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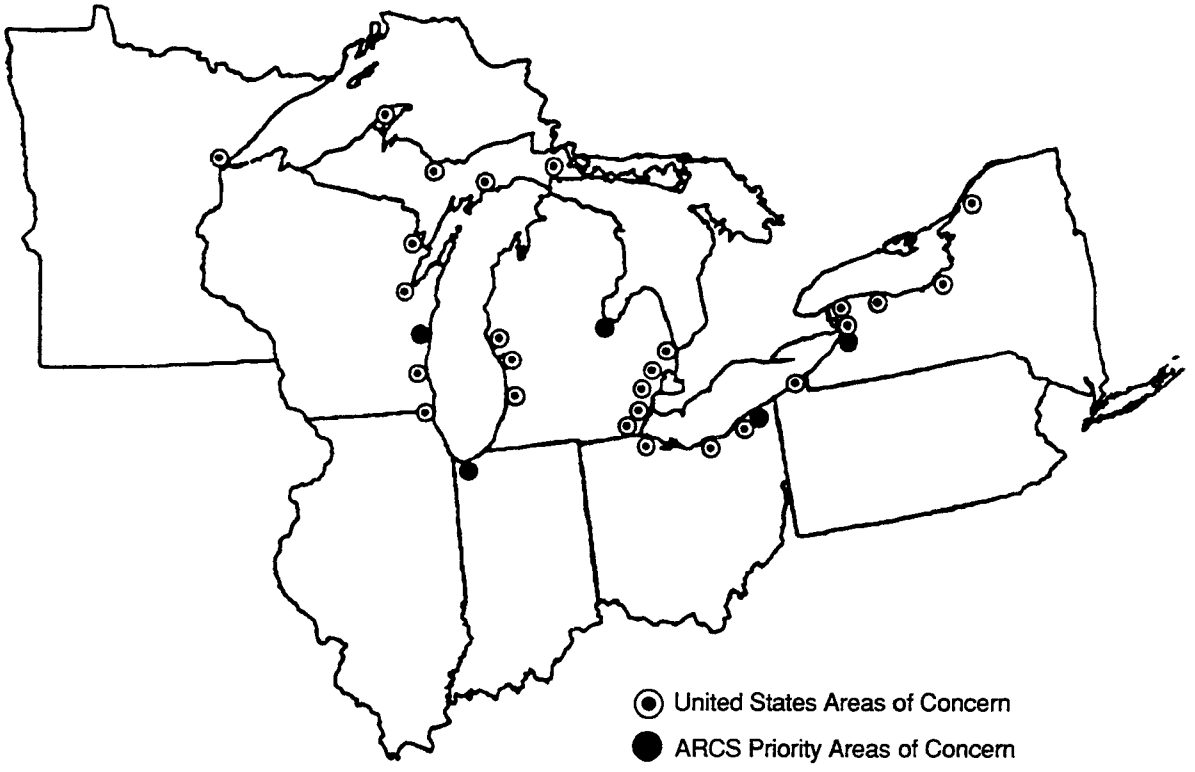
BASELINE HUMAN HEALTH RISK ASSESSMENT: ASHTABULA RIVER, OHIO, AREA OF CONCERN



BASELINE HUMAN HEALTH RISK ASSESSMENT: ASHTABULA RIVER, OHIO, AREA OF CONCERN

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FOREWORD

Risk assessment has been defined as the characterization of the probability of adverse effects from human and ecological exposures to environmental hazards. Risk assessments are quantitative, chemical-oriented characterizations that can use statistical and biological models to calculate numerical estimates of risk to human health or the environment. The concept of risk assessment is a cornerstone on which the U.S. Environmental Protection Agency builds programs to confront pollution problems in air, water, and soil under the direction of Congressional mandates. One such mandate is the Clean Water Act, which includes a directive to the Agency to study the control and removal of toxic pollutants in the Great Lakes, with emphasis on removal of contaminants from bottom sediments. Charged with performing this study is EPA's Great Lakes National Program Office (GLNPO) located in Chicago, IL. GLNPO administers the Assessment and Remediation of Contaminated Sediments (ARCS) program to examine the problem of contaminated sediments using a multidisciplinary approach involving engineering, chemistry, toxicology, modeling, and risk assessment.

In support of the GLNPO, the Environmental Research Laboratory-Athens began a series of studies under the ARCS program that will culminate in a baseline risk assessment for each of five Great Lakes Areas of Concern (AOC)--Buffalo River, NY, Grand Calumet River, IN, Saginaw River, MI, Ashtabula River, OH, and Sheboygan River, WI. This report describes a baseline human health risk assessment for the population within the Sheboygan River AOC. The assessment, which is based on available environmental data, is designed to provide a conservative estimate of carcinogenic and noncarcinogenic risks to human health under the baseline, no-action alternative.

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PREFACE

This risk assessment was prepared as part of the Assessment and Remediation of Contaminated Sediments (ARCS) program coordinated by the U.S. EPA Great Lakes National Program Office. The work by AScI Corporation was completed under contract no. 68-C1-0012 with the U.S. EPA Environmental Research Laboratory-Athens by Judy Crane, Ph.D. under the supervision of James L. Martin, Ph.D., P.E., AScI Site Manager. This work was performed through the U.S. EPA Center for Exposure Assessment Modeling, Mr. Robert Ambrose, Jr., P.E., Manager.

ABSTRACT

The Assessment and Remediation of Contaminated Sediments (ARCS) program, a 5-year study and demonstration project relating to the control and removal of contaminated sediments from the Great Lakes, is being coordinated and conducted by the U.S. Environmental Protection Agency's (EPA) Great Lakes National Program Office (GLNPO). As part of the ARCS program, baseline human health risk assessments are being performed at five Areas of Concern (AOCs) in the Great Lakes region. The Ashtabula River, located in northeastern Ohio, is one of these AOCs.

In this report, exposure and risk assessment guidelines, developed for the EPA Superfund program, have been applied to determine the baseline human health risks associated with direct and indirect exposures to sediment-derived contaminants in the Ashtabula River AOC. These risks were estimated for noncarcinogenic (e.g., reproductive toxicity, teratogenicity, liver toxicity) and carcinogenic (i.e., probability of an individual developing cancer over a lifetime) effects.

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ACKNOWLEDGMENTS

Several members of the Ashtabula River RAP Advisory Council provided useful information about the Ashtabula River Area of Concern including: Julie Letterhos (RAP Coordinator, Ohio EPA), Carl Anderson (Co-Chair), Leonard Eames, Karla Auker (Ohio EPA), and Jack Phelps (Co-Chair). In addition, Pete Redmon (U.S. EPA Region 5) and Rick Fox (Great Lakes National Program Office) supplied additional information about the Ashtabula River. Jack's Marine provided a courtesy boat tour of the lower Ashtabula River and Harbor. Members of the ARCS Risk Assessment and Modeling Work Group have provided useful feedback for the preparation of this risk assessment. James Martin (AScI Corp.), Bill Sutton (ERL-Athens), and Bob Swank (ERL-Athens) reviewed this document. Finally, I would like to thank Tawnya Robinson (AScI Corp.) for typing the tables in Chapter 3.

CHAPTER 1

EXECUTIVE SUMMARY

1.1 OVERVIEW

The Assessment and Remediation of Contaminated Sediments (ARCS) program, a 5-year study and demonstration project relating to the control and removal of contaminated sediments from the Great Lakes, is being coordinated and conducted by the U.S. Environmental Protection Agency's (EPA) Great Lakes National Program Office (GLNPO). As part of the ARCS program, baseline human health risk assessments are being performed at five Areas of Concern (AOCs) in the Great Lakes region. The Ashtabula River, located in northeastern Ohio, is one of these AOCs.

In this report, exposure and risk assessment guidelines, developed for the EPA Superfund program, have been applied to determine the baseline human health risks associated with direct and indirect exposures to sediment-derived contaminants in the Ashtabula River AOC. These risks were estimated for noncarcinogenic (e.g., reproductive toxicity, teratogenicity, liver toxicity) and carcinogenic (i.e., probability of an individual developing cancer over a lifetime) effects under different exposure scenarios.

1.2 STUDY AREA

The lower 3.2 km of the Ashtabula River and the Ashtabula Harbor have been severely impacted by industrial pollution, especially from contaminant loads transported into the river from Fields Brook, a Superfund site. Dredging of the federal navigation channel was stopped in 1964 due to severe contamination problems in the channel and to a lack of agreement among several agencies on how to safely dispose of the dredged sediments. Consequently, sediments have built up in the lower river to the point where it is becoming increasingly difficult for people with deep draft boats to navigate the river.

The contamination and sedimentation problems in this area have been of concern to the International Joint Commission (IJC); the IJC designated this region as an AOC in 1987. In response, the Ohio EPA has nearly completed the Stage One Remedial Action Plan (RAP) to identify contamination problems in the Ashtabula River AOC (Ohio EPA, 1991).

Within the AOC, the Ashtabula River is bordered by nine marinas and yacht clubs in the small city of Ashtabula. The lower river is used by recreational boaters and charter boat operators as an access point to Lake Erie. Sport fishing is very popular in the Ashtabula Harbor and in the nearshore Lake Erie area. The Ohio Department of Health and Ohio EPA issued a fish advisory in 1983

recommending that no fish caught in the river from the 24th St. Bridge to the harbor mouth be eaten (Woodward-Clyde Consultants, 1991). Despite these warnings, some people still fish in the AOC.

1.3 EXPOSURE ASSESSMENT

This assessment focused on only one pathway by which residents of the lower Ashtabula River were likely to be exposed to sediment-derived contaminants: the consumption of contaminated fish. Other exposure pathways were determined to be either incomplete (e.g., ingestion of sediments) or insignificant in terms of risk (e.g., ingestion of surface water during infrequent swimming events).

Woodward-Clyde Consultants (WCC) conducted the most recent survey (1990) of contaminant levels in fish inhabiting the Ashtabula River AOC. In this study, 12 carp, 16 small/large mouth bass, and 16 bluegill were collected from four sites in the AOC and were analyzed for a variety of contaminants. Data obtained from composite samples of fish collected from two of the sites were used in the exposure assessment: 1) the Ashtabula River just downstream from Fields Brook (3 carp, 2 large mouth bass, and 11 bluegills), and 2) the Ashtabula Harbor (4 carp, 1 small mouth plus 3 large mouth bass, and 5 bluegills). The carp were analyzed as whole fish while the other two species were analyzed as skin-on fillets. The collection and data analysis of the fish appears to have gone through a rigorous QA/QC program at WCC. Thus, the data were deemed usable for this baseline risk assessment.

Noncarcinogenic and carcinogenic risks were estimated for three different exposure scenarios: typical (average), reasonable maximum (i.e., the maximum exposure that is reasonably expected to occur at a site), and subsistence exposures. The subsistence pathway was chosen for a small segment of the population that may be relying on the consumption of fish from the area for their main source of protein. Different consumption rates were applied to each scenario (Table 1.1), and it was assumed that only fish collected and consumed from the Ashtabula River AOC were contaminated. In addition, the exposure duration varied with the exposure scenario. Typical exposures were assumed to occur over a period of 9 years; reasonable maximum and subsistence exposures were assumed to occur over a period of 30 years. Noncarcinogenic effects were averaged over the same time period as the exposure duration, whereas carcinogenic effects were averaged over a lifetime (i.e., 70 years). For all three exposure scenarios, exposures were determined for each chemical and added for each pathway. This assumption of additivity did not account for any synergistic or antagonistic effects that might occur among chemicals.

Several heavy metals and organic compounds that were detected in some or all of the fish samples were included in the exposure assessment (i.e., chromium, copper, mercury, silver, zinc, polychlorinated biphenyls (PCBs), 1,1,2,2-

TABLE 1.1. AMOUNT OF FISH ASSUMED TO BE CONSUMED PER PERSON PER DAY FROM THE ASHTABULA RIVER AOC

Exposure Scenario	Total Amount of Fish Consumed* (g/day)	x FI** =	Amount of Ashtabula R. Fish Consumed (g/day)
Typical	19.2	0.10	1.9
Reasonable Maximum	54	0.25	13.5
Subsistence	132	0.7	92.4

* Sources: Typical (West et al., 1989); Reasonable Maximum (USEPA, 1991a); Subsistence [Pao et al. (1982) cited in USEPA (1989a)]

** FI = fraction of fish ingested from the Ashtabula River (study assumption)

tetrachloroethane, tetrachloroethene, and trichloroethene). In addition, noncarcinogenic and/or carcinogenic toxicity values were either available or under review for this set of contaminants. Thus, the exposure and toxicity information could be integrated into the risk assessment.

1.4 RISK ASSESSMENT

1.4.1 Determination of Risk

This baseline risk assessment did not characterize absolute human health risks, rather it identified potential sources of unacceptable risks. Risk estimates were determined for both noncarcinogenic and carcinogenic endpoints.

Noncarcinogenic effects were evaluated by comparing an exposure level over a specified time period with a reference dose (RfD)[1] derived from a similar

[1] The RfD provides an estimate of the daily contaminant exposure that is not likely to cause harmful effects during either a portion of a person's life or their entire lifetime (USEPA, 1989a).

exposure period (otherwise known as a hazard quotient (HQ)). Thus, HQ = exposure level/RfD. An HQ value of less than 1 indicates that exposures are not likely to be associated with adverse noncarcinogenic effects. HQ values between 1 and 10 may be of concern, particularly when additional significant risk factors are present (e.g., other contaminants at levels of concern) (USEPA, 1988a). The sum of more than one HQ value for multiple substances and/or multiple exposure pathways is represented by the Hazard Index (HI).

Carcinogenic risks were estimated as the incremental probability of an individual developing cancer over a lifetime as a result of exposures to potential carcinogens. This risk was computed using average lifetime exposure values that were multiplied by the oral slope factor[2] for a particular chemical. The resulting carcinogenic risk estimate generally represents an upper-bound estimate, because slope factors are usually based on upper 95th percentile confidence limits. Carcinogenic effects were summed for all chemicals in an exposure pathway. This summation of carcinogenic risks assumed that intakes of individual substances were small, that there were no synergistic or antagonistic chemical interactions, and that all carcinogens produced the same effect (i.e., cancer). The EPA believes it is prudent public health policy to consider actions to mitigate or minimize exposures to contaminants when estimated excess lifetime cancer risks exceed the 10^{-5} to 10^{-6} range, and when noncarcinogenic health risks are estimated to be significant (USEPA, 1988a).

1.4.2 Noncarcinogenic Risks

Noncarcinogenic risks, as represented by the Hazard Index (HI), were below levels of concern (i.e., less than 1) for most of the typical and reasonable maximum exposure scenarios (Table 1.2). For fish collected from the Ashtabula Harbor, only the consumption of whole carp under the subsistence exposure scenario resulted in a significant risk. The subsistence consumption of large mouth bass fillets, bluegill fillets, and whole carp collected from below Fields Brook could pose a potential noncarcinogenic risk to anglers and their families; the reasonable maximum consumption of carp at this site was also of concern. The estimated risks were mostly attributable to methyl mercury and copper contamination. Methyl mercury has been shown to cause central nervous system effects in humans at the lowest adverse effect level of 0.003 mg/kg/day (IRIS data baseretrieval for methyl mercury, 1992). Information about the types of noncarcinogenic effects one might experience from chronic exposure to copper is not available at this time from the IRIS data base.

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[2] Slope factors are estimated through the use of mathematical extrapolation models, most commonly the linearized multistage model, for estimating the largest possible linear slope (within 95% confidence limits) at low extrapolated doses that is consistent with the data (USEPA, 1989a).

TABLE 1.2. NONCARCINOGENIC RISKS BASED ON THE HAZARD INDEX (HI) FOR EACH EXPOSURE SCENARIO

	Exposure Scenario		
Site and Fish Type	Typical	RME*	Subsistence
Ashtabula Harbor			
S/L Mouth Bass Fillet	0.02	0.1	0.9
Bluegill Fillet	0.02	0.1	0.8
Whole Carp	0.05	0.3	2
Ashtabula R. (Downstream from Fields Brook)			
L. Mouth Bass Fillet	0.02	0.1	1
Bluegill Fillet	0.04	0.2	2
Whole Carp	0.4	3	20

* RME = Reasonable Maximum Exposure

1.4.3 Carcinogenic Risks

A carcinogenic risk estimate could not be calculated for the consumption of small/large mouth bass collected from the Ashtabula Harbor and for bluegills collected from both the river and harbor; this was because no carcinogens were detected in these fish fillets (Table 1.3). The upper-bound carcinogenic risk estimates associated with the consumption of large mouth bass fillets collected below Fields Brook were below concern levels (i.e., less than 10^{-6}) under all three exposure scenarios. Methylene chloride was the only carcinogen detected in the bass for which a toxicity value was available. The consumption of whole carp was of concern at both the harbor and river under all three exposure scenarios. The carcinogenic risk from consuming carp was attributable to PCB contamination. There is a possibility that people who ingest, inhale, or have dermal contact with certain PCB mixtures may have a greater chance of incurring liver cancer; however, this statement is based on suggestive evidence rather than on verified data (IRIS data base retrieval for PCBs, 1992).

The human health risks attributable to carp consumption were probably overestimated because the risk estimates were based on data derived from whole carp instead of fillets. In addition, the data were also based on raw fish; different

TABLE 1.3. CARCINOGENIC RISKS FOR THE CONSUMPTION OF FISH IN THE ASHTABULA RIVER AOC

Site and Fish Type	Exposure Scenario		
	Typical	RME*	Subsistence
Ashtabula Harbor			
S/L Mouth Bass Fillet	-	-	-
Bluegill Fillet	-	-	-
Whole Carp	4E-06	9E-05	6E-04
Ashtabula R. (Downstream from Fields Brook)			
L. Mouth Bass Fillet	5E-09	1E-07	7E-07
Bluegill Fillet	-	-	-
Whole Carp	2E-05	5E-04	3E-03

* RME = Reasonable Maximum Exposure

preparation and cooking techniques may reduce concentrations of hydrophobic organic contaminants (e.g., PCBs) in fish if the fat is trimmed away prior to cooking.

1.4.4 Uncertainties

Several assumptions and estimated values were used in this baseline risk assessment that contributed to the overall level of uncertainty associated with the noncarcinogenic and carcinogenic risk estimates. As with most environmental risk assessments, the uncertainty of the risk estimates probably varied by around an order of magnitude or greater. The uncertainties were addressed in a qualitative way for the parameters and assumptions that appeared to contribute the greatest degree of uncertainty. One of the greatest sources of uncertainty was the assumption that exposure intakes and toxicity values would not change during the exposure duration. This assumed that human activities and contaminant levels would remain the same over the exposure duration, and that toxicity values would not be updated.

CHAPTER 2

INTRODUCTION

Sediments in the Great Lakes have become a repository for a variety of nutrients and contaminants, mostly as a result of industrial and municipal pollution. More stringent pollution control measures have generally reduced point sources of contamination during the past twenty years. However, problems remain with nonpoint sources of pollution (ranging from agricultural runoff to groundwater contamination) and with permit violations of effluent dischargers. In some areas of the Great Lakes, contaminated sediments now represent the primary source of anthropogenic chemicals to the aquatic environment. Consequently, concern has been raised about what remediation measures, if any, are needed to deal with the problem of contaminated sediments. In addition, these contaminants may pose a potential health risk to aquatic life, wildlife, and to human populations residing in the area of concern.

The 1987 amendments to the Clean Water Act, in Section 118(c)(3), authorize the U.S. Environmental Protection Agency's (EPA) Great Lakes National Program Office (GLNPO) to coordinate and conduct a 5-year study and demonstration project relating to the control and removal of contaminated sediments from recommended areas in the Great Lakes region. To achieve this task, GLNPO has initiated the Assessment and Remediation of Contaminated Sediments (ARCS) program. The overall objectives of the ARCS program (USEPA, 1991b), for selected Areas of Concern (AOCs), are to:

1. Assess the nature and extent of contaminated sediments,

2. Evaluate and demonstrate remedial options (e.g., removal, immobilization, and advanced treatment technologies) as well as the "no action" alternative,

3. Provide risk assessments for humans, aquatic life, and wildlife exposed to sediment-related contaminants, and

4. Provide guidance on the assessment of contaminated sediment problems and on the selection and implementation of necessary remedial actions in the Areas of Concern and other locations in the Great Lakes.

