PHASE 2 REPORT
FURTHER SITE CHARACTERIZATION AND ANALYSIS

VOLUME 2F - REVISED HUMAN HEALTH RISK ASSESSMENT

HUDSON RIVER PCBs REASSESSMENT RI/FS

NOVEMBER 2000

For

U.S. Environmental Protection Agency
Region 2
and
U.S. Army Corps of Engineers
Kansas City District

Book 1 of 1
Upper Hudson River
Mid-Hudson River

TAMS Consultants, Inc.
Gradient Corporation

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
REGION 2
290 BROADWAY
NEW YORK, NY 10007-1866

November 29, 2000

To All Interested Parties:

The U.S. Environmental Protection Agency (USEPA) is pleased to release the Revised Baseline Human Health Risk Assessment (Revised HHRA), which is part of Phase 2 of the Reassessment Remedial Investigation/Feasibility Study (Reassessment RI/FS) for the Hudson River PCBs Superfund Site. The Revised HHRA evaluates current and future cancer risks and non-cancer health hazards posed by PCBs in the Upper and Mid-Hudson River in the absence of remediation of PCBs in sediments of the Upper Hudson River and institutional controls, such as the fish consumption advisories currently in place.

The Revised HHRA shows that cancer risks and non-cancer health hazards to people who eat fish from the Upper and Mid-Hudson River are at levels that are above USEPA's levels of concern. USEPA strongly recommends that everyone follow the current fish consumption advisories.

On May 30–31, 2000, USEPA, through its contractor, Eastern Research Group (ERG), convened a panel of independent scientific experts to conduct a peer review of the August 1999 Human Health Risk Assessment for the Upper Hudson River and the March 2000 Responsiveness Summary for that report. In conjunction with this Revised ERA, USEPA is issuing a Response to Peer Review Comments on the Human Health Risk Assessment for the Upper Hudson River. The November 2000 Response to Peer Review Comments describes how USEPA incorporated the peer review comments or provides the technical rationale for not incorporating a comment.

The Revised HHRA combines into a single report the August 1999 HHRA for the Upper Hudson River, the March 2000 Responsiveness Summary, and the November 2000 Response to Peer Review Comments. The Revised HHRA also includes revisions to the December 1999 HHRA for the Mid-Hudson River and the August 2000 Responsiveness Summary for that report. USEPA is using the results of the Revised HHRA to establish acceptable PCB exposure levels, which will in turn be used to develop remedial alternatives for the PCBs in the sediments of the Upper Hudson River.

If you need additional information regarding the Revised HHRA or the Reassessment RI/FS, please contact Ann Rychlenski at 212-637-3672.

Sincerely yours,

William McCabe

Richard L. Caspe, Director
Emergency and Remedial Response Division

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LIST OF ACRONYMS

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1-D	One Dimensional
2-D	Two Dimensional
ATSDR	Agency for Toxic Substances and Disease Registry
CDI	Chronic Daily Intake
CERCLA	Comprehensive Environmental Response, Compensation, and Liability Act
COC	Chemical of Concern
COPC	Chemical of Potential Concern
CSF	Carcinogenic Slope Factor
CT	Central Tendency Exposure
CV	Coefficient of Variation
EPC	Exposure Point Concentration
ERA	Baseline Ecological Risk Assessment
ERG	Eastern Research Group
FDA	Food and Drug Administration
FS	Feasibility Study
GE	General Electric Company
GLSFTFA	Great Lakes Sport Fish Task Advisory
GM	Geometric Mean
GSD	Geometric Standard Deviation
HHRA	Human Health Risk Assessment
HHRASOW	Human Health Risk Assessment Scope of Work
HI	Hazard Index for Non-Cancer Health Effects
HQ	Hazard Quotient for Non-Cancer Health Effects
IRIS	Integrated Risk Information System
ISC	Industrial Source Complex
ISCLT	Industrial Source Complex Long Term
ISCST	Industrial Source Complex Short Term
LOAEL	Lowest Observed Adverse Effects Level
LN	Log
MF	Modifying Factor
NCP	National Oil and Hazardous Substances Pollution Contingency Plan
NOAA	National Oceanic and Atmospheric Administration
NOAEL	No Observed Adverse Effects Level
NPL	National Priorities List
NYSDEC	New York State Department of Environmental Conservation
NYSDOH	New York State Department of Health
PAHS	Polycyclic Aromatic Hydrocarbons
PCB	Polychlorinated Biphenyl
RAGS	Risk Assessment Guidance for Superfund
RBMR	Revised Baseline Modeling Report
RfD	Reference Dose

LIST OF ACRONYMS

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RI	Remedial Investigation
RI/FS	Remedial Investigation/Feasibility Study
RM	River Mile
RME	Reasonable Maximum Exposure
ROD	Record of Decision
SARA	Superfund Amendments and Reauthorization Act of 1986
TCDD	2,3,7,8-Tetrachlorodibenzo-p-dioxin
TEF	Toxicity Equivalency Factor
TSCA	Toxic Substances Control Act
UCLM	Upper Confidence Limit on the Mean
UF	Uncertainty Factor
USEPA	United States Environmental Protection Agency
USF&W	United States Fish and Wildlife

REVISED HUMAN HEALTH RISK ASSESSMENT: UPPER AND MID-HUDSON RIVER
EXECUTIVE SUMMARY
NOVEMBER 2000

This document presents the Revised Human Health Risk Assessment (Revised HHRA) for the Upper and Mid-Hudson River, which is part of Phase 2 of the Reassessment Remedial Investigation/Feasibility Study (Reassessment RI/FS) for the Hudson River PCBs site in New York. The Revised HHRA quantitatively evaluates cancer risks and non-cancer health hazards from exposure to polychlorinated biphenyls (PCBs) in the Upper Hudson River, which extends from Hudson Falls, New York to the Federal Dam at Troy, New York, and in the Mid-Hudson River, which extends from the Federal Dam to Poughkeepsie, New York. The Revised HHRA evaluates current and future risks to young children, adolescents, and adults under baseline conditions, which means in the absence of any remedial action and institutional controls such as the current fish consumption advisories. The Revised HHRA uses current U.S. Environmental Protection Agency (USEPA) policy and guidance as well as additional site data and analyses to update USEPA's 1991 baseline risk assessment.

On May 30-31, 2000, USEPA, through its contractor Eastern Research Group (ERG), convened a panel of independent scientific experts to conduct a peer review of the August 1999 Human Health Risk Assessment for the Upper Hudson River, consistent with the Agency's Peer Review Handbook (USEPA, 1998d). The peer reviewers were asked to respond to USEPA's charge questions, which covered each component of the risk assessment. The specific charge questions and information about the peer review are presented in the "Report on the Peer Review of the Hudson River PCBs Human Health Risk Assessment" (ERG, 2000) (the "Peer Review Report"). In conjunction with this Revised HHRA, USEPA is issuing a Response to Peer Review Comments on the Human Health Risk Assessment (USEPA, 2000h). Consistent with Peer Review Handbook, the Response to Peer Review Comments describes where in the Revised HHRA USEPA incorporated the peer review comments or provides the technical rationale for not incorporating a comment.

USEPA uses risk assessment as a tool to evaluate the likelihood and degree of chemical exposure and the possible adverse health effects associated with such exposure. The basic steps of the Superfund human health risk assessment process are the following: 1) Data Collection and Analysis to determine the nature and extent of chemical contamination in environmental media, such as sediment, water, and fish; 2) Exposure Assessment, which is an identification of possible exposed populations and an estimation of human chemical intake through exposure routes such as ingestion, inhalation, or skin contact; 3) Toxicity Assessment, which is an evaluation of chemical toxicity including cancer and non-cancer health effects from exposure to chemicals; and 4) Risk Characterization, which describes the likelihood and degree of chemical exposure at a site and the possible adverse health effects associated with such exposure.

Consistent with USEPA regulations, the risk managers in the Superfund program evaluate the cancer risks and non-cancer health hazards to the reasonably maximally exposed (RME) individual in the decision-making process. The Revised HHRA shows that cancer risks and non-cancer health hazards to the RME individual associated with ingestion of PCBs in fish from the Upper Hudson River and Mid-Hudson River are above levels of regulatory concern. Cancer risks and non-cancer health hazards from other exposure pathways are generally below levels of concern. The results of the Revised HHRA will help establish acceptable exposure levels for use in developing remedial alternatives to address the PCBs in contaminated sediments in the Upper Hudson River, which is Phase 3 (Feasibility Study) of the Reassessment RI/FS.

UPPER HUDSON RIVER

DATA COLLECTION AND ANALYSIS

USEPA previously released reports on the nature and extent of contamination at the Hudson River PCBs Site as part of the Reassessment RI/FS (*e.g.*, February 1997 Data Evaluation and Interpretation Report, July 1998 Low Resolution Sediment Coring Report, Database for the Hudson River PCBs Reassessment RI/FS [Release 5.0, October 2000], January 2000 Revised Baseline Modeling Report (RBMR), and associated responsiveness summaries). The Reassessment RI/FS documents provide current and future concentrations of PCBs in fish, sediments, and river water and form the basis of the site data collection and analyses used in conducting the Revised HHRA.

EXPOSURE ASSESSMENT

Adults (over 18 years old), adolescents (aged 7-18), and young children (aged 1-6) are potentially exposed to PCBs in the Upper Hudson River due to fishing and recreational activities (swimming, wading), as well as from living adjacent to the Upper Hudson River and inhaling volatilized PCBs in the air. Cancer risks and non-cancer health hazards were calculated for each of these segments of the exposed population. To provide information on the possible variability of PCB exposure, both an average (central tendency) exposure estimate and an RME estimate were calculated. The RME is the maximum exposure that is reasonably expected to occur in the Upper Hudson River under baseline conditions and is not a worst-case exposure scenario.

The exposure pathways identified are ingestion of fish, incidental ingestion of sediments, dermal contact with sediments and river water, and inhalation of volatilized PCBs in air. For these exposure pathways, central tendency and RME estimates were calculated using point estimate analyses, whereby a single point estimate was selected for each exposure factor used in the calculations of cancer risks and non-cancer health hazards. Incidental ingestion of river water while swimming was not quantitatively evaluated because the river water meets federal drinking water standards for PCBs established under the Safe Drinking Water Act.

In addition to the point estimate analysis, a Monte Carlo analysis was performed to provide a range of estimates of the cancer risks and non-cancer health hazards associated with the fish ingestion pathway. The Monte Carlo analysis helps to evaluate variability in exposure parameters (*e.g.*, differences within a population's fish ingestion rates, number of years an angler is exposed, body weight) and uncertainty (*i.e.,* a lack of complete knowledge about specific variables).

Ingestion of Fish

For fish ingestion, both central tendency and RME estimates were developed for each of the parameters needed to calculate the cancer risks and non-cancer health hazards. Based on the 1991 New York Angler survey of fish consumption by licensed anglers (Connelly *et al.*, 1992), the central tendency fish ingestion rate was determined to be 4.0 grams per day, or about six half-pound meals per year and the RME fish ingestion rate was determined to be 31.9 grams per day, or about 51 half-pound meals per year for adults. Fish ingestion rates for adolescents and young children were reduced based on the ratio of adolescent or child body weight to that of an adult.

For the point estimate analyses, cancer risks and non-cancer health hazards to an adult, adolescent, and young child angler were calculated. Population mobility data from the U.S. Census Bureau for the five counties surrounding the Upper Hudson River and fishing duration data from the 1991

New York Angler survey were used to determine the length of time an angler fishes in the Upper Hudson River (*i.e.*, exposure duration). The exposure duration for fish ingestion was 12 years for the central tendency exposure estimate (total of 6, 3, and 3 years for adult, adolescent, and young child exposures, respectively), 40 years for the RME exposure estimate for cancer (total of 22, 12, and 6 years for adult, adolescent, and young child exposures, respectively). The exposure duration of 7 years for the RME exposure estimate for non-cancer health assessment was selected because it is an exposure period for chronic non-cancer health effects that yields a high end average daily dose based on the modeled decline in PCB concentration with time. Standard USEPA default factors were used for angler body weight. Future concentrations of PCBs in fish were derived from forecasts presented in the RBMR, which were then grouped by fish species and averaged over species for the entire Upper Hudson River. PCB losses during cooking were assumed to be 20% for the central tendency exposure estimate and 0% (no loss) for the RME estimate, based on studies reported in the scientific literature.

In the Monte Carlo analysis, each exposure parameter (*e.g.*, ingestion rate, exposure duration, body weight) was represented by a range of values, each with an assigned probability, rather than as a single point estimate. Cancer risks and non-cancer health hazards were calculated for anglers beginning at age 10. Differences in the length of time an angler fishes the Upper Hudson (exposure duration) were obtained from the 1991 New York Angler survey and the U.S. Census data. Differences in angler body weight through time were obtained from national health surveys summarized in the scientific literature. Future concentrations of PCBs in fish were derived from the RBMR. Fish species consumption variability was evaluated based on consumption patterns determined from the 1991 New York Angler survey and within-species PCB concentrations were averaged over location within the Upper Hudson River. The variability and uncertainty in fish ingestion rates was examined by considering surveys of fish ingestion rates from four independent studies. Uncertainty in PCB cooking loss was determined from a review of the scientific literature.

Due to the lack of sufficient information available to define quantitative uncertainty distributions for several important exposure factors, an explicit two-dimensional Monte Carlo analysis which examines variability and uncertainty separately could not be performed. Instead, an expanded one-dimensional (1-D) analysis was completed using a sensitivity/uncertainty analysis. Each 1-D Monte Carlo simulation, consisting of 10,000 simulated anglers, examined variability of PCB intake due to inherent variability of exposure among individuals within the population. Ranking the 10,000 outcomes for cancer risk or non-cancer health hazards from each separate simulation provides an estimate of the distribution of cancer risk and non-cancer health hazards for an individual within the angler population. The 1-D analyses were then repeated for a range of possible input distributions for important exposure variables. A total of 72 separate combinations of the variable input parameters was examined in the 1-D analysis.

Other Exposure Pathways

For direct exposure to river water and sediment, there are no available data that quantify the frequency of exposure. The central tendency exposure estimates for adults and young children (aged 1-6) were assumed to be one day every other week for the 13 weeks of summer (7 days/year) and for the RME were assumed to be one day per week for the 13 weeks of summer (13 days/year). Adolescents (aged 7-18) were assumed to have about three times more frequent exposure, with a central tendency exposure estimate of 20 days/year and an RME estimate of 39 days/year. In addition, an avid recreator scenario was evaluated where the central tendency exposure estimate for adults, adolescents, and young children was assumed to be approximately twice a week for 6 months of the year (52 days/year), and the RME was assumed to be four times a week for 6 months of the year (104 days/year). The concentrations of PCBs in water and sediment were derived from the RBMR.

The cancer risks due to possible inhalation of PCBs in air were evaluated for both recreational users of the river (swimmers and waders) as well as for residents living adjacent to the Upper Hudson River. The concentrations of PCBs in air were calculated from a combination of historical monitoring data and modeled emissions from the river using a USEPA-recommended air dispersion model. Non-cancer health hazards from inhalation of volatilized PCBs could not be calculated due to a lack of toxicity factors for this exposure pathway.

TOXICITY ASSESSMENT

The toxicity assessment is an evaluation of the chronic (7 years or more) adverse health effects from exposure to PCBs (USEPA, 1989b). Consistent with Superfund guidance, two types of adverse health effects were evaluated: 1) the incremental risk of developing cancer due to exposure to chemicals and 2) the hazards associated with non-cancer health effects, such as reproductive impairment, developmental disorders, disruption of specific organ functions, and learning problems. The cancer risk is expressed as a probability and is based on the cancer potency of the chemical, known as a cancer slope factor, or CSF. The non-cancer health hazard is expressed as the ratio of the chemical intake (dose) to a Reference Dose, or RfD. The chronic RfD represents an estimate (with uncertainty spanning perhaps an order of magnitude or greater) of a daily exposure level for the human population, including sensitive populations (*e.g.*, children), that is likely to be without an appreciable risk of deleterious effects during a lifetime. Chemical exposures exceeding the RfD do not predict specific diseases. USEPA's Integrated Risk Information System, known as IRIS, provides the primary database of chemical-specific toxicity information used in Superfund risk assessments. The most current CSFs and RfDs for PCBs were used in calculating cancer risks and non-cancer health hazards for all pathways. For fish ingestion, the pathway determined to be of greatest concern, CSFs of 2 $(mg/kg\text{-}day)^{-1}$ and 1 $(mg/kg\text{-}day)^{-1}$ were used for the RME and central tendency exposure, respectively, and an RfD for Aroclor 1254 of 2×10^{-5} mg/kg-day was used for the RME and central tendency exposures.

PCBs are a group of synthetic organic chemicals consisting of 209 individual chlorinated biphenyls called congeners. Some PCB congeners are considered to be structurally similar to dioxin and are called dioxin-like PCBs. USEPA has classified PCBs as probable human carcinogens, based on a number of studies in laboratory animals showing liver tumors. In IRIS, USEPA characterizes the data for exposures to PCB mixtures as "inadequate but suggestive" of causing cancer in humans. As stated in USEPA's 1996 reassessment of PCB cancer toxicity, the uncertainty around the CSF estimates extends in both directions, *i.e.*, contributing to potential underestimates or overestimates of cancer potency. However, the CSFs developed by the USEPA represent plausible upper bound estimates, which means that the USEPA is reasonably confident that the actual cancer risk will not exceed the estimated risk calculated using the CSF (USEPA, 1986; 1996c).

As recommended by the peer reviewers (ERG, 2000), the Revised HHRA examines recent PCB toxicity studies published since 1996. Selected epidemiological and population studies are summarized in Appendix D. These published studies indicate both positive and negative causal relationships between PCB exposure and cancer. However, inherent limitations in the studies, as discussed in this Revised HHRA, continue to be debated. Several recent national and international human epidemiological studies of non-cancer health effects (*i.e.*, developmental and neurotoxic effects), which are summarized in Appendix D, are being reevaluated by USEPA as part of the IRIS process in addition to animal toxicity studies and supporting studies. Until the Agency's reevaluation is completed, it is unclear whether the RfD will change and the effect of that change, if any, on the conclusions of the Revised HHRA.

RISK CHARACTERIZATION

Point Estimate Calculations

Ingestion of fish contaminated with PCBs resulted in the highest lifetime cancer risks. The RME estimate of the increased risk of an individual developing cancer averaged over a lifetime (childhood through adulthood), based on the exposure assumptions in this Revised HHRA, is 1×10^{-3}, or one in 1,000 increased chance of developing cancer. The RME cancer risks associated with the dioxin-like PCBs are comparable. The central tendency (average) estimate of cancer risk is 3×10^{-5}, or 3 in 100,000. For probable human carcinogens such as PCBs, acceptable exposure levels for Superfund are generally those that represent an incremental upper bound lifetime cancer risk to an RME individual of between 10^{-4} and 10^{-6} (1 in 10,000 to 1 in 1,000,000).

Estimated RME and central tendency cancer risks relating to PCB exposure in sediment and water while swimming or wading, or from inhalation of volatilized PCBs in air by residents living near the river, are much lower than those for fish ingestion, falling generally at the low end, or below, the range of 10^{-4} to 10^{-6}. A summary of the point estimate calculations for cancer risk is presented in the table below.

Point Estimate Cancer Risk Summary – Upper Hudson River*

Pathway	Central Tendency Risk	RME Risk
Ingestion of Fish		
Total*	3×10^{-5} (3 in 100,000)	1×10^{-3} (1 in 1,000)
Adult	1×10^{-5} (1 in 100,000)	6×10^{-4} (6 in 10,000)
Adolescent	7×10^{-6} (7 in 1,000,000)	4×10^{-4} (4 in 10,000)
Child	1×10^{-5} (1 in 100,000)	4×10^{-4} (4 in 10,000)
Exposure to Sediment		
baseline recreator	2×10^{-7} (2 in 10,000,000)	2×10^{-6} (2 in 1,000,000)
avid recreator	1×10^{-6} (1 in 1,000,000)	9×10^{-6} (9 in 1,000,000)
Exposure to Water		
baseline recreator	3×10^{-8} (3 in 100,000,000)	2×10^{-7} (2 in 10,000,000)
avid recreator	1×10^{-7} (1 in 10,000,000)	1×10^{-6} (1 in 1,000,000)
Inhalation of Air	2×10^{-8} (2 in 100,000,000)	1×10^{-6} (1 in 1,000,000)

**Total risk for young child (aged 1-6), adolescent (aged 7-18), and adult (over 18).*

The evaluation of non-cancer health effects involved comparing the average daily exposure levels (dose) to determine whether the estimated exposures exceed the Reference Dose. The ratio of the site-specific calculated dose to the RfD for each exposure pathway is summed to calculate the Hazard Index (HI) for the exposed individual. An HI of one (1) is the reference level established by USEPA above which concerns relating to non-cancer health effects must be evaluated.

Ingestion of fish resulted in the highest HI values. The RME HI is 104, 71, and 65, for the young child, adolescent, and adult, respectively, compared to the Agency's level of concern of an HI of 1 or less. The central tendency non-cancer HIs are 12, 8, and 7 for the young child, adolescent, and adult, respectively, which are also above USEPA's level of concern.

Non-cancer health hazards due to inhalation of PCBs were not calculated because IRIS does not contain a toxicity value for inhalation of PCBs. A summary of the point estimate non-cancer health hazards is presented below.

Point Estimate Non-Cancer Hazard Summary – Upper Hudson River*		
Pathway	**Central Tendency Non-Cancer Hazard Index**	**RME Non-Cancer Hazard Index**
Ingestion of Fish		
Adult	7	65
Adolescent	8	71
Young Child	12	104
Exposure to Sediment		
baseline recreator	0.03	0.04
avid recreator	0.2	0.3
Exposure to Water		
baseline recreator	0.01	0.02
avid recreator	0.06	0.1
Inhalation of Air**	Not Calculated	Not Calculated

**Values for young child or adolescent, which are higher than adult for these pathways.*

***Non-cancer hazards were not calculated for the inhalation pathway due to a lack of non-cancer toxicity values for this pathway.*

Consistent with the National Oil and Hazardous Substances Pollution Contingency Plan, USEPA uses the RME cancer risks and non-cancer HI values in determining whether conditions at a site are above or below levels of concern. The central tendency cancer risks and non-cancer health hazards are provided to more fully characterize the variability/uncertainty of cancer risks and non-cancer health hazards among individuals within the potentially exposed population, by describing the health effects associated with average exposure. The uncertainty of the calculated cancer risks and non-cancer HIs may exceed an order of magnitude (*e.g.*, 10-fold), however the results from the Monte Carlo Analysis suggest the uncertainty is less than 100-fold.

Because this Revised HHRA is for current and future exposures to PCBs from the Upper Hudson River (*i.e.*, exposures beginning in 1999), the estimated cancer risks and non-cancer health hazards in this assessment are incremental and overlay possible cancer risks and non-cancer health hazards due to prior exposures to PCBs from the River. Furthermore, the Revised HHRA does not examine exposures to environmental contaminants from other possible sources. In other words, any exposure of individuals to PCBs from the Upper Hudson River before 1999 or from sources other than the Hudson River would be in addition to the cancer risks and non-cancer health hazard estimates presented in the Revised HHRA.

Monte Carlo Estimates

In the August 1999 HHRA, USEPA presented the results of its Monte Carlo analysis for the fish ingestion pathway. That analysis, which was composed of 72 combinations of input parameters for 10,000 simulated anglers (for a total of 720,000 computer simulations), showed that USEPA's RME point estimates were appropriately within the high-end ($\geq 90^{th}$ percentile) for cancer risks and non-cancer health hazards (see, comparison of point estimates and Monte Carlo analysis, below). Because the peer

reviewers did not recommend any changes to the most important input parameters for the fish ingestion pathway (*e.g.*, fish ingestion rate, exposure duration, exposure point concentration in fish) and due to the extensive level of effort required to perform Monte Carlo analysis, USEPA did not conduct another Monte Carlo analysis for the Revised HHRA. Nevertheless, suggestions from the peer reviewers to better explain and describe the August 1999 Monte Carlo analysis were incorporated into the Revised HHRA. Furthermore, as discussed in the March 2000 Human Health Risk Assessment Responsiveness Summary (USEPA, 2000b), there was a 2-fold, or less, difference in the modeled PCB concentrations in fish when the August 1999 results are compared to those used in this Revised HHRA. Thus, even if the Monte Carlo analysis had been updated to account for the revised modeling results, the overall outcome and conclusions would not change substantially.

In the Monte Carlo analysis, a distribution of cancer risks and non-cancer health hazards was calculated for the fish ingestion pathway. The tables below summarize the low-end (5^{th} percentile), midpoint (50^{th} percentile), and high-end ($\geq 90^{th}$ percentile) cancer risks and non-cancer health hazards. The base case, low estimate, and high estimate of the 72 combinations examined are given at each risk percentile. The interpretation of the risks at any particular percentile is given by the following example. For the base case in the table below, the calculated incremental cancer risk at the 95^{th} percentile is 9×10^{-4}, which means that the cancer risks for 95 percent of the population is equal to or less than 9×10^{-4}, or 9 in 10,000. Conversely, the cancer risks for 5 percent of the population exceed this value. For any particular risk percentile, the "low" and "high" estimates represent the lowest and highest cancer risk and HI estimates of the 72 combinations examined.

Monte Carlo Cancer Risk Summary For Fish Ingestion – Upper Hudson River

Risk Percentile	Low Estimate	Base Case	High Estimate
5^{th} Percentile	7×10^{-7}	5×10^{-6}	5×10^{-5}
50^{th} Percentile	1×10^{-5}	6×10^{-5}	4×10^{-4}
90^{th} Percentile	7×10^{-5}	5×10^{-4}	2×10^{-3}
95^{th} Percentile	1×10^{-4}	9×10^{-4}	3×10^{-3}
99^{th} Percentile	3×10^{-4}	4×10^{-3}	1×10^{-2}

Monte Carlo Non-Cancer Hazard Index Summary - Fish Ingestion – Upper Hudson River

Risk Percentile	Low Estimate	Base Case	High Estimate
5^{th} Percentile	0.1	1	7
50^{th} Percentile	2	11	51
90^{th} Percentile	5	31	117
95^{th} Percentile	11	82	233
99^{th} Percentile	19	136	366

Comparison of Point Estimates and Monte Carlo Analysis

The Monte Carlo base case scenario is the one from which point estimate exposure factors for fish ingestion were drawn, thus the point estimate RMEs and the Monte Carlo base case estimates can be compared. Similarly, the point estimate central tendency (average) and the Monte Carlo base case midpoint (50^{th} percentile) are comparable. For cancer risk, the point estimate RME for fish ingestion (1×10^{-3}) falls approximately at the 95^{th} percentile from the Monte Carlo base case analysis. The point estimate central tendency value (3×10^{-5}) and the Monte Carlo base case 50^{th} percentile value (6×10^{-5}) are similar. For non-cancer health hazards, the point estimate RME for fish ingestion (104 for young child) falls between the 95^{th} and 99^{th} percentiles of the Monte Carlo base case. The point estimate central tendency HI (12 for young child) is approximately equal to the 50^{th} percentile of the Monte Carlo base case HI of 11.

Mid-Hudson River

DATA COLLECTION AND ANALYSIS

The data used to assess cancer risks and non-cancer health hazards in the Mid-Hudson include the data used for the Upper Hudson assessment, as well as forecasts of future concentrations of PCBs below the Federal Dam at Troy as reported in the Revised Baseline Ecological Risk Assessment (USEPA, 2000f) and its responsiveness summary (USEPA, 2000g). The revised model forecasts were based on revised PCB loads to the Lower Hudson as summarized in the RBMR (USEPA, 2000a).

EXPOSURE ASSESSMENT

For the fish ingestion pathway, the same ingestion rate was used for the Mid-Hudson as was adopted for the Upper Hudson, based on the state-wide 1991 New York Angler survey (Connelly *et al.*, 1992). The exposure durations were calculated in a similar manner, based on population mobility data from the U.S. Census for the six counties surrounding the Mid-Hudson River, and were found to be essentially the same as those derived for the Upper Hudson (*i.e.*, 40 years for RME cancer assessment, 12 years for CT cancer and non-cancer assessments). The same exposure duration of 7 years for RME non-cancer assessment was used in the Mid-Hudson as in the Upper Hudson, based on a high-end chronic average daily dose. Exposure point concentrations of PCBs in fish were derived based on relative species intake for species found in the Mid-Hudson River, as reported in the 1991 New York Angler survey.

Other Exposure Pathways

For direct exposure to river water and sediment, there are also no available data to quantify the frequency of exposure in the Mid-Hudson. The exposure frequencies for the Mid-Hudson were assumed to be the same as those for the Upper Hudson. No avid recreator or resident inhalation of volatilized PCBs was evaluated based on the results for the Upper Hudson River, where PCB concentrations are higher. However, due to community concerns, the cancer risks and non-cancer health hazards due to ingestion of river water as a drinking water source were evaluated. The concentrations of PCBs in water and sediment were derived from the RBMR (USEPA, 2000a) and Revised ERA (USEPA, 2000f).

TOXICITY ASSESSMENT

The toxicity values used to assess cancer risks and non-cancer health hazards in the Mid-Hudson are the same Agency consensus values from IRIS described above that were used for the Upper Hudson.

RISK CHARACTERIZATION

As with the Upper Hudson, ingestion of fish resulted in the highest cancer risks and non-cancer health hazards. Cancer risks and non-cancer health hazards for the Mid-Hudson are approximately one-half of those for the Upper Hudson, as summarized in the tables below.

Point Estimate Cancer Risk Summary – Mid-Hudson River

Pathway	Central Tendency Risk	RME Risk
Ingestion of Fish		
Total*	1×10^{-5} (1 in 100,000)	7×10^{-4} (7 in 10,000)
Adult	6×10^{-6} (6 in 1,000,000)	3×10^{-4} (3 in 10,000)
Adolescent	3×10^{-6} (3 in 1,000,000)	2×10^{-4} (2 in 10,000)
Young Child	5×10^{-6} (5 in 1,000,000)	2×10^{-4} (2 in 10,000)
Swimming/Wading Exposure to Sediment*	2×10^{-8} (2 in 100,000,000)	2×10^{-7} (2 in 10,000,000)
Swimming/Wading Exposure to Water*	9×10^{-9} (9 in 1,000,000,000)	6×10^{-8} (6 in 100,000,000)
Consumption of Drinking Water*	3×10^{-8} (3 in 100,000,000)	1×10^{-7} (1 in 10,000,000)

**Total risk for young child (aged 1-6), adolescent (aged 7-18), and adult (over 18).*

Point Estimate Non-Cancer Hazard Summary – Mid-Hudson River

Pathway	Central Tendency Non-Cancer Hazard Index	RME Non-Cancer Hazard Index
Ingestion of Fish		
Adult	3	34
Adolescent	4	37
YoungChild	6	53
Exposure to Sediment*	0.002	0.004
Exposure to Water*	0.005	0.007
Consumption of Drinking Water*	0.01	0.02

**Values for young child and adolescent, which are higher than adult for these pathways.*

MAJOR FINDINGS OF THE REVISED HHRA

The Revised HHRA evaluated both cancer risks and non-cancer health hazards to young children, adolescents and adults posed by PCBs in the Upper and Mid-Hudson River. USEPA has classified PCBs as probable human carcinogens and known animal carcinogens. Other long-term adverse health effects of PCBs observed in laboratory animals include a reduced ability to fight infections, low birth weights, and learning problems. The major findings of the report are:

- Eating fish is the primary pathway for humans to be exposed to PCBs from the Upper and Mid-Hudson.
- Under the RME scenario for eating fish from the Upper Hudson, the calculated total cancer risk (40 years of exposure apportioned as a young child, adolescent, and then adult) is 1×10^{-3}, or one in 1,000. This excess cancer risk is 1,000 times higher than USEPA's goal of protection and ten times higher than the highest cancer risk level allowed under the federal Superfund law.
- For non-cancer health effects, the RME scenario for eating fish from the Upper Hudson results in a Hazard Index to a young child of 104, a level of exposure to PCBs that is more than 100 times higher than USEPA's reference level (Hazard Index) of one. HIs for the adolescent and adult are 71 and 65, respectively, which are 71 and 65 times higher than the reference level of one.
- The RME cancer risks and non-cancer health hazards are expected to be above USEPA's generally acceptable levels for the 40-year exposure period that begins in 1999, assuming baseline conditions of no remediation and no institutional controls.
- The RME cancer risks and non-cancer health hazards from ingestion of fish in the Mid-Hudson are about one-half the cancer risks and non-cancer Hazard Indices from ingestion of fish in the Upper Hudson. Nonetheless, these values are above USEPA's levels of concern.
- The central tendency cancer risks and non-cancer health hazards from eating fish in the Upper and Mid-Hudson, which are based on consumption of about one meal every two months, are also above USEPA's levels of concern.
- Cancer risks and non-cancer health hazards from being exposed to PCBs in the river through skin contact with contaminated sediments and river water, incidental ingestion of sediments, inhalation of PCBs in air, and consumption of river water as a drinking water source are generally within or below USEPA's levels of concern.

1 Overview of Upper Hudson River Risk Assessment

1.1 Introduction

This document presents the Revised Human Health Risk Assessment (Revised HHRA) for the Upper Hudson River as required under the National Oil and Hazardous Substances Pollution Contingency Plan ("NCP") (USEPA, 1990). This assessment quantifies carcinogenic and non-carcinogenic health effects from exposure to polychlorinated biphenyls (PCBs) in the Upper Hudson River, following USEPA risk assessment policies and guidance. Consistent with the NCP, this assessment evaluates both current and future risks to young children, adolescents and adults based on the assumption of no remediation or institutional controls (*i.e.*, baseline conditions).

On May 30–31, 2000, USEPA, through its contractor, Eastern Research Group (ERG), convened a panel of independent scientific experts to conduct a peer review of the August 1999 Human Health Risk Assessment for the Upper Hudson River (HHRA), consistent with the Agency's Peer Review Handbook (USEPA, 1998d). The peer reviewers were asked to respond to USEPA's charge questions, which covered each component of the HHRA and the HHRA overall. The specific charge questions and information about the peer review are presented in the "Report on the Peer Review of the Hudson River PCBs Human Health Risk Assessment" (ERG, 2000) (the "Peer Review Report"). In conjunction with this Revised HHRA, USEPA is issuing a Responsiveness Summary to the Peer Review Report. The Responsiveness Summary describes where in the Revised HHRA USEPA incorporated the peer review comments or provides the technical rationale for not incorporating a comment. The Revised HHRA combines the August 1999 HHRA, the December 1999 HHRA for the Mid-Hudson River, their respective responsiveness summaries, and the Response to Peer Review Comments into a single document.

The risk assessment considers site data collected during the late 1970s and early 1980s, and data collected throughout the Reassessment Remedial Investigation and Feasibility Study (RI/FS), which started in 1990. This assessment relies primarily on data in the database for the Hudson River PCBs Reassessment RI/FS (USEPA, 2000d) as well as extensive data analysis and modeling reports as described in the following documents: the Database Report (USEPA, 1995a); the Preliminary Model Calibration Report (USEPA, 1996a); the Data Evaluation and Interpretation Report (USEPA, 1997d); the Revised Baseline Modeling Report (RBMR), USEPA (2000a); the Revised Baseline Ecological Risk Assessment (Revised ERA), USEPA (2000f); and associated responsiveness summaries.

1.2 Site Background

The Hudson River PCBs Superfund Site extends from Hudson Falls, NY to the Battery (at the southern tip of Manhattan) in New York City. The site covers approximately 200 river miles. The Revised HHRA addresses the Upper Hudson River, which is the area between Hudson Falls, NY and the Federal Dam in Troy, NY, a length of approximately 40 river miles (Plate 1).[1]

From 1957 through 1975, between 209,000 and 1,300,000 pounds of PCBs were discharged to the Upper Hudson River from two General Electric Company (GE) capacitor manufacturing plants: one located in Fort Edward, NY and the other in Hudson Falls, NY (USEPA, 1991a). In 1977, the

[1] A separate risk assessment has been conducted using similar methodologies for the Mid-Hudson River (the area between Federal Dam in Troy, NY and Poughkeepsie, NY), a length of approximately 83 river miles. The Mid-Hudson analysis is presented in Chapter 6 of this report.

manufacture and sale of PCBs within the United States were generally prohibited under provisions of the Toxic Substances Control Act (USEPA, 1978).

In 1973, the Fort Edward Dam was removed, which facilitated the downstream movement of PCB-contaminated sediments (USEPA, 1991a). Because of potential human health risks due to consumption of PCB-contaminated fish, in 1976, the New York State Department of Environmental Conservation (NYSDEC) and the New York State Department of Health (NYSDOH) banned fishing in the Upper Hudson River and limited the recommended number of fish meals consumed for specific species in the Lower Hudson River (NYSDOH, 1995). In 1976, the commercial striped bass fishery in the Hudson River was closed based on elevated PCB levels in striped bass. The ban on fishing in the Upper Hudson River was subsequently changed to a "catch and release" program in August 1996, however advisories against consumption of any fish from the Upper Hudson River remain in effect (NYSDOH, 1999).

In 1984, USEPA issued a Record of Decision (ROD) for the site. The ROD required: 1) an interim No Action decision concerning river sediments; 2) in-place capping, containment and monitoring of Remnant Deposit sediments; and 3) a detailed study of the Waterford Water Works treatment facilities to see if an upgrade or alterations of the facilities were needed (USEPA, 1984a).

In December 1990, USEPA began a reassessment of the interim No-Action decision for the Upper Hudson River sediments based on, among other things, new requirements of the Superfund Amendments and Reauthorization Act of 1986 (SARA) to conduct reviews every five years of remedial decisions for sites where contamination remains on site, and a request by NYSDEC. Due to the size, complexity, and high level of public interest in this project, USEPA decided to conduct the study in three phases and issue reports to the public as work progressed. In August 1991, USEPA released the Phase 1 Report, which compiled and analyzed the existing data and included a preliminary human health risk assessment and an ecological risk assessment (USEPA, 1991a).

1.3 General Risk Assessment Process

The goal of the Superfund human health risk assessment process is to provide a framework for developing the risk information necessary to assist in the determination of possible remedial actions at a site. USEPA uses risk assessment as a tool to characterize the contaminants, evaluate the toxicity of the chemicals, assess the potential ways in which an individual may be exposed to the contaminants, and characterize the cancer risks and non-cancer health hazards at a Superfund site (USEPA, 1989b). In accordance with USEPA guidance, actions at Superfund sites are based on an estimate of the reasonable maximum exposure (RME) expected to occur under both current and future conditions at the site. The RME is defined as the highest exposure that is reasonably expected to occur at a site. USEPA guidance also recommends that the Agency estimate risks based on central tendency (CT), or average, exposures at a site (USEPA, 1995b). The RME and CT exposures are used to estimate cancer risks and non-cancer health hazards.

A systematic framework for human health assessment was first outlined in 1983 by the National Academy of Sciences (NRC, 1983). Building upon that foundation, the risk assessment process described in USEPA's "Risk Assessment Guidance for Superfund Volume I Human Health Evaluation Manual (Part A)" (USEPA, 1989b) and subsequent Agency guidance consists of the following components:

- *Data Collection and Analysis* - involves gathering data, including the use of models as necessary, to define the nature and extent of contamination.

- *Exposure Assessment* - entails an estimate of the magnitude of actual and/or potential human exposures, the frequency and duration of these exposures, and the pathways (*i.e.*, inhalation, ingestion, and dermal contact) by which people are potentially exposed.

- *Toxicity Assessment* - examines the type of adverse health effects associated with chemical exposure, and the relationship of the magnitude of exposure and the health response.

- *Risk Characterization* - summarizes the results from the first three steps of the assessment (both quantitative and qualitative) and a discussion of the uncertainties in the analysis.

1.4 Discussion of 1991 Phase 1 Risk Assessment

In 1991, USEPA issued the Phase 1 Report - Interim Characterization and Evaluation for the Hudson River PCB Reassessment RI/FS, including a quantitative risk assessment for the Upper Hudson River and a qualitative risk assessment for the Lower Hudson River (USEPA, 1991a). The Phase 1 Risk Assessment identified potential cancer risks and non-cancer health hazards associated with regular consumption of fish from the Upper Hudson River exceeding guidelines established in the NCP for acceptable exposure levels.

The Phase 1 Upper Hudson River human health risk assessment evaluated current and potential future risks from ingestion of fish, ingestion of drinking water, dermal contact with sediments, dermal contact with river water, and incidental ingestion of sediments. A map of the Upper Hudson River study area is shown in Plate 1.

The cancer risks from ingestion of fish were 2×10^{-2} (*i.e.*, an excess cancer risk of 2 in 100) using the 1986-1988 95% Upper Confidence Limit on the Mean (95% UCLM) PCB concentration in fish (12.0 mg/kg), and 2×10^{-3} (2 in 1,000) using the 30-year projected mean PCB concentration in fish (1.5 mg/kg) (USEPA, 1991a). The non-cancer Hazard Index (HI) for ingestion of fish was 51 using the 1986-1988 95% UCLM PCB concentration, and 6 using the 30-year projected mean PCB concentration in fish.

As described in the NCP (USEPA, 1990), "[f]or known or suspected carcinogens, acceptable exposure levels are generally concentration levels that represent an excess upper bound lifetime cancer risk to an individual of between 10^{-4} to 10^{-6} using information on the relationship between dose and response." The cancer risks calculated in Phase 1 exceeded the range defined in the NCP; the non-cancer Hazard Index exceeded one (1), indicating an exceedance of the Reference Dose (RfD), or the level at which no adverse chronic health effects are expected to occur.

The cancer risk from drinking water was 6×10^{-6} (6 in 1,000,000), which is within the acceptable risk range defined in the NCP, and the non-cancer HI was less than one (USEPA, 1991a). Cancer risks from dermal exposure to river sediment, incidental ingestion of river sediment, and dermal contact with river water totaled 8.8×10^{-6} (about 9 in 1,000,000), which is also within the acceptable risk range, and the non-cancer HI was also less than one (USEPA, 1991a). Risks from other pathways including ingestion of vegetables and meat and inhalation of volatilized PCBs were evaluated qualitatively in the Phase 1 risk assessment.

The Phase 1 human health risk assessment for the Lower Hudson River qualitatively evaluated current and potential cancer risks and non-cancer health hazards from ingestion of fish, based on the

findings in the Upper Hudson River. The assessment concluded that the risks from ingestion of fish would be similar to those found in the Upper Hudson River.

1.5 Objectives of Phase 2 Risk Assessment

Phase 2 of the Reassessment RI/FS consists of six major reports, one of which is the human health risk assessment. Since the Phase 1 Risk Assessment, additional data and information have become available and have been incorporated into this Revised HHRA, as follows:

- Additional data have been collected that provide PCB concentrations in water, sediment, fish and other biota.
- PCB concentration trends in environmental media have been forecast using linked PCB fate, transport, and bioaccumulation models.
- Major fish ingestion surveys published in the scientific literature have been reviewed to determine the most appropriate fish ingestion rate for the Reassessment RI/FS.
- The cancer toxicity of PCBs has been reviewed by USEPA and the scientific community resulting in updated cancer slope factors for PCBs (USEPA, 1996c). The revised cancer slope factors for PCBs are lower than those in effect when the Phase 1 assessment was completed based on new animal studies and revisions in USEPA's cancer guidelines (USEPA, 1996b). A reassessment of PCB non-cancer toxicity is underway.

The objectives of the Phase 2 risk assessment are to update the findings from Phase 1 (that cancer risks and non-cancer health hazards from fish ingestion outweigh other pathways of exposure), taking into consideration the additional information highlighted above, and to provide estimates of risks both to the RME, or high-end risk estimates ($\geq 90^{th}$ to 99^{th} percentiles), as well as estimates of risks to the average exposed individual, or central tendency (CT) risk estimates (50^{th} percentile).

1.6 Data Collection and Analysis

The data collection and analysis for the Upper Hudson River is provided in reports previously released by USEPA on the nature and extent of contamination at the Hudson River PCBs Site as part of the Reassessment RI/FS (*e.g.*, February 1997 Data Evaluation and Interpretation Report, July 1998 Low Resolution Sediment Coring Report, July 2000 Database for the Hudson River PCBs Reassessment RI/FS [Release 5.0], January 2000 RBMR, and associated responsiveness summaries).

This HHRA evaluates potential cancer risks and non-cancer health hazards associated with PCBs only as the contaminants of concern. This is supported by the data collection and analysis efforts. As discussed in the RBMR (USEPA, 2000a), in addition to monitoring for PCBs, fish collected by NYSDEC at the site have been analyzed for total DDT, total chlordane, total endrin, total endosulfan, dieldrin, aldrin, mirex, total heptachlor, total hexachlorobenzene, toxaphene, methoxychlor, individual polycyclic aromatic hydrocarbons (PAHs), cadmium, mercury, dioxins, and dibenzofurans. These analytes were found to be present at relatively low levels or below detection limits (Sloan, 1999), confirming that PCBs are the primary contaminants of concern in the Hudson River. The identification of PCBs as the contaminants of concern is also reasonable in light of the purpose of the Reassessment RI/FS, which is limited to the Hudson River PCBs site.

