EVALUATION OF PAH SEDIMENT CONTAMINATION IN USEPA REGION V AND THE GREAT LAKES

EPA 905-R-87-117

EVALUATION OF PAH SEDIMENT CONTAMINATION IN USEPA REGION V AND THE GREAT LAKES

Submitted by:

Science Applications
International Corporation
8400 Westpark Drive
McLean, Virginia 22102

Submitted to:

Howard Zar, Project Monitor
U.S. Environmental Protection Agency
Region V
230 S. Dearborn Street
Chicago, Illinois 60604

EPA Contract No. 68-04-5035, D.O. #012
SAIC Project No. 2-813-06-193-12

September 1987

ABSTRACT

The nature and extent of polycyclic aromatic hydrocarbon (PAH) contamination of sediments in 384 reported sampling sites in USEPA Region V and the Great Lakes was characterized and compared. Sampling sites were geographically aggregated into 10 regions and 35 associated subregions, to facilitate comparisons.

Based on exposure assumptions and available toxicological and physical mobility data for PAHs, human carcinogenic, human non-carcinogenic and acute aquatic toxicity risk scores were calculated for each PAH at each reported sampling site. These scores were then aggregated to derive mean risk scores for sites, subregions and regions. Comparative ranking scores for sampling sites within subregions, and subregions within regions, were then calculated.

Due to the sparcity of available toxicological parameters on PAHs, assumptions were necessary to derive risk scores. These assumptions involved determinations of carcinogenicity, UCRs, ADIs, and acute aquatic life criteria. These assumptions probably resulted in the calculation of risk scores considerably higher than those which would be derived from the same sites if adequate data were available. For this reason, the computed risk scores should be used for the purposes of comparative rankings rather than derivation of absolute risks.

The highest PAH sediment concentrations were generally measured in the vicinity of coke plants or otherwise unidentified steel industry outfalls, however elevated concentrations were also reported near petroleum handling and storage facilities, storm sewer or CSO outfalls, POTW discharges, waste disposal and dredge sediment disposal sites, and miscellaneous industrial discharges.

Of the 10 regions compared, the highest human carcinogenic, human non-carcinogenic and acute aquatic toxicity risk scores were calculated from the northwest Indiana region, which includes the Grand Calumet River and the Indiana Harbor/Canal. The mean PAH sediment concentration of the Indiana Region is more than four times that of the second and third ranking regions (Ohio Steel and Milwaukee regions, respectively). With respect to mean human

carcinogenic, non-carcinogenic and aquatic toxicity risk scores, the second/third ranking regions are the Milwaukee/Ohio Steel, Ohio Steel/Milwaukee and Ohio/Ohio Steel regions, respectively.

Based on available data, it is observed that PAHs settle out within very short distances downstream of the points of discharge. Related observations include the influence of sample site selection on accurate estimation of true PAH sediment concentrations, particularly as relates to refinement of risk calculation and interpretation procedures.

Due to the length and complexity of the report, a separate, comprehensive summary chapter is provided (Chapter 7).

TABLE OF CONTENTS

TABLE OF CONTENTS (Continued)

TABLE OF CONTENTS (Continued)

LIST OF FIGURES

LIST OF FIGURES (Continued)

LIST OF TABLES

LIST OF TABLES (Continued)

1. BACKGROUND INFORMATION ON PAHS

Polycyclic aromatic hydrocarbons (PAHs) are complex organic molecules formed by incomplete pyrolysis of organic matter. Although acute and chronic health effects have been demonstrated, little is known about the distribution of PAHs in the aquatic environment.

This report has been prepared to characterize the nature and extent of PAH contamination of sediments in USEPA Region V and the Great Lakes basin. This characterization includes calculation of crude sediment scores, reflecting potential risks to human health and aquatic life, and cross-comparison of various sediment sampling sites based on these calculated risk scores. It is emphasized that the characterization of risks is based on currently available information on PAH mobility in the aquatic environment and toxicological effects on human health and aquatic life. As this information is quite limited, at present, the assignment of risks in this report has involved a number of assumptions. These assumptions are discussed in the text.

It should be emphasized that evidence of PAHs causing significant increases in the incidences of tumor formation in fish populations is drawn from field studies of aquatic systems which are heavily contaminated with other potential carcinogens as well. Evidence that PAHs alone, even at extremely high levels, can cause increases in the incidence of tumor formation is inadequate, particularly for PAH levels that organisms could typically be expected to be exposed to in even highly contaminated aquatic systems. Field studies should be conducted to determine the degree to which PAHs alone, at levels which are encountered in highly contaminated aquatic systems, increase the incidences of tumor formation and/or other adverse effects in aquatic organisms.

This report is based on available PAH data, supplied by various USEPA sources. Geographically, these data include sites in USEPA Region V and the Great Lakes Basin.

Due to the length and complexity of this report, a separate, comprehensive summary is included, as Chapter 7.

Chapter 1 is divided into five sections, describing: physical, chemical and toxicological properties (1.1), sources (1.2), environmental fate and transport (1.3), PAH toxicity to humans and other mammals (1.4), and PAH toxicity to aquatic life (1.5).

1.1 PHYSICAL, CHEMICAL AND TOXICOLOGICAL PROPERTIES

Structures and various physical, chemical and toxicological properties are listed in Table 1-1 for PAHs that are frequently analyzed for in the environment. Structures and physical and chemical properties were taken primarily from USEPA (1981a). Most of the octanol/water partition coefficients (K_{ow}) listed in USEPA (1981a) for PAHs were estimated using the fragment constant method (Hansch and Leo in USEPA 1981a and in Lyman et al. 1982). Most of the organic fraction adjusted sediment/water partition coefficients (K_{oc}) listed for PAHs in USEPA (1981a) were calculated from K_{oc} values using the following equation (Hasset et al. 1980 in USEPA [1981a]):

$$K_{oc} = K_{ow} \ 10^{-0.317} \qquad (1\text{-}1)$$

The above equation was derived from a linear regression of log K_{oc} on log K_{ow} for various types of sediments and PAHs.

Most of the bioconcentration factors (BCF) listed in Table 1-1 were taken from USEPA (1980a). The bioconcentration factors listed in USEPA (1980a) for PAHs and those listed in Table 1-1 which were not listed in USEPA (1980a) were calculated from K_{oc} values using the following equation:

$$BCF = (3.0/7.6) \ K_{ow}^{0.85} \ 10^{-0.7} \qquad (1\text{-}2)$$

The above equation was derived from a linear regression of log BCF on log K_{ow} for fish with a 7.6% lipid content and various types of organics (Veith et al. 1979 in USEPA 1980a). The factor (3.0/7.6) is included in the equation to account for the difference between the lipid content of the experimental fish and the estimated consumption weighted mean lipid content of fish and shellfish consumed in the United States (USEPA 1980a).

Table 1-1. PAH Physical-Chemical and Toxicological Data[1]

PAH compound	Structure	MW	Vapor P (torr)	H ($atm\text{-}m^3\text{-}mol^{-1}$)	Solubility (mg/l)	K_{ow} (unitless)	K_{oc}[2] (unitless)	BCF[3] (unitless)	UCR[4] $(mg/kg/day)^{-1}$	ADI[5] (mg/kg/day)	Acute AQ[6] toxicity (mg/l)
Acenaphthene		154	1.55×10^{-3}	9.1×10^{-5}	3.42	9.6×10^{-3}	4.6×10^{3}	1.9×10^{2}	NC	5.9×10^{-3}	<1.7
Acenaphthylene		152	2.9×10^{-2}	1.5×10^{-3}	3.93	5.3×10^{-3}	2.5×10^{3}	1.2×10^{2}	NC	5.9×10^{-3}	<1.7
Anthracene		178	2.4×10^{-4}	1.3×10^{-3}	4.5×10^{-2}	2.8×10^{-3}	1.4×10^{4}	4.8×10^{2}	NC	5.9×10^{-3}	<1.7
Benzo[a]anthracene		228	2.2×10^{-8}	1.0×10^{-6}	5.7×10^{-3}	4.1×10^{-3}	2.0×10^{5}	4.6×10^{3}	11.5	C	<1.7
Benzo[b]fluoranthene		252	5.0×10^{-7}	1.2×10^{-5}	1.4×10^{-2}	1.15×10^{-3}	5.5×10^{5}	1.1×10^{4}	11.5	C	<1.7
Benzo[k]fluoanthene		252	5.0×10^{-7}	3.9×10^{-5}	4.3×10^{-3}	1.15×10^{-3}	5.5×10^{5}	1.1×10^{4}	11.5	C	<1.7
Benzo[g,h,i]perylene		276	1.03×10^{-10}	1.4×10^{7}	2.6×10^{-4}	3.2×10^{-3}	1.6×10^{6}	2.7×10^{4}	NC	5.9×10^{-3}	<1.7
Benzo[a]pyrene		252	5.6×10^{-9}	4.9×10^{-7}	3.8×10^{-3}	1.15×10^{-3}	5.5×10^{5}	1.1×10^{4}	11.5	C	<1.7
Chrysene		228	6.3×10^{-9}	1.1×10^{-7}	1.8×10^{-3}	4.1×10^{-3}	2.0×10^{5}	4.6×10^{3}	11.5	C	<1.7
Dibenzo[a,h]anthracene		278	1.0×10^{-10}	7.3×10^{-8}	5.0×10^{-4}	6.9×10^{-3}	3.3×10^{6}	4.5×10^{3}	11.5	C	<1.7
Fluoranthene		202	5.0×10^{-6}	6.5×10^{-6}	2.6×10^{-1}	7.9×10^{-4}	3.8×10^{4}	1.2×10^{3}	NC	5.9×10^{-3}	<4.0

Table 1-1. PAH Physical-Chemical and Toxicological Data[1]
(continued)

PAH compound	Structure	MW	Vapor P (torr)	H ($atm\text{-}m^3\text{-}mol^{-1}$)	Solubility (mg/l)	K_{ow} (unitless)	K_{oc}[2] (unitless)	BCF[3] (unitless)	UCR[4] $(mg/kg/day)^{-1}$	ADI[5] (mg/kg/day)	Acute AQ[6] toxicity (mg/l)
Fluorene		116	1.3×10^{-2}	1.1×10^{-3}	1.69	8.1×10^{-3}	3.9×10^{3}	2.8×10^{2}	NC	5.9×10^{-3}	<1.7
Indeno(1,2,3-cd)pyrene		276	1.0×10^{-10}	7.0×10^{-8}	5.3×10^{-4}	3.2×10^{-6}	1.6×10^{6}	2.7×10^{4}	11.5	C	<1.7
Naphthalene		128	8.7×10^{-2}	4.6×10^{-4}	31.4	1.95×10^{-3}	9.4×10^{2}	27.0	NC	0.26	<2.3
Phenanthrene		178	9.6×10^{-4}	2.3×10^{-4}	1.0	2.8×10^{-4}	1.4×10^{4}	4.9×10^{2}	NC	5.9×10^{-3}	<1.7
Pyrene		202	2.5×10^{-6}	5.1×10^{-6}	1.3×10^{-1}	8.0×10^{-4}	3.8×10^{4}	1.1×10^{3}	NC	5.9×10^{-3}	<1.7

[1]Physical-chemical data were taken primarily from USEPA (1981a).

[2]K_{oc} generally was calculated using K_{ow} and the equation $\log K_{oc} = 1.00 \log K_{ow} - 0.37$ (Hasset et al. 1980 in USEPA 1981a).

[3]BCF was calculated using K_{ow} and the equations $\log BCF^1 = 0.85 \log K_{oc} - 0.70$ and $BCF = BCF^1 (3.0/7.6)$ (USEPA 1980a).

[4]$11.5\ (mg/kg/day)^{-1}$ is the UCR for benzo[a]pyrene (Fed. Reg. 1980 in Salee 1984). All known or highly suspected carcinogens are all assumed to have the same UCR as benzo[a]pyrene because other values of UCR are not available.

[5]5.9×10^{-3} mg/l is the ADI for fluoranthene (USEPA 1984 in Salee 1984). All known or generally considered non-carcinogens, except naphthalene, are assumed to have the same ADI as fluoranthene because other values of ADI (except for naphthalene) are not available.

[6]There are no acute or chronic aquatic toxicity criteria established for any of the PAHs. Concentrations of acenaphthene, fluoranthene, and naphthalene as low as 1.7 mg/l have been reported to cause acute toxic effects in aquatic animals (USEPA 1980b,c,d).

Values of toxicological parameters for most of the PAHs have not yet been determined. Benzo[a]pyrene is reported to have a unit carcinogenic risk factor (UCR) of 11.5 kg/day/mg (Federal Register 1980 in Salee 1984) but no other UCRs for PAHs could be found in the literature. The only available acceptable daily intakes (ADI) are for fluoranthene (5.9×10^{-3} mg/kg/day) and naphthalene (0.26 mg/kg/day) (USEPA 1984 in Salee 1984). No acute or chronic water quality criteria for the protection of aquatic life have been established for any of the PAHs (USEPA 1980a, b, c, and d). Concentrations of acenaphthene, napthalene, and fluoranthene as low as 1.7 mg/L, 2.3 mg/L, and 4.0 mg/L, respectively have been reported to cause acute toxic effects in some aquatic organisms (USEPA 1980b, d, c).

For the purpose of scoring and ranking regions, subregions, sediment sampling sites and PAHs according to potential risks to human and aquatic life, the following assumptions were made in assigning values of UCRs, ADIs and acute aquatic criteria to the PAHs in Table 1-1:

1. PAHs were assumed to be either carcinogens or non-carcinogens based upon available evidence for carcinogenicity (SAIC 1984).

2. "Carcinogenic" PAHs were all assigned UCR values of 11.5 kg/day/mg which is the reported UCR for benzo[a]pyrene (Federal Register 1980 in Salee 1984).

3. "Non-carcinogenic" PAHs other than naphthalene were assigned ADI values of 5.9×10^{-3} mg/kg/day which is the reported ADI value for fluoranthene. Naphthalene has a reported ADI value of 0.26 mg/kg/day (USEPA 1984 in Salee 1984).

4. No acute aquatic life criteria have been established for any of the PAHs. However, PAHs other than fluorathene and naphthalene were assumed to have acute aquatic life criteria of 1.7 mg/L based on the no-effect level for acenaphthene (USEPA 1980b). Acute aquatic life criteria for naphthalene and fluoranthene were assumed to be 2.3 mg/L and 4.0 mg/L, respectively, based on no-effect levels (USEPA 1980d,c). The scoring and ranking methodology is described in Chapter 3.

Sufficient data are presently unavailable with which to formulate precise estimates of risks to human health and aquatic life from PAHs in sediments. Although estimates of such risks are developed in this report, these estimates are probably several orders of magnitude high than actual risks and are presented for the purposes of ranking and comparing sites (see also Section 3.1).

1.2 SOURCES OF PAHS TO THE AQUATIC ENVIRONMENT

PAHs are formed by the incomplete combustion (pyrolysis) of organic materials; the naturally occurring diagenesis of sedimentary organic material, which produces fossil fuels; and by plant and microbe biosynthesis (Neff 1979, 1985; Woo and Arcos 1981).

Potentially major point sources of PAH pollution to the aquatic environment include effluents from the following (Neff 1979, 1985; Woo and Arcos 1981; Chapter 2 of this report):

- Coke and coke byproduct production facilities
- Iron and steel blast furnace and sintering operations
- Wood preserving facilities using creosote
- Metal finishing rinsing operations
- Aluminum-forming facilities
- Facilities using high temperature furnances such as metal smelters and foundries (particularly those using a Soderberg electrode which consists of anthracite, coketar pitch and anthracene)
- Oil refineries
- Organic chemical manufacturers which use solid or liquid hydrocarbon feedstock.

Potentially major non-point sources of PAH pollution to the aquatic environment include the following (Neff 1979, 1985; Woo and Arcos 1981; Chapter 2 of this report):

- Petroleum and petroleum product spills and leaks during transport or storage
- Surface runoff from roads and contaminated soil
- Leaching from hazardous waste sites, including dredged sediment piles
- Discharges from boat motors and ship engines
- Atmospheric deposition of PAH contaminated particulates

Potentially major sources of PAH contaminated particulates to the atmosphere include emissions from: coal and oil burning electric power plants, coal and wood burning heat furnances, refuse burning, coke and coke byproducts production, oil refineries, and internal combustion engines (Woo and Arcos 1981).

A further discussion of point and non-point sources of PAH pollution to the aquatic environment is given in Chapter 2.

1.3 ENVIRONMENTAL FATE AND TRANSPORT OF PAHS IN AQUATIC SYSTEMS

The following discussion on environmental fate and transport of PAHs was taken primarily from the reviews of USEPA (1979a) and Neff (1979, 1985) unless otherwise referenced. A brief overview is followed by more specific discussions of sediment/water partitioning; volatilization; hydrolysis and chemical oxidation; photolysis; biodegradation; and bioaccumulation, metabolism and excretion by aquatic organisms.

In general, much more of the total PAH in aquatic systems is bound to sediment or suspended solids than is dissolved in the water column. Sediment/water partitionings of PAHs generally increase with decreasing aqueous solubility and increasing molecular weight, number of rings, octanol/water partition coefficient and sediment organic content. The primary transport mechanism for PAHs in aquatic systems appears to be by transport of PAH contaminated suspended solids. Volatilization under turbulent conditions and from shallow water bodies may remove substantial amounts of the lower molecular weight and ring number PAHs dissolved in the water column within several days. However, for most PAHs under most conditions, volatilization over periods well exceeding 1 month may be required to remove substantial proportions of the dissolved PAH from water (Southworth 1979). Volatilization half-lives increase (e.g., rates decrease) with decreasing system turbulence and increasing PAH molecular weight, number of rings and Henry's constant.

PAHs are resistant to hydrolysis but some PAHs, such as benzo[a]anthracene and benz[a]pyrene, may be suspectible to chemical oxidation by photochemically produced reactive species. Photolysis half-lives for many PAHs are less than 24 hours under optimal conditions but increase substantially with increasing water depth, water turbidity, humic content and sorption to sedi-

ments. Some PAHs appear to be susceptible to at least aerobic biodegradation but rates of biodegradation greatly decrease with increasing molecular weights and number of rings. Although predicted bioconcentration factors based on the octanol/ water partition coefficient are moderately high for many PAHs (e.g., $>10^3$ for PAHs with 4 or more rings), the actual values for fish may generally be substantially lower due to metabolism of the PAHs (USEPA 1980a).

1.3.1 Sediment/Water Partitioning of PAHs

Sediment/water equilibrium partition coefficients (K_D) for the adsorption of different PAHs to the same sediment type generally increase with decreasing aqueous solubility and increasing molecular weight, number of rings and octanol/water partition coefficient. However, the concentration of various PAHs in sediments do not generally follow any particular order with respect to molecular weight and numbers of rings. One of the reasons is that the concentration of any given PAH in sediment at equilibrium will be equal to the product of the sediment/water partition coefficient times the concentration of the given PAH in water. The sediment/water partition coefficient generally increases with increasing molecular weight and number of rings, however the concentrations of the higher molecular weight PAHs in water are frequently less than those of the lower molecular weight PAHs due to lower aqueous solubilities and/or loading rates. Another reason is that aquatic systems are frequently far from either equilibrium or steady state conditions.

Sediment/water equilibrium partition coefficients for the adsorption of the same PAH to different sediments will generally increase with increasing organic content of the sediment. However, the organic normalized equilibrium partition coefficient (K_{OC}), which is the ratio of the sediment/water partition coefficient to the organic fraction of the sediment, should remain relatively constant for the same PAH absorbed to different sediments (USEPA 1981a, Lyman et al. 1982). Estimates of K_{OC} can be obtained from K_{OW} values using equation (1-1) as discussed in Section 1.1. Estimates of K_{OC} values increase with increasing molecular weight and number of rings, ranging from 9.4×10^2 for naphthalene to 5.5×10^5 for benzo[a]pyrene (Table 1-1). The K_{OC} values listed in Table 1-1 correspond to estimated K_D values, ranging from 47 for naphthalene to 2.8×10^4 for benzo[a]pyrene (5 rings), in sediments containing 5% organic carbon.

The relatively low aqueous solubilities and relatively high K_{OC} values for most PAHs indicate that a much larger proportion of the total PAH in an aquatic system will generally be absorbed to sediments and suspended solids than will be in solution in the water column. This is particularly true for PAHs with 4 or more rings and for sediments with high organic carbon. Both laboratory and field data have generally shown that the ratios of total PAH absorbed to sediments and suspended solids, to total PAH dissolved in the water column, are generally greater than 10^3 for PAHs with 3 or more rings.

The relatively high sediment/water partitioning for most PAHs suggest that the primary mechanism of transport for those PAHs in aquatic systems is by transport of PAH contaminated sediment. This had led to speculation that PAH concentrations in sediments may be higher in areas of suspended solids deposition (e.g. areas of reduced current and turbulence) than in other areas of aquatic systems (Eadie et al. 1982).

1.3.2 Volatilization

Volatilization half-lives for PAHs in water generally increase with increasing molecular weights and number of rings due to the corresponding decrease in Henry's constant and diffusion constants in air and water (Southworth 1979). Volatilization half-lives for a given PAH from water decrease with increasing turbulence in either the air or water. For example, based on laboratory determinations of mass transfer coefficients for PAH's in air and water, Southworth (1979) predicted volatilization half-lives for various PAHs as a function of river current and wind speed. Estimated volatilization half-lives for naphthalene, anthracene, benzo[a]anthracene and benzo[a]pyrene decreased from approximately 80, 300, 2200 and 7000 hours, respectively, for a river current of 0.1 m/sec and wind speed of 0.75 m/sec to approximately 3.2, 16, 150 and 430 hours, respectively, at a river current of 1 m/sec and wind speed of 4 m/sec. Volatilization half-lives from rivers with depths typically >1 m would be proportionally longer.

With the exception of 2 and 3 ring compounds under turbulent conditions and in shallow water, it appears that PAH volatilization half-lives are too long for volatilization alone to prevent substantial proportions of dissolved PAH from being transported many km downstream from the point of input (South-

worth 1979). However, over time periods exceeding one month, volatilization alone could possibly account for a substantial decrease in dissolved PAH concentrations, at least for PAHs with no more than 5 rings.

1.3.3 Hydrolysis and Chemical Oxidation

PAHs do not readily undergo nucleophillic aromatic substitution and therefore are not predicted or known to undergo hydrolysis in natural waters.

Major naturally occurring oxidizing species in natural waters are thought to include the peroxy free radical $RO_2\cdot$ and singlet oxygen $^1O_2$ which are generated photochemically (USEPA 1981a). The half-life for the chemical oxidation of a PAH by both $RO_2\cdot$ and $^1O_2$ can be estimated from the following equation:

$$t_{1/2} = \frac{0.693}{k_{^1O_2}\,[^1O_2] + k_{RO_2\cdot}\,[RO_2\cdot]} \qquad (1\text{-}3)$$

where

$k_{^1O_2}$ = second order rate constant for the chemical oxidation of the given PAH by $^1O_2$

$[^1O_2]$ = molar concentration of singlet oxygen

k_{RO_2} = second order rate constant for the oxidation of the given PAH by RO_2

$[RO_2\cdot]$ = molar concentration of peroxy free radical.

Estimated second order rate constants for the chemical oxidation of numerous PAHs by $^1O_2$ and $RO_2\cdot$ are listed in USEPA (1981a). Estimates of PAH half-lives due to chemical oxidation by $^1O_2$ and $RO_2\cdot$ can be obtained by substituting the values for those rate concentrations, along with assumed $^1O_2$ and $RO_2\cdot$ concentrations of 1×10^{-12}M and 1×10^{-9}M, respectively, in sun-exposed natural waters, into equation (1-3). Using this method, estimates of PAH half-lives due to chemical oxidation by $^1O_2$ and $RO_2\cdot$ range from 40 days for anthracene and pyrene and 56 days for benzo[a]anthracene and benzo[a]pyrene to 5.8×10^4 years for naphthalene. Therefore, based on the estimated second order rate constants listed in USEPA (1981a), chemical oxidation by $^1O_2$ and $RO_2\cdot$ does not appear to be a significant removal process for PAHs in natural waters. However, experimentally determined 2nd order rate constants for the

oxidation of benzo[a]anthracene and benzo[a]pyrene by RO_2. (Smith et al. 1978) are much larger than the estimated values listed in USEPA (1981a). If the experimentally determined values of the second order rate constants for the oxidation of benzo[a]anthracene and benzo[a]pyrene are substituted into equation (1-3), estimated half-lives of 3.8 hours and 4.7 days are obtained for benzo[a]anthracene and benzo[a]pyrene, respectively. Therefore, it appears that at least some PAHs may be susceptible to oxidation in natural water by photochemically produced oxidants, whereas others (such as naphthalene and phenanthrene) are not. PAHs appear to be readily oxidized to quinones by both chlorine and ozone during the treatment of drinking water (USEPA 1979a). Half-lives for many PAHs at exposure concentrations of ozone and chlorine typically used in water treatment are generally less than one hour.

1.3.4 Photolysis

Most PAHs appear to be susceptible to rapid rates of direct aqueous photolysis under optimal conditions (e.g. dissolved in shallow clean water, exposed to midday sunlight or to intense sun lamps). For example, Zepp and Schlotzhauer 1979 (in Lyman et al. 1982) reported aqueous photolysis half-lives for seven PAHs with 4 or less rings ranging from 0.68 hours for pyrene to 70 hours for naphthalene. Smith et al. (1978) reported aqueous photolysis half-lives for benzo[a]anathracene and benzo[a]pyrene of 3.3 hours and 1 hour, respectively. Other groups have reported similar results under optimal conditions. However, rates of aqueous photolysis decrease with increasing water depth, turbidity, humic concentrations, and PAH adsorption to sediments. Therefore, under actual field conditions, aqueous photolysis half-lives may be much longer than those reported under optimal conditions.

1.3.5 Biodegradation of PAHs by Microorganisms

Biodegradation rates are generally much greater for PAHs with no more than 3 rings than for PAHs with 4 or more rings. In addition, biodegradation rates are generally greater in continuously contaminated water or sediment than in previously uncontaminated water or sediment. For example, biodegradation half-lives for naphthalene, anthracene, benzo[a]anthracene and benzo[a]-pyrene in hydrocarbon contaminated sediment were reported to be 5,280, 7.0 x

10^3 and 21 x 10^3 hours, respectively (Herlis and Schwall 1978 in USEPA 1979a). Biodegradation half-lives for the same PAHs in previously uncontaminated sediment were 10 to 400 times longer. The failure of Smith et al. (1978) to develop any bacteria cultures capable of degrading benzo[a]anthracene or benzo[a]pyrene may indicate that long acclimation times are required before bacteria are able to degrade PAHs.

Biodegradation rates are reported to be much greater under aerobic conditions than anaerobic conditions (Neff 1985) and may be generally greater for PAHs absorbed to sediment than for PAHs dissolved in the water column (USEPA 1979a).

1.3.6 PAH Bioaccumulation, Metabolism, and Excretion by Aquatic Organisms

Based on somewhat limited data, the rate at which aquatic organisms can metabolize and/or excrete PAHs appears to generally follow the order: fish > crustaceans >> bivalve mollusks >>> microalgae (Neff 1985). The ability of the higher aquatic organisms to metabolize and/or excrete PAHs rapidly is reflected in the relatively short times ($\leq$ 24 hours in most cases) required to reach steady state PAH concentration in tissues of fish and the crustacean Daphnia continuously exposed to PAHs (Herbes and Risi 1978; Southworth et al. 1978; Lee et al. 1972b; Anderson et al. 1974b all in Neff 1985). Most of the PAH in fish was removed by metabolism whereas most of the PAH in Daphnia was removed through excretion.

Bivalve mollusks do not readily metabolize PAHs nor do they generally excrete PAHs as rapidly as Daphnia Therefore, they may bioaccumulate PAHs to a greater extent than most fish or crustaceans. Nevertheless, PAH contaminated mollusks appear to be able to remove most of the PAH in their tissue (presumably primarily by excretion) within a few days to several weeks after being placed in uncontaminated water (Neff 1985). Microalgae reportedly bioaccumulate and retain PAHs to a greater extent than the higher organisms due possibly to irreversible binding of the PAHs to the cell wall (Neff 1985).

Steady state bioconcentration factors for the uptake of different PAHs by aquatic organisms from water generally increase with increasing molecular weight, number of rings, and octanol/water partition coefficient. However,

the concentrations of various PAHs in the tissues of aquatic organisms do not generally follow any order with respect to molecular weight or number of rings. For example, phenanthrene (3 rings) had a much higher concentration in the tissues of fish taken from the Black River than did other PAHs with 2, 3, 4 or more rings (Bauman et al. 1982). One of the reasons is that the steady state concentration of any given PAH in the tissues of an aquatic organism will be equal to the product of the bioconcentration factor times the concentration of the PAH in water. Although the bioconcentration factor generally increases with increasing molecular weight, the concentration of the higher molecular weight PAHs in water are frequently less than those of the lower molecular weight PAHs due to lower aqueous solubility and/or loading rates. Other reasons may include non-steady state conditions and contributions of PAHs from other media such as food and sediment.

For organisms such as Daphnia or bivalve mollusks, which appear to remove PAH's primarily through excretion, the increase in bioconcentration factors with increasing octanol/water partition coefficient can be explained by the fact that the octanol/water partition coefficient is generally a good indicator of a chemical's partitioning between the lipid containing tissues of aquatic organisms and water. In the case of fish species which appear to remove PAH in their tissues primarily by metabolism, the increase in bioconcentration factors with increasing molecular weight and number of rings may be due to a corresponding decrease in the ability of the organism to metabolize PAHs with high molecular weights and number of rings.

The bioconcentration factors listed in Table 1-1 for PAHs and edible fish and shellfish with a mean lipid content of 3% were taken primarily from USEPA (1980a). They were computed by substituting values of the octanol/water partition coefficient into equation (1-2) which was derived from data on the bioconcentration of various types of organics by fish. However, the organics upon which the equation is based are not PAHs and are probably generally more resistant to metabolism by fish. Therefore, the estimated bioconcentration factors listed in Table 1-1 may be somewhat high for species of fish or shellfish which readily metabolize PAHs and for PAHs with 3 or less rings which are readily metabolized (USEPA 1980a). However, they may be reasonably accurate for PAHs with 5 or more rings which are not readily metabolized or for orga-

nisms which do not readily metabolize PAHs. For example, the octanol/water partition coefficient is a good predictor of bioconcentration in Daphnia which do not readily metabolize PAHs (Southworth et al. 1978 in USEPA 1979a).

Based on limited data, it appears that bioconcentration factors defined by the ratio of PAH concentrations in organisms to those in food or sediment are substantially lower than those based on the ratio of PAH concentration in organisms to those in water (Neff 1985). However, since PAH concentrations in sediment are generally much greater than those in water, a substantial accumulation of PAH in organisms from the sediment may sometimes occur. The extent to, and mechanism by which PAHs are accumulated in an aquatic organism, will primarily depend on whether the organism is benthic. Benthic organisms may accumulate PAHs from sediment by direct physical contact during feeding, through consumption of other PAH contaminated benthic organisms, or through respiration in interstitial water or water near the sediments which have been contaminated by deadsorption from the sediment (Black 1983). Generally, non-benthic organisms will probably accumulate PAHs primarily during respiration in contaminated water. However, in most cases, the concentrations of PAH in the general water column will be far less than PAH concentrations in interstitial water or water near the sediments.

1.4 PAH TOXICITY TO HUMANS AND TERRESTRIAL TEST ANIMALS

Various PAHs have long been suspected of being carcinogenic to humans based on their relatively high concentrations in media known to be carcinogenic such as chimney soot, coal tar, and cigarette smoke (Woo and Arcos, 1981). Although numerous studies have shown that various PAHs can induce malignant tumors in laboratory animals (primarily mice), most of the studies have involved dermal exposure or subcutaneous injection (LaVore and Hecht 1981; Woo and Arcos 1981). The oral administration of a limited number of PAHs have been shown to induce stomach tumors in laboratory animals but the concentrations of PAH required is generally quite high (>30 ppm) (Woo and Arcos 1981). Furthermore, in a review of PAH carcinogenicity, Grice et al. (1981) indicated that only benzo[a]anthracene, benzo[a]pyrene, dibenzo[a,h]-anthracene, 7,12-dimethylbenzo[a]anthracene, and methylcholanthrene had been conclusively shown to induce malignant tumors after oral administration. The

most frequently encountered effects of the oral administration of those PAHs to laboratory animals were leukemia, forestomach tumors, hepatoma, pulmonary adenoma, and mammary tumors (Grice et al. 1981). It is currently believed that mixed function oxidases catalyze the production of carcinogenic metabolic products of PAH, which are believed to initiate carcinogenesis through first covalently binding to DNA (Woo and Arcos 1981). The only PAH for which a unit carcinogenic risk (UCR) has been established is benzo[a]pyrene (11.5 kg/day/mg) (Federal Register 1980 in Salee 1984).