As one part of the ARCS program, baseline human health risk assessments for exposure to sediment-derived contaminants are being prepared for five AOCs: Ashtabula River, OH; Buffalo River, NY; Grand Calumet River/Indiana Harbor Canal, IN; Saginaw River, MI; and Sheboygan River, WI (Figure 2.1). The objectives of these risk assessments are to: 1) estimate the magnitude and

frequency of human exposures to sediment-derived contaminants in the AOC, and 2) estimate the risk of adverse effects resulting from both typical and reasonable maximum exposures (i.e., the highest exposure that is reasonably expected to occur at a site) to contaminants. Risk estimates are determined for both noncarcinogenic (i.e., chronic or subchronic effects) and carcinogenic (i.e., probability of an individual developing cancer over a lifetime) effects resulting from direct and indirect exposures to sediment-related contaminants. These risk estimates are made using conservative assumptions about exposure scenarios when complete data are not available. Thus, the risk estimates are designed to be overprotective of human health.

This document presents a baseline human health risk assessment for the Ashtabula River AOC. The next chapter describes the AOC and its contamination problems. Successive chapters describe the risk assessment framework and provide details on how the exposure and risk estimates were generated. The final chapter gives a qualitative assessment of the uncertainties associated with the risk estimates.

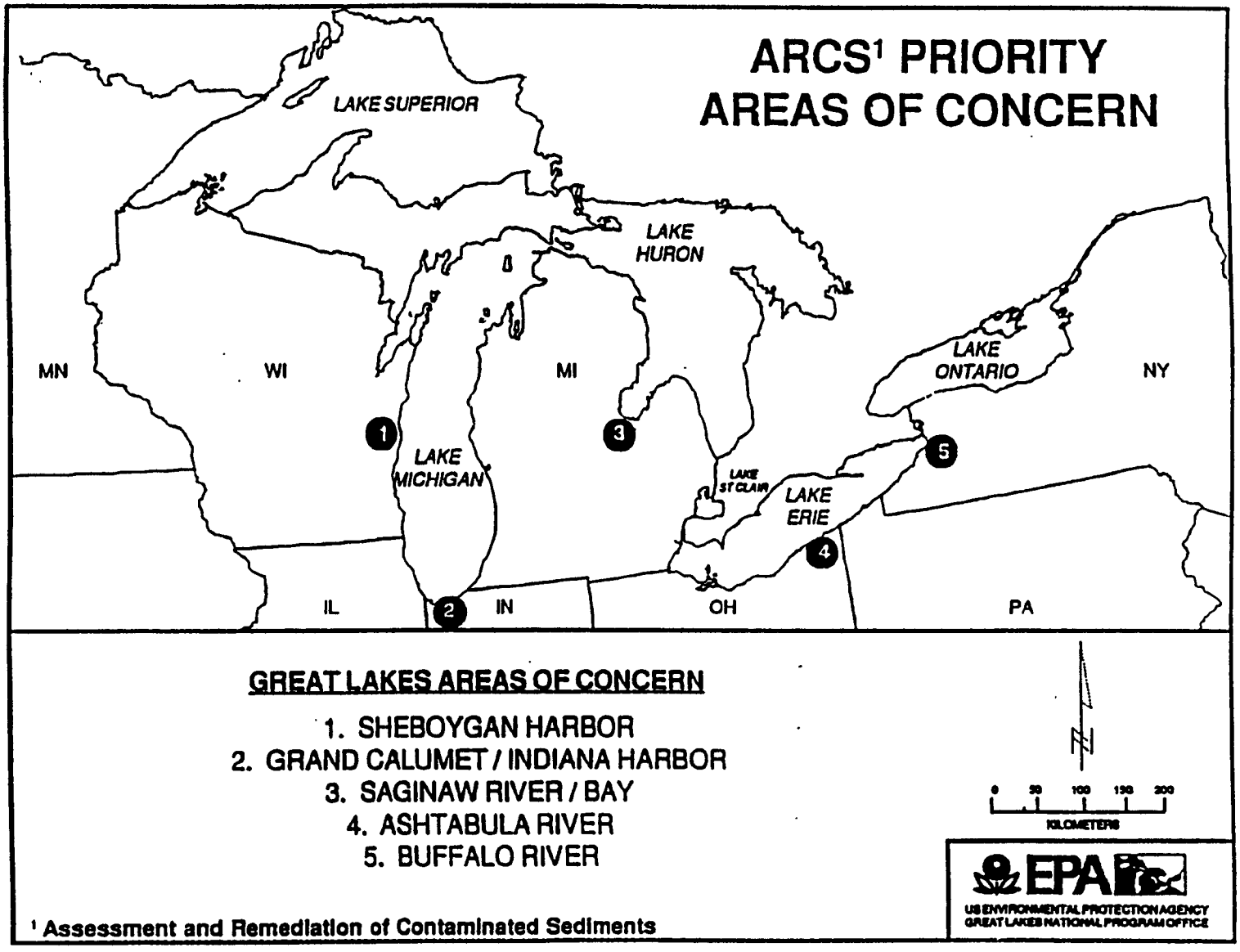

Figure 2.1. Map of ARCS priority Areas of Concern (USEPA, 1991b).

CHAPTER 3

ASHTABULA RIVER AREA OF CONCERN

3.1 ENVIRONMENTAL SETTING

The lower 3.2 km of the Ashtabula River and the Ashtabula Harbor in northeastern Ohio (Figure 3.1) have been identified as one of 43 Great Lakes AOCs by the International Joint Commission. This ranking is given to areas where environmental quality is degraded and designated uses of the water are impaired. The Ashtabula River AOC has been severely impacted by industrial pollution, especially from contaminant loads transported into the river from Fields Brook, a Superfund site. A Stage One Remedial Action Plan (RAP) for the Ashtabula River AOC has been prepared by the Ohio EPA, in cooperation with the Ashtabula River RAP Advisory Council, to identify impaired uses in the AOC (Ohio EPA, 1991). The RAP will also serve as a guidance document for future remedial clean-up measures. The RAP was the major source of information for this chapter, and the reader should refer to it for a thorough description of the physical site as well as for detailed information on the sources and extent of multimedia contamination in the AOC.

Within the AOC, the Ashtabula River is bordered by nine marinas and yacht clubs in the small city of Ashtabula (Figure 3.2). The lower river is used by recreational boaters and charter boat operators as an access point to Lake Erie. However, it is becoming increasingly difficult for people with deep draft boats to navigate the lower river due to the accumulation of sediments in the channel; in some areas, the water column is only 0.6 m deep (Ohio EPA, 1991). The river has not been dredged for about 30 years because of a decline in commercial shipping in the area (Ohio EPA, 1991). In addition, a suitable solution has not been reached between the U.S. Army Corps of Engineers, U.S. EPA, City of Ashtabula, and other parties for deciding how to dredge and dispose of contaminated sediments in the AOC.

The hydrologic and topographic features of the Ashtabula River have enhanced the settling and deposition of suspended particulate matter (SPM) (and associated contaminants) to the sediments. Low flow conditions prevail approximately 90% of the time in the river with an average flow of 4.5 cubic meters per second (Ohio EPA, 1991). In addition, the AOC lies within the former lake bottom of Lake Erie where the vertical gradient in this 4.8 to 8 km wide band is only 76 cm per km (Ohio EPA, 1991). Thus, a net accumulation of sediments arises from these conditions of low flow, narrow vertical gradient, and high SPM load. The resuspension of these sediments by boat propellers or wind induced waves may be important mechanisms for reintroducing contaminants back into the water column.

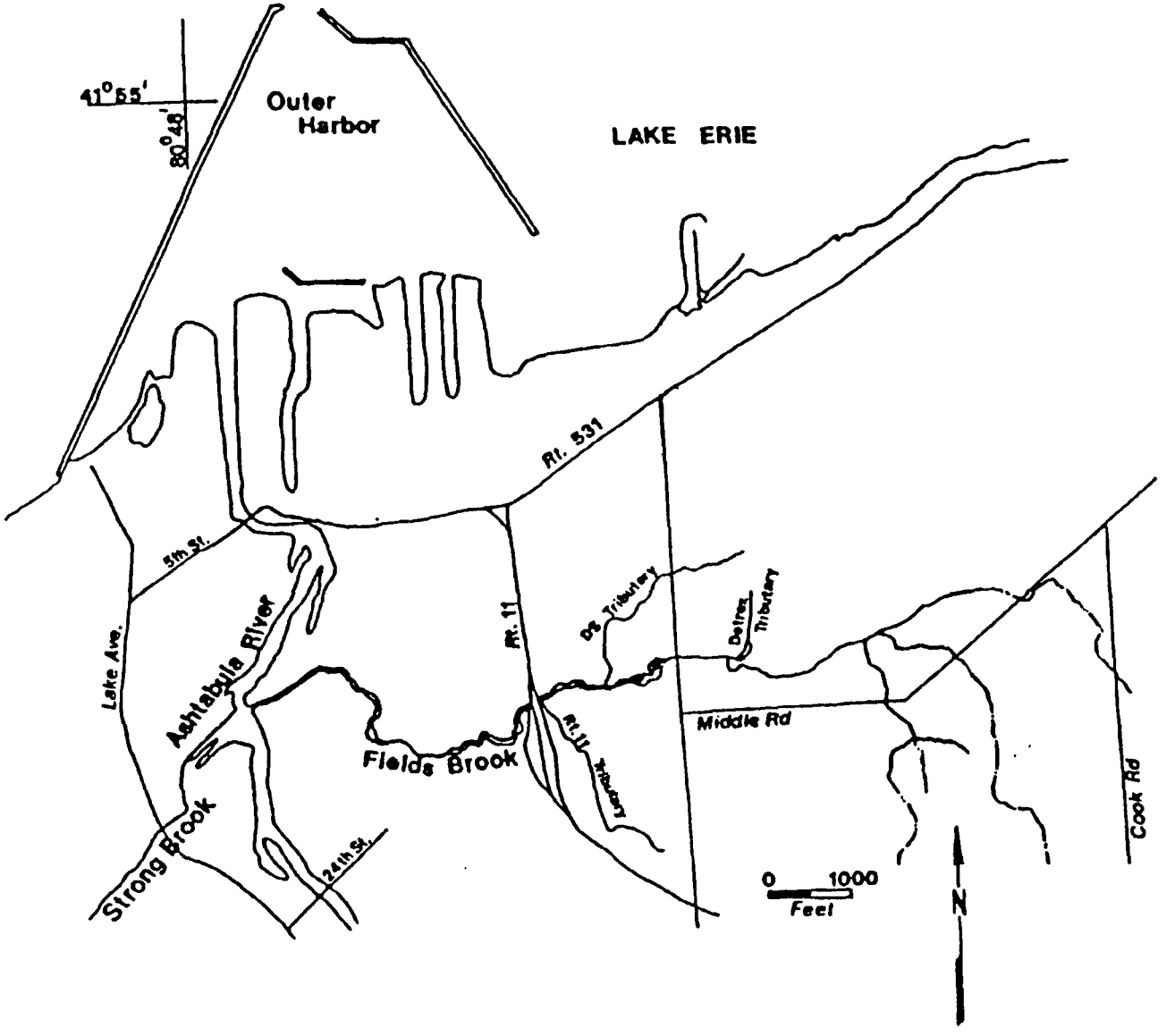

Figure 3.1. Boundaries of the Ashtabula River Area of Concern (Ohio EPA, 1991).

1. Ashtabula Public Water Supply Intake
2. CEI Intake
3. ASHCO Intake
4. Walnut Beach
5. Lakeshore Park
6. Point Park
7. Sutherland Marine
8. Marshall Marine
9. Ashtabula Yacht Club
10. Kister Marine
11. Jack's Marine North
12. Riverside Yacht Club
13. Harbor Yacht Club
14. Jack's Marine
15. Recreation Unlimited
16. Community Boating Center

* Water Supply Intake
● Recreational Facility

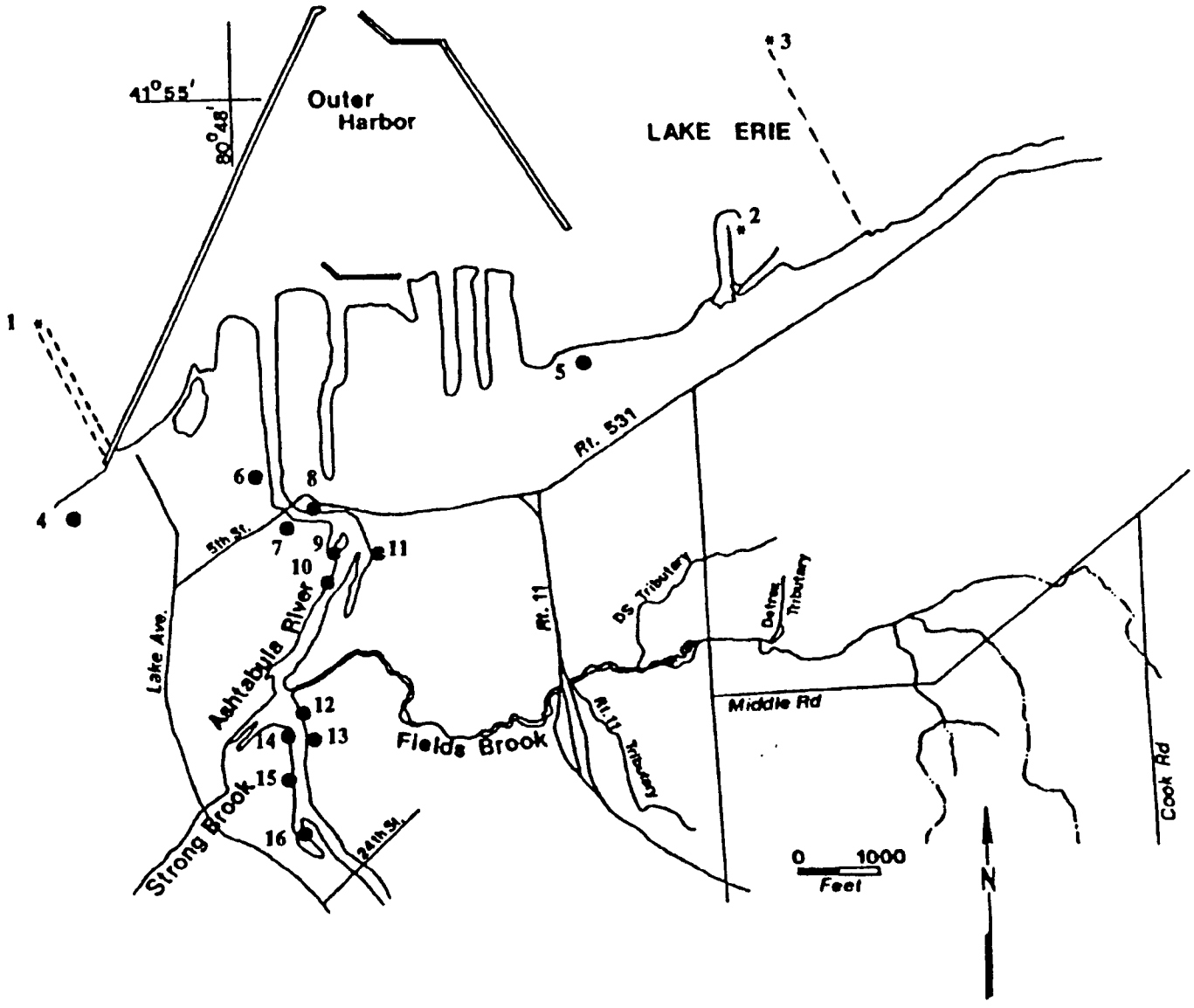

Figure 3.2. Location of water supply intakes and recreational facilities in the Ashtabula River AOC (Ohio EPA, 1991).

Most of the Ashtabula River discharge flows north through the Ashtabula Harbor mouth to an area adjacent to the Lake Erie Central Basin sediment depositional area [Thomas and Mudrock (1979) cited in the RAP (Ohio EPA, 1991]. The river discharge typically remains within 2.4 km of the shoreline and moves eastward due to prevailing southwest winds [U.S. Department of Health, Education, and Welfare (1965) cited in Ohio EPA (1991)]. Thus, suspended sediment loads (and associated contaminant loads) transported from the Ashtabula River may be deposited in the nearshore area of Lake Erie. Although the nearshore area is included in the AOC of the RAP, it will not be included in this risk assessment because of insufficient information on contaminant levels in the water column and sediments.

The Ashtabula River drainage basin (approximately 355 km^2) is the smallest watershed of all the major tributaries entering Lake Erie (Ohio EPA, 1991). The basin includes mostly rural and agricultural land in northeastern Ohio. The city of Ashtabula, with a 1990 census population of 21,633, is the only urban area in the watershed that has contributed extensively to contamination in the AOC (Ohio EPA, 1991). The industrial zone of Ashtabula is concentrated around Fields Brook and is dominated by several chemical industries and waste disposal sites (Figure 3.3). Another industrial area exists by the river mouth where large quantities of coal are stored at the Conrail coal dock; coal is the major commodity shipped from Ashtabula. The next section will describe some of the contamination problems in the Ashtabula River AOC arising from this industrial development.

3.2 CONTAMINATION PROBLEMS

The Ashtabula River AOC has a history of contamination problems due to previously unregulated industrial and municipal discharges of metals and organic chemicals into Fields Brook and the Ashtabula River beginning in the late 1940s (Ohio EPA, 1991). The greatest source of contamination to the AOC today is through the release of in-place pollutants contained in the sediments (Ohio EPA, 1991). Other secondary sources include point sources (e.g., effluent discharges, combined sewer overflows) and nonpoint sources (e.g., industrial and agricultural runoff). The location of point source dischargers in the Ashtabula River AOC is shown in Figure 3.3, and their recent record of compliance with final NPDES permit limits is given in Table 3.1. Fines have been levied against some of the industries who have violated their NPDES permits.

Twenty-four unregulated hazardous waste sites have been identified in the Ashtabula River AOC (Ohio EPA, 1991), and runoff from these waste sites could contribute to the contaminant load in Fields Brook and the Ashtabula River. Strong Brook, a small tributary entering the Ashtabula River west of Jack's Marine, has been an important source of oil and grease, lead, and zinc in the past [Aqua Tech (1979) cited in Ohio EPA (1991)]. The highest concentrations of zinc and lead in the sediments of the AOC are still found in Strong Brook (WCC, 1991).

TABLE 3.1. CONTINUED

	Discharger	Compliance	Action
18.	Vygen	No	Currently operating under interim limits. Compliance schedule in permit.
19.	SCM #1	No	Findings and Orders - 4/91
20.	SCM #2	No	Findings and Orders - 4/91

Thus, this small brook may continue to provide a source of zinc and lead to the rest of the AOC.

Other possible sources of contamination to the Ashtabula River AOC do not appear to be important. Groundwater contamination has not been observed to intrude on the Ashtabula River; this is because there is little groundwater recharge to the river due to the low permeability of surface deposits (Ohio EPA, 1991). Agricultural runoff and other upstream watershed inputs do not appear to contribute substantially to contaminant loads in the river although much of the sediments in the AOC have originated from these upstream sources. Regular air quality standards (i.e., ozone, total suspended particulates, sulfur dioxide, nitrogen dioxide, carbon monoxide, and lead) have not been violated in Ashtabula County (Ohio EPA, 1991). It is unlikely that atmospheric contamination contributes significantly to the contaminant burden in the AOC because of the small size of the watershed. In addition, no studies have been conducted to evaluate whether the AOC itself may serve as a source of contaminants to the atmosphere via the volatilization of hydrophobic organic compounds (HOCs) (e.g., PCBs).