2 Exposure Assessment

The objective of the exposure assessment is to estimate the cancer risks and non-cancer health hazards from human exposure to PCBs within the study area. USEPA guidance (USEPA, 1989a,b; 1991b; 1992a,b,c; 1995b; 1996b; 1997a,e,f) provides the framework adopted to conduct the exposure assessment for this risk assessment.

The population of concern in this Revised HHRA consists of the inhabitants of the towns, cities, and rural areas surrounding the Upper Hudson River who may fish or engage in activities that will bring them into contact with the river. In the discussion that follows, certain terms used by risk assessors are introduced to define specific subgroups of this population. For example, members of the population who fish are described as the "angler" population. In addition, specific types of activities (*e.g.*, recreation) give rise to the use of the term "recreator" to describe another possible segment of the exposed population. The term "receptor" or "receptor population" is used to describe these subgroups of the exposed population. This definition of several receptor population groups does not suggest that these represent distinct individuals or even separate populations. Thus, individuals in the population of concern may fall within each of the "angler," "recreator," and "resident" receptor groups described below and throughout this Revised HHRA. Distinguishing separate receptor groups does not imply these populations are mutually exclusive, but rather the receptor groups are defined for convenience of distinguishing different PCB exposure possibilities.

Human exposures to PCBs in the environment are quantified by determining the concentration of PCBs in environmental media (air, water, sediment, and fish), which humans may then ingest or otherwise contact resulting in PCB uptake into the body. The exposure assessment process involves determining the concentration of PCBs in the environmental media of concern and combining this information with estimates of human exposure to the environmental media. The variability of environmental concentrations, the likelihood of exposure occurring *via* particular pathways, and the frequency and duration of human exposure are all components of the exposure assessment.

USEPA guidance and policy call for an evaluation of cancer risks and non-cancer health hazards to the average, or CT individual and an evaluation of cancer risks and non-cancer health hazards to the RME individual. An estimate for the RME individual can be obtained by determining estimates of likely "high-end" exposure factors and then combining these high-end factors with average factors to come up with a point estimate, or single value, for the reasonable maximum exposure. Alternatively, the RME can be estimated using probabilistic methods, often involving a technique termed Monte Carlo analysis (USEPA, 1997a). A Monte Carlo analysis does not estimate the RME based on single point estimates for each exposure factor, but rather draws repeated plausible exposure factor values from a probability distribution characterizing each factor, and combines these repeated samples to develop a distribution of exposure estimates. This distribution of PCB exposure contains an explicit estimate of the probability associated with any particular PCB exposure (intake) estimate, such that the RME can be determined based on estimates from the high-end ($\geq 90^{th}$ percentile) of the distributions of exposure results calculated using the Monte Carlo procedure. An estimate for the CT individual can be obtained by determining estimates of average exposure factors to calculate point estimate values for cancer risks and non-cancer health hazards, or by considering the mid-point (50^{th} percentile) of the Monte Carlo distributions.

In this Revised HHRA, point estimates of exposure (and the calculated cancer risks and non-cancer health hazards) are developed for the CT and RME exposures for all exposure pathways that are considered to be complete (see next section). The point estimate method used, described in the Risk Assessment Guidance for Superfund - Part A (USEPA, 1989b), is the same as the approach that was used

in the Phase 1 risk assessment, taking into consideration the important new information outlined in Section 1.5. In addition, a Monte Carlo exposure analysis is conducted for the fish ingestion pathway, the pathway shown in the Phase 1 risk assessment to yield the highest exposure to PCBs. For clarity, the point estimate exposure analysis is presented in this chapter (Chapter 2) of the report. The Monte Carlo exposure analysis for the fish ingestion pathway is presented in Chapter 3. Because some of the point estimate exposure factors (*e.g.*, fish ingestion rate, exposure duration, *etc*.) are based upon the sources of information and probability distributions for these factors derived in Chapter 3, the reader is referred to the Monte Carlo analysis for further details on these exposure factors where they are discussed more fully.

Section 2.1 summarizes the environmental media, potential receptors, and exposure pathways of PCB intake for the Revised HHRA. The framework for calculating human intake resulting from PCB exposures is presented in Section 2.2. The PCB exposure point concentrations used to estimate PCB intake are summarized in Section 2.3. Finally, the exposure factors and algorithms used to calculate PCB intake, and estimates of PCB intake for each complete exposure pathway, are summarized in Section 2.4. In this report, exposure assessment information is tabulated in the format prescribed by USEPA's Risk Assessment Guidance for Superfund Part D (USEPA, 1997e), in order to promote transparency and consistency in presenting risk assessment information to the public.

2.1 Exposure Pathways

For cancer risks and non-cancer health hazards to be present, a complete exposure pathway for chemical contact and intake must exist. A complete pathway requires a source and mechanism for release of constituents, a transport or retention medium, a point of potential human contact (exposure point) with the affected medium, and an exposure route (*e.g.*, ingestion, dermal contact, inhalation) at the exposure point (USEPA, 1989a,b; 1992a).

If any one of these elements is missing, the pathway is not considered complete and is not evaluated further. For example, if human activity patterns and/or the location of potentially exposed individuals relative to the location of affected media prevents human contact, then that exposure pathway is not complete and there is no health risk in such instances. For exposure pathways that are complete, the human health risks are determined by the degree of chemical exposure combined with the chemical's toxicity. Consistent with the NCP, the exposure scenarios examined in this Revised HHRA assume baseline conditions of no remediation and no institutional controls, such as fishing restrictions or consumption advisories, which are designed to limit environmental exposures.

The Upper Hudson River study area for this Revised HHRA includes urban, suburban, and rural areas along the river. During boating, fishing, and other recreational activities members of the Upper Hudson River study area population may become exposed to PCBs if they consume fish caught from the river, or as they come into contact with river water and river sediments; they could also inhale PCBs that may be released from the water into the air. Potential exposure pathways considered in this Revised HHRA are summarized in Table 2-1, identifying those which are "complete" and warranted exposure and risk calculations in this study. The following sections describe site-specific elements that make up the complete exposure pathways that are evaluated in this Revised HHRA.

2.1.1 Potential Exposure Media

Humans may be exposed to PCBs from the site either through direct ingestion or contact with media containing PCBs. In addition, PCB exposure can result from the transfer of PCBs from one

medium to another (*e.g.*, from water into air). PCBs have been detected, monitored and modeled extensively at the site. The exposure media that are considered the most potentially significant sources of PCB exposure at the site include the following:

Fish. Fish bioaccumulate PCBs, and as the results of the Phase 1 risk assessment indicate, ingestion of fish is likely to be the predominant pathway for human exposure to PCBs in the Upper Hudson River.

Sediment. Swimming, wading, and boating along the Hudson are recreational activities that would likely give rise to contact with sediment. Therefore, sediment is a potential exposure medium at the site.

River Water. Similar to river sediment, exposure to surface water from the Upper Hudson River is likely to occur during recreational activities and river water is thus considered a potential exposure medium.

Air. PCBs that volatilize from the river water may be inhaled by both recreators and residents living near the river. This medium is being considered in this assessment in order to update information presented in the Phase 1 risk assessment and address concerns raised by the public regarding potential inhalation of PCBs.

The actual determination of the relative importance of each of these potential exposure media, and those which may or may not pose a significant cancer risk and non-cancer health hazards, is determined based on the results of the quantitative exposure and risk analysis.

2.1.2 Potential Receptors

The population of concern in the evaluation of the Upper Hudson River consists of the inhabitants of the towns, cities, and rural areas surrounding the river. Within the potentially exposed population, the following "receptor" groups have been defined for the purpose of quantifying the potential PCB exposures within the population as a whole. These receptor groups should not be interpreted as distinct population subgroups, rather they are defined for convenience of presenting the exposure and risk analysis.

Anglers. The analysis from the Phase 1 Report (USEPA, 1991a) revealed that estimated PCB intake through consumption of fish from the Hudson River is the most significant pathway of human exposures to PCBs at the site. Therefore, much of the effort for the Revised HHRA is focused on refining the estimates of PCB exposure to anglers consuming fish. The angler population is defined as those individuals who consume self-caught fish from the Upper Hudson, in the absence of the Hudson-specific fish consumption advisories. The assessment of fish consumption by the angler population includes young children (aged 1-6), older children and adolescents (aged 7-18), and adults (over 18). Prenatal and neonatal exposures were evaluated qualitatively.

Recreators. Recreators along the Upper Hudson River are another potential receptor population group defined in this Revised HHRA This receptor population includes individuals participating in recreational activities along the river such as swimming, wading, boating, picnicking, *etc.* Because recreational activity patterns change with the age of the population, exposure by young children (aged 1-6), older children and adolescents (aged 7-18), and adults (over 18) are considered separately.

Residents. Although both of the above receptor groups include residents of the Upper Hudson River study area, a third receptor group, termed "residents," has been assigned for the purpose of assessing long-term exposure to PCB-contaminated air for that portion of the population living in close proximity to the river.

While quantifying the current and future size of the exposed population is difficult, it is possible to provide perspectives on the approximate size. According to the 1990 U.S. Census (1990a) data, the total population of the five Upper Hudson counties is approximately 750,000. An estimated 78,628 fishing licenses were issued to anglers in the five counties surrounding the Upper Hudson during 1998-1999 fishing season (NYSDEC, 2000). The number of fishing licenses does not include children under age 15, who are not required to have licenses, or friends and families who eat fish caught by an angler. According to Connelly *et al*. (1990), an estimated 10,310 anglers fished on the Upper Hudson River in 1988 and, according to Jackson (1990), angling effort in New York State appears to be increasing over time. This information is provided for perspective only—USEPA Superfund guidance and the NCP calls for the calculation of incremental cancer risks and non-cancer health hazards to the RME and CT individual.

2.1.3 Potential Exposure Routes

An exposure route is the means, or mechanism, of contact with an exposure medium, such as fish, river water, or sediment. Typical routes of exposure include dietary intake, inadvertent or incidental ingestion or intake of environmental media, air inhalation, *etc.* For anglers in the Upper Hudson River area, fish ingestion (*e.g.*, dietary intake) is the potential exposure route evaluated in this risk assessment. Routes of exposure under a recreational use scenario include absorption of PCBs *via* dermal contact with sediments, incidental ingestion of PCBs contained in sediments during subsequent hand to mouth contact, dermal contact with river water, and inhalation of air. Inhalation of air is also a potential exposure route for residents who live in close proximity to the Upper Hudson River. Ingestion of river water from the Upper Hudson was not quantitatively evaluated in the risk assessment because this exposure route was found to have *de minimus* (insignificant) risk, using reasonable maximum exposure assumptions, in the Phase 1 assessment (USEPA, 1991a). Furthermore, the current and projected future PCB concentrations in the Upper Hudson River are below the federal drinking water maximum contaminant level (MCL) of 0.05 µg/L established under the Safe Drinking Water Act. Each of these exposure routes is summarized in Table 2-1.

In addition to the above-mentioned routes of exposure, other potential pathways exist by which individuals may be exposed to PCBs originating from the Upper Hudson River. Such pathways include exposure from eating home-grown crops grown in floodplain soils, incidental ingestion of floodplain soils, and consumption of local beef, dairy products, or other non-fish biota (*e.g.*, turtles, ducks, *etc.*) in the vicinity of the Upper Hudson. These exposure pathways are outside the scope of the Reassessment RI/FS, which is focused on the PCBs in the sediments, and thus there are limited data to assess some of these exposure pathways. Although insufficient data exist to assess these exposure pathways quantitatively, the discussion below indicates they are unlikely to be significant pathways for PCB intake.

For the past 25 years, the New York State Department of Agriculture and Markets (NYSDA&M) has analyzed more than 18,200 samples of cow's milk within the state and has not found any detection of PCBs above the detection limit of 0.6 ppm (lipid normalized).[2] The information was obtained directly from Dr. Rudnick of NYSDA&M (Rudnick, 1999). The sample results are not contained in any

[2] This detection limit is significantly less than the FDA limit of 1.5 ppm (lipid normalized) (FDA, 1996).

computerized database, which would permit analysis by geographic area, and it would be resource-intensive to determine the location and number of dairy farms along the Upper Hudson River that are represented in this state-wide database. However, a representative of the NYSDOH confirmed that the samples represent individual farms, not composite samples of milk from more than one farm (Montione, 2000).

Possible exposure to PCBs *via* indirect uptake in produce impacted by airborne fallout or plant uptake has been raised as another pathway to consider. For example, USEPA is aware of two abstracts concerning the Chicago "urban plume" of PCBs (Eisenreich *et al*., 1996; Baker *et al*., 1996). While such information is relevant to PCBs in general, due to the different hydrodynamics between Lake Michigan and the Upper Hudson River, USEPA believes the information is not directly relevant to assessing human health risk to individuals exposed to PCBs in the Upper Hudson River. Moreover, in the 1980s, Dr. Buckley from the Boyce Thompson Institute at Cornell University collected data on PCBs in forage crops (corn and hay) grown in an area with PCB-contaminated soil. He found that levels of PCBs on these crops (sources of animal food) were below the U.S. Department of Agriculture regulatory level of 0.2 mg/kg for forage crops (Buckley and Tofflemire, 1983). Based on this information, the risk from ingestion of foods other than Hudson River fish is likely to be minimal, and collection of additional PCB data from vegetables, meat, eggs and milk is not warranted at this time.

In addition, a few snapping turtles in the Upper Hudson River have been found to contain PCBs (Stone *et al.*, 1980; Olafsson *et al.*, 1983). Stone *et al.* (1980) found PCB concentrations from ten snapping turtles, collected from the Hudson River in 1976-1978, to range from 306 to 7,990 mg/kg PCBs in fat tissue, 0.54 to 683 mg/kg PCBs (wet weight) in liver, and 0.2 to 27.6 mg/kg PCBs (wet weight) in muscle. Olafsson *et al.* (1983) reported a PCB concentration in fat tissue of 3,608 mg/kg from one snapping turtle from the Upper Hudson River near Hudson Falls, NY. In addition, five turtle samples from the Upper Hudson collected by NYSDEC are reported in the Database for the Hudson River PCBs Reassessment RI/FS [Release 5.0]. In these five samples taken at RM 176 in 1998, the Aroclor 1260 concentration ranged from 3.0 - 156 ppm.

While these sparse data provide an indication that turtles do indeed have appreciable concentrations of PCBs in their tissues, because of the small number of turtles that have been analyzed, the data may not be representative. It is also unknown whether turtles are regularly caught and consumed by local residents. Nonetheless, there is currently a state-wide consumption advisory for women of childbearing age, infants, and children under the age of 15 to avoid eating snapping turtles or soups made with their meat (NYSDOH, 1999a, p. 14). Consumption of fish is considered to be a more likely important dietary pathway for PCB intake from the Upper Hudson River. While the PCB intake from possible occasional consumption of other non-fish biota (such as turtles), cannot be ruled out entirely and is a possible concern, it would likely result in lower overall PCB intake compared to intake from fish consumption because fish consumption is considered to occur more frequently within the angler population. Based on the high concentrations of PCBs found in the turtles, any PCB intake from ingestion of turtles would pose cancer risks and non-cancer health hazards in addition to those evaluated in this Revised HHRA.

2.2 Quantification of Exposure

In this section of the risk assessment, the basic approach for calculating human intake levels resulting from exposures to PCBs is presented. Exposure estimates represent the daily dose of a chemical taken into the body, averaged over the appropriate exposure period. Chemical intake is expressed in

terms of a dose, having units of milligram chemical per kilogram body weight per day (mg/kg-day). In general, quantitative exposure estimates involve the following:

- determination of exposure point concentrations (the concentrations of PCBs in environmental media at the point of human exposure);
- identification of applicable human exposure models and input parameters (exposure frequency, duration, *etc.*); and
- estimation of human intakes using exposure algorithms.

The primary source for the exposure algorithms used in the risk assessment is USEPA's Risk Assessment Guidance for Superfund, Part A (RAGS) (USEPA, 1989b) and exposure guidance and guidelines. The generalized equation for calculating chemical intakes is:

$$I = \frac{C \times CR \times EF \times ED \times CF}{BW \times AT}$$

where:

I	=	*Intake* - the amount of chemical at the exchange boundary (mg/kg body weight/day)
C	=	*Exposure Point Concentration* - the chemical concentration contacted over the exposure period at the exposure point (*e.g.*, mg/kg-fish)
CR	=	*Contact Rate* - the amount of affected medium contacted per unit time or event (*e.g.*, fish ingestion rate in g/day)
EF	=	*Exposure frequency* - describes how often exposure occurs (days/year)
ED	=	*Exposure duration* - describes how long exposure occurs (yr)
CF	=	*Conversion factor* - (kg/g)
BW	=	*Body weight* - the average body weight over the exposure period (kg)
AT	=	Averaging time - period over which exposure is averaged (days); over a lifetime for evaluating cancer risks and over the appropriate exposure duration for evaluating non-cancer health hazards.

Exposure parameters (*e.g.*, contact rate, exposure frequency, exposure duration, body weight) describe the exposure of a receptor for a given exposure scenario. These values are the input parameters for the exposure algorithms used to estimate chemical intake (USEPA, 1989b; 1991b; 1992a, 1997f). The general equation above is slightly modified for each pathway. The specific exposure parameters for each pathway are summarized and discussed in detail in Section 2.4.

For each of the potentially complete exposure pathways identified in Table 2-1, both CT and RME exposure estimates are calculated in this Revised HHRA. The RME is the maximum exposure that is reasonably expected to occur at the site (USEPA, 1989b, 1990). A combination of Agency-recommended values and site-specific values were used for the input parameters. According to USEPA guidance (1992a, 1995b), CT estimates are intended to reflect central or average estimates (*e.g.*, 50^{th} percentile) of exposure or dose, while RME estimates are intended to reflect exposure of persons at the upper end ("above about the 90^{th} percentile") of the distribution. As described in the NCP (USEPA,

1990), the RME, or high-end, exposure estimates should be within the range of possible exposures, and not beyond. Therefore, the RME exposure estimate is not a worst-case scenario.

High-end risk estimates, according to USEPA (1992a, 1995b), are defined as "plausible estimates of the individual risk for those persons at the upper end of the risk distribution." When a sufficient database is available, USEPA (1992a, 1995b) recommends reporting exposures "at a set of selected percentiles of the distributions, such as 90^{th}, 95^{th}, and 98^{th} percentile." The use of the 90^{th} to 95^{th} percentile estimates of exposure parameters for the RME, or high end, exposure assessment for the Upper Hudson is consistent with this guidance, and reflects the upper range of exposures, but not the maximum possible exposure.

2.3 Exposure Point Concentrations

A typical baseline Superfund risk assessment includes an evaluation of those chemicals at a contaminated site that pose a potential health concern, or chemicals of potential concern (COPCs). In this Revised HHRA, PCBs are identified as the COPCs and later as Chemicals of Concern (COC), because the Revised HHRA is being conducted as part of USEPA's Reassessment of its 1984 interim No Action decision for the PCB-contaminated sediments in the Upper Hudson River, and because fish data show that PCBs are detected at greater concentrations than other contaminants. As discussed in the RBMR, in addition to monitoring for PCBs, fish collected by NYSDEC at the site have been analyzed for total DDT, total chlordane, total endrin, total endosulfan, dieldrin, aldrin, mirex, total heptachlor, total hexachlorobenzene, toxaphene, methoxychlor, individual polycyclic aromatic hydrocarbons (PAHs), cadmium, mercury, dioxins, and dibenzofurans. These analytes were found to be present at relatively low levels or below detection limits (Sloan, 1999), confirming that PCBs are the primary contaminants of concern in the Hudson River. Consequently, no screening of COPCs was performed for this assessment. Thus, the USEPA RAGS Part D format tables (Tables 2-2 through 2-5), which for a typical risk assessment would include information necessary to determine COPCs, are not needed and are included only for consistency.

Another consideration that shapes the determination of the exposure point concentrations (EPCs) in this Revised HHRA is the time- and space-dependency of the PCB concentrations in fish, sediment, and water. Moreover, the EPC for PCBs in each of these media is based upon modeled projections of future concentrations in each medium (although the models are based upon a large monitoring record). As a result, the typical approach used in Superfund risk assessments of calculating an upper confidence limit on a mean concentration, or 95% UCLM (USEPA, 1992c), in some instances no longer strictly applies. One reason for its inapplicability is that the 95% UCLM calculation is based upon the notion that the estimate of the mean EPC from a finite sample set is uncertain and is a function of the number of samples available to estimate the true mean. However, when a model is used to predict the EPC there is no corollary to sample size; with a model an almost unlimited number of model-predicted values can be calculated. As the number of model-projected concentration estimates increases (in time or space), the model mean and model 95% UCLM (calculated from the finite number of model estimates) converge to the same value. Only if model inputs are varied to reflect environmental variability of the model input parameters, and repeated model estimates of the mean are obtained over the range of parameters, can an average and 95% upper confidence limit on the modeled means be calculated.

2.3.1 PCB Concentration in Fish

Because the Revised HHRA examines current and future cancer risks and non-cancer health hazards, and because the concentration of PCBs in fish changes over time and location, the EPC for PCBs in fish necessarily relies upon model predictions. Three factors have an influence on the EPC in fish:

1. The concentration of PCBs for any particular species varies from year to year, but overall it declines over time.

2. The concentration of PCBs within the same fish species varies with location in the Upper Hudson River, with higher concentrations upstream (Thompson Island Pool) compared to downstream.

3. The concentration of PCBs varies among different fish species.

Thus, even though fish are considered a single exposure medium, each of the above factors will influence the calculation of a single EPC.

Summary of Modeled PCB Concentration Results

Appendix A presents the Executive Summary from the RBMR (USEPA, 2000a) and several figures from the RBMR in order to provide perspective on the modeling performed to predict future PCB concentrations in fish, sediment and water. In the RBMR, a fate and transport model (HUDTOX) was used to predict concentrations of PCBs in water and sediment and several bioaccumulation models were used to predict concentrations of PCBs in fish. One of the bioaccumulation models adopted an empirical prediction of bioaccumulation based on a bi-variate correlation analysis of PCB concentrations in sediment and the water column with those measured in fish. Another bioaccumulation model was a mechanistic food web model called FISHRAND, which used the historical measurements of PCBs in fish, water, and sediment in order to calibrate the model to fish species in the Upper Hudson River. In both cases, the bioaccumulation models rely upon predictions of future PCB concentrations in the water column and sediments (from HUDTOX) to predict future trends of PCB concentration in fish. In this Revised HHRA, the FISHRAND model predictions (USEPA, 2000a) were used to estimate EPCs for fish.

As described in the RBMR, the FISHRAND model was calibrated using the extensive database for the Hudson River PCBs Reassessment RI/FS (USEPA, 2000d, Release 5.0). The database contains approximately 750,000 measurements for sediments, fish and aquatic biota, surface water flow and surface water quality from the USEPA, NYSDEC, USGS, NOAA and GE. Almost 350,000 of these records contain data acquired by USEPA as part of the Phase 2 Reassessment RI/FS sampling effort. The remaining records contain data from a large number of historical and ongoing monitoring efforts in the Hudson River.

Using FISHRAND, model predictions were provided for six fish species: brown bullhead, largemouth bass, white perch, yellow perch, pumpkinseed, and spottail shiner. These species were selected to get a representative distribution of bottom feeders, species at the top of the food chain, and semi-piscivorous species (USEPA, 2000a). Model estimates of Total PCB concentration in each species were based on all PCB congeners with three or more chlorine molecules, *i.e.*, Tri+ PCB concentrations (USEPA, 2000a). The model provides estimates of PCB concentrations in fish fillets (skin on) for the larger fish species modeled (*i.e.*, brown bullhead, largemouth bass, white perch, and yellow perch). The fillet represents the portion of the fish most commonly consumed. The model provides estimates of PCB concentrations in whole fish for the smaller species *(i.e.*, pumpkinseed and spottail shiner); however,

these species are not considered in the Revised HHRA because they are not typically consumed. Although white perch migrate to the lower two lock pools of the Upper Hudson River to spawn, they are typically found in the Lower Hudson River, not the Upper Hudson River for the remainder of the year (USEPA, 1999d, Appendix D). In addition, no measured data are available for white perch to calibrate the model results in the Upper Hudson (USEPA, 2000a).

Modeled predictions of future PCB concentrations in fish are available for three locations in the Upper Hudson River: Thompson Island Pool (approximately River Mile 189); Stillwater Dam (approximately River Mile 168); and near the Federal Dam (approximately River Mile 154). These three locations correspond to the monitoring locations where data are available for model calibration. In general, the concentrations of PCBs in all fish species decrease with river mile, such that concentrations in the vicinity of the Thompson Island Pool are the highest (see Figures 2-1 through 2-3).

The Revised HHRA evaluates current (1999) and future exposures to PCBs in fish. PCB concentrations in fish were forecast for the period 1999 to 2067. Forecasts from 1999 to 2039 were used in deriving the EPC in fish for the point estimate RME cancer assessment, which has an exposure duration of 40 years; forecasts from 1999-2067 were used in deriving the EPC in fish for the Monte Carlo RME cancer assessment, which has an exposure duration of up to 70 years (see Sections 2.4.1 and 3.2.4). Figures 2-1 through 2-3 show the average annual PCB concentrations over time and location for each of the three modeled species used in the Revised HHRA.

In addition to the annual mean PCB concentrations, the model predictions include the 50^{th} and 95^{th} percentile annualized concentration. These percentiles represent percentiles of the entire distribution of PCB concentration ranges within species, not the range, or uncertainty, on the mean PCB concentration in fish. Thus, this information is insufficient to provide an estimate of the upper confidence limit on the mean, or 95% UCLM, concentration of PCBs in the various fish species. However, the ratio of the model predicted 95^{th} percentile is a factor of 2- to 3-fold greater than the 50^{th} percentile concentration on average (the maximum ratios for each species are nearly identical to their average ratios) (see Figures 7-10 through 7-12 of the RBMR). Given this modest spread of concentration from the 50^{th} to 95^{th} percentile of the entire distribution, the 95% UCLM concentration would not be expected to be significantly greater than the mean concentration. Therefore, the modeled mean concentration of PCBs was used for the EPC in fish.

Figures 2-7 through 2-9 show measured annual average PCB concentrations in brown bullhead, largemouth bass, and yellow perch, respectively, as reported by NYSDEC for the 20-year period from 1980 to 1999, together with model forecast values. As these figures show, the forecast values generally project a somewhat more rapid decline in PCB concentration in fish than what is shown by the NYSDEC monitoring data. Thus, while the model calibration results indicate that the model generally matches the historical measurements (within an approximate factor of 2-fold when compared on a lipid basis), the projections into the future are somewhat more uncertain given uncertainties in the model boundary conditions (such as upstream loadings) combined with the fact that the forecast PCB concentrations in fish also rely upon modeled predictions of future PCB concentrations in water and sediment. A HUDTOX model validation was conducted to compare predicted and observed water column concentrations for Tri+ using a dataset acquired in 1998 for the Upper Hudson River by GE. Results indicated good agreement at both the Thompson Island Dam and Schuylerville over an entire year, spanning a range of environmental conditions in the river. Several approaches (detailed in Appendix A) were used to validate the FISHRAND model and generally good agreement was identified across all three species implying confidence in the performance of the model.

Concentration Averaged Over Locations

With the exception of some limited information in the NYSDOH 1996 study of Hudson River anglers (NYSDOH, 1999b), there is insufficient information to quantify fishing preference or frequency at specific locations within the Upper Hudson River. Consequently, projected PCB concentrations in fish were averaged over the three locations that were modeled. This averaging essentially presumes a uniform likelihood of fishing at any location within the Upper Hudson River study area. A sensitivity analysis is included in Chapter 5 to examine how the exposure and cancer risk and non-cancer health hazard estimates vary on a location-by-location basis.

The PCB concentrations, averaged over location, for each of the three modeled species used in the Upper Hudson River risk assessment are summarized in Figure 2-4. Modeled PCB concentrations for brown bullhead are the highest for approximately the first 10 years; the modeled PCB concentration in largemouth bass and yellow perch are comparable to one another.

PCB Concentration Weighted by Species-Consumption Fractions

In order to take into account the species individuals actually eat from the Upper Hudson River, species-specific intake patterns, derived from the 1991 New York Angler survey (Connelly *et al.*, 1992), were used to calculate the concentration of PCBs ingested in fish. That is, each species of fish has a characteristic PCB concentration, and the average concentration an angler consumes will, in part, be based on the relative percentages of the different fish species consumed.

A summary of the Connelly *et al.* (1992) survey results is provided in Table 3-3, and is described briefly here. A complete discussion of the 1991 New York Angler survey is found in Chapter 3. A total of nine specific species, plus a tenth category denoted "other," were included in the Connelly *et al.* (1992) survey. Of the nine species in the survey, salmon and trout are not commonly found in the Upper Hudson River study area. In addition, very few catfish (there is a separate category for bullhead) were caught in the 1991/2 and 1996 creel surveys of Hudson River anglers (NYSDOH, 1999b). Therefore, salmon, trout and catfish, along with the unidentified "other" category, were excluded when determining species ingestion weighting factors. The six species from the Connelly *et al.* (1992) survey that are potentially caught and eaten in the Upper Hudson River (bass, walleye, bullhead, carp, eel, and perch), were grouped in order to develop the fish ingestion weights from which the weighted concentration term was developed. Carp and eel, which are bottom feeders, were grouped with brown bullhead as Group 1. Walleye, which is similar to bass based on its large size and piscivorous diet, was grouped with the bass as Group 2.[3] Group 3 is perch, for which yellow perch modeled concentrations were used. Using this approach, the concentrations of PCBs in fish species that were not modeled (*i.e.*, carp and eel, walleye and some bass) were approximated based on the two species consumed that were modeled (brown bullhead and largemouth bass), so that consumption of the non-modeled species could be included in the species-weighted EPCs. Table 3-4 summarizes species-group intake percentages by summing the frequency percentage of the individual species in each group.

The point estimate EPCs in fish were derived using the species ingestion fractions shown in Table 3-4 multiplied by the PCB concentrations in each of the three modeled fish species. Thus, the point estimate of the weighted EPC is:

$$EPC = EPC_{Group1} \times 0.44 + EPC_{Group2} \times 0.47 + EPC_{Group3} \times 0.09$$

[3] The Connelly *et al.* (1992) survey did not specify what specific species were included in "bass." Presumably, this category includes both largemouth and smallmouth bass. The category may include striped bass, and other bass species as well.

The EPC values for fish are summarized in Tables 2-6 through 2-8 for each of the three modeled locations. An overall EPC for the entire Upper Hudson River was calculated by averaging over the three locations. As summarized in Tables 2-12a through 2-12c, for the evaluation of cancer risks and non-cancer health hazards, the CT EPCs are 3.0 mg/kg for the adult and 3.3 mg/kg PCBs for the adolescent and young child. These EPCs were calculated by averaging the species-weighted concentration distribution over 6, 3, and 3 years for the adult, adolescent, and young child exposures, respectively. The exposure durations for each age group sum to 12 years, the 50^{th} percentile exposure duration estimate.

The RME EPCs for the evaluation of cancer risks are 2.0, 2.5, and 3.0 mg/kg PCBs for adult, adolescent, and child exposures, respectively. These EPCs were calculated by averaging the species-weighted concentration distribution over 22, 12, and 6 years for adult, adolescent, and young child exposures, respectively. The RME exposure durations sum to 40 years, the 95^{th} percentile exposure duration estimate. The RME exposure EPCs for the evaluation of non-cancer health hazards are 2.9 mg/kg for adult and adolescent (averaged over the 7-year chronic exposure) and 3.0 mg/kg PCBs for the young child (averaged over the 6-year exposure duration). The determination of these particular exposure durations is described in Section 2.4.1. and Section 3.2.4.

It may be counter-intuitive that the RME EPC is lower than the CT EPC. This fact is a direct result of the general trend of a projected decline in concentrations of PCBs in fish over time. Due to this decline over time, the average concentration over a longer exposure duration is less than the average concentration over a shorter time period. However, the total lifetime PCB dose, which combines average concentration, exposure duration, and other intake factors, is greater for the high-end (RME) individual than for the average (CT) individual.

2.3.2 PCB Concentration in Sediment

Just as is the case for fish, PCB concentrations in sediment in the Upper Hudson River change as a function of location and time. PCB concentrations in surficial (0 - 4 cm) sediment layers were modeled over time and distance assuming baseline conditions of a constant-upstream source of PCBs of 13 ng/L and the results were presented for Total PCBs and Tri+ PCBs (USEPA, 2000a). The predicted Total PCB concentrations were used to calculate EPCs in sediment.

The modeled concentrations of PCBs in sediment are available for five different river mile segments of the Upper Hudson River. Model predictions were differentiated into cohesive and non-cohesive sediment classes for each river segment. Figure 2-5 shows the time trend of segment-averaged Total PCB concentrations in sediment weighted by relative cohesiveness and non-cohesiveness for RM 189, RM 168 and RM 154. The average PCB concentration in sediment over the entire Upper Hudson is also shown in Figure 2-5.

The modeled results for cohesive and non-cohesive sediments were adjusted into a single PCB concentration in sediment by weighting the respective areal extent of cohesive and non-cohesive sediment. Figures 5-4(A-D) of the RBMR (USEPA, 2000a) show that the cohesive sediments tend to occur in areas along the margins of the river channel or in near-shore areas where human contact might be more frequent. However, the non-cohesive sediments predominate on a total area basis, even in near-shore areas of the river. Nonetheless, the weighting is unlikely to affect the EPC in sediment because the cohesive and non-cohesive sediments do not differ appreciably in PCB concentration. A sensitivity analysis of avid recreator exposure to potentially higher PCB concentrations in selected near-shore environments is presented in Chapter 5.

As summarized in Tables 2-13 through 2-15, the CT EPCs of 6.6, 7.2, and 7.2 mg/kg PCBs in sediment for adult, adolescent, and young child exposures, respectively, were calculated by averaging the modeled sediment concentration over 5, 3, and 3 years for adult, adolescent, and young child exposure durations, respectively. These average exposure durations for each age group sum to 11 years, the 50th percentile residence duration in the five counties surrounding the Upper Hudson. The high-end exposure EPCs of 3.8, 5.2, and 6.4 mg/kg PCBs in sediment for adult, adolescent, and young child exposures, respectively, were calculated by averaging the modeled sediment distribution over 23, 12, and 6 years for adult, adolescent, and young child exposure durations, respectively. These RME exposure durations sum to 41 years, the 95th percentile residence duration in the five counties surrounding the Upper Hudson.

2.3.3 PCB Concentration in River Water

Similar to the sediment results, the RBMR provides modeled future concentrations of PCBs in the water column over time and distance, assuming baseline conditions of a constant-upstream source of PCBs and provides results for Total PCBs and Tri+ PCBs (USEPA, 2000a). The modeled Total PCB concentrations were used to calculate the EPCs in river water.

The model predictions were presented for five different river mile segments in the Upper Hudson River. Figure 2-6 shows the time trend of the modeled concentrations of PCBs for RM 189, RM 168 and RM 154, including an overall decline in the modeled concentrations over the 70 year modeling period as well as a declining concentration gradient from up-river to down-river locations.

As summarized in Tables 2-16 through 2-18, the CT EPCs of 4.6×10^{-5}, 4.8×10^{-5}, and 4.8×10^{-5} mg/L PCBs for adult, adolescent, and young child exposures, respectively, were calculated by averaging the modeled PCB concentrations in river water 5, 3, and 3 years for adult, adolescent, and young child exposure durations, respectively. These average exposure durations for each age group sum to 11 years, the 50th percentile residence duration in the five counties surrounding the Upper Hudson. The high-end exposure EPCs of 3.4×10^{-5}, 4.0×10^{-5}, and 4.5×10^{-5} mg/L PCBs for adult, adolescent, and young child exposures, respectively, were calculated by averaging the modeled river water distribution over 23, 12, and 6 years for adult, adolescent, and young child exposure durations, respectively. These RME exposure durations sum to 41 years, the 95th percentile residence duration in the five counties surrounding the Upper Hudson.

2.3.4 PCB Concentration in Air

The Phase 1 Report (USEPA, 1991a) provides a discussion of a number of studies that have documented PCB measurements in air in the Upper Hudson River study area and elsewhere in New York State. A wide range of PCB concentrations in air are reported for the Upper Hudson River, with data collected in the early to late 1980s generally exhibiting concentrations of PCBs in air on the order of 0.1 $\mu g/m^3$, or less (see, Table B.3-21 of Phase 1 Report). Although the air studies indicate PCBs exist in the atmosphere of the Upper Hudson, the studies do not necessarily identify the contribution of PCBs in the air that is derived from PCB-contaminated river water, which would link those PCBs to the site.

In order to evaluate the potential quantitative exposure *via* inhalation of volatilized PCBs from the Upper Hudson River, the following three different approaches were considered:

1. Historical measurements in 1980-81 of PCBs released to the air from the Upper Hudson River near Lock 6 were examined (Buckley and Tofflemire, 1983).

2. The results of the 1991 air monitoring study conducted during remediation of the PCBs in the Remnant Deposit sediments near Fort Edward (released subsequent to the Phase 1 Report) were evaluated.

3. PCB releases from the Upper Hudson River water column were estimated using diffusion and volatilization equations.

Buckley and Tofflemire 1980-81 Study

Airborne PCB concentrations were monitored at two locations above the Lock 6 Dam (RM 186.2) during the period of 1980-81 (Buckley and Tofflemire, 1983). These monitoring locations were chosen by the authors to represent areas expected to have elevated concentrations of PCBs in air, owing to the turbulence of the water in the dam spillway, which promotes air exchange and increased potential for PCB volatilization. Seven samples were taken at a height of 1 meter and two samples were taken at a height of 4.5 meters. Table B-1 (Appendix B) summarizes the PCB concentrations measured at two locations (A and B) above the Lock 6 Dam. Results of Aroclor-specific concentrations for each sample time were summed to get a Total PCB value, assigning one-half the detection limit to non-detected values. Summing all Aroclors to estimate Total PCBs likely overstates the Total PCB concentration. Given the small sample size and historical nature of the results, no adjustment was attempted that would correct for possible overestimating the Total PCB concentration. Aroclor 1242 was detected in all samples. The Total PCB concentration ranged from 0.033 $\mu g/m^3$ to 0.530 $\mu g/m^3$. The highest detected value may be an outlier result, and was described by the authors as "atypical." The mean of the nine samples is 0.11 $\mu g/m^3$.

Although this study provides evidence suggesting PCBs in air could be attributed to releases from the water column, the study results cannot be used directly to assess current (1999) and future exposure to PCBs because the concentration of PCBs in the water column in 1980-81 was much greater than current (expected future) concentrations.

Remnant Deposit Remediation Air Monitoring 1991

As part of the Remnant Deposit Remediation monitoring, Harza Engineering performed air monitoring studies for GE from January through November 1991 (Harza, 1992). The first five months of the monitoring program focused on two miles of the Upper Hudson River in the Fort Edward area and monitored PCB concentrations in air during construction. After containment was achieved, the remaining monitoring program (June through November 1991) focused on the Remnant Deposits for the first six weeks and then to residential areas for the remainder of the program. Between June and mid-July, one sampler operated on, or adjacent to, each Remnant Deposit; from mid-July to the end of November, three fixed-location stations (A2, A3, and A4) operated in residential areas (Harza, 1992). Concurrent with the air monitoring, PCBs were monitored in the water column of the Upper Hudson River.

A total of 985 airborne PCB samples were collected during the 1991 construction monitoring period. Of these samples, only 13 samples, or 1.3%, had PCB concentrations above the limit of quantification. PCB concentrations (only Aroclor 1242 was detected in 1991) ranging from 0.03 to 0.13 $\mu g/m^3$ were detected during this monitoring program. Table B-2 (Appendix B) presents all detected

air sampling results and corresponding river water samples collected in the same vicinity and at approximately the same time as the detected air sample results.

A number of factors suggest that the PCBs detected in air were emanating largely from the Upper Hudson River and less likely from the four Remnant Deposits or other sources. First, all PCB levels were below the detection limit throughout the first four months of 1991, when the construction activities were occurring, and such activities would tend to promote airborne releases of PCBs. Second, the surfaces of the Remnant Deposits were covered when these detections occurred (Harza, 1992). Third, PCBs were detected in air only when high PCB concentrations were also detected in the water column samples.

These data can be used to estimate an empirical water to air transfer coefficient, representing the ratio of the PCB concentration in air divided by the PCB concentration in water. Using the detected PCB concentrations in air and water summarized in Table B-2, empirical air-water transfer coefficients range from 0.02 to 0.4 ($\mu g/m^3$ per $\mu g/L$), with a median value of 0.09, and an average value of 0.15 ($\mu g/m^3$ per $\mu g/L$).

In accordance with widely used transport equations used to estimate volatile release of chemicals to air (see discussion of modeling below), at equilibrium the chemical release to the air is linearly proportional to the chemical concentration in water. Using this principle, the empirical transfer coefficients provide one means of estimating the PCB concentration in air that corresponds to the predictions of future PCB concentrations in the water column. As discussed earlier, the mean predicted PCB concentration in the water column is 24 ng/L (0.024 $\mu g/L$). Applying the median empirical transfer coefficient (0.09), an empirical estimate of the PCB concentration in air associated with an average 0.024 $\mu g/L$ in the water column is 0.002 $\mu g/m^3$. A high-end estimate of the PCB concentration in air, based on the 95^{th} percentile estimate of the water column PCB concentration of 0.042 $\mu g/L$ and the highest empirical transfer coefficient of 0.4, is 0.017 $\mu g/m^3$.

Modeled PCB Concentrations in Air

Published modeling approaches were also used to evaluate PCB concentrations in air, as summarized in Appendix B. Two approaches were used to estimate the PCB flux from the river. One of these approaches is based on the commonly used two-layer film resistance model as described in Achman *et al.* (1993) and Bopp (1983), and other standard texts. This model describes the volatilization of chemicals as a process of chemical diffusion through a water boundary layer on the water-side of the air-water interface, volatilization at the interface, then diffusion through the air boundary layer on the air-side of the interface. The PCB flux using this model is linearly proportional to the PCB concentration in water, yielding a "normalized" flux rate (mass of chemical per unit concentration in water). Using physical-chemical parameters determined by Bopp (1983) for tri- and tetrachlorobiphenyls, the normalized PCB flux rate is estimated to be:

$$\text{Normalized PCB Flux (two-film model):}\ \ 2.7 \times 10^{-3}\ (ng/m^2\text{-sec per ng/L})$$

The second modeling approach is an empirical approach based on a number of field studies conducted examining the flux of PCBs from water bodies to the atmosphere (Nelson *et al.*, 1998; Hornbuckle *et al.*, 1994, Achman *et al.*, 1993; Hornbuckle *et al.*, 1993). Given the complexity of the physical processes controlling the volatilization flux, the estimates using the two-film resistance model were compared with field measurements conducted by Achman *et al.* (1993) in Lake Michigan. Based upon field measurements from June through October, 1989, Achman *et al.* measured the flux of PCBs on 14 separate days, under a range of field conditions (temperature, wind speed, *etc.*). The Total PCB concentration in water measured during the study period ranged from 0.35 ng/L to 7.8 ng/L; measured

PCB flux rates ranged from 13 to 1,300 ng/m^2-day (1.5×10^{-4} to 1.5×10^{-2} ng/m^2-sec). The average normalized PCB flux rate (based on the 14 measurements) was:

Normalized PCB Flux (empirical): 1.2×10^{-3} (ng/m^2-sec per ng/L)

The modeled flux rate using the physical-chemical parameters from Bopp (1983) and the empirical PCB flux rate estimates compare favorably. The two-layer film model estimate is used in the following discussion to estimate the PCB concentration in air in the immediate vicinity of the Upper Hudson River.

The PCB emission estimates provided the PCB source term for the Industrial Source Complex (ISC) air dispersion model (USEPA, 1995c) that was used to estimate PCB concentrations in air in the vicinity of the Upper Hudson River. The ISC model is recommended as a preferred model by the USEPA for use in regulatory and permitting applications. The ISC model was developed by USEPA for determining atmospheric pollutant concentrations associated with point, line, area and volume sources of emission.

Two separate versions of the ISC model are available to allow analysis of both long-term and short-term air quality impacts. The primary difference between the two models is the type of weather data needed as input. The short-term version, ISCST, was designed to calculate contaminant concentrations over time periods as short as one hour. The ISCST model can be used to calculate ambient concentrations over longer time periods (for example one year), simply by averaging the hourly predictions over the appropriate averaging period. Because the ISCST predictions are based upon more detailed meteorologic inputs, the predictions from the ISCST model are considered more accurate than those estimated using the ISCLT (long-term) model. The current ISC Short Term model, ISCST3 Version 97363 (USEPA, 1995c as updated) was used to estimate the concentration of PCBs in the vicinity of the Upper Hudson River for purposes of the exposure assessment.