There is very little data concerning the non-carcinogenic effects of PAHs. Several PAHs are known or suspected skin and/or mucous membrane irritants to humans and/or laboratory animals (Clements 1985). PAH effects on skin include hyperplasia, hyperkeratosis, ulceration, and chronic dermatitis (Clements 1985). Sub-chronic oral exposure of rats and mice to acenaphthene causes loss of body weight, blood alterations, and mild morphological damage to the kidneys and liver (Clements 1985). Oral LD 50's of acenaphthene for rats and mice are 10 g/kg and 2.18 g/kg respectively (Clements 1985). Naphthalene is a teratogen to mice causing retarded cranial ossification and heart development. Inhalation of naphthalene can cause headaches, nausea and kidney damage in humans and laboratory test animals. Inhalation or ingestion of naphthalene can cause optical neuritis, injuries to the cornea and opacities of the lens (Clements 1985). Oral LD 50s for rats and mice are 1250 mg/kg and 580 mg/kg respectively (Clements 1985). Acceptable daily intakes have only been established for fluoranthene (5.9×10^{-3} mg/kg/day) and naphthalene (0.26 mg/kg/day) (USEPA 1984 in Salee 1984).

1.5 TOXICITY TO FRESHWATER AQUATIC ORGANISMS

The data on the acute toxicity of PAHs to freshwater aquatic organisms is extremely limited. Acenaphthene is reported to have a 48 hour EC 50 for Daphnia magna of 41,200 mg/L, a 96 hour LC 50 for bluegills of 1,700 mg/L and 96 hour EC 50s of 530 mg/L (chlorophyll a) and 520 mg/L (cell numbers) for the freshwater algae Selenastrium capricornutum (USEPA 1980b). Fluorathene is reported to have a 48 hour EC 50 of 325,000 ug/L for Daphnia magna, a 96 hour LC 50 of 3,980 ug/L for bluegills, and 96 hour EC 50s of 54,600 ug/L (chlorophyll a) and 54,400 ug/L (cell numbers) for the freshwater algae Selenastrium

capricornutum (USEPA 1980c). Napthalene is reported to have a 48 hour EC 50 of 8,579 ug/L for Daphnia magna, a flow-through 96 hour LC 50 of 2,300 ug/L for rainbow trout, flow-through 96 hour LC 50s of 4,900 ug/L and 8,900 ug/L for fathead minnows at 14 C and 24 C, respectively, and an EC 50 (cell numbers) of 33,000 ug/L for the freshwater algae Chlorella vulgaris (USEPA 1980d). Brown et al. (1975 in USEPA 1980a) reported that continuous exposure of bluegills to a benzo[a]anthracene concentration of 100 ug/L caused a 87% mortality rate. Hutchinson et al. (1980) in Neff (1985) reported 3 hour EC 50s (rate of photosynthetic CO_2 fixation) of 9,600 ug/L, 1,700 ug/L, 4,500 ug/L, 900 ug/L, 200 ug/L and 200 ug/L for the freshwater microalgae Chlamydomonas angulosa exposed to naphthalene, 1-methylnapthalene, 2-methylnapthalene, phenanthrene, anthracene and pyrene, respectively. Corresponding 3 hour EC50 values for the freshwater microalgae Chlorella vulgaris were 19,200 ug/L, 5,100 ug/L, 900 ug/L, 1,200 ug/L, 500 ug/L and 300 ug/L, respectively.

Data on the acute toxicity of PAHs to saltwater organisms is a little more extensive and shows that such organisms are generally more sensitive to PAHs than similar types of freshwater organisms. Data on the acute toxicity of PAHs to both freshwater and saltwater organisms indicates that the acute toxicity of PAHs with 4 or less rings generally increases with increasing molecular weight and number of rings (Neff 1985). However, PAHs with 5 or more rings are generally not acutely toxic, perhaps at least partly due to their low aqueous solubility (Neff 1985).

A chronic value of 620 ug/L has been reported for fathead minnows exposed to naphthalene in an embryo-larval test (USEPA 1980d). No other chronic values for freshwater species were reported in the various PAH water quality criteria documents (USEPA 1980a, b, c, d). Neff (1985) summarizes reported effects of 2 and 3 ring PAHs on primarily saltwater organisms exposed to sublethal concentrations. Observed effects included: retarded larval development for mud crabs, reduction in offspring for marine copepods, impaired movement in blue crabs, lesion development in oysters, histological changes in the livers of mullets and gill hyperplasia and gill filament hemorrhages in mummihog fish.

There is some circumstantial evidence that PAHs in heavily contaminated aquatic systems may induce the formation of malignant and/or benign epidermal

and/or liver tumors in bottom feeding fish (Brown et al. 1973 in Bauman et al. 1982; Bauman et al. 1982; Black 1983). Brown et al. (1973) in Bauman et al. (1982) reported that 12% of the brown bullheads collected from the Fox River in Illinois (which is contaminated with PAHs) had tumors compared to only 2% for brown bullheads collected from the relatively unpolluted Lake of the Woods in Ontario. Bauman et al. (1982) reported that 1.2% of the 2 year old and 33% of the greater than 3 year old brown bullheads collected from the heavily PAH contaminated Black River at Lorain, Ohio, had liver tumors compared to none of the 2 year or 3 year and older brown bullheads collected from the unpolluted Buckeye Lake. In addition, many of the fish collected from the Black River had lip and dermal tumors. Black (1983) reported that 8.9% of 305 freshwater drum collected from heavily PAH polluted regions of the Niagara River System had dermal tumors compared to 2.3% of 89 freshwater drum collected from relatively unpolluted regions of Lake Erie. Black et al. (1985) reported that 17% of 28 large adult brown bullheads collected in the Buffalo River had dermal or liver tumors.

Although the field studies described above indicate that PAHs in heavily polluted aquatic systems may induce the formation of tumors in fish, the evidence is only circumstantial since other toxic and potentially carcinogenic organics and heavy metals are also present. Unfortunately, there is very little direct evidence for or against the postulate that PAHs can induce tumor formation in fish. The limited evidence available suggests that PAHs do not readily induce tumor formation in fish, but may do so in some fish if they are exposed to high concentrations of PAH over extended time periods. Skin tumors were induced in two out of three species of fish by dermal exposure to 0.5 mg of both methylcholanthrene and benzo[a]pyrene twice a week for 3-6 months. However, 10 injections of 1 mg/injection benzo[a]pyrene failed to induce skin tumors over a 4 month period (Ermer 1970 in Neff 1985). Also, neither total injections of 40 mg of 7-12-dimethylbenz[a]anthracene (DMBA) and 20 mg of 3-methylcholanthrene, nor the addition of 120 mg DMBA per 100 g food for 56 weeks induced tumors in either guppies or zebra fish (Pliss and Khudoley 1975 in Bauman et al. 1982). Black et al. (1985) reported that 8 of 22 brown bullheads developed dermal papillomas (benign skin tumors) during dermal exposure to a 5% organic extract of PAH contaminated Buffalo River sediment once a week over an 18 month period.

2. SOURCES OF PAH POLLUTION TO AQUATIC SYSTEMS

A brief overview of sources of PAHs to aquatic systems was presented in Section 1.2. In this chapter, a more detailed discussion is presented. Chapter 2 is divided into the following nine sections: General Point Sources (2.1), the Wood Preserving Industry (2.2), the Iron and Steel Industry (2.3), the Metal Finishing Industry (2.4), the Aluminum Forming Industry (2.5), the Metal Foundries Industry (2.6), the Non-ferrous Metals Manufacturing Industry (2.7), Petroleum Refineries (2.8), and Non-point Sources (2.9).

2.1 GENERAL POINT SOURCES

Table 2-1 presents PAH data (in ug/L) for raw and treated wastewater for 17 of the 34 industrial categories (based on SIC codes) currently under regulatory control or consideration by the EPA (USEPA 1980e). The PAH data for the other 17 industrial categories were either negligible or not available (USEPA 1980e). Table 2-1 lists the following information for each industrial category and type of wastewater (USEPA 1980e):

- The number of PAHs analyzed for in the effluent (#C)
- The number of effluents analyzed (#A)
- The number of effluents in which at least 1 PAH was detected (#D)
- The percentage of effluents analyzed in which at least 1 PAH was detected (%D)
- The total (top number) and mean ± std. dev. (bottom numbers) of minimum detected PAH concentrations (Min D)
- The total (top number) and mean ± std. dev. (bottom numbers) of maximum detected PAH concentrations (Max D)

TABLE 2-1. MEAN PAH CONCENTRATIONS (ug/L) IN WASTEWATER FROM VARIOUS INDUSTRIES

	RAW WASTEWATER								TREATED WASTEWATER							
INDUSTRY	#C	#A	#D	%D	MIN. D	MAX. D	MEAN D	MEAN	#C	#A	#D	%D	MIN. D	MAX.D	MEAN D	MEAN
Timber Products Processing Industry (Wood Preserving)	14	168	164	98%	120 8.9+ 8.8	220,000 16,000+ 19,000	39,000 2,800+ 3,500	36,000 2,600+ 3,100	16	131	131	100%	130 8.7+ 2.8	140,000 9,500+ 13,000	17,000 1,100+ 1,500	17,000 1,100+ 1,500
Iron & Steel Manufacturing	8	197	178	80%	47 <5.9+ 5.8	71,000 8,900+ 7,000	<13,000 <1,600+ 1,800	<8,800 <1,100+ 960	9	99	81	82%	<29 <3.6+ 2.6	4,200 526+ 598	<640 <80+ 110	<590 <74+ 110
Metal Finishing[2]	8	197	147	74.6%	ND ND	64,000 8,000+ 19,000	5,400 680+ 1,300	5,400 680+ 1,300	NA	NA	NA	NA	NA NA	NA NA	NA NA	NA NA
Aluminum Forming	8	49	25	51%	170 21+ 12	<3,300 <410+ 460	<1,100 <140+ 140	<850 <110+ 120	9	145	47	32 4%	<20 <2.8+ 3.4	150,000 22,000+ 41,000	21,000 3,000+ 5,600	8,000 1,000+ 2,100
Foundries	15	795	157	20%	<62 <5.2+ 2.8	<30,000 <2,500+ 4,900	<5,600 <470+ 970	<1,200 <78+ 110	15	795	146	18%	<75+ <7.5+ 2.7	28,000 <2,500+ 3,400	<2,400 <220+ 290	<570 <38+ 58
Non-Ferrous Metals Manufacturing[2]	13	760	97	12.8%	ND ND	27,000 2,100+ 3,100	930 72+ 110	930 72+ 110	13	607	41	6.8%	ND ND	980 75+ 77	61 4.7+ 4.8	61 4.7+ 4 8
Petroleum Refineries	9	185	25	13.5%	1,500 190+ 300	5,400 670+ 750	3,300 410+ 503	390 49+ 46	9	189	12	6.3%	7.5 1.5+ 2.6	18 3.5+ 2.9	12 2.3+ 2.3	1.0 .011+ 0.14
Organic Chemicals[3] Plastics, and Synthetic Resin	14	165	--	--	-- --	-- --	-- 590+ 590	--	13	--	--	--	-- -- --	-- -- --	-- 17+ 12	-- -- --
Textile Mills[4]	8	481	20	4.2%	2.8 0.71+ 0.76	4,700 670+ 1,600	140 20+ 28	140 20+ 28	8	504	24	5.0%	2.0 0.5+ 0.5	14 1.8+ 1.0	4.6 0.58+ 0.61	4.6 0.58+ 0.61

TABLE 2-1 MEAN PAH CONCENTRATIONS (ug/L) IN WASTEWATER FROM VARIOUS INDUSTRIES (continued)

	RAW WASTEWATER								TREATED WASTEWATER							
INDUSTRY	#C	#A	#D	%D	MIN. D	MAX D	MEAN D	MEAN	#C	#A	#D	%D	MIN D	MAX.D	MEAN D	MEAN
Auto and Other Laundries	6	50	20		2 4 0.60± 0 74	960 240± 270	<200 <50± 58	<84 <14± 21	6	15	14	93%	19 3.7± 5 8	160 31± 32	60 12± 8 9	59 9 9± 9 3
Battery Manufacturing[2]	3	39	19	13%	ND ND	<69 <23± 12	<30 <10± 0	<30 <10± 0	1	16	13	81%	ND ND	15 15	2 0 2 0	2 0 2.0
Leather Tanning and Finishing	8	144	25	17%	<56 <7 0± 3.9	<370 <46± 58	<200 <25± 27	<69 <8.6± 14	2	12	8	67%	2.8 1.4± 0	<20 <10± 0.0	<12 <6 2 +0.0	<8.7 <4.1± 0.0
Paint and Ink Formulation	8	12	7	58%	<45 <9.0± 2.2	<55 <11± 2.7	<50 <10± 1.9	<28 <5.5± 4.8	7	7	4	67%	55 11± 1.0	55 11± 1 0	55 11± 1 0	42 8.4 +4.5
Photographic Equipment/Supplies	15	137	23	17%	24 3.4± 1.8	730 81± 150	290 32± 59	48 3.2± 7.1	3	29	8	28%	1.0 0.50± 0.0	11 3.7± 2 3	6.6 2.2± 1.2	1.8 0.6± 0.2
Pulp and Paperboard Mills[2]	3	54	5	9.3%	ND ND	18 6 0± 1.0	4.5 1.5± 0.5	4.5 1.5± 0.5	2	19	1	5.3%	ND ND	1.0 1.0	0.33 0 33	0.32 0.16
Coil Coating	14	1092	91	8.3%	<120 <10± 0	<850 <71± 105	<140 <12± 8.2	<20 <1 4± 2.9	15	205	52	25%	<10089 <10± 0	55 <8.9± 4.5	10 5.5± 4.7	0.78± 1.0
Coal Mining	15	723	47	6.5%	26 2.9± 3.6	270 30± 43	87 9.7± 6.3	11 0.88± 0.97	15	787	18	2.3%	47 5.9± 4.4	64 7.1± 5.1	56 6 2± 4.7	2.6 0.22 ±0.27

1. The first number listed under MinD, Max D, Mean D and Mean is total PAH over all PAH. The second and third numbers listed under each heading is average PAH concentration ± standard deviation over all PAHs.
2. Minimums and means were based on total number of samples, not detections
3. No other information was provided
4. Means computed from medians

ND = Non detecteds
#C = Number of chemicals analyzed for
#A = Number of effluents analyzed
#D = Number of effluents analyzed in which at least 1 chemical was detected
%D = Percent of effluent analyzed in which at least 1 chemical was detected
Min D = Minimum concentration detected
Max D = Maximum concentration detected
Mean D = Mean concentration detected
Mean = Mean concentration included ND = 0.0

Source: USEPA (1980e)

- o The total (top number) and mean ± std. dev. (bottom numbers) of mean detected PAH concentrations (Mean D)

- o The total (top number) and mean ± std. dev. (bottom numbers) of mean PAH concentrations including non-detecteds as zero concentrations.

Table 2-1 does not include naphthalene data.

Table 2-1 arrays the industrial categories on the basis of decreasing mean PAH concentrations in raw wastewater. The mean of the mean PAH concentrations in raw wastewater is much greater for the Timber Products Processing, Iron and Steel Manufacturing, and Metal Finishing Industries than for the other industrial categories. The mean of the mean PAH concentrations in treated wastewater is much greater for the Timber Products Industry than for the Iron and Steel Industry and all other industrial categories listed in Table 2-1 except the Aluminum Forming Industry. The data for the Aluminum Forming Industry appears to be anamalous since the mean of the mean PAH concentrations is much greater in treated than in raw wastewater. The data for the Aluminum Forming Industry will be discussed in greater detail in Section 2.4.

Descriptions of the first 7 industrial categories listed in Table 2-1 are given in Sections 2.2-2.8 and more detailed effluent data are contained in Appendix A.

2.2 TIMBER PRODUCTS PROCESSING INDUSTRY (WOOD PRESERVING)

The Timber Products Processing Industry is divided into 15 subcategories, of which 6 are under regulatory control: Wood Preserving (Waterborne or Non-pressure), Wood Preserving (Steam), Wood Preserving (Boulton), Insulation Board Manufacturing, Hardboard Manufacturing (SIS) and Hardboard Manufacturing (S2S) (EPA 1980f, 1983a, 1981b). PAH data was presented in the Treatability Manual only for the Steam and Boulton Wood Preserving subcategories (USEPA 1980f, 1983a).

As of 1981, the wood preserving subcategories were under BPT limitations which impose a no-discharge (to natural water) of wastewater pollutant limitation on the Wood Preserving (Waterborne or Non-pressure) and Wood Preserving (Boulton) subcategories, and establish numerical limits on the discharge to natural water of COD, total phenol, oil and grease, and pH for the Wood Preserving (Steam) sub-category (USEPA 1981b). As of 1981, EPA was promulgating new source performance standards (NSPS) that require no discharge of wastewater pollutants. In addition, existing EPA pre-treatment standards for existing sources (PSES) include a 100 mg/L limitation on oil and grease for wood preserving facilities that discharge to POTWs (USEPA 1981b). It was believed that the PSES oil and grease limitation was sufficient to prevent substantial quantities of PAHs from passing though POTWS (USEPA 1981b).

According to the American Wood Preservers Association, 43 wood preserving facilities existed within the EPA Region V States as of 1978 (AWPA 1978 in USEPA 1981b). Of these 43 facilities, 12 are in Wisconsin, 8 are in Illinois, 7 each are in Ohio and Minnesota, 5 are in Indiana, and 4 are in Michigan. However, the PCS data base, which is a compilation of facilities holding NPDES permits, lists only 4 wood preserving facilities in the States comprising Region V (Appendix A, Table 1). Therefore, the other 39 wood preserving facilities in Region V appear to be currently exempt from NPDES permitting due to discharge to POTWs and/or no direct discharge to natural bodies of water. However, those facilities discharging to POTWs should also be under PSES restrictions.

The wood preserving process consists of (USEPA 1981f; 1983a; 1981b):

- Conditioning the wood to reduce its moisture content and increase its permeability to preservatives, and
- Impregnating the wood with preservatives.

Methods of conditioning the wood include:

- Drying at ambient temperatures in open air
- Kiln drying
- Exposing the wood to organic vapors which transfer their heat of vaporation to the wood upon condensation thereby vaporizing water in the wood (vapor drying process)

- Steaming the wood at elevated pressure in a retort followed by application of a vacuum (steaming process)

- Heating the wood in a preservative bath under reduced pressure in a retort (Boulton process).

After the wood is pre-conditioned, it is impregnated with preservatives using either pressure processes, which force the preservative into the wood in closed systems under pressure, or non-pressure processes, which generally involve simply immersing the the wood in open tanks containing a preservative solution, whether heated or at ambient temperature (USEPA 1981b). Wood preserving facilities which use the vapor drying, steaming, or Boulton pre-conditioning processes generally use pressure processes to impregnate the wood since the pre-conditioning and the impregnation can take place in the same closed system (USEPA 1981b). Wood preserving facilities which use non-pressure impregnation processes generally use open air or kiln drying for pre-conditioning.

Preservatives used in wood preserving facilities include creosote, creosote-coal tar mixtures, cresote-petroleum mixtures, pentachlorophenol and water soluble inorganic salts of copper, chromium, and arsenic. Creosote and creosote mixtures together accounted for over 50% of preserved wood production in 1978 (USEPA 1981b).

EPA classifies wood preserving facilities according to a combination of factors including type of pre-conditioning impregnation process and preservatives used. The subcategories are as follows (USEPA 1981b):

- Wood Preserving (Waterborne or Non-pressure) - includes all facilities using non-pressure impregnation processes regardless of preservatives used and all facilities using only water soluble preservatives, such as inorganic salts, regardless of the type of preconditioning or impregnation used

- Wood Preserving (Steam) - includes all facilities using steam pre-conditioning processes and using either only oil type preservatives or a combination of oil type and inorganic salt preservatives

- Wood Preserving (Boulton) - includes all facilities using the Boulton pre-conditioning method.

Wood preserving facilities which use creosote, or creosote mixture preservatives are the ones most likely to have elevated concentrations of PAHs in their effluents since creosote contains high levels of PAHs. However, it is possible that wood preserving facilities which use the vapor drying pre-conditioning process may also have elevated levels of PAHs in their effluents since the most common types of organics used to form the vapors are derivatives of petroleum and coal tar.

Table A-2A of Appendix A lists minimum detected, maximum, mean detected, and mean PAH concentrations in raw and BPT treated wastewater effluents from the Timber Processing Industry (presumably from wood preserving facilities) (USEPA 1980e). The column headings have the same meaning as for Table 2-1. It can be seen in Appendix A, Table A-2A that the PAH concentrations in the treated wastewater remain relatively high, with the mean of the mean PAH concentrations being only slightly less than 50% of that for the raw wastewater.

Table A-2B of Appendix A lists ranges and medians of PAH concentrations in raw and treated wastewater effluents from facilities in the Wood Preserving (Steam) and Wood Preserving (Boulton) Subcategories (USEPA 1980f; 1983a). It can be seen from Appendix A, Table A-2B that maximum and median PAH concentrations in tested effluents from facilities in the Wood Preserving (Steam) Subcategory are much greater than those from facilities in the Wood Preserving (Boulton) Subcategory. The mean wastewater volume generated per day by 8 Boulton facilities was reported to be 21,210 L/day which is almost identical to the 22,450 L/day for 22 steaming facilities (USEPA 1981b). Multiplying the mean of the two mean wastewater volumes generated per day times the total of mean PAH concentrations (excluding naphthalene) in treated wastewater from the Timber Processing Industry (1200 ug/L from Appendix A, Table A-2B) yields an estimated mean total PAH loading (excluding naphthalene) of 26.2 g/day from combined Boulton and Steam facilities. However, as will be shown in Section 2.3, the estimated loading rates for wood preserving facilities are much smaller than those for by-product recovery cokemaking facilities.

2.3 IRON AND STEEL MANUFACTURING INDUSTRY

The Iron and Steel Manufacturing Industry is divided into 25 subcategories, most of which can be grouped into one of 5 basic steps involved in the production of steel (USEPA 1981f):

- Coke production - Approximately 99% of the coke, which is primarily used to reduce iron ore, is produced by the byproduct recovery process (The byproduct recovery process consists of heating bituminous coal in the absence of air to drive off volatile components which converts the coal into coke. The volatile components of the coal are collected and processed into various chemicals and chemical mixtures including crude coal tar, pitch of tar, creosote oils, naphtha solvent, benzene, toluene, xylene, phenol, cresols, and naphthalene.)

- Iron production - The coke produced in the byproduct recovery process is heated with iron ore and limestone in a blast furnace to reduce iron ore to iron.

- Steel production - Iron, scrap steel, limestone, fluorspar, dolomite, iron ores and alloying substances such as ferromanganese are heated in either a basic oxygen furnace, an open hearth furnace or an electric furnace to produce steel.

- Sintering - In the sintering process, waste fines containing high amounts of iron which are collected from the blast and steel production furnaces are combined with coke fines and limestone to agglomerate the iron fines and prepare them for recycle to the blast furnace.

- Steel forming and finishing.

A list of blast furnaces and steel mills with NPDES permits that are within states comprising Region V of EPA is presented in Table A-3A of Appendix A. As of 1980, the Iron and Steel Manufacturing Industry consisted of 1,405 direct, 238 indirect and 220 zero dischargers nationwide (USEPA 1980f).

Table A-3B of Appendix A lists minimum, maximum, mean detected and mean PAH concentrations in raw and treated (BPT) wastewater effluents from the Iron and Steel Manufacturing Industry as a whole (USEPA 1980e). It can be seen from Table A-3B, Appendix A, that the BPT treatment processes are fairly effective in removing PAHs as the mean of the mean PAH concentrations in treated wastewater is less than 10% of that in raw wastewater.

Table A-3C, of Appendix A lists median detected, maximum, mean detected and mean PAH concentrations in raw and treated wastewater effluents from 3 subcategories of the Iron and Steel Manufacturing Industry (USEPA 1980f). PAH concentrations in the effluents from the other subcategories were either negligible (compared to the 3 subcategories listed in Appendix A, Table A-3C) or were not available (USEPA 1980f). As can be seen from Appendix A, Table

A-3C, the total and mean of mean PAH concentrations (excluding naphthalene) in raw wastewater are, suprisingly, slightly greater for the Blast Furnace Subcategory than for the Byproduct Cokemaking Subcategory. However, if the naphthalene concentrations had been included the order would be reversed. Equally suprising is that the total and mean of PAH concentrations (excluding naphthalene) in treated wastewater are greatest for the Sintering Subcategory, which had the lowest PAH values in raw wastewater. Furthermore, the total and mean of mean PAH concentrations in treated wastewater are greater than those for raw wastewater from the sintering subcategory. However, the total and mean of mean PAH concentrations in treated wastewater from both the byproduct recovery cokemaking and the blast furnace iron subcategories are much less than 10% of those in raw wastewater.

The average raw wastewater flow per unit coke production for the byproduct recovery cokemaking subcategory is 0.38 m^3/Mg of coke produced (USEPA 1980f). Daily coke production capacities of 59 cokemaking plants range from approximately 520 Mg/day to 21,000 Mg/day (EPA 1980f) which, when multiplied by the average raw wastewater flow per unit coke production (0.38 m^2/Mg), corresponds to a wastewater flow per day range of 200 m^3/day to 8,000 m^3/day assuming 100% of capacity is utilized. Assuming that the mean total PAH concentration (without naphthalene) in treated wastewater from byproduct recovery cokemaking plants is equal to the total of mean PAH concentrations (without naphthalene) for the byproducts recovery cokemaking subcategory listed in Appendix A, Table A-3C (440 ug/L), the wastewater discharge range of 200 m^3/day (200 x 10^3 L/day) to 8,000 m^3/day (= 8,000 x 10^3 L/day) corresponds to a total PAH (without naphthalene) loading rate range of 88 g/day to 3.5 kg/day, assuming that 100% of capacities are utilized.

The average daily production capacity of 21 wet sintering plants ranges from 450 Mg/day to 11,000 Mg/day (USEPA 1980f). Treated wastewater discharges per unit production average 0.55 m^3/Mg (USEPA 1980f). Multiplying the range of average daily production capacities by the average treated wastewater discharges per unit production yields a range of average treated wastewater discharge per day of 250 m^3/day to 6,100 m^3/day. Multiplying the range of wastewater discharges per day by the total mean PAH concentration (excluding naphthalene) in treated wastewater from the Sintering Subcategory (770 ug/L = 770

mg/M^3 from Appendix A, Table A-3C) gives a range of total PAH loadings per day (excluding naphthalene) of 190 g/day to 4.7 kg/day. That is comparable to the range of total PAH loadings (excluding naphthalene) that were calculated for the Byproduct Recovery Cokemaking Subcategory.

The average daily production of 55 blast furnaces plants is, assuming 260 production days/yr, approximately 7600 Mg/day (USEPA 1980f). Average treated wastewater discharges per unit production range from 0 m^3/Mg (for plants with water recycle) to 27.9 m^3/Mg of iron produced (USEPA 1980f). Multiplying the average daily production by the range of treated wastewater discharges per unit production gives a range of treated wastewater discharges per day of 0 to 2.1 x 10^5 m^3/day. Multiplying the range of wastewater discharges per day by the total mean PAH concentration in treated wastewater from the Blast Furnace Iron Subcategory (130 ug/L = 139 mg/m^3 from Appendix A, Table A-3C) gives an estimated range of total PAH loadings per day (excluding naphthalene) of 0 kg/day to 29.5 kg/day. The minimum is lower and the maximum is greater than those for the Byproduct Recovery Cokemaking and Sintering Subcategories.

The preceeding calculations indicate that PAH loadings from the Sintering and Blast Furnace Iron Subcategories may possibly sometimes be comparable or even greater than those of the Byproduct Recovery Cokemaking Subcategory. However, the PAH concentrations in sediments below cokeoven outfalls are generally much higher than those below other steel industry outfalls or any other outfalls.

2.4 METAL FINISHING INDUSTRY

Nationwide, the Metal Finishing Industry consist of approximately 10,561 direct, 2,909 indirect, and no "zero" dischargers (USEPA 1983a). Depending upon the products involved, as few as 1 and as many as 45 separate unit operations may be involved in the machining, fabrication, and finishing of metal products (USEPA 1983a). Wastewaters from metal finishing facilities are classified by EPA into one of the following 7 categories depending upon the primary component of the wastes: common metals, precious metals, complexed metals, chromium (hexavalent), cyanide, oils, and solvents (USEPA 1983a).

Table A-4A of Appendix A presents minimum, maximum, and mean concentrations of PAHs in raw wastewater from the Metal Finishing Industry as a whole. The total and mean of the mean PAH concentrations in raw wastewater are the third highest among the industrial categories listed in Table 2-1 and are much greater than for the fourth highest industrial category.

Table A-4B of Appendix A presents ranges, medians, and means of PAH concentrations in raw common metals and raw oily wastewaters (USEPA 1983a). No data were presented for other types of wastewater. As would be expected, the maximums, medians, and means of PAHs in the raw oily wastewater are uniformly much greater than those in the raw common metals wastewater. No data were presented for treated oily or common metals wastewater. The contribution of oily wastewater to total wastewater flows from the metal finishing facilities examined ranged from 0.0% to 32% with a mean of 6.4% (EPA 1983a).

2.5 ALUMINUM FORMING INDUSTRY

No description of the Aluminum Forming Industry was available in the references that were used (USEPA 1980e,f; 1981c, 1983a).

Table A-5 of Appendix A presents minimum detected, maximum, mean detected and mean concentrations of PAHs in raw and treated wastewater from the Aluminum Forming Industry (USEPA 1980e). The total and mean of the mean concentrations of PAHs in the treated wastewater are the second highest among the industrial categories listed in Table 2-1. Furthermore, they are approximately an order of magnitude greater in the treated wastewater than in the raw wastewater, due to increases in the anthracene and phenanthrene concentrations. However, the concentrations of the other PAHs are much lower in the treated wastewater than in the raw wastewater.

It is possible that the "raw wastewater" and "treated wastewater" designations were inadvertently reversed for the anthracene and phenanthrene data in USEPA (1980e). However, if the concentrations of anthracene and phenanthrene in treated wastewater from the Aluminum Forming Industry are typically as great as listed in Appendix A, Table A-5 the Aluminum Forming Industry could represent a major source of at least anthracene and phenanthrene pollution depending of course on the volumes of wastewater discharged.

2.6 FOUNDRY INDUSTRY

The Foundry Industry consists of 1,050 direct, 498 indirect, and 450 zero dischargers engaged in forming metal objects by the pouring or injection of molten metal into molds (USEPA 1980f). The Foundry Industry has been divided into 9 subcategories based primarily on the type of metal processed: Iron and Steel, Aluminum, Zinc, Copper, Magnesium, Lead, Tin, Titanium, and Nickel (USEPA 1980f).

Table A-6A of Appendix A presents minimum detected, maximum, mean detected, and mean PAH concentrations in raw and treated wastewater from the Foundry Industry as a whole (USEPA 1980e). The mean of mean PAH concentrations in raw wastewater ranks 5th (and in treated wastewater 4th) among the industrial categories listed in Table 2-1.

The relatively high ranking of the Foundry Industry as a whole appears to depend primarily upon contribution from the Aluminum Casting Subcategory. Table A-6B of Appendix A presents ranges, medians, and means of PAH concentrations in raw wastewater from 5 of the 9 subcategories (EPA 1980f). Data for the other 4 subcategories and for treated wastewater were not presented. It can be seen from Appendix A, Table A-6B that PAH concentrations in the raw wastewater from the Aluminum Casting Subcategory are generally much higher than for the other subcategories. Some of the PAH concentrations, such as for chrysene and benzo[a]anthracene, are comparable to PAH concentrations present in raw wastewaters from the Iron and Steel Manufacturing Industry and the Wood Preserving subcategories.

2.7 NON-FERROUS METALS MANUFACTURING INDUSTRY

The Non-ferrous Metals Manufacturing Industry consists of 129 direct, 79 indirect and 215 zero dischargers involved in the smelting and refining of non-ferrous metals (USEPA 1980f). The industry has been divided into 61 subcategories based on the type of metal processed and on whether the metal is recovered from ore (primary) or scrap (secondary). Only the following 12 of the 61 subcategories were under regulatory control as of 1980: Primary Aluminum, Secondary Aluminum, Primary Columbium, Primary Tantalum, Primary Copper,

Secondary Copper, Primary Lead, Secondary Lead, Secondary Silver, Primary Tungsten, Primary Zinc, and Primary Cadmium (USEPA 1980f).

Table A-7A of Appendix A presents minimum, maximum, and mean PAH concentrations for the Non-ferrous Metals Industry (the 12 subcategories regulated) as a whole (USEPA 1980e). The mean of mean PAH concentrations in raw wastewaters is comparable to that in the raw wastewater from the Foundry Industry. However, the mean of mean PAH concentrations in treated wastewater is much lower that that in treated wastewater from the Foundry Industry.

Table A-7B of Appendix A presents the range and means of PAH concentrations in raw wastewaters from 9 of the 12 subcategories currently regulated (EPA 1980f). PAH data was not presented for the other subcategories. Maximums and means of PAH concentrations were generally greater in raw wastewaters from the Copper and Primary Aluminum Subcategories than in the other subcategories of the Non-ferrous Metals Manufacturing Industry listed in Appendix A, Table A-6B.

