3 and 23.1 μg/g, respectively. Somewhat higher values were reported in toxicity studies with mink (55.6 and 37.7 μg/g) by Aulerich et al. (1974) and with otter (39.0 and 33.0 μg/g) by O'Connor and Nielson (1980). Interestingly, mercury residues in tissues from wild animals that are suspected to have died from mercury poisoning are about twice those of animals that died from experimental intoxication (Wren, 1985, 1991). Such discrepancies may be due to kinetic-based differences among exposed animals (see Section 2.3.1.3 of this Volume). Perhaps the most valid comparison that can be made at this time is that between apparently unaffected wild animals and wild animals that have died from mercury poisoning.

4.5   Factors Relevant to the Interpretation and Use of Mercury Toxicity Data

Although several excellent studies of methylmercury toxicity to selected wildlife species have been carried out, the available data are, in general, quite limited, and the extent to which these results can be extrapolated from the laboratory to the field and from one species to another remains in question. Two related issues that may contribute substantially to this uncertainty are singled out for special attention. These are hepatic demethylation as a mechanism for detoxification of methylmercury and the ameliorative effects of dietary selenium.

The protective effect of selenium against methylmercury toxicity to birds has been known for over twenty-five years (Ganther et al., 1972). Koeman et al. (1973) found that mercury and selenium occur in a 1:1 molar ratio in the livers of several marine mammal species. Previously, it had been shown that much of the mercury in the livers in marine mammals existed in an inorganic form. It is now known that these observations are related. Although efforts to elucidate the exact mechanism continue, selenium has been shown to bind mercury after hepatic demethylation of methylmercury. The compounds formed in this manner probably include both mercury-selenoproteins and HgSe (Palmisano et al., 1995; Cavalli and Cardellicchio, 1995).

Thus, it appears that many vertebrate species possess a capability to detoxify and sequester mercury originating as methylmercury in the diet. Moreover, the extent to which this capability is developed appears to be related to an animal's feeding habits and is most highly developed in fish-eating marine mammals and the carnivorous polar bear (Dietz et al., 1990). Correlations between selenium and mercury have also been reported for several seabirds, although the Se/Hg ratio may be higher than 1:1 (Elliott et al., 1992). The capacity of this system to detoxify methylmercury is largely unknown. Variable detoxification among individuals of a single species (pilot whales) has been demonstrated; lactating females demonstrated a significantly diminished detoxifying capability (Caurant et al., 1996).

The demethylating capabilities of birds and mammals that inhabit terrestrial and freshwater ecosystems are less well known. Methylmercury constituted 46% of total mercury in the livers of mink fed a diet of methylmercury-contaminated fish (Jernelöv et al., 1976). There was no obvious relationship between levels of liver mercury and selenium. Similar values were reported by Wren et al. (1986) for mink (53%) and otter (34%). Barr (1986) found that methylmercury comprised 4-27% of total mercury in livers from loons taken from mercury-contaminated waters in northwestern Ontario. Selenium concentrations were not measured. Interestingly, the percentage of methylmercury did not vary with the gradient of site contamination, as might be expected if the demethylating system was saturated at particularly high exposure levels. A positive correlation between liver mercury and selenium was reported in the goldeneye, but no attempt was made to identify mercury species (Eriksson et al., 1989). Although limited to a single study, evidence suggests that demethylation of methylmercury also occurs in some birds of prey (Norheim and Forslic, 1978).

Additional evidence that this detoxifying pathway is related to animal feeding habits is provided by Fimreite (1974). Among adult ducks, fish-eating mergansers exhibited the lowest levels of methylmercury as a percent of total (12% in the liver). Methylmercury constituted 32%, 38% and 52% of total mercury in the livers of goldeneyes, mallards and pintails. Moreover, this detoxifying ability appears to develop early in life. Methylmercury levels as a percent of total in livers taken from ducklings were 27%, 49%, 53% and 58% in the merganser, mallard, goldeneye and pintail. Methylmercury levels in breast muscle from all four species as a percent of total were essentially identical, averaging about 60%.

The protective effect of selenium against mercury toxicosis may vary with lifestage and the chemical form of selenium. Selenium as selenomethionine (10μg/g) protected adult male mallards against the toxic effects of methylmercury (10μg/g) in the diet. However, a combination of these treatments in hen mallards resulted in adverse reproductive effects greater than those seen with mercury or selenium alone. These effects included reduced hatching success and survival of ducklings, including an increase in teratogenic impacts (Heinz and Hoffman, 1996). Methylmercury in the diet greatly increased selenium storage in tissues. The livers of male mallards fed only selenium contained 9.6μg/g selenium, whereas in mallards fed both selenium and methylmercury, the livers contained an average of 114μg/g selenium. This observation is important because high concentrations of selenium are known to produce teratogenic effects in wild birds (Ohlendorf et al., 1986). The ecological significance of these findings remains to be determined. Data summarized above suggest that, among duck species, mallards possess less capability to detoxify methylmercury than piscivorous mergansers and goldeneyes. In addition, the levels of mercury and selenium employed in this study are well above those known to cause toxic effects when applied separately.

To summarize, many, if not most, birds and mammals possess a capability to detoxify methylmercury, and the activity of this system appears to be related to an animal's feeding habits. This conclusion is significant for at least two reasons: (1) the toxicity of methylmercury to birds and mammals may be highly dependent upon the availability of dietary selenium and (2) most toxicity tests with birds conducted to date have been carried out using non-piscivorous species that may not possess a well-developed demethylating capability.

4.6　Combined Effects of Mercury and Other Chemical Stressors

In most aquatic systems mercury is but one of many potential chemical stressors. Using current assessment methods, there is a general tendency to evaluate the toxic potential of compounds applied individually. A notable exception is the use of toxic equivalency factors (TEQs) to predict the combined impact of compounds that act through an Ah receptor-mediated mode of action (PCBs, dioxins). Applying this approach to a mixture of mercury and PCBs would be difficult, however, due to differences in chemical modes of action.

It is of interest, therefore, to note that the effects of PCBs and methylmercury, singly and in combination, have been evaluated in mink (Wren et al., 1987a,b). Growth and survival of kits were reduced by a combined exposure to PCBs (Arochlor® 1254) and methylmercury at concentrations that individually produced no response. The authors of these studies described this outcome as a "synergistic effect." Given the limited number of dose levels (0.0, 0.5 and 1.0 µg/g), however, it would be difficult to rule out an additive response.

5. ASSESSMENT OF THE RISK POSED BY AIRBORNE MERCURY EMISSIONS TO PISCIVOROUS AVIAN AND MAMMALIAN WILDLIFE

5.1   Scope of the Assessment

As described in Chapter 2 of this Volume, mercury bioconcentrates, bioaccumulates and biomagnifies in aquatic food chains. These processes result in mercury residues in fish that are much higher than concentrations in the water in which they live, thereby providing an enriched contaminant source for piscivorous avian and mammalian wildlife. Existing data permit a general treatment of mercury exposure and effects on such populations. A more accurate assessment of the risk posed by mercury to a specific group of animals occupying a given location requires the collection of necessary supporting information such as food habits, migratory behavior, breeding biology, and mercury residues in preferred prey items.

A general summary of ecological risk assessment methods is provided by U.S. EPA (1996) in its Proposed Guidelines for Ecological Risk Assessment. The data needs of these methods vary widely and dictate to a considerable degree which methods can be applied to a given situation. Guidance is provided in Section 5.2 on the risk assessment methods that may be most applicable to airborne mercury emissions, given the nature and extent of currently existing information. Additional guidance is provided in Section 5.3 based on a review of published assessments for piscivorous species living in the Great Lakes region, south Florida, central Ontario, and coastal regions of Georgia, South Carolina and North Carolina.

The scope of the present Report was intended to be national in scale. It was determined, therefore, that any effort to assess the risk of mercury to a given species living in a defined location would be inappropriate. Instead, an effort was made to compare mercury exposure and effects in a general way using data collected from throughout the country and in so doing to develop qualitative statements about risk.

Consistent with this broader-scale approach, an effort is made in Section 5.4 to derive a wildlife criterion level (WC) for mercury that is protective of piscivorous wildlife. This WC is defined as the concentration of mercury in water that, if not exceeded, protects avian and mammalian wildlife populations from adverse effects resulting from ingestion of surface waters and from ingestion of aquatic life taken from these surface waters. The health of wildlife populations may, therefore, be considered the assessment endpoint of concern. Although not generally derived for the purpose of ecological risk assessment, WC values incorporate the same type of exposure and effects information used in more standard approaches. Such calculations also provide for a simple assessment of risk in any given situation, i.e., by determining whether the concentration of mercury in water exceeds the criterion value.

Calculation of a WC for mercury is based upon the use of a wildlife reference dose approach, combined with knowledge of the extent to which mercury becomes concentrated in aquatic food chains. The methods used to calculate this criterion value are based on those described in the Proposed Great Lakes Water Quality Guidance for the Great Lakes Water Quality Initiative (U.S. EPA, 1993c) and implemented in the final Water Quality Guidance for the Great Lakes System (U.S. EPA, 1995b), henceforth referred to as the "Proposed Guidance" and "Final Guidance," respectively. When originally implemented in support of the Great Lakes Water Quality Initiative (GLWQI), this approach yielded a single measurement endpoint, which was the total mercury concentration in water that was believed to be protective of piscivorous wildlife. In the present assessment, an effort is made to update the WC for mercury by calculating its value using data for methylmercury. It should be noted that a methylmercury-based WC can still be related to total mercury residues

in fish or water through the use of appropriate conversion factors. By convention, mercury concentrations in environmental media (and in dosing solutions) are usually expressed as $\mu g/g$ of elemental mercury, regardless of the identity of the mercury species. This convention is retained throughout the present analysis.

Methylmercury BAFs for trophic levels 3 and 4 (forage fish and larger, piscivorous fish, respectively) are estimated in Appendix D of Volume III. This information is summarized in Section 5.4.2 of the present Volume. It is recognized that there is considerable natural variability with respect to the accumulation of mercury in aquatic food chains, which contributes in turn to variability in trophic relationships and BAFs. In addition, there is a lack of understanding of fundamental processes that contribute to methylation of mercury and subsequent bioaccumulation in aquatic organisms. Additional uncertainty derives from ongoing improvements in sampling technique and analytical methodology. A review of uncertainties associated with the derivation of WC values is provided in Section 5.4.11. In general, the same uncertainties apply to any risk assessment effort for mercury in wildlife.

Tempering these uncertainties is a large and growing volume of both laboratory and field data for mercury. From the perspective of WC development, field data are of particular interest. The GLWQI stipulates that when sufficient field data are available, field-derived BAFs should take precedence over values estimated from laboratory studies or by employing empirical relationships (e.g., correlation with chemical hydrophobicity). The focus of the BAF analysis in this Volume is on incorporating recent field data into the revised GLWQI approach. The results of this effort are summarized in Section 5.4.2.3.

5.2   Summary of Relevant Risk Assessment Methodologies

Perhaps the most comprehensive type of risk assessment that can be attempted is a comparison of statistical distributions of exposure and effects information. In essence, risk is determined from the degree of overlap of these distributions. Linearization of the effects and exposure distributions simplifies such comparisons. This is generally accomplished by log transformation of the cumulative exposure and effects distributions (U.S. EPA, 1996; SETAC, 1994). A particularly good example of such an assessment is provided by Solomon et al. (1996) for atrazine in aquatic systems.

The data requirements of such an approach are extensive. Moreover, it is critically important that effects information be collected under conditions that are comparable to the exposure data. For this reason, the approach is most easily applied in circumstances where the effects are expressed after a relatively short period of exposure and the compound of interest does not bioaccumulate. Both of these criteria are satisfied for a compound like atrazine.

Mercury presents a far greater challenge by virtue of the fact that it bioaccumulates for extended periods of time and because toxic effects occur only after sufficient body residues are attained. Moreover, the limited data collected to date permit the characterization of a dose-response curve for only three or four wildlife species.

A more feasible approach to assessing chemical risk to wildlife species involves the comparison of a point estimate of effect with a statistical distribution of exposure (U.S. EPA, 1996). The data needs of this approach include one or a few toxicity studies from which an appropriate toxicity endpoint can be determined and sufficient exposure data to define the distribution. In the simplest application of this approach for a compound such as mercury (for which the diet is the primary route of uptake), exposure would be expressed as a residue concentration in prey. Risk would then be characterized as the probability that exposure (prey concentration) would exceed a given effect level. Alternatively, exposure can be characterized as a contact rate

(mass of compound consumed/kg bw/d). Although more data intensive, this latter approach is preferred because it better reflects the long-term nature of the exposure.

An even simpler approach to wildlife risk assessment expresses risk as the ratio of exposure and effects point estimates. Often referred to as the "hazard quotient" method, this approach is by far the most commonly used of all current techniques. It may also be the most intuitive, since risk is inferred by the simple fact of a ratio approaching or exceeding 1.0. The disadvantage of this approach is that is does not permit a probabilistic assessment of risk. Moreover, because this approach is generally used when more detailed data are lacking, risk assessors often adjust the effect level downward using one or more "safety factors."

In the following Section, several published efforts to assess the risk of mercury to wildlife are reviewed. These efforts illustrate the point that while information needed to perform such assessments are extremely limited, effects information are in general more limited than exposure data.

5.3 Review of Published Efforts to Estimate the Risk of Mercury to Wildlife

5.3.1 Risk of Mercury to Bald Eagles in the Great Lakes Region

Bowerman et al. (1994) compared feather mercury data with measures of reproductive performance to evaluate the risk of mercury to bald eagles in the Great Lakes Region. Although no attempt was made to develop a quantitative estimate of risk, it was determined that there was no association between mercury residues in feathers and either productivity or nesting success. On this basis, it was concluded that mercury was not affecting bald eagle reproduction. A conclusion of this type may be characterized as a qualitative statement of risk.

5.3.2 Risk of Mercury to Bald Eagles in Michigan

Giesy et al. (1995) used a hazard quotient approach to characterize the risk to bald eagles posed by mercury and several organic compounds at locations above and below dams on three Michigan rivers. An exposure point estimate for mercury was calculated from measured concentrations in fish and an egg:fish biomagnification factor (set equal to 1.0). Hazard quotients ranging from 0.15 to 0.98 were calculated for mercury at study sites on the three rivers. The highest quotients were calculated for sites above the dams due to the presence of higher mercury levels in fish. The authors concluded that mercury does not pose a significant threat to eagles living in this region. This conclusion was based upon the opinion that the NOAEC level used in the analysis (0.5 µg mercury/g egg) was conservative, as well as the suggestion that eagles consume only small quantities of the most contaminated fish species (yellow perch and walleye) living in these rivers. Hazard quotients for PCBs and TCDD (equivalents) were much greater than 1.0 (ranging from 7.6 to 76) at all sites downstream from the dams.

5.3.3 Risk of Mercury to Loons in Central Ontario

Scheuhammer and Blancher (1994) assessed the risk of mercury to loons by comparing residues in fish collected from central Ontario lakes with a threshold value for reproductive impairment. A strength of this assessment is that the toxic effects point estimate was also determined in a study of wild loons (Barr, 1986). The fish selected for this analysis were of a size appropriate to predation by loons. Care was also taken to survey lakes of the type preferred by breeding loons. Among the lakes surveyed, up to 30% contained fish which exceeded the toxicity threshold, depending upon the species of fish chosen.

5.3.4   Risk of Mercury to Mink in Georgia, North Carolina, and South Carolina

Osowski et al. (1995) assessed the risk of mercury, PCBs and several chlorinated organic pesticides to mink in the coastal regions of southeastern U.S. The risk associated with mercury was determined by comparing residue levels in kidney tissue with levels that had been associated previously with toxic effects. Unfortunately, the threshold effect level (tissue residue) was not given. It is difficult, therefore, to critically evaluate the author's conclusion that residues "were in the range of those known to cause impacts to reproduction, growth, and behavior in wild mink."

5.3.5   Risk of Mercury to Mink in Michigan

A second assessment for mink was conducted by Giesy et al. (1994) for animals living on three rivers in lower Michigan. In this assessment, an effort was made to calculate a hazard quotient using published toxicity data for mink (Wobeser, 1976a,b) and measured residues in fish collected from the study sites. Interestingly, hazard quotients greater than 1.0 were calculated at all three sites (range of 1.2-6.6). However, the significance of this finding was minimized because hazard quotients calculated for PCBs and TCDD-like compounds tended to be higher. In this regard, it is of interest to note previous studies in which mercury and PCBs appeared to act "synergistically" in toxicity studies with mink (see Section 4.6 of this volume).

5.3.6   Risk of Mercury to Great Egrets in south Florida

Sundlof et al. (1994) reported on another researcher's use of the hazard quotient method to assess the risk of mercury to great egrets in south Florida. The actual assessment was conducted as part of a Masters degree research program (Jurczyk, 1993). For this assessment, a published LOAEL for reproductive effects in loons (Scheuhammer, 1991) was compared to a methylmercury consumption rate calculated using measured residues in local fish and shellfish. Based upon this analysis, it was concluded that great egrets were consuming 3.9 times the LOAEL, thus placing the population at risk.

5.4   Calculation of a Criterion Value for Protection of Piscivorous Wildlife

5.4.1   Procedure Used to Develop Criterion Values for Wildlife in the Water Quality Guidance for the Great Lakes System

The WC for mercury is defined as the concentration of total mercury in surface water that, if not exceeded, protects both avian and mammalian wildlife that use the water as a drinking or foraging source. Thus, the WC is the highest aqueous concentration of mercury that causes no significant reduction in growth, reproduction, viability or usefulness (in a commercial or recreational sense) of a population of animals exposed over multiple generations. For the purpose of this analysis, the term "aqueous concentration" refers to the concentration of methylmercury in filtered water, including both the freely dissolved form and methylmercury that is associated with dissolved organic material.

The equation used in this analysis to calculate a WC for mercury is identical to that described in the Proposed Guidance (U.S. EPA, 1993c) and implemented in the final Water Quality Guidance for the Great Lakes System (U.S. EPA, 1995b):

$$WC = \frac{(TD \ x \ [1/UF]) \ x \ Wt_A}{W_A \ + \ [(FD_3)(F_A \ x \ BAF_3) \ + \ (FD_4)(F_A \ x \ BAF_4)]}$$

where

WC	=	wildlife criterion value (pg/L; after converting from μg/L)
Wt_A	=	average species weight (g)
W_A	=	average daily volume of water consumed (L/d)
F_A	=	average daily amount of food consumed (g/d)
FD_3	=	fraction of the diet derived from trophic level 3
FD_4	=	fraction of the diet derived from trophic level 4
BAF_3	=	aquatic life bioaccumulation factor for trophic level 3 (L/g; methylmercury concentration in fish/methylmercury in water)
BAF_4	=	aquatic life bioaccumulation factor for trophic level 4 (L/g; methylmercury concentration in fish/methylmercury in water)
TD	=	tested dose (μg/g bw/d)
UF	=	uncertainty factor

A similar equation was first used by the State of Wisconsin to set Wild and Domestic Animal Criteria (State of Wisconsin, 1989). The entire approach, including both the equation and data requirements for its parameterization, was later modified by U.S. EPA for incorporation into the Proposed Guidance (U.S. EPA, 1993c) and Final Guidance (U.S. EPA, 1995b). The method, in its current form, was reviewed in 1992 at a workshop entitled "The National Wildlife Criteria Methodologies Meeting," which was sponsored by U.S. EPA (U.S. EPA, 1994). Subsequently, the method was used to develop interim Tier I WC for four compounds (PCBs, DDT, dieldrin, and mercury) in the Great Lakes Basin (U.S. EPA, 1993b). These criteria have received public comment. The method has been reviewed by EPA's Science Advisory Board on two occasions, most recently in June of 1994. Detailed descriptions of the method, including comparisons with other proposed methods for setting wildlife criterion values, are given elsewhere (U.S. EPA 1993c, 1994).