As described in Appendix B, a one kilometer (1,000 meter) stretch of river, with an approximate width of 200 meters (a typical width in the Thompson Island Pool area), was modeled.[4] Using the projected average PCB concentration in the Upper Hudson River of 24 ng/L (described earlier) and the normalized flux of 2.7×10^{-3} ng/m^2 per ng/L, the PCB flux estimate for the modeled source area (1000 m $\times$ 200 m) is 13 µg/sec.

The exposure point concentration estimate for PCBs in air depends greatly on the distance from the river. The normalized average downwind PCB concentration modeled using ISCST is estimated to be approximately 70 pg/m^3 per µg/sec at the immediate river edge (downwind), and drop by 10-fold within 200 meters downwind. The average concentration within 50 to 200 meters of the river shoreline is 9 pg/m^3 per µg/sec (Appendix B).

Using the PCB flux just described (13 µg/sec), and the normalized average concentration within 200 meters of shore (9 pg/m^3 per µg/sec), gives a PCB concentration in air of 117 pg/m^3, or 0.00012 µg/m^3. For comparison, if the empirical estimate of PCB flux from the Lake Michigan study (Achman *et al*., 1993) were used (1.2×10^{-3} ng/m^2-sec per ng/L), the predicted PCB concentration in air within the region 50 to 200 meters from the river shoreline would be 0.00005 µg/m^3.

[4] It should be noted that it is not necessary to model the entire Upper Hudson River. Given the general north-south orientation of the River, the model results are very stable in the east-west direction. Had a longer stretch of river been modeled, the PCB emission rate would have been scaled to the appropriate increase in surface area. The PCB flux per unit area (which is the term that drives the dispersion model), remains approximately constant.

Estimated Exposure Point Concentration in Air

In summary, there are limited data available that provide site-specific information necessary to estimate future PCB concentrations in air that are attributable to PCB releases from the Upper Hudson River. Based on the foregoing discussion, the following range of PCB concentrations in the air for locations near the Upper Hudson River can be reasonably linked to releases from the water column:

Measurements (1980-81):	0.11 $\mu g/m^3$ (mean) 0.53 $\mu g/m^3$ (maximum)
Measurements (1991):	0.03 $\mu g/m^3$ (minimum detected) 0.13 $\mu g/m^3$ (maximum detected)
Empirical Estimate: (1991 Remnant Monitoring)	0.002 $\mu g/m^3$ (central est.) 0.017 $\mu g/m^3$ (high-end est.)
Modeled Estimates:	0.00012 $\mu g/m^3$ (mean water column source) 0.00021 $\mu g/m^3$ (high-end water column source)

The 1980-81 air measurements cannot be used to assess potential current and future PCB exposures because PCB concentrations in the water column were much greater in 1980-81 than current and projected future concentrations. Similarly, to the extent the detected concentration range of PCBs in air measured in 1991 are associated with releases from the water column, the PCB concentrations in the water column were between one and two orders of magnitude higher in 1991 than they are expected to be in 1999-2020. Thus, using the 1991 measurements directly would likely substantially overstate the airborne PCB concentrations.

The ISCST3 modeled estimates of PCB concentration in air summarized above yield the lowest estimated concentrations of volatilized PCBs in air. Of the two steps in the air model (first determining the flux rate of PCBs from the water column then using this flux in the ISCST model), modeling the flux rate is the most uncertain. The diffusion coefficients in the flux model are highly dependent on the degree of turbulence in the water column, especially at the air-water interface. The measured flux rates from the Lake Michigan study could be expected to underpredict flux from the Hudson River, which is a flowing, more turbulent, water body. Yet, even if the Lake Michigan flux rates were increased by as much as an order of magnitude, the predicted PCB concentration in air would be 0.0005 $\mu g/m^3$.

Notwithstanding the large range of airborne concentration estimates, a central estimate EPC of 0.001 $\mu g/m^3$ was estimated as the midpoint between the modeled concentration (0.00012 $\mu g/m^3$) and the empirical transfer coefficient estimate (0.002 $\mu g/m^3$). For the RME value, the high-end empirical transfer coefficient estimate of 0.017 $\mu g/m^3$ was chosen as the EPC. These values are summarized in Table 2-11.

Note that PCB-contaminated sediment and floodplain soil also could potentially contribute to PCBs in air. The contribution of PCBs in air from contaminated sediment and floodplain soil was not quantified for several reasons:

- The contribution is expected to be minor compared to the concentrations of PCBs in air that were obtained during periods of high activity (*i.e.*, Remnant Deposit remediation);

- the calculated cancer risks from inhalation of volatilized PCBs are *de minimus* (*i.e.*, insignificant); and

- consistent with the scope of the Reassessment RI/FS, the Revised HHRA addresses the cancer risks and non-cancer health hazards from PCBs in Upper Hudson River water and sediments, not floodplain soils.

The uncertainty associated with concentrations of PCBs in air from all sources, which could include river sediments periodically exposed to air, is discussed in Section 5.3.

2.4 Chemical Intake Algorithms

The following sections describe the calculation of PCB intake for each complete exposure pathway, including the algorithms and exposure parameters. Complete tabulations of the exposure factors for each exposure pathway and receptor scenario are found in Tables 2-12 through 2-24.

2.4.1 Ingestion of Fish

As has been noted earlier, the Revised HHRA contains both point estimate and Monte Carlo exposure estimates of PCB exposure *via* fish ingestion. For both the point estimate calculations and the Monte Carlo assessment (Chapter 3), the intake and cancer risks and non-cancer health hazards are calculated for an adult, adolescent, and young child angler. This section summarizes the exposure calculations and factors for the point estimate analysis. Many of the point estimate factors were selected based upon the analysis and derivation of their respective probability distributions; these are derived and described in detail in Chapter 3.

The fish ingestion point estimate intake is calculated as:

$$\text{Intake}_{\text{fish}}(\text{mg / kg - d}) = \frac{C_{\text{fish}} \times IR \times (1 - LOSS) \times FS \times EF \times ED \times CF}{BW \times AT}$$

where:

C_{fish}	=	Concentration of PCBs in fish (mg/kg)
IR	=	Annualized fish ingestion rate (g/day)
LOSS	=	Cooking loss (g/g)
FS	=	Fraction from source (unitless fraction)
EF	=	Exposure frequency (days/year)
ED	=	Exposure duration (years)
CF	=	Conversion Factor (10^{-3} kg/g)
BW	=	Body weight (kg)
AT	=	Averaging time - period over which exposure is averaged (days); over a lifetime for evaluating cancer risks and over the appropriate exposure duration for evaluating non-cancer health hazards.

Exposure factor values for the CT and RME point estimate calculations for this pathway are summarized in Tables 2-12a through 2-12c. Site-specific considerations in selecting these factors are discussed below.

Fraction from Source (FS). The Revised HHRA examines possible exposure for the population of anglers who consume self-caught fish from the Upper Hudson River. The exposure and cancer risks and non-cancer health hazard analyses assume the Upper Hudson River accounts for 100% of the sportfish catch of the angler (FS=1). As noted below, the fish ingestion rate is based upon consumption of sportfish, such that it excludes fish that may be purchased and then consumed. Given the large geographic area encompassed by the Upper Hudson River, it is not unreasonable to assume that a sizeable population of anglers could catch and consume a substantial percentage of their sportfish from this stretch of River. The 40-mile extent of the Upper Hudson River contains a variety of fish species commonly consumed by anglers and can support a sizeable sport fishery. Clearly, reducing this exposure factor would lead to reductions in the estimates of cancer risk and non-cancer health hazards. However, as discussed in Section 5.3.1, this exposure factor must be considered in the context of other factors, such as the fish ingestion rate, in order to examine the reasonableness of the overall intake of sportfish from the Upper Hudson River.

Exposure Frequency (EF). Because the fish ingestion rate is based on an annualized average ingestion over one year, an implicit exposure frequency value of 365 days/year is used in the intake calculation. This does not imply consumption of fish for 365 days per year.

Exposure Duration (ED). While Superfund risk assessments typically use the length of time that an individual remains in a single residence as an estimate for exposure duration, such an estimate is not necessarily a good predictor of exposure duration for an angler, because an individual may move from one residence to another and continue to fish in the same location, or an individual may chose to stop angling irrespective of the location of his or her home. Furthermore, given the large size of the Hudson River PCBs Superfund site, an individual may move from one place of residence to another and still remain within the Upper Hudson area and continue to fish from the Upper Hudson River. For the purposes of defining the angler population likely to fish the Upper Hudson River, it was assumed this population would be most likely to constitute residents from the five counties surrounding the Upper Hudson River (Albany, Rensselaer, Saratoga, Warren, and Washington). Furthermore, the 1988 New York Angler survey (see Chapter 3 discussion) found that the average distance traveled by New York anglers was 34 miles, providing further support for assuming that the majority of the angler population for the Upper Hudson River is likely to reside in the five surrounding counties.

Given the above considerations, the ED (angling duration) for the fish consumption pathway is not based solely upon a typical residence duration. Instead, as described in Section 3.2.4, an angler is assumed to continue fishing until any one of the following occurs:

- the individual stops fishing;
- the individual moves out of the area, or dies.

The 1991 New York Angler survey of over 1,000 anglers (Connelly *et al.*, 1992) was used to estimate fishing patterns within the population of New York anglers (see Section 3.2.1). The U.S. Census (1990a) provided the data for county to county mobility, which was used to estimate the range of residence durations within the five counties surrounding the Upper Hudson River (see Section 3.2.4).

As described in Section 3.2.1, the 50th percentile of the fishing duration distribution is 12 years and the 95th percentile is 40 years. These values were used as basis for the CT and RME point estimates, respectively. For comparison, 9 years, and 30 years are default ED factors for Superfund risk assessments based on national statistics of population mobility alone (USEPA, 1989a,b, 1991b, 1997f).

Body Weight (BW). The average adult body weight used in the intake equation was 70 kg, taken from USEPA (1989a,b; 1991b). Note that the adult body weight found in the 1997 Exposure Factors Handbook (USEPA, 1997f) is 71.8 kg. Because USEPA's derivation of the PCB cancer toxicity factors was based upon a 70 kg adult in extrapolating the animal data to humans, this assessment uses the prior 70 kg body weight value for consistency (USEPA, 1996b). Similarly, the average young child and adolescent body weights of 15 and 43 kg, respectively, were taken from USEPA (1991b, 1997f).

Averaging Time (AT). A 70-year lifetime averaging time of 25,550 days was used for cancer calculations (70 years × 365 day/year) (USEPA, 1989a,b; 1991b) even though the 1997 Exposure Factors Handbook (USEPA, 1997f) indicates that 75 years is the most current estimate. This is because the most current cancer slope factor is based on an average lifetime of 70 years (USEPA, 1996b,c). Had a 75 year averaging time been used, this would effectively decrease the calculated intake of PCBs in fish by 7%.

Non-cancer averaging times are not averaged over a lifetime, but rather over a period of time equating to a chronic level of exposure (USEPA, 1989a,b). Chronic exposures are those that exceed the subchronic exposure duration (*i.e.*, chronic exposure is 7 years or more). Because the PCB concentration in fish declines for the projected 70-year period covered by this risk assessment, the average concentration over time declines as the exposure period increases. Thus, the average concentration (and by extension, average PCB intake in terms of mg/kg-day) in a 7-year exposure period (USEPA, 1989a,b) is greater than the average concentration over 40 years. This leads to the somewhat counter-intuitive result that the average daily dose decreases as the exposure duration increases. For cancer risk evaluation, which is based upon a lifetime averaging period, this lower average daily dose still yields a higher overall PCB intake, simply because the intake is accumulated over the lifetime. For the evaluation of non-cancer health hazards to the RME adult or adolescent, it is inappropriate to extend the averaging time to an exposure duration greater than 7 years. Exposure durations longer that 7 years would result in a lower average daily dose, and thus would not be representative of an RME exposure.

Based on the foregoing considerations, the averaging time for the non-cancer health hazard assessment was set to 2,555 days (7 years × 365 days/year) for the RME adult and adolescent, and 2,190 days (6 years × 365 days/year) for the RME young child. The non-cancer averaging time for the CT estimate was set to 2,190 days for the adult and 1,095 days for the adolescent and young child (ED × 365 days/ year).

Concentration of PCB in Fish (C_{fish}). As described earlier in Section 2.3.1, the PCB concentration in fish was determined based on the modeled total PCB concentration results presented in the RBMR (USEPA, 2000a), combined with the fish consumption patterns as defined by the 1991 New York Angler survey (Connelly *et al.*, 1992). For the evaluation of cancer risks and non-cancer health hazards, the CT EPCs are 3.0 mg/kg for the adult and 3.3 mg/kg PCBs for the adolescent and young child. These EPCs were calculated by averaging the species-weighted concentration distribution over 6, 3, and 3 years for the adult, adolescent, and young child exposures, respectively. The exposure durations for each age group sum to 12 years, the 50th percentile exposure duration estimate. The high-end exposure EPCs for the evaluation of cancer risks are 2.0, 2.5, and 3.0 mg/kg PCBs for the adult, adolescent, and young child exposures, respectively. These EPCs were calculated by averaging the species-weighted concentration distribution over 22, 12, and 6 years for the adult, adolescent, and young child exposures, respectively. The RME exposure durations sum to 40 years, the 95th percentile exposure duration

estimate. The RME exposure EPCs for the evaluation of non-cancer health hazards are 2.9 mg/kg for the adult and adolescent (averaged over the 7-year chronic exposure) and 3.0 mg/kg PCBs for the young child (averaged over the 6-year exposure duration). It should be noted that the apparent contradiction in EPCs, whereby the RME EPC is lower than the CT EPC, is a direct result of the declining PCB concentration in fish over time. Due to this decline over time, the average concentrations over exposure durations are less than the average concentrations averaged over shorted time periods.

Fish Ingestion Rate (IR). The fish ingestion rate is based upon an estimate of the long term average consumption of self-caught fish in the angler population, expressed as an annualized daily average rate in units of grams of fish per day (g/day). It is important to note that the ingestion of fish from all sources (*e.g.*, self-caught plus purchased fish) is necessarily greater than or equal to the ingestion rate of only self-caught fish. Because the Revised HHRA examines the cancer risks and non-cancer health hazards of PCB intake from Hudson River fish only, the focus is only on self-caught fish.

As described in Section 3.2.1, the fish ingestion rate is based upon a survey of over 1,000 New York anglers (Connelly *et al.*, 1992) who caught and consumed fish in 1991. For the adult exposure, the CT fish ingestion rate is the 50th percentile of the empirical distribution (4.0 g/day) and the RME ingestion rate is the 90th percentile (31.9 g/day).[5] For a one-half pound serving, these ingestion rates represent approximately 6.4 and 51 fish meals per year, respectively.

For the adolescent and young child, fish ingestion rates were estimated to be approximately 2/3 that of an adult for an adolescent, and 1/3 that of an adult for a young child (see Tables 2-12a through 2-12c). This approach yields ingestion rates generally consistent with the limited information provided in USEPA Exposure Factor's Handbook (USEPA, 1997f). According to Table 10-1 of the Exposure Factor's Handbook (USEPA, 1997f), the 95th percentile intake for children aged 0-9 is 16.5 g/d, compared to the RME value used in the Revised HHRA of 10.6. g/d for children aged 1-6. For adolescents aged 10-19, the 95th percentile intake in USEPA's Exposure Factors Handbook (USEPA, 1997f) is 26.8 g/d, which compares favorably with the 21.3 g/d used in the Revised HHRA for adolescents aged 7-18.

Cooking Loss (LOSS). Numerous studies have examined the loss of PCBs from fish during food preparation and cooking. A review of the available literature is discussed in detail in Section 3.2.3 and a brief summary is presented here.

Experimental results range considerably, both between various cooking methods and within the same method. Cooking losses, expressed as percent loss based on total PCB mass before and after cooking, as high as 74 percent were reported in one study (Skea *et al.,* 1979). Several studies reported net gains of PCBs (Moya *et al.*, 1998; Armbruster *et al.*, 1987).[6]

Despite a wide range of data covering 12 studies, it is not possible to determine the key factors that influence the extent of PCB cooking losses. PCB losses from cooking may be a function of the cooking method (*i.e.*, baking, frying, broiling, *etc*.), the cooking duration, the temperature during cooking, preparation techniques (*i.e.*, trimmed *vs.* untrimmed, with or without skin), the lipid content of the fish, the fish species, the magnitude of the PCB contamination in the raw fish, the extent to which lipids separated during cooking are consumed, the reporting method, and/or the experimental study design. In addition, personal preferences for various preparation and cooking methods and other related habits (such as consuming pan drippings) may result in consumption of PCBs "lost" from the fish upon cooking.

[5] A fish ingestion rate of 30 grams per day was used in the Phase 1 risk assessment which was the USEPA-recommended median value at the time of that report (USEPA, 1989a).
[6] It is likely that the net gain is within the experimental measurement error and essentially indicates zero loss.

The 12 studies reviewed (Section 3.2.3 and Table 3.5) support the conclusion that cooking loss may be zero to 74 percent. Despite the rather wide range of cooking loss estimates, most PCB losses were between 10 and 40 percent. A value of 20% (midpoint of 0% - 40%) was selected as the central tendency point estimate for cooking loss. For the RME, no cooking loss (LOSS = 0%) was selected to include the possibility that pan drippings are consumed along with the fish.

2.4.2 Ingestion of Sediment

For the sediment ingestion pathway, intake is calculated as:

$$\text{Intake}_{\text{ingestion}}\,(\text{mg / kg - d}) = \frac{C_{sed} \times IR \times FS \times EF \times ED \times CF}{BW \times AT}$$

where:

C_{sed}	=	Concentration of PCBs in sediment (mg/kg)
IR	=	Sediment ingestion rate (mg/day)
FS	=	Fraction from source (unitless fraction)
EF	=	Exposure frequency (days/year)
ED	=	Exposure duration (years)
CF	=	Conversion factor (10^{-6} kg/mg)
BW	=	Body weight (kg)
AT	=	Averaging time - period over which exposure is averaged (days); over a lifetime for evaluating cancer risks and over the appropriate exposure duration for evaluating non-cancer health hazards.

Exposure factor values for the CT and RME calculations for this pathway are summarized in Tables 2-13 through 2-15. Site-specific considerations in selecting these factors are discussed below.

PCB Concentration in Sediment (C_{sed}). As described in Section 2.3.2, the RBMR (USEPA, 2000a) contains 70-year projections of the PCB concentration in sediment. The CT EPCs of 6.6, 7.2, and 7.2 mg/kg PCBs for adult, adolescent, and young child exposures, respectively, were calculated by averaging the modeled mean sediment distribution over 5, 3, and 3 years for adult, adolescent, and young child exposures, respectively. These average exposure durations for each age group sum to 11 years, the 50th percentile residence duration in the five counties surrounding the Upper Hudson. The RME EPCs of 3.8, 5.2, and 6.4 mg/kg PCBs for adult, adolescent, and young child exposures, respectively, were calculated by averaging the modeled sediment distribution over 23, 12, and 6 years for adult, adolescent, and young child exposures, respectively. These RME exposure durations sum to 41 years, the 95th percentile residence duration in the five counties surrounding the Upper Hudson.

Sediment Ingestion Rate (IR). This factor provides an estimate of incidental intake of sediment that may occur as a result of hand-to-mouth activity. In the absence of site-specific ingestion rates, USEPA-recommended median values for daily soil ingestion, rather than high-end values, were used for this factor to account for the shorter timeframes spent by recreators at the Hudson River. The incidental ingestion rate for children is 100 mg/day and for adults and adolescents is 50 mg/day. These values are also the reported median estimates of soil intake found in USEPA's current Exposure Factors Handbook

(USEPA, 1997f).[7] The incidental soil (sediment) ingestion rate provides an estimate of the ingestion that may occur integrated over a variety of activities, including ingestion of indoor dust.

Exposure Frequency (EF). Exposure to river sediments is most likely to occur during recreational activities. However, there are no site-specific data to provide an indication of the likely frequency of recreational activities along the Upper Hudson River, nor are there general population studies that provide usable information. Therefore, two cases were considered in this risk assessment: a "recreator" and an "avid recreator". For the recreator scenario, an assumption that recreational activities are likely to be most frequent during the summer months is made, and an estimate of one day per week during the 13 weeks of summer is considered a reasonable estimate of the RME value for adults (*i.e.*, 13 days per year). This same frequency was used for the young child (aged 1-6), assuming that the young child would most likely be accompanied by an adult. For the adolescent (aged 7-18), who is not as likely to be accompanied by an adult, it was assumed recreational frequency was three-fold greater than the adult/child frequency (*i.e.*, 39 days per year). The RME values were reduced by 50% for the CT exposure. The RME exposure frequency factors used are approximately 2- to 3-fold higher than the values used in the 1991 Phase 1 risk assessment (USEPA, 1991a).

For the avid recreator, an exposure frequency of 104 days/year was assumed for all age groups as the RME estimate, which corresponds to approximately 4 days per week for 6 months (26 weeks) of the year. This exposure frequency was also assumed for adults and adolescents in the Rogers Island Site Risk Assessment (USEPA, 1999h). An exposure frequency of 52 days/year (50% of the RME value) was used as the CT estimate.

Exposure Duration (ED). The RME exposure duration for sediment ingestion in recreational scenarios is 41 years, and the CT exposure duration is 11 years, which correspond to the 95th and 50th percentiles, respectively, of the residence duration determined for the five counties surrounding the Upper Hudson (see Section 3.2.4.3 and Figure 3-5a). The RME exposure duration is 6 years for the young child, 12 years for the adolescent, and 23 years for the adult (summing to 41 years), and the CT exposure duration is 3 years for the young child, 3 years for the adolescent, and 5 years for the adult (which sum to 11 years). Note that these values are somewhat greater than values determined from nationwide statistics, which indicate 30 years is the 95th percentile and 9 years is the 50th percentile residence duration at one location (USEPA, 1997f). Also, note that the exposure durations for the recreator (41 years for RME and 11 years for CT) based on residence duration are very similar to the exposure duration for the angler (40 years for RME and 12 years for CT) based on angling duration.

Body Weight (BW). Age-specific body weights were used. The mean BW for children aged 1 to 6 is 15 kg, the mean body weight for adolescents aged 7-18 is 43 kg, and the mean adult body weight is 70 kg (USEPA, 1989a,b; 1991b).

Averaging Time (AT). For all recreational exposure calculations, a 70-year lifetime averaging time of 25,550 days (365 days × 70 years) was used for cancer evaluations. Non-cancer averaging times are equal to the ED multiplied by 365 days/year (USEPA, 1989a,b).

[7] In the Phase 1 risk assessment, a value of 200 mg/day was used as the sediment ingestion rate for children, and 100 mg/day for adolescents and adults, was used as outlined in USEPA's Standard Default Guidance and RAGS Part A (USEPA, 1989b and 1991b).

2.4.3 Dermal Contact with Sediment

For the sediment dermal contact, absorbed doses are used (USEPA, 1999f). Dermal intake (the amount absorbed into the body) is calculated as:

$$\text{Intake}_{\text{dermal}}\,(\text{mg / kg - d}) = \frac{C_{sed} \times DA \times AF \times SA \times EF \times ED \times CF}{BW \times AT}$$

where:

C_{sed}	=	Concentration PCBs in sediment (mg/kg),
DA	=	Dermal absorption fraction (unitless),
AF	=	Sediment/skin adherence factor (mg/cm^2),
SA	=	Skin surface area exposed (cm^2/exposure event),
EF	=	Exposure frequency (exposure events/year),
ED	=	Exposure duration (years),
CF	=	Conversion factor (10^{-6} kg/mg)
BW	=	Body weight (kg)
AT	=	Averaging time - period over which exposure is averaged (days); over a lifetime for evaluating cancer risks and over the appropriate exposure duration for evaluating non-cancer health hazards.

Exposure factor values for the CT and RME calculations for the dermal contact pathway are summarized in Tables 2-13 through 2-15. Site-specific considerations in selecting these factors are discussed below.

PCB Concentration in Sediment (C_{sed}). As described in Section 2.3.2, the RBMR (USEPA, 2000a) contains 70-year projections of the PCB concentration in sediment. The CT EPCs of 6.6, 7.2, and 7.2 mg/kg PCBs for adult, adolescent, and young child exposures, respectively, were calculated by averaging the modeled mean sediment distribution over 5, 3, and 3 years for adult, adolescent, and young child exposures, respectively. These average exposure durations for each age group sum to 11 years, the 50^{th} percentile residence duration in the five counties surrounding the Upper Hudson. The RME EPCs of 3.8, 5.2, and 6.4 mg/kg PCBs for adult, adolescent, and young child exposures, respectively, were calculated by averaging the modeled sediment distribution over 23, 12, and 6 years for adult, adolescent, and young child exposures, respectively. These RME exposure durations sum to 41 years, the 95^{th} percentile residence duration in the five counties surrounding the Upper Hudson.

Dermal Absorption Fraction (DA). The dermal absorption fraction represents the amount of a chemical in contact with skin that is absorbed through the skin and into the bloodstream. The dermal absorption rate of 14% used is based on the *in vivo* percutaneous absorption of PCBs from soil by rhesus monkeys (Wester *et al.*, 1993 and USEPA, 1996c).

Soil/Skin Adherence Factor (AF). The sediment adherence values were obtained from USEPA's March 1999 Draft Dermal Risk Assessment Guidance (USEPA, 1999f) which, among other studies, relies upon data published by Kissel *et al.* (1998). That study represents a continuation of dermal adherence studies that provide the basis for the current exposure factors recommended by USEPA in its 1997 Exposure Factors Handbook (USEPA, 1997f).

The data in Kissel *et al.* (1998) include soil/skin adherence factors for a range of activities and individuals (*i.e.*, transplanting of bedding plants, laying of pipe by adults, children's play, *etc*.). For each of these activities, Kissel lists measured dermal adherence (soil loadings) on four body parts (hands, forearms, lower legs, and faces). Area weighted adherence factors for the Kissel, *et al.* (1998) study, and others, are presented in the March 1999 Draft Dermal Risk Assessment Guidance (USEPA, 1999f). The area-weighted sediment/skin adherence values for adults and children are determined by summing the soil loading rates of each body part (hands, forearms, lower legs and face) multiplied by their respective surface area, and dividing by the sum of the surface areas. The resulting 50^{th} percentile sediment/skin adherence factor for children is 0.2 mg/cm^2, and 0.3 mg/cm^2 for adults (USEPA, 1999f). These adherence factors are for children playing in wet soil, and adults whose soil loadings were measured for reed gathering activities. These activities, which represent active contact with soil, are appropriate surrogates for activities in which recreators in the Upper Hudson River may contact sediment. The soil adherence factor for adolescents was taken as the midpoint between the child and adult factors.

Skin Surface Area Exposed (SA). For children and adolescents, the mean surface area of hands, forearms, lower legs, feet, and face were calculated by multiplying the total body surface area (averaged between males and females) by the percentage of total body surface area that make up the relevant body parts (USEPA, 1997f). For children, the mean surface area of the hands, forearms, lower legs, feet, and face is 2,792 cm^2 (using data for the category 6 to 7 years old); for adolescents, the mean surface area of the hands, forearms, lower legs, feet, and face is 4,263 cm^2 (for 12 years old); for adults, the mean surface area of hands, forearms, lower legs, feet, and face is 6,073 cm^2 (USEPA, 1997f). In the Phase 1 risk assessment, the corresponding exposure factors used were: 3,931 cm^2, 7,420 cm^2, and 5,170 cm^2 for child, adolescent, and adult surface areas, respectively. These prior values were based upon the surface area of the child/adolescent legs, feet, arms, and hands, and adult lower legs and feet, forearms, and hands.

Exposure Frequency (EF). As described above, there are no site-specific data to provide an indication of the likely frequency of recreational activities along the Upper Hudson River, nor do general population studies exist. The exposure frequency factors (Tables 2-13 through 2-15) for dermal contact are the same as those for incidental ingestion of sediment described in the preceding section.

Exposure Duration (ED). The ED for sediment dermal contact in recreational scenarios is the same as that described in the previous section for incidental ingestion.

Body Weight (BW). Age-specific body weights were used. The mean BW for children aged 1 to 6 is 15 kg, the mean BW for adolescents aged 7-18 is 43 kg, and the mean adult body weight is 70 kg (USEPA, 1989a,b; 1991b).

Averaging Time (AT). For all recreational exposure calculations, a 70-year lifetime averaging time of 25,550 days (365 days × 70 years) was used for cancer assessments. Non-cancer averaging times are equal to the ED multiplied by 365 days/year (USEPA, 1989a,b).

2.4.4 Dermal Contact with River Water

For the river water dermal contact pathway, dermal intake (the amount absorbed into the body) is calculated as:

$$\text{Intake}_{\text{water}}(\text{mg / kg - d}) = \frac{C_w \times K_p \times SA \times DE \times EF \times ED \times CF}{BW \times AT}$$

where:

C_w	=	Concentration of PCBs in water (mg/l)
K_p	=	Chemical-specific dermal permeability constant (cm/hr)
SA	=	Skin surface area exposed (cm^2)
DE	=	Duration of event (hr/d)
EF	=	Exposure frequency (d/year)
ED	=	Exposure duration (years)
CF	=	Conversion factor (10^{-3} L/cm^3)
BW	=	Body weight (kg)
AT	=	Averaging time - period over which exposure is averaged (days); over a lifetime for evaluating cancer risks and over the appropriate exposure duration for evaluating non-cancer health hazards.

Exposure factor values for the CT and RME point estimate calculations for the dermal contact with river water pathway are summarized in Tables 2-16 through 2-18. Site-specific considerations in selecting these factors are discussed below.

PCB Concentrations in River Water (C_w). As described in Section 2.3.3, the RBMR (USEPA, 2000a) contains 70-year projections of the PCB concentration in river water. The CT EPCs of 4.6×10^{-5}, 4.8×10^{-5}, and 4.8×10^{-5} mg/L PCBs for adult, adolescent, and young child exposures, respectively, were calculated by averaging the mean modeled river water distribution over 5, 3, and 3 years for adult, adolescent, and young child exposures, respectively. These average exposure durations for each age group sum to 11 years, the 50^{th} percentile residence duration in the five counties surrounding the Upper Hudson. The RME EPCs of 3.4×10^{-5}, 4.0×10^{-5}, and 4.5×10^{-5} mg/L PCBs for adult, adolescent, and young child exposures, respectively, were calculated by averaging the modeled river water distribution over 23, 12, and 6 years for adult, adolescent, and young child exposures, respectively. These RME exposure durations sum to 41 years, the 95^{th} percentile residence duration in the five counties surrounding the Upper Hudson.

Permeability Constant (K_p). In the absence of experimental measurements for the dermal permeability constant for PCBs, it was estimated to be 0.48 cm/hr based on the value for hexachlorobiphenyls reported in the 1999 Draft Dermal Risk Assessment Guidance (USEPA, 1999f).

Skin Surface Area Exposed (SA). As a conservative estimate of possible exposure, 100% of the full-body surface area was assumed to come into contact with water. The surface areas for adults, adolescents, and young children, respectively are: 18,150 cm^2, 13,100 cm^2, and 6,880 cm^2 (USEPA, 1997f).

Duration of Event (DE). For all recreator scenarios, 2.6 hours/day was used as the river water dermal exposure time, which is the national average duration for a swimming event (USEPA, 1989a,b).

Exposure Frequency (EF). As described above, there are no site-specific data to provide an indication of the likely frequency of recreational activities along the Upper Hudson River, nor do general population studies exist that provide usable information. The EF factors (Tables 2-16 through 2-18) for

dermal contact with water while swimming are the same as those for incidental ingestion and dermal contact with sediments described in the preceding sections.

Exposure Duration (ED). The ED for river water dermal contact in recreational scenarios is the same as that described in the previous sections for incidental ingestion and dermal contact with sediment.

Body Weight (BW). Age-specific body weights were used. The mean BW for children aged 1 to 6 is 15 kg, the mean body weight for adolescents aged 7-18 is 43 kg, and the mean adult (over 18 years old) body weight is 70 kg (USEPA, 1989a,b; 1991b).

Averaging Time (AT). For all recreational exposure calculations, a 70-year lifetime averaging time of 25,550 days (365 days × 70 years) was used for cancer evaluations. Non-cancer averaging times are equal to the ED multiplied by 365 days/year (USEPA, 1989a,b).

2.4.5 Inhalation of PCBs in Air

For the inhalation pathway, intake is calculated as:

$$\text{Intake}_{\text{inhalation}}\,(\text{mg / kg - d}) = \frac{C_{\text{air}} \times \text{IR} \times \text{DE} \times \text{EF} \times \text{ED} \times \text{CF}}{\text{BW} \times \text{AT}}$$

where:

C_{air}	=	Concentration of the chemical in air ($\mu g/m^3$),
IR	=	Inhalation rate (m^3/hr)
DE	=	Duration of event (hrs/day)
EF	=	Exposure frequency (days/yr)
ED	=	Exposure duration (yrs)
CF	=	Conversion factor (10^{-3} mg/μg)
BW	=	Body weight (kg)
AT	=	Averaging time - period over which exposure is averaged (days); over a lifetime for evaluating cancer risks and over the appropriate exposure duration for evaluating non-cancer health hazards.

Exposure factor values for the CT and RME calculations for the inhalation of PCBs in air pathway are summarized in Tables 2-19 through 2-24. Site-specific considerations in selecting these factors are discussed below.

PCB Concentrations in Air (C_{air}). The EPCs, summarized in Section 2.3.4 and Appendix B, were estimated for areas in the immediate proximity of the Upper Hudson River. The CT EPC is 1×10^{-6} mg/m^3, the RME EPC is 1.7×10^{-5} mg/m^3.

Inhalation Rate (IR). For adult residents, the inhalation rate used is 20 m^3/day, which is the recommended value for long term exposure assessments for Superfund risk assessments (USEPA, 1991b). The inhalation rate for young children (10 m^3/day) and adolescents (13.5 m^3/day) used to calculate inhalation are current recommendations in the 1997 Exposure Factors Handbook for long term exposures (USEPA, 1997f).[8] The same values were used in both CT and RME calculations.

[8] These values are based on children aged 6-8 years and the average male/female adolescent 12-14 year age category.

For all recreational scenarios, the mean inhalation rate values for short-term, moderate activities were used: 1.6 m^3/hr for adults and adolescents, and 1.2 m^3/hr for young children (USEPA, 1997f).

Exposure Frequency (EF). Because residents may be exposed to PCB-affected air when performing activities outside their homes as well as when they are inside (through outside air exchange), a RME scenario assuming exposure 24 hours a day, 350 days a year was used (which assumes 2 weeks away from the residence). The EF for inhalation of air during recreational activities is the same as those for incidental ingestion of sediment and dermal contact with sediment and river water.

Exposure Duration (ED). The ED for the inhalation pathway is the same as that described in the previous sections for contact with sediment and water.

Body Weight (BW). Age-specific body weights were used. The mean BW for children aged 1 to 6 is 15 kg, the mean body weight for adolescents aged 7-18 is 43 kg, and the mean adult (over 18 years old) body weight is 70 kg (USEPA, 1989a,b, 1991b).

Averaging Time (AT). A 70-year averaging time of 25,550 days was used for cancer evaluations (365 days/year × 70 years). Non-cancer averaging times are equal to the exposure duration multiplied by 365 days/year (USEPA, 1989a,b).

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3 Monte Carlo Exposure Analysis of Fish Ingestion Pathway

A Monte Carlo analysis was conducted pursuant to the Agency's guidance on probabilistic analysis for risk assessment (USEPA, 1997a). The purpose of the Monte Carlo analysis is to estimate a probability distribution of PCB exposure among members of the angler population and to quantify the extent to which important sources of uncertainty affect the precision of these estimates. When combined with the toxicity information described in Chapter 4, the range of PCB exposure is translated into a range of cancer risks and non-cancer health hazards (Chapter 5).

USEPA's recently published and peer reviewed draft Probabilistic Risk Assessment Guidance document (USEPA, 2000e), currently being revised in response to the peer review input, provides a tiered approach for evaluating the need for a probabilistic risk assessment (*e.g.*, Monte Carlo analysis), the pathways to be evaluated, and the level of analysis. This document recommends that probabilistic risk assessment be used for those pathways posing the greatest cancer risks and non-cancer health hazards. Based on an analysis of the results from the point estimate analysis, only the cancer risks and non-cancer health hazards associated with fish ingestion were evaluated using Monte Carlo analysis.

As described earlier, USEPA's guidance for Superfund risk assessments and USEPA policy recommends an evaluation of RME. In the preceding section, the point estimate method of estimating the RME was outlined, which consists of combining high-end and appropriate average exposure estimates for exposure factors such that the combination of factors yields an estimate of an individual who may experience a reasonable maximum exposure. While the RME is widely used to capture exposures in the high-end of the distribution (above the 90^{th} percentile), in practice it is rare that the precise probability associated with the RME can be determined. That is, the result is clearly a "high-end" estimate of exposure, but it is difficult to determine whether the high-end is the 90^{th} percentile, 95^{th} percentile, or 99^{th} percentile, *etc.* within a population.

Monte Carlo simulation methods provide an alternative approach to estimating the RME using probabilistic analysis. The advantage of Monte Carlo methods is that, given sufficient data on parameter distributions, they can provide an explicit estimate of the likelihood, or probability, associated with the entire range of exposure -- this quantitative estimate of the probability of exposure translates into a quantitative estimate of the probability of cancer risk or non-cancer hazard, as discussed in Chapter 5. The Monte Carlo exposure analysis is a more resource-intensive analysis and requires more detailed information, in the form of probability distributions, to describe the range of plausible values for the exposure factors and, consequently, to provide a distribution of cancer risks and non-cancer health hazards.

After the exposure factor distributions have been determined, performing the Monte Carlo simulation is relatively straightforward: the range and relative likelihood of exposure is calculated by sampling exposure factors from their respective probability distributions. The simulation randomly selects a value from each parameter's distribution and calculates the corresponding PCB intake, repeating this process many times. The collection of computed PCB intake values approximates the exposure distribution for the population of interest.

Although the actual simulation process is straightforward, the significant challenge of a Monte Carlo analysis lies in developing the probability distributions of each exposure factor. The majority of the discussion in this chapter examines the information sources used to derive the distributions for each of the exposure factors and outlines the uncertainties involved in deriving the input probability. In the August 1999 HHRA, USEPA presented the results of its Monte Carlo analysis for the fish ingestion pathway.

That analysis, which was composed of 72 combinations of input parameters for 10,000 anglers (for a total of 720,000 computer simulations), showed that USEPA's RME point estimates were appropriately within the high-end ($\geq 90^{th}$ percentile) for cancer risks and non-cancer health hazards (see, comparison of point estimates and Monte Carlo analysis in Chapter 5). Because the peer reviewers did not recommend any changes to the most important exposure parameters for the fish ingestion pathway (*e.g.*, fish ingestion rate, exposure duration, exposure point concentration in fish) and due to the extensive level of effort required to perform a Monte Carlo analysis, USEPA did not conduct another Monte Carlo analysis for the Revised HHRA. Nevertheless, suggestions from the peer reviewers to better explain and describe the August 1999 Monte Carlo analysis were incorporated into this Revised HHRA. Furthermore, as discussed in the March 2000 Human Health Risk Assessment Responsiveness Summary (USEPA, 2000b), there was a 2-fold, or less, difference in the modeled PCB concentrations in fish when the August 1999 results are compared to those used in this Revised HHRA. Thus, even if the Monte Carlo analysis had been updated to account for the revised modeling results, the overall outcome and conclusions would not change substantially.

3.1 Discussion of Variability and Uncertainty

This section highlights the distinction between two important concepts in the analysis, variability and uncertainty, each of which contribute to variations in the exposure calculations. It is important to segregate the influence of variability and uncertainty in the context of the Monte Carlo analysis because they are different concepts: variability addresses the issue of whether there are members of the population with a particularly elevated level of intake (and by extension cancer risks and non-cancer health hazards), whereas uncertainty affects the precision of the intake estimates.

Exposure factors can vary among the population, and they can be uncertain due to limited information. Parameter variability is an inherent reflection of the natural variation within a population (*e.g.*, true differences in fish ingestion rates, exposure duration, body weight, *etc.*). Uncertainty represents a lack of perfect knowledge about specific variables, models, or other factors. Uncertainty can be reduced through further study, measurements, *etc.*, whereas variability cannot. Further study of the variability of the characteristics affecting exposure within a population can improve the accuracy with which the variability can be modeled and thus can improve the accuracy of exposure estimates and resulting cancer risk and non-cancer hazard estimates.

The exposure factor parameters used to estimate chemical intake, in concept, have multiple possible values for any of three reasons. First, a parameter's true value may be uncertain, but may not vary substantially across different members of the population. In this case, the parameter has one "true" value for all members of the population of interest, but that value is not known precisely. Second, a parameter's value may vary from member to member of the population, but be treated as known with relative certainty. For example, the distribution of human body weights within a population clearly varies, yet given a sufficient number of measurements the variability may be determined with accuracy. Third, a quantity may both be uncertain and vary from member to member of the population. In practice, most exposure factors fall into this third category. Assessments need to address both variability in a population and scientific uncertainty in the cancer risk and non-cancer health hazard estimates. The effects of these factors need to be addressed separately and not mixed together in an assessment to develop a single risk distribution. There are different alternatives for presenting information on variability and uncertainty, depending on the available data and assessment needs.

If the distinction between uncertainty of an exposure factor and true variability among the population were not distinguishable, then a single probability distribution for each exposure factor would

be all that is needed for a Monte Carlo analysis. In this instance, a "one-dimensional" (1-D) Monte Carlo analysis would proceed repeatedly drawing randomly selected values for each stochastic parameter (*i.e.*, a random sample reflecting a combination of uncertainty and variability). For each set of values drawn, the simulation computes an intake, repeating this process a large number of times. The resulting set of intake (exposure) estimates can be plotted as a histogram that approximates the range and relative likelihood of the plausible exposure that may exist in the modeled population. However, this approximation to the probability distribution of exposure (and cancer risks and non-cancer health hazards) generated by a one-dimensional Monte Carlo simulation has embedded within it both variability and uncertainty. Because it reflects both uncertainty and variability, it is broader than the true distribution of cancer risks and non-cancer health hazards. Moreover, it cannot be thought of as representing the risk that would occur for any one individual within a population.

A two-dimensional (2-D), or nested Monte Carlo simulation addresses this problem by conducting a large number of separate one-dimensional (1-D) simulations. For each 1-D simulation, a fixed set of randomly selected values is assigned to each of the uncertain parameters. Values for variable parameters are permitted to vary within each 1-D simulation. Each 1-D simulation produces a large number of intake estimates (*e.g.*, 1,000 to 10,000 or more such estimates) representing the set of PCB intake incurred by members of a population, given the fixed values assigned to each uncertain parameter for that simulation.

The results of a 2-D analysis can be used to quantify the distribution of plausible cancer risks and non-cancer health hazards for representative members of the population. For example, the range of plausible cancer risks for the "median individual" (*i.e.*, the individual whose risk is greater than the cancer risk for one-half of the population, and less than the cancer risk for the other half) is estimated by collecting the median cancer risk values from each of the repeated 1-D simulations, where the 1-D simulations are repeated using alternative exposure parameter distributions or ranges, reflecting their uncertainty.

In the Scope of Work for the Phase 2 HHRA (USEPA, 1998a), a 2-D Monte Carlo analysis had been proposed in order to explicitly address uncertainty and variability. The 2-D analysis involves: (1) defining probability distributions that reflect the parameter variability (*i.e.*, true differences in fish ingestion, exposure frequency, exposure duration, body weight, *etc.* within an exposed population), and (2) evaluating the uncertainty associated with the exposure factor distributions. Thus, the first component (variability analysis) of this process yields a probability distribution that conveys information on the range of risk experienced by individuals within a population, and allows a quantitative estimate of the RME individual (such as the 95^{th} percentile exposure and cancer risk and non-cancer health hazards). The second component (uncertainty analysis) is intended to provide quantitative estimates of the accuracy of the predictions. Uncertainty in the exposure parameter estimates affects the precision of the resulting cancer risk and non-cancer health hazard estimates. The more reliable the available information is to define the exposure factor probability distributions, the narrower the range of Monte Carlo exposure estimates for any particular exposure percentile; conversely, greater uncertainty in the exposure factor distributions leads to wider range in the cancer risk and non-cancer health hazard estimates.