The main source of PAHs in the wastewaters from the Primary Aluminum Subcategory may be from the production and rinsing of the anodes used in the reduction of aluminum ores to aluminum (USEPA 1983b; Neff 1979). The anodes (both pre-baked and Soderberg) are made from coal tar pitch, coke, anthracite and anthracene oil, all of which contain elevated levels of PAHs (USEPA 1983b; Neff 1979).

2.8 PETROLEUM REFINERIES

During 1976, the Petroleum Refinery Industry consisted of 182 direct, 48 indirect, and 55 zero discharges (USEPA 1980f). Petroleum refineries convert crude petroleum to various petroleum products including propane, gasoline, jet fuels, heating and lubricating oils, asphalt, coke, and various coke by-products (USEPA 1981c).

Table A-8A of Appendix A presents minimum detected, maximum, mean detected and mean PAH concentrations in raw and treated wastewater from the Petroleum Refinery Industry as a whole (USEPA 1980e). A substantial proportion of the Petroleum Refinery Industry is engaged in the pyrolysis of organic matter through the distillation of crude petroleum and/or coal. Furthermore,

relatively high levels of PAH have been reported in the higher boiling petroleum and coal distillation factions (Neff 1979, 1985). Therefore, it is somewhat suprising that the concentrations of PAHs in raw wastewater from the Petroleum Refinery Industry listed in Appendix A, Table A-8A are generally lower than those for six other industrial categories listed in Table 2-1. In addition, the concentrations in treated wastewater are among the lowest of any of the industrial subcategories. Since petroleum refineries are generally complex facilities engaged in numerous types of processes (USEPA 1983c), the relatively low concentrations of PAHs in the raw and treated wastewater listed in Appendix A, Table A-8A may be due to unrepresentative sampling or failure to sample wastewater discharges most likely to have elevated PAH concentrations. The extremely low PAH concentrations in treated wastewater may also be due to effective wastewater treatment.

Despite the relatively low PAH concentrations listed for wastewater from the Petroleum Refinery Industry in Appendix A, Table A-8A the processes that take place in petroleum refineries (e.g., distillation of crude petroleum and coal) do produce elevated levels of PAHs in at least the higher boiling factions as will be further discussed in Section 2.9. Therefore, it is possible that PAH levels in wastewater from such processes are at least occasionally relatively high. Furthermore, even if PAH levels in the treated wastewaters discharged from petroleum refineries are generally low, leakage of crude petroleum and petroleum products with elevated PAH levels from the storage facilities at petroleum refineries may sometimes occur. Therefore, a list of petroleum refineries within the States that comprise EPA Region V is provided in Appendix A, Table A-8B.

2.9 NON-POINT SOURCES

Potentially major non-point sources of PAH pollution to the aquatic environment were listed in Section 1.2. Among those listed were petroleum and petroleum product spills and leakage during transport and storage. Some crude petroleum and petroleum products have various PAH concentrations well exceeding one ppm (mg/kg). Neff (1979) compiled reported benzo[a]pyrene and other PAH concentrations in crude petroleum and petroleum products. Table A-9 of Appendix A, which is taken from Neff (1979), lists the concentrations of var-

ious PAHs in 2 crude oils and in 2 fuel oils. Table A-10 of Appendix A which is also taken from Neff (1979), lists concentrations of benzo[a]pyrene in various crude oils and petroleum products. In general, the tables show that PAH concentrations in crude oil are lower than those in the high boiling fraction petroleum products (e.g. asphalt, Bunker C residual oil), but higher than the PAH concentrations in the lower boiling fraction petroleum products (e.g., gasoline, light fuel oils). In addition, concentrations of the 2 and 3 ring PAHs in crude petroleum and petroleum products are generally much greater than concentrations of the 4 or more ring PAHs.

Neff (1979) estimates that petroleum spillage accounts for as much as 74% of the total PAH input and 4.3% of the benzo[a]pyrene input to the marine environment. Although the percentages of total PAH and benzo[a]pyrene input to freshwater systems by petroleum spillage or leakage are probably less than those for the marine environment, a major source of PAH pollution to the marine environment is thought to be freshwater river runoff (NAS 1976 in Neff 1979). In addition, moderate to heavy amounts of grease and oil have been reported in many sediment samples taken from heavily PAH polluted areas such as the Detroit River, the Milwaukee estuary, the Black River near Lorain, the Grand Calumet River and the Buffalo River. Sources of petroleum and petroleum product inputs to freshwater systems include spillage and leakage during transport and storage (Neff 1979) and discharges from boat and ship engines (Woo and Arcos 1981).

Surface runoff from roads and contaminated soils may also contribute substantial amounts of PAH to some freshwater systems (Neff 1979; Woo and Arcos). Such surface runoff can enter freshwater systems directly (through overland flow) or indirectly (through storm sewer overflows and discharges). Sources of PAHs in surface runoff from roads include leaching of asphalt surfaces, dissolution of carbon black residues from tires, and dissolution of vehicle exhaust condensates (Neff 1979, 1985). Sources of PAH in leachate from contaminated soils (other than those associated with hazardous waste sites) include atmospheric deposition and possibly plant biosynthesis (Neff 1979, 1982; Woo and Arcos, 1981).

Atmospheric deposition is predicted to account for substantial PAH inputs to freshwater systems with large surface areas such as the Great Lakes.

Atmospheric dry and wet depositions of PAH contaminated particulates are thought to be the primary source of PAHs to the Great Lakes (Eadie et al. 1982). Although many of the tributaries flowing into the Great Lakes are heavily contaminated with PAH-laden suspended solids, tributaries are predicted to account for only 10-25% of the PAH input to the Great Lake by atmospheric deposition (Eadie et al. 1982). Atmospheric deposition may also account for significant PAH inputs to water bodies with small surface areas during periods of heavy rainfall, since much of the PAH in surface runoff during heavy rainfall may be due to washout (Woo and Arcos 1981).

Other non-point sources of PAH to freshwater systems may include hazardous waste disposal sites and dredged sediment piles (NRTC 1984). Dredged sediment piles are frequently located at or near the banks of the water body from which they were dredged and may contain extremely high PAH concentrations. PAHs from dredged sediment piles may reenter the water body through leaching or through erosion and subsequent transport of PAH contaminated sediment from the dredge pile. PAHs from hazardous waste sites near the ground surface may also enter nearby water bodies through leaching or erosion of contaminated soil. PAHs at deeply buried hazardous waste sites may be leached into the groundwater and enter surface water through groundwater outcroppings.

3. PAH CONCENTRATION IN SEDIMENTS AND ASSOCIATED HUMAN CARCINOGENIC, HUMAN NON-CARCINOGENIC AND ACUTE AQUATIC TOXICITY RISK SCORES

Chapter 3 presents PAH sediment concentration data for each individual sampling site and individual PAH, as well as the associated human carcinogenic, human non-carcinogenic and acute aquatic toxicity risk scores for each PAH in each sediment sample taken from sites in USEPA Region V and at other sites near the Great Lakes. The data were classified into the following 10 regions and associated subregions for purposes of presentation and discussion:

- Indiana region - 35 sampling sites in Lake Michigan Harbors and Tributaries in Indiana (3 subregions: Indiana Harbor, Indiana Harbor Canal, and Grand Calumet River)
- Detroit region - 43 sampling sites (4 subregions: Detroit River and Tributaries, St. Clair River and Lake, Raisin River, and Lake Erie off Raisin River)
- Milwaukee region - 12 sampling sites (No subregions)
- Ohio Steel region - 23 sampling sites in the Black and Mahoning Rivers in Ohio (2 subregions: Black River at Lorrain, and Mahoning River)
- Ohio region - 42 sampling sites in Lake Erie Harbors and Tributaries in Ohio (5 subregions: Toledo, Cuyahoga River at Cleveland, Fairpoint Harbor, Ashtabula, and Conneaut Harbor)
- Michigan region - 33 sampling sites in Lake Michigan Harbors and Tributaries in Michigan (5 subregions: Manistique, Manistee, Muskegon and New Buffalo Harbor, Kalamazoo River, and St. Joseph Harbor)
- Open Lake Michigan region - 12 sampling sites well offshore in Lake Michigan (No subregions)
- Lake Superior region - 8 sampling sites in Lake Superior Harbors and Tributaries (2 subregions: Ashland, and Miscellaneous)
- Buffalo region - 151 sampling sites in the Niagara River system (9 subregions: Upper Niagara River and Chippawa Canal, Tonawanda Canal, Two Mile Creek, Lower Niagara River, Scajaquada Creek, Buffalo River, Buffalo Harbor, Lake Erie near Buffalo, and Rush and Smokes Creek)
- New York and Pennsylvania region - 25 sampling sites in Lake Ontario and Lake Erie Harbors and Tributaries in Pennsylvania and New York, excluding Buffalo (3 subregions: Rochester, Oswego and Olcott, and Dunkirk and Erie).

Data were collected and analyzed from each of the 384 individual reported sampling sites within these regions/subregions.

Chapter 3 is divided into 12 sections. The methodology used to generate human carcinogenic, human non-carcinogenic and aquatic toxicity risk scores from PAH sediment concentration data is described in Section 3.1. Each of the following 10 sections, 3.2-3.11, addresses one of the 10 regions listed above. Tables listing PAH sediment concentrations and human carcinogenic, human non-carcinogenic and acute aquatic toxicity scores associated with the PAH concentration or mean concentration at each sampling site were created for each region and are all contained in Appendix B. For most regions, a table describing the sediment sampling sites is also included in Appendix B. Figures showing sampling site locations are included for some regions and are incorporated with text in the appropriate section.

3.1 METHODOLOGY USED TO GENERATE HUMAN CARCINOGENIC, HUMAN NON-CARCINOGENIC AND ACUTE AQUATIC TOXICITY RISK SCORES ASSOCIATED WITH PAH SEDIMENT CONCENTRATIONS OR MEAN CONCENTRATIONS AT EACH SEDIMENT SAMPLING SITE

In order to rank regions, subregions within regions, sampling sites within subregions and PAHs within subregions according to potential risks to human and aquatic life, a methodology was developed to assign a human carcinogenic, human non-carcinogenic and acute aquatic toxicity risk score to each PAH sediment concentration or mean concentration at each sediment sampling site for which PAH sediment concentration data were available. Although the computations involved in deriving each of the risk scores parallel those which could be used to calculate actual risks if sufficient data were available, the resulting risk scores should only be used for comparative or ranking purposes. In most cases, the resulting risk scores probably exceed actual risk by several orders of magnitude and should not be taken as even worst case estimates of actual risk to human or aquatic life associated with the concentration of PAHs in sediments. There is, at present, insufficient data available to formulate precise estimates of the actual risks to human or aquatic life associated with PAH concentrations in sediments.

As described in Section 1.1, the PAHs listed in Table 1-1 were classified as either carcinogens or non-carcinogens based on the available evidence for

carcinogenicity (SAIC 1984). Human carcinogenic risk scores (x 10^4) were calculated for each carcinogenic PAH sediment concentration or mean concentration at each sediment sampling site from the following equation:

$$XC_{ij} = \frac{(UCR_j)\ (6.5 \times 10^{-3}\ kg/day)\ (BCF_j)\ (C_{ij})\ (10^4)}{(K_{ocj})\ (f_{oc})\ (70\ kg)} \qquad (3\text{-}1)$$

where:

XC_{ij} = human carcinogenic risk score (x 10^4) associated with the concentration of carcinogenic PAH j in sediment at sampling site i (unitless)

UCR_j = unit carcinogenic risk for PAH_j (assumed to be equal to 11.5 mg/kg/ day for all carcinogenic PAHs)

6.5×10^{-3} kg/day = assumed fish consumption per day by adult male

BCF_j = estimated bioconcentration factor for the uptake of carcinogenic PAH j from water by fish with a consumption weighted mean lipid content of 3% (unitless)

C_{ij} = concentration of carcinogenic PAH_j in sediment at sampling site i (mg/kg)

K_{ocj} = estimated organic carbon and adjusted sediment water partition coefficient for carcinogenic PAH j (unitless)

f_{oc} = organic fraction of sediment (assumed to be 0.05 in absence of data)

70kg = assumed weight of an adult male

(10^4) = multiplication factor used to prevent the computer from rounding small values down to zero.

The human carcinogenic scores computed from equation (3-1) are products of the unit carcinogenic risk factor times the daily dose of carcinogenic PAH due to the consumption of contaminated fish. Equation (3-1) was derived using the following assumptions:

- Humans are exposed to PAHs in the aquatic system only though the consumption of contaminated fish
- Fish are at steady state with respect to PAH tissue concentrations and become contaminated with PAHs only by uptake from the water column

- The concentrations of PAHs in the water column are equal to those that would be present in interstitial water at equilibrium with the PAH contaminated sediment.

The last assumption is primarily responsible for the human carcinogenic risk scores computed from equation (3-1) probably being several orders of magnitude greater than actual risks. The concentrations of PAHs in the water column even close to the sediments are much smaller than those that would be present in interstitial water at equilibrium with the sediment. However, without knowledge of the hydrodynamic characteristics of a water body, it is not possible to estimate the concentration of PAHs in the water column as a function of distance from, and PAH concentrations in, the sediment. Even if the hydrodynamic characteristics were known, such estimates would require modeling.

Since the only PAH for which a UCR has been establised is benzo[a]pyrene, the UCRs of the other PAHs had to be assumed to be equal to the UCR for benzo-[a]pyrene (11.5 day/kg/mg). Therefore, since the human carcinogenic risk scores computed from equation (3-1) are proportional to the UCR, they are positively biased for PAHs less carcinogenic than benzo[a]pyrene and are negatively biased for PAHs more carcinogenic than benzo[a]pyrene.

Human non-carcinogenic risk scores (x 10^2) were calulated for each non-carcinogenic PAH sediment concentration or mean concentration at each sediment sampling site from the following equation:

$$XN_{ij} = \frac{(6.5 \times 10^{-3}\ \text{kg/day})\ (BCF_j)\ (C_{ij})\ (10^2)}{(ADI_j)\ (K_{ocj})\ (f_{oc})\ (70\ \text{kg})} \qquad (3\text{-}2)$$

where

XN_{ij} = human non-carcinogenic risk score (x 10^2) associated with the concentration of non-carcinogenic PAH j in sediment at sampling site i (unitless)

ADI_j = acceptable daily intake of non-carcinogenic PAH j (mg/kg/day)

(10^2) = multiplication factor used to prevent the computer from rounding small values to zero.

All of the other parameters in equation (3-2) are the same as defined for equation (3-1).

The human non-carcinogenic risk scores computed from equation (3-2) are the ratios of the daily doses of non-carcinogenic PAHs, due to the consumption of contaminated fish, to the ADIs. Equation (3-2) was derived using the same assumptions listed above for deriving equation (3-1). Again, the assumption that PAH concentrations in the water column are equal to those that would be present in interstitial water in equilibrium with the sediment is primarily responsible for the human non-carcinogenic risk scores probably being much higher than actual risks.

The ADIs for all of the non-carcinogenic PAHs except naphthalene were assumed to be equal to that of fluoranthene (5.9×10^{-3} mg/kg/day) except for naphthalene (0.26 mg/kg/day). Therefore, since the non-carcinogenic risk scores computed from equation (3-2) are inversely proportional to the ADI, they are positively biased for non-carcinogens less toxic than fluoranthene and negatively biased for non-carcinogens more toxic than fluoranthene.

Acute aquatic toxicity risks scores ($\times 10^3$) were calculated for each PAH sediment concentration or mean concentration at each sediment sampling site from the following equation:

$$Y_{ij} = \frac{C_{ij}\ (10^3)}{(ACRI_j)\ (K_{ocj})\ (f_{oc})} \qquad (3\text{-}3)$$

where

Y_{ij} = acute aquatic toxicity risk score for PAH j in sediment at sampling site i

$(ACRI_j)$ = acute aquatic life criteria for PAH j

(10^3) = multiplication factor used to prevent the computer from rounding down $\overline{Tow}$ values to zero.

All of the other parameters in equation (3-3) are the same as defined for equation (3-1).

The acute aquatic toxicity risk scores computed from equation (3-3) are the ratios of PAH concentrations in the water column to acute aquatic crite-

ria, assuming that the PAH concentrations in the water column are equal to those that would be present in interstitial water in equilibrium with the sediment. That assumption may again make acute aquatic toxicity risk scores much higher than actual risks.

There have been no acute aquatic life criteria developed for any of the PAHs. Therefore, all of the PAHs except fluoranthene and naphthalene were assumed to have acute aquatic life criteria of 1.7 mg/L based on a no effect level of acenaphthene on bluegills (USEPA 1980b). Acute aquatic criteria for napthalene and fluoranthene were assumed to be 2.3 mg/L and 4.0 mg/L, respectively, based on no effect levels (USEPA 1980c, d). Therefore, since the acute aquatic life risk scores are inversely proportional to the assumed acute aquatic criteria, they will be positively biased for PAHs (other than naphthalene and fluoranthene) less toxic than acenaphthene and negatively biased for PAHs more toxic than acenaphthene.

3.2 INDIANA REGION: SAMPLING SITES IN LAKE MICHIGAN HARBORS AND TRIBUTARIES IN INDIANA

The Indiana region is divided into the following 3 subregions: Indiana Harbor, Indiana Harbor Canal and the Grand Calumet River.

Sediment sampling sites for PAH sediment concentration data in the Indiana region are described in Appendix B, Table B-1A. Site ACOE was sampled by the Army Corps of Engineers in 1985 (ACOE 1985). Sites LTI2 and LTI 5-1 were sampled by Limno-Tech, Inc. in 1984 (LTI 1984). Sites 501 through 514 were sampled by the Central District Office (State of Indiana?) in 1980 (Appendix C of USEPA 1982a). Sites C2 through C17 were sampled by HydroQual, Inc. in 1983 (HydroQual 1984). The Indiana Harbor Canal Sites at Canal Street, the Forks, Indianapolis Blvd. and Columbus Drive were sampled by USEPA in 1977 (USEPA ~~19~~82a). The Grand Calumet River sites 100 through 35,600 were sampled by USEPA in 1972 (USEPA 1982a). The site numbers represent feet from a culvert located at the headwaters of the East Branch of the Grand Calumet River. Table B-1B of Appendix B lists potentially significant point sources of pollution for the Indiana region (USEPA 1982a).

Figure 3-1 is a map of the Grand Calumet River, Indiana Harbor Canal and Indiana Harbor showing river miles and general direction of flow (HydroQual 1984). Figure 3-2 is a map of the Grand Calumet River, Indiana Harbor Canal and Indiana Harbor showing sampling sites S01 through S14 and major point sources of pollution (USEPA 1982a). (The point source ID numbers in Figure 3-2 correspond to those listed in Appendix B, Table B-2B.) Figure 3-3 is a map of the Grand Calumet River, Indiana Harbor Canal and Indiana Harbor showing sediment sampling sites C2 through C17 (HydroQual 1984).

PAH sediment concentration data (mg/kg) for the Indiana region are listed in Appendix B, Table B-1C. Human carcinogenic (x 10^4), human non-carcinogenic (x 10^2) and acute aquatic life (x 10^3) risk scores associated with the PAH sediment concentration data from the Indiana region are listed in Appendix B, Tables B-1D, B-1E and B-1F, respectively.

3.3 DETROIT REGION

The Detroit region is divided into the following 4 subregions: Detroit River and Tributaries, St. Clair River and Lake, Raisin River, and Lake Erie off the Raisin River.

Sediment sampling sites for PAH sediment concentration data in the Detroit region are described in Appendix B, Table B-2A. Sites within the Detroit River subregion were sampled by GLNPO in 1982 (USEPA 1985a and STORET). Sites within the St. Clair River/Lake and Raisin River subregion were sampled in 1981 and were described in STORET. Sites within the Lake Erie subregion were sampled by the Great Lakes National Research Lab of NOAA prior to 1982 (Eadie et al. 1981).

Figure 3-4 is a map of the Detroit River and Tributaries subregion showing the sites sampled by GLNPO in 1982 (USEPA 1985a).

PAH sediment concentration data (mg/kg) for the Detroit region are listed in Appendix B, Table B-2B. Human carcinogenic (x 10^4), human non-carcinogenic (x 10^2), and acute aquatic life (x 10^3) risk scores associated with the Detroit region PAH sediment concentration data are listed in Appendix B, Tables B-2C, B-2D and B-2E, respectively.

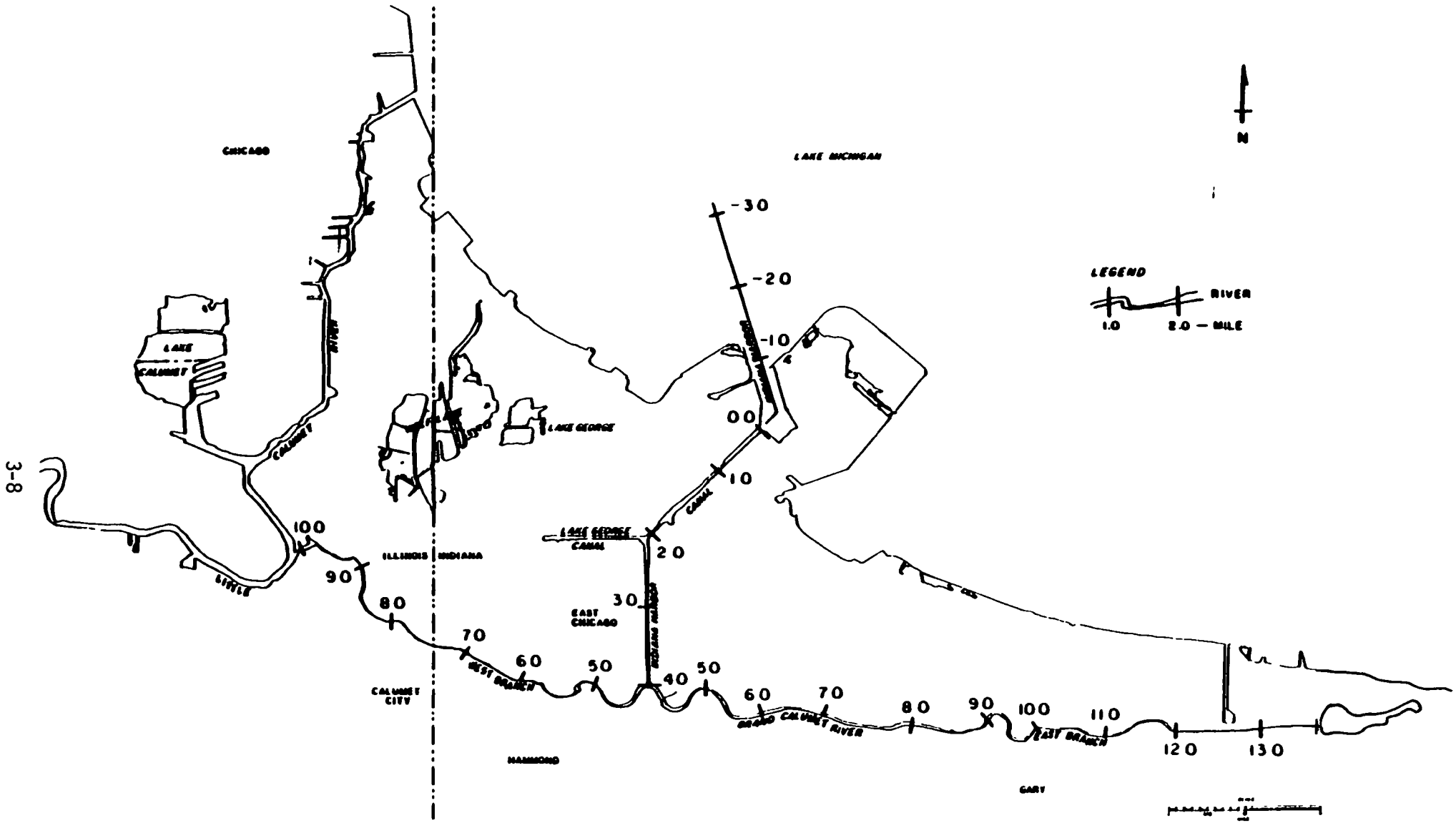

Figure 3-1 Map of the Grand Columet River, Indiana Harbor Canal and Indiana Harbor Showing River Miles and General Direction of Flow. Source: Hydroqual (1984)

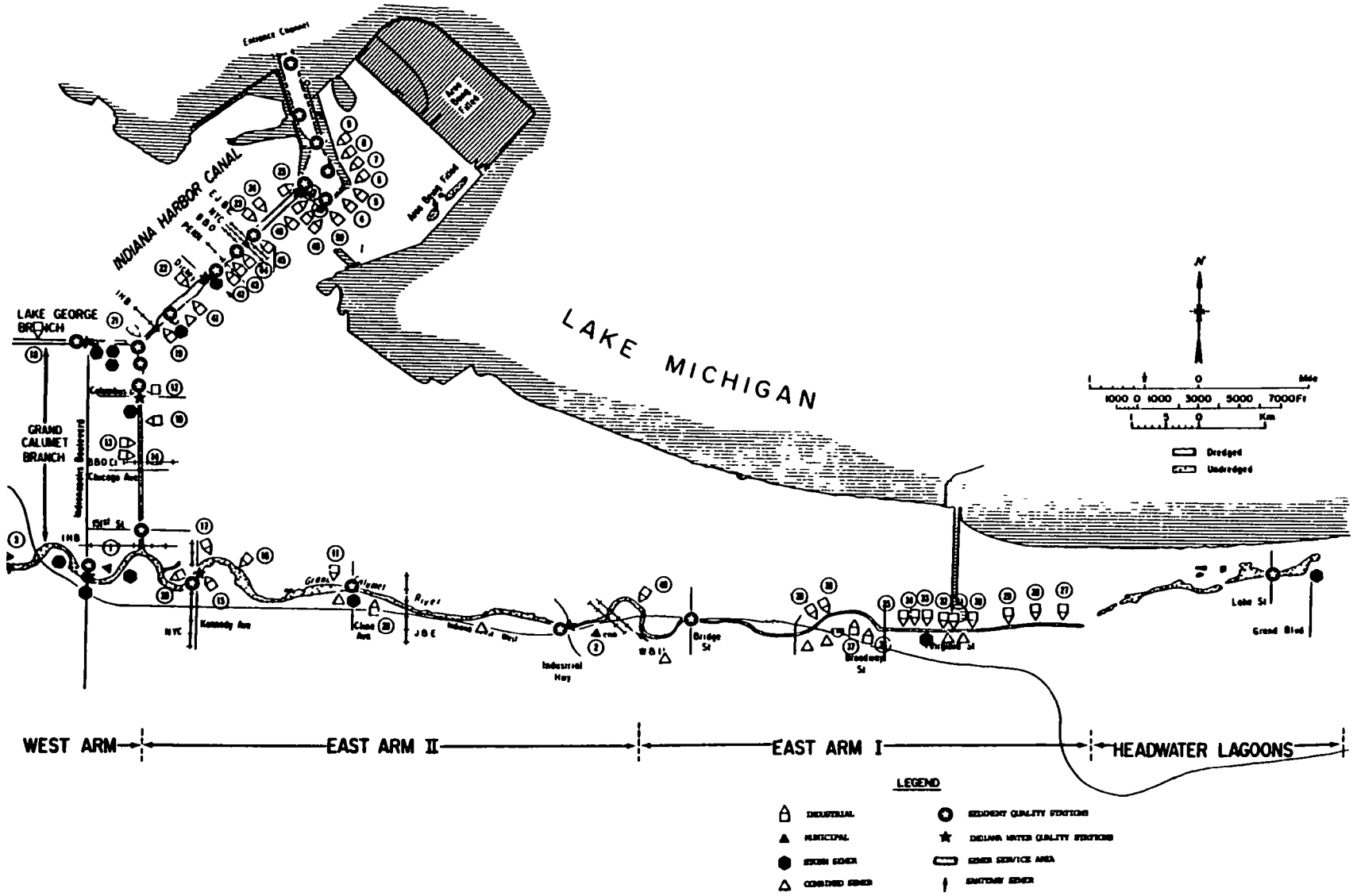

Figure 3-2 Map of the Grand Calumet River, Indiana Harbor Canal and Indiana Harbor Showing Sediment Sampling Sites in the Study Conducted by GLNPO(S) and Major Point Sources of Pollution. Numbers are identified in Table 3-1B.
Source: EPA (1982a)

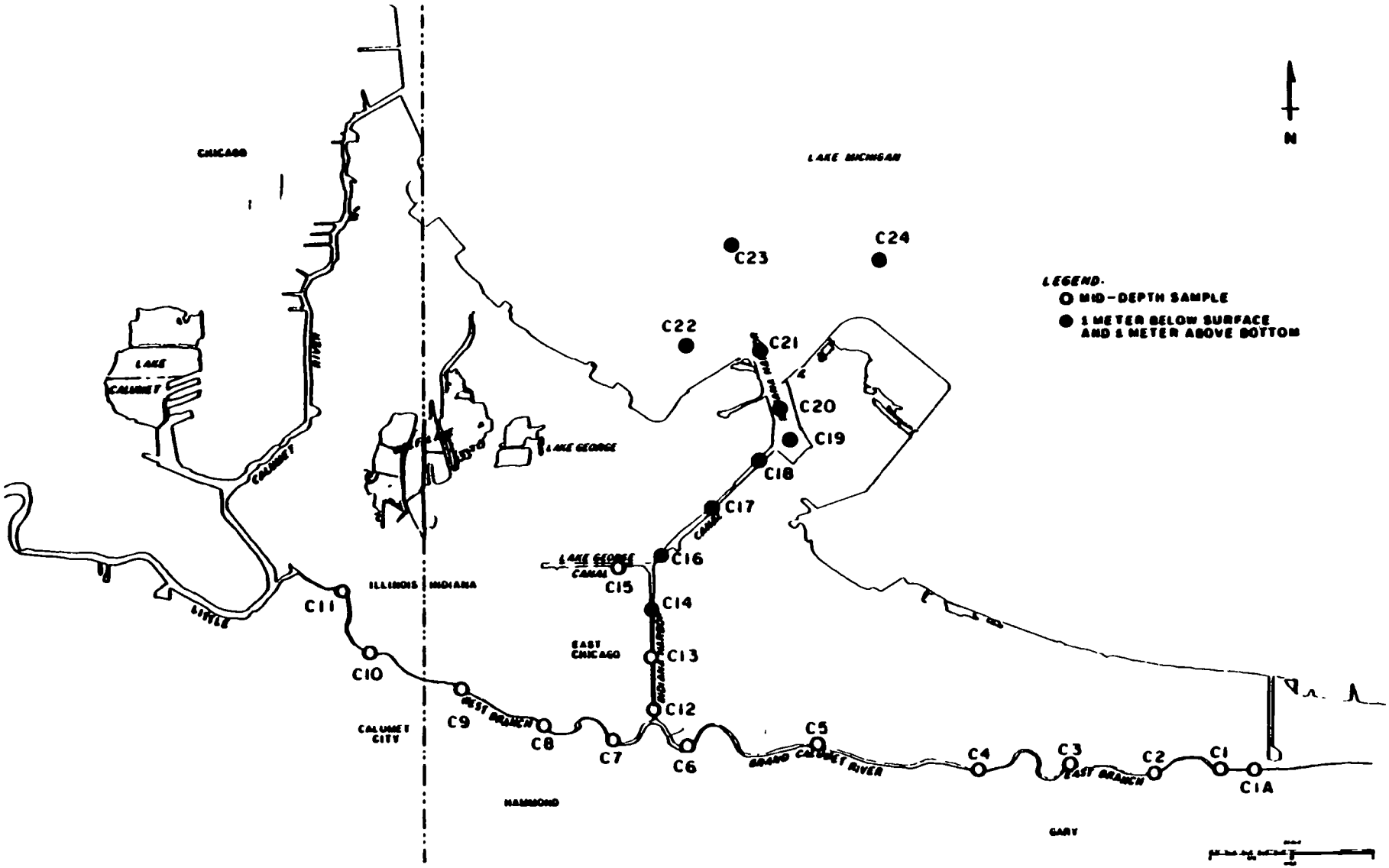

Figure 3-3 Map of the Grand Columet River, Indiana Harbor Canal and Indiana Harbor Showing Sediment Sampling Sites in the Study Conducted by Hydroqual. Source: Hydroqual (1989)

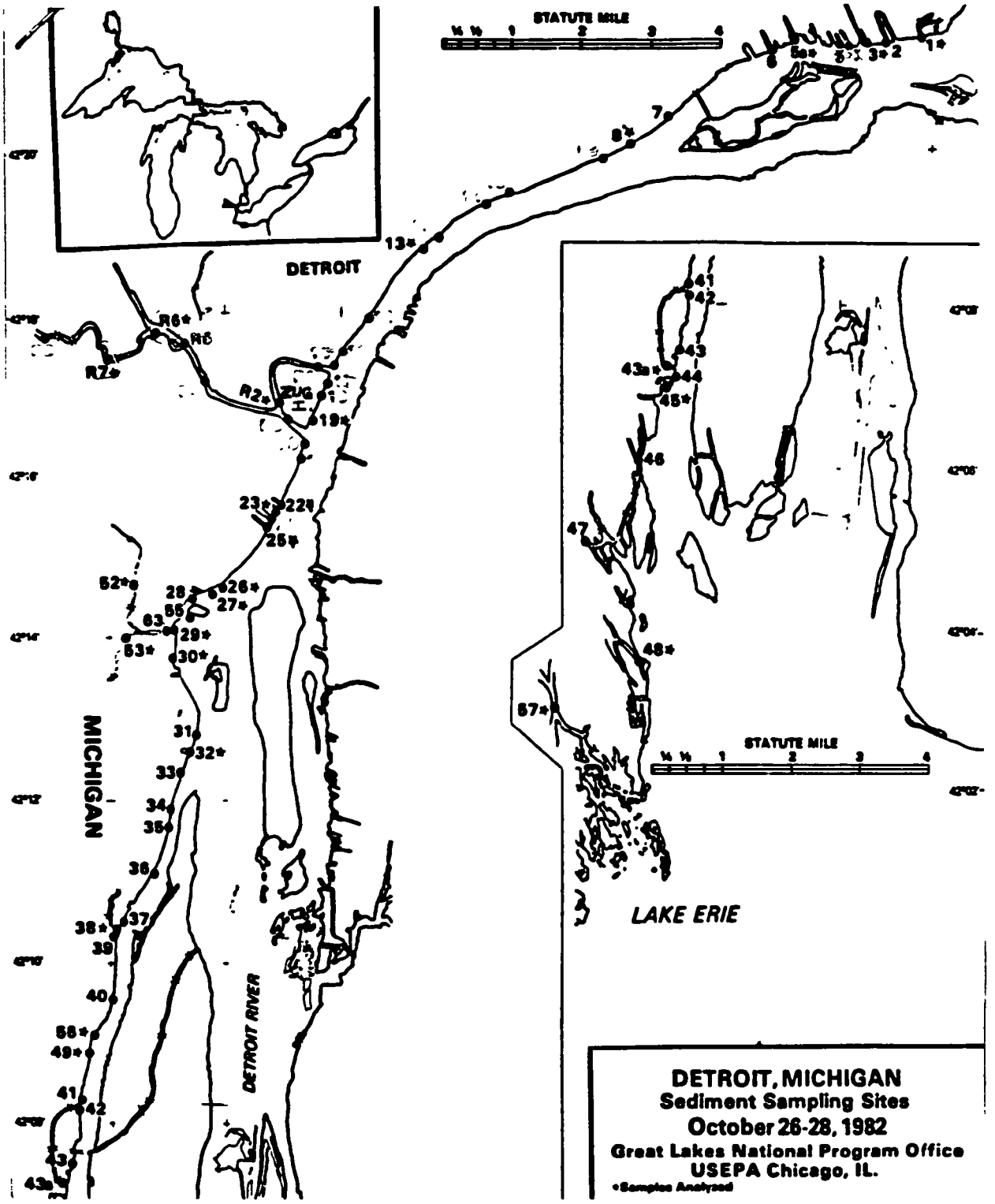

Figure 3-4 Map of the Detroit River and Tributaries Showing Sediment Sampling Sites in the Study Conducted by GLNPO in 1982
Source: EPA (1985a)

3.4 MILWAUKEE REGION

The Milwaukee region is not divided into subregions.