A number of contaminants have been detected in the sediments, water column, and fish tissue collected from the Ashtabula River (Table 3.2) (Ohio EPA, 1991). The sediments have become a repository for metals, pesticides, nutrients, PCBs, polyaromatic hydrocarbons (PAHs), and a number of other chlorinated organic compounds. The contaminants of greatest concern in the AOC, because of their prevalence and potential toxicity, include PCBs, mercury, zinc, hexachlorobenzene, hexachlorobutadiene, chromium, and volatile organic compounds (Ohio EPA, 1991).

While a portion of these contaminants will become permanently buried in the sediments, contaminants in the surface sediment layer may be released to the water column through processes such as bioturbation, molecular diffusion, and resuspension. Furthermore, ionic species of chemicals (e.g., metals) in the sediments may undergo changes in solubility due to changes in reaction kinetics, pH, redox conditions, and other variables. In addition, benthic (i.e., bottom

TABLE 3.2. POLLUTANTS IDENTIFIED IN THE ASHTABULA RIVER AREA OF CONCERN SINCE 1975 (Y = YES; N = NO; X = POLLUTANT DETECTED IN MEDIUM) (ADAPTED FROM THE STAGE ONE RAP (OHIO EPA, 1991))

PARAMETER	PRIORITY POLLUTANT	WATER			SEDIMENT			FISH
		OUTER HARBOR	ASHTABULA RIVER	FIELDS BROOK	OUTER HARBOR	ASHTABULA RIVER	FIELDS BROOK	
Inorganics								
Aluminum	N	X	X	X	X	X	X	-
Arsenic	Y	-	-	-	X	X	X	X
Barium	N	X	X	X	X	X	X	-
Beryllium	Y	-	-	-	-	X	X	X
Cadmium	Y	X	X	X	X	X	X	-
Chromium	Y	X	X	X	X	X	X	-
Copper	Y	X	X	X	X	X	X	X
Cyanide	Y	-	X	-	X	X	X	-
Iron	N	X	X	X	X	X	X	-
Lead	Y	X	X	X	X	X	X	X
Manganese	N	X	X	X	X	X	X	-
Mercury	Y	X	X	X	X	X	X	X
Nickel	Y	X	-	-	X	X	X	-
Nitrogen (Ammonia)	N	X	X	X	X	-	-	-
Nitrate + Nitrite	N	X	X	X	X	-	-	-
Phosphorus	N	X	X	X	X	-	-	-
Oil and Grease	N	-	-	-	X	X	-	-
Silver	Y	-	-	-	-	X	X	X
Zinc	Y	X	X	X	X	X	X	X
Total Dissolved Solids	N	X	X	X	-	-	-	-
Phenols	Y	-	-	-	X	X	-	-
Organics								
Aldrin + Dieldrin	Y	X	X	-	-	X	-	-
PCBs	Y	-	-	-	X	X	X	X

TABLE 3.2. CONTINUED

PARAMETER	PRIORITY POLLUTANT	WATER			SEDIMENT			FISH
		OUTER HARBOR	ASHTABULA RIVER	FIELDS BROOK	OUTER HARBOR	ASHTABULA RIVER	FIELDS BROOK	
PAHs								
Acenaphthene	Y	-	-	-	X	-	-	-
Anthracene	Y	-	-	-	X	X	-	-
Benzo(a)anthracene	Y	-	-	-	X	X	-	-
Benzo(a)pyrene	Y	-	-	-	X	X	X	-
Benzo(b)fluoroanthene	Y	-	-	-	X	X	-	-
Chrysene	Y	-	-	-	X	X	-	-
Fluoranthene	Y	-	-	-	X	X	X	X
Fluorene	Y	-	-	-	X	X	-	-
Naphthalene	Y	-	-	X	X	X	-	-
Phenanthrene	Y	-	-	-	X	X	X	-
Pyrene	Y	-	-	-	X	X	-	-
2-chloronaphthalene	-	-	-	-	-	X	-	-
Benzo(k)fluoranthene	Y	-	-	-	-	X	-	-
Other Organics								
Acetone	N	-	-	-	-	X	X	-
Benzene	Y	-	-	-	-	-	X	-
Bis(2-ethylhexyl phthalate	Y	-	-	-	X	X	X	-
2-butanone	N	-	-	-	-	X	X	-
Butylbenzyl phthalate	Y	-	-	-	-	X	X	-
Chlorobenzene	Y	-	-	-	-	X	-	-
Chloroform	Y	-	-	X	-	-	X	-
1,1-dichloroethene	Y	-	-	-	-	-	X	-
Diethyl phthalate	Y	-	-	X	-	-	X	-
Dimethyl phthalate	Y	-	-	-	-	X	X	-
Di-n-butyl phthalate	Y	-	-	-	-	X	X	-

TABLE 3.2. CONTINUED

PARAMETER	PRIORITY POLLUTANT	WATER			SEDIMENT			FISH
		OUTER HARBOR	ASHTABULA RIVER	FIELDS BROOK	OUTER HARBOR	ASHTABULA RIVER	FIELDS BROOK	
Other Organics (Continued)								
Ethylbenzene	Y	-	-	-	-	X	X	-
Fluorotrichloromethane	N	-	-	-	-	-	X	-
Hexachlorobenzene	Y	-	-	X	-	X	X	X
Hexachlorobutadiene	Y	-	-	X	-	X	X	X
Hexachloroethane	Y	-	-	-	-	X	X	-
Methylene chloride	Y	-	X	X	X	X	X	-
1,2-Dichlorobenzene	Y	-	-	X	-	-	-	-
1,3-Dichlorobenzene	Y	-	-	X	-	X	-	-
1,4-Dichlorobenzene	Y	-	-	X	-	X	-	-
n-nitrosodiphenylamine	Y	-	-	X	-	-	-	-
Carbon tetrachloride	Y	-	-	X	-	-	-	-
Octachlorostyrene	N	-	-	-	-	X	-	X
Xylene	N	-	-	-	-	X	X	-
Pentachlorobenzene	N	-	-	-	-	-	-	X
1,1,1,2-tetrachloroethane	N	-	-	-	-	X	-	-
1,1,2,2-tetrachloroethane	Y	X	X	X	-	-	X	-
Tetrachloroethene	Y	X	X	X	-	X	X	X
1,2-transdichloroethene	N	-	-	X	-	-	X	-
1,1,2-trichloroethane	Y	-	-	X	-	-	X	-
1,1,1-trichloroethane	Y	-	-	-	-	-	X	-
Trichloroethene	Y	X	X	X	-	X	X	X
Toluene	Y	-	-	-	-	X	X	-
Vinyl chloride	Y	-	-	-	-	-	X	-
1,2,4-trichlorobenzene	N	-	-	-	-	X	-	-
1,2-dichloroethane	Y	-	-	X	-	-	-	-
1,3,5-trichlorobenzene	N	-	-	-	-	-	-	-
1,2,3,4-tetrachlorobenzene	N	-	-	-	-	-	-	-

dwelling) organisms may ingest sediments while feeding which may result in biological transformations of some contaminants. Thus, the cycling of these contaminants in the environment will affect their availability for biotic uptake and human exposure.

The physical-chemical properties of the contaminants detected in the Ashtabula River AOC affect their fate in the environment. Hydrophobic organic compounds, like PCBs, are especially persistent and ubiquitous in the environment due to their low aqueous solubilities, high octanol-water partition coefficients, high molecular weights, etc. HOCs preferentially partition to organic-rich particles in the water column and sediment, but will also partition to a lesser extent to dissolved and colloidal phases in the porewater and water column. Thus, HOCs are often difficult to detect in the water column because they are usually present at very low concentrations. Since HOCs preferentially partition into the lipids of organisms and will biomagnify through the higher orders of the food chain, fish can provide a good indication of contamination problems in the area.

Unlike HOCs, ionic (e.g., metal species) and polar compounds are more susceptible to the solvating properties of water. However, metal complexes can precipitate out of the water column under certain conditions, and both ionic and polar compounds may become associated with suspended particulate matter which may also settle out of the water column. Besides settling, contaminants may undergo a variety of other processes (e.g., volatilization, photolysis, advective transport, uptake into biota, hydrolysis, oxidation, microbial biotransformation) depending on the physical-chemical properties of the chemical. It is beyond the scope of this risk assessment to describe the importance of these mechanisms in the Ashtabula River and Harbor.

Contamination of the water column by metals has been a problem for some areas of the Ashtabula River AOC. Ambient Water Quality Standards (WQS) have recently been violated in the Ashtabula River for copper, cadmium, iron, lead, and zinc (Table 3.3) (Ohio EPA, 1991). However, the Ashtabula River is not used as a drinking water source, and these violations do not appear to pose an immediate threat to human health.

The upper turning basin, including the area near the mouth of Fields Brook, is the most contaminated area of the Ashtabula River (Figure 3.4). Most of the toxic sediments in the AOC are found in this area and are covered by 1.2 to 3.6 m of moderately to heavily polluted sediments. Current U.S. EPA classification lists the upper turning basin and area immediately downstream from Fields Brook as toxic, most of the river channel as heavily polluted, and the outer harbor as non-to-moderately polluted (Ohio EPA, 1991). Sediments in the outer harbor may be dredged and disposed of in Lake Erie approximately 3.2 km from the east breakwater light, when necessary. However, PCBs appear to be migrating from the river to the harbor and this could curtail future dredging operations if PCB levels continue to increase in the harbor sediments (Ohio EPA, 1991).

TABLE 3.3. A SUMMARY OF AMBIENT WATER QUALITY STANDARD VIOLATIONS MEASURED IN THE ASHTABULA RIVER AOC (ALL CONCENTRATIONS IN UG/L) (ADAPTED FROM THE STAGE ONE RAP (OHIO EPA, 1991))

Site	Concentrations						Ohio WQS		U.S. EPA Criteria		
Parameter	Max.	Min.	Mean	Standard Deviation	Number of Samples	Detection Limits	Aquatic Life	Human Health[1]	Aquatic Life	Human Health	GLWQA Objectives
Ashtabula River*											
Copper	129	ND	13.5	30.6	23	2.4	16(2)	----	16(2)	----	5(9)
Cadmium	3.5	ND	0.15	0.7	23	3.2	1.8(1)	----	1.5(1)	----	0.2(1)
Iron	4850	353	900	860	23	13.7	1000(2)	----	1000(2)	----	300(23)
Lead	7.8	ND	1.29	2.26	23	1.5	11	----	4.9(4)	----	25
Zinc	62.3	6.8	16.0	14.6	23	1.4	140	----	141	----	30(2)
Endosulfan	0.13	ND	0.01	0.03	23	0.05	.003(2)	2.0	.056(2)	159	
Lake Erie, (Water Intake)*											
Copper	35.5	4.8	17.5	0.75	12	2.4	16(7)	1000	16(7)	----	5(11)
Iron	4810	892	2502	1610	12	13.7	1000(8)	----	1000(8)	300(12)	300(12)
Zinc	41.0	6.0	19.7	9.81	12	1.4	140	5000	141	----	30(2)
Total Diss. Solids (mg/l)	227	169	194.2	17.1	12	10	1500	----	----	----	200(4)
Mercury	0.5	ND	0.05	0.15	12	0.4	0.2(1)	0.012(1)	0.012(1)	0.146(1)	0.2(1)
Bis(2-ethylhexyl)phthalate	13.0	ND	1.08	3.59	12	10	8.4(1)	59	----	50000	0.6(1)

1 Human health standards for the Ashtabula River are based on surface water concentrations that could bioaccumulate in fish tissue making fish consumption potentially deleterious to human health. Human health standards for Lake Erie include consumption of water as well since Lake Erie is designated as a public water supply.

* Woodward Clyde Consultants (1991)

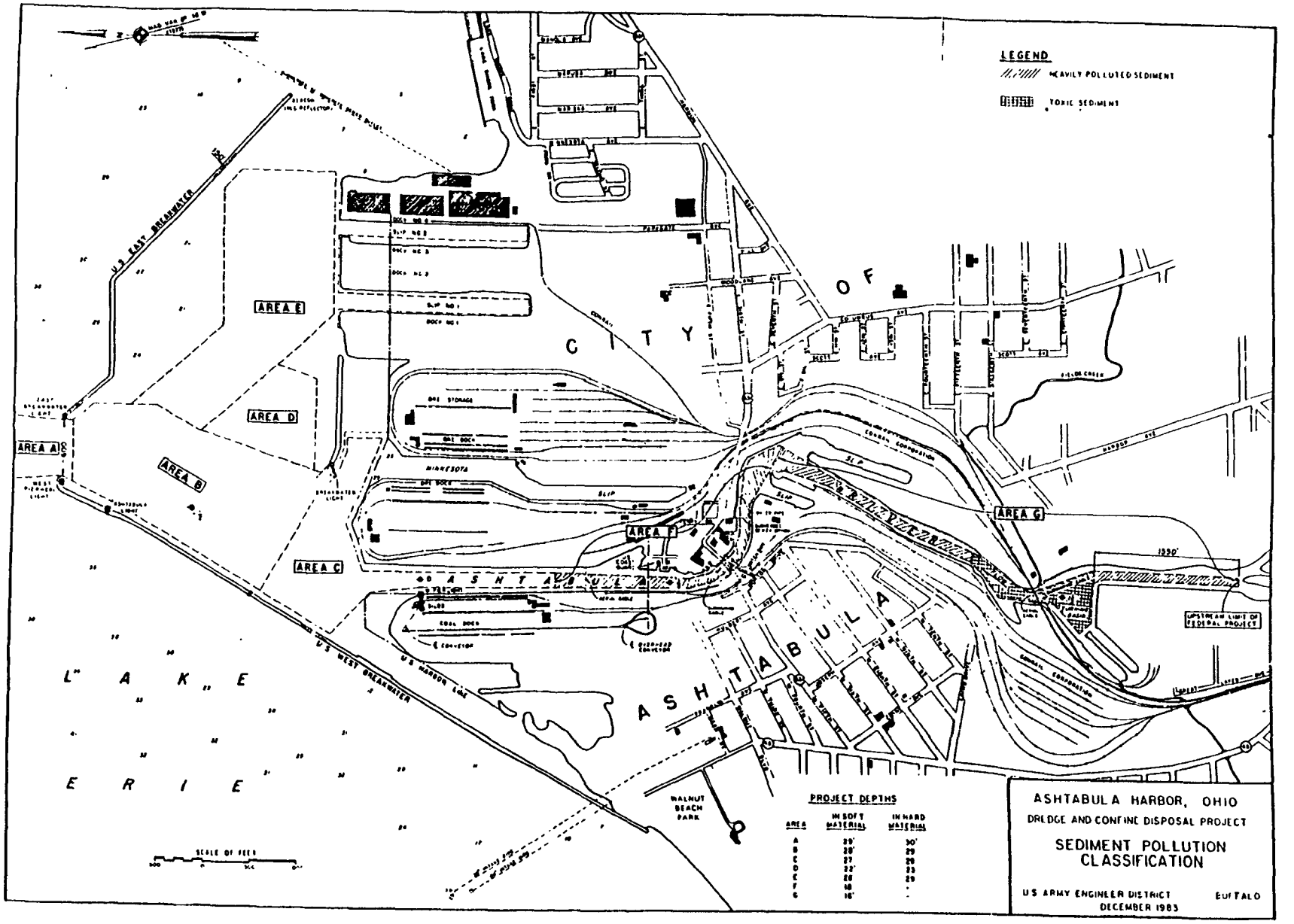

Figure 3.4. **Sediment pollution classification of the Ashtabula River AOC (Ohio EPA, 1991).**

The latest sediment sampling of the Ashtabula River AOC was conducted during 1989 and 1990 by Woodward-Clyde Consultants (WCC). The WCC investigation involved an intensive sampling effort of 115 stations located in Lake Erie, the harbor, the main navigation channel, outside the channel, in slips off the river itself, and upstream from the navigation channel. The draft Stage One RAP has provided a synopsis of WCC's results (Ohio EPA, 1991). A few major points to make about this study are: 1) metal (especially iron and arsenic) and PCB contamination is extensive at nearly all sites in and around the main channel; 2) pollutant concentrations generally increase with depth to some maximum level (PCB concentrations as high as 660 mg/kg were observed in a buried layer of sediment in the navigation channel); 3) all contaminants show a decreasing concentration gradient with distance downstream from Fields Brook, especially for organics; 4) heavily polluted (i.e., >10 mg/kg) concentrations of PCBs were never detected in the surficial sediments; and 5) very high concentrations of chromium, copper, lead, and zinc were observed downstream from the 5th St. Bridge (WCC, 1991).

3.3 RECREATIONAL USES

The greatest recreational opportunities on the Ashtabula River and Harbor involve fishing and boating. Recreational land along the Ashtabula River AOC consists mostly of marina facilities and launching ramps that provide an access point to Lake Erie. There are currently nine marinas and yacht clubs in the area with an estimated 1200 slips (Figure 3.2). Fishing for perch and walleye has improved greatly in Lake Erie, and there are now more than 50 registered fishing charters operating out of Ashtabula (Ohio EPA, 1991). The marinas and improved fisheries in Lake Erie have contributed to local efforts to increase tourism and to promote economic development in the region.

The economic base of Ashtabula has been weakened by industrial layoffs and closures. Ashtabula, like other small cities in the Great Lakes region that are undergoing economic problems, is searching to increase tourism by capitalizing on their access to one of the Great Lakes, on the history of the town, and on the natural resources of the area. The area is being promoted for sport fishing in Lake Erie, deer hunting, snowmobiling, visiting historical covered bridges in the county, and enjoying the scenic beauty of the area (Ashtabula Area Chamber of Commerce, 1991). In addition, Ashtabula is trying to promote its small size, rural lifestyle, and easy access to the major metropolitan centers of Cleveland, Erie and Youngstown/Warren to people and businesses who might want to settle there.

Although sport fishing in Lake Erie is very popular, fishing also occurs in the Ashtabula Harbor. The species composition of fish in the harbor is typical of the warmwater fish community in Lake Erie river mouths (Ohio EPA, 1991). The protected areas of the harbor usually contain relatively large numbers of yellow perch, white bass, pumpkinseed, white crappie, goldfish and emerald shiner. The more open water areas contain lower densities of gizzard shad, yellow perch, carp,

goldfish, brown bullhead, and emerald shiner. Fish migrate to and from the lower Ashtabula River when water conditions are favorable. Spawning migration runs for walleye and smallmouth bass occur in the spring [U.S. Fish and Wildlife Service (1984) cited in Ohio EPA (1991)]. The construction of vertical bulkheads at the commercial shipping facilities from River Mile 0.7 to the mouth of the river severely affected the ability of this portion of the river to support a diverse fish community.

Swimming is another recreational activity that takes place near the Ashtabula River AOC. Two official swimming beaches are located along the nearshore of Lake Erie. Walnut Beach, located west of the harbor mouth, has a large sand beach. Lakeshore Park, located east of the harbor mouth, has a smaller beach with recurring erosion problems. Both beaches consistently meet bacteriological water quality standards for bathing waters (Ohio EPA, 1991). Boaters may occasionally anchor and swim in the outer harbor by a sheltered sand bar near the west breakwater; this sand bar is also accessible by car, but it would probably be used only occasionally for swimming because Walnut Beach is nearby.

No swimming areas or beaches exist along the lower 3.2 km of the Ashtabula River. Although the river is classified by the State of Ohio for primary contact, the poor water quality of the river does not make it aesthetically pleasing for swimming. In addition, parts of the banks of the river are walled off; thus it is not likely that anyone would be exposed to contaminants through contact with river bank sediments. Some sporadic swimming may occur in the river from people jumping off their boats and swimming in the water. In addition, a railroad bridge, located just downstream from where Fields Brook enters the Ashtabula River, is used infrequently by kids who jump off the bridge into the river (J. Letterhos, Ohio EPA, personal communication, 1991). Water skiing is not likely to take place on the lower Ashtabula River because of the shallow depth of the river and busy boat traffic. Other activities that may result in immersion in the water, such as wind surfing, are unlikely to occur in the river because of heavy boat traffic and flow reversals in the surface water.