An examination of the GLWQI equation reveals both a hazard and an exposure component. The equation includes a term TD for "tested dose." In this Report, data were reviewed to determine an appropriate NOAEL, which was used for the TD. In the absence of a NOAEL, a LOAEL was used with the addition of an appropriate factor (UF_L) to indicate uncertainty around the toxic threshold. An uncertainty factor (UF_A) also may be used to provide a margin of safety when applying data from a species other than the species of concern. A third uncertainty factor (UF_S) may be used to extrapolate from subchronic to chronic exposures. Additional adjustments may be warranted by toxicokinetic or toxicodynamic considerations. Collectively, the application of the UF to the TD results in the estimation of a "reference dose" (RfD) for subsequent calculation of the WC.

The WC for mercury derived in support of the GLWQI was expressed as the total mercury concentration in filtered water. Although it was recognized at the time that methylmercury is the form of mercury that

bioaccumulates in fish, few laboratories possessed the analytical capability to speciate mercury in water from natural sources.

A WC for mercury was calculated in the Proposed Guidance using fixed values for all parameters in the equation. Species-specific WC values (WC_s) were calculated for each of the wildlife species of concern (eagle, herring gull, kingfisher, mink, otter). Intermediate WC values (WC_i) were then obtained for avian and mammalian wildlife by calculating the geometric mean of values for contributing species. The final WC (WC_f) was set equal to the lowest of the two resulting intermediate values and, for mercury, was driven by the calculations for avian species.

The WC_f for mercury derived in the Proposed Guidance is 1300 pg/L. A comparison of the GLWQI criteria for birds and mammals with those derived in this Report is presented in Section 5.4.8 of this Volume.

For the present analysis, a decision was made to consider all but one of the wildlife species considered in the Proposed Guidance. Herring gulls, which are indigenous to the Great Lakes region, are not evaluated in this Report. The herring gull was replaced in the present analysis by the common loon (*Gavia immer*). The other avian wildlife for which WC values are calculated are the bald eagle (*Haliaeetus leucocephalus*), osprey (*Pandion haliaetus*) and belted kingfisher (*Ceryle alcyon*). The mammalian wildlife for which WC are calculated are the mink (*Mustela vison*) and river otter (*Lutra canadensis*). Each of these species was originally selected after consideration of the following: (1) their exposure to bioaccumulative contaminants; (2) their relevance to Great Lakes ecosystems; (3) the availability of information with which to calculate criterion values; and (4) the evidence for accumulation and/or adverse effects.

Several other wildlife species would satisfy most or all of the selection criteria presented in the GLWQI. Notable examples include the double-crested cormorant (*Phalacrocorax auritus*), Forster's tern (*Sterna forsteri*), wood stork (*Mycteria americana*), raccoon (*Procyon lotor*), snapping turtle (*Chelydra serpentina*), and American alligator (*Alligator mississippiensis*). Exposure factors for a large number of wildlife species are available in a recently published handbook (U.S. EPA, 1993a). A critical evaluation of these data as they pertain to the development of WC is also available (U.S. EPA, 1995a). Allometric equations may also be used to calculate both feeding and drinking requirements (see for example Calder and Braun, 1983; Nagy, 1987). In time, the inclusion of other species, including both amphibians and reptiles, may be appropriate, particularly if an effort is made to calculate WC on a regional basis or if the species used in the present analysis are not representative of the ecosystem of concern. The present analysis is intended, however, to be national in scope. Each of the species selected for this analysis is distributed over large portions of the country (see species distributions in Section 3.3 of this Volume), and in these locations each species is closely tied to water resources via aquatic food chains.

Finally, this analysis differs from that of the GLWQI insofar as WC values are calculated on a "dissolved" (freely dissolved and associated with DOC) methylmercury basis. A review of literature collected over the last several years suggests that there is now sufficient information available to estimate BAFs for mercury on a methylmercury basis. Previously, it was thought that much of the variation around BAFs estimated on a total mercury basis could be attributed to differences among water bodies in the proportion of total mercury existing as the methylated form. The goal of the present analysis was to calculate a WC for the bioaccumulating form of mercury, thereby yielding an estimate with the lowest possible variation around the mean.

5.4.2   Bioaccumulation Factors (BAFs) for Magnification of Methylmercury in Aquatic Food Chains
        5.4.2.1 Definition of BAFs and Overview

        The bioaccumulation factor (BAF) for any given trophic level is defined as the ratio of methylmercury concentration in fish flesh divided by the concentration of dissolved methylmercury in the water column. The BAF represents the accumulation of mercury in fish of a specific trophic level from both direct uptake from water and predation on contaminated organisms. The BAF is a principal input variable in the GAS ISC3 exposure model used in Volume III of this Report to link estimates of mercury deposition to exposure levels for fish-consuming species.

        In this Report, BAFs are estimated for trophic level 3 (foraging fish) and trophic level 4 (piscivorous fish), which are designated as BAF_3 and BAF_4, respectively. BAF_4 is estimated by three different methods and BAF_3 is estimated by two different methods. The result, or output, of each estimation method is a distribution of BAF values, each associated with some degree of likelihood. The three methods by which BAF_4 is estimated are: a modified GLWQI method, a $BAF \times PPF$ method, and a direct field-derived method from measured BAFs at trophic level 4. BAF_3 is estimated by the modified GLWQI method and directly from measured BAFs at trophic level 3. These methods are summarized in Section 5.4.2.2 of this Volume and described in detail in Appendix D to Volume III (Appendix D also describes two BAF approaches for total mercury). BAF_4 is intended to be representative of the random selection of a trophic level 4 fish from a random lake in a random geographical location. It is meant to be used to estimate the concentration of methylmercury in such a randomly-selected fish when multiplied by the dissolved methylmercury concentration. BAF_3 performs the same function for trophic level 3 fish.

        The general approach used in this analysis was based on probabilistic methods, as described in Appendix D to Volume III. This approach was taken to allow quantitative expression of the overall variability surrounding the various estimates of the BAFs and to determine the relative sensitivity of the estimates to specific individual variables.

        5.4.2.2 BAF Estimation Methods

        Modified GLWQI Method

        The GLWQI method is essentially the same as that in the Proposed Guidance (U.S. EPA, 1993c), modified to consider only methylmercury, and based entirely on field-derived BCFs and PPFs. The formula is given in equation 1.

$$BAF_n = BCF \times FCM_n \qquad\qquad (1)$$

where

        n   is the trophic level for which the BAF is estimated,

        BCF   is the weighted-average bioconcentration factor (BCF) for dissolved methylmercury at
                    trophic level 1, and

        FCM_n is the food-chain multiplier representing the cumulative biomagnification of
                    methylmercury from trophic level 2 to trophic level n, n=[3,4].

The formulas for FCM_3 and FCM_4 are given in equations 2 and 3, respectively.

$$FCM_3 = PPF_2 \times PPF_3 \tag{2}$$

$$FCM_4 = PPF_2 \times PPF_3 \times PPF_4 \tag{3}$$

where

> PPF_2 is the predator-prey factor at trophic level 2 representing the biomagnification of methylmercury in zooplankton as a result of feeding on contaminated phytoplankton,

> PPF_3 is the same for trophic level 3 fish feeding on contaminated organisms, and

> PPF_4 is the same for trophic level 4 fish feeding on trophic level 3 fish.

Distributions were assigned to each of the variables in equations 1-3 based on data available in the published literature. The basis and description of the distribution for each variable are described in Appendix D of Volume III. The nominal values for some of the variables are not the same as presented in the Proposed Guidance (U.S. EPA, 1993c) due to differing assumptions and approaches to data analysis.

BAF × PPF Method

The formula for the calculation of BAF_4 by this method is given in equation 4.

$$BAF_4 = BAF_3 \times PPF \tag{4}$$

where

> BAF_3 is the field-measurement-derived distribution for the BAF at trophic level 3 and

> PPF_4 is the same as for the GLWQI method.

Field-derived (Direct) Method

This method estimates BAF_3 and BAF_4 directly from measurements of BAFs in field studies. The derivation of the BAF distributions is described in Appendix D of Volume III.

5.4.2.3 Results of BAF Simulations and Recommended Values

Results of the probabilistic simulations for each of the methods are given in Table 5-1, which shows representative statistics for each BAF output distribution. All of the statistics are given as the geometric equivalents (antilogs) of the actual values generated by the simulations. There is a large variance in the distributions, which cannot be separated into variability in BAFs and uncertainty in their estimation.

Table 5-1
Summary of Methylmercury Bioaccumulation Factors for Trophic Levels 3 and 4
(mean, 5%, and 95% values)

	BAF_3		BAF_4		
Recommended	1,600,000		6,800,000		
Method	Direct Field-derived	GLWQI	BAF_3 x PPF_4	Direct Field-derived	GLWQI
Median (GM[a])	1,600,000	1,300,000	7,820,000	6,800,000	6,500,000
5th pctl	461,000	71,500	1,960,000	3,260,000	331,000
95th pctl	5,410,000	2,440,000	31,100,000	14,200,000	129,000,000
GSD[b]	2.12	5.88	2.32	1.56	6.13

[a] Geometric Mean
[b] Geometric Standard Deviation

The recommended BAFs are those developed from field data at each trophic level. Values estimated using the GLWQI methodology are similar in each case to those estimated from field data but show much greater variability. This greater variability is not surprising given the greater number of variables and paucity of data for the GLWQI approach (see Appendix D of Volume III). Only four field-derived data points were available to characterize the BAF_3 and BAF_4 distributions. In each case, however, these data points were in relatively good agreement, resulting in narrower statistical distributions that those associated with the GLWQI and BAF_3 x PPF_4 approaches.

The GLWQI stipulates that when high quality field data are available, BAFs developed from these data should take precedence over values estimated using laboratory data. At the time of its development, the field data needed to estimate BAFs for the GLWQI were not available. Recently collected field data are thought to be sufficient to generate accurate estimates of mean BAFs for trophic levels 3 and 4. Confidence in estimates of the geometric standard deviations is lower. Additional data from a broader array of ecosystem types are needed to better characterize the shapes of these distributions.

5.4.2.4 Sensitivity Analysis

A limited sensitivity analysis was conducted to examine the influence of distribution form on the BAFs estimated by the direct field-derived method. The analysis investigated the impact on the output of assuming the BAFs were distributed normally rather than lognormally. The difference in the two assumptions was small, with slightly higher median estimates for the normal distributions and slightly higher upper percentiles for the lognormal. The empirical data more closely matched the lognormal form. This analysis is presented in Appendix D of Volume III.

5.4.2.5 Uncertainty and Variability

Generally, in the representation of the input and output distributions, there are no distinctions as to size or species of fish, location or type of lake (eutrophic or oligotrophic), water column pH, or absolute mercury concentrations (in fish or water). The available data are insufficient to make these distinctions. Field data are heavily biased towards northern (oligotrophic) lakes and somewhat towards smaller (younger) fish.

There is no distinction between variability and uncertainty in the BAF_4 distributions. That is, the variability in the output distributions reflects both naturally variable processes and the uncertainty around those processes. For example, the BAF_4 distributions include variability in the BAF associated with variations in fish size combined with measurement uncertainties.

Perhaps the greatest source of variability is that of model uncertainty; i.e., uncertainty introduced by failure of the model to account for significant real-world processes. In lake surveys conducted within a relatively restricted geographic region, large differences can exist between lakes with respect to mercury concentrations in a given species of fish (see for example Cope et al., 1990; Grieb et al., 1990; Sorenson et al., 1990; Jackson, 1991; Lange et al., 1993). Although much of this variability can be attributed to local biogeochemical processes that determine the percentage of total mercury that exists as the methylated form, additional sources of variability undoubtedly exist. In addition, it has been repeatedly shown that mercury in fish accumulates throughout the lifetime of the individual (Scott and Armstrong, 1972; MacCrimmon et al., 1983; Wren et al., 1983; Mathers and Johansen, 1985; Skurdal et al., 1985, Wren and MacCrimmon, 1986; Sorenson et al., 1990; Jackson, 1991; Gutenmann et al., 1992; Glass et al., 1993, Suchanek et al., 1993; Lange et al., 1993). Reported BAF values for a given species may, therefore, vary as a function of the ages of the animals examined. As a result, some researchers have suggested that comparisons between lakes should be made using "standardized" fish values (e.g., a value for a hypothetical 1 kg northern pike), typically derived by linear regression of residue data collected from individuals of varying size and/or age (Wren and MacCrimmon, 1986; Sorenson et al., 1990; Meili et al., 1991). An additional source of variability is seasonal variation of dissolved methylmercury in the water column. While the concentration of methylmercury in fish flesh is presumably a function of the varying water concentration, specific values for BAF_4 and BAF_3 are generally calculated from single representative values.

5.4.2.6 Conclusions

BAFs derived from adequate data collected at a site of concern should be used in lieu of the estimated values presented in this Report. The criteria for defining the adequacy of data are discussed in the Data Quality Objectives section of Appendix D in Volume III. When such values are not available, the use of the geometric mean values from the BAF_3 and BAF_4 output distributions generated from the direct field-derived distributions is the recommended approach. Use of the geometric mean, rather than the arithmetic mean, is a consequence of the assumption that BAFs are distributed in nature as the logarithm of the observed value. The recommended approach is more direct and less variable than the GLWQI method and involves fewer assumptions. The recommendation as to the use of the (geometric) mean value of these distributions is based on the inability to distinguish among various sources of uncertainty and variability in the output distributions, with consequent problems of interpretation of specific percentiles. Because the exposure concern is for repeated ingestion of contaminated fish, the mean, rather than the median, is the appropriate value. The median is only useful when the concern is the random selection of a single fish.

Reducing the uncertainty in the BAFs generated by these methods will require the collection of more data representative of the critical factors underlying the observed variability and the inclusion of additional terms to explicitly model those factors. For example, the inclusion of an age/size regression term would account for a substantial portion of the variability in both BAF_4 and PPF_4.

5.4.3   Exposure Parameters

Exposure parameters for the present analysis are shown in Table 5-2. The scientific basis for parameters that apply to the mink, otter, kingfisher, osprey and eagle is reviewed elsewhere (U.S. EPA 1993a, 1995a). The weight of loons was calculated as the average of values reported by Barr (1986) for adult males and females, and the feeding rate was taken from Barr (1973). Data provided by Barr (1996) suggest that, when given the opportunity, loons feed almost exclusively on live fish and that these fish belong almost exclusively to trophic level 3.

Table 5-2
Exposure Parameters for Mink, Otter, Kingfisher, Osprey, and Eagle

Species	Body Wt. (WtA) kg	Ingestion Rate (F_A) kg/d	Drinking Rate (W_A) L/d	Trophic Level of Wildlife Food Source	% Diet at Each Trophic Level
Mink	0.80	0.178	0.081	3	90
Otter	7.40	1.220	0.600	3,4	80,20
Kingfisher	0.15	0.075	0.017	3	100
Loon	4.00	0.800	0.120	3	100
Osprey	1.50	0.300	0.077	3	100
Eagle	4.60	0.500	0.160	3,4	74,18

For this analysis, it was assumed that prey not attributed to trophic levels 3 and 4 were derived from non-aquatic origins and do not contain mercury. Were these prey to contain mercury, WC values calculated for the relevant species would decrease. BAFs for trophic levels 3 and 4 were assigned the values recommended in Section 5.4.2.3 of this Volume.

5.4.4   Summary of Health Endpoints for Avian and Mammalian Wildlife

The avian chronic TD value was derived from studies by Heinz (1975, 1976a,b, 1979) in which three generations of mallard ducks (*Anas platyrhynchos*) were dosed with methylmercury dicyandiamide (0, 0.5 and 3.0 ppm) (see Section 4 of this Volume). The lowest dose, 0.5 ppm (78 μg/kg bw/d), resulted in adverse effects on reproduction and behavior and was designated as a chronic LOAEL. As no NOAEL was reported, a UF_L of 3 was used according to methodology described in U.S. EPA (1995b). In a departure from the GLWQI, a decision was made not to adjust this value further using a species-to-species uncertainty factor (UF_A) greater than 1.0. Although no toxicity data are available for any of the bird species of interest, a review of the literature suggests

that piscivorous birds possess a greater capability to detoxify methylmercury than do non-piscivorous birds (see Section 4 of this volume). Adjusting the TD for mallards even lower is, therefore, unjustified.

The mammalian chronic NOAEL was derived from studies of subchronic exposure by Wobeser (1973, 1976a,b) in which mink were dosed with mercury in the form of mercury-contaminated fish (0.22 and 0.33 ppm, naturally incorporated into fish; 1.1, 1.8, 4.8, 8.3 and 15.0 ppm, spiked into the diet). Effects observed include histopathologic lesions in nerve tissue at 1.1 ppm and higher doses. Anorexia, ataxia and death occurred at 1.8 ppm and higher doses. The dose of 0.33 ppm (55 μg/kg bw/d) was selected as the NOAEL for subchronic exposure. As this was a less than lifetime study, a UF_S of 3 was applied to the TD or NOAEL. The value of this uncertainty factor is less than the value employed in the GLWQI (10). However, the authors of the GLWQI also identified 1.1 ppm as the NOAEL, whereas this analysis considers the histopathological lesions seen in the 1.1 ppm dose group an adverse toxic effect. The subchronic NOAEL/UF_S is 18.3 μg/kg bw/d, which is approximately equal to the chronic NOAEL (20 μg/kg bw/d) estimated from long-term feeding studies with domestic cats (Charbonneau et al., 1974).

Based on the information above, the TDs used for calculation of a WC for mercury were:

For avian wildlife - A LOAEL of 78 μg/kg bw/d.

For mammalian wildlife - A NOAEL of 55 μg/kg bw/d.

Dividing the avian TD by a UF_L of 3 yields an avian RfD of 26 µg/kg bw/d. A mammalian RfD of 18 µg/kg bw/d was calculated by dividing the mammalian TD by a UF_S of 3.

5.4.5   Calculation of Wildlife Criterion Values

WC values were calculated for each of the wildlife species of concern using exposure values recommended in Section 5.4.4.4. Calculations of WC values for each of the selected species follow.

The mean of the two WC_s values calculated for mammals is 50 pg/L. The mean of the four avian values is 74 pg/L. The lowest of these is the WC_i calculated for mammalian species. Therefore, the WC_f for methylmercury is 50 pg/L.