Sediment sampling sites for PAH sediment concentration data in the Milwaukee region are described in Appendix B, Table B-3A. Sites within the Milwaukee region were sampled by GLNPO in 1980 (USEPA 1985b). Figure 3-5 is a map of the Milwaukee Estuary showing the sites sampled by GLNPO in 1980 (USEPA 1985b).

PAH sediment concentration data (mg/kg) for the Milwaukee region are listed in Appendix B, Table B-3B. Human carcinogenic (x 10^4), human non-carcinogenic (x 10^2), and acute aquatic life (x 10^3) risk scores associated with the PAH sediment concentration data from the Milwaukee region are listed in Appendix B, Tables B-3C, B-3D, and B-3E, respectively.

3.5 OHIO STEEL REGION: SAMPLING SITES IN THE BLACK AND MAHONING RIVERS

The Ohio Steel region is divided into the following two subregions: Black River/Lorain and Mahoning River.

Sediment sampling sites for PAH sediment concentration data in the Ohio Steel region are described in Appendix B, Table B-4A. Sites LOR82-07 through LOR82-18 were sampled in 1982 and are described in STORET. Samples USS1 through USS5 were sampled by Environmental Research Group, Inc. in 1984 (ERG 1984). Black River site B-1 was sampled by Bauman et al. (1982). Black River site B-2 was sampled by Black et al. (1985). Sites 19466 (2) through 19476 (12) were sampled in 1974 by the National Field Investigation Center of EPA located in Cincinnati (USEPA 1974). Mahoning River sites Warren, Youngstown and Struthers were sampled in 1975 by EPA region V (USEPA 1977).

Figure 3-6 is a map of the Black River/Lorain area showing sampling sites USS1 through USS5. Figure 3-7 is a map of the Black River/Lorain area showing sampling sites 19466 (2) through 19476 (12).

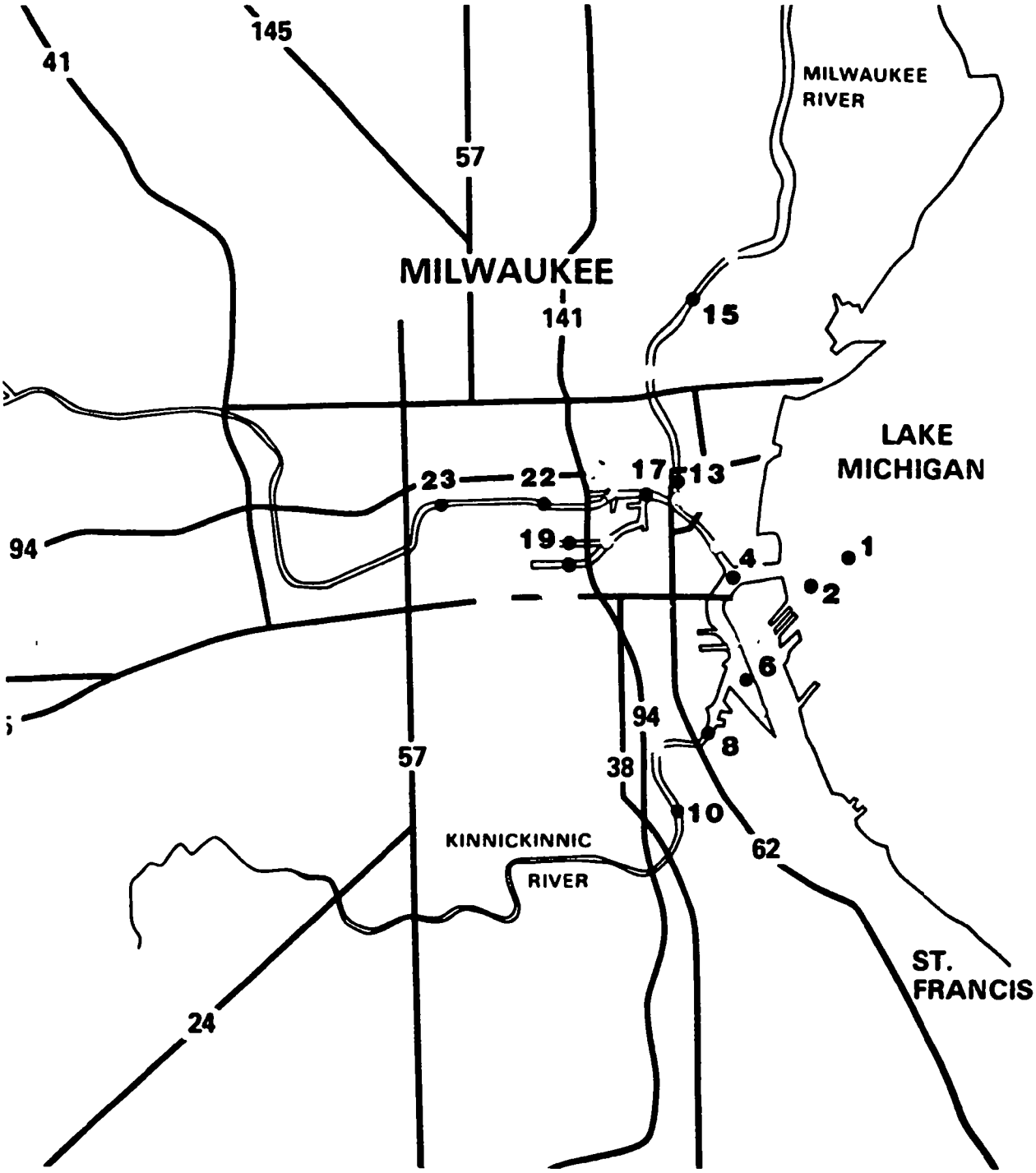

Figure 3-5 Map of the Milwaukee Estuary Showing Sediment Sampling Sites in the Study Conducted by GLNPO in 1980. Source: EPA (1985b)

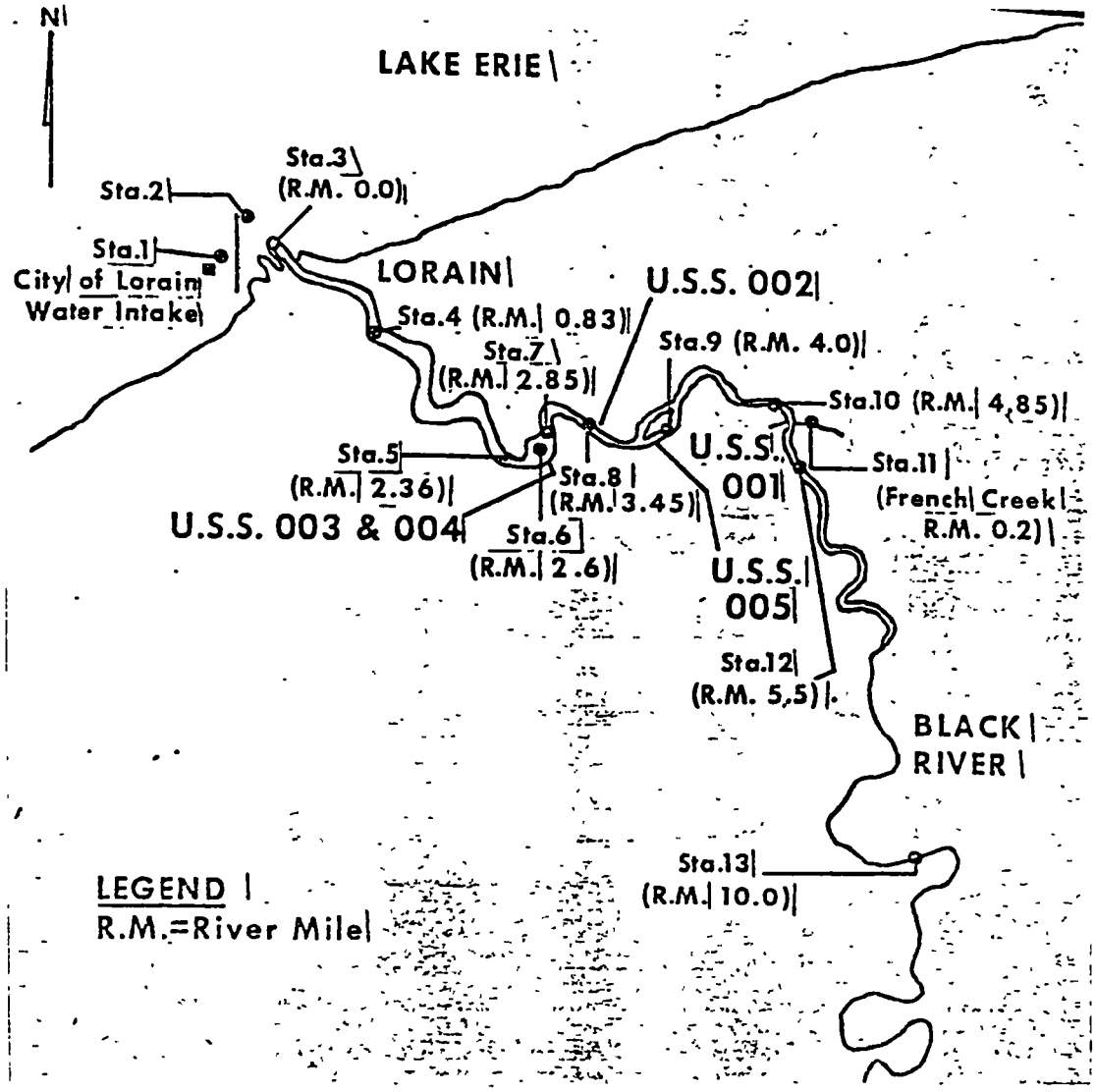

Figure 3-6 Map of the Black River in the Lorain Area Showing Approximate Sediment Sampling Sites in the Study Conducted by EPA in 1974. Source: USEPA (1974)

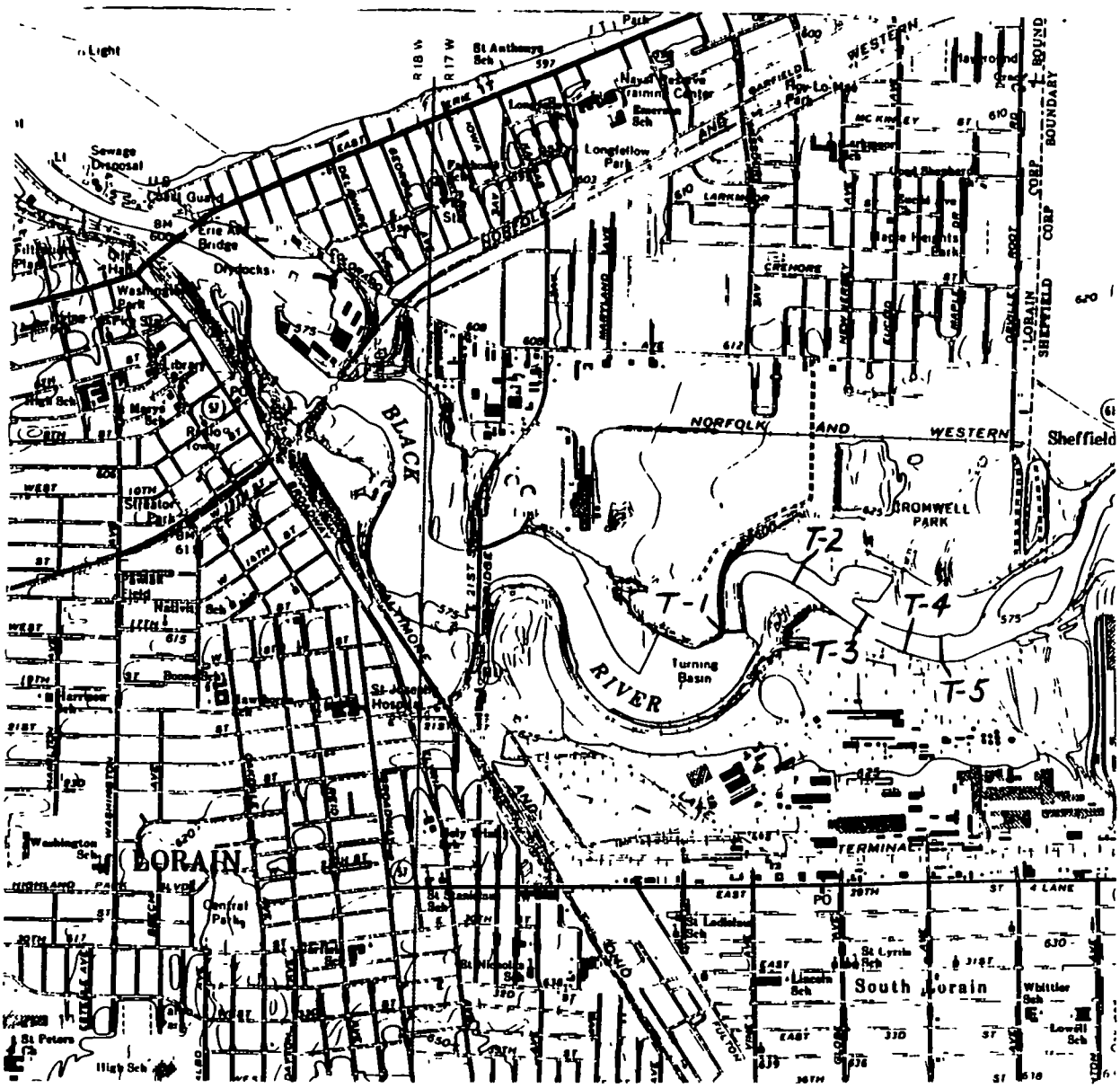

Figure 3-7 Map of the Black River in the Lorain Area Showing Sediment Sampling Sites in the Study Conducted by ERG in 1984. Source: ERG (1984)

PAH sediment concentration data (mg/kg) for the Ohio Steel region are listed in Appendix B, Table B-4B. Additional PAH sediment concentration data for the Black River are reported by ATEC (1983), however these data are not included in this report. Human carcinogenic (x 10^4), human non-carcinogenic (x 10^2) and acute aquatic life (x 10^3) risk scores associated with the PAH sediment concentration data of the Ohio Steel region are listed in Appendix B, Tables B-4C, B-4D, and B-4E, respectively.

3.6 OHIO REGION: LAKE ERIE HARBORS AND TRIBUTARIES IN OHIO (EXCLUDING BLACK AND MAHONING RIVERS)

The Ohio region is divided into the following five subregions: Toledo, Cuyahoga River (Cleveland), Fairpoint Harbor, Ashtabula and Conneaut Harbor.

Sediment sampling sites for PAH sediment concentration data in the Ohio region are described in Appendix B, Table B-5A. All of the sites except those in Ashtabula were sampled in 1982 (STORET 1985). The sites in Ashtabula were sampled in 1979 (STORET 1985). All of the site descriptions were taken from STORET (1985).

PAH sediment concentration data (mg/kg) for the Ohio region are listed in Appendix B, Table B-5B. Human carcinogenic (x 10^4), human non-carcinogenic (x 10^2) and acute aquatic life (x 10^3) risk scores associated with the PAH sediment concentration data from the Ohio region are listed in Appendix B, Tables B-5C, B-5D, and B-5E, respectively.

3.7 MICHIGAN REGION: LAKE MICHIGAN HARBORS AND TRIBUTARIES IN MICHIGAN

The Michigan region is divided into the following five subregions: Manistique, Manistee, Muskegon and St. Joseph Harbor.

Sediment sampling sites for PAH sediment data in the Michigan region are described in Appendix B, Table B-6A. All of the sites except those in New Buffalo Harbor were sampled in 1981 (STORET). Sites in New Buffalo Harbor were sampled in 1977 (STORET 1985). All of the site descriptions were taken from STORET (1985).

PAH sediment concentration data (mg/kg) for the Michigan region are listed in Appendix B, Table B-6B. Human carcinogenic (x 10^4), human non-carcinogenic (x 10^2) and acute aquatic life (x 10^3) risk scores associated with the PAH sediment concentration data from the Michigan region are listed in Appendix B, Tables B-6C, B-6D and B-6E, respectively.

3.8 OPEN LAKE MICHIGAN REGION

The Open Lake Michigan region is not divided into subregions.

PAH sediment concentration data (mg/kg) for the Open Lake Michigan region are listed in Appendix B, Table B-7A. Figure 3-8 is a map of Lake Michigan showing sampling site locations T1 through T11 which were sampled prior to 1977 (Cahill 1977 in Eadie 1984). Sites NOAA 24, NOAA 45 and NOAA 60 were sampled by the Great Lakes National Research Lab of NOAA prior to 1983 and are located several km offshore from Grand Haven Michigan (Eadie et al 1982).

Human carcinogenic (x 10^4), human non-carcinogenic (x 10^2) and acute aquatic life (x 10^3) risk scores associated with the PAH sediment concentration data from the Open Lake Michigan region are listed in Appendix B, Tables B-7B, B-7C and B-7D, respectively.

3.9 LAKE SUPERIOR REGION

The Lake Superior region is divided into the following two subregions: Ashland and Miscellaneous.

PAH sediment concentration data (mg/kg) for the Lake Superior region are listed in Appendix B, Table B-8A. All of the sites except the single open Lake Superior site were sampled by GLNPO in 1981 (USEPA 1984a). All three of the Ashland sampling sites are located in Lake Superior, but just a few feet offshore. The Ashland sites ASH 81-01, ASH 81-03 and ASH-05 are located just off a power plant outfall, a sewage treatment plant and the mouth of a small creek, respectively. The single Black River (Michigan) site BRH 81-03 is located at the mouth of the river in the center of the harbor. Site LAN 81-03 is located in Lake Superior just offshore of a submerged sewer outfall at L'Arse, Michigan. Site Lan 81-04 is also located in Lake Superior just offshore of L'Arse but not at a sewer outfall. Site LAN 81-07 is located in the

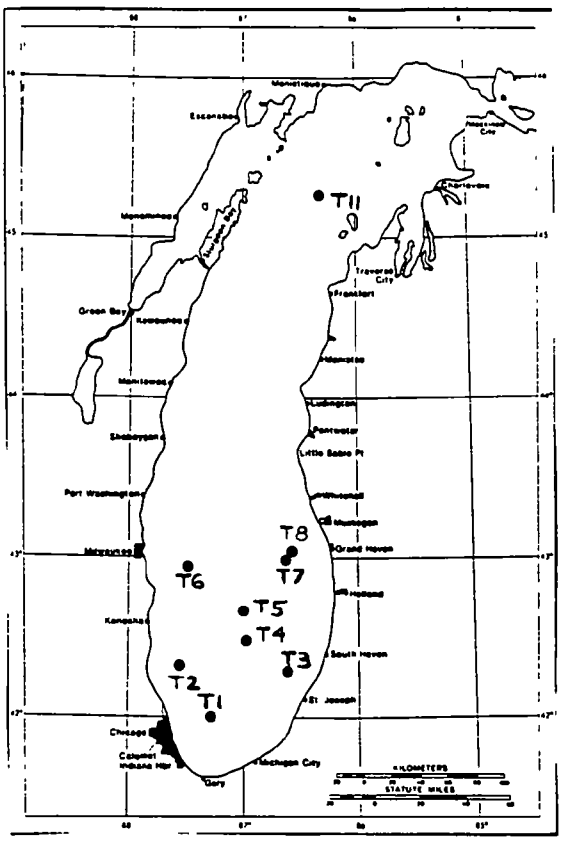

Figure 3-8 Map of Lake Michigan Showing Sediment Sampling Sites in a Study Reported by Cahill in 1977. (Source: Eadie 1984)

Fall River close to the mouth and L'Arse Harbor. The data for the open Lake Superior site was reported by Gschwend and Hites (1981 in Eache 1984) but no description of the site was given.

Human carcinogenic (x 10^4), human non-carcinogenic (x 10^2) and acute aquatic life (x 10^3) risk scores associated with the sediment concentration data from the Lake Superior region are listed in Appendix B, Tables B-8B, B-8C, and B-8D, respectively.

3.10 BUFFALO REGION: NIAGARA RIVER SYSTEM

The Buffalo region is divided into the following subregions: Upper Niagara River and Chippawa Canal, Tonawanda Canal, Two Mile Creek, Lower Niagara River, Scajaquada Creek, Buffalo River, Buffalo Harbor, Lake Erie (Buffalo), and Rush and Smokes Creeks.

Sediment sampling sites for PAH sediment concentration data in the Buffalo region are described in Appendix B, Table B-9A. Sites identified with numbers G1 through G67 were sampled by the Great Lakes National Program Office (GLNPO) of EPA Region V in 1981 (USEPA 1984b; NRTC 1984). Sites identified with numbers N1 through N30 were sampled by the New York State Department of Environmental Conservation (NYSDEC) in 1981 (EPA 1985c; NRTC 1984). Sites identified with numbers N31 through N43 were sampled by NYSDEC in 1982 (NRTC 1982). Table 3-9B of Appendix B lists potentially significant point sources of pollution along the Niagara River System (NRTC 1984). Table B-9C of Appendix B lists potentially significant hazardous waste disposal sites along the Niagara River System.

Figures 3-9, 3-10, 3-11, 3-12, 3-13, and 3-14 are maps of the Niagara River System showing the direction of flow, potentially significant point source of pollution, potentially significant disposal sites, sampling sites G1 through G-67, sampling sites N1 through N43 and sampling sites with figure ID numbers begining with U, respectively.

PAH sediment concentration data (mg/kg) for the Buffalo region are listed in Appendix B, Table B-9D. Human carcinogenic (x 10^4), human non-carcinogenic (x 10^2), and acute aquatic life (x 10^3) risk scores associated with the PAH sediment concentration data from the Buffalo region are listed in Appendix B, Tables B-9E, B-9F, and B-9G, respectively.

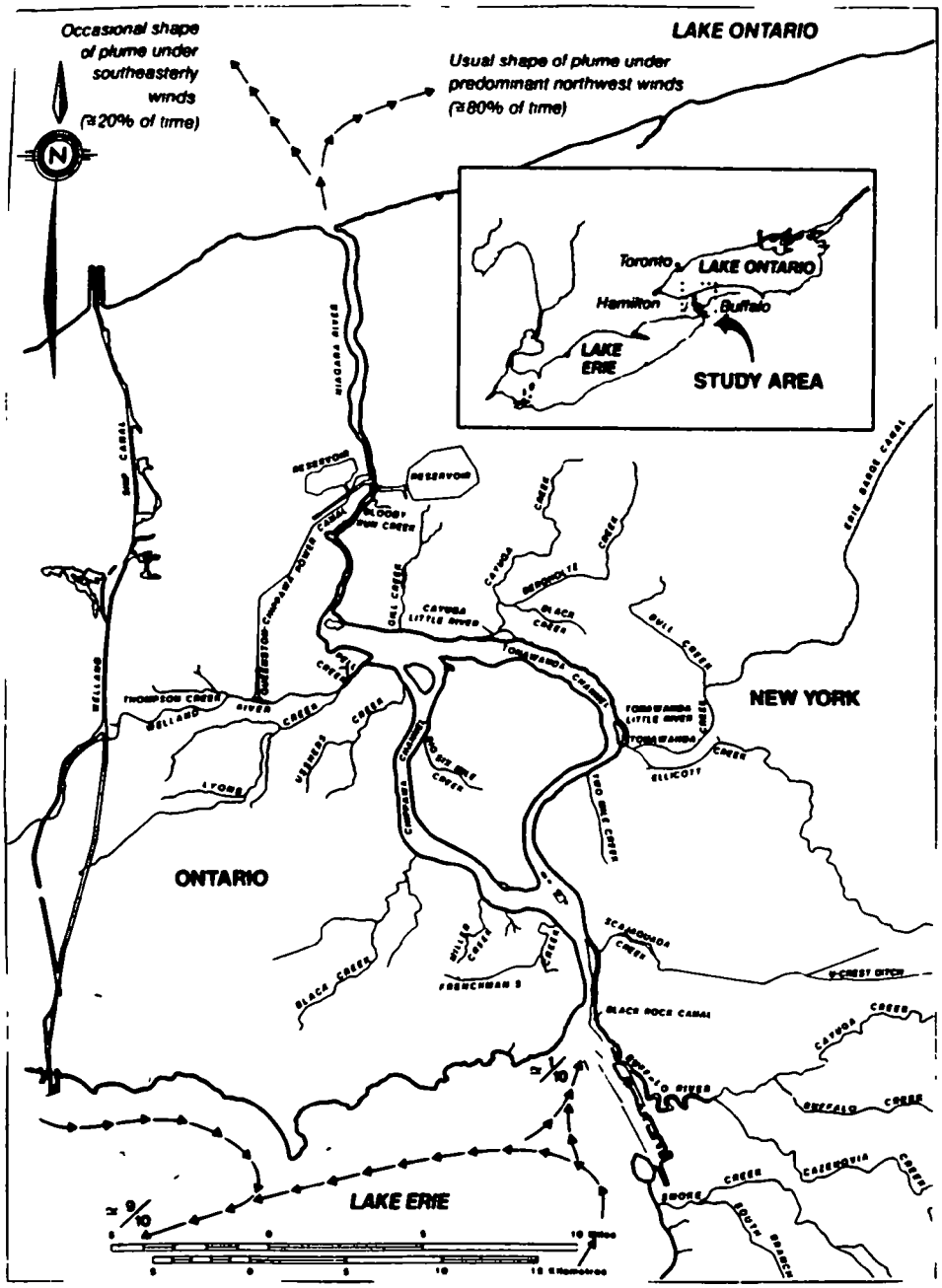

Figure 3-9 Map of Niagara River System Showing the Direction of Flow. (Source: NRTC 1984)

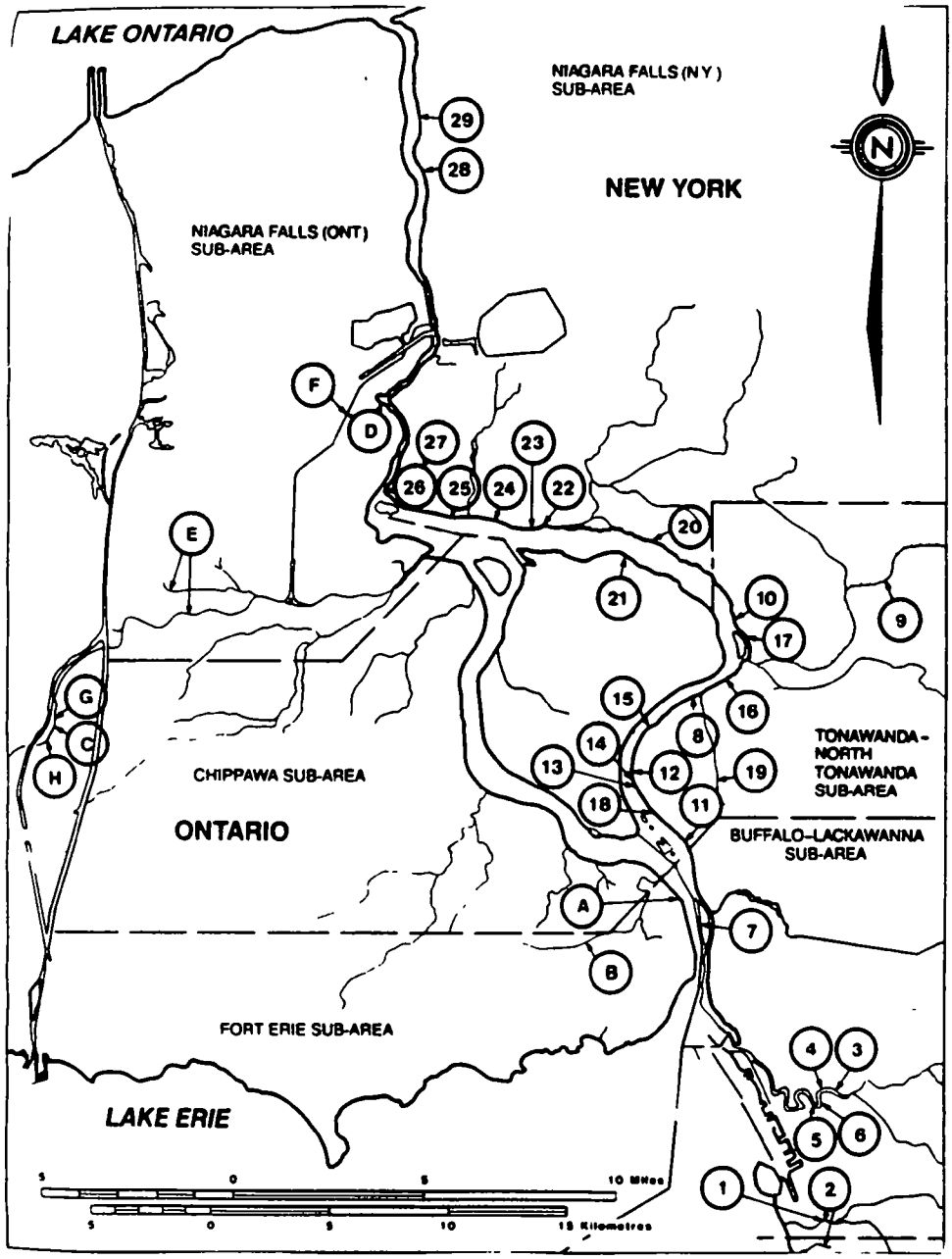

Figure 3-10 Map of the Niagara River System Showing Potential Point Sources of Pollution. Numbers are Identified in Table 3-9B. (Source: NRTC 1984)