While a nested 2-D Monte Carlo provides a framework for evaluating both the variability of exposure within a population and provides a quantitative estimate of the accuracy of the exposure, the information required to conduct the analysis is substantial. Modeling variability and uncertainty separately requires not only a probability distribution defining the variability for a particular parameter, but also a quantitative measure of the uncertainty for that probability distribution. For example, consider modeling the variability of a particular exposure parameter, such as fish ingestion, as a lognormal random variable with parameters μ (mean) and σ^2 (variance). To accomplish a fully 2-D analysis, quantitative

uncertainty distributions for both the mean and variance would be necessary. In other words, not only is a probability distribution of fish ingestion required, but probability distributions for plausible values of μ and σ^2 are also required (requiring three probability distributions, one for fish ingestion and one each for μ and σ^2). Clearly, such an approach requires much more information than a 1-D analysis.

For the reasons described later in this section, an explicit 2-D analysis was not performed due to insufficient information available to define quantitative uncertainty distributions for several important exposure factors. The analysis conducted is an enhanced 1-D Monte Carlo analysis of the *variability* of exposure as a function of the variability of individual exposure factors. The second component of the analysis includes an uncertainty/sensitivity analysis for the important exposure variables. This sensitivity analysis examines changes in the predicted bottom line distribution of population variability when alternative assumptions are made for the distribution of exposure factors. A total of 72 separate combinations of the variable input parameters were examined in the uncertainty analysis. Thus, the likely precision of each percentile of the exposure estimate distribution is not characterized by a specific probability, but rather the range of exposure estimates for each percentile is presented to give the reader an estimate of how wide or narrow is the range of the exposure estimates given the combined uncertainty of the exposure factors.

Consistent with USEPA policy (USEPA, 1997a), the variability and/or uncertainty associated with chemical toxicity was not included quantitatively in this Monte Carlo analysis. USEPA recognizes the uncertainty inherent in the determination of cancer and non-cancer toxicity factors, and the uncertainty is factored into the determination of the toxicity factors when they are published in USEPA's Integrated Risk Information System (IRIS). The USEPA's Risk Assessment Forum held a colloquia on this issue in September of 2000; no specific guidance has been issued by the Agency regarding possible methods for including toxicity uncertainty within a Monte Carlo analysis. A discussion of the uncertainty associated with toxicity values is presented in Chapter 4 and in the discussion of uncertainties in Chapter 5.

3.2 Derivation of Exposure Factor Distributions

The Monte Carlo analysis calculates chemical intake *via* fish ingestion based upon the basic intake equation defined in Section 2.3.1, which is repeated here for ease of reference:

$$Intake_{fish}(mg/kg\text{-}d) = \frac{C_{fish} \times IR \times (1 - LOSS) \times FS \times EF \times ED}{BW \times AT} \times CF$$

where:

C_{fish}	=	Species weighted concentration of PCBs in fish (mg/kg)
IR	=	Annualized fish ingestion rate (g/day)
LOSS	=	Cooking loss (g/g)
FS	=	Fraction from source (unitless fraction)

EF	=	Exposure frequency (days/year),
ED	=	Exposure duration (years),
CF	=	Conversion Factor (10^{-3} kg/g)
BW	=	Body weight (kg),
AT	=	Averaging time - period over which exposure is averaged (days); over a lifetime for evaluating cancer risks and over the appropriate exposure duration for evaluating non-cancer health hazards.

For the point estimate exposure analysis, several parameters (C_{fish} and IR in particular) were based on weighted average inputs based upon species ingestion rates. The Monte Carlo analysis does not adopt weighted averages for these exposure factors. Consequently, the calculation of PCB intake from fish ingestion for the Monte Carlo simulation is the summation of the annualized intake over the exposure duration and over all fish species as shown in the following equation:

$$Intake = \sum_{f} \sum_{y=1999}^{1999+ED-1} \left(\frac{C_{f,y} \times IR_a \times PCT_f \times (1 - LOSS) \times FS \times EF}{BW_a \times AT} \right) \times CF \qquad [3\text{-}1]$$

where:

Intake	=	PCB intake from all fish species over the exposure duration (mg/kg-day)
$C_{f,y}$	=	PCB concentration in fish species *f* in year *y* (mg/kg)
IR_a	=	Fish ingestion rate (g/day) at age *a* (*a = y - year of birth*)
PCT_f	=	Fraction of annual fish ingestion for species *f* (unitless fraction)
LOSS	=	PCB cooking loss (g/g)
FS	=	Fraction from source (unitless fraction)
EF	=	Exposure frequency (days/year)
ED	=	Exposure duration (years)
CF	=	Conversion factor (10^{-3} kg/g)
BW_a	=	Body weight (kg) at age *a* (*a = y - year* of birth)
AT	=	Averaging time - period over which exposure is averaged (days); over a lifetime for evaluating cancer risks and over the appropriate exposure duration for evaluating non-cancer health hazards.

In this form of the intake equation, ED, referred to here as the incremental ED, is the number of years until the individual stops fishing in the Upper Hudson River because the angler stops fishing altogether or the angler moves out of the region (or dies). The total dose over the exposure duration is given by summing over the three modeled fish species consumed (denoted by subscript *f*).

The variables in the above equation for which probability distributions or sensitivity analysis ranges were developed include:

IR_a	ingestion rate
$C_{f,y}$	concentration of PCBs in fish[9]
PCT_f	percent of species *f* consumed
LOSS	cooking loss
ED	exposure duration (*e.g.*, fishing duration)
BW_a	body weight

Parameters that were treated as constants in the Monte Carlo analysis, set to the same values as they were in the point estimate analysis, were the following:

FS	Fraction from source (100%)
EF	Exposure frequency
AT	Averaging time

A discussion of the derivation of the variable exposure factors is presented in the following subsections.

3.2.1 Fish Ingestion Rate

The fish ingestion rate term represents the amount of fish an individual consumes on average within the year, annualized such that it is expressed in units of grams of fish per day (g/day). Upper Hudson River anglers are defined as all individuals who would consume self-caught fish from the Upper Hudson River at least once per year in the absence of fish consumption advisories. The population in question therefore includes a range of infrequent to frequent anglers, who may fish for sport (recreational) or for sustenance (food source).

Based on a review of the available literature and consideration of a number of scientific issues relevant to fish ingestion rates, a probability distribution of fish consumption rates was determined using data from the 1991 New York Angler survey (Connelly *et al.*, 1992) to represent Upper Hudson River anglers. The statistics and percentiles for this distribution are summarized in Table 3-1. The point estimate exposure calculations used the 50^{th} percentile of the distribution (4.0 g/day) and the 90^{th} percentile (31.9 g/day) ingestion rates, corresponding to approximately 6.4 and 51 one-half pound meals per year, respectively. The entire distribution of fish ingestion rates was used in the Monte Carlo analysis to represent variability of fish consumption patterns among the angler population. The fish ingestion rate for anglers 10 to 18 years of age was scaled to the adult ingestion rates as the ratio of the body weight at a particular age, divided by adult body weight. A discussion of the fish ingestion surveys reviewed, and the derivation of the ingestion rate distribution selected, is presented in the following sections.

3.2.1.1 Summary of Fish Ingestion Rate Literature

Self-caught fish ingestion rates can vary based on many factors, including: the type of water body (flowing *vs.* still, freshwater *vs.* saltwater), the available fish species, the type of consumer (commercial *vs.* recreational), the preference for specific species, the effect of fish consumption advisories, weather, and the distance of the angler from the water body (reviewed in USEPA, 1997f). Numerous scientific studies of various water bodies (lakes, rivers, streams, *etc*.) have been conducted to identify fishing patterns (frequency, fishing practices, fish species preference, *etc*.) and fish consumption rates. Because the Upper Hudson River is a flowing body of water, the review of fish ingestion literature

[9] The Monte Carlo calculations presented are from the December 1999 HHRA, which used the modeled PCB concentrations from the Baseline Modeling Report (USEPA, 1999d).

focused on studies of anglers fishing in inland flowing waterbodies, also emphasizing studies conducted in the Northeast.

Fish ingestion studies can be either "creel" surveys, where anglers are interviewed in person while fishing, or mail surveys, where anglers (often identified as individuals with fishing licenses) are sent questionnaires in the mail (reviewed in USEPA, 1992d). Creel surveys typically involve interviews with anglers at the dockside requesting information about the fishing activities (fish preference, consumption rates, cooking methods, age, gender, frequency of fishing the specific water body, *etc.*). This survey method can provide information on both licensed and unlicensed anglers, depending upon who is interviewed. Mail surveys typically involve sending questionnaires to licensed anglers requesting information on fishing practices, preferred rivers, lakes or streams, fish consumption, and other information. However, if mailing addresses are obtained from lists of licensed anglers, unlicensed anglers will not be represented. A third type of survey, diary surveys, where participants are asked to record the frequency of fish ingestion, the types of fish eaten, and the meal size, require more effort on the part of the survey participants, but are generally assumed to yield more accurate results because the potential recall bias found in the other survey methods is minimized.

1988 New York Angler Survey (Connelly et al., 1990). In 1988, researchers at Cornell University performed a statewide mail survey to determine New York State anglers' fishing experiences during 1988 (Connelly *et al.*, 1990). Over 10,000 licensed anglers returned completed surveys regarding fishing preferences and interests. A subset of 200 individuals who did not respond to the mail survey was contacted by telephone to account for potential non-response bias. An estimated 26,870 anglers fished in the Hudson River in 1988. The mean distance traveled by anglers fishing in the Hudson was 34 miles. The mean number of fishing trips per Hudson angler was 8.6 trips, and the mean trip duration was 1.2 days. For all New York anglers, the mean age at which they began fishing regularly was 13.3 years of age. Although anglers were asked to estimate their total annual consumption of fish (fresh or saltwater, sport-caught or purchased), they were not specifically asked about the quantity of self-caught freshwater fish consumed.

1991 New York Angler Survey (Connelly et al., 1992). In 1991, researchers at Cornell University performed another statewide mail survey to determine New York anglers' awareness and knowledge of fishing advisories, and to determine fish consumption patterns during the 1991 fishing season (Connelly *et al.*, 1992). A total of 1,030 licensed anglers returned completed surveys. A subset of 100 individuals who did not respond to the mail survey was contacted by telephone to account for potential non-response bias. Anglers were also asked to report the number of fish caught and consumed in 1991 according to fish species and fishing location. The overall mean ingestion rate for New York anglers was 11 sport-caught fish meals in 1991. Analysis of the raw survey data also allowed determination of fish ingestion rates for specific locations or for categories of fishing locations (*i.e.*, rivers *vs.* lakes). About 85% of New York anglers were aware of fish consumption advisories, and almost half reported that they would eat more sport-caught fish if there were no problems with contaminants. Most New York anglers reported starting fishing at an early age; the mean age at which anglers began fishing was 14 years of age.

1992 Lake Ontario Diary Study (Connelly et al., 1996). Researchers at Cornell University performed a 12-month diary study targeting Lake Ontario anglers fishing in 1992 (Connelly *et al.*, 1996). The goal of the study was to provide accurate estimates of fish consumption among Lake Ontario anglers and to evaluate the effect of Lake Ontario fish consumption advisories. Participants were asked to record all fish consumption and fishing trips for an entire year (1992). Participation was encouraged even if anglers intended to fish infrequently to reduce bias toward only avid anglers. Participants were also contacted by telephone to follow-up every three months. A total of 1,202 anglers agreed to participate initially, but only 516 completed their diary for the entire year. Adjustments were made to account for

those with less than a full year participation to address potential biases. In January, 1992, participants were also asked to complete a questionnaire asking for 12-month recall of their 1991 fish consumption, which allowed for comparison of results from mail (recall) surveys and diary studies.

Based on the diary results, average daily consumption of sport-caught fish from all sport sources for Lake Ontario anglers was 2.2 g/day for the 50th percentile, and 17.9 g/day for the 95th percentile (Connelly *et al.*, 1996). For fish from all sources (sport-caught and purchased fish), the average daily consumption for Lake Ontario anglers was 14.1 g/day for the 50th percentile, and 42.3 g/day for the 95th percentile. The overall average sport-caught meal size was 232 g/meal, or approximately one-half pound. The 1991 12-month recall mail questionnaires yielded higher fish ingestion rates than those resulting from the diary data, suggesting that recall bias results in overestimates of fish ingestion (Connelly *et al.*, 1996; Connelly and Brown, 1995). Over 95% of the participants were aware of the New York State fish consumption advisories, and 32% indicated that they would eat more fish if there were no fish consumption advisories.

Additional Connelly Surveys (Connelly and Knuth, 1993; Connelly et al., 1993). In 1993, researchers at Cornell published two studies – one which evaluated angler knowledge and response to Great Lakes health advisories and assessed communication techniques (Connelly and Knuth, 1993), and one which evaluated health advisory awareness and associated behaviors among Lake Ontario anglers (Connelly *et al.*, 1993). Both reports focused specifically on Great Lakes anglers.

1996 and 1991-1992 Hudson Angler Surveys (NYSDOH, 1999b; Barclay, 1993). The NYSDOH conducted a creel survey of Hudson River anglers in 1996 (NYSDOH, 1999b). This survey used a slightly modified version of the questionnaire and interviewing technique used in a 1991-1992 creel survey of Hudson River anglers conducted by the Hudson River Sloop Clearwater organization (Barclay, 1993). A total of 460 Hudson River anglers were interviewed in the two surveys combined; of these, 132 anglers were from the area between Hudson Falls and the Federal Dam at Troy (the Upper Hudson River). For the following discussion, the 1991-1992 and 1996 surveys are combined and considered a single survey.

Of the Upper Hudson River anglers, over 85% were male; almost all (97%) were Caucasian. About 17% of the anglers were under age 20, and almost 10% were age 60 and older. Half of those surveyed had a New York State fishing license, 8% did not have a license, and 42% did not respond. All of the anglers interviewed from the Upper Hudson River were fishing from shore, and not from a boat. About half of the anglers in the Upper Hudson River area had caught any fish at the time of the interview; the most commonly reported fish caught included smallmouth bass, largemouth bass, and white perch. Blue crabs were caught only south of Catskill, not in the Upper Hudson River (NYSDOH, 1999b).

About two-thirds of the Upper Hudson River anglers were aware of official health advisories about eating fish from the Hudson. Only one angler reported food as a main reason for fishing; most anglers were fishing primarily for recreation or other similar reasons. About 92% reported that they never eat their catch, and similarly about 90% reported never giving their catch away to others. Only about 14% of Upper Hudson River anglers reported having eaten fish from the Hudson in the past; of those, about 37% reported eating fish once per week, about 19% reported eating fish 2-3 times per month, another 19% reported eating fish once per month, and 25% reported eating fish less than once per month (NYSDOH, 1999b).

About two thirds of the Upper Hudson River anglers reported fishing two times or less in the previous week; six percent reported fishing 7 times in the previous week. On a monthly basis, about half reported fishing three times or less in the previous month; about 12% reported fishing 20 or more times in

the previous month. Anglers were not asked about their total number of fishing trips per year (NYSDOH, 1999b).

1993 Maine Angler Survey (Ebert et al., 1993). Ebert and colleagues conducted a mail survey of licensed Maine anglers. A total of 1,612 licensed anglers returned completed surveys. Anglers were questioned about the number of fish caught and consumed from flowing and standing water bodies and the number of fishing trips completed in the 1990 season. The study authors developed a distribution of fish ingestion rates assuming that all freshwater fish caught by the angler is shared equally with other household members, with the 50^{th} percentile (median) fish consumption from flowing waters equaling 0.99 g/day, and the 95^{th} percentile equaling 12 g/day. Assuming that only the angler consumes fish and there is no sharing in the household yielded a distribution with the 50^{th} percentile (median) fish consumption from flowing waters equaling 2.5 g/day, and the 95^{th} percentile equaling 27 g/day.

1990 Mid-Hudson Angler Survey (Jackson, 1990). A survey of Hudson River anglers fishing between Stuyvesant and Kingston (within the mid-Hudson) was conducted by researchers at Cornell University in 1990 (Jackson, 1990). From May to August, 1990, they interviewed 413 individuals fishing from shore and 265 individuals fishing from boats to determine fish species preferences, the percentage of anglers that keep and eat Hudson River fish, awareness of fish advisories, and various other characteristics. Over half (57.1%) of the anglers were fishing for "anything", 28.6% were fishing for large or small mouth bass, and 9.3% were fishing for striped bass. Of those interviewed, most were male between the ages of 31 and 60 (82% male, 18% female; 8% <16 years, 10.8% 16-20 years, 29.1% 21-30 years, 44.6% 31-60 years, and 7.5% >60 years). There were significant differences between shore and boat anglers; shore anglers tended to be younger, more casual anglers (*i.e.*, fishing for anything), while boat anglers tended to be older and fishing for specific targeted species. Tournaments are popular in this stretch of the Hudson; almost three-quarters of the boat anglers were practicing for or participating in a tournament.

1998 Survey of Hudson River Striped Bass Fishery (Peterson, 1998). The recreational striped bass fishery is an important social and economic resource to residents of eastern New York state (Peterson, 1998). Based on creel surveys of boat and shore anglers on the Hudson, and interviews with more than 2,700 Hudson anglers conducted from April through June of 1997, the New York Cooperative Fish and Wildlife Research Unit at Cornell University estimated that the striped bass fishery supported more than 145,842 angler trips in 1997 (Peterson, 1998). They further estimated that 112,757 striped bass were caught, of which 14,163 (12.5%) were harvested (caught and kept). However, because striped bass are predominantly only located downstream of the Federal Dam in Troy (River Mile 154), striped bass is quantitatively evaluated later in the risk assessment for the Mid-Hudson River (Chapter 6).

3.2.1.2 Fish Ingestion Rate Distribution

Selection of the most appropriate data set for determining a distribution of fish ingestion rates for the Upper Hudson River involved consideration of a variety of factors. Ideally, site-specific fish ingestion data would be the preferred source of information. However, the objective of this baseline risk assessment is to evaluate exposures to PCBs in fish *in the absence of Hudson-specific health advisories on fish consumption*. Hudson-specific fish ingestion information can not be collected at the present time while a catch and release advisory for all fish from the Upper Hudson River remains in place. Thus, while the 1996 and 1991-1992 Hudson Angler Surveys provide useful site-specific information, they can not be used to determine fish ingestion rates for the Upper Hudson River because they were conducted while fish advisories recommended eating no fish from the Upper Hudson River; fishing was prohibited in the Upper Hudson River during the 1991-1992 survey. Therefore, the other fish ingestion studies were reviewed to determine the study most appropriate to serve as a surrogate for the Upper Hudson River.

For angler fish ingestion rates, it is important to consider a variety of factors, including the type of waterbody (marine *vs.* freshwater, flowing *vs.* still water, single waterbody *vs.* multiple waterbodies), the climate, fishing regulations, and the availability of desired fish species (reviewed in Ebert *et al.*, 1994). It is also important to consider any potential biases introduced by the survey method. All survey methods involve some uncertainties and potential biases. Long term mail survey may involve uncertainties in individuals ability to recall their behaviors over time. Diary surveys depend on individuals consistency in recording their behaviors and accuracy of record keeping may decrease with time. Connelly and Brown (1995) have reported results where mail recall estimates exceeded diary survey estimates, particularly for frequent anglers. Creel surveys (interviewing anglers "on location") have the advantage of providing data specific to active users of a resource, but are thus more likely to interview frequent anglers (Price *et al.*, 1994).

The review of available fish ingestion studies were first limited to those focusing on recreational anglers (as opposed to fish consumption of the general population that includes consumption of purchased fish) fishing on waterbodies in the Northeast. As just indicated, the two Hudson-specific studies (NYSDOH, 1999b; Barclay, 1993) cannot be used because the information was collected while advisories against consumption of all fish from the Upper Hudson River were in place. The 1990 Mid-Hudson angler survey (Jackson, 1990) and the 1998 survey of the Hudson River striped bass fishery (Peterson, 1998) focus on the lower and mid-Hudson areas and are similarly impacted by the fishing advisories, and therefore cannot be used to develop a distribution of fish ingestion rates for the Upper Hudson River (striped bass are uncommon in the Upper Hudson). The 1988 New York Angler Survey (Connelly *et al.*, 1990) did not collect information on ingestion rates of self-caught freshwater fish. The additional Connelly surveys (Connelly and Knuth, 1993; Connelly *et al.*, 1993; Connelly *et al.*, 1996) focused on fish caught in the Great Lakes, and are not the preferred source of information for developing Upper Hudson River fish ingestion rates due to differences in the types of waterbodies and the primary species present.

The two remaining studies, the 1991 New York Angler survey (Connelly *et al.*, 1992) and the 1993 Maine angler survey (Ebert *et al.*, 1993), are both comprehensive mail surveys of licensed anglers. Summary statistics for total fish ingestion rates from flowing waterbodies, as well as a distribution of ingestion rates, were presented by the study authors for the 1993 Maine angler survey. The distribution of fish ingestion rates from the Connelly *et al.* (1992) study was calculated by analyzing the raw survey data from the 1991 New York Angler survey.

The 1991 New York Angler survey was selected as the primary source of information for the Monte Carlo analysis of fish ingestion rates for Upper Hudson River anglers because the climate and characteristics of other New York waterbodies are more likely to be similar to the Upper Hudson River than Maine waterbodies. Because the Maine survey asked respondents only about total fish consumption from all flowing waterbodies, and not from individual waterbodies separately, it is not possible to screen the Maine dataset for more "Hudson-like" rivers and streams. Furthermore, in the 1991 New York survey, survey information was collected from a subset of non-respondents over the phone, allowing for correction of non-response bias. Such information was not collected in the 1993 Maine survey. As discussed in a later section, the Maine angler survey was used for the sensitivity analysis performed for this assessment.

Further reasoning for selecting the New York Angler survey (Connelly *et al.*, 1992) is the fact that it reasonably matches the demographics of the Upper Hudson angler population surveyed in an independent study. The demographic data from the 1996 Survey of Hudson River Anglers (NYSDOH, 1999b) compare favorably with the 1991 New York Angler survey. The table below compares the demographic information from the site-specific Hudson Angler Survey to the New York Angler survey.

Demographic Factor	Hudson Angler Surveys – Upper Hudson Region (NYSDOH, 2000)	New York Angler Survey (Connelly *et al*., 1992)
Age	<24yrs: 26% 25-34yrs: 25% 35-44yrs: 26% 45-59yrs: 13% >60yrs: 10%	16-25yrs: 9% 26-30yrs: 10% 31-40yrs: 29% 41-50yrs: 25% 51-65yrs: 20% >66yrs: 6%
Gender	Male: 90% Female: 10%	Male: 88% Female: 12%
Race	Caucasian: 97% African American: 2.3% Other: 0.7%	Caucasian: 94.7% African American: 2.9% Other: 2.4%
Average # in Household	3 people	N/A
Household Income	< $10K: 15% $10-$29K: 39% $30-$49K: 23% $50-$69K: 5%	<$30K: 23% $30-$45K: 24% $45-$65K: 25% >$65K: 28%

Note: Not all percentages add to 100% due to "no response" answers to particular survey questions.

While the above comparison does not provide exact metrics from which to compare the two surveys, it does indicate general similarities between the two survey populations with respect to age, gender, and race. The demographic factor differing the most between the two survey populations is household income where the percentage of the population with incomes less than $30,000 was approximately 2-fold higher for the 1991-1992 and 1996 angler population than the 1991 angler survey.

Based on the foregoing considerations, USEPA selected the 1991 New York Angler survey (Connelly *et al.*, 1992) as the primary source for the development of fish consumption rates. Survey responses reporting consumption of an unknown amount of fish were not included in the derivation of the fish ingestion rate distribution. Total ingestion rates greater than 1,000 meals of fish per year were also excluded from the resulting distribution, as such responses seem implausible, given that three meals every day would total 1,095 meals. In addition, only non-zero ingestion rates were included in the analysis (42.7% of the responses indicated they ate none of their fish).

Connelly *et al.* (1992) reported fish ingestion as number of meals of fish eaten. These data were converted to reflect fish ingestion rates in terms of g/day, assuming a meal size of one-half pound (227 grams). This assumption is consistent with the finding by Connelly *et al.* (1996) that the overall average sport-caught meal size among Lake Ontario anglers was 232 g/meal, or approximately one-half pound. A half-pound meal size is also consistent with typical assumptions about meal size made by state agencies and the GLSFATF or Great Lakes Sport Fish Advisory Task Force (Cunningham *et al.*, 1990; GLSFATF, 1993; NYSDOH, 1999b).

The responses indicating consumption of fish from flowing water bodies were used to derive the fish ingestion rate distribution; responses indicating consumption of fish from non-flowing water bodies were not included. In addition, many of the survey responses included consumption of fish from unknown water bodies. For these responses, the fish ingestion rates were scaled based on the following:

$$IR_{scaled} = IR_{Flowing} + IR_{Unknown} \times \frac{IR_{Flowing}}{IR_{Flowing} + IR_{Non-Flowing}}$$

A total of 226 responses formed the basis of the ingestion rate distribution for the 1991 New York Angler survey respondents. For the non-respondents, the type of water body was not reported. For this cohort, the ingestion rate was scaled drawing a random scaling factor, based on the equation above, from the distribution of respondent values.

Figure 3-2a provides a probability plot of the respondent results. The x-axis of this plot (z-value) is the number of standard deviations from the central value (median). The y-axis is the natural log of the ingestion rate. Data that are lognormal will fall on a straight line. The median ingestion rate for the respondents is approximately 4.4 grams per day.

The 1991 New York Angler survey ingestion rates were also corrected for non-response bias. A total of 100 of the 913 non-respondents were interviewed by telephone. Of these 100 interviews, 55 indicated they consumed at least one or more meals of their catch. Figure 3-2b provides a probability plot of the 55 non-respondent ingestion rates. The median ingestion rate for this group is approximately 3.11 grams per day.

Although both distributions appear to be approximately lognormal, they failed several statistical "goodness of fit" tests. Because the survey responses were categorical (*i.e.*, discrete number of meals eaten per year), many of the responses that clustered at the low end of the ingestion distribution (those for responses indicating a single meal per year), tended to cause the data to fail the statistical goodness of fit test. The results for respondents and non-respondents were combined and this combined distribution for the entire population was the basis for the ingestion rate probability distribution for the Monte Carlo simulation. Figure 3-2c shows the probability plot for the combined data set. The median ingestion rate for the combined data sets is 4.1 grams per day. The entire empirical dataset (281 responses) was used to generate 1,000 random samples (with replacement) for the Monte Carlo analysis (*i.e.*, a fitted lognormal distribution was not adopted). Summary statistics and percentiles for the fish ingestion rates distribution are summarized in Table 3-1.

3.2.1.3 Sensitivity/Uncertainty Analysis of Fish Ingestion Rates

As the foregoing discussion of the many surveys of fish catch and ingestion from multiple locations in the country indicates, fish ingestion rates vary among anglers, and the rates determined from independent surveys differ from one another. As a sensitivity/uncertainty analysis, the Monte Carlo simulations were conducted using the fish ingestion study results from three other surveys. Summary statistics for each of these studies are provided in Table 3-2.

The fish ingestion rates based on the 1991 New York Angler survey are consistent with the range of ingestion rates found in the fish ingestion studies that provide the foundation of the generic ingestion rates recommended by USEPA in its 1997 Exposure Factors Handbook (USEPA, 1997f). The values in the Exposure Factors Handbook are based on fish ingestion studies from several different freshwater locations within the country. The RME adult ingestion rate (31.9 g/day) is similar to the fish ingestion rate used by NYSDOH (30 g/day) in setting the fish consumption advisories for the Hudson River and the fish ingestion rate used in the Phase 1 risk assessment (30 g/day).

In the current USEPA 1997 Exposure Factors Handbook (USEPA, 1997f), the recommended fish ingestion rates for adult recreational freshwater fish consumption are 8 g/day (50th percentile) and 25 g/day (95th) percentile. These values are based on composite information from the following studies:

- 1992 Maine Angler Survey (Ebert *et al.*, 1993)
- 1992 Lake Ontario Diary Study (Connelly *et al.*, 1996)
- 1989 Michigan Sport Angler survey (West *et al.*, 1989)

As the summary in Table 3-2 indicates, the median fish ingestion value from the 1991 New York Angler study (4.0 g/day) is between the Michigan 1989 study result for recreational fish ingestion (10.9 g/day), and the 1992 Lake Ontario study value for sportfish ingestion (2.2 g/day), and the 1993 Maine Angler study value adjusted for angler consumption of self-caught fish (2.5 g/day). The 95th percentile fish ingestion rate based on the 1991 New York Angler survey (63.4 g/day) is greater than the corresponding 95th percentile ingestion rates for the three above studies. The 90th percentile from the 1991 New York Angler Survey (31.9 g/day) appears to be more consistent with the 95th percentiles of the other studies summarized in Table 3-2. Plots of the relative frequency distributions of fish ingestion for the four studies used in the sensitivity/uncertainty analysis are provided in Figures 3-3a through 3-3d. For each of the three additional studies used in this analysis, fish ingestion was modeled as a lognormal variable with distribution parameters summarized on the respective figures.[10]

The central and high-end fish ingestion rates for all flowing waterbodies from the 1993 Maine Angler Survey (Ebert *et al.*, 1993), particularly the results assuming that only the angler consumes sport-caught fish and that fish is not shared in the household, are reasonably consistent with the results for all flowing waterbodies from the 1991 New York Angler survey (Connelly *et al.*, 1992). Compared to the 1992 Lake Ontario diary study (Connelly *et al.*, 1996), the ingestion rates for sport caught fish are also reasonably consistent, although the values from the 1991 New York Angler survey are somewhat higher. This may be due to differences between Great Lakes anglers and other New York State anglers, or may reflect the fact that the 1992 Lake Ontario study was based on diary records (believed to be more accurate) while the 1991 New York Angler survey was a mail recall survey (possibly biased high due to recall bias). The difference between the two studies is greater for the 95th percentile values, consistent with the findings of Connelly and Brown (1995) that recall bias tended to result in greater overestimation of fishing activities among more frequent anglers than among less frequent anglers. The 95th percentile fish ingestion rate for flowing waterbodies from the 1991 New York Angler survey (Connelly *et al*., 1992) is somewhat higher than the 95th percentile fish ingestion rate for Lake Ontario anglers for fish from *all* sources (including sport-caught and store-bought fish). Although the above factors may be suggestive that the rates from the 1991 New York Angler survey may be overestimates, the differences could also be attributable to the different types of water bodies covered by the two surveys, and possible differences in fishing patterns among residents of the two states. The 90th percentile ingestion rate from the 1991 New York Angler survey (Connelly *et al*., 1992) was adopted as the RME IR point estimate.

Comparison to the 1996 and 1991-1992 Hudson angler surveys (NYSDOH, 1999; Barclay, 1993) is more complicated. While these studies focused on anglers fishing along the Hudson River, which is of direct interest for this risk assessment, the fact that a catch and release program is in place and current advisories recommend eating no fish from the Upper Hudson River has likely impacted fish ingestion

[10] The distribution parameters for the Connelly *et al.* (1996) and West *et al.* (1989) studies were estimated by the best-fit line through the percentiles reported in the 1997 Exposure Factors Handbook (USEPA, 1997f) fit to a lognormal distribution. The R-squared for these regressions were 0.98 and 0.96, respectively. The distribution parameters for the 1992 Maine Angler survey were reported by Ebert *et al*. (1993).

rates. Very few Upper Hudson River anglers currently eat fish from the Upper Hudson; 92% reported never eating their catch. Only 14% reported eating Hudson fish in the past; of those, 6 respondents reported eating fish once per week, 6 respondents reported eating fish one to three times per month, and 4 respondents reported eating fish less than once per month. However, it is difficult to extrapolate these values to annual average ingestion rates, due to seasonal variations in freshwater fishing. Nonetheless, despite the uncertainties in interpreting the fish ingestion data from the Hudson angler surveys, the distribution of fish ingestion rates from the 1991 New York Angler survey seems reasonable, and appears to span the range of consumption rates reported in the Hudson angler surveys.

3.2.1.4 Discussion of Additional Considerations

Licensed Versus Unlicensed Anglers. The 1991 New York Angler survey, used to generate a distribution to represent fish ingestion rates for the Upper Hudson River, was sent only to licensed anglers. Therefore, unlicensed anglers were not represented in the survey. It is somewhat uncertain how well unlicensed anglers are represented. However, given that the distribution of fish ingestion rates from the 1991 New York Angler survey spans the range of consumption rates reported in the Hudson River angler surveys, which included both licensed and unlicensed anglers (as discussed above), it seems likely that unlicensed anglers are reasonably well represented. In addition, the fish ingestion rates used compare favorably with those from other studies, as discussed above.

Highly Exposed Subpopulations. Subpopulations of highly exposed or less exposed anglers have not been explicitly characterized, but instead are assumed to be represented in the fish ingestion rate distribution. For example, the 99th percentile fish ingestion rate from the 1991 New York Angler survey is 393 meals per year, or more than one fish meal per day (Table 3-1). Furthermore, even those responses up to 1,000 meals per year were included from the 1991 New York Angler survey. Although it is possible that there are subsistence or highly exposed individuals who do not obtain fishing licenses, and therefore would not have been captured in the 1991 New York Angler survey or included in the generated distribution of ingestion rates, there are no known, distinct subpopulations that may be highly exposed (such as a Native American community) in the Upper Hudson River area.

Review of the limited literature on subsistence or highly exposed angler populations supports the assumption that these subpopulations are likely to be adequately represented in the total distribution of fish ingestion rates developed for Upper Hudson River anglers. As presented in a thesis by Wendt entitled "Low Income Families' Fish Consumption of Freshwater Fish Caught From New York State Waters," low-income families in 12 counties throughout New York, including Albany and Rensselaer counties were interviewed (Wendt, 1986). Wendt reported that between 9% and 49% of the low-income families in each county ate freshwater fish from New York State waters. Wendt then conducted a more in-depth survey of low-income families in Wayne County, New York, bordering Lake Ontario and determined fish consumption rates. The average consumption rate was 17.5 meals per year, or 10.9 g/day. In comparison, the arithmetic average consumption rate from the distribution selected to represent Upper Hudson River anglers is 27.8 meals per year, or 17.3 g/day.

As another surrogate for highly exposed angler populations, fish ingestion rate values for Mohawk women, members of a Native American community living along the St. Lawrence River where PCB fish contamination is present, who may be more dependent on local fish and game than other subpopulations, were also considered. Fitzgerald *et al.* (1995) reported the mean number of local fish meals per year consumed by Mohawk women (one year before a pregnancy) was 27.6 meals per year, which falls between the 80th and 90th percentiles of the distribution of fish ingestion rates developed for Upper Hudson River anglers.

Effect of Fish Consumption Advisories. The NYSDOH issues health advisories on eating sportfish from New York State rivers, lakes and streams. It is likely that the fish consumption advisories currently in place throughout New York State, and those in the past, have effected fish ingestion rates from the 1991 New York Angler survey. Almost half of the respondents in the 1991 New York Angler survey indicated they would eat more sport-caught fish if there were no contamination problems (Connelly *et al.*, 1992). The general state-wide advisory limits the number of sport-caught fish eaten from New York waters to no more than one meal per week (*e.g.*, NYSDOH, 1999a). For the Upper Hudson River, from Hudson Falls to the Federal Dam at Troy, there is a specific recommendation to eat no fish. Below the Federal Dam, there is a specific recommendation that women of child-bearing age and children under the age of 15 years eat no fish, and advisories recommending restrictions on quantities and species consumed for the remaining population.

However, fish advisories are not 100% effective in preventing or limiting fish consumption. Based on an analysis of the raw survey data from the 1991 New York Angler survey (Connelly *et al.*, 1992), there was no significant difference in the mean number of freshwater fish meals eaten when comparing New York waterbodies with full, partial, or no advisories, despite the expectation that the fishing advisories would likely suppress fish ingestion rates to some degree.

To characterize fish ingestion rates that have not been influenced by the Hudson-specific health advisories to eat no fish, this baseline risk assessment uses fish ingestion rates from the 1991 New York Angler survey (Connelly *et al.*, 1992) for all flowing waterbodies without specific fish consumption advisories. The effect of general, non-specific NYSDEC and NYSDOH fishing regulations that would be in effect regardless of PCB contamination levels in the Upper Hudson River inherently will be taken into account because these regulations also apply to the New York flowing waterbodies surveyed in the 1991 New York Angler survey.

Women and Children Anglers. Although young children and adolescents under age 15 are not required to have fishing licenses in New York State, several sources indicate that many children consume sport-caught freshwater fish (Connelly *et al.*, 1990; Connelly *et al.*, 1992; Wendt, 1986). However, ingestion rates of freshwater fish specific for children are not available. The 1991 New York Angler survey provides data on the age at which anglers begin fishing, and this information has been incorporated into the exposure duration modeling to generate both the length of exposure and also the age at which exposure begins. For each modeled angler whose exposure begins during childhood (as shown in Figure 3-4c, approximately 16% of the anglers in the 1991 New York Angler survey were 10 years old, or younger), the same distribution of number of meals per year generated for adult anglers was used, simply scaled according to body weight, on a year by year basis. Thus, children are represented in this risk assessment to the same extent that they are represented in the New York angler populations. Similarly, although fewer women tend to fish than men, women anglers are represented in this risk assessment to the same extent that they are represented in the 1991 New York angler populations.

Recall Bias. The 1991 New York Angler survey (Connelly *et al.*, 1992), as well as the other mail recall surveys, may be subject to recall bias. It is difficult for many individuals to remember accurately their activities over an entire year. When asked about recreation participation over a long period of time (*i.e.*, one year), respondents tend to overestimate their activities (reviewed in Connelly and Brown, 1995; Westat, 1989). With respect to fishing specifically, Connelly and Brown (1995) found that anglers reported significantly higher rates of fish consumption and numbers of days fished in 12-month mail recall surveys compared to 12-month diary studies. The difference was greater for anglers who fished more frequently than those who fished less frequently. These results suggest that the data from the 1991 New York Angler survey (Connelly *et al.*, 1992), used to generate the distribution of fish ingestion rates

used in the base case analysis in this risk assessment, are more likely not likely to underestimate actual ingestion rates, particularly for more frequent anglers.

Single Versus Multiple Waterbodies. By deriving the distribution of fish ingestion rates from the data for all flowing waterbodies from the 1991 New York Angler survey, it was conservatively assumed that the amount of fish an individual would consume from the Upper Hudson River, a single waterbody, is equal to the amount of fish consumed by New York anglers from all flowing waterbodies. Although this assumption may overestimate fish ingestion rates for anglers who fish in multiple water bodies (including the Upper Hudson River), many of the respondents in the 1991 New York survey fished in only one or two locations; 35.5% fished in only one location and 21% fished in only two (Connelly *et al.*, 1992). For anglers who fish only the Upper Hudson River, the ingestion rate distribution used would not necessarily overestimate their fish consumption rate.

3.2.2 PCB Concentration in Fish

As described earlier in Section 2.3.1, there are several important environmental factors that affect the determination of the EPC in fish ($C_{f,y}$) and therefore influence the variability of PCB intake *via* fish ingestion:

1. The concentration of PCBs in any particular species varies for a particular year, but overall it declines over time.

2. The concentration of PCBs within the same fish species varies depending on the location in the Upper Hudson River (higher concentrations upstream than downstream within the same fish species)

3. The PCB concentration varies among different fish species.

Within Species Annual Variability ($C_{f,y}$)

As was discussed in Section 2.3.1., the variability of model-predicted 50th (median) and 95th percentile PCB concentration within fish for any particular year varies by approximately a factor of 2- to 3-fold. It is unknown to what degree the modeled range represents true variability that is expected among fish of the same species, and to what extent the modeled range is a function of model uncertainty. Regardless of the contribution these two factors may represent, the modest range between the 50th and 95th percentile predictions is not anticipated to yield large differences in the mean PCB concentration in fish that are ingested. This conclusion is supported by an examination of the historical sampling results as well.

Based on the historical monitoring data summarized in the Phase 1 Report (Tables B.3-16 through B.3-18), the coefficient of variation (CV), which is the ratio of the standard deviation divided by the mean, of the measured PCB content in brown bullhead and largemouth bass is generally less than 1.0, and typically around 0.7. Compared to this, the upstream to downstream difference in PCB concentration within a given fish species and year is on the order of 2 to 3-fold. Thus, for an angler who consumes a large amount of fish (*i.e.*, someone at greatest risk), the within-species coefficient of variation is typically less than the variation in concentration attributable to fishing either up- or downstream (*i.e.*, fishing location component of variability). Furthermore, the difference in PCB concentration across fish species is also on the order of 2-fold, again greater than the within species coefficient of variation. Thus, even if the within-species annual variability of PCB concentration in fish were included quantitatively in the

Monte Carlo analysis, it would likely be overshadowed by the larger variability in concentration across locations and species.

For the above reasons, the within species PCB concentration for any particular year ($C_{f,y}$) was set to the mean modeled concentration for that species and year for the intake calculated using Equation [3-1]. The variability (randomness) of PCB ingestion from fish was modeled based on the variability in the species consumed, which is accounted for by the PCT_f term in Equation [3-1].

Variability of Species Ingested (PCT_f)

As described in Section 2.3.1, the fish species consumption patterns for the point estimate exposure calculations were based on a weighted average of the species consumed. The species consumption weights were based on the 1991 New York Angler surveys (Connelly *et al.*, 1992) which provided information on the fish species caught and consumed by the surveyed anglers.

For the Monte Carlo analysis, the survey responses from all respondents were used to develop a distribution of fish species ingestion patterns. The same criteria applied to fish ingestion, only those angler responses indicating consumption of at least one and fewer than 1,000 meals from flowing water bodies only, were used to derive the species ingestion distribution. This survey group consists of 226 respondents.

A summary of the species ingestion responses for these respondents is presented in Table 3-3. As described earlier in Section 2.3.1, these species were grouped such that only those responses indicating consumption of fish potentially inhabiting the Upper Hudson River were used. These responses were grouped such that each of the three modeled species provided a surrogate for the concentration of any fish within the group.

The fish species reported consumed by the 226 respondents were grouped into one of three groups according to the groupings given in Table 3-4. For the Monte Carlo analysis, random samples (with replacement) were drawn from this empirical distribution of 226 respondents. This distribution ranges from respondents indicating consumption of a single species, to respondents indicating consumption of multiple species.

3.2.3 Cooking Loss

Numerous studies have documented a loss of PCBs from fish due to cooking (Armbruster *et al.*, 1987; Armbruster *et al.*, 1989; Moya *et al.*, 1998; Puffer and Gossett, 1983; Salama *et al.*, 1998; Schecter *et al.*, 1998; Sherer and Price, 1993; Skea *et al.*, 1979; Smith *et al.*, 1973; Wilson *et al.,* 1998; Zabik *et al.*, 1979; Zabik *et al.*, 1995a; Zabik *et al.*, 1995b; Zabik *et al.*, 1996; Zabik and Zabik, 1996). These studies were reviewed to determine if the extent of PCB losses during cooking have been adequately characterized in the scientific literature to support a quantitative estimate of cooking losses for risk assessment purposes.

A summary of the cooking loss estimates for each of these studies is provided in Table 3-4. As this table shows, experimental results range considerably, both between various cooking methods and within the same method. Most PCB losses (expressed as percent loss based on Total PCB mass before and after cooking) were between 10 and 40 percent. Losses as high as 74 percent were reported in one study (Skea *et al.,* 1979). Net gains of PCBs were reported in several studies (Moya *et al.*, 1998;

Armbruster *et al.*, 1987).[11] Overall, these studies support the conclusion that some PCBs are lost during cooking. Consistent with this conclusion, both the NYSDOH and the GLSFATF recommend proper methods of trimming, skinning, and cooking fish to remove fat and reduce levels of PCBs and other contaminants (NYSDOH, 1999a; GLSFATF, 1993).

The extent of PCB cooking losses has not been well characterized in the published literature, and quantitative estimates of cooking losses remain uncertain. There were no consistent differences in PCB losses between cooking methods in the studies reviewed. Although losses from baking were greater than losses from pan-frying in two studies where the same fish type was used for both cooking methods (Armbruster *et al.*, 1987; Salama *et al.*, 1998), the study by Salama *et al.* (1998) only used one fish per cooking method, and is therefore of limited significance. It is difficult to make comparisons between different fish types, as different preparation and cooking methods were often used for different fish types. With regards to preparation technique, while data from Zabik *et al.* (1979) and Salama *et al.* (1998) showed greater losses of PCBs from fish cooked with the skin off as compared to skin on, Zabik *et al.* (1995a) observed minimal differences in PCB losses between fish with skin on or skin off.

Based on the available data, it is not possible to quantify the importance of specific factors influencing the extent of PCB cooking losses. PCB losses from cooking may be a function of the cooking method (*i.e.*, baking, frying, broiling, *etc.*), the cooking duration, the temperature during cooking, preparation techniques (*i.e.*, trimmed *vs.* untrimmed, with or without skin), the lipid content of the fish, the fish species, the magnitude of the PCB contamination in the raw fish the reporting method, and/or the experimental study design. The extent of reduction of PCBs due to cooking may also depend on the PCB homologues present in the fish. Zabik *et al.* (1994), as cited in Zabik and Zabik (1996), found that cooking losses of pentachloro-, hexachloro- and heptachlorobiphenyls are greater than losses for PCB homologues with either more or fewer chlorines. Differences among the techniques used for extracting and measuring PCBs are another factor that could contribute to the observed differences in cooking loss between studies.