For the mink:

$$WC_S = \frac{(TD \ x \ [1/(UF_A \ x \ UF_S \ x \ UF_L)]) \ x \ Wt_A}{W_A + [(0.9)(F_A \ x \ BAF_3)]}$$

$$WC_S = \frac{(0.055 \ mg/kg/d \ x \ [1/(1 \ x \ 3 \ x \ 1)]) \ x \ 0.8 \ kg}{0.081 \ L/d + [(0.9) \ (0.178 \ kg/d \ x \ 1,600,000)]}$$

$$WC_S = 57 \ pg/L$$

For the otter:

$$WC_S = \frac{(TD \; x \; [1/(UF_A \; x \; UF_S \; x \; UF_L)]) \; x \; Wt_A}{W_A \; + \; [\, (0.8) \; (F_A \; x \; BAF_3) \; + \; (0.2) \; (F_A \; x \; BAF_4)\,]}$$

$$WC_S = \frac{(0.055 \; mg/kg/d \; x \; [1/(1 \; x \; 3 \; x \; 1)]) \; x \; 7.4 \; kg}{0.60 \; L/d \; + \; [\, (0.8) \; (1.22 \; kg/d \; x \; 1,600,000) \; + \; (0.2) \; (1.22 \; kg/d \; x \; 6,800,000)]}$$

$$WC_S = 42 \; pg/L$$

For the kingfisher:

$$WC_S = \frac{(TD \; x \; [1/(UF_A \; x \; UF_S \; x \; UF_L)]) \; x \; Wt_A}{W_A \; + \; [\, (1.0) \; (F_A \; x \; BAF_3)\,]}$$

$$WC_S = \frac{(0.078 \; mg/kg/d \; x \; [1/(1 \; x \; 1 \; x \; 3)]) \; x \; 0.15 \; kg}{0.017 \; + \; [\, (1.0) \; (0.075 \; x \; 1,600,000)\,]}$$

$$WC_S = 33 \; pg/L$$

For the loon:

$$WC_S = \frac{(TD \; x \; [1/(UF_A \; x \; UF_S \; x \; UF_L)]) \; x \; Wt_A}{W_A \; + \; [\, (1.0) \; (F_A \; x \; BAF_3)\,]}$$

$$WC_S = \frac{(0.078 \; mg/kg/d \; x \; [1/(1 \; x \; 1 \; x \; 3)]) \; x \; 4.0 \; kg}{0.012 \; L/d \; + \; [\, (1.0) \; (0.8 \; kg/d \; x \; 1,600,000)\,]}$$

$$WC_S = 82 \; pg/L$$

For the osprey:

$$WC_S = \frac{(TD \ x \ [1/(UF_A \ x \ UF_S \ x \ UF_L)]) \ x \ Wt_A}{W_A + [\ (1.0) \ (F_A \ x \ BAF_3) \ ]}$$

$$WC_S = \frac{(0.078 \ mg/kg/d \ x \ [1/(1 \ x \ 1 \ x \ 3)]) \ x \ 1.5 \ kg}{0.077 \ L/d + [\ (1.0) \ (0.3 \ kg/d \ x \ 1,600,000) \ ]}$$

$$WC_S = 82 \ pg/L$$

For the bald eagle:

$$WC_S = \frac{(TD \ x \ [1/(UF_A \ x \ UF_S \ x \ UF_L)]) \ x \ Wt_A}{W_A + [\ (0.74) \ (F_A \ x \ BAF_3) + (0.18) \ (F_A \ x \ BAF_4 \ ]}$$

$$WC_S = \frac{(0.078 \ mg/kg/d \ x \ [1/(1 \ x \ 1 \ x \ 3)]) \ x \ 4.6 \ kg}{0.16 \ L/d + [\ (0.74) \ (0.5 \ kg/d \ x \ 1,600,000) + (0.18) \ (0.5 \ kg/d \ x \ 6,800,000)]}$$

$$WC_S = 100 \ pg/L$$

5.4.6 Calculation of Mercury Residues in Fish Corresponding to the Wildlife Criterion Value

The WC for methylmercury, along with appropriate BAFs, can be used to calculate corresponding mercury residues in fish. Using the recommended BAFs presented in Table 5-1, a WC of 50 pg/L corresponds to methylmercury concentrations in fish of 0.077 μg/g and 0.346 μg/g for trophic levels 3 and 4, respectively.

5.4.7 Calculation of the Wildlife Criterion Value for Total Mercury in Water

A WC for total mercury can be calculated using an estimate of dissolved methylmercury as a proportion of total dissolved mercury in water. Mercury speciation data from filtered water samples are reviewed in Appendix D of Volume III. Based upon a survey of these data, the best current estimate of methylmercury as a proportion of total is 0.078. Using this value, a methylmercury WC of 50 pg/L corresponds to a total dissolved mercury concentration of 641 pg/L. An additional correction is needed if the WC is to be expressed as the amount of total mercury in unfiltered water. The available data, although highly variable, suggest that on average

total dissolved mercury comprises about 70 percent of that contained in unfiltered water (Back and Watras, 1995; Driscoll et al., 1995; Mason and Sullivan, 1997; Watras et al., 1995a). Making this final correction results in a WC of 910 pg/L (unfiltered, total mercury), which is approximately 70 percent of the value published previously in the GLWQI.

5.4.8   Calculation of a Wildlife Criterion for the Florida Panther

Estimates of the NOAEL and LOAEL in domestic cats were not used in the derivation of a WC for Florida panthers, but were presented instead to provide a comparison with other mammals. The chronic NOAEL for cats (20 μg/kg bw/d) is close to that derived from mink data (18.3 μg/kg bw/d). Cats, therefore, do not appear to be uniquely sensitive or insensitive to the toxic effects of mercury.

Derivation of a WC to protect the panther is complicated by the possibility that prey items (e.g., the raccoon) accumulate mercury to an even greater extent than the fish represented by trophic level 4. Other prey (e.g., deer) probably contain relatively lower levels of mercury. Calculation of a WC protective of the panther, therefore, requires collection of additional information on the diet of this species and mercury residues contained therein. These residues would then have to be related to corresponding levels in water through the use of PPFs (e.g., raccoon/fish or other aquatic biota) and BAFs (aquatic biota/water). Existing data are insufficient to support such an analysis but could be collected and developed for this purpose.

5.4.9   Comparison of GLWQI Criteria with WC Derived in this Report

The evaluation of data and calculation of WC values in this Report was done in accordance with the methods published in the draft GLWQI (U.S. EPA 1993a). The availability of additional data and differences in interpretation of those data led to differences in the calculated values of the WC in this Report and those published in the final GLWQI (U.S. EPA 1995b). Both evaluations employed the same methodology as described in Section 5.4.1 of this Volume. Both used the same studies as the basis for WC calculation: for birds, the three generation reproduction study in mallards (Heinz, 1974, 1975, 1976a,b, 1979) and, for mammals, the subchronic dietary studies in mink (Wobeser et al., 1976a,b). In addition to these studies, this Report also relies on Wobeser's dissertation (Wobeser, 1973), which provided some additional information that was augmented by discussions with the author.

To provide a basis for comparing methylmercury WC values derived in this Report with values calculated in the GLWQI, it was necessary to convert all methylmercury values to corresponding total mercury estimates (see Section 5.4.6 of this Volume). Table 5-3 presents a comparison between the WC values calculated in the GLWQI (U.S. EPA, 1995b) and this Report (converted to total mercury in unfiltered water). All of the WC values calculated in this Report are lower (i.e., more conservative) than those published in the GLWQI. All species-specific WC values, however, differ by a factor of three or less. Expressed as total mercury, the WC derived in this Report is approximately 70 percent of the WC derived in the GLWQI.

In the evaluation of effects in birds, both the GLWQI and this Report identified a LOAEL for reproductive effects in the second generation of mallards exposed to 0.5 ppm mercury in diet (Heinz 1976b, 1979). This LOAEL was adjusted to 0.078 mg/kg bw/d by applying an average food ingestion rate for treated mallards of 0.156 kg/kg/d. In calculating the wildlife reference dose, the GLWQI used a UF_A of 3 and a UF_L of 2. This Report used a UF_A of 1 and a UF_L of 3 (see Section 5.4.11.2 for a discussion of UF_L).

In the effects assessment for piscivorous mammals, both the GLWQI and this Report used data on mink administered mercury in the diet. The GLWQI identified a NOAEL of 1.1 ppm. At this dietary

Table 5-3
Species-specific Wildlife Criteria Calculated in the Great Lakes Water Quality Initiative
(GLWQI)[a] and in the Mercury Study Report to Congress

Species	Wildlife Criterion (pg/L)	
	GLWQI	Mercury Study Report to Congress
Mink	2880	1038
Otter	1930	764
Kingfisher	1040	598
Osprey	Not done	1498
Eagle	1920	1818

[a] U.S. EPA, 1995b

exposure, there were changes in the liver, lesions in the central nervous system, and axonal degeneration; moreover, two of the animals in this treatment group were observed at the end of treatment to move slowly by comparison to other mink. The study authors reported their opinion that mink treated at 1.1 ppm in the diet for longer than the study would be expected to show clinical signs of nervous system damage. Animals treated at the next dose, 1.8 ppm, were observed with anorexia, ataxia and increased mortality. Based on these considerations, this Report considered 1.1 ppm to be a LOAEL and, as described in Section 4.3, used data from the first part of the study to identify a NOAEL of 0.33 ppm. This Report also used data from Wobeser (1973) to establish the weights of female mink and kits used in this part of the study; this resulted in slight differences in conversion of dose in ppm diet to μg/kg bw/d

In its assessment of exposure to birds through consumption of prey, the GLWQI made assumptions that were appropriate to the Great Lakes region. In particular the GLWQI assumed that mercury contaminated herring gulls constitute 6% of the diet of bald eagles. As this Report is a nationwide assessment, use of this region-specific assumption was not considered appropriate; eagles were assumed to consume non-fish prey, with no mercury contamination, as 8% of the total diet. The largest numerical difference in the exposure assessment between the GLWQI and this Report is in the calculation of BAFs. The GLWQI used a BAF of 27,000 for trophic level 3 and a BAF of 140,000 for trophic level 4. Total mercury BAFs corresponding to the methylmercury-based values reported in Table 5-1 (and assuming that methylmercury constitutes 7.8 % of total mercury) are 124,800 and 530,400 for trophic levels 3 and 4, respectively.

Thus, the differences between the WC in the GLWQI and in this Report are a result of several factors. First, this Report uses more recent data to derive BAFs. The Supplementary Information Document to the final Water Quality Guidance for the Great Lakes System noted that a preliminary draft of the Mercury Report to Congress was available but was not used because it had not been completed at the time the final guidance was published (U.S. EPA 1995b, p. 144). Second, the GLWQI appropriately used some region-specific assumptions that were not used in this nationwide assessment (e.g., consumption of herring gulls by eagles). Third, different toxicity endpoints were used in this Report. In the GLWQI, a risk-management decision was made to base the

WC on endpoints that comprise direct effects on growth, reproduction, or development. In this Report, more sensitive endpoints were considered with the goal of assessing a greater range of toxic effects. Finally, different uncertainty factors were employed in the two assessments. In general, uncertainty factors used in the GLWQI are more conservative than those used in this Report.

5.4.10 Uncertainty Analysis

A formal analysis of uncertainty around the WC estimate was not attempted. Such an analysis would require specification of numeric distributions for each of the parameters in the equation. Data for several of the parameters in the equation, in particular the NOAEL and UF estimates, are presently sufficient to generate point estimates only. A partial uncertainty analysis has been conducted for the bioaccumulation part of the WC approach (see Appendix D of Volume III).

5.4.11 Sensitivity Analysis

In a sensitivity analysis, an attempt is made to characterize the extent to which a calculated value changes with changes in the parameters upon which its calculation depends. Examination of the equation for calculation of WC values suggests that a proportional relationship exists between the WC and the NOAEL, UF or Wt_A. The relationships between the WC and parameters that appear in the denominator are not as apparent and must be explored by varying these parameters one-by-one in systematic fashion. The analysis is also complicated by the variable relationship that exists between FD_3 and FD_4. In the otter and eagle, FD_3 and FD_4 tend to be reciprocal (although in the eagle these values do not add up to 1). In the mink, however, FD_3 is assigned a value of less than 1, and the remainder of the diet is assumed to consist of prey that are not aquatic in origin and are not contaminated with mercury.

Nevertheless, general conclusions can be reached regarding the sensitivity of WC estimates to changes in these parameters. These can be described as follows:

- A decrease in any parameter that appears in the denominator will have a larger effect on WC than an equivalent percentage-wise increase.

- When BAF_3 appears alone in the denominator, a percentage-wise increase in BAF_3 or FD_3 will cause a less than proportional decrease in the WC; conversely a decrease in BAF_3 or FD_3 will cause a greater than proportional increase in the WC.

- When both BAF_3 and BAF_4 appear in the denominator, an equivalent percentage-wise change in BAF_4 (and by extension PPF_4) has a greater impact on the WC than a change in BAF_3, but in either case, the effect is less than proportional.

- If BAF_3 and BAF_4 are both allowed to change (holding PPF_4 constant), a percentage-wise increase in BAF_3 (and by extension BAF_4) will have a less than proportional effect on WC, while a decrease in BAF_3 will have a greater than proportional impact.

- Under all circumstances, a percentage-wise increase in F_A will cause a less than proportional decrease in WC, while a decrease in F_A will cause a greater than proportional increase in WC.

- Owing to its small contribution to the analysis as a whole, large changes in W_A have a very small impact on WC.

With the exception of F_A, it is not possible to conclude that, for all species, the WC is most sensitive to one or the other of the parameters in the denominator of the equation. For species that feed at one trophic level, all parameters other than F_A have the potential to change WC in a proportional or greater than proportional manner. For species that feed at two trophic levels, the BAF at the lower trophic level becomes relatively less important, but it may still have a large impact on WC if the percentage of the diet represented by this lower trophic level is large (e.g., in the mink).

5.4.12 Uncertainties Associated with the Wildlife Criteria Methodology

Efforts to develop WC values for the protection of piscivorous wildlife are relatively recent in origin, and the methods employed for this purpose continue to undergo modification and refinement. Owing to the complexity of natural systems, uncertainties associated with the development of WC values are to be expected. Additional uncertainties derive from the relative scarcity of wildlife toxicity information and the necessity of extrapolating individual-based effects to higher levels of biological organization (e.g., populations).

Uncertainties associated with the WC methodology have been reviewed elsewhere (U.S. EPA, 1994). Rather than repeat this information, this Report attempts to focus on those areas that are especially pertinent to the development of a WC for mercury. These uncertainties are described below in no particular order.

5.4.12.1 Limitations of the Toxicity Database

Substantial uncertainties underlie most of the toxicity data for mercury in wildlife. Comparison of NOAELs and LOAELs between species requires adoption of unproven assumptions about the uptake, distribution, elimination, and toxic effects of mercury. Conclusions based upon extrapolation from one species to another are, therefore, tenuous. Additional uncertainties are a result of extrapolating from LOAELs to NOAELs and from subchronic endpoints to chronic endpoints. In some instances, there may also be a need to account for the possibility that test results do not adequately protect the most sensitive individuals. This may be particularly germane to the case of the Florida panther, where there is concern for individual animals.

Toxicity studies utilizing "naturally incorporated" mercury are complicated by the possibility that mercury is accompanied by other contaminants that are exerting some or all of the observed effect. Ideally, it is desirable to compare the effects of mercury that has been incorporated naturally with effects that are due to mercury that has been spiked into a prepared diet. By spiking mercury into the diet, the researcher can better control the dose to the animal. The bioavailability of mercury in such a formulation may be different from that which exists naturally. However, Charbonneau et al. (1976) demonstrated that the bioavailability and toxicity of methylmercury to cats is equivalent whether given in contaminated fish or spiked in the diet.

EPA cannot test all wildlife species of interest. The use of uncertainty factors for species extrapolation is likely, therefore, to continue. Existing information can be used, however, to suggest which species should be singled out for testing. Information of this type is reviewed in this document in several locations and includes species distribution, natural history considerations, and exposure factors.

Finally, comparisons between wildlife and human NOAELs are complicated by differences in the ability of a given study to reveal an adverse effect when it occurs. For wildlife, most of the endpoints selected can be

considered severely adverse or frank effects. Very few studies to date have been designed to study subtle adverse effects or precursors to adverse effects in wildlife. Developmental neurotoxicity endpoints are of particular interest due to their demonstrated sensitivity in humans. The question, therefore, arises: what would the LOAEL or NOAEL for a given wildlife species be if the researcher was looking for (or was able to detect) these more subtle effects? One approach to this question is to examine the results of studies in which both frank and more subtle effects were observed and determine the corresponding difference between dosage levels.

5.4.12.2 LOAEL-to-NOAEL Uncertainty Factor UF_L

In determining the WC for mercury exposure in wildlife, a chronic NOAEL is the preferred value for the TD. In cases where studies do not identify a NOAEL, the data are examined to identify a LOAEL. This LOAEL is then adjusted using a LOAEL-to-NOAEL uncertainty factor (UF_L) to estimate a wildlife RfD. A UF_L of 3 or 10 (based on EPA reference dose methodology) is typically applied when a LOAEL is used in the absence of a NOAEL.

In determining the RfD for human exposure to methylmercury, a large number of laboratory animal studies on methylmercury toxicity were summarized as supporting data. Results from many of these studies permitted estimation of both a LOAEL and a NOAEL. These studies were examined in an effort to determine the most appropriate UF_L for wildlife exposure to mercury.

The studies examined are summarized in Volume V of this Report. Nineteen studies were selected as being the most relevant and appropriate for determining a UF_L. Selection criteria included the following:

- methylmercury toxicity to nonhuman mammals;

- oral exposure (with preference given to dosing in food or drinking water); and

- chronic or subchronic exposure durations (with exceptions for reproductive and developmental toxicity where such distinctions are less relevant).

Cancer and genotoxic endpoints were not included because tumors are not often reported in wildlife toxicity studies. Endpoints included in the analysis included lethality, neurotoxicity, renal toxicity, gastrointestinal toxicity, immunotoxicity, developmental toxicity and reproductive toxicity (see Table 5-4). Data abstracted from the studies include the species and sex of the test subjects, toxicologic endpoint, LOAEL, NOAEL and the ratio between them. The LOAEL:NOAEL ratios were not segregated by endpoint because there was an insufficient number of studies at most endpoints to determine statistical significance.

The ratios of LOAEL-to-NOAELs for laboratory animal studies are plotted versus frequency in Figure 5-1. These ratios can be thought of as the reduction in the LOAEL necessary to estimate the corresponding NOAEL. Figure 5-1 illustrates that the majority of ratios lie between one and two (n=6) and between four and five (n=9). Only one ratio of the 19 plotted was greater than 10. A ratio of five indicates that the NOAEL observed following exposure to methylmercury is 5-fold less than the

Table 5-4
**Analysis of LOAEL-to-NOAEL Uncertainty Factor**

Endpoint Species and Sex		LOAEL (mg/kg/day)	NOAEL (mg/kg/day)	RATIO LOAEL:NOAEL	Study
Lethality					
B6C3F1 Mouse	M	0.69	0.60	1.15	Mitsumori et al., 1990
Neurotoxicity					
Rat (Wistar)	M & F	0.25	0.05	5.0	Munro et al., 1980
Cat	sex NS	0.046	0.020	2.3	Charbonneau et al., 1976
Monkey (*Macaca fasicularis*)	M & F	0.03	0.02	1.5	Sato and Ikuta, 1975
Monkey (*Macaca artoides* and *M. nemestrina*)	M & F	0.5	0.4	1.25	Evans et al., 1977
Renal Toxicity					
Mouse (ICR)	M F	0.72 0.62	0.15 0.11	4.8 5.6	Hirano et al., 1986
Mouse (B6C3F1)	M F	0.14 0.6	0.03 0.13	4.7 4.6	Mitsumori et al., 1990
Gastrointestinal Toxicity					
Mouse (B6C3F1)	M	0.69	0.14	4.9	Mitsumori et al., 1990
Immunotoxicity					
Rabbit (New Zealand White)	M & F	0.4	0.04	10.0	Koller et al., 1977
Developmental Toxicity					
Rat (Charles River)	F	4.0	0.2	20.0	Nolen et al., 1972
Rat (Wistar)	F	0.25	0.05	5.0	Khera and Tabacova, 1973
Rat (Charles River)	F	1.4	0.7	2.0	Fowler and Woods, 1977
Rat (Wistar)	offspring of both sexes	0.6	0.2	3.0	Schreiner et al., 1986

Table 5-4 (continued)
Analysis of LOAEL-to-NOAEL Uncertainty Factor

Endpoint Species and Sex		LOAEL (mg/kg/day)	NOAEL (mg/kg/day)	RATIO LOAEL:NOAEL	Study
Reproductive Toxicity					
Rat (Wistar)	M	0.5	0.1	5.0	Khera, 1973
Mouse (ICR)	M	0.72	0.15	4.8	Hirano et al., 1986
Mouse (B6C3F1)	M	0.68	0.14	4.9	Mitsumori et al., 1990
Monkey *(Macaca facicularis)*	M	0.065	0.047	1.4	Mohamed et al., 1987
Monkey *(M. facicularis)*	F	0.06	0.04	1.5	Burbacher et al., 1988

NS - Not stated.