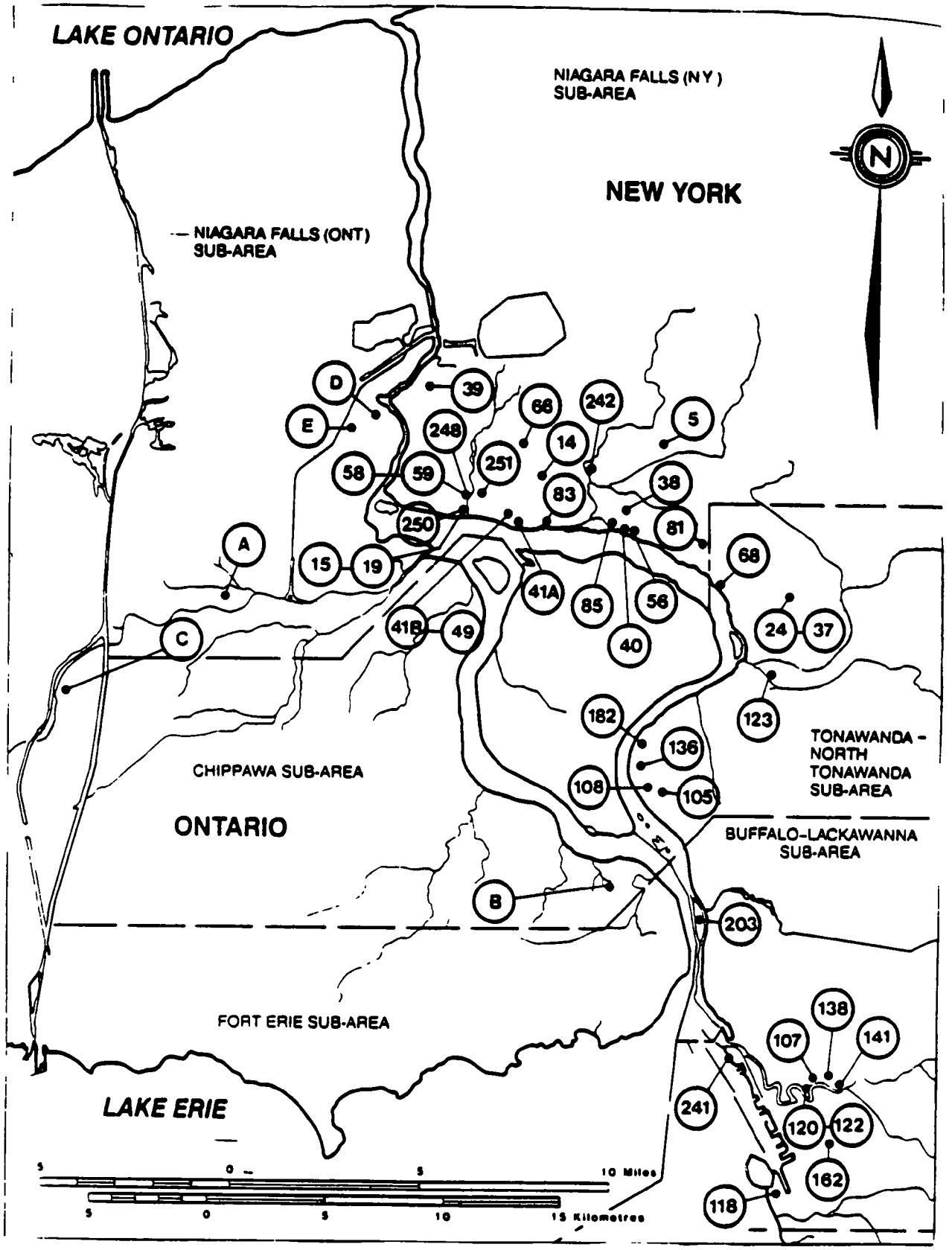

Figure 3-11 Map of the Niagara River System Showing Hazardous Waste Disposal and Landfill Sites. (Source: NRTC 1984)

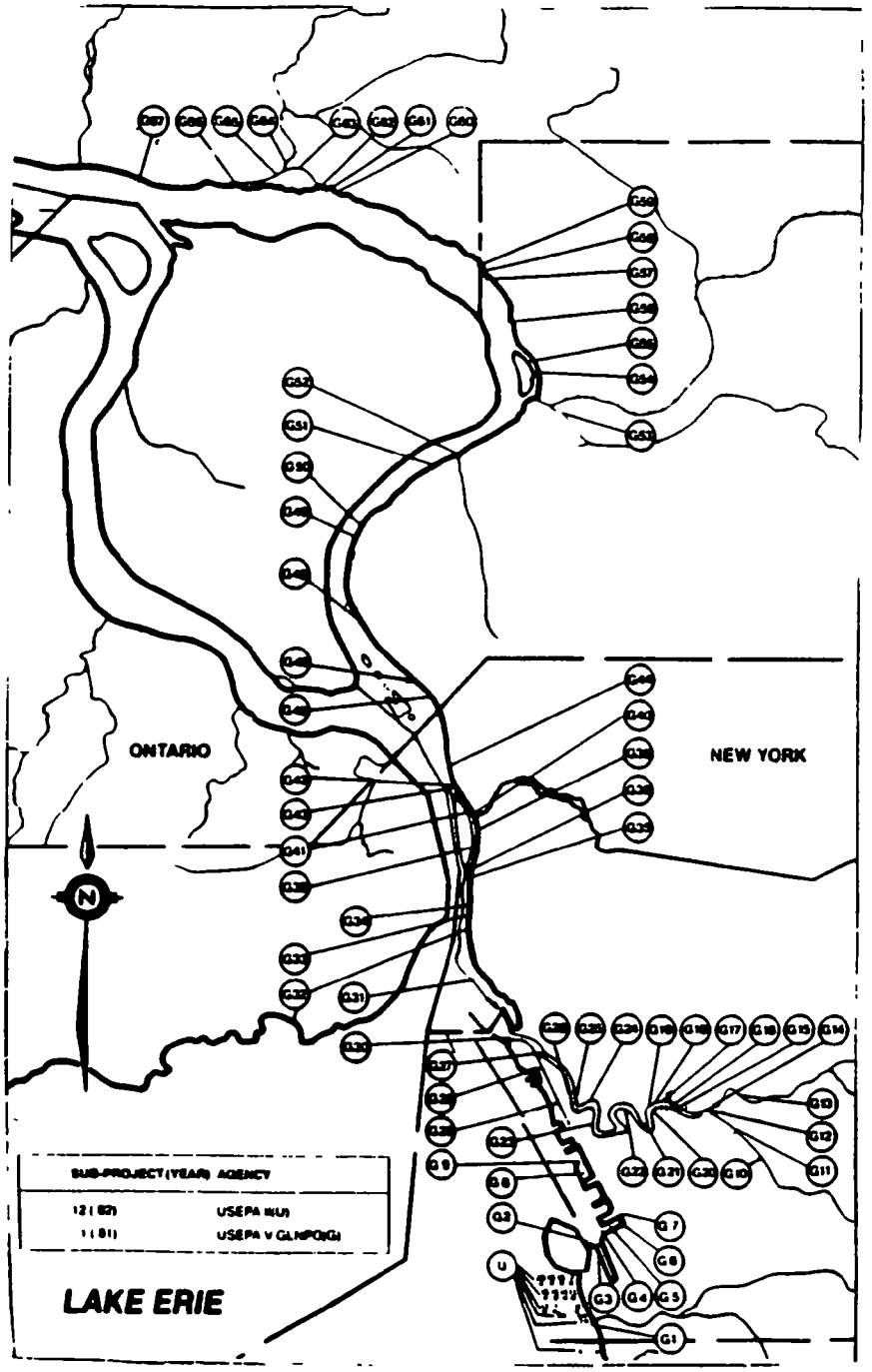

Figure 3-12 Map of Niagara River System Showing Sediment Sampling Sites in Studies Conducted by GLNPO in 1981 (G) and EPA Region II in 1982 (U). (Source: NRTC 1984)

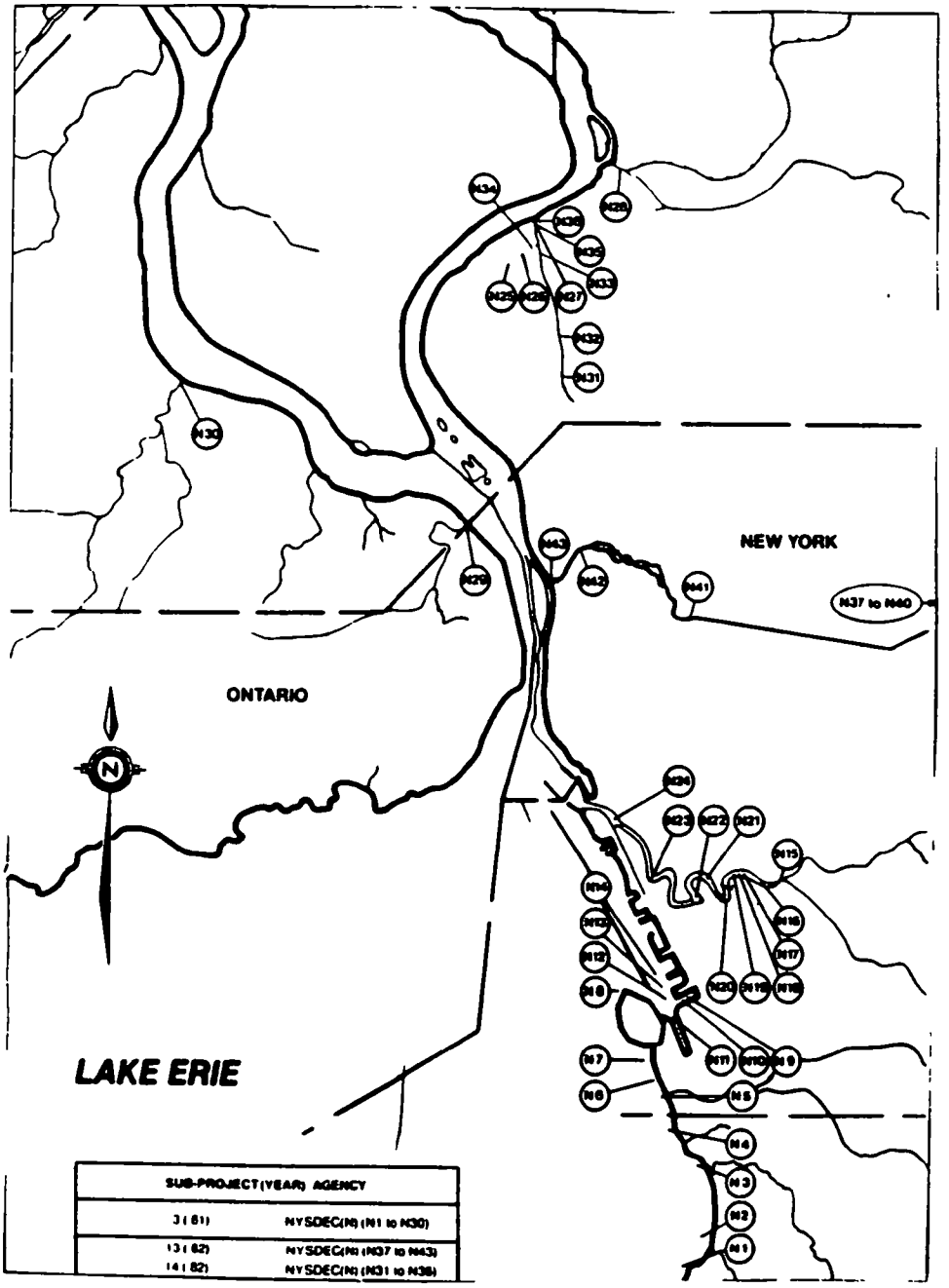

Figure 3-13 Map of Niagara River System Showing Sediment Sampling Sites in Studies Conducted by the New York State Department of Environmental Control (NYSDEC) in 1981 (N1 to N30) and 1982 (N31 to N43). (Source: NRTC 1984)

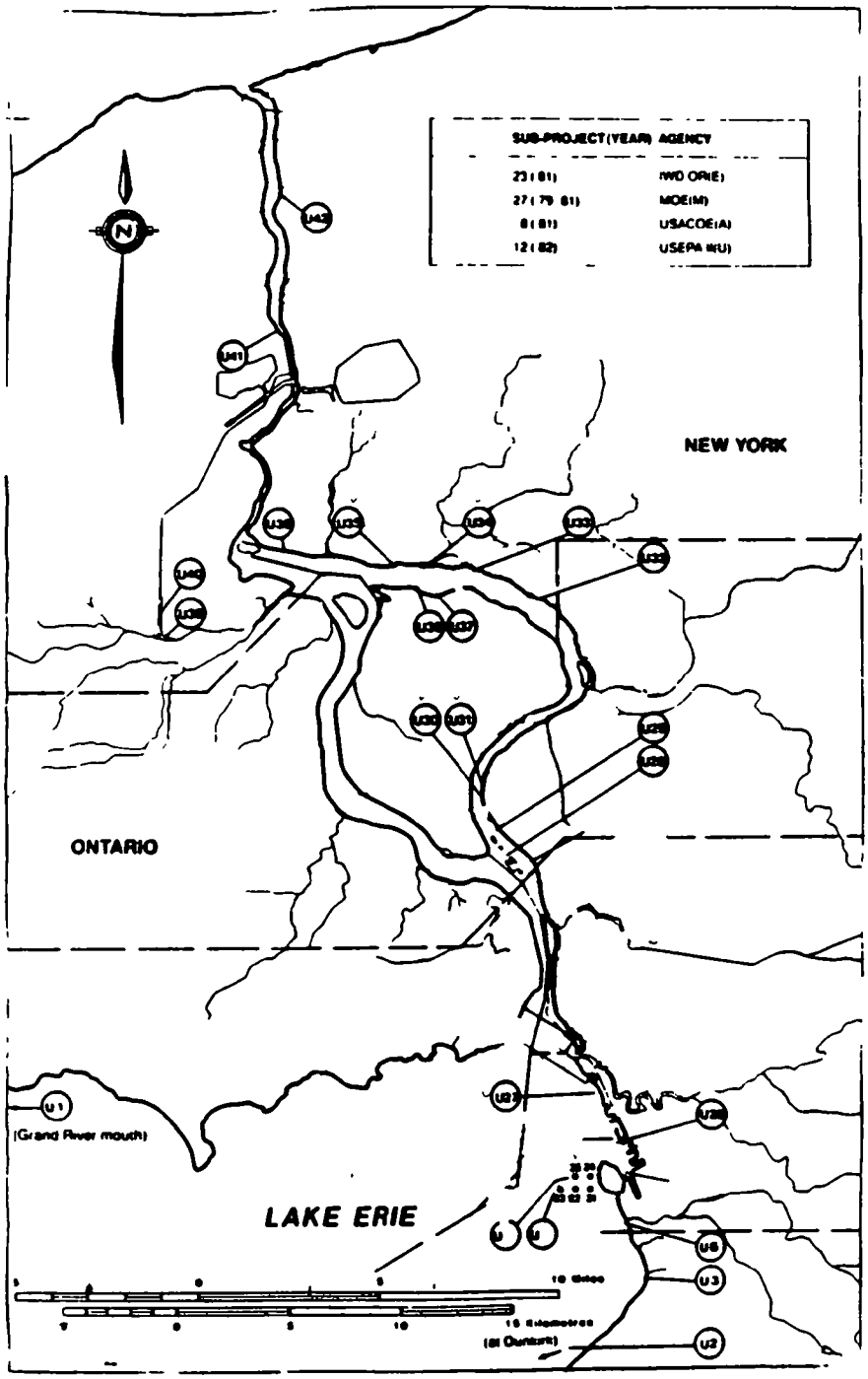

Figure 3-14 Map of Niagara River System Showing Sediment Sampling Sites in a Study Conducted by EPA Region II in 1982(U). (Source: NRTC 1984)

3.11 NEW YORK AND PENNSYLVANIA REGION: LAKE ONTARIO AND LAKE ERIE HARBORS AND TRIBUTARIES IN NEW YORK STATE (EXCLUDING BUFFALO) AND PENNSYLVANIA

The New York State and Pennsylvania region is divided into the following three subregions: Rochester (Genesee River); Oswego (Wine Creek) and Olcott (18 Mile Creek); and Dunkirk and Erie.

Sediment sampling sites for PAH sediment concentration data in the New York State and Pennsylvania region are described in Appendix B, Table B-10A. All of the Rochester, Oswego and Olcott sites were sampled by GLNPO in 1981 (USEPA 1984c). All of the Dunkirk and Erie sites were sampled by GLNPO in 1982 (USEPA 1984c).

PAH sediment concentration data for the New York State and Pennsylvania region are listed in Appendix B, Table B-10B. Human carcinogenic (x 10^4), human non-carcinogenic (x 10^2), and acute aquatic life (x 10^3) risk scores associated with the PAH sediment concentration data for this region are listed in Appendix B, Tables B-10C, B-10D, and B-10E, respectively.

4. RANKING OF REGIONS, SUBREGIONS WITHIN REGIONS, AND SAMPLING SITES AND PAHS WITHIN SUBREGIONS

Chapter 4 presents equations for computing mean PAH sediment concentrations for all PAHs combined, mean human carcinogenic risk scores, mean human non-carcinogenic risk scores and mean acute aquatic toxicity risk scores for regions, subregions within regions, and sampling sites within subregions. Mean sediment concentrations and risk scores are also computed for specific PAHs within subregions. Rankings of regions, subregions within regions, and sampling sites and PAHs within subregions are presented in Appendix C. The rankings are presented in order of decreasing mean PAH sediment concentration, mean human carcinogenic risk scores, mean human non-carcinogenic risk scores, and mean acute aquatic toxicity risk scores.

Sufficient data are presently unavailable to support derivation of precise estimates of risks to aquatic life and human health from PAHs in sediments. Although estimates of such risks are developed in this report, these estimates are probably several orders of magnitude higher than actual risks and are presented only for the purposes of ranking and cross-comparison of sampling sites (see also Section 1.1 and 3.1).

4.1 EQUATIONS USED FOR COMPUTING MEAN PAH SEDIMENT CONCENTRATIONS, MEAN HUMAN CARCINOGENIC RISK SCORES, MEAN HUMAN NON-CARCINOGENIC RISK SCORES AND ACUTE AQUATIC TOXICITY RISK SCORES

The general equation that was used to compute the mean PAH sediment concentration, mean human carcinogenic risk score, mean human non-carcinogenic risk score, and mean acute aquatic toxicity risk score over all PAHs, sampling sites and subregions for a given region R is given by:

$$\bar{X}_{ijk} = \frac{\sum_k \sum_j \sum_i X_{ijk}}{N_R} \qquad (4\text{-}1)$$

where

$\bar{X}_{ijk}$ = mean PAH sediment concentration ($\bar{C}_{ijk}$), or mean human carcinogenic risk score ($\bar{\bar{X}}\bar{C}_{ijk}$) or mean human non-carcinogenic risk score ($\bar{\bar{X}}\bar{N}_{ijk}$) or mean acute aquatic toxicity risk score ($\bar{Y}_{ijk}$) over all PAHs, sampling sites, and subregions for a given region

X_{ijk} = PAH sediment concentration (C_{ijk}) or human carcinogenic risk score (XC_{ijk}) or human non-carcinogenic risk score (XN_{ijk}) or acute aquatic toxicity risk score (Y_{ijk}) for PAH j at sampling site i within subregion k

N_R = total number of PAH data for region R if computing $\bar{C}_{ijk}$ or $\bar{X}_{ijK}$; or total number of carcinogenic PAH data for region R if computing $\bar{\bar{XC}}_{ijk}$; or total number of non-carcinogenic PAH data for region R if computing $\bar{\bar{XN}}_{ijk}$.

In calculating $\bar{X}_{ijk}$, X_{ijk} was set equal to zero for non-detected PAHs and the non-detected PAHs were included in computing N_R.

The general equation that was used to compute the mean PAH sediment concentration, mean human carcinogenic risk score, mean human non-carcinogenic risk score or mean acute aquatic toxicity risk score over all PAHs j and sampling sites i for a given subregion k=K of region R is given by

$$\bar{X}_{ijK} = \frac{\sum_j \sum_i X_{ijK}}{N_{RK}} \qquad (4\text{-}2)$$

where

$\bar{X}_{ijK}$ = mean PAH sediment concentration ($\bar{C}_{ijK}$), or mean human carcinogenic risk score ($\bar{\bar{XC}}_{ijK}$) or mean human non-carcinogenic risk score ($\bar{\bar{XN}}_{ijk}$) or mean acute aquatic toxicity risk score ($\bar{Y}_{ijK}$) over all PAHs j and sampling sites i for a given subregion k=K of region R

X_{ijK} = PAH sediment concentration (C_{ijK}) or human carcinogenic risk score (XC_{ijK}) or human non-carcinogenic risk score (XN_{ijK}) or acute aquatic toxicity risk score (Y_{ijK}) for PAH j at sampling site i within subregion k=K

N_{RK} = total number of PAH data over the given subregion k=K of region R if computing $\bar{C}_{ijK}$ or $\bar{Y}_{ijK}$; or total number of carcinogenic PAH data over the given subregion k=K of region R if computing $\bar{\bar{XC}}_{ijK}$; or total number of non-carcinogenic PAH data over the given subregion k=K of region R if computing $\bar{\bar{XN}}_{ijK}$.

The general equation that was used to compute the mean PAH sediment concentration, mean human carcinogenic risk score, mean human non-carcinogenic risk score or mean acute aquatic toxicity risk score over all PAHs j for a given sampling site i=I within a given subregion k=K of region R is given by:

$$\bar{X}_{IjK} = \frac{\sum_{J} X_{IjK}}{N_{RKI}} \qquad (4\text{-}3)$$

where

$\bar{X}_{IjK}$ = mean PAH sediment concentration ($\bar{C}_{i=I,j,k=K}$) or mean human carcinogenic risk score ($\bar{X}_{IjK}$) or mean human non-carcinogenic risk score ($\bar{\bar{X}}\bar{N}_{IjK}$) or mean acute aquatic toxicity risk score ($\bar{Y}_{IjK}$) for all PAHs j over a given sampling site i=I within subregion k=K or region R

X_{IjK} = PAH sediment concentration (C_{IjK}) or human carcinogenic risk score (XC_{IjK}) or human non-carcinogenic risk score (XN_{IjK}) or acute aquatic toxicity risk score (Y_{IjK}) for PAH j at the given sampling site i=I within the given subregion k=K of region R

N_{RKI} = total number of PAH data points over the given sampling site i=I within the given subregion k=K of region R if computing $\bar{C}_{IjK}$ or $\bar{Y}_{IjK}$; or total number of carcinogenic PAH data over the given sampling site i=I within the given subregion k=K of region R if computing $\bar{\bar{X}}\bar{C}_{IjK}$; or total number of non-carcinogenic PAH data over the given sampling site i=I within the given subregion k=K of region R if computing $\bar{\bar{X}}\bar{N}_{IjK}$.

The general equation that was used to compute the mean PAH sediment concentration, mean human carcinogenic risk score, human non-carcinogenic risk score or mean acute aquatic toxicity risk score over all sampling sites i for a given PAH j=J within a given subregion k=K of region R is given by:

$$\bar{X}_{iJK} = \frac{\sum_{I} X_{iJK}}{N_{RJK}} \qquad (4\text{-}4)$$

where

$\bar{X}_{iJK}$ = mean PAH sediment concentration ($\bar{C}_{iJK}$) or mean human carcinogenic risk score ($\bar{\bar{X}}\bar{C}_{iJK}$) or mean human non-carcinogenic risk score ($\bar{\bar{X}}\bar{N}$

$_{iJK}$) or mean acute aquatic toxicity risk score ($\bar{Y}_{iJK}$) over all sampling sites i for a given PAH j=J within a given subregion k=K of region R

X_{iJK} = PAH sediment concentration (C_{iJK}), human carcinogenic risk score (XC_{iJK}), human non-carcinogenic risk score (XN_{iJK}) or acute aquatic toxicity risk (Y_{iJK}) for a given PAH j=J at sampling site i within a given subregion k=K of region R

N_{RKJ} = total number of the given PAH j=J data over all sampling sites i within the given subregion k=K of region R.

4.2 RANKINGS

Regions, subregions within regions, sampling sites within subregions and PAHs within subregions are ranked in decreasing order of mean PAH sediment concentration, mean human carcinogenic risk score, mean human non-carcinogenic risk score and mean acute aquatic toxicity risk score in Appendix C, Tables 1, 2, 3 and 4, respectively. The rankings are discussed in Chapter 5.

5. DISCUSSION AND SUMMARY OF THE PAH SEDIMENT CONCENTRATION DATA AND ASSOCIATED RISK SCORES AND RANKINGS

This chapter presents a summary and discussion of the PAH sediment concentrations and associated risk scores and ratings which are presented in Chapters 3 and 4. The 10 sections of Chapter 5 each address one of the 10 regions. The regions are discussed in order of increasing overall ranking score. Subregions within a region and sampling sites within a subregion are also discussed in order of increasing overall ranking scores.

The overall ranking score of a given region, subregion, or sampling site is equal to the sum of its rankings with respect to the highest mean PAH sediment concentration, the highest mean human carcinogenic risk score, the highest mean human non-carcinogenic risk score, and the highest mean acute aquatic toxicity risk score.

5.1 INDIANA REGION

Of the 10 regions discussed in this report, the Indiana region ranks first with respect to mean PAH sediment concentration (51.5 mg/ kg), mean human carcinogenic risk score (1.22×10^{-4}), mean human non-carcinogenic risk score (0.686), mean acute aquatic toxicity risk score (9.81×10^{-2}), and overall ranking score (sum of regional rankings = 1 + 1 + 1 + 1 = 4). The Indiana region is a heavily industrialized steel producing area with numerous outfalls from various steel industry processes located along the East Branch of the Grand Calumet River, the Indiana Harbor Canal, and Indiana Harbor.

Table 5-1 lists mean PAH sediment concentrations (mg/kg), mean human carcinogenic risk scores ($\times 10^4$), mean human non-carcinogenic risk scores ($\times 10^2$), mean acute aquatic toxicity risk scores ($\times 10^3$), overall ranking scores and associated rankings (in parenthesis) for subregions and sediment sampling sites in the Indiana region. The subregions and sampling sites within subregions are listed in the order of increasing overall ranking score. Descriptions of the sediment sampling sites in the Indiana region were presented in Appendix B, Table B-1A and cross-referenced to Figures 3-2 and 3-3. Potentially significant point sources of pollution were presented in Appendix B, Table B-1B, and cross-referenced to Figure 3-2. Maps of the

Table 5-1. Summary of Mean PAH Sediment Concentrations, Mean Risk Scores, and Rankings for Subregions and Sampling Sites in the Indiana Region

Region, subregion, site	River mile	Sediment concentration (mg/kg) and (ranking)	Carcinogenic risk score ($\times 10^4$) and (ranking)	Non-carcinogenic risk score ($\times 10^2$) and (ranking)	Acute aquatic toxicity risk score ($\times 10^3$) and (ranking)	Overall ranking score and (ranking)
Indiana Region	--	**51.5 (1)**	**1.22 (1)**	**68.6 (1)**	**98.1 (1)**	**4 (1)**
Grand Calumet River Subregion	--	88.6 (1)	2.02 (1)	150 (1)	75.7 (2)	5 (1)
6975	12.7	960 (3)	23.2 (1)	1,700 (3)	647 (3)	10 (1)
4560	13.1	1,030 (2)	4.03 (5)	2,200 (2)	840 (2)	11 (2)
10800	12	2,650 (1)	0.31 (13)	5,840 (1)	2,220 (1)	16 (3)
3300	13.4	150 (4)	12.2 (2)	110 (6)	42.1 (7)	19 (4)
C3	10	97.3 (6)	6.36 (3)	110 (7)	94.2 (5)	21 (5)
12900	11.5	133 (5)	0.31 (14)	286 (4)	109 (4)	27 (6)
26900	8.9	84.8 (7)	0.88 (10)	171 (5)	65.1 (6)	28 (7)
C4	8.6	15.1 (12)	1.02 (9)	17.0 (9)	15.7 (9)	39 (8)
C7	4.6W	14.8 (13)	1.09 (8)	15.0 (10)	10.1 (10)	41 (9)
C5	6.8	16.5 (11)	1.73 (7)	10.4 (13)	9.11 (11)	42 (10)
100	14	22.2 (10)	0.26 (16)	44.1 (8)	16.8 (8)	42 (11)
8700	11.4	77.0 (8)	4.70 (4)	0.00 (17)	0.02 (17)	46 (12)
C2	11	10.4 (14)	0.49 (11)	13.5 (11)	8.15 (12)	48 (13)
500	13.9	39.0 (9)	2.38 (6)	0.00 (18)	0.01 (18)	51 (14)
35600	6.7	7.75 (15)	0.27 (15)	12.1 (12)	4.62 (13)	55 (15)
C9	6.9W	0.66 (17)	0.04 (17)	0.68 (14)	0.48 (14)	62 (16)
2100	13.6	7.00 (16)	0.43 (12)	0.00 (19)	0.00 (19)	66 (17)
S01	>14	0.28 (18)	0.02 (18)	0.38 (15)	0.16 (15)	66 (18)
S04	5.5W	0.21 (19)	0.01 (19)	0.19 (16)	0.05 (16)	70 (19)

Table 5-1. Summary of Mean PAH Sediment Concentrations, Mean Risk Scores, and Rankings for Subregions and Sampling Sites in the Indiana Region
(continued)

Region, subregion, site	River mile	Sediment concentration (mg/kg) and (ranking)	Carcinogenic risk score ($\times 10^4$) and (ranking)	Non-carcinogenic risk score ($\times 10^2$) and (ranking)	Acute aquatic toxicity risk score ($\times 10^3$) and (ranking)	Overall ranking score and (ranking)
Indiana Harbor Subregion	--	61.4 (2)	1.36 (2)	31.1 (2)	98.1 (1)	7 (2)
ACOE	--	226 (1)	4.95 (1)	114 (1)	1,420 (1)	4 (1)
LTI5-1	--	3.55 (2)	0.20 (2)	3.08 (2)	2.34 (2)	8 (2)
S11	--	0.88 (3)	0.03 (3)	0.80 (3)	1.27 (3)	12 (3)
LTI2	--	0.01 (4)	0.00 (4)	0.01 (4)	0.01 (4)	16 (4)
Indiana Harbor Canal Subregion	--	8.40 (3)	0.43 (3)	9.16 (3)	5.07 (3)	12 (3)
Columbus Drive	2.5	28.7 (2)	3.05 (1)	23.0 (3)	12.8 (3)	9 (1)
Canal Street	1.2	15.3 (4)	2.32 (2)	10.9 (5)	13.5 (2)	13 (2)
Indianapolis Blvd.	--	55.3 (1)	0.00 (12)	57.4 (1)	33.5 (1)	15 (3)
Forks	2.1	22.3 (3)	0.28 (8)	24.1 (2)	10.1 (5)	18 (4)
S08	1.8	8.27 (5)	0.28 (7)	11.0 (4)	10.7 (4)	20 (5)
S09	0.0	7.17 (6)	0.41 (5)	7.46 (6)	2.43 (7)	24 (6)
C16	2.0	6.00 (8)	0.45 (4)	5.52 (8)	3.53 (6)	26 (7)
S13	--	6.03 (7)	0.58 (3)	4.37 (9)	1.65 (11)	30 (9)
S14	--	5.43 (9)	0.34 (6)	5.73 (7)	2.19 (9)	31 (8)
C14	2.5	2.94 (11)	0.22 (10)	2.53 (10)	2.22 (8)	39 (10)
C13	3.2	3.10 (10)	0.23 (9)	2.41 (11)	2.09 (10)	40 (11)
C17	1.2	1.01 (12)	0.08 (11)	0.82 (12)	0.49 (12)	41 (12)

Indiana region showing river miles, general direction of flow, locations of potentially significant pollution point sources and locations of sampling sites were presented in Figures 3-1, 3-2, and 3-3, respectively.

5.1.1 Grand Calumet River Subregion

Of the three reported subregions in the Indiana region, the Grand Calumet River subregion ranks first with respect to mean PAH sediment concentration (88.6 mg/kg), mean human carcinogenic risk score (2.02×10^{-4}), mean human non-carcinogenic risk score (1.50), and overall ranking score (sum of subregional rankings = 1 + 1 + 1 + 2 = 5). It ranks second with respect to mean acute aquatic toxicity risk score (7.56×10^{-2}).

Of the 19 reported sediment sampling sites in the Grand Calumet River subregion, the 1st through 4th ranking sites (with respect to the smallest overall ranking score) are sites 6975, 4560, 10800, and 3300, which are approximately located at river miles 12.7, 13.1, 12, and 13.4, respectively. U.S. Steel coke plant outfalls 005, 007, 010, sintering plant outfall 015, and blast furnace outfall 017 are located at river miles 13.5, 13.3, 13.1, 12.9, and 12.9, respectively. Therefore, the four highest ranking sampling sites in the highest ranking subregion of the highest ranking region are located at or just below coke plant, sintering plant, and blast furnace outfalls.

The mean PAH sediment concentrations for the 4 highest ranking sites are 2650, 1030, 960, and 150 mg/kg, respectively. However, all four of the highest ranking sites were sampled in 1972 and, therefore, may not reflect current conditions. Of the five U.S. Steel outfalls listed above, only the blast furnace outfall 017 was described as discharging any process wastewater in 1983 (HydroQual 1984). The other four outfalls were described as discharging only cooling (non-contact) water.