The wide variation in PCB losses observed, both between and within studies, the lack of an association with various factors which could affect PCB losses, and the fact that personal preferences for various preparation and cooking methods and other related habits (such as consuming pan drippings which may serve to reintroduce PCBs lost during cooking into the fish meal) are poorly defined, highlights that there are many uncertainties associated with estimating losses of PCBs from fish. It is not possible to develop a probability distribution representing the variability of cooking loss expected either among different consumers, or due to different preparation methods. Thus, for the Monte Carlo analysis, cooking loss was held constant. However, for the sensitivity, or parameter uncertainty analysis, the following range of cooking loss were examined:

RME Exposure:	0%
CT estimate:	20%
Low-end exposure estimate:	40%

Although it is possible that PCBs volatilized during cooking could be inhaled, in the absence of any scientific studies in this area, it is not possible to quantify the potential cancer risks and non-cancer health hazards from this pathway. Based on a qualitative assessment of the cooking frequency for fish, the temperatures used in the cooking, the various cooking practices used, and the relatively lower PCB toxicity *via* inhalation compared to ingestion, the cancer risks and non-cancer health hazards from inhalation while cooking are unlikely to be significant compared to the ingestion of fish.

[11] It is likely that the net gain is within the experimental measurement error and essentially indicates zero loss.

3.2.4 Exposure Duration

While Superfund risk assessments typically use the length of time that an individual remains in a single residence as an ED, such an estimate may not be a good predictor of angling duration for the Upper Hudson River, because an individual may move into a nearby residence and continue to fish in the same location, or an individual may chose to stop angling irrespective of the location of their home.

For the fish consumption pathway, ED is defined as the number of years, starting in 1999, that an individual consumes fish from the Upper Hudson River. The angler population has been defined as those individuals who consume self-caught fish from the Hudson River at least once per year, in the absence of a fishing ban or fish consumption advisories. Although the population of anglers who fish from the Upper Hudson River is likely to include individuals from a large geographic area, it was assumed that individuals residing in any of the five counties surrounding the Upper Hudson River would be the most frequent anglers (recall the 1988 New York Angler, Connelly *et al*., (1990), survey reports the mean distance traveled by anglers fishing in the Hudson was 34 miles). For members of this population of anglers, exposure is assumed to continue until one of the following occurs:

- The individual stops fishing;
- The individual moves out of the area; or dies.

Information regarding the age distribution of New York anglers, including the number of years fished, and when anglers began fishing, was obtained from the 1991 New York Angler survey (Connelly *et al.*, 1992). The probability of moving into and out of any of the five counties surrounding the Upper Hudson River was derived from data on county-to-county mobility (U.S. Census, 1990a).

As described in the following subsections, determining the distribution of ED for the angler population involves the following computational steps:

1. *Section 3.2.4.1*. The individual's current age and age at which he or she began fishing is randomly drawn from a distribution developed from information contained in the 1991 New York Angler survey conducted by Connelly *et al.* (1992).

2. *Section 3.2.4.2*. The time remaining until an individual stops fishing, which is a function of current age and the age at which the individual started fishing, is derived from the 1991 New York Angler survey data (Connelly *et al*., 1992).

3. *Section 3.2.4.3*. The time remaining until that individual moves out of the Upper Hudson counties (one of the five counties comprising the Upper Hudson region) is drawn from a distribution developed from the 1990 U.S. Census In-Migration data tape (U.S. Census, 1990a). This distribution describes the time until an individual moves out of the region as a function of current age.

As was discussed earlier in Section 2.4.1, the 50th percentile exposure duration was determined to be 12 years, and the 95th percentile exposure duration is 40 years. The derivation of the distribution is described below.

3.2.4.1 Joint Distribution for Current Age and Fishing Start Age

The joint distribution for current age and the age at which individuals started fishing (the "fishing start age") were characterized from the 1991 New York Angler survey (Connelly *et al*., 1992). For each of the 1,030 survey respondents, the survey lists the current age and the age at which the respondent started fishing. In addition to the 1,030 respondents, there were also 913 non-respondents, of whom 100 were surveyed by telephone. However, the follow-up survey of the non-respondents did not record the age at which these individuals started fishing.

From the 1991 New York Angler survey, the probability that a randomly selected angler started fishing at age *s* and is currently age *c* is denoted *P(s,c)* can be computed as:

$$P(s,c) = \frac{N(s,c)}{\sum_{i,j} N(i,j)} \qquad [3\text{-}2]$$

where

P(s,c) = probability of starting fishing at age *s* for individual who is currently age *c*
N(s,c) = number of survey individuals who started fishing at age *s* and are now age *c*

The summation in the denominator of Equation [3-2] is simply the summation over all the anglers in the survey. Before conducting these calculations, two adjustments were made to the data, as described below.

Adjustment 1: Data Sparseness. The data were aggregated into 10-year age groups because the value of *N(s,c)* was often small or 0 for some age groups, thus compromising the robustness of the calculated value, *P(s,c)*. Thus, both *s* and *c* were rounded to the nearest value of 10. This aggregation puts a lower limit of 10 years on the age at which individuals start fishing, and hence a lower limit on the age at which exposure may begin. If children younger that 10 years old fish or consume fish caught by others, this aggregation will underestimate childhood exposure.

Adjustment 2: Connelly follow-up survey of non-respondents. The Connelly *et al.* (1992) respondent data (N = 1,030) were adjusted to reflect the non-respondent data (N = 913). As noted in Section 3.2.1.1, Connelly *et al.* (1992) resurveyed 100 of the non-respondents and reports the ages of these individuals. However, the non-respondent survey results do not report the age at which non-respondents started fishing. In order to include the non-respondent information in Equation [3-2], the results for the 1,030 initial respondents were therefore adjusted by multiplying *N(s,c)* in Equation [3-2] by an scaling factor (k_c) computed as:

$$k_c = \frac{\frac{913}{100} \times NR(c) + \sum_{s \in all\ start\ ages} N(s,c)}{\sum_{s \in all\ start\ ages} N(s,c)} \qquad [3\text{-}3]$$

where *NR(c)* is the number of resurveyed non-respondents who report their current age to be *c*. This adjustment is based upon the following assumptions:

- The current age of the entire non-respondent group (913) mirrors the current age of the 100 surveyed non-respondents; the factor 913/100 is simply a weighting factor that conveys this adjustment.

- The distribution of the current age for the non-response group is similar to the distribution of current age for the survey respondents.

Discussion of Assumptions

There are several basic assumptions made in deriving the joint distribution for current age and fishing start age, which are summarized here.

- The angler population is a steady state population, meaning that the age profile of this population remains unchanged over time.

- A corollary to the steady state assumption is that the 1991 New York Angler survey is representative of anglers in 1999.

- Information about the 913 non-respondent group can be inferred from the information gathered from 100 non-respondents who were contacted by Connelly *et al.* (1992).

- Connelly *et al.* (1992) report the current age for the non-respondents, but not the age at which they started fishing. Therefore, the results from the respondents were stratified by current age as a surrogate. The validity of this approach rests on the assumption that the response rate depends statistically on current age but not the age at which an individual starts fishing.

- Although the 1991 New York Angler survey (Connelly *et al.*, 1992) provided information about the reported age at which each angler started fishing, the analysis required grouping the starting age into 10-year age groups. Thus, all starting ages between 5 and 15 years were categorized in the "10 year" age group. This aggregation required an assumption that no one began fishing before 5 years of age when, in fact, 2.9% of the respondents reported starting fishing before age 5.

The 1991 New York Angler (Connelly *et al.*, 1992) survey results suggest that the assumption of the age profile of the angler population remaining constant over time is not strictly true, even after adjustment to reflect the data from resurveyed non-respondents. Specifically, it appears that the survey under-counted the number of young anglers (age 10). The constructed distribution was adjusted, although it is not clear if the adjustment is sufficient to represent all young anglers. However, there are no studies that have evaluated fishing populations over time. The cross-sectional design of the Connelly *et al.* (1992) study provides a representative indication of fishing activities in the future and is believed to be a reasonable use of available data.

Upper Hudson River Angler Populations Considered

For the purpose of characterizing current and future cancer risks and non-cancer health hazards, the starting year for the exposure calculations is 1999. Two populations of anglers were considered in the exposure analysis, because it was unclear *a priori* which group might have a longer possible exposure duration. The two groups considered were:

- *The population of all anglers currently living in the five counties of the Upper Hudson region.* For this population, all data from the 1991 New York Angler survey were used to calculate the joint distribution for current age and fishing start age.

- *The population of anglers living in the five counties who started fishing in 1999*: Analysis of the 1991 New York Angler survey data was restricted to individuals who "recently" started fishing. Ideally, these data would include only those anglers whose start age and current age are exactly the same (*i.e.*, individuals who started fishing within the last year). However, restricting the analysis to these individuals resulted in too small a data set. All anglers whose *rounded* fishing start age and current age were the same were used for this analysis.

Evaluation of the data for both possible population groups showed that the exposure duration distributions for these two groups did not differ appreciably. Therefore, the Monte Carlo analysis was based upon the "all angler" category, which is the larger set of the 1991 New York Angler survey respondents.

3.2.4.2 Time Remaining Until an Individual Stops Fishing

The time remaining until an individual stops fishing was also based upon the 1991 New York Angler survey (Connelly *et al.*, 1992). Because time until an individual stops fishing was not directly available from the Connelly *et al.* (1992) survey, it was estimated using the start age and current age of the respondents. The probability that an individual whose start age is s and whose current age is $c \geq s$ stops fishing within the next T years, designated $F(s,c,T)$, is

$$F(s,c,T) = \frac{N(s,c) - N(s,c+T)}{N(s,c)} \qquad [3\text{-}4]$$

where as defined in the previous section, $N(s,c)$ is the number of individuals in the survey who started fishing at age s and are now age c.

The reasoning underlying Equation [3-4] is that $N(s,c)$ is the number of individuals in a cohort that started fishing at age s and who are now age c, and $N(s,c+T)$ is the number of individuals remaining in this cohort T years in the future. Since the number of individuals who will remain in this cohort T years in the future is unknown, the number of individuals who started fishing at age s and who are currently $c+T$ years of age serves as a surrogate. This approach presumes that the angler population is in a "steady state," meaning that $N(s,c)$ remains unchanged over time for all values of s and c. From this assumption, it also follows that:

- $F(s,c,T)$ must remain unchanged over time; and

- $N(s,c) \geq N(s,c,T)$ for all positive values of T.

Before making these calculations, three adjustments were made to the data. The first two, to address data sparseness and to incorporate the Connelly *et al*. (1992) follow-up survey of non-respondents, are identical to the adjustments described in Section 3.2.4.1. A third adjustment was made to preserve the assumption of steady state. It turns out that even after adjustment of the Connelly *et al.* (1992) data to reflect non-respondents, the condition $N(s,c) \geq N(s,c,T)$, which follows from the steady state assumption,

failed to hold true in some cases. There are several possible reasons for this phenomenon, among which are:

- The steady state assumption is not strictly true, and the number of individuals that started fishing at age *s*, *T+c* years ago exceeds the number of individuals who started fishing *c* years ago at age *s*;
- The Connelly *et al*. (1992) survey, even after adjustment for non-respondents, still under counts the number of individuals in some age groups.
- The condition may fail due to the sparseness of data for some age groups (*e.g.*, it could be an artifact of sample size and the necessity to aggregate data).

Although the steady state assumption may not strictly hold, it is believed to be a reasonable approximation. To adjust the survey data so that they are consistent with the steady state assumption (and in order to make it possible to calculate valid values for $F(s,c,T)$), the adjusted counts of survey respondents ($N_{Adj}(s,c)$) were set equal to the maximum of N(s,c) and N(s,c+10). In cases where this adjustment was necessary, the resulting estimate of $F(s,c,10)$ is 0.

The above adjustment may err on the side of understating the probability that an individual will stop fishing within some time period since the value of $N_{Adj}(s,c)$ may exceed $N(s,c+10)$. On the other hand, in cases where the survey under-reported $N(s,c,T)$ for some relatively small value of *T*, these calculations will overstate the probability that individuals who started fishing at age *s* and whose current age is *c* will soon stop fishing.

Summary of Fishing Cessation Probability

A frequency histogram of fishing cessation probability is shown in Figure 3-4a. This figure indicates the relative frequency of those anglers who will stop fishing in the given number of years. Thus, approximately 24% of the angler population is estimated to cease fishing in 10 years, approximately 23% in 20 years, 20% in 30 years, *etc.* Approximately 1% are estimated to cease fishing in 70 years.

Figures 3-4a, 3-4b, 3-4c, and 3-4d summarize the fishing cessation age, starting age, current age, and total fishing duration frequency histograms for the angler population. Note that P(s,c) and F(s,c,T) represent conditional probability functions, and cannot be represented with a single histogram.

3.2.4.3 Determination of Residence Duration

The second determinant of total exposure duration is the residence duration in any of the five counties surrounding the Upper Hudson, which are Albany, Rensselaer, Saratoga, Warren, and Washington. When an individual moves out of these five counties, regular fishing in the Upper Hudson River is assumed to stop.

The distribution for the time remaining until an individual moves out of the Upper Hudson Region is given by estimating the one-year probability that an individual moves out of the region, and then combining these one-year probabilities to calculate the likelihood that an individual will move out of the area over a more extended time period. Specifically, designate $p_{k,n}$ to be the probability that an individual who is now age *k* moves out of the area in exactly *n* years. Then $p_{k,n}$ can be computed from the 1-year move probabilities as

$$p_{k,n} = \left[\prod_{i=1}^{n-1} (1 - p_{k+i-1,1}) \right] \times p_{k+n,1} \qquad [3\text{-}5]$$

where the product (indicated by the Π symbol) is taken over a series of terms indexed by the subscript *i*. Note that the product within the brackets is the probability that the individual does not move outside the region during the next *n-1* years, while the term following the brackets is the probability that the individual moves in year *n*. Finally, the 1-year probability, $p_{k,1}$, is computed as the number of individuals age *k* who move out of the region in a single year divided by the number of individuals age *k* who lived in the region at the beginning of the year.

Data from the 1990 In-Migration portion of the County-to-County Migration Files published by the U.S. Census Bureau (1990a) were used to compute the 1-year move probabilities. For each of a series of age groups (ages 5-9, 10-14, 15-19, 20-24, 25-29, 30-34, 35-44, 45-54, 55-64, 65-74, 75-84, and 85+), those files quantify the number of current (1990) residents in every U.S. county who have resided in that county during the preceding five year period (1985 to 1989), and the number of current residents who moved into the county during the preceding five year period. For the latter group, the data quantify how many residents came from each outside county.

In order to estimate the probability of moving into or out of the counties bordering the Upper Hudson, the following census information was used:

1. The number of individuals in 1990 who had resided within the five counties since 1985;

2. The number of individuals in 1990 who had moved to their current residence from one of the other four counties bordering the Upper Hudson; and

3. The number of individuals in 1990 who had moved to their current residence from a county outside the Upper Hudson counties.

The sum of the first and second categories is the number of individuals in 1990 who had been living within that region during the preceding five years.

If the age categories divide the population into five year increments, then it is by definition true that

$$Start_{1985\text{-}90,k} + In_{1985\text{-}90,k} - Out_{1985\text{-}90,k} = End_{1985\text{-}90,k+1} \qquad [3\text{-}5]$$

where

$End_{1985\text{-}90,k+1}$ = Number of individuals in age category *k+1* at the end of the 1985 to 1990 period.

$Start_{1985\text{-}90,k}$ = Number of individuals in age category *k* who lived in the region at the beginning of the 1985-1990 period.

$In_{1985\text{-}90,k}$ = Number of individuals in age category *k* who moved into the region during the past 5 years.

$Out_{1985\text{-}90,k}$ = Number of individuals in age category k who moved out of the region during the past 5 years.

The In-Migration files do not report the value of $End_{1985\text{-}90,k}$. However, under the assumption that the populations in the Upper Hudson counties are in steady state, the number of individuals in age category k at the beginning of the 1985 time period is equal to the number of individuals in the same age category at the end of that time period. Hence, $End_{1985\text{-}90,k+1}$ is assumed to equal $Start_{1985\text{-}90,k+1}$, and Equation [3-5] can be rewritten,

$$Start_{1985\text{-}90,k} + In_{1985\text{-}90,k} - Out_{1985\text{-}90,k} = Start_{1985\text{-}90,k+1} \quad [3\text{-}6]$$

From Equation [3-6], the value of $Out_{1985\text{-}90,k}$ can be calculated as,

$$Out_{1985\text{-}90,k} = (Start_{1985\text{-}90,k} - Start_{1985\text{-}90,k+1}) + In_{1985\text{-}90,k} \quad [3\text{-}7]$$

Finally, the probability that an individual in age category k moves out of the region during a five-year period, denoted $p(k)$, is computed as:

$$p(k) = \frac{Out_{1985-90,k}}{Start_{1985-90,k} + In_{1985-90,k}}$$

Two computational issues must be noted. First, 1-year move probabilities cannot be directly computed using the In-Migration data because the data reflect mobility over a five-year time period. The number of individuals moving out of an area in a single year were assumed to equal the number who move out over a five-year time period divided by five. The 1-year move probabilities were applied to all ages within category k. Second, because the age categories for ages 35 or above are reported in 10-year increments, while those for ages 34 and below are reported in five-year increments, one-half the value reported for $Start_{1985\text{-}90,35\text{-}44}$ was used in the computation of $Out_{1985\text{-}90,30\text{-}34}$.

Tables 3-8 through 3-12 detail the In-Migration data for each of these five counties separately, and Table 3-13 summarizes the counts summed over these five counties. Table 3-14 lists the values used to compute the 1-year move probabilities, and Table 3-15 provides an overall summary of the move probabilities. Figure 3-5a provides a frequency histogram of the residence duration. The overall frequency distribution for total exposure duration (the combination of fishing duration probability and residence duration probability) is shown in Figure 3-5b.

Assumptions for Residence Duration Estimates

Two basic assumptions were made here in order to estimate the probability distribution of residence duration (and likelihood of moving out of the five counties):

- The population's age distribution was assumed to be at steady-state and not change over time.

- The probability that an individual moves was assumed to depend only on his or her current age and not on the length of time he or she has already lived in the area. If the conditional probability of moving out of the area is lower for individuals who have already lived in the area for a long period of time, it is possible that the approach adopted will underestimate the fraction of the population whose residence times are very long.

It is of course likely that the population is not strictly at steady state. However, an adjustment for non-steady state conditions is not apparent, because it would require projecting future trends with historical data. Forecasting future trends was deemed to be a greater source of uncertainty than an assumption of steady state.

The exposure duration distribution ranges from 10 years to 60 years, with a 50th percentile value of 12 years, and a 95th percentile value of 40 years. For comparison, current USEPA recommendations for the exposure duration parameter for Superfund risk assessments are 9 years (median) and 30 years (high-end) based on population mobility statistics for the general public (USEPA, 1991b). There are uncertainties inherent in the derivation of the exposure duration, which may over- or under-estimate cancer risks and non-cancer health hazards. However, the values, which are based on county-specific data, are reasonable when compared to national mobility statistics, and also cover the possibility of extended exposure from consuming fish, as long as 60 years (*i.e.*, from age 10 to 70), as reported by anglers in the state-wide 1991 New York Angler survey (Connelly *et al.,* 1992).

3.2.5 Body Weight

The probability distribution of the variation of BW within the population was drawn from published studies of adult and child/adolescent body weights. Brainard and Burmaster (1992) report that the BW distributions for males between the ages 18 and 74 years and for females between the ages of 18 and 74 are lognormal. The Brainard and Burmaster (1992) results and the calculated lognormal distribution summary statistics appear in Table 3-16.

Finley *et al.*(1994) report the arithmetic means ($\bar{x}$) and arithmetic standard deviations (s_x) of the BW distributions for individuals aged 1 to 18 years, and for all individuals greater than 18 years of age. Because the authors do not specify the form of these distributions, they are assumed to be lognormal based on the lognormality of the adult body weights found by Brainard and Burmaster (1992). Assuming a lognormal distribution of BW, the geometric mean (GM) and geometric standard deviation (GSD) can be calculated from their arithmetic counterparts by,

$$GM = \exp\left(\ln \bar{x} - GSD^2/2\right)$$

$$GSD = \exp\left(\sqrt{\ln\left(1 + s_x^2/\bar{x}^2\right)}\right)$$

Because body weights can be measured very accurately and the distribution of body weights in the population has been extensively studied and well characterized (*e.g.,* Finley *et al.,* 1994 and Brainard and Burmaster, 1992), the uncertainty associated with this parameter's estimate is likely to be negligible. No sensitivity analysis was deemed necessary for this parameter.

It was assumed that for each individual in the population, BW is perfectly correlated over time. That is, individuals whose BW is high at one age will have a high BW at other ages, while those with a low body weight at one age will have a low body weight at other ages. To implement this temporal correlation, each simulated individual was assigned a weight distribution percentile, and this body weight percentile was assigned to the simulated individual throughout the ED. For example, the individual who has the median population BW at age 1 was assigned the median population BW during the remainder of his or her simulated lifetime, ensuring that individual body weights in the population are correlated over time.

3.3 Summary of Simulation Calculations

The Monte Carlo exposure calculation sequence is shown in Figure 3-1. Each simulation consisted of 10,000 samples, where each sample represents a simulated angler. A summary of the base case and sensitivity analysis distribution inputs is provided in Section 3.3.1. Section 3.3.2 summarizes the numerical stability of the Monte Carlo calculations. The risk estimates that correspond to the Monte Carlo exposure analysis are presented in Chapter 5, following the discussion of PCB toxicity factors in Chapter 4.

3.3.1 Input Distributions Base Case and Sensitivity Analysis

As described above, the Monte Carlo exposure analysis was conducted to examine the RME for the fish ingestion pathway. The probability distributions derived for this analysis are aimed at determining the variability of exposure among the angler population. Throughout the derivation of the input distributions, a recognition of the uncertainty involved in estimating the distributions has been presented. Because insufficient information is available to characterize the uncertainty by means of a fully 2-D Monte Carlo analysis, a sensitivity/uncertainty analysis was performed as an alternative means to address the approximate precision of the analysis.

The sensitivity analysis involved repeating the Monte Carlo analysis for separate input distributions for each of the variable parameters. The 72 combinations evaluated included the following:

Parameter*	***Base Case***	***Sensitivity Analysis***
Fish Ingestion (4)	1991 New York Angler Survey Empirical Ingestion Distribution	1992 Maine Angler (Ebert *et al.*, 1993) 1989 Michigan (West *et al.*, 1989) 1992 Lake Ontario (Connelly *et al*, 1996)
Exposure Duration (2)	Minimum of Fishing Duration and Residence Duration	Residence Duration only
Fishing Location (3)	Average of 3 Modeled Locations	Thompson Island Pool Waterford/Federal Dam
Cooking Loss (3) (no variability modeled)	20% (midpoint of typical range)	0% (high-end exposure) 40% (low-end exposure)

**Numbers in parentheses indicate number of combinations*

In the table below, parameter values for all the exposure factors are listed to provide insight on the degrees of variability and uncertainty that each contributes to the overall PCB exposure estimates. Because each of these factors vary independently, their combined effect can only be accurately assessed by examining the results of the Monte Carlo analysis (Section 5.2). A brief discussion of each factor follows.

Fish Ingestion Rate. The fish ingestion rate for the point estimate calculations was selected from the 1991 New York Angler survey (Connelly *et al*., 1992). There is an approximate 2-fold range, above and below the mean value from the 1991 New York Angler survey, for the additional studies used to examine uncertainty of this parameter. The high end estimates are approximately 4-fold to 10-fold higher than their respective mean values. As this comparison indicates, the combined variability and uncertainty of fish ingestion rates can affect cancer risk and non-cancer health hazard calculations by at least an order of magnitude.

Exposure Point Concentrations (EPC). The variability of the EPCs was evaluated based on (1) differences in concentration at the three different locations within the Upper Hudson River and (2) differences in concentration among the fish species consumed by anglers (species bioaccumulate PCBs in different amounts). In the matrix below, the variation in EPC values is shown as a function of location (*e.g.*, the values presented are averaged at each location over the three fish species modeled). As this comparison shows, the range in EPC values as a function of the location of catch on the Upper Hudson varies by less than 10-fold over the 12-year averaging period, and varies by approximately three-fold for longer averaging periods (*i.e.*, 40 years). Although not shown in this comparison, the variation as a function of different fish species is less than the variation in concentration at upstream *versus* downstream locations (see Figure 2-4).

As discussed earlier, the multiple sources of possible model uncertainty precluded a quantitative analysis of the uncertainty of the forecast PCB values in fish. The FISHRAND model in general was able to match lipid-based measured PCB concentrations within a factor of approximately 2-fold (Appendix A, see also, USEPA, 2000a). Despite the reasonable calibration to measured data, the forecasts also rely upon a combination of PCB forecasts in the water column and surface sediments and upstream continuing PCB loads. Thus, while the uncertainty of modeling PCB body burdens in fish may be modest, the combined effect of forecasting many variables into the future appears to yield PCB concentrations in fish that are more likely to underestimate, rather than over-estimate, future trends in fish (see Figures 2-7 through 2-9).

Fraction of Fish from Upper Hudson. This exposure factor was held constant at 100%, reflecting the definition of the potentially exposed population as those anglers that catch and consume fish at least once per year. While this definition may limit the size of the population, over 10% of the anglers surveyed in the Hudson Angler surveys (NYSDOH, 1999b; Barclay *et al*., 1993) indicated they consumed their Hudson River catch. Additional discussion of this factor is provided in Section 5.3.1.

Cooking Loss. This factor was treated as uncertain, with three values selected for the Monte Carlo analysis: 0%, 20% and 40%. As discussed earlier, the differences in cooking methods, type of fish, and many other factors precluded defining the variability in this factor as a function of either fish species or preparation methods. Thus, cooking loss was treated as a fixed (no variability) but uncertain parameter in the Monte Carlo analysis.

Exposure Duration. Both variability and uncertainty were considered for this parameter. The variability was defined by an empirical probability distribution. For the base case analysis, the empirical distribution was defined as the conditional probability distribution of fishing duration and residence duration in the five Upper Hudson Counties. The fishing duration distribution was drawn from the 1991 New York Angler Survey data (Connelly *et al*., 1992). The residence duration distribution was derived from the 1990 census data for the five counties surrounding the Upper Hudson River (U.S. Census Bureau, 1990a). As the 50^{th} and 95^{th} percentile values from these respective empirical distributions shown below indicate, there was little difference in the exposure duration distribution for these two cases. It is likely that the uncertainty in this exposure factor is larger than this analysis would suggest. However, as discussed earlier, it is difficult to quantify the uncertainty.

Body Weight. As described in Section 2.2, chemical intake is converted to a dose, which is simply the intake expressed as a function of body weight. Body weight clearly varies within the angler population, as it does in any population. The uncertainty in the body weight exposure factor was considered minor compared to the sources of uncertainty considered for other exposure factors, thus no

uncertainty in its probability distribution was modeled. The 50th and 95th percentile values from its lognormal distribution for adults are shown in the matrix below.

Summary of Monte Carlo Exposure Parameter Uncertainty and Variability Analysis			
Exposure Parameter*	***Mean***	***High End***	***Comment or Source of Information for Variability/Uncertainty Analysis***
Fish Ingestion Rate[a] (grams/day)	4.0 2.5 2.2 10.9	31.9 27 17.9 38.7	1991 NY Angler survey – approx. LN(4.0, 5.0) 1993 Maine Angler survey – LN(2.5, 4.25) 1992 L. Ontario survey – LN(1.98, 3.95) 1989 Michigan survey – LN(7.9, 3.16)
Exposure Point Concentration[b] (mg/kg)	2.5 4.6 1.2 0.5	1.6 3.0 2.0 0.9	12 & 40-year averages – Averaged over 3 Locations 12 & 40 year averages – RM 189 12 & 40 year averages – RM 168 12 & 40 year averages – RM 154
Fraction of Fish from Upper Hudson (%)	100%	NA	Defined based on exposed population (no variability/uncertainty modeled)
Cooking Loss (%)	0% 20% 40%	No variability	0% -- RME point estimate and high end intake factor for Monte Carlo 20% -- CT point estimate 40% -- lower bound intake factor for Monte Carlo
Exposure Duration (years)	12 11	40 41	Minimum of fishing duration and residence duration Residence duration
Body Weight[c] (kg) Adult Age 10	(Median) 69.3 36.3	95th Pct. 100 50.3	Lognormal by age class: LN(69.28, 1.25) LN(36.26, 1.22)

Notes:

[a]Fish ingestion values are for adults; values for children and adolescents scaled by body weight. Distribution parameters for lognormal distribution, LN(GM, GSD), where GM and GSD are the geometric mean and geometric standard deviation.

[b]Mean and high end for PCB concentration in fish are averages for 3 fish species averaged over mean and high end exposure durations (see text). Additional source of variability modeled in the Monte Carlo analysis was based on PCB variations of individual fish species.

[c]Modeled as lognormal with given GM and GSD, *e.g.*, LN(GM, GSD) (see Table 3-16 for other age groups). Point estimate 70 kg used for adults for both CT and RME calculations.

The Monte Carlo exposure analysis examines variability (and sensitivity/uncertainty) only of PCB intake. The intake is translated into health risk by combining the intake results with PCB toxicity factors for both cancer risk and non-cancer health hazard evaluations. Thus, the intake results are scaled linearly by the corresponding toxicity factors. A discussion of the base case Monte Carlo analysis results is presented in Section 5.2 and the sensitivity analysis is discussed in Section 5.3.3.

3.3.2 Numerical Stability Analysis

The Monte Carlo simulations were implemented using SAS version 6.12. A total of 10,000 iterations were performed for each of the 72 scenarios evaluated.

In order to investigate the numerical stability of the Monte Carlo calculations, 100 independent trials, each of 10,000 iterations, were run. As shown below, the small coefficients of variation, which is the standard deviation (s_x) divided by the mean ($\overline{x}$)for various PCB intake percentiles shows that 10,000 samples is sufficient to produce stable numerical results.

Numerical Stability Results (100 Simulations of 10,000 iterations)	
Statistic	*Coefficient of Variation* ($s_x/\overline{x}$)
5th percentile	2.9%
25th percentile	2.3%
50th percentile	1.9%
90th percentile	2.6%
95th percentile	3.8%
99th percentile	6.0%

At the 50th percentile (median) intake, the standard deviation of the 100 simulations (each consisting of 10,000 simulated anglers) was within 1.9% of the mean. For the tails of the intake estimates, the standard deviation of the 95th percentile intake was within 3.8% of the mean, and for the 99th percentile within 6% of the mean.

4 Toxicity Assessment

PCBs are a group of synthetic organic chemicals that contain 209 individual chlorinated biphenyl compounds (also known as congeners) with varying harmful effects. There are no known natural sources of PCBs in the environment. PCBs enter the environment as mixtures containing a variety of individual components (congeners) and impurities that vary in toxicity. Commercially available PCB mixtures are known in the U.S. by their industrial trade name, Aroclor. The name Aroclor 1254, for example, means that the molecule contains 12 carbon atoms (the first 2 digits) and approximately 54% chlorine by weight (second 2 digits). The manufacture processing and distribution in commerce of PCBs in the U.S. was restricted beginning in October 1977 because of evidence that PCBs build up in the environment and cause harmful health effects (USEPA, 1978).

At sufficient dose levels, PCBs have been demonstrated to cause a variety of adverse health effects, both carcinogenic and noncarcinogenic. These health effects include cancer, liver toxicity, reproductive toxicity, developmental effects, neurotoxicity, immunotoxicity, dermal toxicity, thyroid effects, and endocrine effects as described in USEPA's IRIS toxicity profiles (USEPA, 1999a-c) and reviewed by Safe (1994) and ATSDR (1997). The toxicity of PCBs for both cancer and non-cancer health effects is summarized in more detail in Appendix D.

USEPA has classified PCBs as "B2" probable human carcinogens based on liver tumors found in female rats exposed to Aroclor 1260, 1254, 1242, and 1016, and in male rats exposed to Aroclor 1260 and suggestive evidence from human epidemiological data (USEPA, 1999c). USEPA has also derived reference doses for Aroclors 1016 and 1254 based on non-cancer health effects, such as reduced birth weight (Aroclor 1016) and impaired immune function, distorted finger and toe nail beds, and occluded Meibomian glands located in the eyelid (Aroclor 1254).

It is also important to recognize that commercial PCBs tested in laboratory animals were not subject to prior selective retention of persistent congeners through the food chain (*i.e.*, laboratory test animals were fed Aroclor mixtures, not environmental mixtures that had been bioaccumulated). According to USEPA's analysis of published studies, bioaccumulated PCBs appear to be more toxic than commercial PCBs and appear to be more persistent in the body (USEPA, 1996c; 1999c).

Potential non-cancer health hazards and cancer risks posed by exposure to PCBs are evaluated using toxicity values, which are determined from systemic toxicity for non-cancer health effects (oral RfDs and inhalation Reference Concentrations (RfCs)), or chemical dose-response relationships for carcinogenicity (cancer slope factors, or CSFs). Following an external and internal peer review process, the profiles presented in USEPA's Integrated Risk Information System (IRIS) consensus database summarize the toxicity of the individual chemicals.

4.1 EPA's IRIS Non-cancer Toxicity Values for PCBs

The chronic RfD represents an estimate (with uncertainty spanning perhaps an order of magnitude or greater) of a daily exposure level for the human population, including sensitive subpopulations, that is likely to be without an appreciable risk of deleterious effects during a lifetime. USEPA derives RfDs by first identifying the highest dose level that does not cause observable adverse effects (the no-observed-adverse-effect-level, or NOAEL). If a NOAEL was not identified, a lowest-observed-adverse-effect-level, or LOAEL, may be used. This dose level is then divided by uncertainty factors to calculate an RfD. There are four standard uncertainty factors that can be used when calculating an RfD:

- An up-to-10-fold factor to account for the variation in sensitivity among members of the human population.

- An up-to-10-fold factor to account for the uncertainty involved in extrapolating from animal data to humans.

- An up-to-10-fold factor to account for the uncertainty involved in extrapolating from less than chronic NOAELs to chronic NOAELs.

- An up-to-10-fold factor to account for the uncertainty involved in extrapolating from LOAELs to NOAELs.

An additional modifying factor can also be applied to the calculation of the RfD. The modifying factor (MF) is an additional uncertainty factor that is greater than zero and less than or equal to 10. The magnitude of the MF depends upon an assessment of the scientific uncertainties of the study and the database used in deriving the RfD that are not explicitly treated above; *e.g.*, completeness of the overall data base and number of species tested.

The IRIS consensus database provides oral RfDs for two Aroclor mixtures, Aroclor 1016 and Aroclor 1254 (summarized in Table 4-1). There is no RfD available for Total PCBs and Aroclor 1248. The RfD for Aroclor 1016 is 0.00007 (7×10^{-5}) mg/kg-day, based on the NOAEL for reduced birth weight in a monkey reproductive bioassay, and an uncertainty factor of 100. This RfD is more stringent than the former RfD of 0.0004 used in the Phase 1 risk assessment.

The RfD for Aroclor 1254 is 0.00002 (2×10^{-5}) mg/kg-day, based on the LOAEL for impaired immune function, distorted growth of finger nails and toe nails, and inflamed and prominent Meibomian glands in the rhesus monkey, and an uncertainty factor of 300. This RfD is more stringent than the former RfD of 0.0004 used in the Phase 1 risk assessment.

For both Aroclor 1016 and Aroclor 1254, the USEPA reports "medium" confidence in the toxicity studies on which the RfDs are based, the overall toxicity database, and the RfDs themselves.

Although there is an IRIS file for Aroclor 1248, the USEPA determined the available health effects data to be inadequate for derivation of an oral RfD (USEPA, 1999e). However, a brief summary of the principal findings of animal studies is included in the IRIS file (USEPA, 1999e). Results of the studies showed impairment of reproduction in female rhesus monkeys lasting more than 4 years after dosing, reduced birth weight for infants, facial acne and edema, swollen eyelids, and hair loss.

Due to various environmental processes, PCB mixtures present in the environment no longer resemble the Aroclor mixture originally released into the environment. Therefore, although the GE facilities historically used primarily Aroclor 1242 in their operations, the PCBs present in Upper Hudson River fish, sediment, and river water do not have the same distribution of PCB congeners as any of the commercial Aroclor mixtures. However, since RfD values are only available for Aroclor mixtures and not Total PCBs, it was necessary to choose the Aroclor mixture most similar to the PCBs present in Upper Hudson River fish, sediment, and river water.

The PCB homologue distribution of sediment and water samples is predominately dichloro- through pentachlorobiphenyls, as reported in the Hudson River Data Evaluation and Interpretation Report

(USEPA, 1997d). This distribution is more similar to Aroclor 1016 than to Aroclor 1254. Therefore, for the purposes of this Revised HHRA, PCBs in sediment and water samples were considered to be most like Aroclor 1016. The Aroclor 1016 RfD (7×10^{-5} mg/kg-day) was used to evaluate non-cancer toxicity for ingestion of Upper Hudson River sediment, dermal contact with Upper Hudson River sediment, and dermal contact with Upper Hudson River water.

The PCB homologue distribution in fish differs from the sediment and water samples due to differential bioaccumulation of PCB congeners with higher chlorination levels. Trichloro- through hexachlorobiphenyls contribute to the majority of fish tissue PCB mass as reported in the (USEPA, 2000a). This distribution is more similar to Aroclor 1254 than to Aroclor 1016. Therefore, for the purposes of this HHRA, PCBs in fish were considered to be most like Aroclor 1254. The Aroclor 1254 RfD (2×10^{-5} mg/kg-day) was used to evaluate non-cancer toxicity for ingestion of Upper Hudson River fish for both the point estimate and probabilistic assessments. Consistent with USEPA policy (USEPA, 1997a), uncertainty and variability in the toxicity values are not quantitatively evaluated in the Monte Carlo analysis.

The Aroclors tested in laboratory animals were not subject to prior selective retention of persistent congeners through the food chain. For exposure through the food chain, therefore, non-cancer health hazards can be higher than those estimated in this assessment.

As indicated in Table 4-2, there are no RfCs currently available for either Total PCBs or any of the Aroclor mixtures (USEPA, 1999a-c). Therefore, inhalation exposures to PCBs are evaluated only for cancer (using the CSF), and not quantified for non-cancer health hazards.

4.2 EPA's IRIS Values for PCB Cancer Toxicity

The cancer slope factor, or CSF, is a plausible upper bound estimate of carcinogenic potency used to calculate cancer risk from exposure to carcinogens, by relating estimates of lifetime average chemical intake to the incremental probability of an individual developing cancer over a lifetime. The CSFs developed by the USEPA are plausible upper bound estimates, which means that the USEPA is reasonably confident that the actual cancer risk will not exceed the estimated risk calculated using the CSF.

USEPA has classified PCBs as "B2" probable human carcinogens, based on liver tumors found in female rats exposed to Aroclors 1260, 1254, 1242, and 1016, and in male rats exposed to Aroclor 1260 and suggestive evidence from human epidemiological data (USEPA, 1996c; 1999c). In IRIS, which summarizes the Agency's review of cancer toxicity data (USEPA, 1996c; 1999c), both upper-bound and central-estimate CSFs are listed for three different tiers of PCB mixtures (Aroclors 1260, 1254, 1242, and 1016). These PCB mixtures contain overlapping groups of congeners that span the range of congeners most often found in environmental mixtures. The CSFs are based on the USEPA's reassessment of the toxicity data on the potential carcinogenic potency of PCBs in 1996 (USEPA, 1996c; Cogliano, 1998) and were derived following the proposed revisions to the USEPA Carcinogen Risk Assessment Guidelines (USEPA, 1996b), including changes in the method of extrapolating from animals to humans and changes in the categories for classifying the carcinogenic potential of chemicals. The CSF reassessment was also externally peer-reviewed. The first tier, "High Risk and Persistence," applicable to food chain exposures, sediment or soil ingestion, dust or aerosol inhalation, dermal exposure (when an absorption factor is applied), early-life exposure, and mixtures with dioxin-like, tumor promoting, or persistent congeners, has upper-bound and central-estimate CSFs of 2.0 and 1.0 $(mg/kg\text{-}day)^{-1}$, respectively. The second tier, "Low Risk and Persistence," applicable to ingestion of water-soluble congeners, inhalation of evaporated

congeners, and dermal exposure (if no absorption factor has been applied), has upper-bound and central-estimate CSFs of 0.4 and 0.3 $(mg/kg\text{-}day)^{-1}$, respectively. The third tier, "Lowest Risk and Persistence," applicable only to mixtures where congeners with more than four chlorines comprise less than one-half percent of the Total PCBs, has upper-bound and central-estimate CSFs of 0.07 and 0.04 $(mg/kg\text{-}day)^{-1}$, respectively.

Consistent with the recommended values in IRIS, the first tier upper-bound and central-estimate CSFs of 2.0 and 1.0 $(mg/kg\text{-}day)^{-1}$ are used to evaluate cancer risks for the upper-bound and central-estimate exposures to PCBs *via* ingestion of Upper Hudson River fish, ingestion of Upper Hudson River sediments, and dermal contact with Upper Hudson River sediments (Table 4-3). These CSFs are lower than the former value of 7.7 $(mg/kg\text{-}day)^{-1}$ used in the Phase 1 risk assessment as a result of new scientific data and changes in the methods for calculating the CSF as indicated in the proposed Carcinogen Guidelines (USEPA, 1996b). The second tier upper-bound and central-estimate CSFs of 0.4 and 0.3 $(mg/kg\text{-}day)^{-1}$ are used to evaluate cancer risks for the upper-bound and central-estimate exposures to PCBs *via* dermal contact with Upper Hudson River water and potential inhalation of PCBs volatilized from the Upper Hudson River (Tables 4-3 and 4-4). In the Phase 1 risk assessment, the former CSF value of 7.7 $(mg/kg\text{-}day)^{-1}$ was used.

For the Monte Carlo analysis of cancer risks *via* fish ingestion, only the upper bound CSF of 2.0 $(mg/kg\text{-}day)^{-1}$ is used. Consistent with USEPA policy (USEPA, 1997a), variability and uncertainty in chemical toxicity is discussed in the risk characterization (Section 5.3.2), but not quantitatively evaluated in the Monte Carlo analysis.

4.3 Toxic Equivalency Factors (TEFs) for Dioxin-Like PCBs

A subset of PCB congeners are considered to be dioxin-like, that is, they are structurally similar to dibenzo-p-dioxins, bind to the aryl hydrocarbon receptor, and cause dioxin-specific biochemical and toxic responses (reviewed in USEPA, 1996c). Several investigators have estimated the carcinogenic potency of these dioxin-like PCB congeners relative to 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD). Dioxins, furans, and dioxin-like PCBs have been associated with numerous adverse health effects, including cancer, developmental and reproductive effects, and immunotoxicity. USEPA has set a CSF of 150,000 $(mg/kg\text{-}day)^{-1}$ for TCDD, based on liver and respiratory tumors in chronically-exposed rats (USEPA, 1997g). It should be noted that USEPA is currently completing its reassessment of the toxicity of TCDD including a Response to Comments from the Science Advisory Board and external peer reviewers. USEPA is currently revising its document to respond to these comments and recommendations.

Dr. Safe proposed TEFs for a number of dioxin-like PCBs based on a review of the available scientific data on the toxicity and mechanisms of action of dibenzo-p-dioxin, dibenzofuran, and PCB congeners (Safe, 1990; Safe, 1994). In 1994, the World Health Organization (WHO) European Center for Environment and Health and the International Program on Chemical Safety (IPCS) published recommended interim TEFs for thirteen dioxin-like PCB congeners based on a comprehensive review of the available scientific literature and consultation with twelve international PCB experts (Ahlborg *et al.*, 1994). The 1994 WHO/IPCS TEFs are summarized in Table 4-5. In 1996, USEPA recommended that the 1994 WHO/IPCS TEFs could be used to supplement analyses of PCB carcinogenicity (USEPA, 1996c). Subsequently, WHO/IPCS held a meeting in 1997 to reevaluate and update TEFs for dioxin-like PCBs (Van den Berg *et al.*, 1998), based on a review of both previously reviewed and new data. The WHO revised TEFs for human health risk assessment were published in 1998 and are also summarized in Table 4-5. Only four TEFs were changed from the 1994 document: the TEF for PCB congener 77 was

reduced from 0.0005 to 0.0001, a TEF for congener 81 was added, and the TEFs for congeners 170 and 180 were withdrawn.