Figure 5-1
LOAEL-to-NOAEL Ratio Distribution
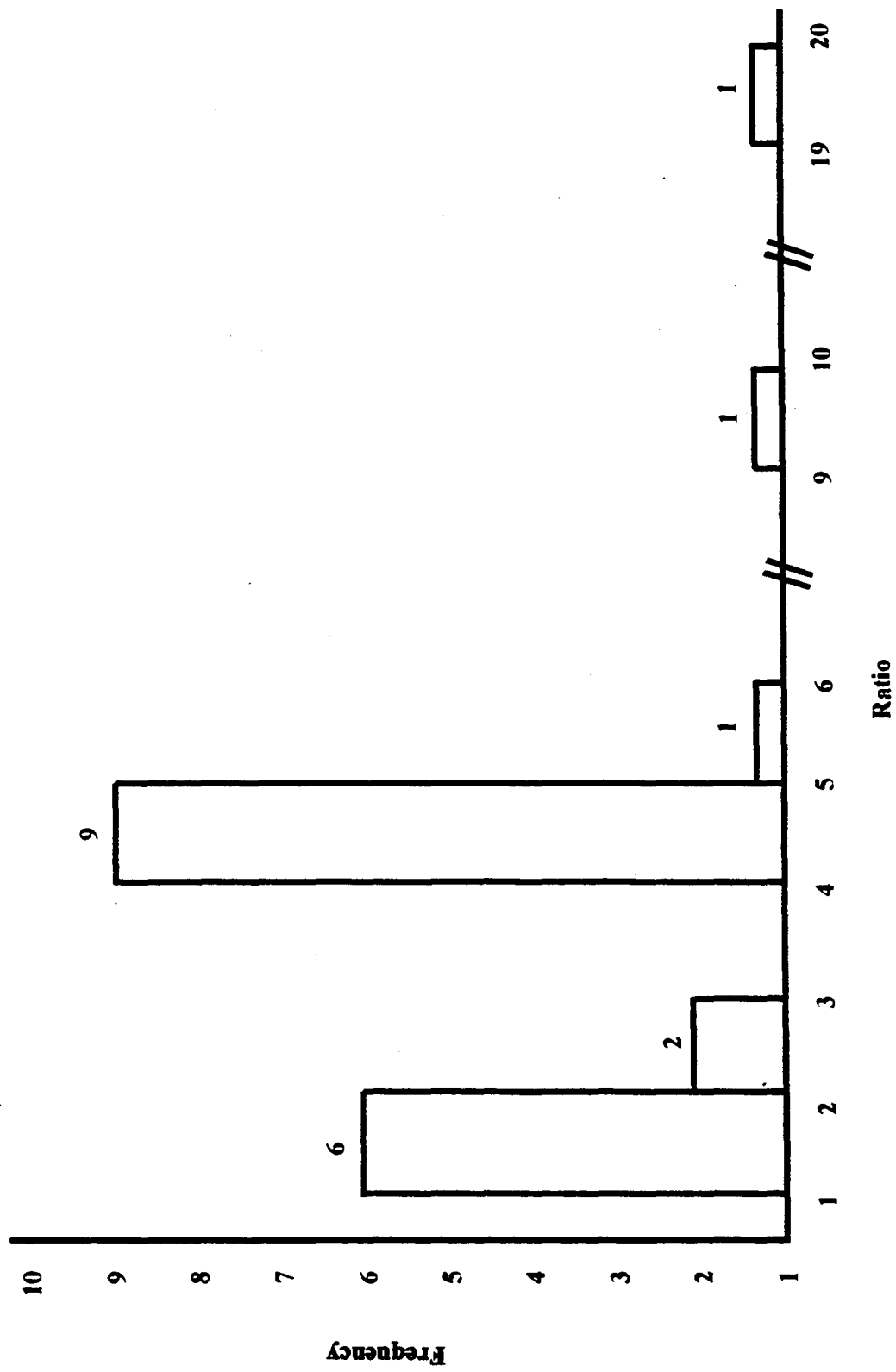
corresponding LOAEL. These data imply that most ratios between LOAELs and their corresponding NOAELs will be less than 10.

A similar analysis of animal toxicity data (Weil and McCollister, 1963) was provided by Dourson and Stara (1983). None of the LOAEL-to-NOAEL ratios from studies of 52 chemical substances exceeded 10. Only two of the 52 ratios exceeded five. The Dourson and Stara (1983) analysis has been cited in support of the use of a variable UF_L of as much as 10 in deriving reference doses for humans. Dourson and Stara (1983) recommended the application of a relatively large UF_L when estimating a NOAEL from a LOAEL for a severe or frank toxicological effect. Conversely, a low UF_L could be applied when the toxicological effect was considered to be relatively mild.

The distribution of LOAEL:NOAEL ratios around two and five primarily reflect the dose spacing selected for the study designs. Two-fold, 5-fold and 10-fold spacing are common in experiments of this type. The most appropriate interpretation of the ratios reported here and by Dourson and Stara (1983) is that the threshold for the toxicologic effects, defined by each study, lies within the bounds of the experimentally derived LOAEL divided by a UF_L and that most of the effects thresholds will be encompassed by using a UF_L of 10 or less. It is also likely that the most appropriate UF_L will vary with the toxicological endpoint selected. For studies that identify only a LOAEL, the principal assumption is that the next lower dose, had it been tested, would be a NOAEL. This assumption is best applied to studies that identify a LOAEL for mild effects. LOAELs for severe or frank effects (which are generally no used for human health risk assessment) require a high degree of professional judgment in applying a UF_L.

The analysis by Dourson and Stara (1983) and the analysis reported here support the UF_L of three selected by the authors of this Report for use with the avian LOAEL. In deriving an RfD for avian species, the authors of the GLWQI used a UF_L of two. Given the substantial uncertainties in all the values used to calculate the WC for mercury exposure, neither two nor three can be considered to be the only correct value.

5.4.12.3 Validity of BCF/BAF Paradigm

A significant shortcoming of the WC for mercury calculated in the GLWQI is its reliance upon BCF values determined in the laboratory. This methodology is based on a bioaccumulation paradigm (steady-state BCF x FCM) that was developed for neutral hydrophobic organic compounds and that may be inappropriate for application to mercury. In addition, the laboratory studies available for estimating BCFs were conducted with fish and not with organisms at the first trophic level (phytoplankton) that begin the bioaccumulation process. The modified GLWQI method uses field data for directly determining BCFs in phytoplankton but must rely on other uncertain assumptions, such as dry weight to wet weight conversion factors, to obtain the appropriate values. The result is increased uncertainty in the results of the GLWQI methodology when compared to direct estimation of BAFs from field data.

Field studies indicate that many, if not most, fish accumulate mercury throughout their lives, often in a nearly linear fashion with age (see for example Scott and Armstrong, 1972; MacCrimmon et al., 1983; Wren et al., 1983; Mathers and Johansen, 1985; Skurdal et al., 1985; Wren and MacCrimmon, 1986; Sorenson et al., 1990; Jackson, 1991; Gutenmann et al., 1992; Glass et al., 1993; Suchanek, 1993; Lange et al., 1993). Moreover, most of the mercury accumulated by fish at trophic level 4 is thought to be taken up from dietary sources. Thus, particularly for long-lived piscivorous fish, a relatively short (one year or less) waterborne exposure cannot duplicate the extent of accumulation that takes place in nature. In addition, the relationship between a concentration of an applied mercury species in the laboratory and the concentrations of multiple species present in the environment (some of which may not be bioavailable) is completely unknown.

The apparent progress to "steady-state" observed in several chronic laboratory studies (see McKim et al., 1976) should not be misinterpreted as an actual steady-state condition, but instead probably reflects growth dilution with rapidly growing fish. Growth dilution will tend to depress BCF values during periods of rapid growth, but as growth rate slows, continued accumulation of mercury will result in an increase in whole-body concentration with age.

5.4.12.4 Selection of Species of Concern

The species identified for the present analysis were selected because they were considered likely to be exposed and not due to their inherent sensitivity to mercury. Lacking toxicity information, little guidance is available concerning which wildlife species are most sensitive to mercury. In addition, there are problems associated with any comparison of laboratory and field data. For example, laboratory data suggest that mercury residues in eggs exceeding 0.5 μg/g are associated with impaired reproduction in mallard ducks (Heintz, 1974, 1976a,b, 1979) and ring-necked pheasant (Fimreite, 1971). In contrast, reproduction in herring gulls appears to be unaffected even when egg residues exceed 10 μg/g (Vermeer et al., 1973). Taken alone, these data suggest that mallards and pheasant are more sensitive to the toxic effects of mercury than are gulls. This may in fact be true; however, such comparisons are complicated by the presence/absence of additional stressors such as confinement, handling and weather, differences between natural and prepared diets, the possible ameliorative effect of selenium, and the interplay between "inherited" (egg) residues and that which the chick consumes. Toxicity can be difficult to observe in a field study, even when it is occurring. In 18 of 38 nests under study by Vermeer et al. (1973), hatching success could not be evaluated for one reason or another.

Clearly, exposure and sensitivity are related. If, for example, a species was, on a delivered dose basis, 10 times more sensitive than the eagle but, due to its dietary habits, received less than 10% of the dose, it would not be expected to show adverse effects at water concentrations protective of the eagle. Pharmacokinetic considerations may also be important. Thus, it has been suggested that birds eliminate a substantial amount of mercury through incorporation into plumage. The frequency and extent to which birds molt may, therefore, impact their apparent sensitivity in an environmental setting. Finally, it has been shown that most, if not all, wildlife possess some capability to detoxify methylmercury by hepatic demethylation. Enhanced demethylation would be particularly important if it represented an adaptive strategy for piscivorous species. The need for toxicity information has already been noted. As such information becomes available, it may be necessary to revise the WC for mercury.

There is also a need to consider animals other than birds and mammals. In particular, there is a need to characterize the exposure of carnivorous reptiles, such as the alligator, that are known to consume considerable quantities of fish and feed on animals (e.g., raccoon) that themselves feed on aquatic biota and are known to accumulate mercury (Roelke et al., 1991).

5.4.12.5 Trophic Levels at Which Wildlife Feed

The dietary preferences of the wildlife species identified for this analysis are shown in Table 5-2. Justification for these assignments can be found in two recent U.S. EPA publications that were developed for the purpose of supporting WC calculations (U.S. EPA 1993a, 1995a). It can be expected, however, that representatives of the same species will be exposed to different levels of mercury due to different feeding habits and/or differences in the availability of specific prey items. For example, bald eagles living on the shores of the Great Lakes may consume significant numbers of herring gulls (Kozie and Anderson, 1991). Since the gulls themselves are piscivores, feeding primarily at trophic level 3, it has been argued that when an eagle consumes a gull, it is feeding at trophic level 4 or higher; the gull/forage fish PPF is thought to be about 10, while the PPF for

fish at trophic level 4 is believed to be approximately 5 (U.S. EPA, 1995a). Eagles living in other parts of the country or migrating into an area during a particular time of year may consume relatively few fish, feeding instead on carrion, including rabbits, squirrels, and dead domestic livestock such as pigs and chickens (Harper et al., 1988). Other populations, however, are critically dependent upon the seasonal availability of fish, particularly spawning salmonids.

The feeding habits of bald eagles are reviewed extensively elsewhere (U.S. EPA, 1993a, 1995a). The intent of this discussion is not to characterize the food preferences of the eagle, but instead to demonstrate how difficult it is to characterize wildlife feeding habits on a nationwide, year-around basis. For some species, such as the kingfisher and river otter, it can be reasonably assumed that fish always comprise a high percentage of the diet. For others, such as the eagle and mink, considerable variations in diet are likely to exist. Still others, such as the Florida panther, consume prey (e.g., the raccoon) that, as a species, consume variable amounts of aquatic biota but that, in south Florida, are thought to represent a close link to the aquatic food chain.

5.4.12.6 Variability in BAFs at each Trophic Level

A concern related to the issue of feeding preference is the possibility that trophic levels presently assigned to the wildlife species in this analysis overestimate the actual extent to which they are exposed to mercury. This is because BAFs are developed to represent the average value for a trophic level when, in fact, piscivorous birds and mammals may be more likely to target prey at the lower end of the size (age) distribution. Thus, eagles are more likely to consume a 1 kg northern pike than a 10 kg individual, yet both are represented in the BAF for trophic level 4. Similarly, kingfishers are probably limited to smaller representatives of trophic level 3 than would be true of an osprey. The reason that these differences are important is that mercury tends to accumulate throughout the life of an individual fish, such that concentrations in an older individual at a given trophic level may far exceed those in a younger individual.

The need to apply BAF estimates on a nationwide basis in this study precludes further refinement. It may, however, be possible to explore this issue by using a probabilistic approach to analyze individual data sets. Specifically, it would be of interest to determine whether percentile information from the resulting output distributions can be related to fish of known size. Eventually, it may be possible to use this or another approach to refine BAF estimates for mercury.

5.4.12.7 Natural History Considerations

Natural exposures are likely to vary in both spatial and temporal domains. This is particularly true of species that migrate, including the bald eagle, osprey, and belted kingfisher. The necessity of incorporating this type of information and the means by which this can be accomplished are open questions.

5.4.12.8 Individuals Versus Populations

The methods used to develop a WC for mercury are based on effects data from individual organisms. The stated assessment endpoint for this Report, however, is the health of wildlife populations. The relationship between individuals and populations is likely to vary with the species and a large number of environmental factors. For some populations, the loss of a significant number of individuals may have little effect, particularly if environmental factors (like carrying capacity) limit population size. Animals that are capable of dispersing over large areas present an additional complication. It is possible, for example, that negative impacts could occur within a given location but would be difficult to observe due to a continuous influx of as yet unaffected individuals. For other populations, in particular those with low fecundity, loss of a relatively few individuals

could have a large impact. Clearly, there is a need to be able to extrapolate toxic effects on individuals to effects on populations. Unfortunately, this type of analysis is complicated by numerous factors and is essentially impossible to apply on a national scale.

Finally, a focus on populations may not always be appropriate, particularly when endangered species are involved. The same may also be true when various factors contribute to the possibility of regional effects. For example, 95% of eagles nationwide might be protected by a WC for avian species, but in a given region mortality could approach 100% if attributes of lakes and rivers in that region contributed to higher than average accumulation of mercury in the aquatic food chain.

5.4.12.9 Species Versus Taxa

The WC developed for mercury in birds was calculated as the geometric mean of values for four species. Similarly, the geometric mean of values for two species was used to represent all mammals. This approach is reasonable if the WC calculated for each species within a taxa are similar, but it would fail to protect species for which the WC value is much lower than the others with which it was averaged.

In the present analysis, WC values calculated for eagles, osprey, loon and kingfisher were within a factor of three of one another. WC values for mink and otter agreed to within a factor of about one and a half. As additional data are gathered, there is a need to identify species that, by virtue of sensitivity and/or exposure, are particularly vulnerable to mercury. Decisions could then be made concerning the advisability of special measures to insure their protection.

5.4.12.10 Discussion of Uncertainties Associated with the Wildlife Criteria Methodology

The existing limited data suggest that BAF values represent an important source of uncertainty in present efforts to calculate water-based WC values, although a lack of toxicity information and incomplete knowledge of what wildlife eat contribute substantially. Considerable progress has been made in understanding and predicting how chemical and biological factors affect mercury bioaccumulation in aquatic biota, and, in time, it may be possible to adjust BAF predictions as needed to represent specific surface waters of concern. The prospect for continuing uncertainty surrounding these estimates argues, however, for adoption of a residue-based approach; i.e., the use of measured mercury residues in fish and wildlife to identify populations at risk.

It is important to recognize that BAF values are calculated as the ratio of a tissue concentration and a water concentration. Emphasis has been placed on problems associated with obtaining the numerator in this equation. However, considerable uncertainty may also exist with respect to the denominator. In several instances, it has been shown that, with improved analytical methods, mercury levels in a given water body tend to come "down," resulting in an increase in the apparent BAF. This "decline" is usually not thought to be real but instead reflects improvements in sampling technique and analytical methods.

It is also unclear which of the mercury species are bioaccumulative and should, therefore, appear in the denominator. The present analysis considers dissolved methylmercury to be the best estimator of bioaccumulation potential in a given water body. Speciation data from a variety of systems suggest that most of the methylmercury in the water column exists as the dissolved form (mean of about 70%) (see Appendix D of Volume III). Nevertheless, questions remain concerning the bioavailability of dissolved methylmercury associated with DOC. Additional refinement of the BAF approach may require methods to identify the "freely dissolved" fraction of methylmercury. A similar approach is now used routinely in BAF calculations with high $\log K_{OW}$ organic compounds.

An effort was made to treat the uncertainty in BAF estimates by using a probabilistic approach. The advantage of this approach is that it explicitly treats known variation in these parameters, thereby providing for the statistical possibility of a high or low end result. In addition, the distributions themselves follow from the processes at work. As more information about mercury is obtained, the distributions themselves can be improved. For example, a skewed BAF distribution for trophic level 4 would be expected from random sampling of a fish population due to the relative scarcity of the oldest individuals. Based upon a survey of published data, the distribution of methylmercury values as a percent of total also appears to be highly skewed. With respect to the definition of these distributions, it is important to recall the possibility of regional bias introduced previously. It could be argued that FCMs based on regression of data for a large number of lakes should be given greater weight (perhaps equal to the number of lakes) than data from a single location. This, however, would only serve to increase the degree of regional bias that is already present.

5.5   Risk of Mercury from Airborne Emissions to Piscivorous Avian and Mammalian Wildlife

5.5.1   Lines of Evidence

Barr (1986) found that 0.3 ppm of mercury in trophic level 3 fish caused adverse effects on reproduction in common loons. In the present Report, an effort was made to calculate a WC for mercury which, if not exceeded, would be protective of piscivorous birds and mammals. The mercury residue in trophic level 3 fish that corresponds to this WC is 0.077 ppm, or about one-fourth the effect level identified by Barr (1986). Based upon a review of two national surveys, the average value for trophic level 3 fish in the continental U.S. was estimated to be 0.052 ppm; however, these surveys may have overestimated the true national average due to a bias toward waters receiving municipal and industrial waste. Nevertheless, recent surveys of lakes that do not receive point source loadings have yielded residue values in forage fish exceeding 0.077 ppm, particularly in regions already impacted by acid deposition (see for example Gerstenberger et al., 1993; Simonin et al., 1994; Driscoll et al., 1994; Lange et al., 1994; Cabana et al., 1994). Although it is difficult to precisely determine an adverse effects level for mercury in forage fish consumed by piscivorous wildlife, this value appears to lie in the range 0.077-0.30 ppm. The exact level may also vary to some degree depending upon the species in question and specific environmental factors.