The 5th through 8th ranking sites in the Grand Calumet River subregion with respect to overall ranking score are C3, 12900, 26900 and C4, which are approximately located at river miles 10, 11.5, 8.8, and 8.6, respectively. These sites are at or within two miles downstream of U.S. Steel outfalls 028, 030, 032, 033, and 034, which are located at river miles 11.8, 11.6, 11.5, 11.3, and 9.2, respectively. Discharges from these outfalls in 1983 included

cooling and/or process water from bar plate mills, basic oxygen steel making furnaces, an atmospheric gas plant, a treatment plant, miscellaneous finishing operations, and hot strip mill recycling processes (HydroQual 1984). Sites 26900 (river mile 8.8) and C4 (river mile 8.6) are also at and just below the Gary POTW outfall at river mile 8.8. The mean PAH sediment concentrations for the 5th through 8th ranking sites range from 15.1 to 132 mg/kg.

The 9th through 14th ranking sites in the Grand Calumet River subregion, with respect to overall ranking score, are C7 (river mile 4.6W), C5 (river mile 6.8), 100 (river mile 14), 8700 (river mile 11.4), C2 (river mile 11), 500 (river mile 13.9), and 35600 (river mile 6.67). The mean PAH sediment concentrations for those sites range from 7.75 to 77.0 mg/kg. Site C7 (river mile 4.6W) is located at the East Chicago POTW outfall (river mile 4.6W) in the West Branch of the Grand Calumet River. Sites C5 (river mile 6.8) and 35600 (river mile 6.7) are located at and just below the Vulcan outfall (river mile 6.8). Sites 100 (river mile 14) and 500 (river mile 13.9) are located a short distance upstream of the first U.S. Steel outfalls 002 and 005 (at river mile 13.5) and are not located downstream of any industrial point source dischargers. Therefore, the moderately high PAH levels at those sites may be due to some limited mixing and dispersion upstream of the U.S. Steel outfalls. Site 8700 (river mile 11.4) is in the vicinity of U.S. Steel outfalls 028 (river mile 11.8), 030 (river mile 11.6), and 032 (river mile 11.3) described previously. Although the mean PAH concentration at site 8700 is comparable to that of site 12900 (river mile 11.5), its overall ranking is much lower due to low rankings with respect to the human non-carcinogenic risk scores (0.0). However, the human non-carcinogenic risk score is 0.0 only because no non-carcinogens were analyzed for at this site. The same is true for sites 500 and 2100. Therefore, the rankings of those sites are somewhat lower than they would be if they had been analyzed for non-carcinogenic PAHs.

The lowest ranking sites (16th through 19th) in the Grand Calumet River subregion, with respect to overall ranking score, are C9 (river mile 7W), 2100 (river mile 13.6), S01 (river mile >14), and S04 (river mile 5.5W), respectively. Site C9 (river mile 7W) is located downstream of the Federal Cement outfall (river mile 6.4W) in the West Branch of the Grand Calumet River. Site S04 (river mile 5.5W) is also located in the West Branch and is

at the Hammond POTW outfall (river mile 5.5W). Sites 2100 (river mile 13.6) and S01 (river mile >14) are located upstream of U.S. Steel outfalls 002 and 005 at river mile 13.5 and are also located upstream of any known industrial point discharge. The low ranking of site 2100 can be partially attributed to the fact that samples from this site were not analyzed for non-carcinogenic PAHs (which resulted in a non-carcinogenic risk score of 0.0). However, the carcinogenic risk score (7.0×10^{-4}) for site 2100 also ranked low (16th). The 15th, 17th, and 18th ranking sites all have mean PAH sediment concentrations less than 1 mg/kg.

The rankings of the sampling sites in the Grand Calumet River subregion can be summarized as follows. The four highest ranking sites, with respect to overall ranking score, are located between river miles 12 and 13.4 in the vicinity of several U.S. Steel coke plant, sintering plant, and blast furnace outfalls. However, data for the four highest ranking sites were reported in 1972 and may not reflect current conditions. The 5th through 8th ranking sites are located between river mile 8.6 and 11.5, at or downstream of several other types of U.S. Steel outfalls and/or the Gary POTW outfall. The 9th through 15th ranking sites are located throughout the Grand Calumet River subregion. Two of the four lowest ranking sites are located in the West Branch of the Grand Calumet River. The other two lowest ranking sites are located upstream of any known industrial point discharger.

5.1.2 Indiana Harbor Subregion

Of the three reported subregions in the Indiana region, the Indiana Harbor subregion ranks first with respect to mean acute aquatic toxicity risk score (9.81×10^{-3}) and second with respect to mean PAH sediment concentration (61.4 mg/kg), mean human carcinogenic risk score (1.36×10^{-4}), mean human non-carcinogenic risk score (0.311), and overall ranking score (2 + 2 + 2 + 1 = 7).

Of the four reported sampling sites in the Indiana Harbor subregion, the highest ranking site with respect to overall ranking score is site ACOE. Site ACOE has by far the highest mean PAH concentration (226 mg/kg) and associated risk scores of the four sampled sites in Indiana Harbor and has the fourth highest mean PAH concentration of all the sites in the Indiana region. Unfor-

tunately, the exact location of site ACOE within Indiana Harbor was not specified (ACOE 1985).

The second highest ranking site in the Indiana Harbor subregion is LTI 5-1, which is located west of the entrance to the Canal in the vicinity of several Inland Steel outfalls including 012, 013, 014, and 015. Discharges from those outfalls include blast furnace blow downs, coke plant cooling water, hearth furnace cooling water, and treated blast furnace process water. Nevertheless, the mean PAH concentration at site LTI 5-1 (3.55) would rank only 17th in the Grand Calumet River subregion. The lowest ranking sites in the Indiana Harbor are S11 and LTI 2, respectively, both of which are located at the entrance to Indiana Harbor at Lake Michigan. Both sites have mean PAH sediment concentrations less than 1 mg/kg.

The ranking of the Indiana Harbor subregion ahead of the Indiana Harbor Canal subregion is due to the mean PAH sediment concentration and associated risk scores at only one site, whose location is not specified. The mean PAH concentrations and associated risk scores of the other three reported sites in the Indiana Harbor subregion are lower than for most of the other reported sites in both the Grand Calumet River and Indiana Harbor subregions. Although the number of sites for which data are reported is too low to support definite conclusions, the relatively low mean PAH sediment concentrations at the two sites at the entrance to the harbor at Lake Michigan may indicate that very little of the PAH contamination from the Grand Calumet River, Indiana Harbor Canal, and Indiana Harbor subregions is entering Lake Michigan via the water column.

5.1.2 Indiana Harbor Canal Subregion

Of the three reported subregions in the Indiana region, the Indiana Harbor Canal subregion ranks third with respect to mean PAH sediment concentration (8.40 mg/kg), mean carcinogenic risk score (4.3×10^{-5}), mean noncarcinogenic risk score (0.092), mean acute aquatic toxicity risk score (5.07×10^{-3}), and overall ranking score (3 + 3 + 3 + 3 = 12). However, as previously discussed, the mean PAH sediment concentration and associated risk scores for most of the 12 reported sites in the Indiana Harbor Canal subregion are substantially greater than those for three of the four reported sites in the Indiana Harbor subregion.

Of the 12 reported sampling sites in the Indiana Harbor Canal subregion, the four highest ranking sites, with respect to overall ranking score, are Columbus Drive (river mile 2.5 or 2.6), Canal Street (between river miles 1 and 2), Indianapolis Boulevard (intersects Lake George Branch), and the Forks (fork between Lake George and Grand Calumet branches of Indiana Harbor Canal at river mile 2.1), respectively. The mean PAH sediment concentrations for those sites are 28.7, 16.3, 55.3, and 22.3 mg/kg, respectively. However, all of the four highest ranking sites were sampled in 1977 and, therefore, may not reflect current conditions.

The sampling site at Columbus Drive (river mile 2.5-2.6), which is the highest ranking site in the Indiana Harbor Canal subregion, is downstream of the Blau-Knox Foundry (river mile 3.3) and Union Carbide (river mile 2.8) outfalls. It is also in the vicinity of the Phillips Pipeline Company outfall (river mile 2.5). The second highest ranking site in the Indiana Harbor Canal subregion is at Canal Street (between river miles 1 an 2). The sampling site at Canal Street is located in the vicinity of a storm sewer overflow, combined sewer overflow, and the American Steel Foundry outfall (river mile 1.6) and is downstream of J & L Steel outfall 001 (river mile 2.1), which discharges process water from flat roll operations. The third highest ranking site is in the Lake George Branch of the Indiana Harbor Canal at Indianapolis Boulevard, downstream of the CF Petroleum outfall. The fourth highest ranking site is at the fork between the Lake George and Grand Calumet branches of the Indiana Harbor Canal, in the vicinity of the J & L Steel outfall 001 described above.

Some of the PAH data for the Indiana Harbor Canal subregion indicate that there may have been some decreases in the PAH levels in sediments since 1977 when the four highest ranking sites in the Indiana Harbor Canal subregion were sampled. For example, the 5th and 7th ranking sites are S08 (river mile 1.8) and C16 (river mile 2.0), which have mean PAH sediment concentrations of 8.27 and 6.00 mg/kg, respectively. They are located between the 4th ranking Forks and the 2nd ranking Canal Street sampling sites, which have mean PAH sediment concentrations of 22.3 and 16.3 mg/kg, respectively. Sampling site S14, which ranks 9th and has a mean PAH sediment concentration of 5.43 mg/kg, is at the same approximate location as the sampling site at Indianapolis Boulevard, which ranks 3rd and has a mean PAH sediment concentration of 55.3 mg/ kg.

Sampling site C14, which ranks 10th and has a mean PAH sediment concentration of 2.94 mg/kg, is at the same approximate location as the Columbus Drive sampling site, which ranks 1st and has a mean PAH sediment concentration of 28.7 mg/kg. In each case, sampling sites which were sampled in 1980 (S08, S14) and in 1983 (C16, C14) had substantially lower mean PAH sediment concentrations and associated risk scores than sampling sites in similar locations which were sampled in 1977 (the four highest ranking sites).

The 6th ranking site in the Indiana Harbor Canal subregion is S09 (river mile 0.0) which is located at the mouth of the Indiana Harbor Canal and is downstream of several steel industry outfalls. The 9th ranking site is S14, which is located in the Lake George Branch upstream of the CF Petroleum outfall. The two lowest ranking sites (11th and 12th) are C13 (river mile 3.2) and C17 (river mile 1.2). Site C13 is located downstream of the Blau Knox Foundry outfall (river mile 3.3). Site C17 is located downstream of several steel industry outfalls, but has a mean PAH sediment concentration of only 1.02 mg/kg. All of the other sites in the Indiana Harbor Canal subregion have mean PAH sediment concentrations greater then 1 mg/kg.

In summary, the four highest ranking sites in the Indiana Harbor Canal subregion are all located within one mile of the fork where the Indiana Harbor Canal divides into the Lake George and Grand Calumet branches (river mile 2.1). Furthermore, each of the three segments of the Indiana Harbor Canal is represented by one of the four highest ranking sites, with one site located at the Fork, one site in the Lake George Branch, one site in the Grand Calumet Branch, and one site in the Indiana Harbor Canal downstream of the Fork. However, all of the four highest ranking sites were sampled in 1977. PAH levels in sediment samples taken at similar sites in 1980 and 1983 were much lower, even though the U.S. Steel Plant is still operating. Therefore, there may have been substantial reductions in the PAH levels present in the effluents since 1977.

5.2 OHIO STEEL REGION

Of the 10 regions discussed in this report, the Ohio Steel region ranks second with respect to mean PAH sediment concentration (11.8 mg/kg), third with respect to mean human carcinogenic risk score (5.5×10^{-3}), second with

respect to mean human non-carcinogenic risk score (0.152), third with respect to mean acute aquatic toxicity risk score, and second with respect to overall ranking score (2 + 3 + 2 + 3 = 10).

The Ohio Steel region is divided into the Black River/Lorain and Mahoning River subregions. The Ohio Steel region was separated from the Ohio region because most of the PAH data in the Black River/Lorain subregion and all of the PAH data in the Mahoning River subregion are for sites in the vicinity of steel industry outfalls. Some of the PAH data in the Cuyahoga River subregion of the Ohio region were also for sites located in the vicinity of steel industry outfalls. Therefore, the Cuyahoga subregion, which is the highest ranking subregion of the Ohio region, might alternately be considered with the Ohio Steel region.

Table 5-2A lists mean PAH sediment concentrations (mg/kg), mean human carcinogenic risk scores (x 10^4), mean human non-carcinogenic risk scores (x 10^2), mean acute aquatic toxicity risk scores (x 10^3), overall ranking scores and associated rankings (in parenthesis) for subregions and sampling sites in the Ohio Steel region. The subregions and sampling sites within subregions are listed in order of increasing overall ranking score. Descriptions of the sediment sampling sites in the Ohio Steel region were presented in Appendix B, Table B-4A. Maps of the Black River/Lorain subregion showing the locations of sampling sites were presented in Figures 3-6 and 3-7.

5.2.1 Black River Subregion

Of the two reported subregions in the Ohio Steel region, the Black River/Lorain subregion ranks first, with respect to mean sediment concentration (11.9 mg/kg), mean carcinogenic risk score (5.5 x 10^{-5}), mean non-carcinogenic risk score (0.155), and overall ranking score (1 + 1 + 1 + 2 = 5). It ranks second with respect to mean aquatic toxicity risk score (7.39 x 10^{-3}).

Table 5-2A. Summary of Mean PAH Sediment Concentrations, Mean Risk Scores, and Rankings for Subregions and Sampling Sites in the Ohio Steel Region

Region, subregion, site	Sediment concentration (mg/kg) and (ranking)	Carcinogenic risk score ($\times 10^4$) and (ranking)	Non-carcinogenic risk score ($\times 10^2$) and (ranking)	Acute aquatic toxicity risk score ($\times 10^3$) and (ranking)	Overall ranking score and (ranking)
Ohio Steel Region	**11.8 (2)**	**0.55 (3)**	**15.2 (2)**	**7.92 (3)**	**10 (2)**
Black River/Lorain Subregion	11.9 (1)	0.55 (1)	15.5 (1)	7.39 (2)	5 (1)
B-2	101 (1)	1.90 (2)	197 (1)	50.0 (1)	5 (1)
LOR82-07	41.0 (2)	4.44 (1)	25.7 (3)	27.1 (2)	8 (2)
USS-4	21.0 (3)	0.78 (4)	35.2 (2)	24.7 (3)	12 (3)
19472(8B)	14.9 (4)	0.96 (3)	16.4 (4)	3.20 (6)	17 (4)
USS-1	9.39 (5)	0.41 (6)	13.8 (5)	6.95 (4)	19 (5)
B-1	8.75 (6)	0.26 (8)	12.0 (6)	3.00 (7)	25 (6)
LOR82-10	3.84 (8)	0.35 (7)	2.86 (10)	4.65 (5)	30 (7)
LOR82-13	3.86 (7)	0.54 (5)	1.94 (13)	1.77 (8)	33 (8)
19470(6B)	2.37 (10)	0.13 (12)	3.61 (8)	0.75 (12)	37 (9)
19469(5C)	3.11 (9)	0.16 (11)	3.83 (7)	0.72 (13)	40 (10)
LOR82-15	2.28 (11)	0.23 (9)	1.44 (14)	1.16 (9)	43 (11)
USS-2	2.00 (13)	0.13 (13)	2.00 (12)	1.13 (10)	48 (12)
19474(10)	1.02 (15)	0.04 (18)	3.31 (9)	0.84 (11)	53 (13)
19467(3B)	1.67 (14)	0.07 (15)	2.74 (11)	0.60 (14)	54 (14)
19476(12)	0.82 (17)	0.06 (16)	0.69 (16)	0.14 (17)	57 (15)
19471(7B)	2.23 (12)	0.18 (10)	0.00 (20)	0.00 (20)	62 (16)
LOR82-18	0.99 (16)	0.10 (14)	0.67 (17)	0.27 (16)	63 (17)
USS-3	1.57 (19)	0.02 (19)	0.81 (15)	0.28 (15)	64 (18)
19466(2)	0.59 (18)	0.04 (17)	0.47 (18)	0.09 (19)	72 (19)
USS-5	0.15 (20)	0.00 (20)	0.25 (19)	0.11 (18)	77 (20)
Mahoning River Subregion	10.8 (3)	0.00[a] (2)	12.8 (2)	14.5 (1)	7 (2)
Struthers	16.1 (1)	0.00[a] (1)	20.9 (1)	23.7 (1)	4 (1)
Youngstown	15.9 (2)	0.00[a] (1)	17.4 (2)	19.4 (2)	7 (2)
Warren	0.05 (3)	0.00[a] (1)	0.0 (3)	0.44 (3)	10 (3)

[a] No analysis for carcinogenic PAHs were performed on samples taken from the Mahoning River.

Of the 20 reported sediment sampling sites in the Black River/Lorain subregion, the five highest ranking sites, with respect to overall ranking score, are B-2, LOR82-07, USS-4, 19472(8B), and USS-1, respectively. Those sites have mean PAH sediment concentrations of 102, 41.0, 21.0, 14.9, and 9.39 mg/kg, respectively. Sites B-2 and USS-4 are located at the U.S. Steel coke plant outfall 002. Site 19472(8B) is located approximately 500 feet downstream of U.S. Steel outfall 002.

Sites LOR87-02 and USS-1 appear to border the downstream end of a sediment dredging disposal site. Although sites LOR87-02 and USS-1 are only 0.5-0.7 miles downstream of U.S. Steel outfall 002, it appears that the main contribution of PAHs to those sites may be from the sediment dredging disposal site. Site USS-2, which is located approximately 0.2 miles closer to U.S. Steel outfall 002, but borders only the front edge of the sediment dredging, ranks 12th. Site USS-3, which is only 700 feet downstream of U.S. Steel outfall 002 (but does not border the sediment dredging waste pile) ranks 19th compared to 2nd for LOR82-07 and 3rd for USS-4.

The higher levels of PAHs at sites LOR82-07 and USS-1, compared to sites USS-2 and USS-3, may also be at least partly due to differences in PAH contaminated sediment deposition rates at these sites. Sites LOR82-07 and USS-1 are located at the head of the turning basin and therefore possibly in areas of reduced current and increased sediment deposition. However, it appears that a large proportion of the PAH discharged at U.S. Steel outfall 002 is deposited in the immediate vicinity of the outfall since sites B-2 and USS-4 (located at the outfall) rank 1st and 3rd, respectively, compared to the 19th ranking for site USS-3, which is located only 700 feet downstream of the outfall. The 20th and lowest ranking site is USS-5, which is located just above U.S. Steel outfall 002.

The location of the 6th ranking site (B-1), which has a mean PAH sediment concentration of 8.75 mg/kg, is not specified (Black et al. 1985). The 7th, 9th, and 10th ranking sites are LOR82-10, 19470(6B), and 19469 (5C), respectively, which are located at or within 0.5 miles downstream of U.S. Steel outfalls 003 and 004, which discharge to the turning basin. The 8th (LOR83-13) and 11th (LOR82-15) ranking sites are located in the vicinity of the Ashland Oil outfall and Lee's Marine Service, respectively. The 13th and 15th ranking

sites are 19474-10 and 19476-12, which are located 0.2 miles downstream and 0.2 miles upstream of U.S. Steel outfall 001, respectively. The 14th (19467-3B) and 19th (19466-2) ranking sites are located at the mouth of the Black River and just off the mouth of the Black River in Lake Erie, respectively. Those sites have mean PAH sediment concentrations of 1.67 and 0.59 mg/kg, respectively. Therefore, it does not appear that very much of the PAH discharged into the Black River reaches Lake Erie. The 15th, 17th, 19th, and 20th ranking sites all have mean PAH sediment concentrations less than one mg/kg.

The PAH sediment concentrations listed for various PAHs and sampling sites USS-1 through USS-5 in Table 5-2B are actually each means of PAH concentrations in four samples taken at equally spaced locations running across and perpendicular to the river flow at each site (ERG 1984). The PAH concentrations for each PAH and each of the four transverse locations at each of the five sampling sites USS-1 through USS-5 are listed in Table 5-2B, along with the mean PAH concentrations for each site. As can be seen from Table 5-1, the PAH concentrations in samples taken from different transverse locations at the same longitudinal sampling site vary widely. For example, PAH concentrations in the sample taken from location 1C are much greater than those for samples taken at locations 1A, 1B, and 1D, even though all four locations are at the same site (USS-1) from a longitudinal or river mile standpoint. Likewise, PAH concentrations in samples taken from locations 4A and 4D are much greater than for those taken at locations 4B and 4C. The large differences in PAH concentration in samples taken from different transverse locations at the same longitudinal site appear to be primarily due to differences in sediment deposition. The highest PAH concentrations at a given site were generally in samples taken from the transverse locations with the highest silt deposition (ERG 1984). The 7th through 14th, 16th and 18th ranking sites have mean PAH sediment concentrations ranging from 1.02 to 3.86 mg/kg.

The results of the ERG (1984) study indicate that care must be taken in sampling sediments for PAH contamination. PAH concentrations in sediments in the ERG (1984) study not only decreased rapidly with longitudinal distance from U.S. Steel outfall 002, but also varied widely between different transverse locations at the same longitudinal distances downstream. Therefore,

Table 5-28. The Dependence of PAH Sediment Concentrations on the Location of Sampling Transverse to the Direction of River Flow for Sampling Sites USS-1 through USS-5 in the Black River/Lorain Subregion

Sample	Acenaphthalene	Acenaphthylene	Anthracene	Benzo[a]anthracene	Benzo[a]pyrene	Benzo[b,k]fluoranthene	Benzo[g,h,i]perylene	Chrysene	Dibenzo[a,h]anthracene	Fluoranthene	Fluorene	Indeno(1,2,3-cd)pyrene	Phenanthrene	Pyrene
USS-1A	0.14	ND	0.16	0.14	0.18	0.19	0.14	0.16	ND	0.34	0.16	0.60	0.31	0.38
USS-1B	0.95	0.36	1.20	1.20	1.10	0.69	0.54	1.60	0.36	2.50	0.95	2.80	2.10	3.40
USS-1C	38.0	0.83	71.0	55.0	4.10	2.40	3.30	50.0	0.73	64.0	36.0	14.0	45.0	69.0
USS-1D	6.12	0.62	4.10	2.70	2.30	0.73	1.40	2.70	0.80	6.0	2.90	5.60	6.70	7.10
USS-1 mean	11 ± 18	0.45 ± 36	19 ± 35	15 ± 27	1.9 ± 1.7	1.0 ± 0.96	1.3 ± 1.4	14 ± 24	0.47 ± 0.37	18 ± 31	10 ± 17	5.8 ± 5.9	14 ± 21	20 ± 33
USS-2A	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.99
USS-2B	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
USS-2C	ND	ND	ND	0.15	ND	ND	ND	0.18	ND	ND	ND	ND	0.50	ND
USS-2D	9.10	ND	8.40	4.60	4.80	8.40	2.80	4.90	2.40	0.70	4.90	31.0	16.0	12.0
USS-2 mean	2.3 ± 4.6	ND	2.1 ± 4.2	1.2 ± 2.3	1.2 ± 2.4	2.1 ± 4.2	0.7 ± 1.4	1.3 ± 2.4	0.6 ± 1.2	0.18 ± 0.35	1.2 ± 2.4	7.8 ± 16	4.1 ± 7.9	3.2 ± 5.9
USS-3A	ND	ND	2.10	1.20	ND	1.0	ND	1.60	ND	3.40	1.10	ND	3.10	3.40
USS-3B	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.24	0.16
USS-3C	ND	ND	0.24	ND	ND	ND	ND	ND	ND	0.54	0.30	ND	0.98	ND
USS-3D	ND	ND	1.0	1.10	1.40	0.93	ND	1.20	ND	2.30	0.40	ND	1.30	2.80
USS-3 mean	ND	ND	0.84 ± 0.94	0.58 ± 0.67	0.35 ± 0.70	0.48 ± 0.56	ND	0.70 ± 0.82	ND	1.6 ± 1.6	0.45 ± 0.47	ND	1.4 ± 1.2	1.6 ± 1.8
USS-4A	39.0	1.90	23.0	12.0	10.0	6.90	3.50	ND	ND	41.0	18.0	24.0	46.0	31.0
USS-4B	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.85	ND	ND	0.44	0.54
USS-4C	ND	ND	0.85	0.40	ND	ND	ND	0.50	ND	1.50	ND	ND	1.30	ND
USS-4D	16.0	104	83.0	43.0	43.0	29.0	ND	55.0	11.0	123	120	84.0	198	1.90
USS-4 mean	14 ± 18	26 ± 52	27 ± 39	14 ± 20	13 ± 20	9.0 ± 14	0.88 ± 1.8	14 ± 27	2.8 ± 5.6	42 ± 57	35 ± 58	27 ± 40	61 ± 93	8.4 ± 15
USS-5A	ND	ND	0.55	0.50	ND	ND	ND	0.56	ND	ND	ND	ND	0.75	ND
USS-5B	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	5.60	ND
USS-5C	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.14	ND
USS-5D	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.15	ND
USS-5 mean	ND	ND	0.14 ± 0.28	0.13 ± 0.26	ND	ND	ND	0.14 ± 0.28	ND	ND	ND	ND	1.7 ± 2.6	ND

based on the results of the ERG (1984) study, it appears that sediment samples should be taken in the immediate vicinity of the outfalls of suspected PAH dischargers and at the first major sediment deposition area downstream. Also, samples should be taken at different transverse locations at the same longitudinal site and either analyzed separately or composited before analysis.

In summary, it appears that the major source of PAHs to the Black River/Lorain subregion was the U.S. Steel coke plant outfall 002. However, it appears that most of the PAH discharged at U.S. Steel outfall 002 is deposited in the immediate vicinity of the outfall (e.g., within 500 feet) and does not appear to affect Lake Erie. Other moderate sources of PAH to the Black River/Lorain subregion may include a sediment dredging disposal site, U.S. Steel outfalls 003 and/or 004, and the Ashland Oil outfall. The PAH concentrations in sediment samples taken in Lake Erie off the mouth of the Black River, at the mouth of the Black River, and upstream of U.S. Steel outfall 002 were relatively low (e.g., $\leq$ 1 mg/kg) compared to those in samples taken from most other sites in the Black River/Lorain subregion.

Sites 19466 through 19474 were sampled in 1974 and may not reflect current conditions. The U.S. Steel coke plant in Lorain has reportedly been shut down (Amendola 1985).

5.2.2 Mahoning River Subregion

Of the 2 reported subregions in the Ohio Steel region, the Mahoning River subregion ranks second (last) with respect to mean PAH sediment concentration (10.8 mg/kg), mean human carcinogenic risk score (0.0), mean non-carcinogenic risk score (0.128) and overall ranking score (2 + 2 + 2 + 1 = 7). It ranks first with respect to mean acute aquatic toxicity risk score (1.45×10^{-2}).

The computed mean carcinogenic risk score for the Mahoning River subregion is 0.0 because none of the 7 PAHs identified in the Mahoning River study are considered to be carcinogenic (USEPA 1977). However, other PAHs were present (but were not identified) which could possibly be carcinogenic.

Of the 3 reported sediment sampling sites in the Mahoning River subregion, the Struthers, Youngstown, and Warren sites rank first, second, and third, respectively, with respect to overall ranking score. The Struthers,

Youngstown, and Warren sampling sites are located in the Mahoning River below coke oven outfalls at the Youngstown Sheet and Tube-Campbell Works (Struthers), Republic (LTV) Steel/Youngstown Plant and the Republic (LTV) Steel/Warren Plant, respectively.

Although all 3 sites are located below coke oven outfalls, the mean PAH sediment concentration and associated risk scores are much greater for samples taken from the Struthers and Youngstown sites than those for samples taken from the Warren site. The mean PAH sediment concentration for the Warren site is only 0.05 mg/kg. The reason appears to be that the coke oven outfall for the Warren plant discharges to a swampy area associated with the Mahoning River whereas the coke oven outfalls for the other two plants discharge directly to the Mahoning River. Therefore, in the case of the Warren site (which is located in the Mahoning River), a substantial porportion of the PAHs discharged may settle out in the swampy area before they reach the Mahoning River (USEPA 1977).

The Struthers, Youngstown and Warren sites were all sampled in 1975 and may therefore not reflect current conditions. Although the Republic (LTV) Steel/Warren Plant is still operating, the Republic (LTV) Steel/Youngstown Plant has reportedly been shut down (Amendola 1985). No information on the current status of the Youngstown Sheet and Tube-Campbell Works (Struthers) was available.

5.3 MILWAUKEE REGION

Of the 10 regions discussed in this report, the Milwaukee region ranks third with respect to mean PAH concentration (10.7 mg/kg), second with respect to mean human carcinogenic risk score (8.4×10^{-5}), third with respect to mean human non-carcinogenic risk score (8.65×10^{-2}), fourth with respect to mean aquatic toxicity risk score and third with respect to overall ranking score (3 + 2 + 3 + 4 = 12). The Milwaukee region is not divided into subregions.

Table 5-3A lists mean PAH sediment concentrations (mg/kg), mean human carcinogenic risk scores ($\times 10^4$), mean human non-carcinogenic risk scores ($\times 10^2$), mean acute aquatic toxicity risk scores ($\times 10^3$), overall ranking scores and assorted rankings (in parenthesis) for sediment sampling sites in

Table 5-3A. Summary of Mean PAH Sediment Concentrations, Mean Risk Scores, and Ranking and Sampling Sites in the Milwaukee Region

Subregions and sampling sites	Mean PAH sediment concentration (mg/kg) and (ranking)	Mean carcinogenic risk score ($\times 10^4$) and (ranking)	Mean non-carcinogenic risk score ($\times 10^2$) and (ranking)	Mean acute aquatic toxicity risk score ($\times 10^3$) and (ranking)	Overall ranking score and (ranking)
Milwaukee Region	**10.7 (3)**	**0.84 (2)**	**8.65 (3)**	**3.62 (4)**	**12 (3)**
MIL 80-15	24.1 (1)	1.88 (1)	20.3 (1)	5.97 (3)	6 (1)
MIL 80-23	21.7 (2)	1.59 (2)	19.1 (2)	6.16 (2)	8 (2)
MIL 80-06	19.9 (3)	1.54 (3)	13.8 (3)	13.6 (1)	10 (3)
MIL 80-08	15.0 (4)	1.22 (4)	11.8 (5)	3.70 (5)	18 (4)
MIL 80-22	15.0 (5)	1.03 (5)	13.7 (4)	5.41 (4)	19 (5)
MIL 80-13	13.5 (6)	1.18 (6)	9.87 (6)	3.59 (6)	23 (6)
MIL 80-17	5.92 (7)	0.45 (7)	4.56 (7)	1.73 (7)	28 (7)
MIL 80-19	4.73 (8)	0.37 (8)	3.43 (8)	1.55 (8)	32 (8)
MIL 80-02	3.04 (9)	0.26 (10)	2.40 (10)	0.51 (10)	39 (9)
MIL 80-10	3.04 (10)	0.21 (11)	2.92 (9)	0.84 (9)	39 (10)
MIL 80-04	2.94 (11)	0.29 (9)	1.83 (11)	0.34 (11)	41 (11)
MIL 80-01	0.14 (12)	0.01 (12)	0.14 (12)	0.01 (12)	48 (12)

the Milwaukee region. Descriptions of the sediment sampling sites in the Milwaukee region were presented in Appendix B, Table B-3A. A map of the Milwaukee region showing the locations of sampling sites was presented in Figure 3-5.

The PAH contamination of sediments appears to be fairly widespread in the Milwaukee region. Of the 12 reported sediment samplings sites in the Milwaukee region, the first and sixth ranking sites (with respect to overall ranking score) are in the Milwaukee River, the second and fifth ranking sites are in the Menomonee River and the third and fourth ranking sites are in the Kinnickinnic River. Furthermore, there is less than a 50% difference in the mean PAH sediment concentrations between the first (24.1 mg/kg) and sixth (13.5 mg/kg) ranking sites. In going from the sixth to seventh ranking site, there is a 56% decrease in the mean PAH sediment concentration, but in going from the seventh (5.92 mg/kg) to the 11th (2.92 mg/kg) ranking site, there is only a 60% decrease in the mean PAH sediment concentration. Oil and grease concentrations exceed 10 g/kg of sediment at the 6 highest ranking sites and at 8 of the 12 reported sites overall.

The mean PAH sediment concentration for the 9th ranking site (MIL 82-02), which is located in Lake Michigan just off the mouth of the confluence of the Milwaukee and Kinnickinnic Rivers, is 3.04 mg/kg. That is comparable to the 2.94 mg/kg for the 11th ranking site (MIL 82-04), which is located at the confluence of the Milwaukee and Kinnickinnic Rivers. However, site MIL 82-02 is also located at the Jones Island STP outfall. Therefore, it is not clear whether the similarities in the mean PAH sediment concentration between MIL 82-04 and MIL 82-02 are due to PAH transport into Lake Michigan or PAH discharges from the Jones Island STP outfall. However, the mean PAH sediment concentration of the 12th and lowest ranking site (MIL 82-01), which is located only slightly north and east of MIL 82-02 in Lake Michigan, is 0.14 mg/kg, which is more than 20 times lower than that for MIL 82-02. Furthermore, site MIL 82-01 is the only one of the 12 sites in the Milwaukee region with a mean PAH sediment concentration of less than one mg/kg. Therefore, even if significant amounts of PAHs are transported into Lake Michigan, it appears that the longitudinal extent of the transport from the mouth of the confluence between the Milwaukee and Kinnickinnic Rivers may be extremely limited.