Dioxin-like PCB congeners are responsible for only part of the carcinogenicity of a Total PCB mixture. To account for the fact that relative concentrations of dioxin-like congeners may be enhanced in environmental mixtures, particularly in fish due to bioaccumulation of more persistent congeners, the 1998 WHO/IPCS TEFs are used in the calculation of cancer risks for dioxin-like PCBs and discussed in the risk characterization, along with the CSF of 150,000 $(mg/kg\text{-}day)^{-1}$ for dioxin, to supplement the evaluation of PCB cancer risks due to consumption of fish (USEPA, 1997g). Note that use of the 1994 WHO/IPCS TEFs would result in similar cancer risk estimates.

4.4 Summary of Additional Key PCB Cancer and Non-cancer Studies

In response to comments received from the peer reviewers, this Revised HHRA contains a summary of several recently published scientific papers on PCB toxicity from human epidemiological studies. Based on an electronic literature search (Medline and Toxline), a number of recently published human and animal studies on PCB toxicity and carcinogenicity were identified. The ATSDR (1997) and USEPA (1999) have recently reviewed a number of published studies, that includes several studies that are also summarized in Appendix D, of the health effects of PCBs, primarily due to PCB intake from fish consumption.

A subset of the recent human epidemiological studies are summarized in the Appendix D in Table D-1, including those epidemiological and population studies suggested by the Peer Review panel, and selected other human studies that are commonly referenced by the scientific community. The results of the studies reported in Table D-1 summarize the study findings as characterized by the author(s). The studies are included to provide updated information published in the scientific literature and to respond to peer review comment, and are not necessarily representative of Agency policies or positions.

The potential impact of these new studies on PCB risk assessment is discussed briefly in the remainder of this section and in more detail in Appendix D. Note that this summary focused primarily on studies published within the past few years and is intended to supplement toxicity summaries presented by USEPA in the PCB IRIS file (USEPA, 1999a,b,c). As part of USEPA's IRIS process, the Agency is currently evaluating the human epidemiological and animal non-cancer studies as part of its reassessment of the current RfDs for PCBs and it would be inappropriate at this time to pre-judge the results of this reassessment regarding the effect of these studies on the calculated RfDs.

4.4.1 Cancer

Recently, Dr. Kimbrough *et al.* (1999) published a paper describing a study of 7,075 male and female workers from two GE capacitor manufacturing plants in New York State. In this study, mortality (deaths) from all cancers was determined for the study group, which comprises 7,075 female and male workers who worked at the GE facilities for at least 90 days between 1946 and 1977. Of the total population of 7,075 workers, there were 586 deaths among the male hourly workers, 380 deaths among the female hourly workers, 177 deaths among the salaried male workers, and 52 deaths among the salaried female workers. No significant elevations in mortality for any site-specific cause were found in the hourly cohort. No significant elevations were seen in the most highly exposed workers. Mortality from all cancers was significantly below expected in hourly male workers and comparable to expected for hourly female workers (Kimbrough *et al.*, 1999). As discussed in Appendix D (Section D.4.1), USEPA has reviewed this study and the preliminary Agency findings are described there.

A summary of occupational and non-occupational studies of populations exposed to PCBs is provided in Appendix D. In several studies (Moysich *et al.*, 1999, Rothman *et al.*, 1997, and Rylander *et al.*, 1995), positive associations were found between PCB blood concentrations and different forms of cancer. Several occupational studies reviewed by USEPA in the IRIS file for PCBs found associations between PCB exposures and different forms of cancer. However, several other studies did not find associations between serum (blood) PCB concentrations and specific diseases (see Appendix D).

In general, the human epidemiological studies are limited based on the population sample sizes, brief followup periods, poor information on exposure to PCBs, selection of control groups, and confounding exposures to other potential carcinogens. Based on USEPA's evaluation of the human epidemiological evidence from several studies (Bertazzi *et al.*, 1987; Sinks *et al.*, 1992; Brown*.*, 1987; NIOSH, 1977, Gustavsson *et al.*, 1986 and Shalat *et al.*, 1989) USEPA concludes that the evidence is inadequate but suggestive of human carciogenicity (USEPA, 1999c).

A discussion of the uncertainties associated with the CSF for PCBs is provided in Chapter 5. In general, the uncertainties around the CSF estimates extend in both directions and may lead to either under-estimates or over-estimates of the cancer potency of PCBs (USEPA, 1996c). However, overall, the CSFs developed by the USEPA represent plausible upper bound estimates, which means that the USEPA is reasonably confident that the actual cancer risk will not exceed the estimated risk calculated from the CSF.

4.4.2 Reproductive and Developmental/Neurotoxic Effects

A number of recent studies have investigated possible developmental and neurotoxic effects in children from pre-natal or post-natal exposures to PCBs. One of the key studies was a longitudinal prospective study on children whose mothers consumed about two meals per month of Lake Michigan fish containing PCBs, in which prenatal PCB exposure was associated with reduced birth weight, smaller head circumference, adverse behavioral outcomes, and cognitive deficits apparent at eleven years of age (Fein *et al*., 1984; Jacobson and Jacobson, 1996; Jacobson and Jacobson, 1997, Schantz, 1996). The North Carolina Breast Milk and Formula project studied a cohort selected from the general population in North Carolina, and found an association between pre-natal PCB exposures and some, but not all, of the effects observed in the Michigan studies (Rogan and Gladen, 1985; Rogan *et al*., 1986; Rogan *et al*., 1991; Gladen and Rogan, 1991). In a cohort of Dutch mother-infant pairs exposed primarily through non-fish food products, effects on growth and development reported to date include lower birth weights and decreased postnatal growth and delays in psychomotor development and neurodevelopment, primarily associated with prenatal PCB and dioxin exposures and not lactational exposures (Sauer *et al*., 1994; Patandin *et al*., 1998; Koopman-Esseboom *et al*., 1996; Huisman *et al*., 1995; Koopman-Esseboom *et al*., 1994; Weisglas-Kuperus *et al*., 1995). Similar developmental and neurotoxic effects have been observed in recent monkey studies of post-natal PCB exposure in young monkeys (*e.g.*, Rice, 1997; 1999).

Possible reproductive effects from maternal or paternal exposures to PCBs have also been investigated in recent studies. Endpoints tested include conception delay, spontaneous fetal death, and changes in menstrual cycle (Buck *et al*., 1997; Courval *et al*., 1999; Stein *et al*., 1999; Mendola *et al*., 1997; Buck *et al*., 1999; Mendola *et al*., 1995). Taken together, the evidence for reproductive effects is inconclusive, and although these results add uncertainty to PCB non-cancer hazard assessment, at this point it is unclear whether these effects would tend to increase or decrease the PCB non-cancer health hazards estimated in this risk assessment.

USEPA is currently evaluating these studies, and others, to determine whether modifications are required to the current RfDs for PCBs. A paper summarizing the available data and USEPA's decisions

regarding the non-cancer health effects of PCBs will be submitted to external independent peer-review. Following this, a chemical file will be developed and submitted for internal USEPA consensus review. At this time, it is premature to determine whether there will be any changes in the current RfDs for PCBs.

4.4.3 Endocrine Disruption

In response to growing concerns about the potential effects of environmental endocrine disruptors on human health, the USEPA's Risk Assessment Forum held several workshops to discuss the current status of knowledge on endocrine disruption at the request of the USEPA Science Policy Council in 1997. As a result of these workshops, USEPA prepared the "Special Report on Environmental Endocrine Disruption: An Effects Assessment and Analysis" (USEPA, 1997b), which is intended to inform Agency risk assessors of the major findings and uncertainties regarding endocrine disruptors and to serve as a basis for a Science Policy Council position statement.

An environmental endocrine disruptor is defined as "an exogenous agent that interferes with the synthesis, secretion, transport, binding, action, or elimination of natural hormones in the body that are responsible for the maintenance of homeostasis, development, and/or behavior" (USEPA, 1997b, pg. 1).

PCBs have been investigated as potential endocrine disruptors, which can affect both cancer and non-cancer health endpoints. For example, some studies have suggested that PCBs increase the risk of breast cancer, while other studies have failed to show an association between PCB exposure and breast cancer (reviewed in USEPA, 1997b). Overall, the USEPA Risk Assessment Forum concluded that it is not possible to attribute a cause and effect association between PCB exposure and breast cancer given the sparse data currently available. Similarly, an association between endometriosis and high levels of PCBs in blood has been reported, but the evidence for a causal relationship is considered weak (reviewed in USEPA, 1997b). Due to the similar structural properties of PCBs and normal thyroid hormones (T_4 and T_3), PCBs may also cause thyroid effects such as hypothyroidism (reduction of thyroid hormones in circulation) *via* competition for receptor binding (reviewed in USEPA, 1997b). The mechanisms of thyrotoxicity associated with PCB exposure may vary and include specific damage to the endocrine gland, interference with hormone transport, and receptor interactions (USEPA, 1997b). For example, in rats, prenatal exposure to some PCBs (specific congeners or mixtures such as Aroclor 1254) have been shown to lower serum T_4 which reduces choline acetyl transferase (ChAT) activity in the hippocampus and basal forebrain. ChAT is involved in the synthesis of acetylcholine, a neurotransmitter considered important to learning and memory (USEPA, 1997b). PCB exposures may also be associated with an increase in thyroid follicular cell adenomas or carcinomas in male rats with a statistically significant trend for Aroclor 1242 and 1254 (Mayes *et al.*, 1998).

There is currently considerable scientific research to understand whether environmental chemicals acting *via* endocrine disruptor mechanisms are responsible for adverse health effects in humans (reviewed in USEPA, 1997b). Because the human body has negative feedback mechanisms to control the fluctuations of hormone levels, exposures to chemicals at the levels found in the environment may be insufficient to disrupt endocrine homeostasis. It is also possible that infants and children are more sensitive to potential endocrine disruptor effects during sensitive windows of development.

The USEPA is concerned about the potential effects of environmental endocrine disruptors on human health and, along with other federal agencies, is currently supporting significant research in this area (USEPA, 1998e). However, "there is little knowledge of or agreement on the extent of the problem," and "further research and testing are needed" (USEPA, 1997b, p. vii). The USEPA Science Policy Council's Interim Position is that "based on the current state of the science, the Agency does not consider endocrine disruption to be an adverse endpoint per se, but rather to be a mode or mechanism of action

potentially leading to other outcomes, for example, carcinogenic, reproductive, or developmental effects, routinely considered in reaching regulatory decisions" (USEPA, 1997b, pg. viii).

Therefore, consistent with current USEPA policy, although PCBs may act as an environmental endocrine disruptor, the available data are insufficient to support a quantitative assessment of endocrine effects in this risk assessment. Potential adverse health effects resulting from PCBs operating through a potential endocrine disruption mechanism of action is an area of uncertainty. Because the IRIS values for PCB cancer risks and non-cancer health hazards do not directly incorporate concerns for endocrine effects, it is possible that cancer risks and non-cancer health hazards in this Revised HHRA have been underestimated.

4.5 Additional Considerations

4.5.1 PCB Half-Life / Body Burden / Timing of Exposure

PCBs are lipophilic, and tend to accumulate in lipid-rich tissues such as the liver, skin, and adipose tissue (ATSDR, 1997). PCBs remain in the body for an extended period of time, even if exposures are stopped abruptly. Because breast milk has a high fat content, a considerable portion of a mother's PCB body burden may be transferred to an infant during breast feeding, making lactation a major route of PCB excretion.

The half-lives of PCB congeners in the human body have not been well established, but are dependent in part on the number and position of chlorines present in each PCB congener. Half-lives tend to increase with the number of chlorines (ATSDR, 1997). One group of scientists have estimated PCB half-lives to range from 5 to 15 years (Patandin *et al.*, 1999). Other scientists have concluded that half-lives for PCB congeners frequently found in blood are unlikely to be less than one year, or greater that ten years (Shirai and Kissel, 1996). It is difficult to measure PCB half-lives even in workers occupationally exposed to PCBs, due to complications with continued low level exposure. ATSDR summarized that PCB congeners can remain in the body for months to years (ATSDR, 1997).

Transfer of maternal PCBs across the placenta and into breast milk can clearly result in significant exposures *in utero* and to a nursing infant (DeKoning and Karmaus, 2000). Exposure to PCBs in breast milk is estimated to be a major contributor to a child's body burden at 42 months of age (Lanting *et al.*, 1998), and to account for over 10% of one's cumulative PCB intake through 25 years of age (Patandin *et al.*, 1999). A mother's body burden of PCBs has been estimated to decrease 20% for every 3-6 months of breast feeding (Patandin *et al*., 1999; Rogan and Gladen, 1985), after which PCB body burdens are gradually restored.

In the case of consumption of PCB-contaminated fish, body burden depends not only on the consumption rate of fish and the PCB concentrations in fish, but also on an individual's age and the temporal pattern of their previous PCB exposures. Thus, at any one time, an individual's body burden is a function of their current and past exposures, and may also be affected by significant fluctuations in an individual's weight. PCB exposures *in utero* are based on the mother's current and past history of PCB exposures. PCB exposures in breast milk depend not only on maternal PCB exposure levels, but can also be significantly influenced by factors such as maternal age, number of children, length of time between children, and duration of breastfeeding (Vartiainen *et al.*, 1998; Rogan *et al.*, 1986).

These unique features of the pharmacokinetics of PCBs raise questions about how to deal with past exposures and how to select the most appropriate averaging time, particularly for evaluating pregnant

mothers and nursing infants, and there are no clear-cut answers. With regard to the question of past exposures, the Revised HHRA is a prospective, not retrospective, assessment, and so by design considers only incremental exposures, not past exposures. Nonetheless, the fact that any previous exposures (either background, or past consumption of PCB-contaminated fish) may still be reflected in an individual's body burden today is considered as a source of uncertainty which may underestimate the cancer risk and non-cancer health hazards.

With regard to nursing infants, PCB levels in breast milk reflect the maternal body burden, and since the maternal body burden in a function of longer-term exposures, an averaging time on the order of years is appropriate. With regard to pregnant women, a shorter averaging time may be more appropriate in evaluating potential developmental and neurotoxic effects from pre-natal exposures, although it depends somewhat on the length of the critical window of exposure in fetuses. If a short window of enhanced susceptibility for effects in fetuses exists (on the order of days or weeks), it is possible that a peak exposure for the mother during that time period could transiently increase maternal blood and transplacental PCB transfer sufficiently to cause adverse effects. If, on the other hand, the critical window of exposure in fetuses is longer (on the order of the entire pregnancy), it is more likely that pre-natal exposures are driven by maternal body burden than by maternal exposures during the pregnancy only these issues should nonetheless be considered as a source of uncertainty.

4.5.2 Exposure to Pregnant Women and Nursing Children

As noted above (Section 4.4.2), a number of recent studies have indicated possible developmental and neurotoxic effects in children from pre-natal or post-natal exposures to PCBs. Although exposures to PCBs from breastfeeding can be significantly higher than *in utero* PCB exposures, most observed developmental effects have been associated with pre-natal exposures and not breast milk exposures (Feeley and Brouwer, 2000; Korrick and Altshul, 1998), suggesting that a developing fetus may be particularly sensitive to PCB exposure. The results from the various studies are not entirely consistent and there are limitations about the study designs. However, the fact that neurobehavioral and learning problems have been observed in animal toxicity studies provides additional support for the findings of developmental and neurotoxicity effects in humans. As described above, USEPA is currently reassessing the non-cancer RfDs for Aroclors 1254 and 1016 and following the reassessment of the scientific literature will determine whether modifications to the RfD are appropriate.

It is important to consider pregnant women and nursing children when conducting risk assessments of PCB exposures. Transfer of maternal PCBs across the placenta and into breast milk can clearly result in significant exposures *in utero* and to a nursing infant (DeKoning and Karmaus, 2000). Exposure to PCBs in breast milk is estimated to be a major contributor to a child's body burden at 42 months of age (Lanting *et al*., 1998) and to account for over 10% of one's cumulative PCB intake through 25 years of age (Patandin *et al*., 1999). However, as discussed in Appendix D, it is unclear which PCB exposure index is most appropriate to evaluate potential adverse health effects from pre-natal and post-natal exposures.

Furthermore, methods to model PCB concentrations in serum or *in utero*/lactational exposures are not well established (Appendix D). PCB exposures *in utero* are based on the mother's current and past history of PCB exposures. PCB exposures in breast milk depend not only on maternal PCB exposure levels, but can also be significantly influenced by factors such as maternal age, number of children, length of time between children, and duration of breastfeeding (Vartiainen *et al.*, 1998; Rogan *et al.*, 1986). A mother's body burden of PCBs has been estimated to decrease 20% for every 3-6 months of breast feeding (Patandin *et al.,* 1999; Rogan and Gladen, 1985), after which PCB body burdens are gradually restored.

Well established methodologies for evaluating PCB exposures in pregnant women and nursing children are not available at this point. Therefore, it is also not possible (through available data or modeling) to make a relevant, direct comparison between exposure levels estimated for Hudson River anglers in this risk assessment (reported in mg/kg-d) and exposure levels for pregnant women and nursing children reported in human studies (typically reported as PCB concentrations in blood or breast milk), without introducing a considerable level of uncertainty. Since developmental and neurotoxic effects have been observed in frequent angler populations consuming fish known to be contaminated with PCBs, and also in general populations with no known sources of significant PCB exposure, it seems plausible that PCB exposures for at least some women consuming Hudson River fish could be in the same range of PCB exposure levels at which developmental and neurotoxic effects have been observed. In part, this is based on concerns regarding the pre-natal exposures which could be quite short (on the order of days to months).

5 Risk Characterization

Risk characterization is the final step of the risk assessment process, which combines the information from the Exposure Assessment and Toxicity Assessment steps to yield estimated non-cancer health hazards and cancer risks from exposure to PCBs. It should be emphasized that because this Revised HHRA is for current and future exposures to PCBs from the Upper Hudson River (*i.e.*, exposures beginning in 1999 and beyond), the estimated risks in this assessment are incremental and overlay possible prior exposures to PCBs from the River. Furthermore, the cancer risks and non-cancer health hazards in this assessment do not include exposures to environmental contaminants from other possible sources.

The risk characterization step also involves an evaluation of the uncertainties underlying the risk assessment process, and this evaluation is included in this section. The risk characterization was prepared in accordance with USEPA guidance on risk characterization (USEPA, 1995b; USEPA, 1992b).

In Section 5.1, the point estimate calculations of non-cancer HIs and cancer risks are presented. The Monte Carlo risk estimates for the base case analysis are summarized in Section 5.2. A discussion of uncertainties inherent to the exposure and toxicity assessments is presented in Section 5.3, along with a quantitative evaluation of the uncertainty in risk characterization for the fish ingestion pathway.

5.1 Point Estimate Risk Characterization

5.1.1 Non-cancer Hazard Indices

The evaluation of non-cancer health effects involves a comparison of average daily exposure levels with established RfDs to determine whether estimated exposures exceed recommended limits to protect against chronic adverse health hazards. A RfD is defined as an estimate (with uncertainty spanning perhaps an order of magnitude or greater) of a daily exposure level for the human population, including sensitive subpopulations, that is likely to be without an appreciable risk of deleterious effects during a lifetime. Chronic RfDs are specifically developed to be protective for long-term exposure to a compound, with chronic duration ranging from seven years to a lifetime (USEPA, 1989a,b).

Potential health hazards from noncarcinogenic effects are expressed as a Hazard Quotient (HQ), which compares the calculated exposure (average daily doses, calculated as part of the exposure assessment in Chapter 2) to the RfD (summarized as part of the toxicity assessment in Chapter 4). Both exposure levels and RfDs are typically expressed in units of mass of PCB intake per kilogram of body weight per day (mg/kg-day). Unlike the evaluation of carcinogenic effects, exposures of less than lifetime duration are not averaged over an entire lifetime but rather the duration of exposure (USEPA, 1989b).

The hazard quotient is calculated by dividing the estimated average daily oral dose estimates by the oral RfD as follows (USEPA, 1989b):

$$Hazard\ Quotient\ (HQ) = \frac{Average\ Daily\ Dose\ (mg/kg-day)}{RfD\ (mg/kg-day)} \qquad [5\text{-}1]$$

High-end (RME) and CT hazard quotients calculated for each exposure pathway (fish ingestion, sediment, and water exposure pathways) are summarized in Tables 5-1 through 5-13. Hazard Quotients

are summed over all COCs (chemicals of concern) and all applicable exposure routes to determine the total HI. In this Revised HHRA, PCBs are the COCs and the HQ for PCBs is equivalent to the HI. The total high-end (RME) and CT HIs for each pathway and receptor are summarized in Tables 5-27 through 5-33.

If a Hazard Index is greater than one (*i.e.*, HI>1), unacceptable exposures may be occurring, and there may be an increased concern for potential non-cancer health effects, although the relative value of an HI above one (1) cannot be translated into an estimate of the severity of the hazard. Ingestion of fish results in the highest HI, with an HI of 12 for the CT estimate, and an HI of 104 for the high-end (RME) estimate, both representing exposures above the reference level (HI>1). These non-cancer HIs represent values for children, which are higher than those for adults and adolescents. As discussed earlier, the average daily dose decreases as the exposure duration increases due to the declining PCB concentration over time. The average concentration over a 7-year exposure period (used as the high-end estimate in this Revised HHRA) is greater than the average concentration over the RME duration of 22 years for adults and 12 years for adolescents (note child RME exposure of 6 years already less and therefore more conservative than a 7-year exposure duration). The HIs for the adolescent and adult are 71 and 65, respectively. Total HIs for the recreational and residential exposure pathways are all below one (1.0). In all cases, the HIs are based on uniform exposure throughout the Upper Hudson River. Uncertainties inherent in these risk estimates are discussed in Section 5.3.

As discussed in Section 4.5.2 and Appendix D, developmental and neurotoxic effects, primarily in children, have been observed in frequent angler populations consuming fish known to be contaminated with PCBs, and also in general populations with no known sources of significant PCB exposure. If women consume fish from the Upper Hudson River, it seems plausible that PCB exposures for at least some of them could be in the same range of PCB exposure levels at which developmental and neurotoxic effects have been observed. This is particularly true because the exposure duration of interest for pre-natal exposures could be quite short (on the order of days to months). Therefore, although there is no established risk assessment methodology for evaluating PCB exposures in pregnant women and nursing children at this time, there is a potential for developmental and neurotoxic effects in these groups that could be associated with the consumption of Hudson River fish.

5.1.2 Cancer Risks

Cancer risks are characterized as the incremental increase in the probability that an individual will develop cancer during his or her lifetime due to site-specific exposure. The term "incremental" implies the risk due to environmental chemical exposure above the background cancer risk experienced by all individuals in the course of daily life. Cancer risks are expressed as a probability (*e.g.*, one in a million, or 10^{-6}) of an individual developing cancer over a lifetime, above background cancer risk, as a result of exposure.

The quantitative assessment of carcinogenic risks involves the evaluation of lifetime average daily dose and application of toxicity factors reflecting the carcinogenic potency of the chemical. Specifically, excess (incremental) cancer risks are calculated by multiplying intake estimates (lifetime average daily doses, calculated in Chapter 2 as part of the exposure assessment) and CSFs (summarized as part of the toxicity assessment in Chapter 4) as follows (USEPA, 1989b):

$$Cancer\ Risk = Intake\left(\frac{mg}{kg-day}\right) \times CSF\left(\frac{mg}{kg-day}\right)^{-1} \qquad [5\text{-}2]$$

As discussed in Chapter 2, exposure levels are expressed as the chronic daily intake averaged over a lifetime of exposure, in units of mg/kg-day (mg of PCB intake per kilogram of human body weight per day). A CSF is an estimate of the upper-bound probability of an individual developing cancer as a result of a lifetime of exposure to a particular level or dose of a potential carcinogen. CSFs are expressed in units that are the reciprocal of those for exposure (*i.e.*, $(mg/kg\text{-}day)^{-1}$). Multiplication of the exposure level by the CSF yields a unitless estimate of cancer risk. The acceptable risk range identified in the NCP 40 CFR § 300.430(e)(2)(i)(A)(2) (USEPA, 1990) is 10^{-4} to 10^{-6} (or an increased probability of developing cancer of 1 in 10,000 to 1 in 1,000,000) refers to plausible upper bound cancer risks.

High-end and central tendency cancer risk estimates calculated for each exposure pathway (fish ingestion, recreational exposure pathways, and residential inhalation) are summarized in Tables 5-14 through 5-26. Total cancer risks are summed over all applicable exposure routes and exposure periods (child through adult). The total RME and CT cancer risks for each pathway are summarized in Tables 5-27 through 5-33.

Ingestion of fish results in the highest cancer risks, 2.9×10^{-5} (2.9 in 100,000) for the central tendency estimate, and 1.4×10^{-3} (1.4 in 1,000) for the high-end estimate. These cancer risks represent the total risks for child (aged 1-6), adolescent (aged 7-18), and adult (over 18 years) exposures. As a further note on the fish ingestion risks, had the 95^{th} percentile fish ingestion rate from the New York Angler survey (63.4 g/day, or 102 meals per year) been used in the analysis, the RME cancer risks for fish ingestion would approximately double (*i.e.*, 2.8×10^{-3} or 2.8 in 1,000).

As indicated earlier, the acceptable cancer risk range established in the NCP is 10^{-4} to 10^{-6}. Thus, the RME fish ingestion results fall outside the NCP acceptable cancer risk range. Estimated cancer risks relating to PCB exposure in either sediment, water, or air are much lower than those for fish ingestion, falling generally at the low end, or below, the range of 10^{-4} to 10^{-6}.

5.1.3 Dioxin-Like Risks of PCBs

To account for the fact that relative concentrations of dioxin-like congeners may be enhanced in environmental mixtures, particularly in fish due to bioaccumulation of more persistent congeners, the 1998 WHO/IPCS TEFs are used in the risk characterization, along with the CSF of 150,000 for dioxin (USEPA, 1997g), to supplement the evaluation of PCB cancer risks due to consumption of fish.

This analysis was performed using the Phase 2 fish data from the Upper Hudson River (River Miles 159-196.9) contained in the Hudson River database. For each Phase 2 fish sample in the Upper Hudson River, the concentrations total (tri+) PCBs, were summarized (Tables 5-34).[12,13] In order to determine the fraction that each dioxin-like congener represented of the Total PCB concentration, the concentration of each dioxin-like PCB congener was divided by the Total PCB concentration for each fish sample (Table 5-35). These fractions were averaged over all the fish samples to determine an average fraction for each dioxin-like congener (Table 5-35, last two rows). These fractions were then multiplied by the high-end Total PCB exposure point concentration used in the risk assessment, to determine the high-end (RME) EPC for each dioxin-like congener (Table 5-36). These exposure point concentrations were then multiplied by the corresponding 1998 WHO/IPCS toxicity equivalency factors TEF to generate a dioxin equivalent (TEQ) for each dioxin-like congener (Table 5-36 last column). The TEQs for each

[12] Note that although PCB congener 81 is considered a dioxin-like PCB congener, it was not analyzed for as part of the analytical program. At the time the analytical sampling methods were determined for the Phase 2 program, a standard for congener 81 was unavailable. The cancer risks for this congener are not included in this cancer risk analysis.

[13] Non-detect values were set to ½ the detection limit if the total detection frequency was greater than 15% (based on professional judgment) for that congener. If the total detection frequency was less than 15%, the value was set to zero.

congener were summed, yielding a high-end total dioxin TEQ of 5.3×10^{-5} mg/kg (Table 5-36, second to last row). The total concentration of the non-dioxin-like PCB congeners was calculated by subtracting the sum of the concentrations of the dioxin-like congeners from the high-end Total PCB exposure point concentration (Table 5-36, last row).

Cancer risks for ingestion of dioxin-like PCBs in fish were calculated similarly to those for PCBs, substituting the dioxin TEQ for the EPC and the dioxin CSF of 150,000 (USEPA, 1997g) for the CSF. The resulting intake and cancer risk estimates are shown in Table 5-38. The RME dioxin-like cancer risk of 1.5×10^{-3} is approximately equivalent to the RME cancer risk calculated without consideration of the dioxin-like congeners, and, similarly, is outside of the acceptable range for cancer risk established in the NCP.

5.2 Monte Carlo Risk Estimates for Fish Ingestion

As described in Section 3.5.1, a total of 72 scenarios were evaluated for the Monte Carlo exposure analysis. The non-cancer health hazards and cancer risk estimates for each scenario were calculated using the same equations outlined in Sections 5.1.1 and 5.1.2, respectively, using Equation [3-1] to calculate PCB intake.

The matrix below summarizes whether PCB intake variability and/or uncertainty were quantitatively evaluated for the fish ingestion pathway in the Revised HHRA. Recall that the variability of PCB intake from fish consumption is based on true differences of fish ingestion tendencies, varying exposure duration, *etc.* for individuals within the exposed population. The uncertainty involved in developing estimates for each of the exposure factors is due to the fact that such estimates are developed from multiple studies or sources, the studies represent a finite data set based on a particular point in time, the study group(s) are from differing geographic locations, *etc.*

Summary of Exposure Parameter Variability and Uncertainty Examined For Fish Ingestion		
Exposure Parameter*	***Variability Modeled?***	***Uncertainty Modeled?***
Fish Ingestion Rate (meals/year)	***YES*** Empirical distribution for NY Angler Survey; lognormal distributions fit to 3 other surveys (Note – variability of ingestion also scaled by body weight for children and adolescents)	***YES*** -- *4 Independent distributions:* (1) NY Anglers (Connelly *et al*., 1992) (2) Maine Angler (Ebert *et al.*, 1993) (3) 1989 Michigan (West *et al.*, 1989) (4) 1992 Lake Ontario (Connelly *et al*, 1996)
Exposure Duration (years)	***YES*** **--** Empirical probability distributions Fishing duration based on NY Angler Survey Residence duration based on 1990 census data for 5 Upper Hudson counties	***YES*** – *2 distributions*: (1) Empirical conditional distribution of the minimum of fishing duration and residence duration (2) Distribution of residence duration only
Exposure Point Concentration (mg/kg)	***YES*** – *3 sources of variability evaluated:* Varies over time (70 year time horizon) 3 modeled fish groups 3 locations	***NO*** Uncertainty of modeled concentrations not evaluated quantitatively. Only variability in the concentration estimates were included.
Fraction of Fish from Upper Hudson (%)	***NO*** Exposed population defined as anglers who catch and consume fish from Upper Hudson	***NO*** Treated as fixed constant (100%) based on definition of potentially exposed population
Cooking Loss (%)	***NO*** Potentially depends on preparation methods, fish type, *etc.* As discussed in the text, published studies don't allow for a quantitative assessment of variability accounting for these factors.	***YES –*** *3 Point Estimates Evaluated* 0% high-end exposure 20% Central tendency 40% low-end exposure
Body Weight (kg)	***YES*** Lognormal distribution as a function of age.	***NO*** Uncertainty is likely minimal and will have small impact on dose estimates
**See text and Section 3.3.1 for specific distribution parameters used.*		

5.2.1 Non-Cancer Health Hazards

For the non-cancer hazard calculations, *Average Daily Dose* in Equation [5-1] was calculated using Equation [3-1], with a maximum exposure duration (ED in Equation [3-1]) of 7 years. This exposure duration limit was selected as the minimum time-period for chronic exposure. Because the Average Daily Dose declines as the exposure duration increases, allowing the intake to be averaged over a longer time-period would underestimate non-cancer health hazards and potentially underestimate the hazard for an RME individual.[14]

[14] The dependency of the intake on ED is due to the time-dependency of PCB concentration in fish.

Each of the 72 scenarios examined consisted of 10,000 simulations of PCB intake (average daily dose), each yielding a distribution of 10,000 intake estimates. From these distributions of intake, low-end, mid-point, and high-end non-cancer hazard index percentiles (5^{th},..., 50^{th}, 90^{th}, 95^{th}, 99^{th}) are summarized in Appendix C.

A relative frequency and cumulative distribution plot for the "base case" analysis is shown in Figure 5-1a. The median HI for the base case Monte Carlo analysis is 11.4, compared with the HI values of 12 (young child), 8 (adolescent), and 7 (adult) for the central point estimates. The 95^{th} percentile HI from the base case Monte Carlo analysis is 137, compared with 104 (young child), 71 (adolescent), and 65 (adult) for the RME point estimates. At the high-end of the base case hazard distribution, the 99^{th} percentile HI is 639; at the low end, the 5^{th} percentile HI is 1.2, and the 10^{th} percentile HI is 1.9. The Monte Carlo analysis of non-cancer health hazards is discussed further in the discussion of uncertainties later in Section 5.3.3.

5.2.2 Cancer Risks

For the cancer risk calculations, *Intake* in Equation [5-2] was calculated using Equation [3-1]. In the case of cancer risks, intake is averaged over a lifetime such that ED in Equation [3-1] was not limited to 7 years, but rather equaled the particular ED value that was sampled from the input probability distribution for this variable on each of the 10,000 iterations.

As was the case for non-cancer health hazards, each of the 72 scenarios examined consisted of 10,000 simulations of PCB intake, resulting in a distribution of 10,000 intake estimates. From these distributions of intake, low-end, mid-point, and high-end cancer risk percentiles (5^{th},..., 50^{th}, 90^{th}, 95^{th}, 99^{th}) are summarized in Appendix C.

A relative frequency and cumulative distribution plot for the "base case" analysis is shown in Figure 5-2a. The median cancer risk for the base case Monte Carlo analysis is 6.4×10^{-5}, which is approximately 2-fold higher than the central point estimate value of 2.9×10^{-5}. The 2-fold difference of these two estimates is directly tied to the fact that the PCB CSF used for the Monte Carlo estimate (2.0 mg/kg-day^{-1}) is 2-fold greater than the CSF used for the central point estimate (1.0 mg/kg-day^{-1}). The 95^{th} percentile cancer risk estimate for the base case Monte Carlo analysis is 8.7×10^{-4} , compared with 1.4×10^{-3} for the RME point estimate. At the high-end of the base case cancer risk distribution, the 99^{th} percentile is 3.7×10^{-3}; at the low end, the 5^{th} percentile is 5.5×10^{-6}, and the 10^{th} percentile 9.6×10^{-6}. The Monte Carlo analysis of cancer risk is discussed further in the discussion of uncertainties later in Section 5.3.3.

5.3 Discussion of Uncertainties

The process of evaluating human health cancer risks and non-cancer health hazards involves multiple steps. Inherent in each step of the process are uncertainties that ultimately affect the final cancer risk and non-cancer health hazard estimates. Uncertainties may exist in numerous areas, including environmental PCB concentration data, derivation of toxicity values, and estimation of potential site exposures. In this section, the significant sources of uncertainty in three of the four risk assessment steps (Exposure Assessment, Toxicity Assessment, and Risk Characterization) are qualitatively discussed, including the strengths, limitations, and uncertainties inherent in key scientific issues and science policy choices. This Revised HHRA accounts for sources of uncertainty in the various components of the risk assessment analysis in order to provide a full understanding of the accuracy and reliability of calculated

risks and hazards. An understanding of the strengths and potential uncertainties of the risk assessment provides the risk manager with additional information for consideration in the risk management decision.

5.3.1 Exposure Assessment

Selection of Exposure Pathways. There are some uncertainties inherent in the selection of exposure pathways quantitatively evaluated in the risk assessment. Fish consumption is the most significant source of cancer risk and non-cancer health effects due to exposure to PCBs in the Upper Hudson River. Anglers also may be exposed to PCBs in sediments and surface water while fishing. However, even if the angler experienced incidental ingestion of sediment, dermal contact with sediment and river water, and inhalation comparable to the adult recreator, such exposure would not measurably increase the cancer risk or non-cancer hazard indices because the fish ingestion pathway cancer risks and non-cancer health hazards outweigh all others by several orders of magnitude.

As discussed in Section 2.1.3, there were insufficient data to evaluate intake of PCBs *via* floodplain soils, ingestion of home-grown crops, beef, dairy products, eggs, turtles, ducks, *etc.* and these potential exposure pathways were not quantitatively evaluated in the risk assessment. Although the magnitude of the potential risks from these pathways cannot be reliably quantified with available information, the risks are likely to be lower than those evaluated quantitatively in this assessment. In addition, evaluation of the inhalation pathway was limited to cancer risks based on the lack of an inhalation RfC for non-cancer health hazards.

Defining the Angler Population. For the purposes of this risk assessment, the angler population is defined as those individuals who consume self-caught fish from the Hudson at least once per year, in the absence of a fishing ban or fish consumption advisories. The start date for the assessment is 1999, which is appropriate because the risk assessment evaluates current and future cancer risks and non-cancer health hazards. Due to the observed trend of decreasing concentrations of PCBs with time, individuals born or moving to the study area (Upper Hudson) after 1999 would be have less exposure to PCBs than the current angler population, so USEPA's approach is appropriately protective of human health. Although this population includes anglers who have been fishing for a long period of time, as well as anglers who may have just started fishing, only exposures occurring in 1999 and later were quantified in the risk assessment. The angler population could have alternatively been defined as the subset of anglers who *began* fishing in 1999 (or recently). During the development of the Monte Carlo analysis, intake was modeled both ways. The results were comparable for both the angler population fishing in the Upper Hudson River in 1999, as well as the subset of anglers who were assumed to *begin* fishing in 1999. Based on the similarity of the two analyses, only a single angler population, based on the full set of data from Connelly *et al.* (1992), was used for the ED analysis.

Cancer risks and non-cancer health hazards to individuals who move into, or are born into the area after 1999 were not quantitatively evaluated in the risk assessment. Similarly, those individuals consuming Upper Hudson River fish caught by a friend or family member or received as a gift were also not quantitatively evaluated. There is little quantitative information available on such exposures. Nonetheless, the cancer risks and non-cancer health hazards for these individuals are expected to be less than the risks for the angler population as defined, because friends and family members of anglers would be expected to have lower fish consumption rates than the angler population evaluated in this risk assessment.

PCB Exposure Concentration in Fish. During Phase 2 of the Reassessment RI/FS, USEPA has expended considerable effort to characterize current and future PCB concentrations in fish. Despite the extensive amount of information developed, there is still some uncertainty in the exposure point PCB

concentrations in fish used in the risk assessment. The source of PCB concentrations in fish is the model forecasts presented in the RBMR (USEPA, 2000a), which was peer reviewed. That report provided information about the variability of predicted PCB concentrations in future years within each modeled fish species. Although there are uncertainties inherent in the modeling approaches (see USEPA, 2000a), there is insufficient quantitative information available about the magnitude of the uncertainties to give a quantitative range of cancer risks and non-cancer health hazards attributable to model uncertainty. Based on the ability of the fish bioaccumulation models to capture the historical observed lipid-normalized PCB measurements in fish, the model uncertainty in PCB projections in fish is not expected to be sufficient to alter the overall conclusions in this risk assessment. Furthermore, the sensitivity/uncertainty analysis conducted for the Monte Carlo analysis provides a measure of the range of exposure and risks as a function of two important factors influencing the exposure point concentration: variations in the fish species caught (different species tend to have different characteristic PCB uptake), and variations in fishing location (the concentration trends decline substantially between the upper and lower reaches of the Upper Hudson River).

Sources of uncertainty in the PCB concentrations in fish used in the assessment include the fact that concentrations were averaged over location, and weighted by species. The weighting of species intake in order to derive an average EPC in fish is a source of uncertainty because there are limited site-specific data available to estimate the species ingestion preferences (*e.g.,* weighting factors). This uncertainty is unavoidable because the angler surveys in the Upper Hudson River (Barclay, 1993 and NYSDOH, 1999b, see pp. 39-40) could not be used to quantify fish consumption by species because the fish consumption advisories that are in place do not represent the baseline conditions considered in the Revised HHRA. The adjustments made to the 1991 New York Angler survey (Connelly *et al.*, 1992) data, such as excluding the "other" category, which may include fish species found in the Upper Hudson, and excluding fish species not found in the Upper Hudson, as well as extrapolating the percent of all fish in flowing water bodies to percent of Hudson species (Table 3-3) also introduces uncertainty in the EPC estimates. While there is some uncertainty associated with grouping three fish species that were not modeled to estimate their PCB concentrations (*i.e.*, walleye, carp, and eel), these fish represent only 9%, 6%, and 2% of the total fish intake, respectively (see Table 3-4). It was deemed preferable to include a more robust amount of information on fish consumption patterns, rather than limit the estimates of species weighting factors to the consumption of only the three modeled species (brown bullhead, bass, and perch).

The fact that there are no modeled future concentrations of PCBs in walleye, carp, eel, and possibly other fish that are consumed from the Upper Hudson is a source of uncertainty that is unavoidable. There are insufficient data to calibrate models of all species. The approach used to approximate concentrations of PCBs in carp, eel, and walleye by grouping them with types of fish for which modeled concentrations are available may lead to either under- or over-estimates of PCB intake *via* fish consumption. The magnitude of the uncertainty is difficult to quantify. For example, if concentrations of PCBs in carp and eel are generally higher than in brown bullhead (Group 1), then the cancer risks and non-cancer health hazards from the intake of these species (carp, 6% and eel, 2%, see Table 3-4) would be higher than calculated. However, based on the relatively low intake percentages reported for carp and eel, the total cancer risks and non-cancer health hazards from ingesting these species are not expected to be substantially greater than those calculated. Conversely, if PCB concentrations in fish not modeled are lower on average than fish actually consumed, the cancer risks and non-cancer health hazards would be correspondingly lower than calculated.

While it is likely that different anglers fish in different locations of the Upper Hudson River there is little information available to quantify these differences, and the presence of current fishing restrictions preclude gathering such information. Nonetheless, an analysis of the risks associated with a possible

population of anglers who fish predominantly in particular stretches in the Upper Hudson River is presented below.

Comparison of Risks and Non-Cancer HI Values At Specific Locations within the Upper Hudson River				
Upper Hudson River Location	**Non-cancer HI (young child)**		**Total Cancer Risk**	
	Central Tendency (CT)	High-End (RME)	Central Tendency (CT)	High-End (RME)
Entire Upper Hudson River – averaged over all 3 river segments	12	104	2.9×10^{-5}	1.4×10^{-3}
Thompson Island Pool (RM 189)	20	183	5.0×10^{-5}	2.5×10^{-3}
Stillwater (RM 168)	10	89	2.5×10^{-5}	1.1×10^{-3}
Federal Dam (RM 154)	5	41	1.1×10^{-5}	5.0×10^{-4}

Note: Exposure factors for these calculations are in Tables 2-12a,b,c; EPCs are given in Tables 2-6, 2-7, 2-8.

As this comparison shows, the cancer risks and non-cancer health hazards are highest at the furthest point upstream (Thompson Island Pool), approximately 2-fold higher than the central tendency and RME scenarios presented in this Revised HHRA, and decrease with river mile.

In addition, a sensitivity analysis of the cancer risks and non-cancer health hazards associated with a possible population of anglers who fish only in the upstream areas of the Upper Hudson River study area, where PCB concentrations in fish are the highest, as well as those who may fish predominantly in the lower reaches where PCB concentrations are the lowest, was also examined in the Monte Carlo analysis (see Section 5.3.3). Fish species-specific consumption frequencies were estimated based on the 1991 New York Angler survey (Connelly *et al*., 1992) from which 226 angler responses report consuming self-caught fish. The variability of fish consumption preference was modeled in the Monte Carlo analysis based on the range of species consumption patterns reflected in that survey.

Fish Ingestion Rate. The primary source used to derive the distribution of fish ingestion for the risk assessment was the 1991 New York Angler survey (Connelly *et al.*, 1992). There are many uncertainties inherent in the fish ingestion rate assumptions used in the risk assessment, the most significant of which are discussed below. Despite these uncertainties, the assumptions regarding fish consumption are believed to be reasonable and health protective; the peer reviewers indicated in their report that the fish ingestion rates used were reasonable (ERG, 2000). The sensitivity analysis conducted for this parameter provides a measure of the range of exposures using several alternative sources of information regarding sportfish ingestion.

As stated at the outset, the intent of the Revised HHRA was to evaluate cancer risks and non-cancer health hazards for Upper Hudson River anglers in the absence of remediation or institutional controls, such as the current Hudson-specific fish consumption advisories. Because there *are* current advisories to eat no fish from the Upper Hudson River, it is not possible to collect site-specific information about the consumption of fish caught in the Upper Hudson River in the absence of fish consumption advisories. Therefore, it was necessary to select fish ingestion rates from sources other than surveys of the Hudson River. There is some uncertainty as to whether data from flowing waterbodies from the 1991 New York Angler survey (Connelly *et al*., 1992) accurately represents Upper Hudson River anglers. Although the fish ingestion rates reported in the New York Angler survey are presumably influenced by general, non site-specific NYSDEC fishing regulations (that would be in effect regardless

of PCB contamination levels in the Hudson), because the survey was state-wide, it is not likely to be unduly affected by the Hudson-specific fish consumption advisories, and thus considered to be a reasonable surrogate for the Upper Hudson.