The effects data, though limited, are remarkable for their consistency; RfDs derived for birds and mammals (mink and domestic cats) are essentially identical. Very few uncertainty factors were used in these calculations, and the uncertainty factor values were small. In addition, the estimated value of UF_L (used to adjust the TD for avian species) was supported by several sources of data. Finally, it should be noted that all wildlife RfDs are greater than the RfD for human health by a factor of about 200 (RfD for human health = 0.1 μg/kg bw/d; see Volume IV). As noted previously, the human health assessment differs from the wildlife assessment in its consideration of subtle cognitive impacts. The possibility also exists that humans are more sensitive than piscivorous wildlife on a delivered dose basis, perhaps due to differences in ability to detoxify methylmercury. Nevertheless, the WC for mercury is unlikely to be grossly "overprotective" (i.e., too low) and may, in some instances, be "underprotective."

5.5.2   Risk Statements

Given the national-scale scope of this Report, quantitative estimates of risk are not possible or appropriate. It is notable, however, that hazard quotients derived by other authors for mink (Giesy et al., 1994) and great egrets (Jurczck, 1993) ranged from 1.2 to 6.6. Such calculations suggest the possibility of local impacts on these two highly exposed populations. As indicated previously, fish residues in some areas exceed calculated WC values for trophic levels 3 and 4. It should be emphasized that these WC values were calculated using geometric mean BAF values; thus, BAFs were higher in approximately half of the systems for which field-data were available. For this reason, and given the small difference between effect (0.3 ppm) and no-effect (0.077 ppm) residue levels, it is likely that individuals of some highly exposed subpopulations (birds and mammals) are consuming fish at or very near adverse effect levels. Additional work is required to establish whether and to what extent impacts are occurring, and what effect local-scale impacts may have on larger species populations. Existing data are insufficient to speculate on the spatial or temporal scale of these possible adverse effects or the potential for recovery. However, the risk of adverse effects is great enough to warrant intensified study of highly exposed wildlife subpopulations, particularly in areas near mercury emissions point sources. Finally, the data suggest that special attention should be given to the possibility that mercury acts in concert with other bioaccumulative contaminants (e.g., PCBs, TCDD) to produce toxic effects at residue levels that, when evaluated separately, would not indicate a problem.

6.   CONCLUSIONS

The following conclusions are presented in approximate order of degree of certainty, based on the quality of the underlying database. The conclusions progress from those with greater certainty to those with lesser certainty.

- Mercury emitted to the atmosphere deposits on watersheds and is translocated to waterbodies. A variable proportion of this mercury is transformed by abiotic and biotic chemical reactions to organic derivatives, including methylmercury. Methylmercury bioaccumulates in individual organisms, biomagnifies in aquatic food chains and is the most toxic form of mercury to which wildlife are exposed.

- The proportion of total mercury in aquatic biota that exists as methylmercury tends to increase with trophic level. Greater than 90% of the mercury contained in freshwater fish exists as methylmercury. Methylmercury accumulates in fish throughout their lifetime, although changes in concentration as a function of time may be complicated by growth dilution and changing dietary habits.

- Piscivorous avian and mammalian wildlife are exposed to mercury primarily through consumption of contaminated fish and accumulate mercury to levels above those in prey items.

- Toxic effects on piscivorous avian and mammalian wildlife due to the consumption of contaminated fish have been observed in association with point source releases of mercury to the environment.

- Concentrations of mercury in the tissues of wildlife species have been reported at levels associated with adverse health effects in laboratory studies with the same species.

- Piscivorous birds and mammals receive a greater exposure to mercury than any other known component of aquatic ecosystems.

- BAFs for mercury in fish vary widely; however, field data are sufficient to calculate representative means for different trophic levels. These means are believed to be better estimates of mercury bioaccumulation in natural systems than values derived from laboratory studies. The recommended methylmercury BAFs for tropic levels 3 and 4 are 1,600,000 and 6,800,000, respectively (dissolved basis).

- Based upon knowledge of mercury bioaccumulation in fish, feeding rates, and the identity of prey items consumed by piscivorous wildlife, it is possible to rank the relative exposure of different piscivorous wildlife species. Of the six wildlife species selected for detailed analysis, the relative ranking of exposure to mercury is: kingfisher > otter > loon = osprey = mink ≥ bald eagle. Existing data are insufficient to estimate the exposure of the Florida panther relative to that of the selected species.

- Local emissions sources (<50 km from receptors) have the potential to increase the exposure of piscivorous wildlife well above that due to sources located more than 50 km from the receptors (i.e., "remote" sources).

- Field data are insufficient to conclude whether the mink, otter, or other piscivorous mammals have suffered adverse effects due to airborne mercury emissions.

- Field data are insufficient to conclude whether the loon, wood stork, great egret, or other piscivorous wading birds have suffered adverse effects due to airborne mercury emissions.

- Field data are suggestive of adverse toxicological effects in the Florida panther due to mercury; however, the interpretation of these data is complicated by the co-occurrence of several other potentially toxic compounds, habitat degradation, and loss of genetic diversity. Field data suggest that bald eagles have not suffered adverse toxic effects due to airborne mercury emissions

- Reference doses (RfDs) for methylmercury, defined as chronic NOAELs, were determined for avian and mammalian wildlife. Each RfD was calculated as the toxic dose (TD) from laboratory toxicity studies, divided by appropriate uncertainty factors. The RfD for avian species is 21 µg/kg bw/d (mercury basis). The RfD for mammalian wildlife is 18 µg/kg bw/d (mercury basis).

- Based upon knowledge of mercury exposure to wildlife and its toxicity in long-term feeding studies, criterion values can be calculated for the protection of piscivorous avian and mammalian wildlife. A wildlife criterion (WC) value is defined as the concentration of total mercury in water which, if not exceeded, protects avian and mammalian wildlife populations from adverse effects resulting from ingestion of surface waters and from ingestion of aquatic life taken from these surface waters.

- The methylmercury criterion for protection of piscivorous avian wildlife is 74 pg/L (mercury basis).

- The methylmercury criterion for protection of piscivorous mammalian wildlife is 50 pg/L (mercury basis).

- The final methylmercury criterion for protection of piscivorous wildlife species is 50 pg/L. This value corresponds to a total dissolved mercury concentration in the water column of 641 pg/L and methylmercury concentrations in fish of 0.077 ppm (trophic level 3) and 0.346 ppm (trophic level 4).

- Modeled estimates of mercury concentration in fish around hypothetical mercury emissions sources predict exposures within a factor of two of the WC. The WC, like the human RfD, is predicted to be a safe dose over a lifetime. It should be noted, however, that the wildlife effects used as the basis for the WC are gross clinical manifestations. Expression of subtle adverse effects at these doses cannot be excluded.

- The adverse effect level (population impacts on piscivorous wildlife) for methylmercury in fish that occupy trophic level 3 lies between 0.077 and 0.3 ppm. A comparison of this range of values with published residue levels in fish suggests that it is probable that individuals of some highly exposed wildlife subpopulations are experiencing adverse toxic effects due to airborne mercury emissions.

There are many uncertainties associated with this analysis, due to an incomplete understanding of the biogeochemistry and toxicity of mercury and mercury compounds. The sources of uncertainty include the following:

- Variability in the calculated BAFs is a source of uncertainty. BAFs given in this Report relate methylmercury in fish to dissolved methylmercury levels in the water column. Methods for the speciation of mercury in environmental samples are rapidly improving but remain difficult to perform. Questions also remain concerning the bioavailability of methylmercury associated with suspended

particulates and dissolved organic material. Local biogeochemical factors that determine net methylation rates are not fully understood. The food webs through which mercury moves are poorly defined in many ecosystems, and may not be adequately represented by a four-tiered food chain model.

- The representativeness of field data used in establishing the BAFs is a source of uncertainty. The degree to which the analysis is skewed by the existing data set is unknown. A disproportionate amount of data is from north-central and northeastern lakes. The applicability of these data to a national-scale assessment is unknown.

- Limitations of the toxicity database present a source of uncertainty.   Few controlled studies of quantifiable effects of mercury exposure in wildlife are available. These are characterized by limited numbers of dosage levels, making it difficult to establish NOAEL and LOAEL values. The toxic endpoints reported in most such studies can be considered severe, raising questions as to the degree of protection against subtle effects offered by reference doses and WC values. Use of less than lifetime studies for prediction of effects from lifetime exposure is a source of uncertainty.

- Concerns exist regarding the possibility of toxic effects in species other than the piscivorous birds and mammals evaluated in this Report. Uncertainty exists about mercury effects in birds and mammals that prey upon aquatic invertebrates and about possible effects on amphibians and aquatic reptiles. Uncertainty also exists about mercury effects in fish. Toxicity to terrestrial ecosystems, in particular soil communities, represents another source of uncertainty.

- Lack of knowledge of wildlife feeding habits is a source of uncertainty. Existing information frequently is anecdotal or confined to evaluations of a particular locality; the extent to which this information can be generalized is open to question. In some instances wherein feeding habits are relatively well characterized (e.g., Florida panther), the extent of mercury contamination of prey is poorly known (e.g., in raccoons).

- While the methods used to assess toxicity focus on individual-level effects, the stated goal of the assessment is to characterize the potential for adverse effects in wildlife populations. Factors that contribute to uncertainty in population-based assessments include these: variability in the relationship between individuals and populations; lack of data on carrying capacity; and relationships of one population, of the same or different species, to another population.

- A focus on populations may not always be appropriate. This could be true for endangered species, which may be highly dependent for the survival of the species on the health of a few individuals. This may also be true for some regional or local populations of widespread species; the local population may be "endangered" and, thus, dependent on the survival of individuals.

- Multiple stressor interactions involving chemical effects are in general poorly known. Even less well known are the possible effects of land and water use practices as they impact water quality and large-scale ecosystem attributes (e.g., community structure and biodiversity).

7.   RESEARCH NEEDS

Mercury is unusual among environmental contaminants in that levels that are likely to cause significant environmental damage exceed those thought to be present "naturally" by less than two (and perhaps closer to one) order(s) of magnitude. Conservative use of uncertainty factors can, therefore, lead to calculation of WC or other similar criterion values that are lower than mercury residues present in even the most pristine systems. With this in mind, there are two general areas within which research progress must be made if environmental assessments are to be improved. The first area pertains to basic information on the fate and effects of mercury in the environment, which would result in reduced use of uncertainty factors and ensure that WC, BAFs, and other estimates are based on a mechanistic understanding of the relevant processes. The second area is an improvement in the ability to detect ecological damage when it is in fact occurring. The present assessment of the "ecological impacts" of anthropogenic mercury emissions is largely limited to consideration of toxic effects on individuals. Models that would permit extrapolation of these results to populations (the simplest extrapolation of individual-based information) do not exist for most species. Further extrapolation to communities and ecosystems is presently out of the question.

Throughout this assessment, uncertainties, discussed above and elsewhere in the text, have limited the scope of possible conclusions. Although lack of sufficient data is a limiting factor in all phases of this assessment, a number of research needs have emerged as being especially important. These needs are presented below in no particular order.

7.1   Process-based Research

Mechanistic information is needed to understand the variability that presently typifies the mercury literature. Laboratory and field studies must be conducted to identify the determinants of mercury accumulation in aquatic food chains and to collect kinetic information that would allow researchers to describe the dynamics of these systems. Areas of uncertainty include: (1) translocation of mercury from watersheds to waterbodies; (2) factors that determine net rates of methylation and demethylation; (3) dietary absorption efficiency from natural food sources; (4) effect of dietary choice; and (5) bioavailability of methylmercury in the presence of dissolved organic material and other potential ligands.

In time, it is anticipated that this information can be used to develop process-based models for mercury bioaccumulation in fish and other aquatic biota. Significant progress in this direction is represented by the Mercury Cycling Model (MCM) (Hudson et al., 1994) and by the GAS-ISC3 model described in Volume III of this Report and employed in the wildlife exposure characterization.

7.2   Wildlife Toxicity Data

There is a need to reduce the present reliance on a relatively few toxicity studies for WC development. Additional data are needed for wildlife that constitute the most exposed organisms in various parts of the country, and in particular there is need to evaluate whether dietary selenium and endogenous demethylating pathways confer protection to piscivorous birds and mammals. Toxicity studies should examine endpoints relevant to the mode of action of methylmercury, including assessments of both reproductive and behavioral effects. There is also a critical requirement for toxicity data (e.g., growth and fecundity) that can be related to effects on populations, including effects on organisms that comprise the lower trophic levels.

7.3   Improved Analytical Methods

Efforts to develop and standardize methods for analysis of total mercury and methylmercury in environmental samples should be continued. Such methods must recognize the importance of contamination, both during the collection of such samples and during their analysis. It is particularly important that mercury measurements, which at present tend to be operationally defined (e.g., "soluble" or "adsorbed to organic material"), be made in such a way that mercury residues in fish can be correlated with the bioavailable mercury pool. Whenever possible, water samples should be filtered to obtain a measure of dissolved mercury species. As validated methods become available, it is important to analyze for both total and methylmercury so that differences between aquatic systems can be definitively linked to differences in methylmercury levels. Analyzing the two mercury species together will contribute to an understanding of existing data, much of which is reported as total mercury.

7.4   Complexity of Aquatic Food Webs

Present efforts to develop WC values for mercury are based on linear, four-tiered food chain models. Research is needed to determine whether this simple paradigm is appropriate and to develop alternatives if field data suggest otherwise. Of particular interest is whether zooplankton and phytoplankton should be modeled as two different trophic levels. Current information for detritivores and benthic invertebrates is extremely limited, even though their importance in mobilizing hydrophobic organic contaminants has been demonstrated.

7.5   Accumulation in Trophic Levels 1 and 2

Ongoing efforts to understand mercury bioaccumulation in aquatic systems continue to be focused on trophic levels 3 and 4, despite the fact that uncertainties in PPFs are relatively small. Additional emphasis should be placed on research at the lower trophic levels. In particular, there is a need to understand the determinants of mercury accumulation in phytoplankton and zooplankton and how rapid changes in plankton biomass impact these values.

7.6   Field Residue Data

High-quality field data are needed to support process-based research efforts and to determine residue concentrations in the fish and other aquatic biota that wildlife eat. Whenever possible, it is desirable to collect residue data at all trophic levels and to analyze mercury levels in the abiotic compartments of a system (e.g., water and sediments). It is particularly important that such measurements be made in a broader array of aquatic ecosystem types (including both lakes and rivers) so that a better understanding of mercury cycling and accumulation can be obtained.

Residue data from wildlife are needed to identify populations that are potentially at risk. Feathers and fur hold considerable promise in this regard due to the potential for "non-invasive" determination of mercury residues. Laboratory research is required, however, to allow interpretation of these data. Factors such as age, sex, and time to last molt are likely to result in variability among individuals of a single population and need to be understood. Whenever possible, tissue samples should be analyzed for both total and methylmercury, as well as selenium. This is especially true of the liver. More attention should be given to analysis of mercury levels in brain tissue, since this is the primary site of toxic action. Sampling efforts with wildlife should be accompanied by analyses of likely food items.

7.7   Natural History Data

The development of WC values requires knowledge of what wildlife eat. Fish sampling efforts are frequently focused on species that are relevant to human consumers but that may be of little significance to wildlife. There is an additional need to collect information for macroinvertebrates and amphibians. Seasonal and spatial effects on predation should be explored and methods developed to describe this information adequately. Additional life history data is needed to characterize fully the nature and extent of exposure to mercury. Complicating factors must be considered, including migratory behaviors and sex-specific differences in distribution and resource allocation. It is particularly important that information be collected to support the development of predictive population models for sensitive species. Such models must account for immigration and emigration, density dependent factors, and the observation that mercury often bioaccumulates as animals age resulting in variable residues in breeding animals from a single population.

8.   REFERENCES

Allard, B. and I. Arsenie (1991). Abiotic reduction of mercury by humic substances in aquatic system - An important process for the mercury cycle. *Water Air Soil Pollut.* 56:457-464.

Allard, M. and P.M. Stokes (1989). Mercury in crayfish species from thirteen Ontario lakes in relation to water chemistry and smallmouth bass (*Micropterus dolomieui*) mercury. *Can. J. Fish. Aquat. Sci.* 46:1040-1046.

Andersson, A. (1979). Mercury in soils. In: *The Biogeochemistry of Mercury in the Environment.* J.O. Nriagu (Ed.), Elsevier/North Holland Biomedical Press, Amsterdam, The Netherlands, pp. 79-106.

Anthony, R.G., M.G. Garrett and C.A. Schuler (1993). Environmental contaminants in bald eagles in the Columbia River estuary. *J. Wildl. Manage.* 57:10-19.

Aulerich, R.J., R.K. Ringer and S. Iwamoto (1974). Effects of dietary mercury in mink. *Arch. Environ. Contam. Toxicol.* 2(1):43-51.

Back, R.C. and C.J. Watras (1995). Mercury in zooplankton of northern Wisconsin lakes: Taxomomic and site-specific trends. *Water Air Soil Pollut.* 80:931-938.

Bahnick, D., C. Sauer, B. Butterworth and D. Kuehl (1994). A national study of mercury contamination in fish. *Chemosphere* 29:537-546.

Bakir, F., S.F. Damluji, L. Amin-Zaki, M. Murtadha, A. Khalidi, N.Y. Al-Rawi, S. Tikriti, and H.E. Dhahir (1973). Methylmercury poisoning in Iraq. *Science* 181: 230-240.

Barr, J.F. (1973). Feeding biology of the common loon (*Gavia immer*) in oligotrophic lakes of the Canadian Shield. Ph.D. dissertation, University of Guelph, Canada.

Barr, J.F. (1986). Population dynamics of the common loon (*Gavia immer*) associated with mercury-contaminated waters in northwestern Ontario. Occasional Paper No. 56, Canadian Wildlife Service.

Barr, J.F. (1996). Aspects of common loon (*Gavia immer*) feeding biology on its breeding ground. *Hydrobiologia* 321:119-144.

Beck, D.L. (1977). Pesticide and heavy metal residues in Louisiana river otter. M.S. thesis, University of Texas, Houston, TX.

Becker, P.H., D. Henning and R.W. Furness (1994). Differences in mercury contamination and elimination during feather development in gull and tern broods. *Arch. Environ. Contam. Toxicol.* 27:162-167.

Becker, D.S. and G.N. Bigham (1995). Distribution of mercury in the aquatic food web of Onondaga Lake, New York. *Water Air Soil Pollut.* 80:563-571.

Belant, J.L. and R.K Anderson (1990). Environmental contaminants in common loons from northern Wisconsin. *Pass. Pigeon* 52:306-310.

Beyer, W.N., E. Cromartie and G.B. Moment (1985). Accumulation of methylmercury in the earthworm, *Eisenia foetida*, and its effect on regeneration. *Bull. Environ. Contam. Toxicol.* 35:157-162.

Bigler, W.J., R.H. Jenkins, R.M. Cumbie, G.L. Hoff and E. Prather (1975). Wildlife and environmental health. Raccoons as indicators of zoonoses and pollutants in southeastern U.S.A. *J. Amer. Med. Assoc.* 167:592-597.

Bishop, C.A., M.D. Koster, A.A. Chek, D.J.T. Hussell and K. Jock (1995). Chlorinated hydrocarbons and mercury in sediments, red-winged blackbirds (*Agelaius phoeniceus*) and tree swallows (*Tachycineta biocolor*) from wetlands in the Great Lakes-St. Lawrence River basin. *Environ. Toxicol. Chem.* 14:491-501.

Bleavins, M.R. and R.J. Aulerich (1981). Feed consumption and food passage in mink (*Mustela vison*) and European ferrets (*Mustela putorius furo*). *Lab. Animal Sci.* 31:268-269.

Bloom, N.S (1992). On the chemical form of mercury in edible fish and marine invertebrate tissue. *Can. J. Fish. Aquat. Sci.* 49:1010-1017.