The PAH sediment concentrations listed for various PAHs and sampling sites MIL 80-02, MIL 80-04, MIL 80-06, MIL 80-08, MIL 80-13, MIL 80-17, MIL 80-19, MIL 80-22, and MIL 80-23 in Appendix B, Table B-3B are actually means of PAH concentrations for various depth fractions of the cores that were taken. Table 5-3B lists PAH concentrations for each PAH and core fraction for each of the sites listed above. As can be seen from Table 5-3B, some maximum PAH concentrations occured in the 30-60 cm and 60-90 cm core fractions as well as in the surface 0-30 cm core fractions. Although the occurrence of maximum PAH concentrations in some sub-surface core fractions may be due in part to some downward PAH transport and/or greater rates of degradation and dissolution near the surface, it may also be due to the more recent deposition of lesser PAH-contaminated sediment overlying the older, greater PAH-contaminated sediment.

The variability of PAH concentrations with depth shown in Table 5-3B indicates the importance of specifying core depth and core fraction when reporting PAH concentration in sediments. PAH concentrations in the surface core fractions (e.g., 0-30 cm) are probably more indicative of risks to benthic organisms and humans than PAH concentrations in the lower core fractions, or PAH concentrations averaged over all core fractions. However, a knowledge of the PAH concentrations as a fraction of depth in the sediment, in addition to a knowledge of PAH concentrations as a function of time, may be of value in determining trends in the PAH contamination of the sediment.

5.4 OHIO REGION

Of the 10 regions covered in this report, the Ohio region ranks fourth with respect to mean PAH sediment concentration (5.81 mg/kg), fifth with respect to mean human carcinogenic risk score (4.2×10^{-5}), fourth with respect to mean human non-carcinogenic risk score (4.07×10^{-2}), second with respect to mean acute aquatic toxicity risk score (1.55×10^{-2}) and fourth with respect to overall ranking score (4 + 5 + 4 + 2 = 15). The second-place ranking of the Ohio region with respect to mean acute aquatic toxicity score is due primarily to extremely high concentrations of naphthalene, acenaphthylene, fluorene, and phenanthrene in sediment samples taken from a single site (CUY 82-18) in the Cuyahoga River below an LTV (Republic) Steel outfall.

Table 5-3B. PAH Concentrations in Various Core Depth Fractions of Sediment Samples Taken in the Milwaukee Region

STORET ID	Core depth (cm)	Acenaph-thalene	Acenaph-thylene	Anthra-cene/phen-anthrene	Benzo[b,k]-fluoranthene	Benzo-[g,h,i]-perylene	Benzo[a]-pyrene	Chrysene	Dibenzo-[a,h]-anthracene	Fluor-anthene	Fluorene	Indeno-(1,2,3-cd)-pyrene	Naphthalene	Pyrene
MIL 80-02a	0-30	ND	ND	3.20	ND	ND	17.0	11.0	ND	13.0	ND	3.50	ND	13.0
MIL 80-02b	30-60	ND	ND	3.30	ND	ND	12.0	9.30	ND	11.0	0.90	0.70	ND	12.0
MIL 80-02c	60-85	ND	ND	0.60	ND	ND	3.0	2.80	ND	1.40	ND	0.70	ND	ND
MIL 80-02	Mean	ND	ND	2.40	ND	ND	10.8	7.70	ND	8.70	ND	1.60	ND	8.80
MIL 80-04a	0-30	ND	ND	1.80	ND	ND	6.50	6.70	ND	7.0	ND	3.60	ND	7.50
MIL 80-04b	30-60	ND	ND	1.60	ND	ND	22.0	5.40	ND	6.0	ND	1.80	ND	6.50
MIL 80-04	Mean	ND	ND	1.70	ND	ND	14.3	6.10	ND	6.50	ND	2.70	ND	7.00
MIL 80-06a	0-30	1.06	1.22	15.3	25.0	7.27	12.4	25.7	5.67	23.2	0.90	7.47	5.68	15.6
MIL 80-06b	30-60	1.06	1.89	24.3	39.1	13.7	23.1	48.8	11.6	42.5	2.21	15.1	12.7	28.5
MIL 80-06c	60-90	4.01	4.34	34.9	50.9	22.3	41.3	76.9	20.6	66.9	2.98	21.8	24.3	48.1
MIL 80-06c	90-110	2.01	2.28	20.4	25.2	8.78	17.2	40.9	10.9	30.4	1.77	9.67	9.22	22.9
MIL 80-06	Mean	2.04	2.45	24.0	35.9	13.4	24.1	48.7	12.3	41.6	1.98	13.9	13.3	29.3
MIL 80-08a	0-30	2.84	0.81	25.5	31.1	6.75	28.3	60.2	6.04	48.4	2.01	8.59	0.61	35.3
MIL 80-08b	30-60	1.19	0.43	15.7	15.2	4.23	11.5	28.4	3.39	25.3	1.46	5.23	0.24	20.0
MIL 80-08	Mean	2.02	0.62	20.6	23.2	5.49	19.9	44.3	4.72	36.9	1.74	6.97	0.43	27.7
MIL 80-13a	0-30	2.58	0.52	21.6	37.4	7.93	22.4	78.3	5.61	46.0	2.44	8.13	2.09	38.3
MIL 80-13b	30-60	0.78	0.54	15.5	21.3	8.64	12.8	46.5	5.79	29.2	1.11	5.76	0.45	23.5
MIL 80-13c	60-85	0.88	0.46	11.8	7.72	2.29	5.42	21.6	1.39	15.4	1.21	2.55	0.92	10.2
MIL 80-13	Mean	1.44	0.51	16.6	23.0	6.52	14.0	50.4	4.43	31.1	1.61	5.65	1.18	24.7
MIL 80-17a	0-30	0.45	0.41	6.85	13.4	3.21	5.92	19.5	8.09	16.8	0.70	7.64	0.40	8.30
MIL 80-17b	30-60	0.78	0.39	9.55	13.7	2.0	6.59	2.05	3.70	18.1	1.05	3.66	0.95	11.9
MIL 80-17c	60-90	0.32	0.20	7.19	7.20	2.78	3.90	19.4	2.50	10.3	0.64	1.75	0.47	8.22
MIL 80-17	Mean	0.52	0.33	7.86	11.4	2.66	5.47	13.7	4.73	15.1	0.80	4.35	0.61	9.47
MIL 80-19a	0-30	0.51	0.36	8.09	9.97	4.79	5.55	19.3	2.59	12.5	0.78	2.92	0.57	10.5
MIL 80-19b	30-60	0.80	0.70	9.45	10.4	3.37	4.99	15.0	10.2	14.3	1.24	8.37	1.07	8.58
MIL 80-19c	60-90	0.21	0.14	1.39	2.44	0.54	1.12	4.17	1.33	2.65	0.25	1.32	0.27	1.83
MIL 80-19	Mean	0.51	0.40	6.31	7.60	2.90	3.89	12.8	4.71	9.82	0.76	4.20	0.64	6.97
MIL 80-22a	0-30	2.23	1.69	31.6	26.0	5.90	15.6	49.8	3.75	44.3	2.35	6.86	0.59	32.8
MIL 80-22b	30-60	1.93	0.84	21.6	22.2	4.74	9.29	2.87	11.4	32.3	2.49	9.82	0.70	17.0
MIL 80-22c	60-85	3.55	1.04	39.9	24.0	6.05	15.8	51.0	4.60	37.6	4.58	5.35	1.80	27.4
MIL 80-22	Mean	1.19	1.19	30.4	24.1	5.53	13.4	33.6	6.70	38.1	3.06	7.46	0.98	25.6
MIL 80-23a	0-30	11.9	<0.5	63.9	40.9	14.5	35.4	117	8.49	84.6	4.27	14.2	ND	88.0
MIL 80-23b	30-60	1.16	0.50	20.5	21.9	3.46	9.02	31.4	8.61	34.2	2.83	8.20	0.26	18.9
MIL 80-23c	60-90	2.06	0.93	32.2	21.2	7.24	12.9	48.8	5.23	39.5	2.50	6.22	0.32	30.4
MIL 80-23d	90-100	2.74	1.32	37.6	29.1	11.0	17.2	64.7	8.08	49.4	3.50	9.04	0.28	38.8
MIL 80-23	Mean	4.81	0.56	38.7	28.1	8.66	18.9	65.6	7.51	47.5	3.23	9.49	0.20	45.1

Source: USEPA (1985b)

Table 5-4 lists mean PAH sediment concentrations (mg/kg), mean human carcinogenic risk scores (x 10^4), mean human non-carcinogenic risk scores (x 10^2), mean acute aquatic toxicity risk scores (x 10^3), overall ranking scores and associated rankings (in parenthesis) for the subregions and sediment sampling sites in the Ohio region. The subregions and sediment sampling sites within subregions are listed in the order of increasing overall ranking score. Descriptions of the sediment sampling sites in the Ohio region are presented in Appendix B, Table B-5A.

5.4.1 Cuyahoga River Subregion

Of the 5 reported subregions in the Ohio region, the Cuyahoga River subregion ranks first with respect to mean PAH sediment concentration (15.2 mg/kg), human carcinogenic risk score (6.8×10^{-5}), human non-carcinogenic risk score (1.21×10^{-2}), acute aquatic toxicity risk score (5.51×10^{-2}), and overall ranking score (1 + 1 + 1 + 1 = 4).

The mean PAH sediment concentration and associated mean risk scores are much higher for the Cuyahoga River subregion that for any other subregion in the Ohio region, and exceed those for the Black River/Lorain and Mahoning River subregions of the Ohio Steel region. The relatively high mean PAH sediment concentration and associated risk scores for the Cuyahoga River subregion are primarily due to the high mean PAH sediment concentration (75.7 mg/kg) for sediment sampling site CUY 82-18 which is located at an LTV (Republic) Steel outfall. Therefore, the Cuyahoga River subregion might alternatively be considered in the Ohio Steel region.

Of the reported sediment sampling sites in the Cuyahoga River subregion, the first ranking site with respect to overall ranking score is site CUY 82-18, discussed above. The second ranking site is CUY 82-18 which is located at the Williams Company outfall. No information was provided in STORET as to the type of industry in which the Williams Company is involved. However, the mean PAH sediment concentration for site CUY 82-18 (5.08 mg/kg) is much lower than for site CUY 82-08 (75.7mg/kg). Sampling site CUY 82-18 is described as being in the vicinity of U.S. Steel but ranks only fifth with respect to overall ranking score and mean PAH sediment concentration (2.42 mg/kg).

Table 5-4. Summary of Mean PAH Sediment Concentrations, Mean Risk Scores, and Rankings for Subregions and Sampling Sites in the Ohio Region

Subregions and sampling sites	Mean PAH sediment concentration (mg/kg) and (ranking)	Mean carcinogenic risk score ($\times 10^4$) and (ranking)	Mean non-carcinogenic risk score ($\times 10^2$) and (ranking)	Mean acute aquatic toxicity risk score ($\times 10^3$) and (ranking)	Overall ranking score and (ranking)
Ohio Region	**5.81 (4)**	**0.42 (5)**	**4.07 (4)**	**15.5 (2)**	**15 (4)**
Cuyahoga River Subregion	15.2 (1)	0.58 (1)	12.1 (1)	55.1 (1)	4 (1)
CUY 82-18	75.7 (1)	2.54 (1)	59.8 (1)	3.30 (1)	4 (1)
CUY 82-08	5.08 (2)	0.56 (3)	4.26 (2)	2.33 (3)	10 (2)
CUY 82-01	4.46 (3)	0.58 (2)	3.26 (3)	1.94 (4)	12 (3)
CUY 82-07	3.70 (4)	0.44 (4)	2.31 (4)	1.73 (5)	17 (4)
CUY 82-13	2.42 (5)	0.22 (6)	1.92 (6)	2.42 (2)	20 (5)
CUY 82-03	2.62 (6)	0.26 (5)	2.11 (5)	1.70 (6)	21 (6)
CUY 82-20	1.26 (7)	0.09 (7)	1.16 (7)	1.61 (7)	28 (7)
Ashtabula Subregion	3.44 (2)	0.26 (3)	3.12 (2)	1.80 (3)	10 (2)
AST 79-15	11.1 (1)	0.62 (2)	11.4 (1)	2.76 (3)	7 (1)
AST 79-16	8.70 (2)	0.73 (1)	7.26 (2)	2.43 (6)	11 (2)
AST 79-06	4.40 (3)	0.03 (6)	5.37 (3)	3.11 (2)	14 (3)
AST 79-05	3.30 (4)	0.06 (5)	3.88 (4)	2.56 (4)	17 (4)
AST 79-12	3.00 (5)	0.00 (7)	1.95 (6)	4.78 (1)	19 (5)
AST 79-03	1.90 (6)	0.03 (6)	1.98 (5)	2.45 (5)	22 (6)
AST 79-07	1.90 (6)	0.06 (5)	1.98 (5)	1.53 (7)	23 (7)
AST 77-03	1.18 (7)	0.10 (3)	1.00 (8)	0.32 (10)	28 (8)
AST 79-14	1.14 (8)	0.06 (5)	1.25 (7)	0.47 (9)	29 (9)
AST 79-04	0.95 (9)	0.07 (4)	0.80 (9)	1.03 (8)	30 (10)
AST 79-10	0.28 (10)	0.00 (7)	0.28 (10)	0.11 (11)	38 (11)
AST 79-17	0.13 (11)	0.00 (7)	0.13 (11)	0.05 (12)	41 (12)

Table 5-4. Summary of Mean PAH Sediment Concentrations, Mean Risk Scores, and Rankings for Subregions and Sampling Sites in the Ohio Region
(continued)

Subregions and sampling sites	Mean PAH sediment concentration (mg/kg) and (ranking)	Mean carcinogenic risk score ($\times 10^4$) and (ranking)	Mean non-carcinogenic risk score ($\times 10^2$) and (ranking)	Mean acute aquatic toxicity risk score ($\times 10^3$) and (ranking)	Overall ranking score and (ranking)
Toledo Subregion	2.41 (3)	0.43 (2)	1.06 (3)	2.01 (2)	10 (3)
TOL 82-07	6.76 (1)	0.79 (1)	4.34 (1)	4.75 (2)	5 (1)
TOL 82-02	5.14 (2)	0.59 (2)	1.64 (3)	2.52 (3)	10 (2)
TOL 82-44	5.13 (3)	0.59 (2)	2.22 (2)	0.57 (8)	15 (3)
TOL 82-31	1.68 (4)	0.18 (3)	0.83 (4)	2.29 (5)	16 (4)
TOL 82-28	0.89 (5)	0.04 (5)	0.30 (6)	5.95 (1)	17 (5)
TOL 82-30A	0.56 (6)	0.05 (4)	0.35 (5)	0.99 (6)	21 (6)
TOL 82-51	0.43 (7)	0.00 (7)	0.29 (7)	2.46 (4)	25 (7)
TOL 82-36A	0.43 (7)	0.00 (7)	0.29 (7)	2.46 (4)	25 (8)
TOL 82-33A	0.29 (8)	0.00 (7)	0.24 (8)	0.89 (7)	30 (9)
TOL 82-13	0.29 (8)	0.00 (7)	0.30 (6)	0.11 (12)	33 (10)
TOL 82-40	0.17 (9)	0.03 (6)	0.15 (9)	0.21 (10)	34 (11)
TOL 82-26	0.14 (10)	0.00 (7)	0.14 (10)	0.20 (11)	38 (12)
TOL 82-09	0.10 (11)	0.00 (7)	0.10 (11)	0.24 (9)	38 (13)
Conneaut Subregion	0.81 (4)	0.09 (4)	0.71 (4)	0.34 (4)	16 (4)
CON 82-02	1.04 (1)	0.09 (1)	0.89 (1)	0.40 (1)	4 (1)
CON 82-09	0.65 (2)	0.00 (2)	0.67 (2)	0.40 (1)	7 (2)
CON 82-05	0.60 (3)	0.00 (2)	0.55 (3)	0.19 (2)	10 (3)
CON 82-03	0.47 (4)	0.00 (2)	0.46 (4)	0.17 (3)	13 (4)
Fairport Harbor Subregion	0.55 (5)	0.07 (5)	0.40 (5)	0.17 (5)	20 (5)
FPH 82-02	1.04 (1)	0.09 (1)	0.89 (1)	0.40 (1)	4 (1)
FPH 82-18	0.82 (2)	0.09 (1)	0.51 (2)	0.13 (2)	7 (2)
FPH 82-04	0.22 (3)	0.03 (2)	0.18 (4)	0.13 (2)	11 (3)
FPH 82-14	0.19 (4)	0.00 (4)	0.19 (3)	0.07 (4)	15 (4)
FPH 82-01	0.17 (5)	0.01 (3)	0.15 (5)	0.04 (5)	18 (5)
FPH 82-07	0.12 (6)	0.00 (4)	0.12 (6)	0.09 (3)	19 (6)

5.4.2 Ashtabula subregion

Of the 5 reported subregions in the Ohio region, the Ashtabula subregion ranks second with respect to mean PAH sediment concentration (3.44 mg/kg), third with respect to mean human carcinogenic risk score (2.6×10^{-5}), second with respect to mean human non-carcinogenic risk score (3.12×10^{-2}), third with respect to mean acute aquatic toxicity risk score (1.80×10^{-3}) and second with respect to overall ranking score.

Of the 12 reported sampling sites in the Ashtabula subregion, sites AST 79-15 and AST 79-16 rank first and second, respectively, with respect to both overall ranking score and mean PAH sediment concentration (11.1 mg/kg and 8.70 mg/kg, respectively). The third, fourth, and fifth ranking sites are AST 79-06, AST 79-05, and AST 79-12, respectively. These sites are all located in the Ashtabula River. The sixth and eighth ranking sites are AST 79-07 and AST 77-03 which are located in the harbor. The three lowest ranking sites (10th through 12th) all have mean PAH sediment concentrations less than 1 mg/kg.

5.4.3 Toledo Subregion

Of the 5 reported subregions in the Ohio region, the Toledo subregion ranks third with respect to mean PAH sediment concentration (2.41 mg/ kg) and mean human carcinogenic risk score (4.3×10^{-5}), third with respect to mean human non-carcinogenic risk score (1.06×10^{-2}), second with respect to mean acute aquatic toxicity risk score (2.01×10^{-3}) and third with respect to overall ranking scores.

Of the 13 reported sampling sites in the Toledo subregion, sites TOL 82-07, TOL 82-02, TOL 82-44, and TOL 82-31 rank first through fourth, respectively with respect to overall ranking score. The mean PAH sediment concentrations for those sites are 6.76, 5.14, 5.13, and 1.68 mg/kg, respectively. The mean PAH sediment concentrations for the other 9 sites are all less than 1 mg/kg. The first four ranking sites are located in Swan Creek, Maumee Bay, Ottawa Creek, and Otter Creek, respectively.

5.4.4 Conneaut Subregion

Of the 5 reported subregions in the Ohio region, the Conneaut subregion ranks fourth with respect to mean PAH sediment concentration (0.81 mg/kg), mean human carcinogenic risk score (9×10^{-6}), mean human non-carcinogenic risk score (7.1×10^{-3}), mean acute aquatic toxicity risk score (3.4×10^{-4}), and overall ranking score.

Only one of the 4 reported sampling sites in the Conneaut subregion has a mean PAH sediment concentration exceeding 1 mg/kg (1.04 mg/kg for CON 82-02). However, all 4 sites are located in the Harbor. Therefore, no information is available on the levels of PAHs in the Conneaut River.

5.4.5 Fairpoint Harbor Subregion

Of the 5 reported subregions in the Ohio region, the Fairpoint Harbor subregion ranks fifth with respect to mean PAH sediment concentration (0.55 mg/kg), mean human carcinogenic risk score (7×10^{-5}), mean human non-carcinogenic risk score (4.0×10^{-3}), mean acute aquatic toxicity risk score (2.0×10^{-2}) and overall ranking score.

Only one of the 6 reported sampling sites in the Fairpoint Harbor subregion exceeds 1 mg/kg (1.04 mg/kg for FPH 82-02). From the latitudes and longitudes given in STORET, it appears that sites FPH 82-02, FPH 82-01, and FPH 82-04 may be in the Grand River.

5.5 NEW YORK STATE AND PENNSYLVANIA REGION (EXCLUDING BUFFALO)

Of the 10 regions covered in this report, the NY/PA region ranks fifth with respect to mean PAH concentration (3.00 mg/kg), fourth with respect to mean human carcinogenic risk score (4.5×10^{-5}), sixth with respect to mean human non-carcinogenic risk score (2.19×10^{-2}), ninth with respect to mean acute aquatic toxicity risk score (1.06×10^{-3}) and fifth with respect to overall ranking score (5 + 4 + 6 + 9 = 24).

Table 5-5 lists mean PAH sediment concentrations (mg/kg), mean human carcinogenic risk scores ($\times 10^4$), mean human non-carcinogenic risk scores ($\times 10^2$), mean acute aquatic life risk scores ($\times 10^3$), overall ranking scores and associated rankings (in parentheses) for the subregions and sediment sam-

Table 5-5. Summary of Mean PAH Sediment Concentrations, Mean Risk Scores, and Rankings for Subregions and Sampling Sites in the New York/Pennsylvania Harbors Region

Subregions and sampling sites	Mean PAH sediment concentration (mg/kg) and (ranking)	Mean carcinogenic risk score ($\times 10^4$) and (ranking)	Mean non-carcinogenic risk score ($\times 10^2$) and (ranking)	Mean acute aquatic toxicity risk score ($\times 10^3$) and (ranking)	Overall ranking score and (ranking)
New York/Pennsylvania Harbors Region	**3.00 (5)**	**0.45 (4)**	**2.19 (6)**	**1.06 (9)**	**24 (5)**
Dunkirk/Erie Subregion	28.8 (1)	2.46 (1)	22.8 (1)	8.69 (1)	4 (1)
DNK 82-06	46.8 (1)	4.42 (1)	37.1 (1)	15.2 (1)	4 (1)
ERH 82-05	10.8 (2)	0.71 (2)	5.68 (2)	0.01 (4)	10 (2)
ERH 82-03	6.35 (3)	0.50 (3)	1.95 (3)	1.61 (2)	11 (3)
ERH 82-01	1.97 (4)	0.00 (5)	0.00 (4)	0.72 (3)	16 (4)
DNK 82-02	1.40 (5)	0.09 (4)	0.00 (4)	0.00 (5)	18 (5)
Rochester Subregion	0.26 (2)	0.05 (2)	0.13 (2)	0.11 (3)	9 (2)
ROC 81-02	0.59 (1)	0.12 (1)	0.29 (1)	0.37 (1)	4 (1)
ROC 81-09	0.31 (5)	0.05 (4)	0.22 (2)	0.33 (2)	13 (2)
ROC 81-14	0.49 (2)	0.09 (2)	0.10 (6)	0.10 (4)	14 (3)
ROC 81-10	0.45 (3)	0.09 (2)	0.07 (9)	0.07 (5)	19 (4)
ROC 81-03	0.20 (7)	0.02 (7)	0.21 (3)	0.16 (3)	20 (5)
ROC 81-12	0.41 (4)	0.07 (3)	0.08 (8)	0.06 (6)	21 (6)
ROC 81-06	0.21 (6)	0.03 (6)	0.19 (4)	0.06 (6)	22 (7)
ROC 81-07	0.20 (7)	0.03 (6)	0.18 (5)	0.07 (5)	23 (8)
ROC 81-11	0.19 (8)	0.04 (5)	0.04 (12)	0.06 (6)	31 (9)
ROC 81-05	0.11 (9)	0.01 (8)	0.09 (7)	0.03 (7)	31 (10)
ROC 81-08	0.10 (10)	0.02 (7)	0.06 (10)	0.02 (8)	35 (11)
ROC 81-01	0.07 (12)	0.01 (8)	0.05 (11)	0.07 (5)	36 (12)
ROC 81-04	0.08 (11)	0.01 (8)	0.05 (11)	0.01 (9)	39 (13)

Table 5-5. Summary of Mean PAH Sediment Concentrations, Mean Risk Scores, and Rankings for Subregions and Sampling Sites in the New York/Pennsylvania Harbors Region
(continued)

Subregions and sampling sites	Mean PAH sediment concentration (mg/kg) and (ranking)	Mean carcinogenic risk score ($\times 10^4$) and (ranking)	Mean non-carcinogenic risk score ($\times 10^2$) and (ranking)	Mean acute aquatic toxicity risk score ($\times 10^3$) and (ranking)	Overall ranking score and (ranking)
Oswego/Olcott Subregion	0.22 (3)	0.04 (3)	0.13 (3)	0.16 (2)	11 (3)
OSW 81-02	1.29 (1)	0.25 (1)	0.63 (1)	0.50 (1)	4 (1)
OLC 81-04	0.15 (2)	0.02 (2)	0.13 (2)	0.18 (3)	9 (2)
OLC 81-01	0.06 (3)	0.00 (4)	0.06 (3)	0.23 (2)	12 (3)
OLC 81-03	0.06 (3)	0.01 (3)	0.05 (4)	0.08 (4)	18 (4)
OLC 81-02	0.01 (4)	0.00 (4)	0.00 (6)	0.06 (5)	19 (5)
OSW 81-01	0.01 (4)	0.00 (4)	0.01 (5)	0.02 (6)	19 (6)
OSW 81-04	0.00 (5)	0.00 (4)	0.00 (6)	0.02 (6)	21 (7)

pling sites in the NY/PA region. The subregions and sediment sampling sites within subregions are listed in order of increasing overall ranking score. Description of the sediment sampling sites in the NY/PA region are presented in Appendix B, Table B-10A.

5.5.1 Dunkirk/Erie Subregion

Of the 3 reported subregions in the NY/PA region, the Dunkirk/Erie subregion ranks first with respect to mean PAH sediment concentration (28.0 mg/kg), mean human carcinogenic risk score (2.46×10^{-4}), mean human non-carcinogenic risk score (0.228), mean acute aquatic toxicity risk score (8.69×10^{-3}) and overall ranking score (1 + 1 + 1 + 1 = 4).

The mean PAH sediment concentrations and associated mean risk scores are much higher for the Dunkirk/Erie subregion than for the other two subregions (Rochester and Oswego/Olcott). The 5 reported sampling sites in the Dunkirk/Erie subregion are ranked in the following order with respect to overall ranking score: DNK 82-06, ERH 82-05, ERH 82-03, ERH 82-01, and DNK 82-02. The mean sediment concentrations for those sites are 46.8, 10.8, 6.35, 1.97, and 1.40 mg/kg, respectively, and are higher than for any of the other reported sites in the NY/PA region. The highest ranking site (DNK 82-06) is located off Dunkirk in Lake Erie at an unidentified outfall beside the main beach. The second highest ranking site (ERH 82-05) is in the Erie area but its specific location is not described in STORET.

5.5.2 Rochester Subregion

Of the 3 reported subregions in the NY/PA region, the Rochester subregion ranks second with respect to mean PAH sediment concentration (0.26 mg/kg), mean human carcinogenic risk score (5.0×10^{-6}), mean human non-carcinogenic risk score (1.3×10^{-3}), and overall ranking score (2 + 2 + 2 + 3 + = 9). It ranks third (last) with respect to mean acute aquatic toxicity risk score (1.10×10^{-4}).

None of the 13 reported sediment sampling sites in the Rochester subregion have mean PAH sediment concentrations exceeding 1 mg/kg even though some of the sites are located downstream of various Kodak outfalls. All 13

sites are located in the Genessee River. The highest ranking site with respect to the lowest overall ranking score in the subregion is ROC 81-02, which is located near a storm sewer.

5.5.3 Oswego/Olcott Subregion

Of the 3 reported subregions in the NY/PA region, the Oswego/Olcott subregion ranks third and last with respect to mean PAH sediment concentration (0.22 mg/kg), mean human carcinogenic risk score (4.0×10^{-6}), mean human non-carcinogenic risk score (1.3×10^{-3}), and overall ranking score (3 + 3 + 3 + 2 = 11). It ranks second with respect to mean acute aquatic toxicity risk score (1.6×10^{-4}).

Of the 7 reported sampling sites in the Oswego/Olcott subregion, the highest ranking site with respect to overall ranking score is site OSW 81-02. Site OSW 81-02 is located in a swampy area which drains to Wine Creek, in Oswego, and is the only site in the Oswego/Olcott region with a mean PAH sediment concentration exceeding 1 mg/kg (1.29 mg/kg). The second highest ranking site in the subregion, and the highest ranking site in the Olcott area, is site OLC 81-04 which is located near the mouth of 18 Mile Creek.

5.6 BUFFALO REGION

Of 10 regions covered in this report, the Buffalo region ranks seventh with respect to mean PAH sediment concentration (2.64 mg/kg), eighth with respect to mean human carcinogenic risk score (1.9×10^{-5}), fifth with respect to mean human non-carcinogenic risk score (2.9×10^{-2}), fifth with respect to mean acute aquatic toxicity risk score (2.16×10^{-3}), and sixth with respect to overall ranking score (7 + 8 + 5 + 5 = 25).