Of the available studies of sportfish ingestion, the 1991 New York Angler survey (Connelly *et al*., 1992) is considered the preferred study to represent Upper Hudson River anglers because, among other reasons outlined in this report, it was conducted in New York and included a large sample size. Other New York waterbodies are likely to be more similar to the Hudson River than waterbodies in other states. The fact that the fish ingestion rates from the 1991 New York Angler survey are reasonably consistent with the results of published studies investigating freshwater fish ingestion rates from other locations in the U.S. lends an additional degree of confidence in the use of the 1991 New York Angler survey data.

Cancer risks and non-cancer health hazards were not specifically quantified for subsistence anglers, unlicensed anglers, or other subpopulations of anglers who may be highly exposed. Although there are no known, distinct subpopulations that may be highly exposed, there is some degree of uncertainty as to whether these subpopulations have been adequately addressed in this risk assessment. However, as discussed in Section 3.2.1.4, based on consideration of fish ingestion rates among low income families (Wendt, 1986), fish ingestion rates reported for licensed and non-licensed anglers from the Hudson River angler surveys (Barclay, 1993; NYSDOH, 1999b), and fish ingestion rates for angler populations in other areas of the country (see Table 3-2), it appears likely that any highly exposed subpopulations are represented within the upper percentiles of the fish ingestion rate distribution used in the Monte Carlo analysis.

The consumption rate chosen is assumed to remain the same from year to year; this approach assumes that fish ingestion rates are perfectly correlated each year. Actual year to year ingestion rates are probably correlated to a high degree, but not perfectly (100%). The assumption of similar year-to-year consumption patterns is supported by the finding that when classified as either low or high avidity (in relation to the median fishing effort), two-thirds of Lake Ontario anglers were classified the same in 1991 and 1992 (Connelly and Brown, 1995). Assuming there is no correlation between yearly ingestion rates would effectively average high-end consumers out of the analysis, and would clearly be inappropriate. Thus, although there are no data available to quantify the correlation between yearly ingestion rates, the approach taken in the risk assessment is reasonable and protective of human health.

While some anglers may consume fish at frequencies less than once per year and some friends or family members of anglers may consume "gift fish" at infrequent intervals, data are insufficient to quantify the fish ingestion rates for these individuals. Nonetheless, consideration of only those anglers who consume self-caught fish from the Hudson at least once per year is protective of human health, because exposure to less frequent anglers, family members, or friends would be lower than the exposure calculated for the angler population.

Fraction from Source. For this assessment, it is assumed that 100% of the sportfish caught and consumed is from the Upper Hudson River. Given the 40-mile extent of the Upper Hudson River and the variety of fish species it can support, a sizeable recreational angler population is considered likely to catch a substantial fraction of their fish from the Upper Hudson (in the absence of fishing restrictions). As noted before, the fish ingestion rate is based upon consumption of sportfish, excluding fish that may be purchased and then consumed. While it is possible that less than 100% of sportfish ingested is from the Upper Hudson, adopting a lower fraction from source would not necessarily change the results significantly. For example, assuming a fraction from source of 0.5 (*i.e.*, 50%) and a 95th percentile ingestion rate (63.4 grams/day) from the 1991 New York Angler survey would result in the same intake

as assuming a 100% fraction from source and a 90th percentile ingestion rate (31.9 grams/day) from the 1991 New York Angler survey.

Angler Exposure Duration. The distribution of angler exposure durations developed for use in the Monte Carlo assessment represents variability among anglers. The uncertainties inherent in developing the exposure duration of anglers were described in Section 3.2.4. For example, it was assumed that the age profile of the angler population remains unchanged over time, and that 1991 angler data is representative of 1999 anglers. Insufficient information is available to evaluate these sources of uncertainty quantitatively. Nonetheless, the resulting point estimates (*e.g.*, a central tendency estimate of 12 years, and an RME estimate of 40 years) are unlikely to underestimate actual exposure durations significantly, and they are not substantially different from the exposure duration values recommended in the USEPA Exposure Factor's Handbook (USEPA, 1997f), which are based on national population mobility statistics.

*PCB Cooking Losses. A*s described in Section 3.2.3, reported cooking losses vary considerably among the numerous studies reviewed. Yet, there is little information available to quantify personal preferences among anglers for various preparation and cooking methods and other related habits (such as consumption of pan drippings). The assumption that there is no loss of PCBs during cooking or preparation, used in the RME point estimate cancer risk and non-cancer health hazard calculations, is conservative, and could overestimate cancer risks and non-cancer health hazards on average. Cooking losses ranging from 0% to 40% were explicitly evaluated in the Monte Carlo analysis.

Sediment Ingestion Rate. There is considerable debate in the scientific community regarding soil ingestion, and work is ongoing to better characterize soil ingestion rates. The soil ingestion rate exposure factor represents *total daily* intake of soil integrated over a variety of activities, including ingestion from both outdoor and indoor sources. In this Revised HHRA, a median ingestion rate (as opposed to a high-end rate) was used for recreational exposures, because the total exposure time is only a fraction of the total day. The median ingestion rates used are likely high-end estimates of incidental sediment ingestion while participating in activities along the Hudson, because other sources (such as at home) also account for soil/sediment ingestion. On the other hand, increased dermal adherence of (wet) sediment compared to (dry) soil could correspond to higher actual ingestion rates for sediment compared to soil.

Sediment/skin adherence factor. This factor represents the amount of sediment that adheres to skin and is available for dermal exposure. Because this value is likely to vary based on one's activity, the values used for this parameter, which are estimates from single activities, are somewhat uncertain. For dermal contact with Upper Hudson River sediments, published adherence factors for adults gathering reeds, and for children playing in wet soils, were used as a surrogate for children. Although it is somewhat uncertain whether these scenarios are representative of contact with Hudson sediments, they appear to be a reasonable use of available data.

Dermal Absorption Value. The PCB dermal absorption rate used in this risk assessment was based on a value published in peer-reviewed literature. Nonetheless, dermal absorption of soil and sediment contaminants is a complicated issue, and there is considerable uncertainty associated with dermal absorption rates. Various factors affect the efficiency of dermal absorption. For example, many compounds are only absorbed through the skin after a long exposure duration (*i.e.*, >24 hours). Since most individuals bathe at least once each day, washing may remove any soil residues adhering to the skin before absorption can occur. Therefore, dermal absorption rates based on studies with long exposure durations tend to overestimate actual absorption. However, soil loadings have also been shown to affect dermal absorption rates; the percentage of dermal absorption may increase as soil loadings decrease. The use of various testing methods also introduces uncertainties; *in vivo* animal studies introduce uncertainties

regarding animal-to-human extrapolation, while *in vitro* studies using human skin introduce uncertainties regarding *in vitro* to *in vivo* extrapolations. Despite these uncertainties, the published dermal absorption values used in this risk assessment provide a reasonable basis to estimate risks for the dermal pathway.

PCB Concentrations in Air. The PCB concentrations in air used in this risk assessment are particularly uncertain. The risk analysis for this pathway should therefore be considered to be a "screening level" analysis. Measurements of PCBs in air in 1991, adjusted to reflect the lower PCB concentrations in the water column at present and predicted into the future, provided one estimate for the exposure point concentration. These measurements were compared with modeled PCB volatilization and dispersion estimates. The two estimation methods provided a very wide range of concentration estimates. Despite the wide range of results, the results of the analysis indicate the volatilization of PCBs from the river is likely to yield *de minimus* human health cancer risks.

Analysis of non-cancer health hazards could not be calculated based on the lack of a toxicity value. However, based on the calculated cancer risks from inhalation of volatilized PCBs, it is not anticipated that this will be a significant non-cancer health hazard.

5.3.2 Toxicity Assessment

The toxicity values used in this risk assessment have been peer reviewed and are the most current values recommended by USEPA in IRIS. As stated in USEPA's reassessment of PCB cancer toxicity, the uncertainty around the CSF estimates extends in both directions, *i.e.*, contributing to possible underestimate or overestimate of cancer potency factors. However, the CSFs developed by the USEPA represent plausible upper bound estimates, which means that the USEPA is reasonably confident that the actual cancer risk will not exceed the estimated risk calculated using the CSF (USEPA, 1986; 1996c).

The current PCB CSF was derived using health protective dose-response models and observed liver tumor rates in rodents, and generates theoretical, upper-bound cancer risk estimates. True cancer risk is likely to be lower and could even be zero (USEPA, 1986). The published occupational and population studies (including the recent Kimbrough *et al.,* [1999] study) indicate both positive and negative causal relationships between PCB exposure and cancer. There are a number of limitations with these studies, including lack of sufficient exposure information, failure to adequately account for co-exposure to other compounds, questions about the appropriateness of the control populations, and inconsistency between studies.

The USEPA used uncertainty factors of 100 and 300, respectively, in deriving the RfDs for Aroclor 1016 and 1254 used in the non-cancer assessment. The RFDs for Aroclors 1254 and 1016 used in the Revised HHRA are currently being evaluated as part of the IRIS reassessment process and it would be inappropriate to prejudge the results of the reassessment at this time. As discussed in Section 4.4.2 and Appendix D, a number of recent national and international studies have reported possible associations between developmental and neurotoxic effects in children from pre-natal or post-natal exposures to PCBs. Although the results from the various studies are not entirely consistent and there are limitations in the study designs, the fact that similar neurobehavioral and learning problems have been observed in animals provides additional support for the findings in humans. Uncertainties with respect to potential developmental and neurotoxic effects are compounded by the fact that the most appropriate averaging time for pregnant mothers and nursing infants is not certain, but could be significantly shorter than the averaging times used in the Revised HHRA, considering the various aspects of PCB pharmacokinetics, and the fact that critical windows of exposure for fetuses could be quite short. This information will be evaluated by USEPA during the non-cancer health toxicity reassessment and it is premature to conclude whether there will be any changes in the current RFDs for PCBs.

The toxicity values (CSFs and RFDs) used in the risk assessment are protective of both males and females. For example, the CSF used in calculating cancer risks is based on an increased incidence of liver tumors in female rats reflecting the potential sensitivity of this gender. The CSF generated based on female rats was higher than that generated for tumors found in male rats. Because cancer risk is a function of exposure and toxicity, the use of the higher slope factor based on data from the female rats is more protective of the general population than using the lower slope factor identified for male rats. Although this is a potential source of uncertainty, it is unlikely to have a significant impact on cancer risks in either direction.

Commercial PCB mixtures tested in laboratory animals were not subject to prior selective retention of persistent congeners through the food chain (such as those found in the Hudson River, and thus there is a potential that cancer risks and non-cancer health hazards have been underestimated. However, since the CSFs are based on animal exposures to a group of PCB mixtures (*i.e.*, Aroclor 1260, 1254, 1242, and 1016) that contain overlapping groups of congeners spanning the range of congeners most often found in environmental mixtures, this source of potential uncertainty is unlikely to have a significant impact.

The fact that any previous exposures (either background, or past consumption of PCB-contaminated fish) may still be reflected in an individual's body burden today is an additional source of uncertainty, and may result in an underestimate of non-cancer health hazards.

Toxic Equivalence Factors (TEFs) for Dioxin-Like PCBs. There is considerable uncertainty regarding the TEF values for the toxicity of dioxin-like PCB congeners. In their publications, WHO indicates that their TEF values represent "*an order of magnitude estimate of the toxicity of a compound relative to TCDD*" (emphasis added) (Van den Berg *et al*., 1998). Also, the TEF analysis assumes that the toxic effects of dioxin-like PCBs are additive. However, this assumption is somewhat uncertain. As discussed in the WHO/ICPS TEF reviews (Ahlborg *et al.,* 1994; Van den Berg *et al.*, 1998), although there is evidence of additivity for Ah receptor mediated responses, interactions between nondioxin-like PCBs and dioxin-like PCBs may be antagonistic, in which case the assumption of additivity is highly conservative. However, evidence of synergistic interactions between PCBs and dioxin also exists (at high concentration levels). It is also important to note that many nondioxin-like PCB congeners have independent mechanisms of toxicity (Hansen, 1998). Although the toxicity of these congeners is likely to be reflected in the toxicity values developed for Total PCBs, the toxicity of each PCB congener has not been fully characterized, and TEF values have not been developed for non-dioxin like congeners.

Research into possible endocrine effects of PCBs is an area of active research to develop toxicological tests to evaluate possible endocrine disruption. Although PCBs may also act as an environmental endocrine disruptor, the available data are insufficient to support a quantitative assessment of endocrine effects in this risk assessment. As discussed in Section 4.4, it is recognized that this is a source of potential uncertainty. Many of the standard toxicity tests performed to date on PCBs were not specifically designed to identify effects of endocrine disruption, and some health endpoints could have been missed by those studies. However, the USEPA Risk Assessment Forum Technical Panel concluded, based on available evidence, that exposure to xenoestrogenic chemicals, at current environmental concentrations, is probably insufficient to evoke an adverse effect in adults (USEPA, 1997b). Additional information is required to understand the mechanism by which the endocrine effects are acting, and to determine if this holds for the human fetus and neonate. Nonetheless, because the IRIS values for PCB cancer risks and non-cancer health hazards do not directly incorporate concerns for endocrine effects, it is possible that cancer risks and non-cancer health hazards in this risk assessment may have been underestimated.

Potential sources of uncertainty in the toxicity assessment are summarized below. The potential sources of uncertainty in the cancer slope factor are directly quoted from the 1996 USEPA Cancer Reassessment for PCBs (USEPA, 1996c). The sources of uncertainty in the non-cancer RfD for Aroclor 1254 are based on the USEPA IRIS file for Aroclor 1254 (USEPA, 1999b).

Summary of Potential Sources of Uncertainty in the Cancer Slope Factor (USEPA, 1996c)

Cancer experimental design and conduct. The rat study (Brunner *et al.*, 1996) is quite extensive in design and conduct, going beyond standard designs for cancer studies in many respects.

Variability in commercial mixture composition. For the four Aroclors tested in female Sprague-Dawley rats (Brunner *et al.*, 1996 and Norback and Weltman, 1985), there is a 30 fold range in potency. This whole range is used to represent environmental mixtures.

Variability across strains. In the four rat strains tested, sensitivity varies up to 15-fold. Potency and cancer slope estimates were derived from a strain covering the middle of this range.

Variability between sexes. Potency and slope estimates were derived from female rats, whose liver response was usually greater than that of males. The greatest response in the liver, however, was in male rats. Greater sensitivity of females was not seen in mice, nor in the thyroid.

Variability across experiments. For the same Aroclor, sex and strain, differences up to four-fold were observed. To reflect this lot-to-lot variability, both estimates were included.

Experimental uncertainty (sample size). Central and upper-bound potency estimates differ by no more than about two-fold. This is a minor source of uncertainty.

Animal to human extrapolation. The use of default cross-species scaling is intended as an unbiased projection not expected to provide conservatism (USEPA, 1992e). Information is lacking to evaluate whether humans are more or less sensitive than rats.

High-to-low dose extrapolation. The use of models that are linear at low doses can potentially overestimate potency by an unknown amount. The rat studies, however, show no evidence of sublinearity in the experimental range.

Route-to-route extrapolation. Information on relative absorption rates suggests that differences in toxicity across exposure routes are small.

Difference between commercial and environmental mixtures. Commercial mixtures released into the environment are altered by environmental processes. Qualitatively, exposure pathway is a reasonably good indicator of whether potency has been decreased or increased. Quantitatively, the percentage change in toxicity is unknown, though the 30-fold range in potency observed for commercial mixtures likely underestimates the range of environmental mixtures.

Persistence and exposure duration. Some PCBs persist in the body and retain biological activity after exposure stops (Anderson *et al.*, 1991). Compared with the current default practice of assuming that less-than-lifetime effects are proportional to exposure duration, rats exposed to the persistent mixture Aroclor 1260 had more tumors while rats exposed to the less persistent Aroclor 1016 had fewer tumors (Brunner *et al.*, 1996). Thus the current default practice can underestimate risks for persistent mixtures.

Human variability in sensitivity. People with decreased liver function can have less capacity to metabolize and eliminate PCBs. Approximately 5% of nursing infants receive a steroid in human milk that further inhibits PCB metabolism and elimination (Calabrese and Sorenson, 1977).

Human variability in exposure. Blood concentrations vary over a 100-fold range (ATSDR, 1993). Highly exposed populations include nursing infants, consumers of game animals contaminated through the food chain, and workers with occupational exposure. There is greater confidence in risk estimates for highly exposed groups.

Summary of Potential Sources of Uncertainty in the Non-Cancer RfD for Aroclor 1254

Selection of most appropriate toxicity study for RfD derivation. USEPA evaluated a wide range of studies in various animal species and an appropriate endpoint was selected.

Selection of most appropriate averaging times. The critical study was subchronic and appropriate uncertainty factors were applied to account for uncertainties in address the exposure duration.

Weight of evidence for developmental/neurotoxic effects. Studies in animals including rhesus monkeys, offspring of female rhesus monkeys exposed *in utero*, and longitudinal studies of children born to mothers who consumed PCB contaminated fish while pregnant were evaluated in the assessment.

Protection for both genders. Data for female and male reproductive function and developmental data in a nonhuman primate species were evaluated in this assessment.

Extrapolation from commercial PCB mixtures to environmental PCB mixtures. See discussion above regarding the environmental changes of PCB mixtures. The use of the current default practice of using commercial mixtures for testing may potentially underestimate non-cancer health hazards for persistent mixtures.

Additional Considerations Relating to Potential Sources of Uncertainty in the Toxicity Assessment

TEFs for dioxin-like PCBs. The TEFs identified by the WHO in 1998 were used in the analysis. This may either under- or overestimate the cancer risks from dioxin exposure based on potential overlaps; however, no attempt was made to combine these risks from dioxin-like and non-dioxin like PCBs.

Weight of evidence for endocrine effects. This is an area of ongoing research and the impacts may either over- or underestimate cancer risks and non-cancer health effects based on on-going research to understand the Mode of Action for endocrine effects.

Previous PCB exposures. This may potentially underestimate the risks for persistent mixtures.

Human epidemiology studies. Published occupational and population studies indicate both positive and negative causal relationships between PCB exposure and cancer and the studies suffer from inherent problems in determining accurate estimates of exposure along with other shortcomings as discussed above and also in Appendix D.

5.3.3 Comparison of Point Estimate RME and Monte Carlo Results for Fish Ingestion

Each of the uncertainties associated with the Exposure and Toxicity Assessment steps in the risk assessment process becomes incorporated into the risk estimates in the Risk Characterization step. As noted earlier, the combination of high-end and average exposure assumptions incorporated into the point

estimates for the RME yields risk estimates that may overestimate actual site risks. A comparison of the central tendency and RME point estimate risks for fish ingestion, with the Monte Carlo estimates, provides a perspective on the variability and uncertainty in the range of risks possible for this pathway under a wide range of scenarios.

A sensitivity/uncertainty analysis consisting of 72 combinations of the important exposure variables for the fish ingestion pathway was performed for the Monte Carlo analysis. A comparison of the base case Monte Carlo results with the point estimate results was presented in Section 5.2. As that comparison showed, the RME cancer risk estimate (1.4×10^{-3}), falls somewhat above the 95th percentile of the base case Monte Carlo distribution of risk. It should be noted that because the peer reviewers did not recommend any changes to the important exposure factors for the fish ingestion pathway, and due to the extensive level of effort required to perform the Monte Carlo analysis, USEPA did not conduct another Monte Carlo analysis for the Revised HHRA. However, as indicated in the March 2000 Responsiveness Summary for the HHRA (USEPA, 2000b), the modest changes in modeled forecasts of PCBs in fish in the RBMR (USEPA, 2000a) as compared to those in the 1999 Baseline Modeling Report (USEPA, 1999d) indicate that any changes in the Monte Carlo results would be correspondingly modest (less than 2-fold).

Tables 5-38 and 5-39 provide a summary of the point estimate HI and cancer risk estimates together with the full range of Monte Carlo estimates. Figures 5-3a and 5-3b plot percentiles for all 72 combinations of the non-cancer HI values and the cancer risks, respectively. In each of these figures, the variability of cancer risk or non-cancer HIs for anglers within the exposed population is plotted on the y-axis for particular percentiles within the population. This variability is a function of variations in fish consumption rates, fishing duration, differences in fish species ingested, *etc.* The uncertainty in the estimates is indicated by the range of either cancer risk or non-cancer HI values plotted on the x-axis. This uncertainty is a function of the 72 combinations of the exposure factor inputs examined in the sensitivity analysis. This analysis provides a semi-quantitative confidence interval for the cancer risk and HI values at any particular percentile. As these figures show, the intervals span somewhat less than two orders of magnitude (*e.g.*, <100-fold).

The central (50th percentile) Monte Carlo HI ranges from a low of 1.8, to a high of 51.5, compared to the CT point estimates of 12 (young child), 8 (adolescent) and 7 (adult). The high-end (95th percentile) Monte Carlo HI ranges from 18.6 to 366, compared to the RME point estimate of 104 (young child), 71 (adolescent) and 65 (adult). A similar comparison for cancer risk indicates the 50th percentile cancer risk estimates range from 9.7×10^{-6} to 4.1×10^{-4}, compared to a CT point estimate of 2.9×10^{-5}. The 95th percentile Monte Carlo cancer risk estimates range from 1.1×10^{-4} to 3.1×10^{-3}, compared to the RME point estimate of 1.4×10^{-3}. A discussion of the sensitivity of the Monte Carlo results as a function of several important exposure factors follows.

Uncertainty in Fishing Locations. For the base case Monte Carlo analysis, and the point estimate analysis, PCB concentrations in fish were averaged over the three locations modeled: Thompson Island Pool (River Mile 189), Stillwater (River Mile 168), and the Federal Dam area (River Mile 154). However, it is possible that an angler would preferentially fish in a single location. To address this possibility, the Monte Carlo analysis considered catching and consuming fish from the most contaminated and least contaminated locations.

As both the historical data and modeling results indicate, the PCB concentration in fish in the Upper Hudson River exhibits a declining concentration from upstream to downstream locations. Of the three locations modeled, Thompson Island Pool had the highest modeled PCB concentrations in fish. Holding all other exposure factors at their base case values, while assuming an angler catches and

consumes fish exclusively from the upstream areas of the Upper Hudson River (using the Thompson Island Pool as a surrogate), yields estimates of non-cancer hazard and cancer risk:

Sensitivity Analysis-Fishing Location			
Outcome	**Point Estimate[a]**	**Base Case Monte Carlo**	**High- End PCB Concentration (Thompson Is. Pool) - Monte Carlo[b]**
Non-Cancer HI	*young child / adolescent / adult*		
Central Tendency (CT)	12 / 8 / 7	11	19
High-End (RME)	104 / 71 / 65	137	226
Total Cancer Risk			
Central Tendency (CT)	2.9×10^{-5}	6.4×10^{-5}	1.0×10^{-4}
High-End (RME)	1.4×10^{-3}	8.7×10^{-4}	1.5×10^{-3}
[a]Point Estimate values based on exposure factors in Tables 2-12a,b,c.			
[b]Refer to Run #4 in Appendix C.			
Base case Monte Carlo = 50th percentile; High-End Monte Carlo = 95th percentile.			

As this comparison shows, the Monte Carlo HI and cancer risk increase by approximately 1.7-fold over their corresponding base case values for this scenario. This ratio is slightly smaller than the point estimate weighted PCB concentrations.

Fish Ingestion Rate. The point estimate and base case Monte Carlo analysis used the 1991 New York Angler survey as the basis for fish ingestion rates. As described in Chapter 3, the New York Angler survey yielded higher estimates of fish ingestion than a number of other studies. The 1992 Maine Angler survey (Ebert *et al.*, 1993) yields the lowest estimate of fish ingestion of the studies examined. An examination of the non-cancer health hazards and cancer risk using the Maine fish ingestion rates yields the following:

Sensitivity Analysis-Fish Ingestion Rate			
Outcome	**Point Estimate[a]**	**Base Case Monte Carlo**	**Using Maine Angler Study Fish Ingestion - Monte Carlo[b]**
Non-Cancer HI	*young child / adolescent / adult*		
Central Tendency (CT)	12 / 8 / 7	11	6
High-End (RME)	104 / 71 / 65	137	85
Total Cancer Risk			
Central Tendency (CT)	2.9×10^{-5}	6.4×10^{-5}	3.4×10^{-5}
High-End (RME)	1.4×10^{-3}	8.7×10^{-4}	5.2×10^{-4}
[a]Point Estimate values based on exposure factors in Tables 2-12a,b,c.			
[b]Refer to Run #28 in Appendix C.			
Base case Monte Carlo = 50th percentile; High-End Monte Carlo = 95th percentile.			

As this comparison shows, the Monte Carlo HI and cancer risk decrease by approximately 2-fold over their corresponding base case values for this scenario. This comparison indicates that adopting a lower estimate of the fish ingestion rate compared with the base case estimate does not change the results significantly.

Exposure Duration. The point estimate and base case Monte Carlo analysis defined exposure duration based on the joint distribution of residence duration and fishing duration. As a sensitivity analysis, residence duration alone was used to examine the non-cancer health hazards and cancer risk under this scenario:

Sensitivity Analysis-Exposure Duration			
Outcome	**Point Estimate[a]**	**Base Case Monte Carlo**	**Exposure Duration based on Residence Duration Only - Monte Carlo[b]**
Non-Cancer HI	*young child / adolescent / adult*		
Central Tendency (CT)	12 / 8 / 7	11	14
High-End (RME)	104 / 71 / 65	137	163
Total Cancer Risk			
Central Tendency (CT)	2.9×10^{-5}	6.4×10^{-5}	1.1×10^{-4}
High-End (RME)	1.4×10^{-3}	8.7×10^{-4}	1.4×10^{-3}

[a]Point Estimate values based on exposure factors in Tables 2-12a,b,c.

[b]Refer to Run #37 in Appendix C.

Base case Monte Carlo = 50th percentile; High-End Monte Carlo = 95th percentile.

As this comparison shows, the Monte Carlo HI increases by approximately 1.2-fold, and the cancer risk increases by approximately 1.6-fold over their corresponding base case values for this scenario. This comparison indicates that adopting a higher estimate of the exposure duration than the base case estimate does not change the results significantly.

Population Risks. Consistent with USEPA's Superfund guidance, this risk assessment does not estimate the number of anglers that consume their catch or the number of women of child-bearing age exposed through consumption of fish because CERCLA requires consideration of risk to an individual with a reasonable maximum exposure. It would be difficult to identify the number of anglers who are consuming fish in the presence of fishing bans and fish consumption advisories, because of the potential for underreporting and the threat of fines for anglers keeping fish from the Upper Hudson River. It is also not possible to project with any certainty the number of potential anglers within various stretches of the river who would consume fish if there were no health advisories in the Upper Hudson River.

5.3.4 Sediment Concentration Variability and Uncertainty

Because the fish ingestion pathway yields the greatest potential PCB intake, it has received the most attention in terms of the analysis of variability and uncertainty using Monte Carlo analysis methods. Clearly, sediment contact and other potential exposure pathways are affected similarly by variable and uncertain factors. In this section, the magnitude of several factors affecting PCB exposures from sediment are examined. This sensitivity analysis is not based on Monte Carlo analysis methods.

For the sediment exposure pathway, the central tendency and RME risks and non-cancer health hazards were calculated based on the assumption of exposure to sediments throughout the Upper Hudson River. That is, the average modeled PCB concentration over the entire stretch was used for the exposure point concentration (averaged over the exposure durations for recreators). However, just as with fish, the PCB concentration shows a marked trend from up-river to down-river locations, with the highest concentrations in the up-river stretches (*e.g.*, RM 189) and much lower concentrations in general in the down-river stretches (*e.g.*, RM 154). This trend is plotted in Figure 2-5. As shown in this figure, the average concentration over all reaches (which was used for the EPCs in our analysis), lies between the highest concentrations predicted for RM 189, and the concentrations predicted for RM 154 (those for RM 168 are not significantly different than the predictions for RM 154).

In the comparison below, the cancer risks and non-cancer HI values are compared for the scenario presented in this Revised HHRA, with several sensitivity calculations using the avid recreator scenario. If an avid recreator is exposed exclusively in areas near the Thompson Island Pool (RM 189), the risks and non-cancer HI values increase 2.5-fold over their respective values when calculated for the entire Upper Hudson. Conversely, for avid recreators who are exposed to sediments in the lowest stretches of the Upper Hudson River (*e.g.*, RM 154), the cancer risks and non-cancer HI values decrease by approximately 5-fold as compared to the values calculated based on the average over the entire Upper Hudson River. Thus, leaving aside model uncertainty, there is an approximate 10-fold range in cancer risks and non-cancer HI values due to exposure variability within the Upper Hudson River.

The possible effect of model uncertainty on the future forecasts of PCB concentrations in surficial sediment was also examined. The RBMR model forecasts are based on river segments, which do not distinguish between nearshore and mid-stream zone within the river. In order to examine available information targeted at nearshore environments, which would be those where contact with sediment would be most prevalent, existing monitoring data for such areas were examined.

As reported in the July 1998 Low Resolution Sediment Coring (LRC) report (USEPA, 1998c), the 95% UCLM of 11 near-shore samples collected in 1994 is 151 mg/kg. Using this value as a starting point, and applying a half-life of 14 years (estimated from the 40% PCB loss in the 10-year period from 1984 to 1994 as reported in the LRC), the average concentration was estimated over time from 1999 and continuing through the appropriate exposure durations for children (6 years), adolescents (12 years) and adults (23 years). In this manner, the following average concentrations for nearshore sediments in the Thompson Island Pool were estimated, taking 1999 as the starting point:

6-year average (children)	97 mg/kg
12-year average (adolescents)	88 mg/kg
23-year average (adults)	67 mg/kg

The cancer risks and non-cancer HI values assuming an avid recreator exposure scenario are summarized below. The RME cancer risk is approximately 5-fold higher than the respective value calculated for the Thompson Island Pool location, and approximately 10-fold higher than the cancer risk for this exposure pathway when calculated over the entire Upper Hudson River. Similar, but somewhat smaller differences, are exhibited for the non-cancer HI values.

Comparison of Sediment Pathway Cancer Risks and Non-Cancer Hazards Avid Recreator Scenario				
Upper Hudson River Location	**Non-cancer HI (young child)**		**Total Cancer Risk**	
	Central Tendency (CT)	High-End (RME)	Central Tendency (CT)	High-End (RME)
Entire Upper Hudson River – averaged over all 3 river segments	0.2	0.3	1×10^{-6}	9×10^{-6}
Thompson Island Pool (RM 189)	0.5	0.75	2.5×10^{-6}	2.2×10^{-5}
Federal Dam (RM 154)	0.04	0.06	2.0×10^{-7}	1.8×10^{-7}
Thompson Island Pool (RM 189) Nearshore Sediments Using Low Resolution Coring Data[a]	--[b]	1.7	--	1×10^{-4}

Exposure factors for these calculations are in Tables 2-13b, 2-14b, 2-15b.

[a] The exposure point concentrations for adults (67 mg/kg), adolescent (88 mg/kg), and children (97 mg/kg) are based on averages over their respective exposure durations (see text).

[b] Not calculated

As this discussion indicates, the variability and uncertainty of the exposure point concentration estimates for sediment range up to as much as approximately 10-fold when both exposure location variability and model uncertainty are considered.

MAJOR FINDINGS OF THE REVISED HHRA FOR THE UPPER HUDSON

The Revised HHRA evaluated both cancer risks and non-cancer health hazards to young children, adolescents and adults posed by PCBs in the Upper and Mid-Hudson River. USEPA has classified PCBs as probable human carcinogens and known animal carcinogens. Other long-term adverse health effects of PCBs observed in laboratory animals include a reduced ability to fight infections, low birth weights, and learning problems. The major findings of the report are:

- Eating fish is the primary pathway for humans to be exposed to PCBs from the Upper Hudson.
- Under the RME scenario for eating fish from the Upper Hudson, the calculated total cancer risk (40 years of exposure apportioned as a young child, adolescent, and then adult) is 1×10^{-3}, or one in 1,000. This excess cancer risk is 1,000 times higher than USEPA's goal of protection and ten times higher than the highest cancer risk level allowed under the federal Superfund law.
- For non-cancer health effects, the RME scenario for eating fish from the Upper Hudson results in a Hazard Index to a young child of 104, a level of exposure to PCBs that is more than 100 times higher than USEPA's reference level (Hazard Index) of one. HIs for the adolescent and adult are 71 and 65, respectively, which are 71 and 65 times higher than the reference level of one.
- The RME cancer risks and non-cancer health hazards are expected to be above USEPA's generally acceptable levels for the 40-year exposure period that begins in 1999, assuming baseline conditions of no remediation and no institutional controls.

- The central tendency cancer risks and non-cancer health hazards from eating fish in the Upper Hudson, which are based on consumption of about one meal every two months, are also above USEPA's levels of concern.
- Cancer risks and non-cancer health hazards from being exposed to PCBs in the river through skin contact with contaminated sediments and river water, incidental ingestion of sediments and inhalation of PCBs in air are generally within or below USEPA's levels of concern.

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6 Mid-Hudson River Risk Assessment

6.1 Overview of Mid-Hudson River Risk Assessment

This chapter presents the baseline HHRA for the Mid-Hudson River and serves as a companion to the Upper Hudson River HHRA presented in the preceding chapters of this report. This final HHRA for the Mid-Hudson River updates the earlier December 1999 HHRA for the Mid-Hudson River (USEPA, 1999g), incorporating information from the August 2000 Responsiveness Summary (USEPA, 2000c). For ease of reference, and in order to minimize confusion with the Upper Hudson HHRA, all tables, figures, and plates referenced in this chapter are found in Appendix E.

6.1.1 Introduction

This assessment quantifies both carcinogenic and non-carcinogenic health effects from exposure to PCBs in the Mid-Hudson River, following USEPA risk assessment policies and guidance. Both current and future cancer risks and non-cancer health hazards to young children, adolescents, and adults were evaluated based on the assumption of no remediation or institutional controls, in accordance with the NCP (USEPA, 1990).

The risk assessment methodology for the Mid-Hudson River parallels the method adopted for the Upper Hudson River HHRA. Therefore, much of the background and details of the risk assessment process is found in the preceding chapters. As indicated earlier (Section 1.6), this Revised HHRA is limited to evaluating potential health risks associated with PCBs.

6.1.2 Site Background

The Hudson River PCBs Superfund Site extends from Hudson Falls, NY to the Battery in New York City. The site covers approximately 200 river miles. This chapter addresses the Mid-Hudson River (Appendix E, Plate 1), which is the area between the Federal Dam in Troy, NY (River Mile 153.5) and the salt water front (approximately River Mile 64).

Because of potential human health risks due to consumption of PCB-contaminated fish, NYSDEC and NYSDOH have made the following general recommendations: 1) Eat no more than one meal (1/2 pound) per week of fish from the Hudson River estuary; 2) Women of childbearing age, infants, and children under the age of 15 should not eat any fish species from the Hudson River; and 3) Follow trimming and cooking advice (NYSDEC, 1999). Additional advisories made specifically for the Hudson River include: 1) Hudson Falls to Troy Dam (Upper Hudson River) -- eat no species; 2) Troy Dam south to bridge at Catskill (Mid-Hudson River) -- eat no species, except American shad (one meal/week), and alewife, blueback herring, rock bass, and yellow perch (one meal/month); 3) Bridge at Catskill south to and including the Upper Bay of NY Harbor (Mid and Lower Hudson River) -- eat American eel, bluefish, striped bass, Atlantic needlefish, rainbow smelt, white perch, carp, goldfish, white catfish, largemouth bass, smallmouth bass, walleye, white catfish, and white perch only one meal/month (NYSDEC, 1999).

6.1.3 Discussion of 1991 Phase 1 Risk Assessment

In 1991, USEPA issued the Phase 1 Report - Interim Characterization and Evaluation for the Hudson River PCB Reassessment Remedial Investigation/Feasibility Study, including a quantitative risk

assessment for the Upper Hudson River and a qualitative risk assessment for the Lower Hudson River (USEPA, 1991a). The risks from ingestion of fish in the Lower Hudson River were qualitatively evaluated, based on the findings in the Upper Hudson River. The assessment concluded that the risks from ingestion of fish would be similar to those found in the Upper Hudson River. The assessment of the Lower Hudson was based on the Thomann PCB bioaccumulation model (USEPA, 1991a).

6.1.4 Objectives of Phase 2 Risk Assessment

The objective of the Phase 2 risk assessment is to quantitatively evaluate current and potential cancer risks and non-cancer health hazards from river water, sediment, and fish in the Mid-Hudson River. This HHRA provides estimates of risks both to the RME individual, or high-end risk (>90^{th} to 99^{th} percentiles), and to the Average Exposed Individual, or central tendency cancer risks and non-cancer health hazards (50^{th} percentile). Since the Phase 1 Risk Assessment, USEPA has conducted extensive modeling efforts in order to forecast PCB concentration trends in environmental media in the Mid-Hudson River region (USEPA, 2000a; 2000f; 2000g). The results from these model forecasts were incorporated into this Phase 2 assessment.

6.2 Exposure Assessment

The objective of the exposure assessment is to estimate the magnitude of human exposure to PCBs in the study area. The same approach and terminology that was used in the Upper Hudson assessment is being adopted here for the Mid-Hudson River HHRA, with the exception that a Monte Carlo analysis was not performed for the fish ingestion pathway for the Mid-Hudson HHRA. Because this Mid-Hudson HHRA parallel the assessment for the Upper Hudson River, the reader should refer to Chapter 2 for additional details.

6.2.1 Exposure Pathways

Those pathways considered in the Upper Hudson River assessment were also considered for this Mid-Hudson HHRA. In general, during boating, fishing, and other recreational activities, members of the Mid-Hudson River study area population may be exposed to PCBs if they consume fish caught from the river, or as they come into contact with river water and river sediments. In addition, the Mid-Hudson River is a source of drinking water and exposure may occur from this pathway. Potential exposure pathways considered are summarized in Table 2-1 (Appendix E), identifying those, which are "complete," and warranted exposure and risk calculations in this study. The following sections briefly summarize the site-specific elements that make up the complete exposure pathways that are evaluated in the Mid-Hudson River HHRA, while the Upper Hudson HHRA discusses the exposure pathways in more detail.

6.2.1.1 Potential Exposure Media

Humans may be exposed to PCBs from the site either through direct ingestion or contact with media containing PCBs. The exposure media that are considered the most potentially significant source of PCB exposure at the site include fish, sediment, and river water. The actual determination of the relative importance of each of these potential exposure media, and those, which may or may not pose a significant health risk, is determined based on the results of the quantitative exposure and risk analysis. As discussed in the Upper Hudson HHRA, PCBs in air (volatilizing from river water) were found to pose minimal risk (10^{-6} or less) in the Upper Hudson region. For the Mid-Hudson River, the total PCB concentration in river water is approximately four times lower than the Upper Hudson such that airborne

PCBs from the river would exhibit a lower concentration (and risk) than determined for the Upper Hudson HHRA. Therefore, cancer risks due to the air pathway is not quantitatively evaluated in the Mid-Hudson River assessment.

6.2.1.2 Potential Receptors

The population of concern in the evaluation of the Mid-Hudson River includes the inhabitants of the towns, cities, and rural areas bordering the river who may fish or engage in activities that will bring them in contact with the river. The six counties include: Albany, Columbia, Dutchess, Greene, Rensselaer and Ulster. From this population, anglers, recreators, and residents were defined as "receptor" groups for the purpose of quantifying the potential PCB exposures within the population as a whole. A detailed description of these receptor categories can be found in Section 2.1.2.

6.2.1.3 Potential Exposure Routes

An exposure route is the means, or mechanism, of contact with an exposure medium. Similar to the Upper Hudson River area, fish ingestion (*i.e.*, dietary intake) is the potential exposure route for anglers evaluated in this risk assessment. Routes of exposure under a recreational use scenario include absorption of PCBs *via* dermal contact with sediments, incidental ingestion of PCBs contained in sediments during subsequent hand to mouth contact, and dermal contact with river water. Consumption of river water as a residential source of drinking water is included in the Mid-Hudson HHRA to address public concerns (the predicted PCB concentrations are well below the MCL).

As summarized in Table 2-1 (Appendix E), several exposure routes are not quantitatively evaluated in this Mid-Hudson HHRA. Cancer risks from the inhalation of air (due to PCBs volatilizing from river water) were not evaluated due to low PCB concentrations present in the Mid-Hudson River. In addition, other potential pathways, such as dietary intake of home-grown crops, consumption of local beef or dairy products, or consumption of snapping turtles are unlikely to be significant pathways for PCB intake, for the reasons discussed in Section 2.1.3.

6.2.2 Quantification of Exposure

This section of the risk assessment summarizes the basic approach for calculating human intake levels resulting from exposures to PCBs. A more detailed explanation of the quantification of exposure can be found in Section 2.2.

The primary source for the exposure algorithms used in the risk assessment is USEPA's Risk Assessment Guidance for Superfund, Part A (RAGS) (USEPA, 1989b). The generalized equation for calculating chemical intakes is:

$$I = \frac{C \times CR \times EF \times ED \times CF}{BW \times AT}$$

where:

I	=	*Intake* - the amount of chemical at the exchange boundary (mg/kg body weight/day)
C	=	*Exposure Point Concentration* - the chemical concentration contacted over the exposure period at the exposure point (*e.g.*, mg/kg-fish)
CR	=	*Contact Rate* - the amount of affected medium contacted per unit time or event (*e.g.*, fish ingestion rate in g/day)
EF	=	*Exposure frequency* - describes how often exposure occurs (days/year)
ED	=	*Exposure duration* - describes how long exposure occurs (yr)
CF	=	*Conversion factor* - (kg/g)
BW	=	*Body weight* - the average body weight over the exposure period (kg)
AT	=	*Averaging time* - period over which exposure is averaged (days)

Exposure parameters (*e.g.*, contact rate, exposure frequency, exposure duration, body weight) describe the exposure of a receptor for a given exposure scenario. These values are the input parameters for the exposure algorithms used to estimate chemical intake. The general equation above is slightly modified for each pathway, and the specific exposure parameters for each pathway are summarized and discussed further in Section 6.2.4.

6.2.3 Exposure Point Concentrations

The EPCs for PCBs in fish, water, and sediment are based upon modeled projections of future concentrations in each medium (although the models are based upon a large monitoring record). As a result, the typical approach adopted in Superfund risk assessments of calculating an upper confidence limit on a mean concentration (*i.e.*, 95% UCLM), no longer strictly applies. In addition, as mentioned above, PCBs were identified as chemicals of concern such that no screening of COPCs was performed for this assessment. Thus, the USEPA RAGS Part D format (Appendix E, Tables 2-2 through 2-4) which, for a typical risk assessment, would include information necessary to determine COPCs, are not needed and are included in this HHRA only for consistency.

This assessment reflects sediment, water column, and bioaccumulation modeling as presented in the RBMR (USEPA, 2000a) and the Revised Baseline Ecological Risk Assessment (Revised ERA) (USEPA, 2000f). All baseline modeling efforts assumed a constant upstream boundary of 13 ng/L PCBs.

6.2.3.1 PCB Concentration in Fish

Because the Mid-Hudson HHRA examines current and future cancer risks and non-cancer hazards, and because the concentration of PCBs in fish changes over time and location, the EPC for PCBs in fish necessarily relies upon model predictions. Three factors have an influence on the exposure point concentration in fish:

1. The concentration of PCBs for any particular species varies for a particular year, but overall it declines over time.

2. The concentration of PCBs within the same fish species varies with location in the Hudson River, with higher concentrations upstream compared to downstream.

3. The concentration of PCBs varies among different fish species.

Thus, even though fish are considered a single exposure medium for the HHRA, each of the above factors will influence the calculation of a single exposure point concentration.

Summary of Modeled PCB Concentration Results

The Revised ERA (USEPA, 2000f) presents a detailed discussion of the PCB bioaccumulation and transport and fate models that have been used by USEPA to predict future trends of PCB concentration in fish. The FISHRAND and Thomann/Farley model predictions used to estimate EPCs for fish were summarized in the Revised ERA. The Thomann/Farley model predictions were used for white perch (age classes 1-7) because it accounts for their migratory behavior. The Thomann/Farley model was not used to determine PCB concentrations in striped bass because it only accounts for migratory striped bass in the Lower Hudson (*i.e.*, below the salt water front) and does not include the Mid-Hudson River study area. The FISHRAND model results were used for the brown bullhead, largemouth bass, and yellow perch. Because striped bass was not specifically modeled in the Mid-Hudson region, the FISHRAND modeled largemouth bass values, scaled by the average ratio of PCB concentration in striped bass over largemouth bass in the NYSDEC monitoring data, were used to estimate future PCB concentrations in striped bass in the Mid-Hudson River (von Stackelberg, 1999). The reader is referred to the Revised ERA (USEPA, 2000f) and the RBMR (USEPA, 2000a) for further information on the bioaccumulation and transport and fate models.