Bloom, N.S. and S.W. Effler (1990). Seasonal variability in the mercury speciation of Onondaga Lake (New York). *Water Air Soil Pollut.* 53:251-265.

Bloom, N.S., C.J. Watras and J.P. Hurley (1991). Impact of acidification on the methylmercury cycle of remote seepage lakes. *Water Air Soil Pollut.* 56:477-491.

Bodaly, R.A., J.W.M. Rudd, R.J.P. Fudge and C.A. Kelly (1993). Mercury concentrations in fish related to size of remote Canadian shield lakes. *Can. J. Fish. Aquat. Sci.* 50:980-987.

Boney, A.D. (1971). Sub-lethal effects of mercury on marine algae. *Mar. Pollut. Bull.* 2:69-71.

Borst, H.A. and C.G. Lieshout (1977). Phenylmercuric acetate intoxication in mink. *Tijdschr. Diergeneesk* 102:495-503.

Boudou, A. and F. Ribeyre (1985). Experimental study of trophic contamination of *Salmo gairdneri* by two mercury compounds - $HgCl_2$ and CH_3HgCl - analysis at the organism and organ levels. *Water Air Soil Pollut.* 26:137-148.

Bowerman, W.W. IV (1993). Regulation of bald eagle (*Haliaeetus leucocephalus*) productivity in the Great Lakes basin: An ecological and toxicological approach. Ph.D. dissertation, Michigan State University, East Lansing, MI.

Bowerman, W.W. IV., E.D. Evans, J.P. Giesy and S. Postupalsky (1994). Using feathers to assess risk of mercury and selenium to bald eagle reproduction in the Great Lakes region. *Arch. Environ. Contam. Toxicol.* 27:294-298.

Boyer, H.A. (1982). Trace elements in the water, sediments, and fish of the upper Mississippi River, twin cities metropolitan area. In: *Contaminants in the Upper Mississippi River.* J.G. Wiener, R.V. Anderson and D.R. McConville (Eds.). Butterworth Publishers, Boston, MA, pp. 195-230.

Braune, B.M. and D.E. Gaskin (1987). Mercury levels in Bonaparte's gulls (*Larus philadelphia*) during autumn molt in the Quoddy region, New Brunswick, Canada. *Arch. Environ. Contam. Toxicol.* 16:539-549.

Braune, B.M. and R.J. Norstrom (1989). Dynamics of organochlorine compounds in herring gulls: III. Tissue distribution and bioaccumulation in Lake Ontario gulls. *Environ. Toxicol. Chem*. 8:957-968.

Burbacher, T.M., M.K. Mohamed and N.K. Mottett (1988). Methylmercury effects on reproduction and offspring size at birth. *Reprod. Toxicol*. 1(4):267-278.

Burger, J., J.A. Rodgers, Jr. and M. Gochfeld (1993). Heavy metal and selenium levels in endangered wood storks *Mycteria americana* from nesting colonies in Florida and Costa Rica. *Arch. Environ. Contam. Toxicol*. 24:417-420.

Burger, J., I.C.T. Nisbet and M. Gochfeld (1994). Heavy metal and selenium levels in feathers of known-aged common terns (Sterna hirundo). *Arch. Environ. Contam. Toxicol*. 26:351-355.

Cabana, G. and J.B. Rasmussen (1994). Modelling food chain structure and contaminant bioaccumulation using stable nitrogen isotopes. *Nature*. 372:255-257.

Cabana, G., A. Tremblay, J. Kalff and J.B. Rasmussen (1994). Pelagic food chain structure in Ontario lakes: a determinant of mercury levels in lake trout (*Salvelinus namaycush*). *Can. J. Fish. Aquat. Sci.* 51:381-389.

Calder, W.A. and E.J. Braun (1983). Scaling of osmotic regulation in mammals and birds. *Am. J. Physiol.* 244:R601-R606.

Carty, A.J. and S.F. Malone (1979). The chemistry of mercury in biological systems. In: *The Biogeochemsitry of Mercury in the Environment*. J.O. Nriagu (Ed.), Elsevier/North-Holland Biomedical Press, Amsterdam, The Netherlands, pp. 433-479.

Caurant, F., M. Navarro and J.-C. Amiard (1996). Mercury in pilot whales: possible limits to the detoxification process. *Sci. Tot. Environ*. 186:95-104.

Cavalli, S. and N. Cardellicchio (1995). Direct determination of seleno-amino acids in biological tissues by anion-exchange separation and electrochemical detection. *J. Chromatog*. 706(A):429-436.

Charbonneau, S.M., I.C. Munro, E.A. Nera, R.F. Willes, T. Kuiper-Goodman, F. Iverson, C.A. Moodie, D.R. Stoltz, F.A.J. Armstrong, J.F. Uthe, H.C. Grice (1974). Subacute toxicities of methylmercury in the adult cat. *Toxic. Appl. Pharm*. 27:569-581.

Charbonneau, S.M., I.C. Munro, E.A. Nera, F.A.J. Armstrong, R.F. Willes, F. Bryce and R.F. Nelson (1976). Chronic toxicity of methylmercury in the adult cat. Interim report. *Toxicol.* 5:337-349.

Choi, M.H., J.J. Cech Jr. and M.C. Lagunas-Solar (1997). Bioavailability of methyl mercury to sacramento blackfish (*Orthodon microlepidotus*): dissolved organic carbon (DOC) effects. *Environ. Toxicol. Chem*. (In press).

Clark, K.E., F.A.P.C. Gobas and D. Mackay (1990). Model of organic chemical uptake and clearance by fish from food and water. *Environ. Sci. Technol*. 24:1203-1213.

Clarkson, T.W. (1972). The pharmacology of mercury compounds. *Ann. Rev. Pharmacol. Toxicol*. 12:375-406.

Clarkson, T.W. (1990). Human health risks from methylmercury in fish. *Environ. Toxicol. Chem*. 9:957-961.

Colborn, T.I. (1991). Epidemiology of Great Lakes bald eagles. *J. Environ. Health Toxicol.* 4:395-453.

Cope, W.G., J.G. Wiener and R.G. Rada (1990). Mercury accumulation in yellow perch in Wisconsin seepage lakes: Relation to lake characteristics. *Environ. Toxicol. Chem*. 9:931-940.

Cranmer, M., S. Gilbert, and J. Cranmer (1996). Neurotoxicity of mercury - indicators and effects of low-level exposure: overview. *Neurotoxicol*. 17:9-14.

Crowder, A. (1991). Acidification, metals and macrophytes. *Environ. Pollut*. 71:171-203.

Crowder, A.A., W. Dushenko and J. Grieg (1988). Metal contamination of wetland food chains in the Bay of Quinte, Ontario. Environment Ontario, Nov. 28-29, 1988. Toronto, Canada, pp. 133-153.

Cumbie, P.M. (1975). Mercury levels in Georgia otter, mink, and freshwater fish. *Bull. Environ. Contam. Toxicol.* 14:193-196.

Dietz, R., C.O. Nielsen, M.M. Hansen and C.T. Hansen (1990). Organic mercury in Greenland birds and mammals. *Sci. Tot. Environ*. 95:41-51.

Dillon, T.M. (1977). Mercury and the estuarine marsh clam, *Rangia cuneata* Gray. I. Toxicity. *Arch. Environ. Contam. Toxicol*. 6:249-255.

Dorfman, D. (1977). Tolerance of *Fundulus heteroclitus* to different metals in salt waters. *Bull. New Jersey Acad. Sci*. 22:21.

Dourson, M.L. and J.F. Stara (1983). Regulatory history and experimental support of uncertainty (safety) factors. *Reg. Toxicol. Pharmacol.* 3:224-238.

Driscoll, C.T., C. Yan, C.L. Schofield, R. Munson and J. Holsapple (1994). The mercury cycle and fish in the Adirondack lakes. *Environ. Sci. Technol.* 28:136A-143A.

Driscoll, C.T., V. Blette, C. Yan, C.L. Schofield, R. Munson and J. Holsapple (1995). The role of dissolved organic carbon in the chemistry and bioavailability of mercury in remote Adirondack lakes. *Water Air Soil Pollut.* 80:499-508.

Dukerschein, J.T., J.G. Wiener, R.G. Rada and M.T. Steingraeber (1992). Cadmium and mercury in emergent mayflies (*Hexagenia bilineata*) from the upper Mississippi River. *Arch. Environ. Contam. Toxicol.* 23:109-116.

Eisler, R. (1987). Mercury hazards to fish, wildlife, and invertebrates: A synoptic review. Publication No. 85 (1.10), U.S. Fish and Wildlife Service, Department of the Interior, Washington, DC.

Eisler, R. and R.J. Hennekey (1977). Acute toxicities of Cd^{+2}, Cr^{+6}, Hg^{+2}, Ni^{+2}, and Zn^{+2} to estuarine macrofauna. *Arch. Environ. Contam. Toxicol.* 6:315-323.

Elliott, J.E., A.M. Scheuhammer, F.A. Leighton and P.A. Pearce (1992). Heavy metal and metallothionein concentrations in Atlantic Canadian seabirds. *Arch. Environ. Contam. Toxicol.* 22:63-73.

Elliott, J.E., R.J. Norstrom and G.E.J. Smith (1996). Patterns, trends, and toxicological significance of chlorinated hydrocarbon and mercury contaminants in bald eagle eggs from the Pacific coast of Canada, 1990-1994. *Arch. Environ. Contam. Toxicol.* 31:354-367.

Ensor, K.L., D.D. Helwig and L.C. Wemmer (1992). Environmental mercury and lead in Minnesota common loons (*Gavia immer*). Minnesota Pollution Control Agency, Water Quality Division, St. Paul, MN.

Environment Canada (1991). Toxic chemicals in the Great Lakes and associated effects: Volume I - Contaminant levels and trends. Department of Fisheries and Oceans, Health and Welfare Canada, Toronto, Canada.

Eriksson, M.O.G., L. Henrikson and H.G. Oscarson (1989). Metal contents in liver tissues of non-fledged goldeneye, *Bucephala clangula*, ducklings: a comparison between samples from acidic, circumneutral, and limed lakes in south Sweden. *Arch. Environ. Contam. Toxicol.* 18:255-260.

Evans, R.D. (1986). Sources of mercury contamination in the sediments of small headwater lakes in south-central Ontario, Canada. *Arch. Environ. Contam. Toxicol.* 15:505-512.

Evans, H.L., R. Garman and B. Weiss (1977). Methylmercury: Exposure duration and regional distribution as determinants of neurotoxicity in nonhuman primates. *Toxicol. Appl. Pharmacol.* 41:15-33.

Facemire, C.F., T.S. Gross and L.J. Guillette, Jr. (1995). Reproductive impairment in the Florida panther: Nature or Nuture? *Environ. Health Perspect.* 103(suppl. 3):79-86.

Fimreite, N. (1970). Effects of methylmercury treated feed on the mortality and growth of leghorn cockerels. *Can. J. Anim. Sci.* 50:387-389.

Fimreite, N. (1971). Effects of methylmercury on ring-necked pheasants. Canadian Wildlife Service Occasional Paper Number 9. Department of the Environment. 39 pp.

Fimreite, N. (1974). Mercury contamination of aquatic birds in northwestern Ontario. *J. Wildl. Manage.* 38:120-131.

Fimreite, N. (1979). Accumulation and effects of mercury on birds. In: *The Biogeochemistry of Mercury in the Environment*. J.O. Nriagu (Ed.), Elsevier/North-Holland Biomedical Press, Amsterdam, The Netherlands, pp. 601-628.

Finley, M.T. and R.C. Stendell (1978). Survival and reproductive success of black ducks fed methyl mercury. *Environ. Pollu.* 16:51-64.

Finley, M.T., W.H. Stickel and R.E. Christensen (1979). Mercury residues in tissues of dead and surviving birds fed methylmercury. *Bull. Environ. Contam. Toxicol.* 21:105-110.

Fischer, R.G., S. Rapsomanikis, M.O. Andreae and F. Baldi (1995). Bioaccumulation of methylmercury and transformation of inorganic mercury by macrofungi. *Environ Sci.Technol.* 29:993-999.

Fjeld, E. and S. Rognerud (1993). Use of path analysis to investigate mercury accumulation in brown trout (*Salmo trutta*) in Norway and the influence of environmental factors. *Can. J. Fish. Aquat. Sci.* 50:1158-1167.

Fleming, W.J., J.A. Rodgers, J.A., Jr., and C.J. Stafford, C.J. (1984). Contaminants in wood stork eggs and their effects on reproduction, Florida, 1982. *Colonial Waterbirds* 7:88-93.

Florida Department of Environmental Regulation (FDER, 1990). Mercury, largemouth bass, and water quality: A preliminary report. Department of Environmental Regulation, Florida.

Florida Panther Interagency Committee (FPIC, 1989). Mercury contamination in Florida panthers. Status Report of the Technical Subcommittee.

Foley, R.E., S.J. Jackling, R.J. Sloan and M.K. Brown (1988). Organochlorine and mercury residues in wild mink and otter: Comparison with fish. *Environ. Toxicol. Chem.* 7:363-374.

Fowler, B.A. and J.S. Woods (1977). The transplacental toxicity of methylmercury to fetal rat liver mitochondria. *Lab. Invest.* 36:122-130.

Francis, D.R. and K.A. Bennett (1994). Additional data on mercury accumulation in northern Michigan river otters. *J. Freshwat. Ecol.* 9:1-5.

Friedmann, A.S., M.C. Watzin, T. Brinck-Johnsen and J.C. Leiter (1996). Low levels of dietary methylmercury inhibit growth and gonadal development in juvenile walleye (*Stizostedion vitreum*). *Aquat. Toxicol.* 35:265-278.

Futter, M.N. (1994). Pelagic food-web structure influences probability of mercury contamination in lake trout (*Salvelinus namaycush*). *Sci. Tot. Environ.* 145:7-12.

Ganther, H.E., C. Goudie, M.L. Sunde, M.J. Kipecky, P. Wagner, S.H. Oh and W.G. Hoekstra (1972). Selenium relation to decreased toxicity of methyl mercury added to diets containing tuna. *Science* 175:1122-1124.

Giesy, J.P., W.W. Bowerman, M.A. Mora, D.A. Verbugge, R.A. Othoudt, J.L. Newsted, C.L. Summer, R.J. Aulerich, S.J. Bursian, J.P. Ludwig, G.A. Dawson, T.J. Kubiak, D.A. Best, and D.E. Tillitt (1995). Contaminants in fishes from Great Lakes-influenced sections and above dams of three Michigan rivers: III. Implications for health of bald eagles. *Arch. Environ. Contam. Toxicol.* 29:309-321.

Giesy, J.P., D.A. Verbrugge, R.A. Othout, W.W. Bowerman, M.A. Mora, P.D. Jones, J.L. Newsted, C. Vandervoort, S.N. Heaton, R.J. Aulerich, S.J. Bursian, J.P. Ludwig, G.A. Dawson, T.J. Kubiak, D.A. Best and D.E. Tillitt (1994). Contaminants in fishes from Great Lakes-influenced sections and above dams of three Michigan rivers. II: Implications for health of mink. *Arch. Environ. Contam. Toxicol.* 27:213-223.

Gilbertson, M., T. Kubiak, J. Ludwig and G. Fox (1991). Great Lakes embryo mortality, edema, and deformities syndrome (GLEMEDS) in colonial fish-eating birds: Similarity to chick-edema disease. *J. Toxicol. Environ. Health* 33:455-520.

Gilmour, C.C. and E.A. Henry (1991). Mercury methylation in aquatic systems affected by acid deposition. *Environ. Pollut.* 71:131-169.

Glass, G.E., J.A. Sorensen, K.W. Schmidt, J.K. Huber and G.R. Rapp, Jr. (1993). Mercury sources and distribution in Minnesota's aquatic resources: Precipitation, surface water, sediments, plants, plankton, and fish. Final report to Minnesota Pollution Control Agency and Legislative Commission on Minnesota Resources, 1989-1991 (Contract Nos. 831479 and WQ/PDS020).

Gobas, F.A.P.C. (1993). A model for predicting the bioaccumulation of hydrophobic organic chemicals in aquatic food webs: Application to Lake Ontario. *Ecol. Model.* 69:1-17.

Godbold, D.L. (1991). Mercury-induced root damage in spruce seedlings. *Water Air Soil Pollut.* 56:823-831.

Goyer, R.A. (1993). Toxic effects of metals. In: *Casarett and Doull's Toxicology. The Basic Science of Poisons*, 4th Ed. M.O. Amdur, J. Doull, and C.D. Klaassen (Eds.), McGraw-Hill, Inc., New York, NY, pp. 623-680.

Grieb, T.M., C.T. Driscoll, S.P. Gloss, C.L. Schofield, G.L. Bowie and D.B. Porcella (1990). Factors affecting mercury accumulation in fish in the upper Michigan peninsula. *Environ. Toxicol. Chem.* 9:919-930.

Grier, J.W. (1974). Reproduction, organochlorines, and mercury in northwestern Ontario bald eagles. *Can. Field Nat.* 88:469-475.

Grubb, T.G., S.N. Wiemeyer and L.F. Kiff (1990). Eggshell thinning and contaminant levels in bald eagle eggs from Arizona, 1977 to 1985. *The Southwestern Naturalist* 35:298-301.

Gutenmann, W.H., J.G. Ebel, Jr., H.T. Kuntz, K.S. Yourstone and D.J. Lisk (1992). Residues of p,p'-DDE and mercury in lake trout as a function of age. *Arch. Environ. Contam. Toxicol.* 22:452-455.

Halbrook, R.S., J.H. Jenkins, P.B. Bush and N.D. Seabolt (1994). Sublethal concentrations of mercury in river otters: Monitoring environmental contamination. *Arch. Environ. Contam. Toxicol.* 27:306-310.

Harper, R.G., D.S. Hopkins and T.C. Dunstan (1988). Nonfish prey of wintering bald eagles in Illinois. *Wilson Bull.* 100:688-690.

Harrison, S.E., J.F. Klaverkamp and R.H. Hesslein (1990). Fates of metal radiotracers added to a whole lake: accumulation in fathead minnow (*Pimephales promelas*) and lake trout (*Salvelinus namaycush*). *Water Air Soil Pollut.* 52:277-293.

Heinz, G.H. (1974). Effects of low dietary levels of methylmercury on mallard reproduction. *Bull. Environ. Contam. Toxicol.* 11:386-392.

Heinz, G.H. (1975). Effects of methylmercury on approach and avoidance behavior of mallard ducklings. *Bull. Environ. Contam. Toxicol.* 13:554-564.

Heinz, G.H. (1976a). Methylmercury: Second-year feeding effects on mallard reproduction and duckling behavior. *J. Wildl. Manag.* 40(1):82-90.

Heinz, G.H. (1976b). Methylmercury: Second-generation reproductive and behavioral effects on mallard ducks. *J Wildl. Manag.* 40(4):710-715.

Heinz, G.H. (1979). Methylmercury: Reproductive and behavioral effects on three generations of Mallard ducks. *J. Wildl. Manage*. 43:394-401.

Heinz, G.H. and D.J. Hoffman (1996). The toxic interactions of mercury and selenium on mallard reproduction. Presentation given at the Wildlife Mercury Workshop, Fairfax, VA, April 12-13, 1996. Co-sponsored by the Wisconsin Department of Natural Resources and Electric Power Research Institute.

Helmke, P.A., W.P. Robarge, R.L. Korotev and P.J. Schomberg (1979). Effects of soil-applied sewage sludge on concentrations of elements in earthworms. *J. Environ. Qual.* 8:322-327.

Hildebrand, S.G., R.H. Strand and J.W. Huckabee (1980). Mercury accumulation in fish and invertebrates of the North Fork Holston River, Virginia and Tennessee. *J. Environ. Qual.* 9:393-400.