3.4 CONTAMINATION OF FISH

3.4.1 Routes of Contamination

One of the primary ways in which people in the Great Lakes region have been exposed to sediment-derived contaminants is through the consumption of contaminated fish. The specific mechanisms by which contaminants may be transferred from sediments to fish are still being elucidated. Part of the problem with determining these mechanisms is that different fish species occupy different habitats in the water column (e.g., benthic (bottom) versus pelagic (open water) habitats) and their diet and metabolism may change with age. This section will examine some of the ways in which fish occupying a river/harbor area of the Great

Lakes may accumulate contaminants, assuming that the major source of pollutants comes from in-place contaminated sediments.

The group of contaminants that have been of major concern in the Great Lakes are hydrophobic organic compounds (HOCs) such as PCBs and DDT. These compounds are persistent in the environment, due to their physical-chemical properties, and will preferentially accumulate in the lipids of organisms relative to other compartments (e.g., muscle, bone). Many of the commercially exploited Great Lakes fish have relatively high amounts of body fat (e.g., lake trout, lake whitefish, and channel catfish), and thus would be expected to contain higher levels of lipid-soluble HOCs than species characterized by low body fat (e.g., yellow perch and suckers) (Kononen, 1989).

The accumulation of contaminants in fish lipids can occur by two routes: 1) diffusion across the gills into the body and 2) transfer from the gut into the body after the consumption of contaminated food (Swackhamer and Hites, 1988). For the first route, the uptake of contaminants from water is functionally dependent on fish respiration and is related to the transfer of dissolved oxygen across the gill surfaces (Weininger, 1978). For the second route, the flux of contaminant transfer through feeding is dependent on the following factors: a) contaminant concentration in food, b) rate of consumption of food, and c) degree to which the ingested contaminant in the food is actually assimilated into the tissues of the organism. The assimilation of pollutants is affected by the desorption and excretion of contaminants from body tissues, and by the growth of the organism (Thomann and Connolly, 1984).

There is some uncertainty as to whether compounds sorbed to sediment particles will be available to fish for uptake. A chemical equilibrium model would assume that contaminant concentrations in the fish and sediments would be in equilibrium through their individual equilibrium coefficients with the water column (Connor, 1984). Studies with marine bottom fish in urban bays seem to indicate that the concentration of organic contaminants in the fish is correlated with the sediment concentration of those compounds (Connor, 1984; Mallins et al., 1984). This correlation may depend on the area's physical flushing capacity (residence time of water in a basin) and the metabolism of the organism (Connor, 1984). Similarly, a good correlation between the types of contaminants found in sediments collected from areas of industrial and urban development with the types of contaminants detected in freshwater carp from the same area has been made (Jaffe et al., 1985). Carp tend to remain in a local territory and, for the most part, are benthic feeders; thus, they would be expected to serve as a reasonable barometer of the types of contaminants (especially organic compounds) found in their aquatic environment. In another study, Brown et al. (1985) hypothesized that PCB concentrations in pelagic (i.e., open water) consumers of benthic-feeding organisms in the Hudson River were largely controlled by PCB levels in the surficial sediments. While the aforementioned studies seem to indicate some causal linkage between contaminant concentrations in sediment and fish, there is

a degree of uncertainty associated with this linkage. One of the difficulties with assessing the impacts of sediment contaminants on fish is that the factors controlling their bioavailability are not well understood, nor is there a basic understanding of trophic transfer from benthic to pelagic food chains (Bierman, 1990).

Due to the difficulty involved with assessing sediment-fish linkages in the field, controlled laboratory experiments have been conducted. Seelye et al. (1982) exposed young-of-the-year perch to a slurry of contaminated sediments for 10-days to simulate the conditions these fish would encounter during dredging. Although the perch accumulated organic compounds and heavy metals from the resuspended sediments, it is not known whether the contaminants in the fish reached steady state. In another experiment, by Kuehl et al. (1987), carp exposed to Wisconsin River sediment for 55 days accumulated 7.5 pg/g 2,3,7,8-TCDD; maintaining exposed fish in clean water for an additional 205 days resulted in the depuration of 32-34% 2,3,7,8-TCDD. The most likely uptake route for 2,3,7,8-TCDD in the carp was through the ingestion of contaminated sediments while feeding (Kuehl et al., 1987). In another experiment, lake trout that were exposed to Lake Ontario sediment and smelt in long term lab experiments appeared to bioaccumulate 2,3,7,8-TCDD primarily through the food chain and secondarily through contact with contaminated sediment (Batterman et al., 1989). These lake trout did not bioaccumulate a significant concentration of 2,3,7,8-TCDD from the water column, even under simulated equilibrium conditions and with low suspended solids concentrations (Batterman et al., 1989).

Recent evidence indicates that concentrations of HOCs in fish are primarily the result of food chain biomagnification and not equilibrium partitioning from the sediments or water column (Oliver and Niimi, 1988; Batterman et al., 1989). In Lake Ontario, samples from all trophic levels in the planktonic (water to plankton to mysid to alewive/smelt to salmonid) and the benthic (water to sediment/suspended sediment to amphipod/oligochaete to sculpin to salmonid) food chains showed classic biomagnification of PCBs with successive trophic levels (Oliver and Niimi, 1988). Thus, the rate at which contaminant concentrations increase with body size will be a function of how efficiently the contaminant is excreted after assimilation (Borgmann and Whittle, 1991). In turn, the assimilation of contaminants in fish will be affected by declines in feeding and clearance rates as growth occurs (Pizza and O'Connor, 1983). Temperature has also been found to affect the accumulation of PCBs in certain adult species of fish because temperature controlled food consumption, growth, and lipid content (Spigarelli et al., 1983).

Other contaminants, such as mercury, are also of concern in the Great Lakes. Unlike HOCs, mercury appears to accumulate in fish tissues through direct uptake from the water column (Gill and Bruland, 1990). The major form of mercury in the water column is the highly toxic methylated mercury species.

Because of the problem of mercury contamination in fish in the Great Lakes region, fish advisories have been issued for certain size classes of sport fish.

3.4.2 Fish and Wildlife Advisories

The Great Lakes jurisdictions have issued consumption advisories for sport fish since the late 1960s and early 1970s. These consumption advisories are based on the relationship between tissue concentrations of contaminants in individual size classes and species of fish and on specific trigger levels. When tissue concentrations exceed some trigger level (usually Food and Drug Administration action levels), consumption advice is issued by the states. The Governors of the Great Lakes States called for the uniform development of fish consumption advisories by the states in the 1986 Great Lakes Toxic Substances Control Agreement (Foran and VanderPloeg, 1989). However, this mandate has not been followed by all states, and this inconsistent consumption advice may serve to confuse the fishing public and those consuming Great Lakes sport fish. Ultimately, confusion about fish consumption advice may result in it being ignored entirely.

The Ohio Department of Health and Ohio EPA issued a fish advisory for the Ashtabula Harbor and lower 3.2 km of the Ashtabula River in 1983. This advisory recommends that no fish caught in the river from the 24th St. Bridge to the harbor mouth be eaten (Ohio EPA, 1991). In addition, signs are posted at several access points to the AOC warning against the consumption of any fish from the lower Ashtabula River. A general advisory against the consumption of carp and channel catfish in Lake Erie is in effect.

Despite these warnings, many anglers do not heed these advisories (J. Letterhos, Ohio EPA, personal communication, 1991). People still fish from the river banks. Fishing is also popular from the west breakwall and along rip rap in the outer harbor. The Ohio Department of Health does not have any specific information on fish consumption patterns and rates of consumption in the Ashtabula River area (T. Shelley, Ohio Department of Health, personal communication, 1991). As will be discussed in Chapter 5, standard fish consumption rates approved by the EPA were used in this risk assessment.

During 1989, an Ohio EPA survey discovered brown bullheads with numerous lip and skin tumors inside the west breakwall in the Ashtabula River (Ohio EPA, 1991). The source of the tumors has not been determined; the U.S. Fish and Wildlife Service is investigating this matter. The Conrail coal storage piles and the coal conveyor spanning the river are sources of coal dust problems at the river mouth. There may be some link between PAHs in the coal dust and tumors in the fish.

Although the Ashtabula River lies on a major migration corridor for ducks and geese, there is currently no information on deformities or tissue

concentrations of contaminants in any birds. Because the Ashtabula River AOC lies within an urban area, hunting for waterfowl would not be occurring there.

3.5 WATER SUPPLY

The drinking water supply for Ashtabula comes from Lake Erie. Very little groundwater is used in Ashtabula County (Ohio EPA, 1991). The Ohio American Water Company provides drinking water to approximately 38,000 people in the city of Ashtabula and surrounding townships. The company has two, 457 m intake pipes in Lake Erie west of the river mouth (Figure 3.2). Concern has been raised that the quality of the water supply may be threatened by river discharges under certain weather conditions or during river dredging.

Sampling at the intake under varying weather conditions was conducted in 1990 to determine if the Ashtabula River plume impacted water quality at the intake (WCC, 1991). Violations of Ohio WQS were noted for copper, iron, and mercury (Table 3.3); however, these metals are frequently violated along the Lake Erie southshore nearshore zone (Ohio EPA, 1991). The WQS for bis(2-ethyl-hexyl) phthalate was also violated. Although raw water sometimes exceeds WQS for metals, finished water meets all drinking water standards (Ohio EPA, 1991). The U.S. Army Corps of Engineers (1988) has calculated that a 40 kilometer per hour northeast wind coupled with a river flow greater than 12.7 cubic meters per second would be needed for the river discharge to affect the water supply intake. These conditions rarely occur.

3.6 HUMAN HEALTH CONCERNS

Several human health studies have been conducted for the Fields Brook area that are relevant to the Ashtabula River AOC population. The Ohio Department of Health conducted a cancer surveillance study of the human population in close proximity to Fields Brook. The study concluded that the total cancer incidence and mortality in the population close to Fields Brook did not differ significantly from the rest of Ohio or the United States [Indian and Hundley (1987) cited in Ohio EPA (1991)]. However, the incidence of mortality of brain and other central nervous system cancers was significantly higher, but it was not known whether exposure to chemicals in the area had contributed to this situation. A follow-up study, the Adverse Reproductive Outcomes Survey, examined congenital anomalies, low birthweight and fetal deaths in the Fields Brook area; these factors did not differ significantly from the rest of Ashtabula County or Ohio [Indian and Rao (1988) cited in Ohio EPA (1991)].

A risk assessment was completed for Fields Brook as part of the CERCLA-Superfund requirements. This assessment determined that public health and welfare may be affected adversely, under existing and future exposure scenarios, by contaminants in Fields Brook and its tributaries (CH_2M Hill, 1986).

Public concern has been raised about uranium contamination in the Ashtabula River AOC. Although uranium was found above background levels in river sediments (2.4-22.3 pCi/g), the concentrations were below Nuclear Regulatory Commission guidelines (i.e., 30 pCi/g) and ruled not to be of concern [U.S. EPA (1990) cited in Ohio EPA (1991)].

CHAPTER 4

RISK ASSESSMENT FRAMEWORK

4.1 CONCEPT OF RISK

People are subject to a number of risks throughout their day which may cause them immediate or delayed harm. Some risks arise from personal choices (e.g., driving a car, participating in sports) while other risks may result from things people have little control over (e.g., breathing urban air, being a victim of a random crime). In terms of human health risks resulting from exposure to some chemical, biochemical, or physical agent, risks are classified into two categories: carcinogenic and noncarcinogenic risks.

Cancer is the leading cause of death for women in the United States, and most cancers, for both men and women, are caused by factors resulting from life style choices (e.g., smoking, drinking alcohol, consuming a diet high in animal fat, being overweight, or staying out in the sun too long (ultraviolet light exposure)) (Henderson et al., 1991). In particular, tobacco (alone or in combination with alcohol) accounts for one of every three cancer cases occurring in the U.S. today (Henderson et al., 1991). Occupational exposures to specific carcinogens (especially asbestos) account for only about 4% of the cancers in the United States (Henderson et al., 1991). Although nonoccupational exposures to environmental contaminants probably cause an even smaller fraction of the cancers reported in the United States, it is important to safeguard the public's health from unnecessary risks. In addition, environmental contaminants may also pose a noncarcinogenic risk to human health.

Noncarcinogenic risks include chronic and subchronic effects to people. Included in this risk category are birth defects, respiratory diseases (e.g., asthma), liver diseases, learning disabilities, etc. One way to examine incidences of these risks in human populations is through epidemiological studies. Three sets of studies of the impacts of human exposure to PCB contaminated fish from the Great Lakes basin--the Michigan Sports Fisherman Cohort, the Michigan Maternal/Infant Cohort, and the Wisconsin Maternal/Infant Cohort were evaluated using epidemiologic criteria (Swain, 1991). The results from comparing the studies against each other, and against comparable data from other geographic locales, strongly suggest a causal relationship between PCB exposure and alterations in both neonatal health status and in early infancy (Swain, 1991). However, there is no evidence that these short-term effects lead to any chronic health effects (Bro, 1989). Possible developmental effects in infants and children will not be addressed in this risk assessment because complex pharmacokinetic models, which are not well developed in the risk assessment field, would have to be used. Thus, it is beyond the scope of the ARCS Program to address this issue in any great depth (USEPA, 1991b).

4.2 RISK FRAMEWORK

Risks associated with environmental exposures to contaminants are difficult to assess because 1) the exposure itself is often difficult to document and 2) the exposure does not always produce immediately observable effects. Due to these difficulties, human health risks associated with exposures to contaminants must often be estimated via scenarios using standard EPA exposure parameters.

The approach used for this baseline human health risk assessment followed exposure and risk assessment guidelines established by the EPA for use at Superfund sites (USEPA, 1988b; 1989a,b; 1991a). Although the Ashtabula River is not a Superfund site, the risk assessment procedures developed for the Superfund Program can be applied to this site to estimate current risks to people residing in the AOC. Unlike the Superfund risk assessments, this risk assessment did not consider risks resulting from future scenarios (e.g., future risks associated with turning a contaminated site into a playground). Instead, this risk assessment was based on the most up-to-date information available to estimate current noncarcinogenic and carcinogenic risks to human populations in the lower 3.2 km of the Ashtabula River and in the Ashtabula Harbor.

The procedures used in this risk assessment are outlined briefly in Figure 4.1. The first step in the process was to obtain information about the Ashtabula River from the draft Stage One RAP (Ohio EPA, 1991) and from other documents. In addition, a search for the latest data on contaminant levels in the environmental media of interest was conducted to characterize the extent of contamination at the site. The next step was to determine the exposure pathways by which people could come in contact with sediment-related contaminants from the river. The most complete and current data sets were then evaluated to judge whether adequate quality assurance/quality control (QA/QC) protocols were followed. Next, based on the exposure pathways and sites of exposures, the most current environmental data were used to determine contaminant intake levels. Intake levels are essentially equivalent to administered doses and are expressed in units of mg chemical/kg body weight-day. These chemical intake levels were then integrated with noncarcinogenic and carcinogenic toxicity data, obtained from verified and interim EPA sources, to estimate the respective human health risks to people in the Ashtabula River AOC. Finally, because of the number of assumptions that went into each step of the risk assessment procedure, a qualitative listing of the uncertainties involved in these assumptions was made.

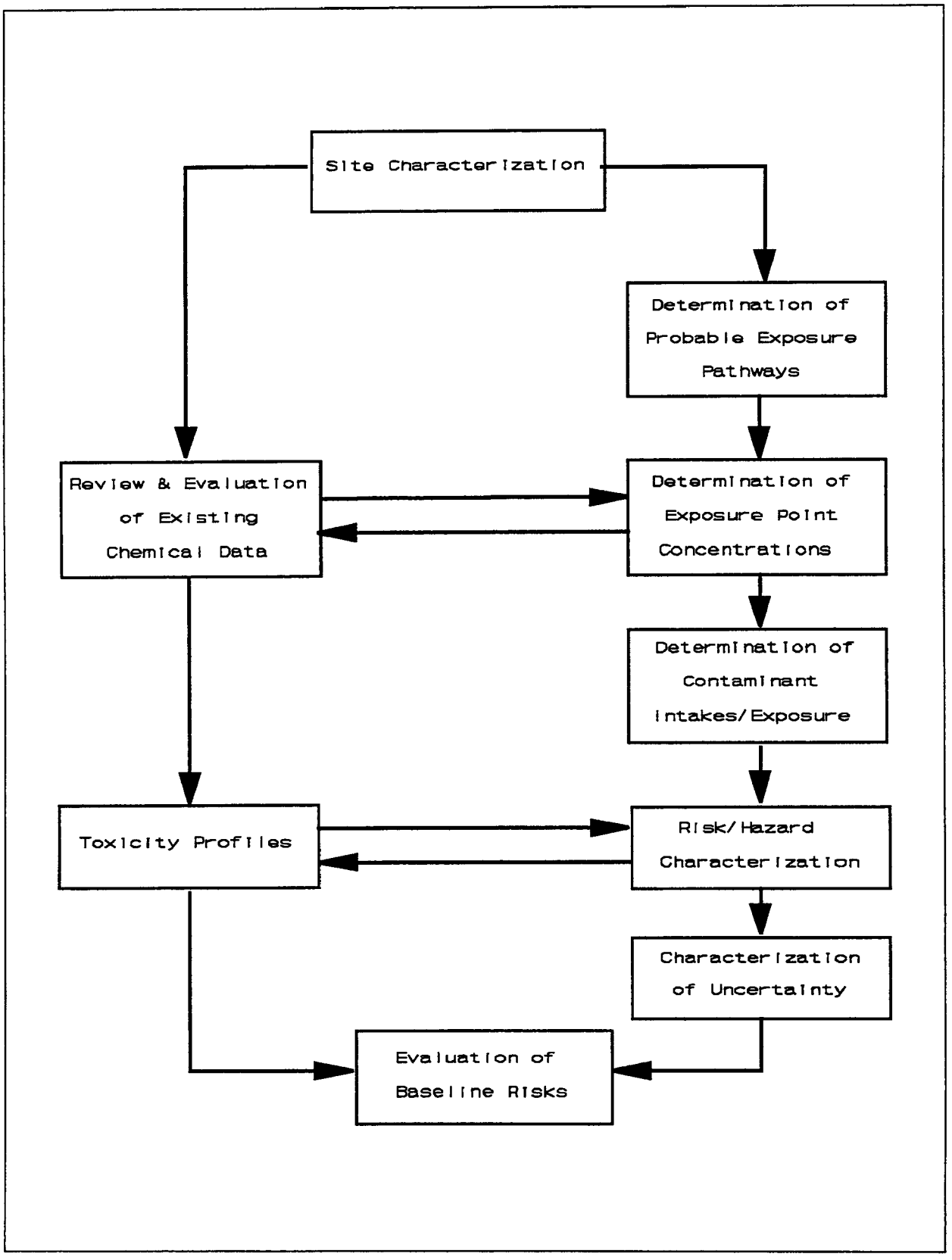

Figure 4.1. Components of baseline human health risk assessments.

CHAPTER 5

EXPOSURE ASSESSMENT

5.1 EXPOSURE PATHWAYS

In this exposure assessment, the magnitude, frequency, duration, and route of direct and indirect exposures of people to sediment-derived contaminants from the Ashtabula River AOC will be determined. Exposures to these pollutants can potentially occur via three pathways: dermal contact, inhalation, and ingestion. Dermal contact involves direct contact of the skin with either contaminated sediments, riverplain soils, or overlying water. Inhalation of airborne vapors or dust may introduce chemicals of potential concern into the respiratory system. Ingestion of contaminants through the consumption of contaminated soils, sediment, or food (e.g., fish, waterfowl) is potentially significant because of the direct transfer of contaminants across the gut.