Model predictions were available for a total of seven fish species: brown bullhead, largemouth bass, striped bass, white perch, yellow perch, spottail shiner, and pumpkinseed. These species were selected in the Revised ERA to get a representative distribution of bottom feeders, species at the top of the food chain, and semi-piscivorous species found in the study area (USEPA, 2000f). Two of these modeled species (spottail shiner and pumpkinseed) were not included in this assessment because they are small fish and are typically not consumed by humans. They were modeled in the Revised ERA as one component of the fish food web that contributes to PCB accumulation higher up in the food chain.

Model estimates of total PCB concentration in each species were based on PCB congeners with three or more chlorine molecules, *i.e.*, Tri+ PCB concentrations. For the larger fish species modeled (*i.e.*, brown bullhead, largemouth bass, striped bass, white perch, and yellow perch), the model provides estimates of PCB concentration in fish fillets (skin on), otherwise the model results are for whole fish for the smaller species. The fillet represents the portion of the fish most commonly consumed by humans.

Modeled predictions of future PCB concentrations in fish from the FISHRAND model are presented in the Revised ERA at three locations along the Mid-Hudson River: River Mile 152 (corresponding to River Miles 153.5 - 123.5); River Mile 113 (corresponding to River Miles 123.5 - 93.5); and River Mile 90 (corresponding to River Miles 93.5 - 63.5). These three locations correspond to locations along the river where fish have been monitored by NYSDEC. Modeled predictions from the Thomann/Farley model (Farley, *et al*., 1999) are presented as an overall average by food web region. Region 1 model results (River Miles 153.5 - 73.5) were used for the Mid-Hudson region. In general, the concentrations for all fish species decrease with River Mile and time. Modeled PCB concentrations in fish for the period 1999 through 2046, were used to estimate present (1999) and future exposure to PCBs in fish. Figures 2-1 through 2-5 (Appendix E) display the modeled mean concentration trend over time by location for each of the 5 modeled species considered in this assessment.

Concentration Averaged Over Locations

With the exception of some limited information in the 1991/92 and 1996 NYSDOH study of Hudson River anglers (NYSDOH, 1999b), there is insufficient information to quantify fishing preference or frequency at specific locations within the entire Mid-Hudson River. Consequently, projected PCB concentrations in fish were averaged over the Mid-Hudson River region. This averaging essentially presumes a uniform likelihood of fishing at any location within the Mid-Hudson River study area.

The PCB concentrations, averaged over location, for each of the modeled species are summarized in Figure 2-6 (Appendix E). Overall, modeled PCB concentrations for striped bass are among the highest, ranging from approximately 2.5 mg/kg to less than 1 mg/kg, while the modeled PCB concentrations in yellow perch are the lowest, ranging from approximately 0.5 mg/kg to 0.25 mg/kg.

PCB Concentration Weighted by Species-Consumption Fractions

In order to take into account the species individuals actually eat from the Mid-Hudson River, species-specific intake patterns, derived from the 1991 New York Angler survey (Connelly *et al.*, 1992) and the 1991/92 and 1996 NYSDOH study of Hudson River anglers (NYSDOH, 1999b), were used as weighting factors to calculate the average concentration of PCBs in fish. That is, the overall average PCB concentration in fish that an angler consumes was based on the relative percent of different fish species consumed, and their respective modeled PCB concentrations.

A complete discussion of the 1991 New York Angler survey is found in Section 3.2.1. A summary of the survey is provided in Table 2-5 (Appendix E), and is briefly described here. A total of 9 specific species, plus a tenth category denoted "other," were included in the Connelly *et al.* (1992) survey. Of the 9 species in the survey, salmon, trout, and walleye are not commonly found in the Mid-Hudson River study area; therefore, these three species, along with the unidentified "other" category, were excluded when determining species ingestion weights. The six species from the Connelly *et al.* (1992) survey that are potentially caught and eaten in the Mid-Hudson River were grouped such that species for which predicted PCB concentrations are unavailable were assigned the PCB concentration of a modeled species that fell within the same group.

The Connelly *et al.* (1992) survey did not distinguish among species included in the "perch" and "bass" categories. Because white perch, yellow perch, largemouth bass, and striped bass are being considered separately for the Mid-Hudson region, an estimated species intake for each was based on adjusting the Connelly *et al.* (1992) ingestion rates using relative catch frequency of the four species. Table 2-6 (Appendix E) summarizes the break-down, which was based on the Mid-Hudson results of the 1991/92 and 1996 NYSDOH study of Hudson River anglers (NYSDOH, 1999b). The results from the NYSDOH survey only account for the amount of each species caught, rather than amount of each species consumed. Other surveys of the Mid-Hudson River region (Jackson, 1990) generally support the results of the NYSDOH survey. Note that although the Jackson (1990) study revealed a higher largemouth bass to striped bass ratio, almost 3/4 of the respondents were targeting black bass (largemouth and smallmouth bass) for a tournament. As a result, the NYSDOH survey results were deemed more appropriate for use. In the NYSDOH (1999b) survey, the white perch catch outnumbers yellow perch about 6:1, while the striped bass catch outnumbers largemouth bass about 3:2.

Table 2-7 (Appendix E) summarizes species-group intake percentages by summing the consumption frequency for individual species in each group. Carp, catfish, and eel were assigned the same PCB concentration as brown bullhead, in part because like bullhead, they tend to spend much of

their time at the bottom of lakes, rivers, and streams. Modeled PCB concentrations are available for each of the remaining species, in the remaining groups.

The point estimate PCB concentrations were derived using the species ingestion fractions shown in Table 2-7 (Appendix E) multiplied by the PCB concentrations in each of the five modeled fish species. Thus, the weighted EPC is:

$$EPC = \sum_{X=1}^{5} \left(EPC_{GroupX} \times Species\,Ingestion\,Fraction_{GroupX} \right)$$

The species-weighted EPC value for fish in the Mid-Hudson River is summarized in Table 2-8 (Appendix E). The EPC for each fish group (EPC_{GroupX}) is the average over all locations within the Mid-Hudson River. The central tendency EPCs of 1.5 mg/kg PCBs for adults and 1.6 mg/kg for adolescents and children were calculated by averaging the species-weighted concentration distribution over the 50th percentile exposure duration estimates of 6 years for adults, 3 years for adolescents, and 3 years for children (summing to 12 years). The high-end exposure EPCs of 1.2 mg/kg PCBs for adults, 1.3 mg/kg for adolescents, and 1.5 mg/kg for children were calculated by averaging the species-weighted concentration distribution over the 95th percentile exposure duration estimates of 22 years for adults, 12 years for adolescents, and 6 years for children (summing to 40 years). The determination of these particular exposure durations is described in Section 6.2.4.1.

It may be counter-intuitive that the high-end EPCs are lower than the central tendency EPCs. This fact is a direct result of the declining PCB concentration in fish. Due to this decline over time, the average concentration over a longer exposure duration is less than the average concentration over a shorter time period. However, the total lifetime PCB dose, which combines concentration, exposure duration, and other intake factors, is greater for the high-end (RME) point estimate.

6.2.3.2 PCB Concentration in Sediment

Just as is the case for fish, PCB concentrations in sediment in the Mid-Hudson River generally decrease as a function of river mile and time. As described in the Revised ERA, PCB concentrations in surficial (0 - 4 cm) sediment were modeled over time and distance. The model predictions for the Mid-Hudson study area were presented for 9 different river mile segments, each approximately 10 miles long, from the Federal Dam (River Mile 153.5) to the salt water front (approximately River Mile 64). The predicted Total PCB concentrations in sediment are plotted in Figure 2-7 (Appendix E).

The exposure point concentrations in sediment were calculated by first averaging the results for Total PCBs in sediment over the 9 model segments (see Figure 2-7), then averaging these values over the central tendency (*i.e.*, 11 years) and RME (*i.e.*, 41 years) exposure durations. Note the exposure duration for this pathway is based only on residence duration, as opposed to a RME of 40 years and a central estimate of 12 years for angling duration, which is a combination of residence duration and fishing duration (see Section 3.2.4). The RME exposure duration is 6 years for children, 12 years for adolescents, and 23 years for adults (summing to 41 years), and the central tendency exposure duration is 3 years for children, 3 years for adolescents, and 5 years for adults (summing to 11 years). The mean of the first 1-4, 5-7, and 8-12 years of these segment averages (0.68, 0.68, and 0.67 mg/kg PCBs) was used as the central tendency point estimate EPCs for children, adolescents, and adults, respectively; the mean of the first 1-7, 8-19, and 20-42 years of these segment averages (0.66, 0.62, and 0.57 mg/kg PCBs) was used as the RME point estimates for children, adolescents, and adults, respectively (Table 2-9, Appendix E).

Again, it may be counter-intuitive that the high-end EPCs are lower than the central tendency EPCs. This fact is a direct result of the declining PCB concentration in sediment over time giving rise to declining EPC estimates as the duration of exposure increases.

6.2.3.3 PCB Concentration in River Water

Similar to the sediment results, the Revised ERA provides model estimated PCB concentrations in the water column over location and time. The water column model predictions for the Mid-Hudson River were presented for 9 river segments, from the Federal Dam (River Mile 153.5) to the salt water front (approximately River Mile 64). The predicted Total whole water PCB concentrations in water are plotted in Figure 2-8 (Appendix E).

The exposure point concentrations in river water were calculated by first averaging the Total PCB concentrations across the 9 model segments, then averaging these values over the central tendency (*i.e.*, 11 years) and RME (*i.e.*, 41 years) exposure durations. The RME exposure duration is 6 years for children, 12 years for adolescents, and 23 years for adults (summing to 41 years), and the central tendency exposure duration is 3 years for children, 3 years for adolescents, and 5 years for adults (which sum to 11 years). The mean of the first 1-4, 5-7, and 8-12 years of these segment averages (1.7×10^{-5}, 1.7×10^{-5}, and 1.5×10^{-5} mg/L PCBs) was used as the central tendency point estimate EPCs for children, adolescents, and adults, respectively; the mean of the first 1-7, 8-19, and 20-42 years of these segment averages (1.4×10^{-5}, 1.2×10^{-5}, and 9.3×10^{-6} mg/L PCBs) was used as the RME point estimates for children, adolescents, and adults, respectively (Table 2-10).

6.2.4 Chemical Intake Algorithms

The calculation of PCB intake for each complete exposure pathway for the HHRA follows the same procedures described in further detail in the Upper Hudson HHRA. Complete tabulations of the exposure factors for each exposure pathway and receptor scenario are found in Tables 2-19 through 2-28 (Appendix E).

6.2.4.1 Ingestion of Fish

The fish ingestion point estimate intake is calculated as:

$$\text{Intake}_{\text{fish}}(\text{mg / kg - d}) = \frac{C_{\text{fish}} \times IR \times (1 - LOSS) \times FS \times EF \times ED \times CF}{BW \times AT}$$

where:

C_{fish}	=	Concentration of PCBs in fish (mg/kg)
IR	=	Annualized fish ingestion rate (g/day)
LOSS	=	Cooking loss (g/g)
FS	=	Fraction from source (unitless fraction)
EF	=	Exposure frequency (days/year)
ED	=	Exposure duration (years)
CF	=	Conversion Factor (10^{-3} kg/g)
BW	=	Body weight (kg)

AT = Averaging time - period over which exposure is averaged (days); over a lifetime for evaluating cancer risks and over the appropriate exposure duration for evaluating non-cancer health hazards.

Exposure factor values for the central tendency and RME point estimate calculations for this pathway are summarized in Table 2-19 (Appendix E). Site-specific considerations in selecting these factors are discussed below.

Fraction from Source (FS). This HHRA examines possible exposure for the population of anglers who consume self-caught fish from the Mid-Hudson River. Thus, the exposure and risk analysis assumes the Mid-Hudson River accounts for 100% of the sportfish catch of the angler (FS=1). As noted below, the fish ingestion rate is based upon consumption of sportfish, such that it excludes fish that may be purchased and then consumed.

Exposure Frequency (EF). Because the fish ingestion rate is based on an annualized average ingestion over one year, an implicit exposure frequency value of 365 days/year is used in the intake calculation. This does not imply consumption of fish 365 days per year.

Exposure Duration (ED). While Superfund risk assessments typically use the length of time that an individual remains in a single residence as an estimate for exposure duration, such an estimate is not likely to be a good predictor of angling duration, because an individual may move into a nearby residence and continue to fish in the same location, or an individual may chose to stop angling irrespective of the location of their home. Furthermore, given the large size of the Hudson River PCBs Superfund site, an individual may move from one place of residence to another, and still remain within the Mid-Hudson area and continue to fish from the Mid-Hudson River. For the purposes of defining the angler population likely to fish the Mid-Hudson River most frequently, it was assumed this population would be most likely to constitute residents from the six counties surrounding the Mid-Hudson River (Albany, Columbia, Dutchess, Greene, Rensselaer, and Ulster). Furthermore, the 1988 New York Angler survey (Connelly *et al*., 1990) found that the average distance traveled by New York anglers was 34 miles, supporting the notion that the majority of the angler population for the Mid-Hudson River is likely to reside in these counties.

Given the above considerations, the exposure duration (angling, or fishing, duration) for the fish consumption pathway is not based solely upon a typical residence duration. Instead, as described more fully in the Upper Hudson HHRA, an angler is assumed to continue fishing until any of the following occur:

- the individual stops fishing;
- the individual moves out of the area, or dies.

The 1991 New York Angler survey of over 1,000 anglers (Connelly *et al.*, 1992) was used to estimate fishing duration habits within the population of New York anglers. U.S. Census data (1990) on county to county mobility provided the source of information to estimate the range of residence durations within the six counties surrounding the Mid-Hudson River (Tables 2-11 through 2-18, Appendix E). As shown in Table 2-18 (Appendix E), the 1-year move probabilities for the Mid-Hudson region are virtually the same (less than 1% difference for any age group) as that for the Upper Hudson region. Given the fact that residence durations for the Mid-Hudson region age categories are essentially the same as those for the

Upper Hudson region, the angling and residence duration distribution derived for the Upper Hudson HHRA apply for the Mid-Hudson HHRA as well.

The 50th percentile of the fishing duration distribution is 12 years and the 95th percentile is 40 years for the Mid-Hudson River region. The RME exposure duration is 6 years for children, 12 years for adolescents, and 22 years for adults (summing to 40 years), and the central tendency exposure duration is 3 years for children, 3 years for adolescents, and 6 years for adults (summing to 12 years). A more complete and detailed discussion of the exposure duration derivation is provided in the Upper Hudson River HHRA (Section 3.2.4).

Body Weight (BW). The average adult body weight used in the intake equation was 70 kg, taken from USEPA (1989a,b). Note that the adult body weight found in the 1997 Exposure Factors Handbook (USEPA, 1997f) is 71.8 kg. Because USEPA's derivation of the PCB cancer toxicity factors was based upon a 70 kg adult in extrapolating the animal data to humans, this assessment uses the prior 70 kg body weight value for consistency.

Averaging Time (AT). A 70-year lifetime averaging time of 25,550 days was used for cancer calculations (70 years × 365 day/year) (USEPA, 1989a,b). In order to avoid possible confusion, a 70 year life expectancy from USEPA RAGS was used as the averaging time for cancer, even though the 1997 Exposure Factors Handbook (USEPA, 1997c) indicates 75 years is the most current estimate. Had a 75 year averaging time been used, this would effectively decrease the calculated intake of PCBs in fish by 7%.

Non-cancer averaging times are not averaged over a lifetime, but rather over a period of time equating to a chronic level of exposure. Therefore, the averaging time for the non-cancer hazard assessment is equal to the exposure duration multiplied by 365 days/ year.

Concentration of PCB in Fish (C_{fish}). As described earlier in Section 6.2.3.1, the PCB concentration in fish was determined based on the modeled Tri+ PCB concentration results, weighted by fish consumption patterns. For the evaluation of cancer risks, the central tendency EPCs are 1.5 mg/kg for adults and 1.6 mg/kg for adolescents and children. These EPCs were calculated by averaging the species-weighted concentration distribution over 6, 3, and 3 years for adult, adolescent, and child exposures, respectively. The exposure durations for each age group sum to 12 years, the 50th percentile exposure duration estimate. The corresponding RME values are 1.2 mg/kg for adults, 1.3 mg/kg for adolescents, and 1.5 mg/kg for child exposures. These EPCs were calculated by averaging the species-weighted concentration distribution over 22, 12, and 6 years for adult, adolescent, and child exposures, respectively. The RME exposure durations sum to 40 years, the 95th percentile exposure duration estimate. It should be noted that the apparent contradiction in EPC, whereby the high-end EPC is lower than the central tendency EPC, is a direct result of the declining PCB concentration in fish over time. Due to this decline over time, the average concentrations over longer exposure durations are less than the average concentrations over shorter periods.

As noted above, the averaging time for the non-cancer hazard assessment was limited to a maximum of 7 years. The 7-year average EPC in fish is 1.5 mg/kg PCBs.

Fish Ingestion Rate (IR). The fish ingestion rate is based upon an estimate of the long term average consumption of self-caught fish in the angler population, expressed as an annualized daily average rate in units of grams of fish per day (g/day). It is important to note that the ingestion of fish from all sources (*e.g.*, self-caught plus purchased fish) is necessarily greater than or equal to the ingestion

rate of only self-caught fish. Because this assessment examines the risk of PCB intake from Mid-Hudson River fish only, the focus is only on self-caught fish.

A full description of the derivation of fish ingestion rates is found in Section 3.2.1, and is briefly summarized here. The fish ingestion rate for this Revised HHRA is based upon a survey of over 1,000 New York anglers (Connelly *et al.*, 1992) who catch and consume fish. For the point estimate exposure and risk calculations, the 50^{th} percentile of the empirical distribution (4.0 g/day) is used as the central tendency point estimate of fish ingestion, and the 90^{th} percentile (31.9 g/day) is the RME ingestion rate. For a one-half pound serving, these ingestion rates represent approximately 6.4 and 51 fish meals per year, respectively.

Cooking Loss (LOSS). Numerous studies have examined the loss of PCBs from fish during food preparation and cooking. A review of the available literature is discussed in Section 3.2.3. Overall, the 12 studies reviewed support the conclusion that cooking loss may be zero to 74 percent. Despite the rather wide range of cooking loss estimates, most PCB losses were between 10 and 40 percent. A value of 20% (midpoint of 0% - 40%) was selected as the central tendency point estimate for cooking loss. In addition, personal preferences for various preparation and cooking methods and other related habits (such as consuming pan drippings) may result in consumption of PCBs "lost" from the fish upon cooking. Thus, for the RME, no cooking loss (LOSS = 0%) was selected for this exposure factor.

6.2.4.2 Ingestion of Sediment

For the sediment ingestion pathway, intake is calculated as:

$$\text{Intake}_{\text{ingestion}}(\text{mg / kg - d}) = \frac{C_{sed} \times IR \times FS \times EF \times ED \times CF}{BW \times AT}$$

where:

C_{sed}	=	Concentration of PCBs in sediment (mg/kg)
IR	=	Sediment ingestion rate (mg/day)
FS	=	Fraction from source (unitless fraction)
EF	=	Exposure frequency (days/year)
ED	=	Exposure duration (years)
CF	=	Conversion factor (10^{-6} kg/mg)
BW	=	Body weight (kg)
AT	=	Averaging time - period over which exposure is averaged (days); over a lifetime for evaluating cancer risks and over the appropriate exposure duration for evaluating non-cancer health hazards.

Exposure factor values for the central tendency and RME point estimate calculations for this pathway are summarized in Tables 2-20 through 2-22 (Appendix E). Site-specific considerations in selecting these factors are discussed below.

PCB Concentration in Sediment (C_{sed}). As described in Section 6.2.3.2, the central tendency point estimates used for PCB concentration in sediment are 0.68, 0.68, and 0.67 mg/kg for children, adolescents, and adults, respectively. The RME point estimates are 0.66, 0.62, and 0.57 mg/kg for children, adolescents, and adults, respectively (see Table 2-9, Appendix E).

Sediment Ingestion Rate (IR). This factor provides an estimate of incidental intake of sediment that may occur as a result of hand-to-mouth activity. In the absence of site-specific ingestion rates, USEPA-recommended median values for daily soil ingestion, rather than high-end values, were used for this factor to account for the shorter timeframes spent by recreators at the Hudson River. The incidental ingestion rate for children is 100 mg/day, and for adults and adolescents the value is 50 mg/day. These values, reported as median estimates of soil intake, are the recommendations found in USEPA's current Exposure Factors Handbook (USEPA, 1997f). The incidental soil (sediment) ingestion rate provides an estimate of the ingestion that may occur integrated over a variety of activities, including ingestion of indoor dust. Thus, these median ingestion rates are likely high-end estimates of incidental sediment ingestion while participating in activities along the Hudson, because other sources (such as at home) also account for soil/sediment ingestion.

Exposure Frequency (EF). Exposure to river sediments is most likely to occur during recreational activities. However, there are no site-specific data to provide an indication of the likely frequency of recreational activities along the Mid-Hudson River, nor are there general population studies that provide usable information. Under the assumption that recreational activities are likely to be most frequent during the summer months, an estimate of one day per week during the 13 weeks of summer is considered a reasonable estimate of the RME value for adults (*i.e.*, 13 days per year). This same frequency was adopted for children (aged 1-6), assuming an adult would most likely accompany them. For adolescents (aged 7-18), who are not as likely to be accompanied by an adult, it was assumed their recreational frequency was three-fold greater than the adult/child frequency (*i.e.*, 39 days per year). The RME values were reduced by 50% for the central tendency exposure calculations.

Exposure Duration (ED). The RME exposure duration for sediment ingestion in recreational scenarios is 41 years, and the central tendency value is 11 years, which correspond to the 95th and 50th percentiles, respectively, of the residence duration determined for the six Mid-Hudson counties. The RME exposure duration is 6 years for children, 12 years for adolescents, and 23 years for adults (summing to 41 years), and the central tendency exposure duration is 3 years for children, 3 years for adolescents, and 5 years for adults (which sum to 11 years). Note that these values are somewhat greater than values determined from nationwide statistics, which indicate 30 years is the 95th percentile, and 9 years is the 50th percentile residence duration at one location (USEPA, 1997f).

Body Weight (BW). Age-specific body weights were used. The mean body weight for children aged 1 to 6 is 15 kg, the mean body weight for adolescents aged 7-18 is 43 kg, and the mean adult body weight is 70 kg (USEPA, 1989a,b; 1997f).

Averaging Time (AT). For all recreational exposure calculations, a 70-year lifetime averaging time of 25,550 days (365 days × 70 years) was used for cancer evaluations. Non-cancer averaging times are equal to the exposure duration multiplied by 365 days/year (USEPA, 1989a,b).

6.2.4.3 Dermal Contact with Sediment

For the sediment dermal contact, absorbed doses are used. Dermal intake (the amount absorbed into the body) is calculated as:

$$\text{Intake}_{\text{dermal}}\,(\text{mg / kg - d}) = \frac{C_{\text{sed}} \times DA \times AF \times SA \times EF \times ED \times CF}{BW \times AT}$$

where:

C_{sed}	=	Concentration PCBs in sediment (mg/kg),
DA	=	Dermal absorption fraction (unitless),
AF	=	Sediment/skin adherence factor (mg/cm^2),
SA	=	Skin surface area exposed (cm^2/exposure event),
EF	=	Exposure frequency (exposure events/year),
ED	=	Exposure duration (years),
CF	=	Conversion factor (10^{-6} kg/mg)
BW	=	Body weight (kg)
AT	=	Averaging time - period over which exposure is averaged (days); over a lifetime for evaluating cancer risks and over the appropriate exposure duration for evaluating non-cancer health hazards.

Exposure factor values for the central tendency and RME point estimate calculations for this pathway are summarized in Tables 2-20 through 2-22 (Appendix E). Site-specific considerations in selecting these factors are discussed below.

PCB Concentration in Sediment (C_{sed}). As described above, the central tendency point estimates used for PCB concentration in sediment are 0.68, 0.68, and 0.67 mg/kg for children, adolescents, and adults, respectively. The RME point estimates are 0.66, 0.62, and 0.57 mg/kg for children, adolescents, and adults, respectively (see Table 2-9, Appendix E).

Dermal Absorption Fraction (DA). The dermal absorption fraction represents the amount of a chemical in contact with skin that is absorbed through the skin and into the bloodstream. The dermal absorption rate of 14% used in this Mid-Hudson HHRA is based on the *in vivo* percutaneous absorption of PCBs from soil by rhesus monkeys (Wester *et al.*, 1993).

Soil/Skin Adherence Factor (AF). The sediment adherence values for the risk assessment were obtained from USEPA's March 1999 Draft Dermal Risk Assessment Guidance (USEPA, 1999f), which among other studies, relies upon data published by Kissel *et al.* (1998). The 50th percentile sediment/skin adherence factor for children is 0.2 mg/cm^2, and 0.3 mg/cm^2 for adults (USEPA, 1999f), as discussed in Section 2.4.3. These adherence factors are for children playing in wet soil, and adults whose soil loadings were measured for reed gathering activities. These activities, which represent active contact with soil, are appropriate surrogates for activities where Mid-Hudson River recreators may contact sediment. The soil adherence factor for adolescents was taken as the midpoint between the child and adult factors.

Skin Surface Area Exposed (SA). For children and adolescents, the mean surface area of hands, forearms, lower legs, feet, and face were calculated by multiplying the total body surface area (averaged between males and females) by the percentage of total body surface area that make up the relevant body parts (USEPA, 1997f). For children, the mean surface area of the hands, forearms, lower legs, feet, and face is 2,792 cm^2 (using data for the category 6<7 years); for adolescents, the mean surface area of the hands, forearms, lower legs, feet, and face is 4,263 cm^2 (for age 12 years); the mean surface area of adult hands, forearms, lower legs, feet, and face is 6,073 cm^2 (USEPA, 1997f).

Exposure Frequency (EF). As described above, there are no site-specific data to provide an indication of the likely frequency of recreational activities along the Mid-Hudson River, nor do general population studies exist that provide usable information. The exposure frequency factors (Tables 2-20 through 2-22, Appendix E) for dermal contact are the same as those for incidental ingestion described in the proceeding section.

Exposure Duration (ED). As explained in the previous section, the exposure duration for sediment dermal contact in recreational scenarios is 41 years, and the central tendency value is 11 years, which correspond to the 95th and 50th percentiles, respectively, of the residence duration determined for the six Mid-Hudson counties.

Body Weight (BW). Age-specific body weights were used. The mean body weight for children aged 1 to 6 is 15 kg, the mean body weight for adolescents aged 7-18 is 43 kg, and the mean adult body weight is 70 kg (USEPA, 1989a,b; 1997f).

Averaging Time (AT). For all recreational exposure calculations, a 70-year lifetime averaging time of 25,550 days (365 days × 70 years) was used for cancer evaluations. Non-cancer averaging times are equal to the exposure duration multiplied by 365 days/year (USEPA, 1989a,b).

6.2.4.4 Dermal Contact with River Water

For the river water dermal contact pathway, dermal intake (the amount absorbed into the body) is calculated as:

$$\text{Intake}_{\text{water}}(\text{mg / kg - d}) = \frac{C_w \times K_p \times SA \times DE \times EF \times ED \times CF}{BW \times AT}$$

where:

C_w	=	Concentration of PCBs in water (mg/l)
K_p	=	Chemical-specific dermal permeability constant (cm/hr)
SA	=	Skin surface area exposed (cm^2)
DE	=	Duration of event (hr/d)
EF	=	Exposure frequency (d/year)
ED	=	Exposure duration (years)
CF	=	Conversion factor (10^{-3} L/cm^3)
BW	=	Body weight (kg)
AT	=	Averaging time - period over which exposure is averaged (days); over a lifetime for evaluating cancer risks and over the appropriate exposure duration for evaluating non-cancer health hazards.

Exposure factor values for the central tendency and RME point estimate calculations for this pathway are summarized in Tables 2-23 through 2-25 (Appendix E). Site-specific considerations in selecting these factors are discussed below.

PCB Concentrations in River Water (C_w). As described in Section 6.2.3.3, the central tendency point estimates used for PCB concentration in the water column are 1.7×10^{-5}, 1.7×10^{-5}, and 1.5×10^{-5} mg/L, for children, adolescents, and adults, respectively. The RME point estimates are 1.4×10^{-5}, 1.2×10^{-5}, and 9.3×10^{-6} mg/L, for children, adolescents, and adults, respectively (Table 2-10, Appendix E).

Permeability Constant (K_p). In the absence of experimental measurements for the dermal permeability constant for PCBs, it was estimated to be 0.48 cm/hr based on the value for hexachlorobiphenyls reported in the 1999 Draft Dermal Risk Assessment Guidance (USEPA, 1999f).

Skin Surface Area Exposed (SA). As a conservative estimate of possible exposure, 100% of the full-body surface area was assumed to come into contact with water. The surface areas for adults, adolescents, and children, respectively are: 18,150 cm^2, 13,100 cm^2, and 6,880 cm^2 (USEPA, 1997f).

Duration of Event (DE). For all recreator scenarios, 2.6 hours/day was used as the river water dermal exposure time, which is the national average duration for a swimming event (USEPA, 1989b).

Exposure Frequency (EF). As described above, there are no site-specific data to provide an indication of the likely frequency of recreational activities along the Mid-Hudson River, nor do general population studies exist that provide usable information. The exposure frequency factors (Tables 2-23 through 2-25, Appendix E) for dermal contact with water while swimming are the same as those for incidental ingestion and dermal contact with sediments described in the proceeding sections.

Exposure Duration (ED). As described in the previous sections, the exposure duration for river water dermal contact in recreational scenarios is 41 years, and the central tendency value is 11 years, which correspond to the 95th and 50th percentiles, respectively, of the residence duration determined for the six Mid-Hudson counties.

Body Weight (BW). Age-specific body weights were used. The mean body weight for children aged 1 to 6 is 15 kg, the mean body weight for adolescents aged 7-18 is 43 kg, and the mean adult body weight is 70 kg (USEPA, 1989a,b; 1997f).

Averaging Time (AT). For all recreational exposure calculations, a 70-year lifetime averaging time of 25,550 days (365 days × 70 years) was used for cancer evaluations. Non-cancer averaging times are equal to the exposure duration multiplied by 365 days/year (USEPA, 1989a,b).

6.2.4.5 Ingestion of River Water

For the river water ingestion pathway, intake is calculated as:

$$\text{Intake}_{\text{water}}(\text{mg / kg - d}) = \frac{C_w \times IR \times EF \times ED}{BW \times AT}$$

where:

C_w	=	Concentration of PCBs in water (mg/L)
IR	=	Ingestion rate (L/d)
EF	=	Exposure frequency (d/year)
ED	=	Exposure duration (years)
BW	=	Body weight (kg)
AT	=	Averaging time - period over which exposure is averaged (days); over a lifetime for evaluating cancer risks and over the appropriate exposure duration for evaluating non-cancer health hazards.

Exposure factor values for the central tendency and RME point estimate calculations for this pathway are summarized in Tables 2-26 through 2-28 (Appendix E). Site-specific considerations in selecting these factors are discussed below.

PCB Concentrations in River Water (C_w). As described in Section 6.2.3.3, the central tendency point estimates used for PCB concentration in the water column are 1.7×10^{-5}, 1.7×10^{-5}, and 1.5×10^{-5} mg/L, for children, adolescents, and adults, respectively. The RME point estimates are 1.4×10^{-5}, 1.2×10^{-5}, and 9.3×10^{-6} mg/L, for children, adolescents, and adults, respectively (Table 2-10).

Ingestion Rate (IR). For the residential scenarios, the 90th percentile and mean drinking water ingestion rates of 2.3 L/day and 1.4 L/day, respectively, were used for adults and adolescents to represent RME and central tendency exposures. Similarly the 90th percentile and mean drinking water ingestion rates of 1.5 L/day and 0.87 L/day were used to represent RME and central tendency exposures for children (USEPA, 1997c).

Exposure Frequency (EF). An exposure frequency of 350 days/year was assumed for residents of all ages.

Exposure Duration (ED). As described in the previous sections, the exposure duration for river water is 41 years, and the central tendency value is 11 years, which correspond to the 95th and 50th percentiles, respectively, of the residence duration determined for the six Mid-Hudson counties.

Body Weight (BW). Age-specific body weights were used. The mean body weight for children aged 1 to 6 is 15 kg, the mean body weight for adolescents aged 7-18 is 43 kg, and the mean adult body weight is 70 kg (USEPA, 1989a,b; 1997f).

Averaging Time (AT). For all residential exposure calculations, a 70-year lifetime averaging time of 25,550 days (365 days × 70 years) was used for cancer evaluations. Non-cancer averaging times are equal to the exposure duration multiplied by 365 days/year (USEPA, 1989a,b).

6.3 Toxicity Assessment

Potential non-cancer hazards and cancer risks posed by exposure to PCBs are evaluated using the most current published USEPA toxicity values, which are summarized in Tables 3-1 and 3-2 (Appendix E) and discussed briefly below. The reader is referred to Chapter 4, Section 5.3.2, and Appendix D for a thorough discussion of PCB toxicity.

6.3.1 Non-cancer Toxicity Values

The chronic RfD represents an estimate of a daily exposure level for the human population, including sensitive subpopulations, that is likely to be without an appreciable risk of deleterious effects during a lifetime. The IRIS database provides oral RfDs for two Aroclor mixtures, Aroclor 1016 and Aroclor 1254. The RfD for Aroclor 1016 is 0.00007 (7×10^{-5}) mg/kg-day, and 0.00002 (2×10^{-5}) for Aroclor 1254 (Table 3-1). There is no RfD available for Total PCBs and Aroclor 1248.

The PCB homologue distribution of sediment and water samples is predominately dichloro- through pentachlorobiphenyls, as reported in the Hudson River Data Evaluation and Interpretation Report (USEPA, 1997d). This distribution is more similar to Aroclor 1016 than to Aroclor 1254. Therefore, for

the purposes of this HHRA, the Aroclor 1016 RfD (7×10^{-5} mg/kg-day) was used to evaluate non-cancer toxicity for ingestion and dermal contact with Mid-Hudson River sediment and water.

The PCB homologue distribution in fish differs from the sediment and water samples due to differential bioaccumulation of PCB congeners with higher chlorination levels. Trichloro- through hexachlorobiphenyls contribute to the majority of fish tissue PCB mass as reported in the Revised ERA (USEPA, 2000f). This distribution is more similar to Aroclor 1254 than to Aroclor 1016. Therefore, the Aroclor 1254 RfD (2×10^{-5} mg/kg-day) was used to evaluate non-cancer toxicity for ingestion of Mid-Hudson River fish.

6.3.2 PCB Cancer Toxicity

The Cancer Slope Factor, or CSF, is a plausible upper bound estimate of carcinogenic potency used to calculate risk from exposure to carcinogens, by relating estimates of lifetime average chemical intake to the incremental risk of an individual developing cancer over a lifetime. In IRIS, both upper-bound and central-estimate CSFs are listed for three different tiers of PCB mixtures. Consistent with the recommended values in IRIS, the first tier upper-bound and central-estimate CSFs of 2.0 and 1.0 $(mg/kg\text{-}day)^{-1}$ are used to evaluate cancer risks for the upper-bound and central-estimate exposures to PCBs *via* ingestion of Mid-Hudson River fish, ingestion of Mid-Hudson River sediments, and dermal contact with Mid-Hudson River sediments. The second tier upper-bound and central-estimate CSFs of 0.4 and 0.3 $(mg/kg\text{-}day)^{-1}$ are used to evaluate cancer risks for the upper-bound and central-estimate exposures to PCBs *via* ingestion and dermal contact with Mid-Hudson River water (Table 3-2, Appendix E).

6.4 Risk Characterization

Risk characterization is the final step of the risk assessment process, which combines the information from the Exposure Assessment and Toxicity Assessment steps to yield estimated non-cancer hazards and cancer risks from exposure to PCBs. A detailed evaluation of the uncertainties underlying the risk assessment process is presented in Chapter 5 of this Revised HHRA. The risk characterization was prepared in accordance with USEPA guidance on risk characterization (USEPA, 1995; USEPA, 1992).

As described in the Chapter 4, some PCB congeners are considered to be structurally similar to dioxin and have been termed "dioxin-like" congeners. A cancer risk analysis for dioxin-like PCB congeners was not performed in the Mid-Hudson HHRA because the findings of the Upper Hudson River HHRA showed that risks from the dioxin-like PCB congeners are approximately equivalent to risks from total PCBs (see Chapter 5). It is expected that a similar finding would hold for the Mid-Hudson River, and in light of the lower concentration of PCBs in the Mid-Hudson River, cancer risks for dioxin-like PCB congeners were not evaluated in the Mid-Hudson HHRA.

6.4.1 Non-cancer Hazard Indices

The evaluation of non-cancer health effects involves a comparison of average daily exposure levels with established Reference Doses (RfDs) to determine whether estimated exposures exceed recommended limits to protect against chronic adverse health hazards. The hazard quotient is calculated by dividing the estimated average daily oral dose estimates by the oral RfD as follows (USEPA, 1989b):

$$Hazard\ Quotient\ (HQ) = \frac{Average\ Daily\ Dose\ (mg/kg-day)}{RfD\ (mg/kg-day)} \quad [6\text{-}1]$$

High-end and central tendency hazard quotients calculated for each exposure pathway (fish ingestion, sediment, and water exposure pathways) are summarized in Tables 4-1 through 4-10 (Appendix E). Hazard Quotients are summed over all COPCs (chemicals of potential concern) and all applicable exposure routes to determine the total Hazard Index (HI). In this Mid-Hudson HHRA, PCBs are the COPCs and the HQ for PCBs is equivalent to the HI. The total high-end and central tendency Hazard Indices for each pathway and receptor are summarized in Tables 4-21 through 4-27 (Appendix E).

If a Hazard Index is greater than one (*i.e.*, HI>1), unacceptable exposures may be occurring, and there may be concern for potential non-cancer effects, although the relative value of an HI above one (1) cannot be translated into an estimate of the severity of the hazard. Ingestion of fish results in the highest Hazard Index, with an HI of 3, 4, and 6 for the central tendency estimates for adult, adolescent, and child exposures, respectively, and an HI of 34, 37, and 53 for the high-end estimates for adult, adolescent, and child exposures, respectively. All cases represent exposures above the reference level (HI>1). Total Hazard Indices for the recreational and residential exposure pathways are all below one. In all cases, the Hazard Indices are based on uniform exposure throughout the Mid-Hudson River.

6.4.2 Cancer Risks

Cancer risks are characterized as the incremental increase in the probability that an individual will develop cancer during his or her lifetime due to site-specific exposure. The quantitative assessment of carcinogenic risks involves the evaluation of lifetime average daily dose and application of toxicity factors reflecting the carcinogenic potency of the chemical. A more detailed explanation of cancer risks can be found in Chapter 5.

The cancer risk is calculated by multiplying the estimated lifetime average daily oral dose estimates by the oral slope factor as follows (USEPA, 1989b):

$$Cancer\ Risk = Intake\left(\frac{mg}{kg-day}\right) \times CSF\left(\frac{mg}{kg-day}\right)^{-1} \quad [6\text{-}2]$$

High-end and central tendency cancer risk estimates calculated for each exposure pathway (fish ingestion, recreational and residential exposure pathways) are summarized in Tables 4-11 through 4-20 (Appendix E). Total cancer risks are summed over all applicable exposure routes and exposure periods (child through adult). The total RME and central tendency cancer risks for each pathway are summarized in Tables 4-21 through 4-27 (Appendix E).

Ingestion of fish results in the highest cancer risks, 1×10^{-5} (1 additional case of cancer in a population of one-hundred thousand) for the central tendency estimate, and 7×10^{-4} (7 additional cancers in a population of ten-thousand) for the high-end estimate. When broken down by age group, cancer risks of 6×10^{-6}, 3×10^{-6}, and 5×10^{-6} were calculated for central tendency adult, adolescent, and child exposures, respectively. Cancer risks of 3×10^{-4}, 2×10^{-4}, and 2×10^{-4} were calculated for RME adult, adolescent, and child exposures, respectively. As a further note on the fish ingestion risks, had the 95th percentile fish ingestion rate (63.4 g/day, or 102 meals per year) been used in the analysis, the RME risks for fish ingestion would approximately double (*e.g.*, 6×10^{-4} for adults).

6.5 Results

A summary of the point estimate cancer risk calculations is presented below. For fish consumption, the RME estimate of the increased risk of an individual developing cancer averaged over a lifetime is 1×10^{-5} for the central tendency estimate, and 7×10^{-4} for the high-end (RME) estimate. For known or suspected carcinogens, acceptable exposure levels for Superfund are generally concentration levels that represent an incremental upper bound lifetime cancer risk to an RME individual of between 10^{-4} and 10^{-6} (USEPA, 1990). The cancer risk associated with RME fish ingestion results falls somewhat outside the NCP acceptable cancer risk range. Estimated cancer risks relating to PCB exposure in sediment and water while swimming or wading, or from consumption of PCBs in drinking water by residents living near the river, are lower than those for fish ingestion, falling below the range of 10^{-4} to 10^{-6}. The central tendency cancer risks are provided to yield information on the variability/uncertainty of the health effects to individuals within the potentially exposed population.

Point Estimate Cancer Risk Summary

Pathway	Central Tendency Risk	RME Risk
Ingestion of Fish		
Total*	1×10^{-5} (1 in 100,000)	7×10^{-4} (7 in 10,000)
Adult	6×10^{-6} (6 in 1,000,000)	3×10^{-4} (3 in 10,000)
Adolescent	3×10^{-6} (3 in 1,000,000)	2×10^{-4} (2 in 10,000)
Child	5×10^{-6} (5 in 1,000,000)	2×10^{-4} (2 in 10,000)
Swimming/Wading Exposure to Sediment*	2×10^{-8} (2 in 100,000,000)	2×10^{-7} (2 in 10,000,000)
Swimming/Wading Exposure to Water*	9×10^{-9} (9 in 1,000,000,000)	6×10^{-8} (6 in 100,000,000)
Consumption of Drinking Water*	3×10^{-8} (3 in 100,000,000)	1×10^{-7} (1 in 10,000,000)

**Total risk for young child (aged 1-6), adolescent (aged 7-18), and adult (over 18).*

A summary of the point estimate non-cancer health hazards is presented below. Ingestion of fish results in the highest Hazard Index, with an HI of 3, 4, and 6 for the central tendency estimates for adult, adolescent, and young child exposures, respectively, and an HI of 34, 37, and 53 for the high-end estimates for adult, adolescent, and young child exposures, respectively. The total HIs for exposure to sediment and water air are all below one.

Point Estimate Non-Cancer Hazard Summary		
Pathway	**Central Tendency Non-Cancer Hazard Index**	**RME Non-Cancer Hazard Index**
Ingestion of Fish		
Adult	3	34
Adolescent	4	37
Young Child	6	53
Exposure to Sediment*	0.002	0.004
Exposure to Water*	0.005	0.007
Consumption of Drinking Water*	0.01	0.02

**Values for young child and adolescent, which are higher than adult for these pathways.*

MAJOR FINDINGS OF THE REVISED HHRA FOR THE MID-HUDSON RIVER

The Revised Mid-Hudson HHRA evaluated both cancer risks and non-cancer health hazards to young children, adolescents and adults posed by PCBs in the Mid-Hudson River. USEPA has classified PCBs as probable human carcinogens and known animal carcinogens. Other long-term adverse health effects of PCBs observed in laboratory animals include a reduced ability to fight infections, low birth weights, and learning problems. The major findings of the report are:

- Eating fish is the primary pathway for humans to be exposed to PCBs from the Mid-Hudson.
- Under the RME scenario for eating fish from the Mid-Hudson, the calculated total cancer risk (40 years of exposure apportioned as a young child, adolescent, and then adult) is 7×10^{-4}, or seven in 10,000. This excess cancer risk is 700 times higher than USEPA's goal of protection and seven times higher than the highest cancer risk level allowed under the federal Superfund law.
- For non-cancer health effects, the RME scenario for eating fish from the Mid-Hudson results in a Hazard Index to a young child of 53, a level of exposure to PCBs that is more than 53 times higher than USEPA's reference level (Hazard Index) of one. HIs for the adolescent and adult are 37 and 34, respectively, which are 37 and 34 times higher than the reference level of one.
- The RME cancer risks and non-cancer health hazards are expected to be above USEPA's generally acceptable levels for the 40-year exposure period that begins in 1999, assuming baseline conditions of no remediation and no institutional controls.
- The central tendency cancer risks and non-cancer health hazards from eating fish in the Mid-Hudson, are based on consumption of about one meal every two months, are also above USEPA's levels of concern.
- Cancer risks and non-cancer health hazards from being exposed to PCBs in the Mid-Hudson River through skin contact with contaminated sediments and river water, incidental ingestion of sediments, and consumption of river water as a drinking water source are generally within or below USEPA's levels of concern.

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