Hill, W.R., A.J. Stewart and G.E. Napolitano (1996). Mercury speciation and bioaccumulation in lotic primary producers and primary consumers. *Can. J. Fish. Aquat. Sci*. 53:812-819.

Hintelmann, H., P.M. Welbourn and R.D. Evans (1995). Binding of methylmercury compounds by humic and fulvic acids. *Water Air Soil Pollut.* 80:1031-1034.

Hirano, M., K. Mitsumori, K. Maita and Y. Shirasu (1986). Further carcinogenicity study on methylmercury chloride in ICR mice. *Jap. J. Vet. Sci.* 48(1):127-135.

Hongve, D., O.K. Skogheim, A. Hindar and H. Abrahamsen (1980). Effects of heavy metals in combination with NTA, humic acid, and suspended sediment on natural phytoplankton photosynthesis. *Bull. Environ. Contam. Toxicol*. 25:594-600.

Huckabee, J.W., J.W. Elwood and S.G. Hildebrand (1979). Accumulation of mercury in freshwater biota. In: *The Biogeochemistry of Mercury in the Environment*. J.O. Nriagu (Ed.), Elsevier/North Holland Biomedical Press, Amsterdam, The Netherlands, pp. 277-302.

Hudson, R.J.M., S.A. Gherini, C.J. Watras, and D.B. Porcella (1994). Modelling the biogeochemical cycle of mercury in lakes: the mercury cycling model (MCM) and its application to the MTL study lakes. In: *Mercury Pollution: Integration and Synthesis*. C.J. Watras and J.W. Huckabee (Eds.), Lewis Publishers, Boca Raton, FL, pp. 473-523.

Hurley, J.P., J. M. Benoit, C.L. Babiarz, M.M. Shafer, A.W. Andren, J.R. Sullivan, R. Hammond and D.A. Webb (1995). *Environ.Sci. Techno.* 29:1867-1875.

Jackson, T.A. (1991). Biological and environmental control of mercury accumulation by fish in lakes and reservoirs of northern Manitoba, Canada. *Can. J. Fish. Aquat. Sci*. 48:2449-2470.

Jernelöv, A., A.-H. Johansson, L. Sörensen and A. Svenson (1976). Methyl mercury degradation in mink. *Toxicol*. 315-321.

Johansson, K., M. Aastrup, A. Andersson, L. Bringmark and A. Iverfeldt (1991). Mercury in Swedish forest soils and waters - Assessment of critical load. *Water Air Soil Pollut*. 56:267-281.

Johnston, T.A., R.A. Bodaly and J.A. Mathias (1991). Predicting fish mercury levels from physical characteristics of boreal reservoirs. *Can. J. Fish. Aquat. Sci*. 48:1468-1475.

Jordan, D. (1990). Mercury contamination: Another threat to the Florida panther. *Fish and Wildlife Service Endangered Species Technical Bulletin* 15(2):1.

Joslin, J.D. (1994). Regional differences in mercury levels in aquatic ecosystems: A discussion of possible causal factors with implications for the Tennessee River system and the northern hemisphere. *Environ. Manage*. 18:559-567.

Jurczyk, N.U. (1993). An ecological risk assessment of the impact of mercury contamination in the Florida Everglades. Master thesis, University of Florida, Gainesville, FL.

Kajiwara, Y., A. Yasutake, T. Adachi, and K. Hirayama (1996). Methylmercury transport across the placenta via neutral amino acid carrier. *Arch. Toxicol*. 70:310-314.

Kerper, L.E., N. Ballatori and T.W. Clarkson (1992). Methylmercury transport across the blood-brain barrier by an amino acid carrier. *Am. J. Physiol. 262*:R761-R765.

Khera, S. (1973). Reproductive capability of male rats and mice treated with methyl mercury. *Toxicol. Appl. Pharm.* 24:167-177.

Khera, K.S. and S.A. Tabacova (1973). Effects of methylmercuric chloride on the progeny of mice and rats treated before or during gestation. *Food. Cosmet. Toxicol.* 11:245-254.

Kim, J.-H., S.E. Lindbert and T.P. Meyers (1995). Micrometeorological measurements of mercury fluxes over background forest soils in eastern Tennessee. *Atmos. Envir*. 27:267-282.

Klaunig, J., S. Koepp, and M. McCormick (1975). Acute toxicity of a native mummichog population (*Fundulus heteroclitus*) to mercury. *Bull. Environ. Contam. Toxicol.* 14:534-536.

Koeman, J.H., W.H.M. Peeters, C.H.M. Koudstaal-Hol, P.S. Thioe and J.J.M. De Goeij (1973). Mercury-selenium correlations in marine mammals. *Nature* 245:385-386.

Koller, L.D., J.H. Exon and B. Arbogast. 1977. Methylmercury: Effect on serum enzymes and humoral antibody. *J. Toxicol. Environ. Health* 2:1115-1123.

Kozie, K.D. and R.K. Anderson (1991). Productivity, diet, and environmental contaminants in bald eagles nesting near the Wisconsin shoreline of Lake Superior. *Arch. Environ. Contam. Toxicol.* 20:41-48.

Kucera, E. (1983). Mink and otters as indicators of mercury in Manitoba waters. *Can. J. Zool.* 61:2250-2256.

Kudo, A., H. Nagase and Y. Ose (1982). Proportion of methylmercury to the total amount of mercury in river waters in Canada and Japan. *Water Res*. 16:1011-1015.

Kuiper, J. (1981). Fate and effects of mercury in marine plankton communities in experimental enclosures. *Ecotoxicol. Environ. Safety* 5:106-134.

Landrum, P.F., M.D. Reinhold, S.R. Nihart and B.J. Eadie (1985). Predicting the bioavailability of organic xenobiotics to *Pontoporeia hoyi* in the presence of humic and fulvic materials and natural dissolved oxygen. *Environ. Toxicol. Chem.* 4:459-467.

Lange, T.R., H.E. Royals and L.L. Connor (1993). Influence of water chemistry on mercury concentration in largemouth bass from Florida lakes. *Trans. Am. Fish. Soc*. 122:74-84.

Lathrop, R.C., P.W. Rasmussen and D.R. Knauer (1991). Mercury concentrations in walleyes from Wisconsin (USA) lakes. *Water Air Soil Pollut*. 56:295-307.

Lee, Y.H., H. Hultberg and I. Andersson (1985). Catalytic effect of various metal ions on the methylation of mercury in the presence of humic substances. *Water Air Soil Pollut*. 25:391-400.

Lee, Y.H. and H. Hultberg (1990). Methylmercury in some Swedish surface waters. *Environ. Sci. Technol*. 9:833-841.

Lindberg, S.E. (1996). Forests and the global biogeochemical cycle of mercury: the importance of understanding air/vegetation exchange processes. In: *Global and Regional Mercury Cycles: Sources, Fluxes and Mass Balances*. W. Baeyens et al. (Eds.), printed in the Netherlands, pp. 359-380.

Lindqvist, O. (1991). Mercury in the Swedish environment. Recent research on causes, consequences and corrective measures. *Water Air Soil Pollut*. 55:1-261.

Lowe, T.P., T.W. May, W.G. Brumbaugh and D.A. Kane (1985). National contaminant biomonitoring program: Concentrations of seven elements in freshwater fish, 1978-1981. *Arch. Environ. Contam. Toxicol.*14:363-388.

MacCrimmon, H.R., C.D. Wren and B.L. Gots (1983). Mercury uptake by lake trout, *Salvelinus namaycush*, relative to age, growth, and diet in Tadenac Lake with comparative data from other Precambrian shield lakes. *Can. J. Fish. Aquat. Sci*. 40:114-120.

Maserti, B.E. and R. Ferrara (1991). Mercury in plants, soil and atmosphere near a chlor-alkali complex. *Water Air Soil Pollut*. 56:15-20.

Mason, R.P., J.R. Reinfelder and F. M.M. Morel (1996). Uptake, toxicity and trophic transfer of mercury in a coastal diatom. *Environ. Sci. Technol.* 30:1835-1845.

Mason, R.P. and K.A. Sullivan (1997). Mercury in Lake Michigan. *Environ. Sci. Technol*. 31:942-947.

Mathers, R.A. and P.H. Johansen (1985). The effects of feeding ecology on mercury accumulation in walleye (*Stizostedion vitreum*) and pike (*Esox lucius*) in Lake Simcoe. *Can. J. Zool*. 63:2006-2012.

May, K., M. Stoeppler and K. Reisinger (1987). Studies in the ratio total mercury/methylmercury in the aquatic food chain. *Tox. Environ. Chem.* 13:153-159.

McGrath, J.T. (1960). *Neurological Examination of the Dog with Clinicopathological Observations. 2nd ed.* Lea and Febiger, Philadelphia, PA.

McKim, J.M., G.F. Olson, G.W. Holcombe and E.P. Hunt (1976). Long-term effects of methylmercuric chloride on three generations of brook trout (*Salvelinus fontinalis*): Toxicity, accumulation, distribution, and elimination. *J. Fish. Res. Bd. Can*. 33:2726-2739.

McMurtry, M.J., D.L. Wales, W.A. Schneider, G.L. Beggs and P.E. Diamond (1989). Relationship of mercury concentrations in lake trout (*Salvelinus namaycush*) and smallmouth bass (*Micropterus dolomieui*) to the physical and chemical characteristics of Ontario lakes. *Can. J. Fish. Aquat. Sci*. 46:426-434.

Meili, M., A. Iverfeldt and L. Hakanson (1991). Mercury in the surface water of Swedish forest lakes - concentrations, speciation and controlling factors. *Water Air Soil Pollut.* 56:439-453.

Meyer, M.W., D.C. Evers, T. Daulton and W.E. Braselton (1995). Common loons (*Gavia immer*) nesting on low pH lakes in northern Wisconsin have elevated blood mercury content. *Water Air Soil Pollut*. 80:871-880.

Meyer, M.W., D.C. Evers and J.H. Hartigan (1996). Relationship of mercury exposure to common loon reproduction in Wisconsin. Presentation given at the Wildlife Mercury Workshop, Fairfax, VA, April 12-13, 1996. Co-sponsored by the Wisconsin Department of Natural Resources and the Electric Power Research Institute.

Mhatre, G.N. and S.B. Chaphekar (1985). The effect of mercury on some aquatic plants. *Environ. Pollut*. 39:297-216.

Michigan Department of Natural Resources (MDNR, 1993). Mercury in Michigan's environment: Environmental and human health concerns. A science report to Governor John Engler. R.D. Sills, Michigan Environmental Science Board, Lansing, MI.

Miskimmin, B.M., J.W.M. Rudd and C.A. Kelly (1992). Influence of dissolved organic carbon, pH and microbial respiration rates on mercury methylation and demethylation in lake water. *Can. J. Fish. Aquat. Sci*. 49:17-22.

Mitsumori, K., M. Hirano, H. Ueda, K. Maita, and Y. Shirasu. (1990). Chronic toxicity and carcinogenicity of methylmercury chloride in B6C3F1 mice. *Fund. Appl. Toxicol.*14:179-190.

Mohamed, M., T. Burbacher and N. Mottet (1987). Effects of methyl mercury on testicular functions in Macaca fascicularis monkeys. *Pharmacol. Toxicol.* 60(1):29-36.

Mosbaek, H., J.C. Tjell and T. Sevel (1988). Plant uptake of airborne mercury in background areas. *Chemosphere* 17:1227-1236.

Munro, I.C., E.A. Nera and S.M. Charbonneau (1980). Chronic toxicity of methylmercury in the rat. *J. Environ. Path. Toxicol.* 3:437-447.

Muramoto, S. and Y. Oki (1984). Influence of anionic surface-active agents on the uptake of heavy metals by water hyacinth (*Eichornia crassipes*). *Bull. Environ. Contam. Toxicol.* 33:444-450.

Nagase, H., Y. Ose, T. Sato and T. Ishikawa (1984). Mercury methylation by compounds in humic material. *Sci. Tot. Environ.* 32:147-156.

Nagy, K.A. (1987). Field metabolic rate and food requirement scaling in mammals and birds. *Ecol. Monogr.* 57:111-128.

National Acid Precipitation Assessment Program (NAPAP, 1990). Acidic deposition: State of science and technology, volume II, aquatic processes and effects. National Acid Precipitation Program, Washington, D.C.

Nature Conservancy (1994). Heritage database. Eastern Heritage Task Force, Boston, MA. Developed under U.S. EPA appropriation 683\40108 for the Office of Air Quality Planning and Standards, Emissions Standards Division, Research Triangle Park, NC.

Niimi, A.J. and G.P. Kissoon (1994). Evaluation of the critical body burden concept based on inorganic and organic mercury toxicity to rainbow trout (*Oncorhynchus mykiss*). *Arch. Environ. Contam. Toxicol.* 26:169-178.

Nilsson, A. and L. Hakanson (1992). Relationships between mercury in lake water, water color and mercury in fish. *Hydrobiologia* 235/236:675-683.

Nolen, G.A., E.V. Buchler, R.G. Geil and E.I. Goldenthal (1972). Effects of trisodium nitrotriacetate on cadmium and methylmercury toxicity and teratogenicity in rats. *Toxicol. Appl. Pharmacol.* 23:222-237.

Norheim, G. and A. Froslic (1978). The degree of methylation and organ distribution in some birds of prey in Norway. *Acta. Pharmacol. Toxicol.* 43:196-204.

Ohlendorf, H.M., D.J. Hoffman, M.K. Saiki, and T.W. Aldrich (1986). Embryonic mortality and abnormalities of aquatic birds: apparent impacts of selenium from irrigation drainwater. *Sci. Tot. Environ.* 52:49-63.

O'Connor, D.J. and S.W. Nielsen (1980). Environmental survey of methylmercury levels in wild mink (*Mustela vison*) and otter (*Lutra canadensis*) from the northeastern United States and experimental pathology of methylmercurialism in the otter. *Worldwide Furbearer Conference Proceedings*, pp. 1728-1745.

Odsjö, T. (1982). Eggshell thinning and levels of DDT, PCB and mercury in the eggs of osprey (*Pandion haliaetus L.*) and marsh harrier (*Circus aeruginosus L.*) in relation to their breeding success and population status in Sweden. Ph.D. dissertation, University of Stockholm, Sweden.

Ogden, J.C. (1994). A comparison of wading bird nesting colony dynamics (1931-1946 and 1974-1989) as an indication of ecosystem conditions in the southern Everglades. In: *Everglades: The Ecosystem and its Restoration*. S.M. Davis and J.C. Ogden (Eds.), St. Lucie Press, Delray, FL, pp.533-570.

Olson, K.R. and R.C. Harrel (1973). Effect of salinity on acute toxicity of mercury, copper, and chromium for *Rangia cuneata* (Pelecypoda, Mactridae). *Contrib. Mar. Sci*. 17:9-13.

Olson, K.R., K.S. Squibb and R.J. Cousins (1978). Tissue uptake, subcellular distribution, and metabolism of $^{14}CH_3HgCl$ and $CH_3^{203}HgCl$ by rainbow trout, *Salmo gairdneri. J. Fish. Res. Bd. Can.* 35:381-390.

Osowski, S.L., L.W. Brewer, O.E. Baker and G.P. Cobb (1995). The decline of mink in Georgia, North Carolina, and South Carolina: The role of contaminants. *Arch. Environ. Contam. Toxicol*. 29:418-423.

Palmisano, F., N. Cardellicchio and P.G. Zambonin (1995). Speciation of mercury in dolphin liver: a two-stage mechanism for the demethylation accumulation process and role of selenium. *Mar. Environ. Res*. 40:109-121.

Parks, J.W., A. Lutz and J.A. Sutton (1989). Water column methylmercury in the Wabigoon/English River-Lake system: Factors controlling concentrations, speciation, and net production. *Can. J. Fish. Aquat. Sci.* 46:2184-2202.

Pauling, L. (1963). College Chemistry, Third Edition. Freeman.

Peakall, D.B. (1988). Known effects of pollutants on fish-eating birds in the Great Lakes of North America. In: *Toxic Contamination in Large Lakes. Vol. I: Chronic Effects of Toxic Contaminants in Large Lakes*. N.W. Schmidtke (Ed.), Lewis Publishers, Inc., Chelsea, MI, pp. 39-54.

Porcella, D.B., C.J. Watras and N.S. Bloom (1991). Mercury species in lake water. In: The deposition and fate of trace metals in our environment. Gen. Tech. Rep. NC-150, S. Verry and S.J. Vermette, U.S. Dept. Agric., Forest Service, North Central Forest Exp. Station, St. Paul, MN, pp. 127-138.

Post, J.R., R. Vandenbos and D.J. McQueen (1996). Uptake rates of food-chain and waterborne mercury by fish: field measurements, a mechanistic model, and an assessment of uncertainties. *Can. J. Fish. Aquat. Sci.* 53:395-407.

Rada, R.G., D.E. Powell and J.G. Wiener (1993). Whole-lake burdens and spatial distribution of mercury in surficial sediments in Wisconsin seepage lakes. *Can. J. Fish. Aquat. Sci*. 50:865-873.

Ribeyre, R. and A. Boudou (1984). Bioaccumulation et repartition tissulaire du mercure - $HgCl_2$ et CH_3HgCl - chez *Salmo gairdneri* apres contamination par voie directe. *Water Air Soil Pollut*. 23:169-186.

Ribeyre, R. And A. Boudou (1994). Experimental study of inorganic and methylmercury bioaccumulation by four species of freshwater rooted macrophytes from water and sediment contamination sources. *Ecotoxicol. Environ. Saf*ety 28:270-286.

Richardson, G.M., M. Egyed and D.J. Currie (1995). Does acid rain increase human exposure to mercury? A review and analysis of recent literature. *Environ.Toxicol.Chem.* 14:809-813.

Rodier, P.M. (1995). Developing brain as a target of toxicity. *Environ. Health Perspec*. 103 (suppl. 6):73-76.

Roelke, M.E., D.P. Schultz, C.F. Facemire, S.F. Sundlof and H.E. Royals (1991a). Mercury contamination in Florida panthers. Florida Game and Fresh Water Fish Commission, Gainesville, FL.

Roelke, M.E., D.P. Schultz, C.F. Facemire and S.F. Sundlof (1991b). Mercury contamination in the free-ranging endangered Florida panther (*Felis concolor coryi*). *Am. Assoc. Zoo. Vet. Annu. Proc*. 277-283.

Roelke, M.M., J.S. Martenson and S.J. O'Brien (1993). The consequences of demographic reduction and genetic depletion in the endangered Florida panther. *Curr. Biol*. 3:340-350.

Saouter, E., L. Hare, P.G.C. Campbell, A. Boudou and F. Ribeyre (1993). Mercury accumulation in the burrowing mayfly (*Hexagenia rigida*) (ephemeroptera) exposed to CH_3HgCl or $HgCl_2$ in water and sediment. *Water Res.* 27:1041-1048.

Sarkar, A. and S. Jana (1986). Heavy metal pollutant tolerance of *Azolla pinnata*. *Water Air Soil Pollut*. 27:15-18.

Sato, T. and F. Ikuta (1975). Long-term studies on the neurotoxicity of small amount of methylmercury in monkeys (first report). In: Tsubaki T, ed. Studies on the Health Effects of Alkylmercury in Japan. Japan: Environment Agency, 63-70.

Scheuhammer, A.M. (1987). The chronic toxicity of aluminum, cadmium, mercury, and lead in birds: A review. *Environ. Pollut*. 46:263-295.

Scheuhammer, A.M. (1988). Chronic dietary toxicity of methylmercury in the zebra finch, *Poephila guttata. Bull. Environ. Contam. Toxicol.* 40:123-130.