The Ashtabula River AOC was toured during 28-29 August 1991 so that researchers could become familiar with the AOC and determine relevant exposure pathways. A meeting of the Ashtabula River RAP Advisory Council, held the evening of August 28, provided a means of informing the Council about this human health risk assessment. For the most part, the Ashtabula River AOC is not accessible for detailed viewing by car. Consequently, Julie Letterhos (RAP Coordinator) arranged a boat tour of the lower Ashtabula River and Harbor area courtesy of Jack's Marine; the tour included Ms. Letterhos and members of the Ashtabula River RAP Advisory Council. In addition, Ms. Letterhos provided a short driving tour of the Ashtabula River AOC and Fields Brook area. The input and cooperation of the Ashtabula River RAP Advisory Council was extremely valuable for evaluating exposure pathways in the AOC.

The potential pathways by which people may be exposed to contaminants from the Ashtabula River AOC are given in Table 5.1. These pathways were then examined to determine whether they were complete or incomplete. A pathway is complete if there is: 1) a source or chemical release from a source, 2) an exposure point where contact can occur, and 3) an exposure route by which contact can occur (USEPA, 1989a). The exposure pathway is incomplete if one of these conditions is not met. Five pathways appear to be incomplete:

1) **Ingestion of contaminated drinking water:** the Ashtabula River is not used as a source of drinking water in the AOC.

2) **Ingestion of sediments:** the ingestion of bottom sediments does not appear to be occurring. Only the bottom sediments near shore would be accessible if, for example, a child reached

TABLE 5.1. POTENTIAL PATHWAYS BY WHICH PEOPLE MAY BE EXPOSED TO SEDIMENT-DERIVED CONTAMINANTS FROM THE ASHTABULA RIVER AOC

INGESTION OF CONTAMINATED:
• Surface Water
• Fish and Wildlife
• Drinking Water
• Sediments
• River Bank/Flood Plain Soils
DERMAL CONTACT WITH CONTAMINATED:
• Surface Water
• Sediments
• River Bank/Flood Plain Soils
INHALATION OF AIRBORNE CONTAMINANTS

into the water and grabbed some sediments; however, no evidence of this behavior was available.

3) **Ingestion of contaminated soils:** the ingestion of contaminated soils from the river banks does not appear to be occurring. The banks are either walled off or are not easily accessible, thus limiting the opportunities for human contact.

4) **Dermal contact with contaminated soils:** the river bank soils are mostly inaccessible to people; thus, this pathway may not be occurring.

5) **Ingestion of wildlife:** hunting is not allowed within the city limits of Ashtabula, including the AOC.

Although five exposure pathways were considered complete in the Ashtabula River (Table 5.2), not all of these exposure pathways may result in significant human health risks. In particular, it was assumed that if insignificant risks were associated with the ingestion of surface water while swimming, then the risk associated with dermal exposure to surface water or sediments in the Ashtabula River AOC would also be insignificant (see Appendix A for the rationale behind this assumption). Under a reasonable maximum exposure scenario in which a 70-kg person swam 3 days/yr for 0.5 hr/event over a 30-year period, the noncarcinogenic and carcinogenic risks resulting from ingesting surface water at a rate of 50 mL/hr could be estimated (Appendix A). The surface water, collected below Fields Brook, contained detectable concentrations of barium, copper,

TABLE 5.2. COMPLETE EXPOSURE PATHWAYS IN THE ASHTABULA RIVER AOC

• Consumption of Contaminated Fish
• Ingestion of Surface Water while Swimming or Playing in the Water
• Dermal Contact with Water while Boating, Fishing, Swimming, etc.
• Dermal Contact with Sediments while Entering or Leaving the Water
• Inhalation of Airborne Contaminants

manganese, zinc, acetone, methylene chloride, and vinyl acetate. The resulting noncarcinogenic risk (Hazard Index (HI) = 0.0003) and upper-bound, lifetime carcinogenic risk (4×10^{-10}) were far below levels of concern (i.e., HI > 1, cancer risk exceeding 10^{-4} to 10^{-6}). Thus, infrequent dermal exposure to water and sediments were also assumed to be insignificant. No swimming areas are designated along the AOC and swimming may only occur infrequently (e.g., if someone jumps off his or her boat into the water).

Although the air pathway is complete, it cannot be quantitatively assessed with the currently available data. In addition, it would be difficult to separate out the contribution of airborne contaminants from the river and that from industrial, municipal, and background sources.

The only complete exposure pathway that will be considered for this risk assessment is the consumption of contaminated fish. Noncarcinogenic and carcinogenic risks will be determined for typical (i.e., average) and reasonable maximum exposures (i.e., the maximum exposure that is reasonably expected to occur at a site), as well as for exposures resulting from subsistence fishing. The subsistence exposure scenario was chosen because of economic problems in the area which might contribute to an underemployed/unemployed person to rely on locally caught fish for their main source of protein.

5.2 DATA USED IN THE EXPOSURE ASSESSMENT

5.2.1 Data Sources

Data on contaminant levels in fish and water were obtained from a recent

study by Woodward-Clyde Consultants (WCC, 1991). WCC also sampled and analyzed sediment cores from the Ashtabula River AOC, but these data were not used in this risk assessment. No assumptions about the temporal and spatial variability of contaminants data in the Ashtabula River and Harbor will be made here because of a general lack of historical data.

Fish samples were collected by WCC at three locations in the lower Ashtabula River and at one site in the harbor (Figure 5.1) from 4-17 October 1990. Thirteen of the planned 16 fish samples were collected during sampling. Ten brown bullheads, collected by the U.S. Fish and Wildlife Service (USFWS) in the harbor, were provided to WCC for analysis. A total of 66 fish were caught: 20 carp, 26 small and large mouth bass, 4 rock bass, and 16 bluegill. Seven of the fish caught had tumors; of this number, one whole fish and two fillets were analyzed for contaminants. The species of fish with tumors were not identified by WCC. Of the total fish caught at the four sites, 12 carp, 16 small/large mouth bass and 16 bluegill were selected at the request of the Ohio EPA for laboratory analysis. The criteria by which fish were selected for analysis was not given. The carp were analyzed as whole fish; the other species were analyzed as fillets. Eight carp had detectable concentrations of PCBs of less than 2 mg/kg, the U.S. Food and Drug Administration recommended limit for PCBs in fish. No fillets had detectable levels of PCBs.

WCC collected water samples at several locations in the Ashtabula River AOC (Figure 5.2). All water samples were grab samples collected mid-stream at mid-depth and at the deepest depth where river water was more than 1 m deep. The samples were analyzed for basic water chemistry parameters, for target compound list compounds (i.e., base/neutral and acid extractable organic compounds, pesticides, PCBs), and for a select group of metals. The only water samples used in this risk assessment were collected just below Fields Brook; the concentrations of contaminants were greatest at this site (see Appendix A).

5.2.2 Data Review

All of the data used in this risk assessment underwent a QA/QC review by Lockheed Engineering and Sciences Company (Lockheed-ESC) under a contract with the EPA Environmental Monitoring Systems Laboratory in Las Vegas, NV. A complete evaluation of the data could not be made because of difficulty with obtaining WCC's QA/QC data. However, it appears that the data were generated following contract laboratory program (CLP) protocols. The CLP protocols are more extensive in their incorporation of QA/QC samples than those specified in the ARCS QA/QC program. In addition, rigorous data verification and validation procedures were used by WCC to ensure that the data were acceptable in terms of the QA/QC requirements as well as correct in terms of data entry and the final released values (i.e., few to no transcription errors). Therefore, it was the opinion of Brian Schumacher, the ARCS QA/QC reviewer (formerly of Lockheed-ESC), that the WCC data should be acceptable for use in this risk assessment; however, the

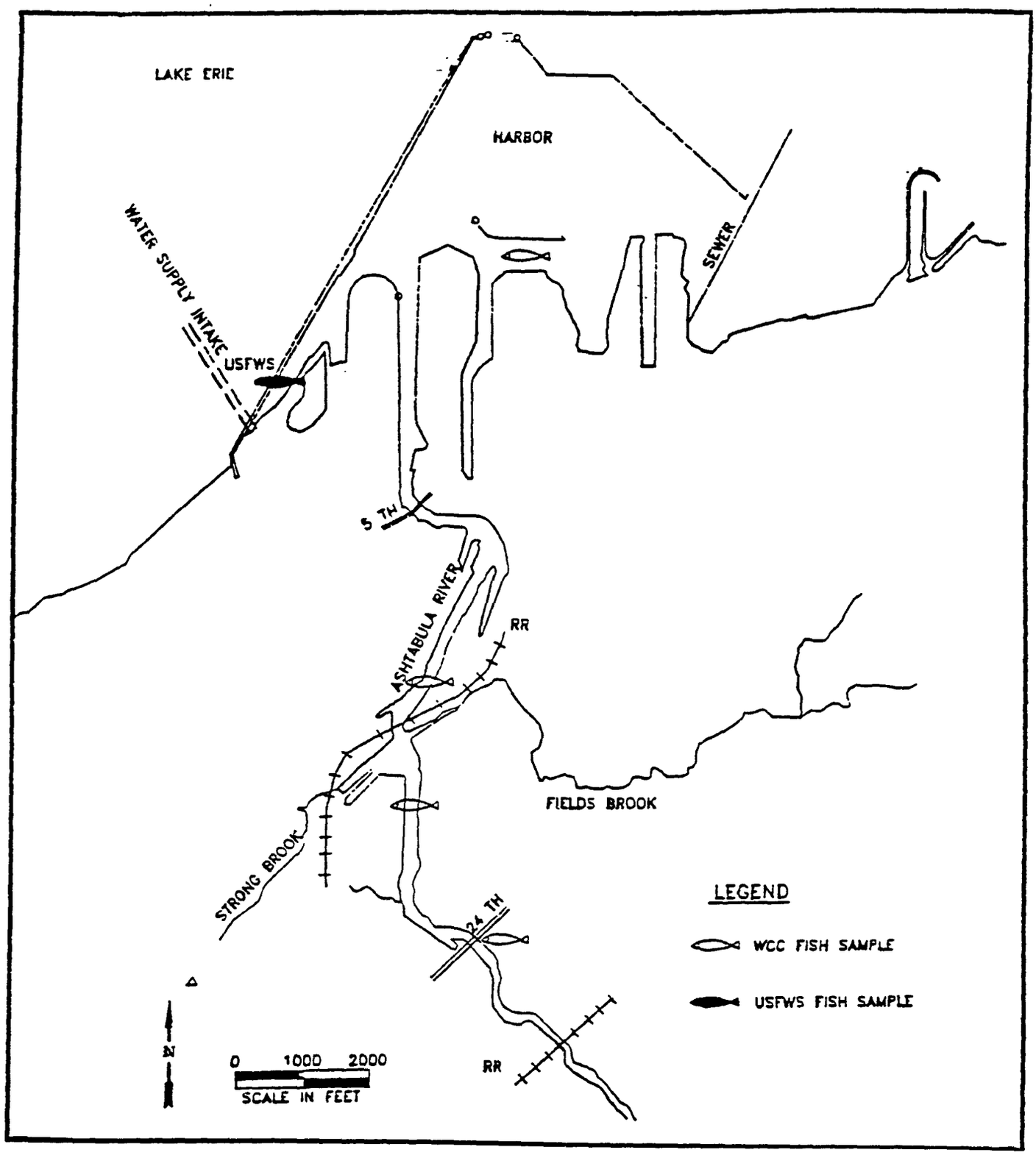

Figure 5.1. Fish sampling locations in the Ashtabula River AOC (WCC, 1991).

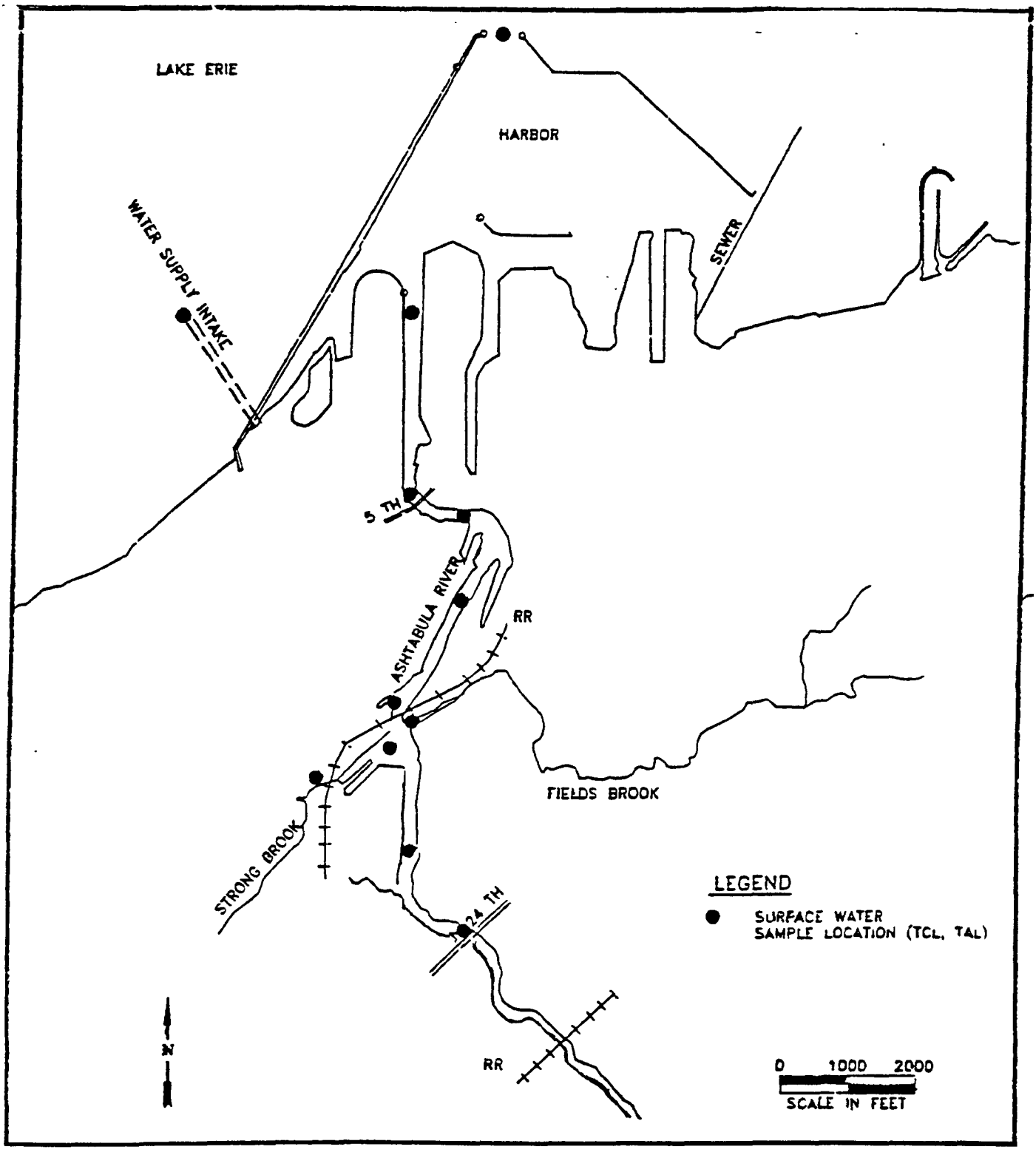

Figure 5.2. Surface water sampling locations in the Ashtabula River AOC (WCC, 1991).

supporting data to confirm its acceptability were not available (B. Schumacher (EPA Environmental Monitoring Systems Laboratory-Las Vegas), personal communication, 1991).

5.2.3 Data Sets

Not all of the fish species sampled by WCC for contaminants were used in this risk assessment because some of the fish were collected upstream from the limits of the Ashtabula River AOC (i.e., 24th Street Bridge). In addition, other fish were collected from the mouth of Strong Brook, upstream from Fields Brook.

Data obtained from composite samples of fish collected from two locations were used in this exposure assessment: 1) the Ashtabula River just downstream from Fields Brook (3 carp, 2 large mouth bass, and 11 bluegills), and 2) the Ashtabula Harbor (4 carp, 1 small mouth plus 3 large mouth bass, and 5 bluegills). Thus, data were obtained from both bottom feeders (i.e., carp) and open water feeders (i.e., bluegills, small/large mouth bass). Although carp are not a favored sport fish, they were included since they generally accumulate high levels of contaminants in water bodies due to their feeding habits and high fat content. Thus, carp were representative of an "upper-bound" level of contaminants in fish. The data for both bluegills and small/large mouth bass were used because both may be consumed by anglers. By determining separate exposures for the consumption of carp, bluegill, and small/large mouth bass, a range of risk estimates could be determined for typical, reasonable maximum, and subsistence exposure scenarios.

The fish were analyzed for a number of contaminants (Table 5.3). The mean contaminant levels of the fish data sets used in the exposure assessment are given in Tables 5.4 and 5.5. The highest contaminant concentrations were generally observed in fish collected below Fields Brook. The only exceptions were for concentrations of 1,1,2,2-tetrachloroethane, tetrachoroethene, and trichloroethene in whole small/large mouth bass collected from the mouth of Strong Brook. However, these data were not used in the exposure assessment since data for bass fillets were available from the Fields Brook site.

5.3 EXPOSURE ASSESSMENT

5.3.1 General Determination of Chemical Intakes

Once the complete exposure pathways were identified and contaminant concentrations for fish were obtained, an exposure assessment could be conducted. Exposures were normalized for time and body weight to determine chemical "intakes," expressed in units of mg chemical/kg body weight-day. For the ingestion of contaminated fish, intakes represent the amount of chemical available for absorption in the gut. The general equation for calculating chemical intakes is given in Table 5.6. Several variables are used to determine intakes, including

TABLE 5.3. LIST OF CONTAMINANTS ANALYZED IN THE FISH USED IN THIS RISK ASSESSMENT

CHEMICAL	DETECTED Yes	No
ORGANICS		
Chlorobenzene		X
1,1,2,2-Tetrachloroethane	X	
Tetrachloroethene	X	
Hexachlorobenzene		X
Hexachlorobutadiene		X
Trichloroethene	X	
Pentachlorobenzene		X
Octachlorostyrene		X
Fluoranthene		X
Phenanthrene		X
PCBs		
Aroclor 1242		X
Aroclor 1248		X
Aroclor 1254		X
Aroclor 1260	X	
INORGANICS		
Arsenic		X
Beryllium		X
Cadmium		X
Copper	X	
Chromium	X	
Lead		X
Mercury	X	
Silver	X	
Zinc	X	

specific information about the exposed population and the period over which the exposure was averaged. Noncarcinogenic effects were averaged over the same time period as the exposure duration [i.e., 9 years for typical exposures and 30 years for reasonable maximum exposures (RME)]. Carcinogenic effects were averaged over a lifetime (i.e., 70 years). Intake variable values were selected so that the combination of all values resulted in an estimate of either the typical, reasonable maximum, or subsistence exposure intakes.

The exposure parameters used in the typical scenario were assumed to be applicable to the general angling population of Ashtabula while the reasonable maximum exposure scenario applied to recreational anglers and their families.

TABLE 5.4. CONTAMINANT CONCENTRATIONS IN WHOLE CARP AND BLUEGILL FILLETS COLLECTED FROM THE ASHTABULA RIVER AOC (WCC, 1991)

Chemical	Whole Carp Ashtabula River (mg/kg)	Whole Carp Ashtabula Harbor (mg/kg)	Bluegill Fillets Ashtabula River (mg/kg)
METALS			
Chromium	2.0E+00	1.5E+00	1.4E+00
Copper	1.7E+01	1.5E+00	5.5E-01
Mercury	4.6E-02	ND	1.7E-01
Silver	3.2E-01	ND	ND
Zinc	9.6E+01	8.3E+01	2.2E+01
ORGANICS			
PCBs (Aroclor 1260)	8.1E-01	1.5E-01	ND
1,1,2,2-Tetrachloroethane	2.8E-02	5.5E-03	ND
Tetrachloroethene	2.7E-01	3.0E-02	ND
Trichloroethene	4.0E-02	7.3E-03	ND

TABLE 5.5. CONTAMINANT CONCENTRATIONS IN BLUEGILL AND SMALL/LARGE MOUTH BASS FILLETS COLLECTED FROM THE ASHTABULA RIVER AOC (WCC, 1991)

Chemical	Bluegill Fillets Ashtabula Harbor (mg/kg)	L. Mouth Bass Fillets Ashtabula River (mg/kg)	S/L Mouth Bass Fillets Ashtabula Harbor (mg/kg)
METALS			
Chromium	1.6E+00	1.2E+00	1.4E+00
Copper	ND	ND	ND
Mercury	7.3E-02	1.5E-01	1.2E-01
Silver	ND	ND	ND
Zinc	1.6E+01	1.1E+01	1.2E+01
ORGANICS			
PCBs (Aroclor 1260)	ND	ND	ND
1,1,2,2-Tetrachloroethane	ND	6.8E-03	ND
Tetrachloroethene	ND	1.2E-01	ND
Trichloroethene	ND	1.3E-02	ND

TABLE 5.6. GENERIC EQUATION FOR CALCULATING CHEMICAL INTAKES (USEPA, 1989a)

$$I = \frac{C \times CR \times EFD}{BW \times AT}$$

where:	
I	Intake = the amount of chemical at the exchange boundary (mg/kg body weight-day)
	Chemical-Related Variables
C	Chemical Concentration = the average concentration contacted over the exposure period (e.g., mg/L)
	Variables that Describe the Exposed Population
CR	Contact Rate = the amount of contaminated medium contacted per unit time or event (e.g., L/day)
EFD	Exposure Frequency and Duration = how long and how often exposure occurs. Often calculated using two terms, EF and ED, where: EF = exposure frequency (days/year) ED = exposure duration (years)
BW	Body Weight = the average body weight over the exposure period (kg)
	Assessment-Determined Variables
AT	Averaging Time = period over which exposure is averaged (days)

The subsistence exposure scenario was chosen for a sensitive subpopulation of people who would be consuming about four 8-ounce servings of locally caught fish per week. At the present time, specific information on fish consumption rates and trends in the Ashtabula River AOC is lacking. Results from the Michigan Sport Anglers Fish Consumption Survey, conducted by West and co-workers at the University of Michigan, may give a better indication of typical ingestion rates of fish by Ashtabula anglers than the default EPA parameter value. West et al. (1989) found that, for their survey conducted during the January-June, 1988 time frame, the average fish consumption was 18.3 g/person/day with a standard total deviation of 26.8 g/person/day; approximately 26% of the sample household

TABLE 5.7. EQUATION USED TO ESTIMATE CONTAMINANT INTAKES DUE TO INGESTION OF FISH

$$Intake = \frac{C \times IR \times FI \times EF \times ED}{BW \times AT}$$

where:	
Intake	Intake Rate (mg/kg-day)
C	Contaminant Concentration (mg/kg)
IR	Ingestion Rate (kg/day)
FI	Fraction Ingested from Contaminated Source (unitless)
EF	Exposure Frequency (days/yr)
ED	Exposure Duration (yr)
BW	Body Weight (kg)
AT	Averaging Time (days)

persons who ate fish consumed between 20-40 g/person/day while another 10% consumed between 40-75 g/person/day. West et al. (1989) estimated a year-round fish consumption rate of 19.2 g/person/day. This exposure assessment used a reasonable maximum ingestion rate of 54 g/person/day; this number seems appropriate because it falls within the upper 10% ingestion rate of the Michigan anglers. This exposure assessment also assumed that the only contaminated fish ingested by local residents came from the Ashtabula River AOC.

5.3.2 Intakes: Ingestion of Contaminated Fish

The equation used to estimate intakes of contaminants due to the ingestion of contaminated fish is provided in Table 5.7. The parameter values used in that equation are given in Table 5.8. Parameter values were obtained from recommended EPA sources except for the fraction of fish assumed to be ingested from the Ashtabula River AOC. This latter parameter had to be estimated, in a conservative way, because of a lack of information about what proportion of the fish consumption was attributable to locally caught fish. Since chemical intake values will be incorporated into the estimation of risk presented in Chapter 7, separate tables of intake values will not be presented here.

TABLE 5.8. PARAMETERS USED IN ESTIMATING CONTAMINANT INTAKES DUE TO INGESTION OF FISH IN THE ASHTABULA RIVER AOC

Var.	Units	Value Used	Comment
IR	kg/day	0.0192	Typical: West et al. (1989)
		0.054	RME: USEPA (1991a)
		0.13	Subsistence fishing: used the 95th percentile daily intakes averaged over 3 days for consumers of fin fish (Pao et al. (1982) cited in USEPA (1989a))
FI	-	0.1	Typical: study assumption
		0.25	RME: study assumption
		0.7	Subsistence fishing: study assumption
EF	day/yr	350	USEPA (1991a)
ED	yrs	9	Typical: USEPA (1989a)
		30	RME and Subsistence: USEPA (1989a)
BW	kg	70	50th percentile average for adult men and women (USEPA, 1989b)
AT:	days	3285	9 yrs x 365 days/yr (typical noncarcinogenic risk)
		10950	30 yrs x 365 days/yr (RME and subsistence noncarcinogenic risk)
		25550	70 yrs x 365 days/yr (carcinogenic risk)

CHAPTER 6

TOXICITY ASSESSMENT

6.1 TOXICITY VALUES

Two types of toxicity values were used in combination with exposure estimates (i.e., chemical intake values) to estimate human health risk. One toxicity value, the reference dose (RfD), provides an estimate of the daily contaminant exposure that is not likely to cause harmful effects during either a portion of a person's life or his/her entire lifetime. The RfD is the toxicity value used most often in evaluating noncarcinogenic effects. The other toxicity value, the slope factor, is used in risk assessments to estimate an upper-bound lifetime probability of an individual developing cancer as a result of exposure to a particular level of a potential carcinogen. In addition, the EPA weight-of-evidence classification scheme indicates the strength of evidence that the contaminant is a human carcinogen (Table 6.1). Slope factors are typically calculated for potential carcinogens in classes A, B1, and B2 as well as for class C on a case-by-case basis. A more detailed description of these toxicity values, summarized from "Risk Assessment Guidance for Superfund. Volume 1. Human Health Evaluation Manual (Part A)" (USEPA, 1989a), is given in Appendix B.

Chronic oral RfD values and oral slope factors were used for the fish ingestion pathway examined in this risk assessment. Toxicity values, which had undergone an EPA review process, were obtained from the EPA's Integrated Risk Information System (IRIS) data base. For chemicals lacking a "verified value," interim toxicity values were obtained from the Health Effects Assessment Summary Tables (HEAST), if available. Table 6.2 lists the toxicity data used for the chemicals of interest. This table also includes the form in which the chemical was administered to the test animal or patient (e.g., drinking water, diet, or gavage) for determination of the oral RfD. In addition, the source of the toxicity value is given. The endpoints of concern for evaluating noncarcinogenic risks are listed in Table B-1 of Appendix B.

6.2 LIMITATIONS

This risk assessment was limited by the current availability of toxicity information. In some cases, toxicity values were not available for some of the chemicals (e.g., RfD value for PCBs). In other cases, toxicity values were available for a particular metal species rather than for the total metal (e.g., mercury). In particular, methyl mercury was assumed to be the major form of mercury present in this system, and chromium VI was assumed to be the major valence state of chromium in the system. For other chemicals (i.e., tetrachloroethene and trichlorethene), the oral slope factors have been withdrawn from IRIS pending further review.

TABLE 6.1. EPA WEIGHT-OF-EVIDENCE CLASSIFICATION SYSTEM FOR CARCINOGENICITY (USEPA, 1989a)

Group	Description
A	Human carcinogen
B1 or B2	Probable human carcinogen B1 indicates that limited human data are available B2 indicates sufficient evidence in animals and inadequate or no evidence in humans
C	Possible human carcinogen
D	Not classifiable as to human carcinogenicity
E	Evidence of noncarcinogenicity for humans

TABLE 6.2. HUMAN HEALTH RISK TOXICITY DATA FOR CHEMICALS OF INTEREST IN THE ASHTABULA RIVER AOC

Chemical	Oral RfD (mg/kg/day)	Form	Source	Carcinogenic Weight of Evidence Class	Source	Oral Slope Factor 1/(mg/kg/day)	Source
"METALS"							
Chromium VI	5.0E-03	drinking water	a	A	a		
Copper	1.3E-03		b	D	a		
Mercury, methyl	3.0E-04	poisonings	a	D	a		
Silver	5.0E-03	oral dose	a	D	a		
Zinc	2.0E-01		b	D	a		
"ORGANICS"							
PCBs				B2	a	7.7E+00	a
1,1,2,2-Tetrachlorethane				C	a	2.0E-01	a
Tetrachloroethene	1.0E-02	gavage	a		Withdrawn pending review		
Trichlorethene	Pending			B2	Withdrawn pending review		

Sources:

a: IRIS (current as of 12/24/91)
b: USEPA (1989c)

CHAPTER 7

BASELINE RISK CHARACTERIZATION FOR THE ASHTABULA RIVER AOC

7.1 PURPOSE OF THE RISK CHARACTERIZATION STEP

The purpose of the risk characterization step is to combine the exposure and toxicity estimates into an integrated expression of human health risk. This section presents the calculated potential human health risks associated with the consumption of contaminated fish from the Ashtabula River AOC under the no action alternative. It is important to recognize that these calculated risk estimates are not intended to be used as actual values. Risk assessment is a regulatory process that provides risk managers with quantitative estimates that are to be used for comparative purposes only. These risk estimates must be interpreted in the context of all the uncertainties associated with each step in the process. Some of the major uncertainties in this risk assessment are addressed in the next chapter.

Two means of expressing the carcinogenic and noncarcinogenic risks of adverse health effects are presented in this chapter. First, chemical specific risks were estimated. Secondly, chemical specific risks were added to estimate the cumulative risk resulting from the consumption of a particular species of fish under one of three exposure scenarios.

7.2 QUANTIFYING RISKS

7.2.1 Determination of Noncarcinogenic Risks

Noncarcinogenic effects do not generally occur below a minimum or threshold level of exposure. These effects are evaluated by comparing a site-specific exposure level over a specified time period with a RfD derived from a similar exposure period (otherwise known as a hazard quotient (HQ)). Thus, HQ = exposure level (or intake)/RfD. Hazard quotients are usually expressed as one significant figure in a nonprobabilistic way. In this risk assessment, HQ values were expressed to two significant figures for each chemical; this was done to reduce round-up error when HQ values were summed for each pathway. An HQ value of less than 1 indicates that exposures are not likely to be associated with adverse noncarcinogenic effects (e.g., reproductive toxicity, teratogenicity, or liver toxicity). As the HQ approaches or exceeds 10, the likelihood of adverse effects is increased to the point where action to reduce human exposure should be considered. Owing to the uncertainties involved with these estimates, HQ values between 1 and 10 may be of concern, particularly when additional significant risk factors are present (e.g., other contaminants at levels of concern). However, the level of concern does not increase linearly as the RfD is approached or exceeded

because RfDs do not have equal accuracy or precision; nor are RfDs based on the same severity of toxic effects (USEPA, 1989a).

In assessing health risks, all HQ values are representative of long term chronic exposures (i.e., exposures assumed to occur over a period of 9 or 30 years). The sum of more than one HQ value for multiple substances and/or multiple exposure pathways is the Hazard Index (HI). This assumption of additivity does not account for any synergistic or antagonistic effects that may occur among chemicals. In addition, no attempt was made to distinguish between risk endpoints (e.g., target organs and related effects) when calculating the HI. Thus, this expression of total risk may be extremely conservative; it would be better to refine the HI to specific endpoints for HQ values greater than one. Additional limitations of HQ values and the segregation of hazard indexes have been described elsewhere (USEPA, 1989a).

7.2.2 Determination of Carcinogenic Effects

Unlike noncarcinogenic effects, carcinogenic effects are thought to pose some degree of risk at all exposure levels. These effects are estimated as the incremental probability of an individual developing cancer over a lifetime as a result of exposure to a potential carcinogen. The carcinogenic risk is computed using average lifetime exposure values that are multiplied by the oral slope factor for each carcinogen of interest. Slope factors are used to convert estimated daily intakes averaged over a lifetime of exposure directly to the incremental risk of an individual developing cancer. The resulting carcinogenic risk estimate is generally an upper-bound estimate, because slope factors are usually based on upper 95th percentile confidence limits. The EPA believes it is prudent public health policy to consider actions to mitigate or minimize exposures to contaminants when estimated excess lifetime cancer risks exceed the 10^{-5} to 10^{-6} range, and when noncarcinogenic health risks are estimated to be significant (USEPA, 1988a).

Carcinogenic effects were summed for all chemicals in an exposure pathway (i.e., consumption of fish). This summation of carcinogenic risks assumes that intakes of individual substances are small, that there are no synergistic or antagonistic chemical interactions, and that all chemicals produce the same effect (i.e., cancer). The limitations to this approach are discussed in detail elsewhere (USEPA, 1989a).

7.3 HUMAN HEALTH RISKS IN THE ASHTABULA RIVER AOC

7.3.1 Typical and Reasonable Maximum Exposures

7.3.1.1 Noncarcinogenic Risks

Based on typical and reasonable maximum exposure (RME) levels over a 9- and a 30-year period, respectively, estimated noncarcinogenic risks were below

levels of concern (i.e., Hazard Index <1) for all three fish species except for carp taken from the Ashtabula River (below Fields Brook) under RME conditions (Tables 7.1-7.6). For these carp, the estimated Hazard Index of 3 was mostly attributable to copper contamination. Thus, high consumption rates of carp from the Ashtabula River near Fields Brook may result in adverse noncarcinogenic effects. Information about the types of noncarcinogenic effects one might experience from chronic exposure to copper is not available at this time from the IRIS data base.

Since some of the chemicals (e.g., PCBs) detected in fish from the Ashtabula River AOC are lacking RfD values, it would be premature to state that no noncarcinogenic risk exists from consuming fish (e.g., bluegills, bass) from the Ashtabula River and Harbor. The noncarcinogenic risk reported here is an estimated risk based on currently available exposure/intake data and toxicity information, and should not be construed as an absolute risk.

7.3.1.2 Carcinogenic Risks

A carcinogenic risk estimate could not be calculated for the consumption of small/large mouth bass collected from the Ashtabula Harbor and for bluegills collected from both the river and harbor because no carcinogens were detected in these fish fillets. However, the carcinogenic risk could be determined, under typical and reasonable maximum exposure scenarios, for the consumption of both large mouth bass and carp collected from the Ashtabula River (below Fields Brook) and for carp from the Ashtabula Harbor.

The upper bound carcinogenic risk estimates associated with the consumption of large mouth bass fillets from the river were below concern levels (i.e., less than 10^{-6}) under typical and RME scenarios (Table 7.7). However, carp collected from both the harbor (Table 7.8) and river (Table 7.9) appear to pose a potential risk to human health; the upper bound cancer risk ranged from 4×10^{-6} to 5×10^{-4}. The potential carcinogenic risk resulting from the consumption of carp was up to an order of magnitude greater for fish collected near Fields Brook than for carp from the harbor. This upper bound risk was attributable to a PCB mixture resembling Aroclor 1260 in the whole carp.

There is a possibility that people who ingest, inhale, or have dermal contact with certain PCB mixtures may have a greater chance of incurring liver cancer; however, this statement is based on suggestive evidence rather than on verified data. Studies with three strains of rats and two strains of mice have verified the carcinogenic toxicity of PCBs through the occurrence of hepatocellular carcinomas (IRIS data base retrieval for PCBs, 1992). This evidence was used to classify PCBs as a probable human carcinogen.

As discussed in Chapter 3, fish will preferentially accumulate PCBs and other hydrophobic organic contaminants in their lipids. Since carp are mostly

benthic feeders that generally reside in a local area, they can be used as an indicator of local contamination problems. In addition, carp have a high lipid content which may readily accumulate contaminants through the ingestion and assimilation of contaminated food and possibly through the consumption of sediment while feeding. It is not possible to estimate how much of the carcinogenic risk is directly attributable to contaminants in the sediments. However, the Stage One RAP (Ohio EPA, 1991) indicates that the sediments in the Ashtabula River AOC are presently the major source of contamination to this area; thus, one could make the conservative assumption that nearly all of the human health risk is attributable to the direct and indirect (e.g., food chain transfer) exposure of fish to contaminants in the sediments.

The carcinogenic risk calculated for the consumption of carp is probably overly conservative because it is based on the assumed consumption of whole carp rather than on fillets. These risk estimates should be updated as new data becomes available, especially for data on contaminant levels in carp fillets. Although carp are generally regarded as an undesirable "trash" fish by many anglers, some people do consume them. In addition, food scientists are examining ways in which carp flesh can be deboned and restructured to form fabricated seafood products (Stachiw et al., 1988); this is of particular interest to Michigan firms as a way of exploiting an underutilized fish.

These noncarcinogenic and carcinogenic risk levels are based on raw fish. Depending on how one prepares and cooks the fish, the risk could be lessened. For the past 20 years, Mary Zabik and coworkers from Michigan State University have been investigating whether cooking methods can reduce pesticide and PCB residues in meat and fish (Smith et al., 1973; Zabik et al., 1979; Zabik et al., 1982; Stachiw et al., 1988). The results have not been consistent between and within species of fish. In one instance, different cooking methods did not result in significant changes in the level of PCBs, DDE, or DDD in cooked carp fillets (Zabik et al., 1982). In another case, cooking resulted in reductions of TCDD in restructured, deboned carp fillets (Stachiw et al., 1988). In order to further assess how cooking techniques may reduce the level of contaminants in fish, the Michigan Department of Public Health and Michigan State University have just begun a 2-year investigation (H. Humphrey, Michigan Department of Public Health, personal communication, 1991). This study will include a variety of sport fish in the Great Lakes (e.g., chinook and coho salmon) for skin-on and skin-off fillets. The results of the Michigan study will be useful for future human health risk assessments for determining better estimates of contaminant levels in cooked fish. At the present time, the following cooking techniques are recommended for reducing the risk of hydrophobic organic contaminants in fish: 1) trim fatty areas, 2) puncture or remove skin before cooking so that fats drain away, or 3) deep-fry trimmed fillets in vegetable oil and discard the oil (Michigan Department of Natural Resources, 1991).

7.3.2 Subsistence Anglers

Subsistence anglers increased their risks to contaminants by nearly an order of magnitude over recreational anglers in the reasonable maximum exposure scenario. The noncarcinogenic risks associated with the separate subsistence consumption of small/large mouth bass and bluegill fillets from the Ashtabula Harbor were below concern levels (i.e., HI<1) (Tables 7.1 and 7.3). The noncarcinogenic risk increased for the same fish species when they were obtained downstream from Fields Brook (HI = 1-2) (Tables 7.2 and 7.4). Whole carp had the highest Hazard Index from the harbor (HI = 2) (Table 7.5) and from the river (HI = 20) (Table 7.6). These risk estimates for carp are probably overestimated since they are based on whole fish instead of fillets. Still, these risk levels are high enough to warrant continued fish advisories in the Ashtabula River AOC. For all three fish species, the estimated noncarcinogenic risk was due mostly to methyl mercury contamination. Methyl mercury has been shown to cause central nervous system effects at the lowest adverse effect level of 0.003 mg/kg/day (IRIS data base retrieval for methyl mercury, 1992).