Scheuhammer, A.M. (1991). Effects of acidification on the availability of toxic metals and calcium to wild birds and mammals. *Environ. Pollut*. 71:329-375.

Scheuhammer, A.M. and P.J. Blancher (1994). Potential risk to common loons (*Gavia immer*) from methylmercury exposure in acidified lakes. *Hydrobiologia* 279-289:445-455.

Schlegel, H., D.L. Godbold and A. Huttermann (1987). Whole plant aspects of heavy metal induced changes in CO_2 uptake and water relations of spruce (*Picea abies*) seedlings. *Physiol. Plant*. 69:265-270.

Schmidt, M. (1987). Atmosphärischer eintrag und interner umsatz von schwermetallen in waldökosystemen. ber. forschungszentr. Waldökosys./Waldst. A 34/37; Göttingen.

Schmitt, C.J. and W.G. Brumbaugh (1990). National contaminant biomonitoring program: Concentrations of arsenic, cadmium, copper, lead, mercury, selenium, and zinc in U.S. freshwater fish, 1976-1984. *Arch. Environ. Contam. Toxicol*. 19:731-747.

Schreiner, G., B. Ulbrich and R. Bass. 1986. Testing strategies in behavioral teratology: II. Discrimination learning. *Neurobehav. Toxicol. Teratol.* 8:567-572.

Scott, M.L. (1977). Effects of PCBs, DDT, and mercury compounds in chickens and Japanese quail. *Fed. Proc.* 36:1888-1893.

Scott, D.P. and F.A.J. Armstrong (1972). Mercury concentration in relation to size in several species of freshwater fishes from Manitoba and Northwestern Ontario. *J. Fish. Res. Bd. Can.* 29:1685-1690.

Sellers, P., C.A. Kelly, J.W.M. Rudd and A.R. MacHutchon (1996). Photodegradation of methylmercury in lakes. *Nature* 380:694-697.

SETAC (1994). Final Report: Aquatic Risk Assessment and Mitigation Dialogue Group. Society of Environmental Toxicology and Chemistry, Pensacola, FL.

Sheffy, T.B. and J.R. St. Amant (1982). Mercury burdens in furbearers in Wisconsin. *J. Wildl. Manage.* 46:1117-1120.

Siegel, S.M., B.Z. Siegel, N. Puerner and T. Speitel (1975). Water and soil biotic relations in mercury distribution. *Water Air Soil Pollut.* 4:9-18.

Siegel, S.M., B.Z. Siegel, C. Lipp, A. Kruckeberg, G.H.N. Towers and H. Warren (1985). Indicator plant-soil mercury patterns in a mercury-rich mining area of British Columbia. *Water Air Soil Pollut.* 25:73-85.

Siegel, S.M., B.Z. Siegel, C. Barghigiani, K. Aratani, P. Penny and D. Penny (1987). A contribution to the environmental biology of mercury accumulation in plants. *Water Air Soil Pollut.* 33:65-72.

Simonin, H.A., S.P. Gloss, C.T. Driscoll, C.L. Schofield, W.A. Kretser, R.W. Karcher and J. Symula (1994). Mercury in yellow perch from Adirondack drainage lakes (New York, U.S.). C.J. Watras and J.W. Huckabee (Eds), In: *Mercury Pollution Integration and Synthesis*, Lewis Publishers, Boca Raton, FL., USA. pp. 457-469.

Singleton, F.L. and R.K. Guthrie (1977). Aquatic bacterial populations and heavy metals - I. Composition of aquatic bacteria in the presence of copper and mercury salts. *Water Res.* 11:639-642.

Skurdal, J., T. Qvenild and O.K. Skogheim (1985). Mercury accumulation in five species of freshwater fish in Lake Tyrifjorden, southeast Norway, with emphasis on their suitability as test organisms. *Environ. Biol. Fish.* 14:233-237.

Slotton, D.G., J.E. Reuter and C.R. Goldman (1995). Mercury uptake patterns of biota in a seasonally anoxic Northern California reservoir. *Water Air Soil Pollut.* 80:841-850.

Solomon, K.R., D.B. Baker, R.P. Richards, K.R. Dixon, S.J. Klaine, T.W. La Point, R.J. Kendall, C.P. Weisskopf, J.M. Giddings, J.P. Giesy, L.W. Hall Jr. and W.M. Williams (1996). Ecological risk assessment of atrazine in North American surface waters. *Environ. Toxicol. Chem.* 15:31-76.

Sorensen, J.A., G.E. Glass, K.W. Schmidt, J.K. Huber and G.R. Rapp, Jr. (1990). Airborne mercury deposition and watershed characteristics in relation to mercury concentrations in water, sediments, plankton, and fish of eighty northern Minnesota lakes. *Environ. Sci. Technol.* 24:1716-1727.

Spalding, M.G., R.D. Bjork, G.V.N. Powell and S.F. Sundlof (1994). Mercury and cause of death in great white herons. *J. Wildl. Manage*. 58:735-739.

Spry, D.J. and J.G. Wiener (1991). Metal bioavailability and toxicity to fish in low-alkalinity lakes: A critical review. *Environ. Pollut*. 71:243-304.

St. Louis, V.L., J.W.M. Rudd, C.A. Kelly, K.G. Beaty, N.S. Bloom, and R.J. Flett (1994). Importance of wetlands as sources of methyl mercury to boreal forest ecosystems. *Can. J. Fish. Aquat. Sci.* 51:1065-1076.

Stanley, R.A. (1974). Toxicity of heavy metals and salts to Eurasian watermilfoil (*Myriophyllum spicatum* L.). *Arch. Environ. Contam. Toxicol*. 2:331-341.

State of Wisconsin (1989). Technical support document for NR 105, 1988. Wisconsin Administrative Code NR 105.07, 1989.

Stickel, L.F., W.H. Stickel, M.A.R. McLane and M. Bruns (1977). Prolonged retention of methyl mercury by mallard drakes. *Bull. Environ. Contam. Toxicol.* 18:393-400.

Stober, Q.J.. R.D. Jones and D.J. Scheidt (1995). Ultra trace level mercury in the everglades ecosystem, a multi-media canal pilot study. *Water Air Soil Pollut.* 80:991-1001.

Stoewsand, G.S., C.A. Bache and D.J. Lisk (1974). Dietary selenium protection of methylmercury intoxication of Japanese quail. *Bull. Environ. Contam. Toxicol.* 11:152-156.

Suchanek, T.H., P.J. Richerson, L.A. Woodward, D.G. Slotton, L.J. Holts and C.E.E. Woodmansee (1993). A survey and evaluation of mercury in: sediment, water, plankton, periphyton, benthic invertebrates and fishes within the aquatic ecosystem of Clear Lake, California. Preliminary Report, Prepared for the U.S. Environmental Protection Agency, Region 9: Superfund Program, by the Institute of Ecology, University of California at Davis, Davis, CA.

Sundlof, S.F., M.G. Spalding, J.D. Wentworth and C.K. Steible (1994). Mercury in livers of wading birds (Ciconiiformes) in Southern Florida. *Arch. Environ. Contam. Toxicol*. 27:299-305.

Suns, K. and G. Hitchin (1990). Interrelationships between mercury levels in yearling yellow perch, fish condition and water quality. *Water Air Soil Pollut*. 50:255-265.

Talmage S.S. and B.T. Walton (1993). Food chain transfer and potential renal toxicity of mercury to small mammals as a contaminated terrestrial field site. *Ecotoxicol*. 2:243-256.

Thomann, R.V. (1989). Bioaccumulation model for organic chemical distribution in aquatic food chains. *Environ. Sci. Technol*. 23:699-707.

Tremblay, A., M. Lucotte and D. Rowan (1995). Different factors related to mercury concentration in sediments and zooplankton of 73 Canadian lakes. *Water Air Soil Pollut.* 80:961-970.

Tremblay, A., M. Lucotte, M. Meili, L. Cloutier and P. Pichet (1996). Total mercury and methylmercury contents of insects from boreal lakes: ecological, spatial and temporal patterns. *Water Qual. Res. J. Can* 31:851-873.

Tremblay, A., M. Lucotte and I. Rheault (1996). Methylmercury in a benthic food web of two hydroelectric reservoirs and a natural lake of northern Quebec (Canada). *Water Air Soil Pollut.* 91:255-269.

U.S. Environmental Protection Agency (U.S. EPA, 1985). Ambient water quality criteria for mercury - 1984. EPA/440/5-84/026. Office of Water, Washington, D.C.

U.S. Environmental Protection Agency (U.S. EPA, 1989). Ecological assessment of hazardous waste sites: A field and laboratory reference. Corvallis, Oregon: Environmental Research Laboratory. EPA/600/3-89/013.

U.S. Environmental Protection Agency (U.S. EPA, 1992a). Peer review workshop on a framework for ecological risk assessment. Risk assessment forum. EPA/625/3-91/002.

U.S. Environmental Protection Agency (U.S. EPA, 1992b). A national study of chemical residues in fish. EPA 823/R-92/008. Office of Water Regulations and Standards, Washington, D.C.

U.S. Environmental Protection Agency (U.S. EPA, 1993a). Wildlife exposure factors handbook. EPA/600/R-93/187a. U.S. EPA Office of Research and Development, Washington, DC.

U.S. Environmental Protection Agency (U.S. EPA, 1993b). Great Lakes water quality initiative criteria documents for the protection of wildlife (proposed) DDT; Mercury; 2,3,7,8-TCDD; PCBs. EPA/822/R-93/006. U.S. EPA Office of Science and Technology, Washington, DC.

U.S. Environmental Protection Agency (U.S. EPA, 1993c). Water quality guidance for the Great Lakes system and correction: Proposed rules. *Fed. Regist.* 58(72):20802-21047 (April 16, 1993).

U.S. Environmental Protection Agency (U.S. EPA, 1994). Draft proceedings of the national wildlife criteria methodologies meeting, April 13-16, 1992, Charlottesville, VA. U.S. EPA Office of Water and Office of Science and Technology, Washington, DC.

U.S. Environmental Protection Agency (U.S. EPA, 1995a). Trophic level and exposure analyses for selected piscivorous birds and mammals. Volume I. Analysis for species of the Great Lakes Basin (Draft). U.S. EPA Office of Science and Technology, Washington, DC.

U.S. Environmental Protection Agency (U.S. EPA, 1995b). Final water quality guidance for the Great Lakes system: Final Rule. *Fed. Regist.* 60(56):15366-15425 (March 23, 1995).

U.S. Environmental Protection Agency (U.S. EPA, 1996). Proposed guidelines for ecological risk assessment. EPA/630/R-95/002B. Risk Assessment Forum, U.S. EPA, Washington, DC.

U.S. Fish and Wildlife Service (U.S. FWS, 1993). Mercury contamination in tissues of Florida bald eagles. Final Project Report. Prepared for the U.S. Fish and Wildlife Service, Department of the Interior, Washington, DC. (December, 1993)

Vermeer, K., F.A.J. Armstrong and D.R.M. Hatch (1973). Mercury in aquatic birds at Clay Lake, Western Ontario. *J. Wildl. Manage.* 37:58-61.

Watras, C.J. and N.S. Bloom (1992). Mercury and methylmercury in individual zooplankton: Implications for bioaccumulation. *Limnol. Oceanogr*. 37:1313-1318.

Watras, C.J., K.A. Morrison, J. Host and N.S. Bloom (1995a). Concentration of mercury species in relationship to other site-specific factors in the surface waters of northern Wisconsin lakes. *Limnol. Oceanogr.*40:556-565.

Watras, C.J., K.A. Morrison and N.S. Bloom (1995b). Mercury in remote Rocky Mountain lakes of Glacier National Park, Montana, in comparison with other temperate North American regions. *Can. J. Fish. Aquat. Sci.* 52:1220-1228.

Watras, C.J., K.A. Morrison and N.S. Bloom (1995c). Chemical correlates of Hg and Methyl-Hg in northern Wisconsin lake waters under ice-cover. *Water Air Soil Pollution*. 84:253-267.

Watras, C.J., K.A. Morrison, J.S. Host and N.S. Bloom (1995). Concentration of mercury species in relationship to other site-specific factors in the surface waters of northern Wisconsin lakes. *Limnol. Oceanogr*. 40:556-565.

Weber, J.H. (1993). Review of possible paths for abiotic methylation of mercury (II) in the aquatic environment. *Chemosphere* 26:2063.

Weil, C.S. and D.D. McCollister (1963). Relationship between short- and long-term feeding studies in designing an effective toxicity test. *Agric. Food Chem.* 11:486-491.

Weis, J.S. and P. Weis (1989). Tolerance and stress in a polluted environment. *BioScience* 39:89-95.

Weis, J.S. and P. Weis (1995). Effects of embryonic exposure to methylmercury on larval prey-capture ability in the mummichog, *Fundulus heteroclitus. Environ. Toxicol. Chem*. 14:153-156.

Welch, L.J. (1994). Contaminant burdens and reproductive rates of bald eagles breeding in Maine. Ph.D. dissertation, University of Maine, Orono, ME.

Wells, J.R., P.B. Kaufman and J.D. Jones (1980). Heavy metal contents in some macrophytes from Saginaw Bay (Lake Huron, USA). *Aquat. Bot.* 9:185-193.

Westermark, T., T. Odsjö, T. and A.G. Johnels (1975). Mercury in bird feathers before and after the Swedish ban on alkyl mercury in agriculture. *Ambio* 4:87-97.

Wiemeyer, S.N., T.G. Lamont, C.M. Bunck, C.R. Sindelar, F.J. Gramlich, J.D. Fraser and M.A. Byrd (1984). Organochlorine pesticide, polychlorobiphenyl, and mercury residues in bald eagle eggs - 1969-1979 - and their relationships to shell thinning and reproduction. *Arch. Environ. Contam. Toxicol.* 13:529-549.

Wiemeyer, S.N., D.M. Bunck and C.J. Stafford (1993). Environmental contaminants in bald eagle eggs - 1980-1984 - and further interpretations of relationships to productivity and shell thickness. *Arch. Environ. Contam. Toxicol.* 24:213-227.

Wiener, J.G., G.A. Jackson, T.W. May and B.P. Cole (1982). Longitudinal distribution of trace elements (As, Cd, Cr, Hg, Pb, and Se) in fishes and sediments in the upper Mississippi River. In: *Contaminants in the Upper Mississippi River.* J.G. Wiener, R.V. Anderson and D.R. McConville (Eds.). Butterworth Publishers, Boston, MA. p. 139-170.

Wiener, J.G., R.E. Martini, T.B. Sheffy and G.E. Glass (1990). Factors influencing mercury concentrations in walleyes in northern Wisconsin lakes. *Trans. Amer. Fish. Soc.* 119:862-870.

Wiener, J.G. and D.J. Spry (1996). Toxicological significance of mercury in freshwater fish. In: *Envrionmental Contaminants in Wildlife: Interpreting Tissue Concentrations.* W.N. Beyer, G.H. Heinz and A.W. Redman-Norwood (Eds.), Special Publication of the Society of Environmental Toxicology and Chemistry, Lewis Publishers, Boca Raton, FL, USA. pp. 297-339.

Winfrey, M.R. and J.W.M. Rudd (1990). Review - Environmental factors affecting the formation of methylmercury in low pH lakes. *Environ. Toxicol. Chem.* 9:853-869.

Wobeser, G. (1973). Aquatic mercury pollution: studies of its occurrence and pathologic effects on fish and mink. Ph.D. dissertation, University of Saskatchewan, Saskatoon, Saskatchewan, Canada.

Wobeser, G. and M. Swift III (1976). Mercury poisoning in a wild mink. *J. Wildl. Dis.* 12:335-340.

Wobeser, G., N.D. Nielsen and B. Schiefer (1976a). Mercury and mink I: The use of mercury contaminated fish as a food for ranch mink. *Can. J. Comp. Med.* 40:30-33.

Wobeser, G., N.D. Nielsen and B. Schiefer (1976b). Mercury and mink II: Experimental methyl mercury intoxication. *Can. J. Comp. Med.* 40:34-45.

Wood, P.B., J.H. White, A. Steffer, J.M. Wood, C.F. Facemire, H.F. Percival (1996). Mercury concentrations in tissues of Florida bald eagles. *J. Wildl. Manage.* 60:178-185.

World Health Organization (WHO, 1989). Environmental health criteria 86: Mercury - environmental aspects.

Wren, C.D. (1985). Probable case of mercury poisoning in a wild otter, *Lutra canadensis*, in northwestern Ontario. *Can. Field Nat.* 99:112-114.

Wren, C.D. (1986). A review of metal accumulation and toxicity in wild mammals. I. Mercury. *Environ. Res.* 40:210-244.

Wren, C.D. (1991). Cause-effect linkages between chemicals and populations of mink (*Mustela vison*) and otter (*Lutra canadensis*) in the Great Lakes basin. *J. Toxicol. Environ. Health* 33:549-585.

Wren, C.D., H.R. MacCrimmon and B.R. Loescher (1983). Examination of bioaccumulation and biomagnification of metals in a precambrian shield lake. *Water Air Soil Pollut.* 19:277-291.

Wren, C.D. and H.R. MacCrimmon (1986). Comparative bioaccumulation of mercury in two adjacent freshwater ecosystems. *Water Res.* 20:763-769.

Wren, C.D., P.M. Stokes and K.L. Fischer (1986). Mercury levels in Ontario mink and otter relative to food levels and environmental acidification. *Can. J. Zool.* 64:2854-2859.

Wren, C.D., D.B. Hunter, J.F. Leatherland, and P.M. Stokes (1987a). The effects of polychlorinated biphenyls and methylmercury, singly and in combination on mink. I: Uptake and toxic responses. *Arch. Environ. Contam. Toxicol.* 16:441-447.

Wren, C.D., D.B. Hunter, J.F. Leatherland, and P.M. Stokes (1987b). The effects of polychlorinated biphenyls and methylmercury, singly and in combination on mink. II: Reproduction and kit development. *Arch. Environ. Contam. Toxicol.* 16:449-454.

Wren, C.D. and G.L. Stephenson (1991). The effect of acidification on the accumulation and toxicity of metals to freshwater invertebrates. *Environ. Pollu.* 71:205-241.

Wright, D.R. and R.D. Hamilton (1982). Release of methyl mercury from sediments: effects of mercury concentration, low temperature, and nutrient addition. *Can. J. Fish Aquat. Sci.* 39:1459-1466.

Xun, L., N.E.R. Campbell and J.W.M. Rudd (1987). Measurements of specific rates of net methyl mercury production in the water column and surface sediments of acidified and circumneutral lakes. *Can. J. Fish. Aquat. Sci.* 44:750-757.

Yannai, S., I. Berdicevsky and L. Duek (1991). Transformations of inorganic mercury by *Candida albicans* and *Saccharomyces cerevisiae*. *Appl. Environ. Micro.* 57:245-247.

Yin, Y., H.E. Allen, Y. Li, C.P. Huang and P.F. Saunders (1996). Adsorption of mercury (II) by soil: effects of pH, chloride, and organic matter. *J. Environ. Qual.* 25:837-844.

Zalups, R.K. and L.H. Lash (1994). Advances in understanding the renal transport and toxicity of mercury. *J. Toxicol. Environ. Health* 42:1-44.

Zelles, L., I. Scheunert and F. Korte (1986). Comparison of methods to test chemicals for side effects on soil microorganisms. *Ecotoxicol. Environ. Safety* 12:53-69.

Zillioux, E.J., D.B. Porcella and J.M. Benoit (1993). Mercury cycling and effects in freshwater wetland ecosystems. *Environ. Toxicol. Chem.* 12:2245-2264.