The carcinogenic risk could only be estimated for large mouth bass fillets (Ashtabula River) and for whole carp (both locations). The estimated cancer risk resulting from the consumption of bass did not appear to be of concern (i.e., less than 1×10^{-6}) (Table 7.7). The estimated cancer risk was much higher for carp collected from the Ashtabula Harbor (6×10^{-4}) (Table 7.8) and by Fields Brook (3×10^{-3}) (Table 7.9). In all cases, the risk was attributable primarily to PCB contamination. These carcinogenic risk levels represent an upper bound risk and are probably overestimated for carp since the risk estimates are based on raw, whole carp. These risk estimates confirm that the greatest human health risk associated with the consumption of fish resulted from fish inhabiting the most contaminated area of the Ashtabula River AOC (i.e., by Fields Brook).

TABLE 7.1. NONCARCINOGENIC RISKS ASSOCIATED WITH CONSUMING SMALL/LARGE MOUTH BASS FILLETS (TAKEN FROM THE ASHTABULA HARBOR) UNDER TYPICAL, REASONABLE MAXIMUM (RME), AND SUBSISTENCE EXPOSURE SCENARIOS

Chemical	Fish Conc. (mg/kg)	Noncarcinogenic Intake (mg/kg-day) Typical	RME	Subsistence	Hazard Index (Intake/RfD) Typical	RME	Subsistence
METALS							
Chromium VI	1.5E+00	3.8E-05	2.7E-04	1.8E-03	0.0076	0.054	0.37
Copper	ND						
Mercury (methyl)	1.2E-01	3.2E-06	2.2E-05	1.5E-04	0.010	0.074	0.51
Silver	ND						
Zinc	1.2E+01	3.0E-04	2.1E-03	1.5E-02	0.0015	0.011	0.073
ORGANICS							
PCBs (Aroclor 1260)	ND						
1,1,2,2-Tetrachloroethane	ND						
Tetrachloroethene	ND						
Trichloroethene	ND						
CUMULATIVE NONCARCINOGENIC RISK					0.02	0.1	0.9

TABLE 7.2. NONCARCINOGENIC RISKS ASSOCIATED WITH CONSUMING LARGE MOUTH BASS FILLETS (TAKEN FROM THE ASHTABULA RIVER DOWNSTREAM FROM FIELDS BROOK) UNDER TYPICAL, REASONABLE MAXIMUM (RME), AND SUBSISTENCE EXPOSURE SCENARIOS

Chemical	Fish Conc. (mg/kg)	Noncarcinogenic Intake (mg/kg-day)			Hazard Index (Intake/RfD)		
		Typical	RME	Subsistence	Typical	RME	Subsistence
METALS							
Chromium VI	1.2E+00	3.2E-05	2.2E-04	1.5E-03	0.0063	0.044	0.30
Copper	ND						
Mercury (methyl)	1.5E-01	4.0E-06	2.8E-05	1.9E-04	0.013	0.092	0.63
Silver	ND						
Zinc	1.1E+01	2.9E-04	2.0E-03	1.4E-02	0.0014	0.010	0.069
ORGANICS							
PCBs (Aroclor 1260)	ND						
1,1,2,2-Tetrachloroethane	6.8E-03	1.8E-07	1.3E-06	8.6E-06			
Tetrachloroethene	1.2E-01	3.0E-06	2.1E-05	1.5E-04	0.0003	0.0021	0.015
Trichloroethene	1.3E-02	3.4E-07	2.4E-06	1.6E-05			
CUMULATIVE NONCARCINOGENIC RISK					0.02	0.1	1

TABLE 7.3. NONCARCINOGENIC RISKS ASSOCIATED WITH CONSUMING BLUEGILL FILLETS (TAKEN FROM THE ASHTABULA HARBOR) UNDER TYPICAL, REASONABLE MAXIMUM (RME), AND SUBSISTENCE EXPOSURE SCENARIOS

Chemical	Fish Conc. (mg/kg)	Noncarcinogenic Intake (mg/kg-day)			Hazard Index (Intake/RfD)		
		Typical	RME	Subsistence	Typical	RME	Subsistence
METALS							
Chromium VI	1.6E+00	4.2E-05	3.0E-04	2.0E-03	0.0084	0.059	0.40
Copper	ND						
Mercury (methyl)	7.3E-02	1.9E-06	1.4E-05	9.2E-05	0.0064	0.045	0.31
Silver	ND						
Zinc	1.7E+01	4.3E-04	3.1E-03	2.1E-02	0.0022	0.015	0.10
ORGANICS							
PCBs (Aroclor 1260)	ND						
1,1,2,2-Tetrachloroethane	ND						
Tetrachloroethene	ND						
Trichloroethene	ND						
CUMULATIVE NONCARCINOGENIC RISK					0.02	0.1	0.8

TABLE 7.4. NONCARCINOGENIC RISKS ASSOCIATED WITH CONSUMING BLUEGILL FILLETS (TAKEN FROM THE ASHTABULA RIVER DOWNSTREAM FROM FIELDS BROOK) UNDER TYPICAL, REASONABLE MAXIMUM (RME), AND SUBSISTENCE EXPOSURE SCENARIOS

Chemical	Fish Conc. (mg/kg)	Noncarcinogenic Intake (mg/kg-day) Typical	RME	Subsistence	Hazard Index (Intake/RfD) Typical	RME	Subsistence
METALS							
Chromium VI	1.4E+00	3.7E-05	2.6E-04	1.8E-03	0.0074	0.052	0.35
Copper	5.5E-01	1.4E-05	1.0E-04	7.0E-04	0.011	0.078	0.54
Mercury (methyl)	1.7E-01	4.5E-06	3.1E-05	2.2E-04	0.015	0.10	0.72
Silver	ND						
Zinc	2.3E+01	5.9E-04	4.2E-03	2.8E-02	0.0030	0.021	0.14
ORGANICS							
PCBs (Aroclor 1260)	ND						
1,1,2,2-Tetrachloroethane	ND						
Tetrachloroethene	ND						
Trichloroethene	ND						
CUMULATIVE NONCARCINOGENIC RISK					0.04	0.2	2

TABLE 7.5. NONCARCINOGENIC RISKS ASSOCIATED WITH CONSUMING WHOLE CARP (TAKEN FROM THE ASHTABULA HARBOR) UNDER TYPICAL, REASONABLE MAXIMUM (RME), AND SUBSISTENCE EXPOSURE SCENARIOS

Chemical	Fish Conc. (mg/kg)	Noncarcinogenic Intake (mg/kg-day) Typical	RME	Subsistence	Hazard Index (Intake/RfD) Typical	RME	Subsistence
METALS							
Chromium VI	1.5E+00	4.0E-05	2.8E-04	1.9E-03	0.0079	0.055	0.38
Copper	1.5E+00	4.0E-05	2.8E-04	1.9E-03	0.030	0.21	1.5
Mercury (methyl)	ND						
Silver	ND						
Zinc	8.3E+01	2.2E-03	1.5E-02	1.0E-01	0.011	0.077	0.53
ORGANICS							
PCBs (Aroclor 1260)	1.5E-01	4.0E-06	2.8E-05	1.9E-04			
1,1,2,2-Tetrachloroethane	5.5E-03	1.4E-07	1.0E-06	7.0E-06			
Tetrachloroethene	3.0E-02	7.9E-07	5.5E-06	3.8E-05	0.00008	0.0006	0.0038
Trichloroethene	7.3E-03	1.9E-07	1.4E-06	9.2E-06			
CUMULATIVE NONCARCINOGENIC RISK					0.05	0.3	2

TABLE 7.6. NONCARCINOGENIC RISKS ASSOCIATED WITH CONSUMING WHOLE CARP (TAKEN FROM THE ASHTABULA RIVER DOWNSTREAM FROM FIELDS BROOK) UNDER TYPICAL, REASONABLE MAXIMUM (RME), AND SUBSISTENCE EXPOSURE SCENARIOS

Chemical	Fish Conc. (mg/kg)	Noncarcinogenic Intake (mg/kg-day)			Hazard Index (Intake/RfD)		
		Typical	RME	Subsistence	Typical	RME	Subsistence
METALS							
Chromium VI	2.0E+00	5.3E-05	3.7E-04	2.5E-03	0.010	0.074	0.51
Copper	1.7E+01	4.5E-04	3.2E-03	2.2E-02	0.35	2.4	17
Mercury (methyl)	4.6E-02	1.2E-06	8.5E-06	5.8E-05	0.0040	0.028	0.19
Silver	3.2E-01	8.4E-06	5.9E-05	4.0E-04	0.0017	0.012	0.081
Zinc	9.6E+01	2.5E-03	1.8E-02	1.2E-01	0.013	0.089	0.61
ORGANICS							
PCBs (Aroclor 1260)	8.1E-01	2.1E-05	1.5E-04	1.0E-03			
1,1,2,2-Tetrachloroethane	2.8E-02	7.4E-07	5.2E-06	3.5E-05			
Tetrachloroethene	2.7E-01	7.1E-06	5.0E-05	3.4E-04	0.0007	0.0050	0.034
Trichloroethene	4.0E-02	1.0E-06	7.4E-06	5.1E-05			
CUMULATIVE NONCARCINOGENIC RISK					0.4	3	20

TABLE 7.7. CARCINOGENIC RISKS ASSOCIATED WITH CONSUMING LARGE MOUTH BASS FILLETS (TAKEN FROM THE ASHTABULA RIVER DOWNSTREAM FROM FIELDS BROOK) UNDER TYPICAL, REASONABLE MAXIMUM (RME), AND SUBSISTENCE EXPOSURE SCENARIOS

Chemical	Fish Conc. (mg/kg)	Carcinogenic Intake (mg/kg-day) Typical	RME	Subsistence	Lifetime Cancer Risk (Intake*Slope Factor) Typical	RME	Subsistence
METALS							
Chromium VI	1.2E+00	4.1E-06	9.5E-05	6.5E-04			
Copper	ND						
Mercury (methyl)	1.5E-01	5.1E-07	1.2E-05	8.1E-05			
Silver	ND						
Zinc	1.1E+01	3.7E-05	8.6E-04	5.9E-03			
ORGANICS							
PCBs (Aroclor 1260)	ND						
1,1,2,2-Tetrachloroethane	6.8E-03	2.3E-08	5.4E-07	3.7E-06	4.6E-09	1.1E-07	7.4E-07
Tetrachloroethene	1.2E-01	3.9E-07	9.2E-06	6.3E-05			
Trichloroethene	1.3E-02	4.4E-08	1.0E-06	7.0E-06			
CUMULATIVE CARCINOGENIC RISK					5E-09	1E-07	7E-07

TABLE 7.8. CARCINOGENIC RISKS ASSOCIATED WITH CONSUMING WHOLE CARP (TAKEN FROM THE ASHTABULA HARBOR) UNDER TYPICAL, REASONABLE MAXIMUM (RME), AND SUBSISTENCE EXPOSURE SCENARIOS

Chemical	Fish Conc. (mg/kg)	Carcinogenic Intake (mg/kg-day)			Lifetime Cancer Risk (Intake*Slope Factor)		
		Typical	RME	Subsistence	Typical	RME	Subsistence
METALS							
Chromium VI	1.5E+00	5.1E-06	1.2E-04	8.1E-04			
Copper	1.5E+00	5.1E-06	1.2E-04	8.1E-04			
Mercury (methyl)	ND						
Silver	ND						
Zinc	8.3E+01	2.8E-04	6.6E-03	4.5E-02			
ORGANICS							
PCBs (Aroclor 1260)	1.5E-01	5.1E-07	1.2E-05	8.1E-05	3.9E-06	9.2E-05	6.3E-04
1,1,2,2-Tetrachloroethane	5.5E-03	1.9E-08	4.4E-07	3.0E-06	3.7E-09	8.7E-08	6.0E-07
Tetrachloroethene	3.0E-02	1.0E-07	2.4E-06	1.6E-05			
Trichloroethene	7.3E-03	2.5E-08	5.8E-07	4.0E-06			
CUMULATIVE CARCINOGENIC RISK					4E-06	9E-05	6E-04

TABLE 7.9. CARCINOGENIC RISKS ASSOCIATED WITH CONSUMING WHOLE CARP (TAKEN FROM THE ASHTABULA RIVER DOWNSTREAM FROM FIELDS BROOK) UNDER TYPICAL, REASONABLE MAXIMUM (RME), AND SUBSISTENCE EXPOSURE SCENARIOS

Chemical	Fish Conc. (mg/kg)	Carcinogenic Intake (mg/kg-day)			Lifetime Cancer Risk (Intake*Slope Factor)		
		Typical	RME	Subsistence	Typical	RME	Subsistence
METALS							
Chromium VI	2.0E+00	6.8E-06	1.6E-04	1.1E-03			
Copper	1.7E+01	5.8E-05	1.4E-03	9.3E-03			
Mercury (methyl)	4.6E-02	1.6E-07	3.6E-06	2.5E-05			
Silver	1.1E-06	1.1E-06	2.5E-05	1.7E-04			
Zinc	9.6E+01	3.3E-04	7.6E-03	5.2E-02			
ORGANICS							
PCBs (Aroclor 1260)	8.1E-01	2.7E-06	6.4E-05	4.4E-04	2.1E-05	4.9E-04	3.4E-03
1,1,2,2-Tetrachloroethane	2.8E-02	9.5E-08	2.2E-06	1.5E-05	1.9E-08	4.4E-07	3.0E-06
Tetrachloroethene	2.7E-01	9.1E-07	2.1E-05	1.5E-04			
Trichloroethene	4.0E-02	1.4E-07	3.2E-06	2.2E-05			
CUMULATIVE CARCINOGENIC RISK					2E-05	5E-04	3E-03

CHAPTER 8

CHARACTERIZATION OF QUALITATIVE UNCERTAINTIES

8.1 INTRODUCTION

A number of assumptions and estimated values are used in baseline human health risk assessments that contribute to the overall level of uncertainty about the risk estimates. For most environmental risk assessments, the uncertainty of the risk estimates varies by at least an order of magnitude or greater (USEPA, 1989a). In this chapter, the key site-related variables and assumptions that contribute the greatest degree of uncertainty will be examined in a qualitative way.

8.2 QUALITATIVE LIST OF UNCERTAINTIES

8.2.1 Data Compilation and Evaluation

The data compilation and evaluation step is one part of the risk assessment process where uncertainties arise. These uncertainties are listed below for the following assumptions and statements.

- **The available data for contaminant levels in fish and water samples collected from the Ashtabula River and Harbor were representative of the true distribution of contaminants in the Ashtabula River AOC.** A moderate level of uncertainty is probably associated with this assumption. Additional sampling over a longer period of time would be needed to look for any temporal or spatial variability in contaminant levels, and to obtain a more representative profile of contaminant concentrations in the media of interest.

- **Contaminant burdens in fish may decrease depending on how the fish is prepared and cooked.** Contaminant levels may be reduced 10-70% depending on how the fish is prepared and cooked (H. Humphrey, Michigan Department of Public Health, personal communication, 1991). Because of this wide range, the uncertainty associated with the resulting overestimation of risk is not well established.

- **The selection of a subset of fish by the Ohio EPA for laboratory analysis was appropriate.** The criteria by which fish were chosen for analysis was not described in the Ashtabula River Report (WCC, 1991). A low to moderate level of uncertainty is probably associated with this assumption.

8.2.2 Exposure Assessment

A number of assumptions were made in the exposure assessment step of this baseline human health risk assessment.

- **An adequate assessment of complete and incomplete exposure pathways was made.** There is a low uncertainty that some exposure pathways were either not identified or else were incorrectly classified as a complete or incomplete exposure pathway.

- **The exclusion of some complete exposure pathways (i.e., dermal exposure to water and sediments) from the exposure assessment was justifiable because of the low probability that these pathways would result in significant human health risks.** The uncertainty associated with this assumption is probably low. The estimated risk from ingesting contaminated water from the Ashtabula River (near Fields Brook) while swimming (3 events per year) was very low, and this pathway usually results in a greater risk than the dermal exposure pathways (for similar exposure frequencies).

- **The complete exposure pathways chosen for the exposure assessment represent the primary pathways by which people in the Ashtabula River AOC were exposed to contaminants.** The pathways chosen were based primarily on observed activities and on available data. A low level of uncertainty is probably associated with not being able to include these incomplete exposure pathways.

- **The assumptions made about exposure frequency and duration variables, body weight, life expectancy, and population characteristics were appropriate.** Many of these assumptions (e.g., body weight, life expectancy, exposure frequency) were based on EPA guidance (USEPA, 1989a,b; 1991a) and probably have a low to moderate level of uncertainty associated with them. A similar level of uncertainty may be attributed to professional judgments about the fraction of fish ingested from contaminated sources.

8.2.3 Toxicity Values

The toxicity values (i.e., oral RfDs and oral slope factors) used in this risk assessment were either verified values obtained from IRIS or interim values obtained from other sources. RfDs and slope factors are subject to change as the result of new information and updates of the IRIS data base. In addition, chemicals will be added to IRIS in the future to expand the data base. Thus, this risk assessment is "dated" to the toxicity values available at the time it was

prepared. Listed below are the uncertainties associated with using these toxicity values.

- **RfD values and slope factors have a certain amount of uncertainty associated with them.** Uncertainty and modifying factors are incorporated into the calculation of RfDs (see Appendix B) and take into consideration factors such as extrapolating data from long-term animal studies to humans, etc. In general, RfD values have an uncertainty range of about one order of magnitude. Since slope factors represent an estimate of an upper-bound lifetime probability of an individual developing cancer, these values are already conservative. Thus, the amount of uncertainty associated with slope factor values may be minimized.

- **A conservative assumption for metal speciation in the Ashtabula River AOC was made for chromium and mercury because toxicity values for the total metal form were not available.** Thus, toxicity values for chromium VI and methyl mercury were used to represent the major forms of these heavy metals. The use of this more toxic chemical species resulted in a conservative estimate of risk. A moderate level of uncertainty is probably associated with this uncertainty.

- **Two organic chemicals were excluded from the risk assessment because their toxicity values have been retracted from IRIS for further review.** The oral slope factors for tetrachloroethene and trichlorethene have been withdrawn from IRIS. The exclusion of these chemicals from the carcinogenic risk assessment was probably minor because the use of the old oral slope factors resulted in a small proportion of the total risk estimate.

8.2.4 Risk Characterization

The uncertainties associated with the risk characterization step are listed below.

- **Exposure intakes and toxicity values will remain the same over the exposure duration.** This assumes that human activities and contaminant levels will remain the same over the exposure duration, and that toxicity values will not be updated. A moderate to high level of uncertainty is probably associated with this assumption since it does not take into consideration the implementation of remedial actions or the deposition of cleaner sediments over contaminated sediments. Furthermore, toxicity values are frequently updated in the IRIS data base as new information becomes available.

The level of uncertainty will probably increase with longer exposure durations.

- **Health risks are additive for both noncarcinogenic and carcinogenic effects.** The uncertainty associated with this assumption is unknown. The toxicity exhibited by a mixture of chemicals may involve synergistic and antagonistic effects. However, no guidelines are available to judge the complex interactions a mixture of contaminants may possess in terms of its potential toxicity to humans. At the present time, standard risk assessment guidance assumes that health risks are additive.

8.3 SUMMARY

Based on the information available, a complete description of the level of uncertainty associated with all of the assumptions and data used in this risk assessment cannot be made. This baseline human health risk assessment was based on data and assumptions that, in reality, represent a snapshot in time. One of the greatest sources of uncertainty in this risk assessment arises from assuming that estimated risks will remain constant over the exposure duration (i.e., 9 years for typical exposures and 30 years for reasonable maximum and subsistence exposures). The overall uncertainty of the risk estimates probably varies by over an order of magnitude. As additional data are collected from the Ashtabula River and as additional (or revised) toxicity values are generated, a better estimate of human health risk can be determined for people living in this area. Thus, updates of this risk assessment will probably reduce the level of uncertainty associated with it.

REFERENCES

Ashtabula Area Chamber of Commerce. 1991. Ashtabula Visitors Guide. Ashtabula Area Chamber of Commerce, Ashtabula, OH.

Batterman, A.R., P.M. Cook, K.B. Lodge, D.B. Lothenbach, and B.C. Butterworth. 1989. Methodology Used for a Laboratory Determination of Relative Contributions of Water, Sediment and Food Chain Routes of Uptake for 2,3,7,8-TCDD Bioaccumulation by Lake Trout in Lake Ontario. Chemosphere. 19:451-458.

Bierman, V.J., Jr. 1990. Equilibrium Partitioning and Biomagnification of Organic Chemicals in Benthic Animals. Environ. Sci. Technol. 24:1407-1412.

Borgmann, U. and D.M. Whittle. 1991. Contaminant Concentration Trends in Lake Ontario Lake Trout (Salvelinus namaycush): 1977 to 1988. J. Great Lakes Res. 17:368-381.

Bro, K.M. 1989. Setting Priorities for Investigating Chemical Contaminants in the Great Lakes. Ph.D. thesis. University of Wisconsin-Madison.

Brown, M.P., M.B. Werner, R.J. Sloan, and K.W. Simpson. 1985. Polychlorinated Biphenyls in the Hudson River. Environ. Sci. Technol. 19:656-661.

CH_2M Hill. 1986. Public Comment - Feasibility Study Fields Brook Site. Sediment Operable Unit, Ashtabula, Ohio. EPA 19.5L46.0, July 3, 1986. Prepared for U.S. Environmental Protection Agency Hazardous Site Control Division under EPA Contract No. 68-01-6692.

Connor, M.S. 1984. Fish/Sediment Concentration Ratios for Organic Compounds. Environ. Sci. Technol. 18:31-35.

Foran, J.A. and D. VanderPloeg. 1989. Consumption Advisories for Sport Fish in the Great Lakes Basin: Jurisdictional Inconsistencies. J. Great Lakes Res. 15:476-485.

Gill, G.A. and K.W. Bruland. 1990. Mercury Speciation in Surface Freshwater Systems in California and other Areas. Environ. Sci. Technol. 24:1392-1400.

Henderson, B.E., R.K. Ross, and M.C. Pike. 1991. Toward the Primary Prevention of Cancer. Science. 254:1131-1138.

Jaffe, R., E.A. Stemmler, B.D. Eitzer, and R.A. Hites. 1985. Anthropogenic, Polyhalogenated, Organic Compounds in Sedentary Fish from Lake Huron and Lake Superior Tributaries and Embayments. J. Great Lakes Res. 11:156-162.

Kononen, D.W. 1989. PCBs and DDT in Saginaw Bay White Suckers. Chemosphere. 18:2065-2068.

Kuehl, D.W., P.M. Cook, A.R. Batterman, D. Lothenbach, and B.C. Butterworth. 1987. Bioavailability of Polychlorinated Dibenzo-p-dioxins and Dibenzofurans from Contaminated Wisconsin River Sediment to Carp. Chemosphere. 16:667-679.

Malins, D.C., B.B. McCain, D.W. Brown, S-L. Chan, M.S. Myers, J.T. Landahl, P.G. Prohaska, A.J. Friedman, L.D. Rhodes, D.G. Burrows, W.D. Gronlund, and H.O. Hodgins. 1984. Chemical Pollutants in Sediments and Diseases of Bottom-dwelling Fish in Puget Sound, Washington. Environ. Sci. Technol. 18:705-713.

Michigan Department of Natural Resources (MDNR). 1991. Michigan Fishing Guide. Michigan Department of Natural Resources, Fisheries Division, Lansing, MI.

Ohio EPA. 1991 (August). Ashtabula River RAP. Stage 1 Draft. Ohio EPA, Division of Water Quality Planning and Assessment.

Oliver, B.G. and A.J. Niimi. 1988. Trophodynamic Analysis of Polychlorinated Biphenyl Congeners and other Chlorinated Hydrocarbons in the Lake Ontario Ecosystem. Environ. Sci. Technol. 22:388-397.

Pizza, J.C. and J.M. O'Connor. 1983. PCB Dynamics in Hudson River Striped Bass. II. Accumulation from Dietary Sources. Aquatic Toxicol. 3:313-327.

Seelye, J.G., R.J. Hesselberg, and M.J. Mac. 1982. Accumulation by Fish of Contaminants Released from Dredged Sediments. Environ. Sci. Technol. 16:459-464.

Smith, W.E., K. Funk, and M.E. Zabik. 1973. Effects of Cooking on Concentrations of PCB and DDT Compounds in Chinook (Oncorhynchus tshawytscha) and Coho (O. kisutch) Salmon from Lake Michigan. J. Fish. Res. Board Can. 30:702-706.

Spigarelli, S.A., M.M. Thommes, and W. Prepejchal. 1983. Thermal and Metabolic Factors Affecting PCB Uptake by Adult Brown Trout. Environ. Sci. Technol. 17:88-94.

Stachiw, N., M.E. Zabik, A.M. Booren, and M.J. Zabik. 1988. Tetrachlorodibenzo-p-dioxin Residue Reduction through Cooking/Processing of Restructured Carp Fillets. J. Agric. Food Chem. 36:848-852.

Swackhamer, D.L. and R.A. Hites. 1988. Occurrence and Bioaccumulation of Organochlorine Compounds in Fishes from Siskiwit Lake, Isle Royale, Lake Superior. Environ. Sci. Technol. 22: 543-548.

Swain, W.R. 1991. Effects of Organochlorine Chemicals on the Reproductive Outcome of Humans who Consumed Contaminated Great Lakes Fish: an Epidemiologic Consideration. J. Toxicol. Environ. Health. 33:587-639.

Thomann, R.V. and J.P. Connolly. 1984. Model of PCB in the Lake Michigan Lake Trout Food Chain. Environ. Sci. Technol. 18:65-71.

U.S. Army Corps of Engineers. 1988. Ashtabula River and Harbor Suspended Sediment Movement During Dredging. Prepared in response to Ohio EPA comments on Final Environmental Impact Statement for dredging Ashtabula Harbor (1987).

U.S. EPA. 1988a. Risk Management Recommendations for Dioxin Contamination at Midland, Michigan. Final Report. Region 5, Chicago, IL. EPA-905/4-88-008.

U.S. EPA. 1988b. Superfund Exposure Assessment Manual. Office of Remedial Response, Washington, D.C. EPA/540/1-88/001.

U.S. EPA. 1989a. Risk Assessment Guidance for Superfund: Human Health Evaluation Manual Part A. Interim Final. OSWER Directive 9285.7-01a.

U.S. EPA. 1989b. Exposure Factors Handbook. Office of Health and Environmental Assessment, Washington, D.C. EPA/600/8-89/043.

U.S. EPA. 1989c. Health Effects Assessment Summary Tables. Fourth Quarter, FY 1989. OERR 9200.6-303-(89-4).

U.S. EPA. 1991a. Risk Assessment Guidance for Superfund. Volume I: Human Health Evaluation Manual. Supplemental Guidance: "Standard Default Exposure Factors." Interim Final (March 25, 1991). OSWER Directive 9285.6-03.

U.S. EPA. 1991b. ARCS: Assessment and Remediation of Contaminated Sediments. 1991 Work Plan. Great Lakes National Program Office, Chicago, IL.

Weininger, D. 1978. Accumulation of PCBs by Lake Trout in Lake Michigan. Ph.D. thesis. University of Wisconsin-Madison, Madison, WI.

West, P.C., J.M. Fly, R. Marans, and F. Larkin. 1989. Michigan Sport Anglers Fish Consumption Survey. University of Michigan School of Natural Resources, Natural Resource Sociology Research Lab Technical Report #1.

Woodward-Clyde Consultants (WCC). 1991 (March 29). Ashtabula River Investigation. Draft Report: Ashtabula, Ohio. Prepared for: The Ashtabula River Group. 86C3609F-510.

Zabik, M.E., P. Hoojjat, and C.M. Weaver. 1979. Polychlorinated Biphenyls, Dieldrin and DDT in Lake Trout Cooked by Broiling, Roasting or Microwave. Bull. Environm. Contam. Toxicol. 21:136-143.

Zabik, M.E., C. Merrill, and M.J. Zabik. 1982. PCBs and Other Xenobiotics in Raw and Cooked Carp. Bull. Environm. Contam. Toxicol. 28:710-715.

APPENDIX A

IMPORTANCE OF OTHER COMPLETE EXPOSURE PATHWAYS IN THE ASHTABULA RIVER AREA OF CONCERN

The dermal exposure of people to water and sediments in the Ashtabula River was assumed to be insignificant based on the frequency with which these exposures would take place and also in comparison to the estimated noncarcinogenic and carcinogenic risk from ingesting surface water while swimming in the Ashtabula River. In this appendix, these assumptions and estimated risk estimates will be described.

Dermal contact with Ashtabula River sediments may occur infrequently because there are no designated swimming areas along shore where someone could wade into the water. Another place where dermal contact with sediments may occur is at the boat ramps when people are putting in or taking out their boats; however, most people would probably be wearing some kind of foot protection to shield their feet from rocks, broken glass, etc. People may have dermal contact with water as their boats travel from the marina areas out to the harbor or Lake Erie.

Although limited dermal exposures to water and sediment may take place in the Ashtabula River, it is more difficult to determine the risks from these pathways than from an ingestion or inhalation pathway. This is because toxicity values are not developed specifically for dermal sorption, nor are absorption rates developed well for contaminants. Thus, the estimation of exposure for the dermal pathway, calculated as an absorbed dose, has a greater amount of uncertainty associated with it than exposures that are based on an actual intake of contaminant into the body. Based on dermal exposures calculated for other more contaminated ARCS sites, dermal exposures to water and sediments in the Ashtabula River are not likely to result in significant noncarcinogenic and carcinogenic risks. In addition, a greater risk is likely to be encountered due to the ingestion of surface water from the Ashtabula River than dermal exposures to it. This is because of the direct intake of contaminants into the gut versus the absorption of contaminants (with varying levels of permeability) across the skin interface.

The human health risk resulting from the ingestion of surface water from the Ashtabula River (collected at the mouth of Fields Brook) was estimated based on exposure and risk assessment guidance developed for the EPA Superfund program (USEPA, 1989a). The noncarcinogenic and carcinogenic intake values were calculated using the following equation:

$$\text{Intake (mg/kg-day)} = \frac{\text{CW x CR x ET x EF x ED}}{\text{BW x AT}}$$

where:

CW = Chemical concentration in water (mg/L)
CR = Contact rate: used 0.05 L/hr
ET = Exposure time: used 0.5 hr/day (study assumption)
EF = Exposure frequency: used 3 days/year (study assumption)
ED = Exposure duration: used 30 years for a reasonable maximum exposure scenario
BW = Body Weight: used 70 kg
AT = Averaging Time: ED x 365 days/year for noncarcinogenic risk (i.e., 1.09×10^4 days) and 70 years x 365 days/year (i.e., 2.56×10^4 days) for carcinogenic risk

The assumptions incorporated into the above equation were either study assumptions, where noted, or else were recommended values given in EPA Superfund Guidance (USEPA, 1989a).

The chemical intake values were incorporated with toxicity estimates to produce the risk estimates. The estimated noncarcinogenic risk is given in Table A.1. The Hazard Index was very low (i.e., HI = 0.0003); thus the consumption of Ashtabula River water during infrequent swimming events appears to pose little noncarcinogenic risk. The upper-bound carcinogenic risk estimate was also quite low (i.e., 4×10^{-10}) (Table A.2). The EPA believes it is prudent public health policy to consider actions to mitigate or minimize exposures to contaminants when estimated excess lifetime cancer risks exceed the 10^{-5} to 10^{-6} range (USEPA, 1988a). Based on this low carcinogenic risk estimate for the ingestion of surface water, dermal exposures to water and sediments were assumed to be insignificant.

TABLE A.1. NONCARCINOGENIC RISK ASSOCIATED WITH THE INGESTION OF WATER FROM THE ASHTABULA RIVER DURING INFREQUENT SWIMMING EVENTS

Chemical	Water Conc. (mg/L)	Noncarc. Intake (mg/kg-day)	Oral RfD (mg/kg-day)	HI Intake/RfD
METALS				
Barium	8.77E-02	2.68E-07	7.00E-02	3.8E-06
Copper	1.29E-01	3.95E-07	1.30E-03	3.0E-04
Manganese	3.19E-01	9.77E-07	1.00E-01	9.8E-06
Zinc	2.03E-02	6.21E-08	2.00E-01	3.1E-07
ORGANICS				
Acetone	8.70E-02	2.66E-07	1.00E-01	2.7E-06
Methylene Chloride	3.90E-02	1.19E-07	6.00E-02	2.0E-06
Vinyl Acetate	4.60E-01	1.41E-06	1.00E+00	1.4E-06
				0.0003

TABLE A.2. CARCINOGENIC RISK ASSOCIATED WITH THE INGESTION OF WATER FROM THE ASHTABULA RIVER DURING INFREQUENT SWIMMING EVENTS

Chemical	Water Conc. (mg/L)	Carcinogenic Intake (mg/kg-day)	Slope Factor 1/(mg/kg/day)	Cancer Risk
METALS				
Barium	8.77E-02	1.10E-07		
Copper	1.29E-01	1.62E-07		
Manganese	3.19E-01	4.01E-07		
Zinc	2.03E-02	2.55E-08		
ORGANICS				
Acetone	8.70E-02	1.09E-07		
Methylene Chloride	3.90E-02	4.91E-08	7.50E-03	3.7E-10
Vinyl Acetate	4.60E-01	5.79E-07		
				4E-10

APPENDIX B

HUMAN TOXICITY ESTIMATES FOR CONTAMINANTS PRESENT IN THE ASHTABULA RIVER AREA OF CONCERN

B.1 TOXICITY ASSESSMENT

The toxicity assessment step is an integral part of the human health baseline risk assessment. This step includes four tasks: 1) gather qualitative and quantitative toxicity information for substances being evaluated, 2) identify exposure periods for which toxicity values are necessary, 3) determine toxicity values (i.e., reference doses (RfDs)) for noncarcinogenic effects, and 4) determine toxicity values (i.e., slope factors) for carcinogenic effects (USEPA, 1989a). The EPA has performed the toxicity assessment step for a limited number of chemicals and these assessments have undergone extensive peer review. Therefore, the toxicity assessment step of this study involves primarily a compilation of available toxicity data.

Once a "verified" toxicity value is agreed upon by the EPA's toxicologists, it is entered into the EPA's Integrated Risk Information System (IRIS) data base; these values are updated as necessary. IRIS is the primary source of toxicity information used in baseline risk assessments. The Health Effects Assessment Summary Tables (HEAST) are the second most current source of toxicity information and include both verified and interim RfD and slope factor values. Interim values are used for chemicals that have not yet been approved by the EPA. Specific EPA workgroups, such as the Carcinogen Risk Assessment Verification Endeavor (CRAVE) and RfD Workgroups, are another source of interim toxicity values. If toxicity values are not available in the aforementioned sources, then interim values from other reports may be used.

This appendix summarizes pertinent toxicity information obtained from IRIS and other sources for chemicals in the lower 3.2 km of the Ashtabula River and Harbor. Also included in this appendix are brief descriptions of the most important toxicity values used to evaluate noncarcinogenic and carcinogenic effects; these subsections were summarized from the EPA guidance document: "Risk Assessment Guidance for Superfund. Volume 1. Human Health Evaluation Manual (Part A)" (USEPA, 1989a).

B.1.1 Noncarcinogenic Chronic Toxicity

The RfD is the toxicity value used most often in evaluating noncarcinogenic effects. RfDs are based on the assumption that thresholds exist for certain toxic effects (e.g., cellular necrosis) but may not exist for other toxic effects (e.g., carcinogenicity). The RfD is defined as an estimate of the daily exposure to the

human population that is likely to be without an appreciable risk of deleterious effects during either a portion of the lifetime (i.e., subchronic RfD or "RfD_s") or during the lifetime (i.e., chronic RfD or "RfD"). This toxicity value has an uncertainty range of about an order of magnitude and includes exposures to sensitive subgroups in the population. For each chemical, the RfD is calculated from the following equation:

$$RfD = \frac{NOAEL \text{ or } LOAEL}{UF \times MF}$$

where:

NOAEL = No-Observed-Adverse-Effect-Level

LOAEL = Lowest-Observed-Adverse-Effect-Level

MF = Modifying Factor

UF = Uncertainty Factor

The NOAEL and LOAEL are derived from dose-response experiments. The NOAEL represents the highest exposure level tested at which no adverse effects occurred (including the critical toxic effect), whereas the LOAEL represents the lowest exposure level at which significant adverse effects occurred. Uncertainty factors usually consist of multiples of ten, with each factor representing a specific area of uncertainty included in the extrapolation from available data. An uncertainty factor of ten is usually used to account for variation in the general population so that sensitive subpopulations are protected. An additional ten-fold factor is usually applied for each of the following extrapolations: from long-term animal studies to humans, from a LOAEL to a NOAEL, and when subchronic studies are used to derive a chronic RfD. A modifying factor (MF), ranging from >0 to 10, is included as a qualitative assessment of additional uncertainties; the default value for the MF is one.

Table B-1 includes the uncertainty and modifying factors, confidence classifications, and critical effects of the contaminants examined for this risk assessment. Uncertainty factors ranged from 3 to 1000, and either a low or medium level of confidence was given for these RfD values. Better estimates of oral RfD values are needed to reduce these levels of uncertainty.

B.1.2 Carcinogenicity

Human carcinogenic risks are usually evaluated for a chemical by using its slope factor (formerly designated as a cancer potency factor) and corresponding weight-of-evidence classification. These variables were listed in Table 6.2 for the Ashtabula River chemicals. Slope factors are estimated through the use of mathematical extrapolation models, most commonly the linearized multistage model, for estimating the largest possible linear slope (within 95% confidence limits), at low extrapolated doses, that is consistent with the data. The slope

TABLE B-1. ORAL RfD SUMMARY FOR CHEMICALS LISTED IN IRIS AS OF 24 DECEMBER 1991

Chemical	UF[1]	MF[2]	Confidence in Oral RfD	Critical Effects
METALS				
Chromium VI	500	1	Low	No effects reported
Copper	Under Review			
Mercury, methyl	10	1	Medium	Central nervous system effects in humans
Silver	3	1	Low	Argyria (bluish-gray discoloration of the skin) in humans
Zinc	Under Review			
ORGANICS				
Tetrachloroethene	1000	1	Medium	Hepatotoxicity in mice, weight gain in rats

[1] UF = Uncertainty Factor [2] MF = Modifying Factor

factor is characterized as an upper-bound estimate so that the true risk to humans, while not identifiable, is not likely to exceed the upper-bound estimate.

The weight of evidence classification for a particular chemical is determined by the EPA's Human Health Assessment Group (HHAG). Chemicals are placed into one of five groups according to the weight of evidence from epidemiological studies and animal studies. These groups are designated by the letters A, B, C, D, and E which represent the level of carcinogenicity to humans (see Table 6.1). Quantitative carcinogenic risk assessments are performed for chemicals in Groups A and B, and on a case-by-case basis for chemicals in Group C.

B.2 UNCERTAINTIES

A number of uncertainties are involved with using toxicity values for estimating noncarcinogenic and carcinogenic risks. Some of these qualitative uncertainties are listed below:

- Using dose-response information from healthy animal or human populations to predict effects that may occur in the general population, including susceptible subpopulations (e.g., elderly, children),
- Using dose-response information from animal studies to predict effects that may occur in human populations,
- Using NOAELs derived from short-term animal studies to predict effects that may occur in humans during long-term exposures,
- Using dose-response information from effects observed at high doses to predict the adverse health effects that may occur following exposure of humans to low levels of the chemical in the environment, and

- Using a toxicity value derived from exposure to a particular chemical mixture (e.g., Aroclor 1260) to represent the level of toxicity for other similar chemical mixtures (e.g., Aroclor 1242, 1248, and 1254).

*U.S.GOVERNMENT PRINTING OFFICE 1993-747-249