Table 5-6 lists mean PAH sediment concentrations (mg/kg), mean human carcinogenic risk scores ($\times 10^4$), mean human non-carcinogenic risk scores ($\times 10^2$), mean acute aquatic toxicity risk scores ($\times 10^3$), overall ranking scores and associated rankings (in parenthesis) for subregions and sampling sites in the Buffalo region. The subregions and sampling sites within subregions are listed in the order of increasing overall ranking score. Descriptions of the sediment sampling sites in the Buffalo region are

Table 5-6. Summary of Mean PAH Sediment Concentrations, Mean Risk Scores, and Rankings for Subregions and Sampling Sites in the Buffalo Region

Subregions and sampling sites	Mean PAH sediment concentration (mg/kg) and (ranking)	Mean carcinogenic risk score ($\times 10^4$) and (ranking)	Mean non-carcinogenic risk score ($\times 10^2$) and (ranking)	Mean acute aquatic toxicity risk score ($\times 10^3$) and (ranking)	Overall ranking score and (ranking)
Buffalo Region	**2.64 (7)**	**0.19 (8)**	**2.92 (5)**	**2.16 (5)**	**25 (6)**
Rush Creek/Smokes Creek Subregion	10.81 (1)	0.67 (2)	7.92 (1)	3.69 (2)	6 (1)
BUF 81-03(B)	21.1 (1)	1.68 (1)	14.1 (1)	7.54 (1)	4 (1)
Smokes Cr.(A)	1.30 (2)	0.17 (2)	0.86 (2)	0.19 (2)	8 (2)
Scajaquada Creek Subregion	8.38 (2)	0.70 (1)	7.25 (2)	6.35 (1)	6 (2)
N37	20.8 (1)	2.02 (1)	13.9 (2)	3.53 (3)	7 (1)
N43	20.2 (2)	0.93 (3)	21.4 (1)	31.0 (1)	7 (2)
N42	11.7 (3)	1.34 (2)	8.01 (3)	5.46 (2)	10 (3)
N41	3.33 (4)	0.23 (4)	3.97 (4)	2.34 (4)	16 (4)
N39	1.50 (5)	0.13 (5)	1.59 (5)	0.93 (5)	20 (5)
N38	1.00 (6)	0.13 (5)	0.79 (7)	0.63 (6)	24 (6)
N40	0.77 (7)	0.09 (6)	1.04 (6)	0.60 (7)	26 (7)
Buffalo Harbor Subregion	4.22 (4)	0.33 (4)	5.37 (4)	3.19 (3)	15 (3)
BUF 81-55	19.2 (1)	1.55 (1)	24.7 (1)	32.8 (1)	4 (1)
3-1	9.57 (2)	0.30 (7)	17.3 (2)	4.98 (3)	14 (2)
3-3	6.42 (4)	0.64 (4)	7.58 (4)	1.99 (6)	18 (3)
BUF 81-60	5.21 (6)	1.18 (2)	4.79 (7)	2.65 (4)	19 (4)
3-2	5.88 (5)	0.22 (9)	8.62 (3)	2.47 (5)	22 (5)
BUF 81-56	4.38 (8)	0.90 (3)	4.53 (9)	5.23 (2)	22 (6)
BUF 81-58	6.64 (3)	0.39 (6)	4.58 (8)	1.64 (8)	25 (7)
B-5 Union	4.58 (7)	0.19 (10)	5.77 (5)	1.91 (7)	29 (8)
BUF 81-51	2.83 (9)	0.11 (12)	5.58 (6)	1.13 (9)	36 (9)
BUF 81-59	2.00 (12)	0.27 (8)	2.97 (11)	1.11 (10)	41 (10)

Table 5-6. Summary of Mean PAH Sediment Concentrations, Mean Risk Scores, and Rankings for Subregions and Sampling Sites in the Buffalo Region
(continued)

Subregions and sampling sites	Mean PAH sediment concentration (mg/kg) and (ranking)	Mean carcinogenic risk score ($\times 10^4$) and (ranking)	Mean non-carcinogenic risk score ($\times 10^2$) and (ranking)	Mean acute aquatic toxicity risk score ($\times 10^3$) and (ranking)	Overall ranking score and (ranking)
BUF 81-57	2.19 (10)	0.57 (5)	1.89 (13)	0.54 (14)	42 (11)
3-5	2.03 (11)	0.09 (13)	3.09 (10)	0.75 (11)	45 (12)
BUF 81-65	1.64 (13)	0.39 (6)	1.52 (15)	0.55 (13)	47 (13)
3-4	1.56 (14)	0.07 (14)	2.40 (12)	0.57 (12)	52 (14)
306	1.20 (15)	0.06 (15)	1.84 (14)	0.42 (15)	59 (15)
BUF 81-54	0.87 (16)	0.14 (11)	0.96 (16)	0.23 (17)	60 (16)
BUF 81-66	0.40 (17)	0.03 (16)	0.72 (17)	0.37 (16)	66 (17)
U27	0.00 (18)	0.00 (17)	0.00 (18)	0.00 (18)	71 (18)
U26	0.00 (18)	0.00 (17)	0.00 (18)	0.00 (18)	72 (19)
Two Mile Creek Subregion	4.43 (3)	0.16 (6)	5.51 (3)	2.79 (5)	17 (4)
N31	10.0 (2)	0.41 (1)	10.8 (2)	6.98 (1)	6 (1)
4-3	13.9 (1)	0.07 (6)	30.3 (1)	5.83 (3)	11 (2)
N36	3.42 (4)	0.27 (2)	3.62 (3)	6.39 (2)	11 (3)
N35	2.68 (5)	0.18 (3)	2.57 (4)	1.53 (5)	17 (4)
4-1	3.75 (3)	0.27 (2)	1.80 (7)	0.15 (8)	20 (5)
N34	1.50 (6)	0.13 (4)	1.08 (8)	2.38 (4)	22 (6)
N33	1.23 (8)	0.09 (5)	1.91 (5)	1.40 (6)	24 (7)
4-2	1.27 (7)	0.04 (7)	1.90 (6)	0.41 (7)	27 (8)
N32	0.00 (9)	0.00 (8)	0.00 (9)	0.00 (9)	35 (9)
Lower Niagara River Subregion	3.07 (5)	0.46 (3)	3.57 (5)	1.44 (6)	19 (5)
BUF 81-31	9.45 (1)	0.73 (4)	13.6 (1)	7.84 (1)	7 (1)
BUF 81-45	6.68 (2)	1.20 (1)	5.64 (3)	1.57 (4)	10 (2)
BUF 81-43	4.79 (3)	1.20 (1)	3.76 (6)	1.39 (5)	15 (3)
BUF 81-32	4.18 (5)	0.48 (6)	6.17 (2)	2.89 (2)	15 (4)

Table 5-6. Summary of Mean PAH Sediment Concentrations, Mean Risk Scores, and Rankings for Subregions and Sampling Sites in the Buffalo Region
(continued)

Subregions and sampling sites	Mean PAH sediment concentration (mg/kg) and (ranking)	Mean carcinogenic risk score ($\times 10^4$) and (ranking)	Mean non-carcinogenic risk score ($\times 10^2$) and (ranking)	Mean acute aquatic toxicity risk score ($\times 10^3$) and (ranking)	Overall ranking score and (ranking)
BUF 81-35	4.47 (4)	1.05 (2)	4.06 (5)	1.14 (8)	19 (5)
BUF 81-41	3.49 (7)	0.68 (5)	4.11 (4)	1.35 (6)	22 (6)
BUF 81-71	2.14 (8)	0.30 (7)	3.66 (7)	1.63 (3)	25 (7)
BUF 81-37	3.84 (6)	1.00 (3)	2.80 (8)	0.68 (10)	27 (8)
BUF 81-40	1.84 (9)	0.28 (8)	2.66 (9)	1.17 (7)	33 (9)
BUF 81-67	1.43 (11)	0.18 (9)	2.18 (10)	0.65 (12)	42 (10)
B-4	1.76 (10)	0.07 (12)	2.07 (11)	0.66 (11)	44 (11)
BUF 81-70	1.21 (12)	0.11 (11)	1.62 (12)	0.77 (9)	44 (12)
BUF 81-44	0.74 (13)	0.16 (10)	0.82 (13)	0.21 (13)	49 (13)
BUF 81-51	0.21 (14)	0.00 (13)	0.49 (14)	0.10 (14)	55 (14)
BUF 81-48	0.06 (15)	0.00 (13)	0.13 (15)	0.02 (15)	58 (15)
Buffalo River Subregion	2.81 (6)	0.15 (7)	2.32 (6)	3.17 (4)	23 (16)
BUF 81-12	15.4 (1)	0.87 (2)	8.79 (3)	78.0 (1)	7 (1)
B-3	7.95 (3)	0.26 (4)	15.3 (1)	4.67 (2)	10 (2)
BUF 81-26	13.3 (2)	2.34 (1)	5.93 (5)	2.57 (4)	12 (3)
B-4	5.46 (5)	0.16 (6)	9.19 (2)	4.56 (3)	16 (4)
1-5	4.76 (6)	0.07 (12)	7.80 (4)	1.91 (5)	27 (5)
1-6	2.78 (11)	0.11 (8)	2.97 (7)	0.80 (7)	33 (6)
B-2	3.68 (8)	0.05 (14)	5.04 (6)	1.25 (6)	34 (7)
1-7	3.65 (2)	0.10 (9)	2.79 (8)	0.54 (12)	41 (8)
BUF 81-27	1.36 (17)	0.42 (3)	0.73 (18)	0.70 (8)	46 (9)
BUF 81-11	4.28 (7)	0.00 (17)	1.52 (12)	0.57 (11)	47 (10)
BUF 81-22	0.96 (19)	0.16 (6)	1.21 (13)	0.66 (9)	47 (11)
1-8	2.21 (13)	0.08 (11)	1.77 (11)	0.35 (16)	51 (12)
1-4	1.99 (14)	0.04 (15)	2.03 (9)	0.42 (13)	51 (13)
1-10	1.90 (15)	0.06 (13)	2.00 (10)	0.38 (14)	52 (14)

Table 5-6. Summary of Mean PAH Sediment Concentrations, Mean Risk Scores, and Rankings for Subregions and Sampling Sites in the Buffalo Region
(continued)

Subregions and sampling sites	Mean PAH sediment concentration (mg/kg) and (ranking)	Mean carcinogenic risk score ($\times 10^4$) and (ranking)	Mean non-carcinogenic risk score ($\times 10^2$) and (ranking)	Mean acute aquatic toxicity risk score ($\times 10^3$) and (ranking)	Overall ranking score and (ranking)
BUF 81-05	6.20 (4)	0.00 (17)	0.97 (15)	0.22 (17)	53 (15)
BUF 81-04	3.14 (9)	0.24 (5)	0.65 (19)	0.16 (21)	54 (16)
BUF 81-19	0.72 (22)	0.12 (7)	0.93 (16)	0.61 (10)	55 (17)
1-9	1.67 (16)	0.04 (15)	1.77 (11)	0.37 (15)	57 (18)
BUF 81-10	2.98 (10)	0.00 (17)	0.58 (20)	0.16 (21)	68 (19)
1-2	0.85 (21)	0.03 (16)	1.11 (14)	0.21 (18)	69 (20)
1-3	1.03 (18)	0.03 (16)	0.91 (17)	0.18 (19)	70 (21)
1-1	0.92 (20)	0.03 (16)	1.11 (14)	0.17 (20)	70 (22)
BUF 81-02	0.33 (24)	0.09 (10)	0.25 (26)	0.10 (23)	83 (23)
BUF 81-13	0.29 (25)	0.00 (17)	0.57 (21)	0.15 (22)	85 (24)
BUF 81-01	0.29 (25)	0.07 (12)	0.27 (24)	0.09 (24)	85 (25)
BUF 81-03	0.70 (23)	0.00 (17)	0.32 (22)	0.08 (25)	87 (26)
BUF 81-08	0.24 (26)	0.05 (14)	0.26 (25)	0.08 (25)	90 (27)
BUF 81-16	0.12 (28)	0.00 (17)	0.30 (23)	0.15 (22)	90 (28)
BUF 81-07	0.16 (27)	0.07 (12)	0.00 (28)	0.00 (27)	94 (29)
BUF 81-53	0.10 (29)	0.00 (17)	0.24 (27)	0.05 (26)	99 (30)
BUF 81-24	0.03 (30)	0.00 (17)	0.00 (28)	0.08 (25)	100 (31)
Tonawanda Canal Subregion	1.34 (7)	0.17 (5)	1.58 (7)	0.81 (8)	27 (7)
BUF 81-72	7.29 (1)	0.02 (13)	15.2 (1)	5.45 (1)	16 (1)
BUF 81-92	4.66 (2)	1.30 (2)	3.45 (2)	0.79 (11)	17 (2)
BUF 81-73	4.49 (3)	1.39 (1)	2.65 (6)	1.11 (7)	17 (3)
BUF 81-78	4.45 (4)	0.94 (3)	3.22 (4)	1.42 (6)	17 (4)
BUF 81-87A	2.58 (5)	0.63 (4)	2.02 (8)	3.16 (3)	20 (5)
BUF 81-74B	1.83 (7)	0.03 (12)	3.14 (5)	3.19 (2)	26 (6)
BUF 81-87	1.95 (6)	0.46 (6)	1.42 (9)	0.62 (12)	33 (7)
BUF 81-74A	1.12 (11)	0.23 (7)	1.27 (10)	1.61 (5)	33 (8)

Table 5-6. Summary of Mean PAH Sediment Concentrations, Mean Risk Scores, and Rankings for Subregions and Sampling Sites in the Buffalo Region
(continued)

Subregions and sampling sites	Mean PAH sediment concentration (mg/kg) and (ranking)	Mean carcinogenic risk score ($\times 10^4$) and (ranking)	Mean non-carcinogenic risk score ($\times 10^2$) and (ranking)	Mean acute aquatic toxicity risk score ($\times 10^3$) and (ranking)	Overall ranking score and (ranking)
BUF 81-76	1.23 (10)	0.07 (11)	2.08 (7)	0.83 (10)	38 (9)
BUF 81-82	1.56 (9)	0.00 (15)	3.32 (3)	0.52 (13)	40 (10)
BUF 81-75	0.91 (12)	0.17 (8)	1.03 (12)	0.92 (9)	41 (11)
BUF 81-83	1.64 (8)	0.47 (5)	0.60 (15)	0.30 (16)	44 (12)
U29	0.67 (14)	0.03 (12)	0.46 (17)	1.82 (4)	47 (13)
BUF 81-81	0.69 (13)	0.11 (9)	0.79 (13)	0.27 (18)	53 (14)
BUF 81-90	0.57 (15)	0.09 (10)	0.63 (14)	0.24 (19)	58 (15)
BUF 81-80A	0.16 (19)	0.00 (15)	0.21 (19)	0.96 (8)	61 (16)
BUF 81-86	0.53 (16)	0.09 (10)	0.56 (16)	0.23 (20)	62 (17)
U35	0.53 (16)	0.01 (14)	1.08 (11)	0.20 (21)	62 (18)
U30	0.25 (18)	0.01 (14)	0.40 (18)	0.43 (15)	65 (19)
BUF 81-89	0.33 (17)	0.09 (10)	0.12 (20)	0.16 (22)	69 (20)
BUF 81-91A	0.03 (20)	0.00 (15)	0.00 (22)	0.50 (14)	71 (21)
BUF 81-91C	0.02 (21)	0.00 (15)	0.00 (22)	0.28 (17)	75 (22)
BUF 81-95	0.01 (22)	0.00 (15)	0.03 (21)	0.01 (23)	81 (23)
BUF 81-91B	0.00 (23)	0.00 (15)	0.00 (22)	0.00 (24)	84 (24)
U36	0.00 (23)	0.00 (15)	0.00 (22)	0.00 (24)	84 (25)
U37	0.00 (23)	0.00 (15)	0.00 (22)	0.00 (24)	84 (26)
U34	0.00 (23)	0.00 (15)	0.00 (22)	0.00 (24)	84 (27)
U32	0.00 (23)	0.00 (15)	0.00 (22)	0.00 (24)	84 (28)
U31	0.00 (23)	0.00 (15)	0.00 (22)	0.00 (24)	84 (29)
U38	0.00 (23)	0.00 (15)	0.00 (22)	0.00 (24)	84 (30)
U33	0.00 (23)	0.00 (15)	0.00 (22)	0.00 (24)	84 (31)

Table 5-6. Summary of Mean PAH Sediment Concentrations, Mean Risk Scores, and Rankings for Subregions and Sampling Sites in the Buffalo Region
(continued)

Subregions and sampling sites	Mean PAH sediment concentration (mg/kg) and (ranking)	Mean carcinogenic risk score ($\times 10^4$) and (ranking)	Mean non-carcinogenic risk score ($\times 10^2$) and (ranking)	Mean acute aquatic toxicity risk score ($\times 10^3$) and (ranking)	Overall ranking score and (ranking)
Lake Erie/Buffalo Subregion	0.93 (8)	0.04 (8)	1.20 (8)	1.34 (7)	31 (8)
U15	8.71 (1)	0.42 (1)	9.24 (1)	12.2 (1)	4 (1)
U10	5.14 (2)	0.15 (2)	6.60 (2)	9.34 (2)	8 (2)
U17	2.15 (3)	0.12 (3)	1.93 (4)	2.50 (4)	14 (3)
U11	1.68 (4)	0.05 (5)	2.16 (3)	3.29 (3)	15 (4)
U13	2.16 (5)	0.06 (4)	0.94 (7)	1.28 (8)	24 (5)
U7	1.09 (7)	0.04 (6)	1.43 (6)	2.06 (5)	24 (6)
2-5	1.15 (6)	0.05 (5)	1.45 (5)	0.40 (10)	26 (7)
U14	1.09 (7)	0.06 (4)	0.92 (8)	1.39 (7)	26 (8)
U12	0.92 (8)	0.05 (5)	0.73 (10)	1.56 (6)	29 (9)
2-8	0.75 (9)	0.03 (7)	0.92 (8)	0.23 (15)	39 (10)
U16	0.55 (11)	0.03 (7)	0.47 (15)	0.90 (9)	42 (11)
2-7	0.54 (12)	0.02 (8)	0.82 (9)	0.24 (14)	43 (12)
U8	0.73 (10)	0.05 (5)	0.62 (12)	0.18 (17)	44 (13)
BUF 81-61	0.40 (15)	0.06 (4)	0.60 (13)	0.22 (16)	48 (14)
2-6	0.49 (13)	0.03 (7)	0.66 (11)	0.16 (18)	49 (15)
U19	0.40 (15)	0.02 (8)	0.32 (17)	0.35 (11)	51 (16)
U20	0.46 (14)	0.03 (7)	0.28 (19)	0.28 (13)	53 (17)
U18	0.39 (16)	0.02 (8)	0.29 (18)	0.29 (12)	54 (18)
2-4	0.33 (17)	0.01 (9)	0.58 (14)	0.14 (19)	59 (19)
U25	0.22 (18)	0.00 (10)	0.46 (16)	0.06 (20)	64 (20)
U21	0.17 (19)	0.01 (9)	0.27 (20)	0.05 (21)	69 (21)
U22	0.16 (20)	0.01 (9)	0.26 (21)	0.05 (21)	71 (22)
2-2	0.15 (21)	0.01 (9)	0.26 (21)	0.06 (20)	71 (23)
U23	0.15 (21)	0.01 (9)	0.23 (22)	0.05 (21)	73 (24)
2-1	0.11 (22)	0.01 (9)	0.20 (23)	0.06 (20)	73 (25)
U9	0.10 (23)	0.00 (10)	0.14 (24)	0.04 (22)	79 (26)

Table 5-6. Summary of Mean PAH Sediment Concentrations, Mean Risk Scores, and Rankings for Subregions and Sampling Sites in the Buffalo Region
(continued)

Subregions and sampling sites	Mean PAH sediment concentration (mg/kg) and (ranking)	Mean carcinogenic risk score ($\times 10^4$) and (ranking)	Mean non-carcinogenic risk score ($\times 10^2$) and (ranking)	Mean acute aquatic toxicity risk score ($\times 10^3$) and (ranking)	Overall ranking score and (ranking)
U5	0.11 (22)	0.01 (9)	0.10 (26)	0.02 (24)	81 (27)
2-3	0.07 (24)	0.00 (10)	0.13 (25)	0.03 (23)	82 (28)
U4	0.06 (25)	0.00 (10)	0.10 (26)	0.01 (25)	86 (29)
U24	0.00 (26)	0.00 (10)	0.00 (27)	0.00 (26)	89 (30)
U6	0.00 (26)	0.00 (10)	0.00 (27)	0.00 (26)	89 (31)
U3	0.00 (26)	0.00 (10)	0.00 (27)	0.00 (26)	89 (32)
U2	0.00 (26)	0.00 (10)	0.00 (27)	0.00 (26)	89 (33)
Upper Niagara River/Chippawa Canal Subregion	0.09 (9)	0.00 (9)	0.13 (27)	0.02 (9)	36 (9)
U-4	0.31 (1)	0.02 (1)	0.32 (1)	0.06 (1)	4 (1)
5	0.08 (2)	0.00 (2)	0.15 (2)	0.03 (2)	8 (2)
6	0.03 (3)	0.00 (2)	0.06 (3)	0.01 (3)	12 (3)
U42	0.00 (4)	0.00 (2)	0.00 (4)	0.00 (4)	16 (4)

presented in Appendix B, Table B-9A, and cross-referenced to Figures 3-12, 3-13, and 3-14. Potentially significant point sources of pollution were listed in Appendix B, Table B-9B and cross-referenced to Figure 3-10. Potentially significant landfill and/or hazardous waste disposal sites, and non-point sources of pollution are listed in Appendix B, Table B-9C and cross-referenced to Figure 3-11. Maps of the Niagara River System (e.g., Buffalo region) showing direction of flow, potentially significant point sources of pollution, potentially significant non-point sources of pollution and locations of sampling sites are presented in Figures 3-9 through 3-14.

5.6.1 Rush Creek/Smokes Creek Subregion

Of the 9 reported subregions in the Buffalo region, the Rush Creek/Smokes Creek subregion ranks first with respect to mean PAH sediment concentration (10.8 mg/kg), second with respect to mean human carcinogenic risk score (6.7×10^{-5}), first with respect to mean human non-carcinogenic risk score (7.92×10^{-2}), second with respect to mean acute aquatic toxicity risk score (3.69×10^{-3}) and first with respect to overall ranking score (1 + 2 + 1 + 2 = 6).

The Rush Creek/Smokes Creek subregion consists of only one sediment sampling site in Rush Creek and one sediment sampling site in Smokes Creek. Smokes Creek empties into Lake Erie just south of Buffalo Harbor. Rush Creek empties into Lake Erie just south of Rush Creek (Figure 3-9).

Of the 2 reported sites in the Rush Creek/Smokes Creek subregion the highest ranking site, with respect to every criteria, is the Rush Creek site which has the highest mean PAH sediment concentration of any reported site in the entire Buffalo region (2.1 mg/kg). The mean PAH sediment concentration for the Smokes Creek site is 1.30 mg/kg. Unfortunatly, specific locations for the Rush Creek and Smokes Creek sites were not described in STORET (1985) or Black (1983). However, Bethlehem Steel outfalls discharge to both Rush and Smokes Creeks (Figure 3-10).

5.6.2 Scajaquada Creek Subregion

Of the 9 reported subregions in the Buffalo region, the Scajaquada Creek subregion ranks second with respect to mean PAH sediment concentration (8.38

mg/kg), first with respect to mean carcinogenic risk score (7.0×10^{-5}), second with respect to mean human non-carcinogenic risk score (7.25×10^{-2}), first with respect to mean acute aquatic toxicity risk score (6.35×10^{-3}), and second with respect to overall ranking score (2 + 1 + 2 + 1 = 6). (Its overall ranking score is actually identical to that of the Rush Creek/Smokes Creek subregion, but it is ranked below that subregion due to a lower mean PAH sediment concentration.) Scajaquada Creek empties into the lower Niagara River/Black Rock Canal north of the Buffalo River (Figure 3-9).

The seven reported sediment sampling sites in the Scajaquada Creek subregion are ranked in the following order with respect to overall ranking score in the subregion: N37, N43, N42, N41, N39, N38, and N40. The mean PAH sediment concentrations for sites N37 and N43 are the second and third highest, respectively, for reported sites in the entire Buffalo region.

The second highest ranking site in the Scajaquada Creek subregion is N43 which is located at the mouth of the Creek. In general, the rankings and mean PAH sediment concentrations of the other sites decrease with increasing distance upstream of the mouth. However, the highest ranking site (with the highest mean PAH sediment concentration) is N37 which is located upstream of at least most of the sites. Unfortunately, even though the Scajaquada Creek subregion ranks second out of 9 subregions in the Buffalo region, no descriptions of the sites were available in NRTC (1984). Furthermore, none of the listed potentially significant point and non-point pollution sources for the Niagara River System (listed in Appendix B, Tables B-9B and B-9C, cross-referenced to Figures 3-10 and 3-11, and taken from NRTC 1984) are located along Scajaquada Creek. Some of the site locations are depicted in Figure 3-13.

5.6.3 Buffalo Harbor Subregion

The Buffalo Harbor subregion ranks fourth with respect to mean PAH sediment concentration (4.22 mg/kg), fourth with respect to mean human carcinogenic risk score (3.3×10^{-5}), fourth with respect to mean human non-carcinogenic risk score (5.37×10^{-2}), third with respect to mean acute aquatic toxicity risk score (3.19×10^{-3}) and third with respect to overall ranking score (4 + 4 + 4 + 3 = 15).

Of the 19 reported sites in the Buffalo Harbor subregion, the five highest ranking sites with respect to both overall ranking scores and mean PAH sediment concentration are BUF 81-55 (G3), 3-1 (N9), 3-3 (N11), BUF 81-60 (G4), and 3-2 (N10), respectively. The mean PAH sediment concentrations for those sites are 19.2, 9.57, 6.42, 5.21, and 5.88 mg/kg, respectively. The sites are all located at the mouths of either the Lackawana or Unionship Canals where they empty into the south end of the harbor (Figure 3-9). The mouths (entrances) to both canals are in the vicinity of Bethlehem Steel.

5.6.4 Two Mile Creek Subregion

Of the 9 subregions in the Buffalo region, the Two Mile Creek subregion ranks third with respect to mean PAH sediment concentration (4.43 mg/kg), sixth with respect to mean human carcinogenic risk score (1.6×10^{-5}), third with respect to mean human non-carcinogenic risk score (5.51×10^{-2}), fifth with respect to mean acute aquatic toxicity risk score (2.79×10^{-3}), and fourth with respect to overall ranking score (4 + 4 + 4 + 3 = 15).

Of the 9 reported sites in the Two Mile Creek subregion, the five highest ranking sites with respect to overall ranking score are N31, 4-3 (N25), N36, N35, and 4-1 (N37), respectively. The mean PAH sediment concentrations for those sites are 10.0, 13.9, 3.42, 2.68, and 3.75 mg/kg, respectively. Site N31 is located upstream in the vicinity of Union Carbide Corp., Linde Div., which manufactures and fabricates cyrogenic hardware. Sites N36, N35, and 4-1 (N37) are located at or just upstream of the mouth of Two Mile Creek.

5.6.5 Lower Niagara River/Black Rock Canal Subregion

Of the 9 subregions in the Buffalo region, the Lower Niagara River/Black Rock Canal subregion ranks fifth with respect to mean PAH sediment concentration (3.07 mg/kg), third with respect to mean human carcinogenic risk score (4.6×10^{-5}), fifth with respect to mean human non-carcinogenic risk score (3.57×10^{-2}), sixth with respect to mean acute aquatic toxicity risk score (1.44×10^{-3}), and fifth with respect to overall ranking score (5 + 3 + 5 + 6 = 18).

Of the 15 reported sites in the Lower Niagara River subregion, the 3 highest ranking sites with respect to both overall ranking score and mean PAH sediment concentration are BUF 81-31 (G40), BUF 81-45 (G32) and BUF 81-43 (G34). The mean PAH sediment concentrations for those sites are 9.45, 6.68, and 4.79 mg/kg, respectively. Site BUF 81-31 (G40) is located at the mouth of Scajaquada Creek which is the second highest ranking subregion in the Buffalo region. Sites BUF 81-45 (G32) and BUF 81-43 (G34) are located in Black Rock Canal south of Squaw Island. There are no significant point sources listed in the vicinity of sites BUF 81-45 (G32) and BUF 81-43 (G34). However, there is a waste disposal site on Squaw Island which contains foundry sand, incinerator residues, trace oils, resins, and municipal wastes (NRTC 1984).

5.6.6 Buffalo River Subregion

Of the 9 subregions in the Buffalo region, the Buffalo River subregion ranks sixth with respect to mean PAH sediment concentration (2.81 mg/kg), seventh with respect to mean human carcinogenic risk score (1.5×10^{-5}), sixth with respect to mean human non-carcinogenic risk score (2.32×10^{-2}), fourth with respect to mean acute aquatic toxicity risk score (3.17×10^{-3}), and sixth with respect to overall ranking score (6 + 7 + 6 + 4 = 23).

Of the 31 reported sites in the Buffalo River subregion, the 6 highest ranking sites with respect to overall ranking score are BUF81-12 (G19), B-3, BUF81-26 (G25), B-1, 1-5 (N19), and 1-6 (N20), respectively. The mean PAH sediment concentrations for these sites are 15.4, 7.95, 13.3, 5.46, 4.76, and 2.78 mg/kg, respectively. Sites BUF81-12 (G19), 1-5 (N19), and 1-6 (N20) are located at or just downstream of Buffalo Color, which manufactures dyes. The Republic Steel and the Donner-Coke outfalls are located only a short distance downstream of the Buffalo Color outfall but appear to be downstream of sites BUF81-12 (G19) and 1-5 (N19). Site 1-6 (N20) is located just downstream of the Republic Steel outfall and may be in the vicinity of the Donner-Coke outfall. The locations of sites B-1 and B-3 in the Buffalo River are not specified by Black (1983) or Black et al. (1985). Site BUF81-26 (G25) is located in the vicinity of a combined sewer.

Although the Buffalo River subregion contains a few sites with relatively high levels of PAH contamination, its overall ranking with respect to the

various criteria is relatively low, compared to a number of other subregions in the Buffalo region, due to the relatively low PAH levels in sites upstream of the Buffalo Color, Republic Steel, and Donner-Coke outfalls and in sites near the mouth of the Buffalo River subregion, 13 of which have mean PAH sediment concentrations below 1 mg/kg.

5.6.7 Tonawanda Canal Subregion

Of the 9 subregions in the Buffalo region, the Tonawanda Canal ranks seventh with respect to mean PAH concentration (1.34 mg/kg), fifth with respect to mean human carcinogenic risk score (1.7×10^{-5}), seventh with respect to mean human non-carcinogenic risk score (1.58×10^{-2}), eighth with respect to mean acute aquatic toxicity risk score (8.1×10^{-4}), and seventh with respect to overall ranking score (7 + 5 + 7 + 8 = 27).

Of the 31 reported sites in the Tonawanda Canal subregion, the 5 highest ranking sites, with respect to both overall ranking score and mean PAH sediment concentration in the subregion, are BUF 81-72 (G45), BUF 81-92 (G66), BUF 81-73 (G46), BUF 81-78 (G52), and BUF 81-87A (G59), respectively. The mean PAH sediment concentrations for these sites are 7.29, 4.66, 4.49, 4.45, and 2.58 mg/kg, respectively. The two highest ranking sites (BUF 81-72 and BUF 81-92) are located at storm sewer overflows. Sites BUF 81-73 (G46), BUF 81-78 (G52), and BUF 81-87A (G59) are located at the Chevrolet outfall, at the mouth of Two Mile Creek (downstream of the Tonawanda WWTP) and at the Hooker Chemical outfall, respectively.

Twenty of the 31 reported sites in the Tonawanda Canal subregion had mean PAH sediment concentrations less than 1 mg/kg or had non-detectable levels of PAH.

5.6.8 Lake Erie (South of Southern Entrance to Buffalo Harbor) Subregion

This subregion is located south of the southern entrance to Buffalo Harbor. Of the 9 subregions in the Buffalo region, the Lake Erie subregion ranks eighth with respect to mean PAH sediment concentration (0.93 mg/kg), mean human carcinogenic risk score (4×10^{-6}), mean human non-carcinogenic risk score (1.20×10^{-2}), and overall ranking score (8 + 8 + 8 + 7 = 31). It

ranks seventh with respect to mean acute aquatic toxicity risk score (1.34×10^{-3}).

Of the 33 reported sites in the Lake Erie/Buffalo Harbor subregion, the 4 highest ranking sites, with respect to both overall ranking score and mean PAH sediment concentration in the subregion, are U15, U10, U17, and U11, respectively. The mean PAH sediment concentrations at those sites are 8.71, 5.14, 2.15, and 1.68 mg/kg, respectively. All four sites are in Lake Erie north of the mouth of Smokes Creek and offshore from the Bethlehem Steel waste disposal site and a sediment dredging disposal site. Of the 29 remaining sites, both north and south of the mouth of Smokes Creek, 25 have mean PAH sediment concentrations below 1 mg/kg.

5.6.9 Upper Niagara/Chippawa Canal

Of the 9 subregions in the Buffalo region, the Upper Niagara River/Chippawa Canal subregion ranks ninth with respect to mean PAH sediment concentration (0.09 mg/kg), mean carcinogenic risk score (0.00), mean non-carcinogenic risk score (1.3×10^{-3}), mean acute aquatic toxicity risk score (2×10^{-5}), and overall ranking score (9 + 9 + 9 + 9 = 36).

Both of the 2 reported sampling sites in the Chippawa Canal and both of the 2 reported sampling sites in the Upper Niagara River have mean PAH sediment concentrations below 1 mg/kg. (Note that use of the terms "Upper" and "Lower" Niagara River are from a geographical rather than river flow perspective. Therefore, the segment of the Niagara River which flows into Lake Ontario is referred to as the Upper Niagara River and the segment which flows from Lake Erie as the Lower Niagara River).

5.6.10 Summary Buffalo Region

Although the Buffalo region is not in Region V of EPA, there is some information which can be derived from the Buffalo region which may be applicable to Region V. In particular, in addition to those sites associated with steel industry outfalls, there are a number of relatively high ranking sites within the Buffalo region which appear to be associated with other types of pollution sources. For example, the highest ranking site in the Two Mile

Creek subregion is in the vicinity of the Union Carbide Corp., Linde Div. Two of the highest ranking sites in the Lower Niagara River/Black Rock Canal subregion are located south of a waste disposal site on Squaw Island. Several of the 6 highest ranking sites in the Buffalo River subregion are at or just below the Buffalo Color outfall. The 2 highest ranking sites in the Tonawanda Canal subregion are in the vicinity of storm sewer overflows. The 4 highest ranking sites in the Lake Erie subregion are offshore of a Bethlehem Steel waste disposal site and a dredged sediment disposal site.

Although the above discussion indicates that sources other than the steel industry may contribute to PAH pollution, it should be pointed out that none of the mean PAH sediment concentrations for any of the above sites are nearly as high as those for sites in the Indiana, Ohio Steel and Ohio regions associated with steel coke oven outfalls. Further, the highest mean PAH concentration in the Buffalo region was associated with 2 Bethlehem Steel outfalls to Rush Creek. In addition, based on mean wastewater discharge flows and PAH concentrations in effluents, NRTC (1984) has estimated PAH loadings to the Niagara River system from a variety of point sources. Of an estimated mean PAH loading of 17.4 kg/day to the Niagara River System in 1981, the following industries were estimated to contribute the daily PAH loadings indicated in parenthesis: Donner-Coke (14.9 kg/day), Bethlehem Steel (1.1 kg/day), Buffalo Sewer Authority WWTP (0.7 kg/day), Union Carbide Linde Div. (0.4 kg/day), and 0.3 kg/day total from 3 separate WWTP's. Therefore, despite the relatively high PAH concentrations in sediment at and just downstream from the Buffalo Color outfall, Buffalo Color is not estimated to contribute significantly to PAH loading based on PAH effluent data. The PAH concentrations in the sediments at and just downstream of the Buffalo Color outfall may be due to some upstream mixing and dispersion from the Donner-Coke outfall. Both Donner-Coke and Bethlehem Steel ceased operations in 1982 and 1983, respectively.

5.7 DETROIT REGION

Of the 10 regions covered in this report, the Detroit region ranks sixth with respect to mean PAH sediment concentration (2.74 mg/kg) and mean human carcinogenic risk score (3.3×10^{-5}), eighth with respect to mean human non-

carcinogenic risk score (1.65×10^{-2}), seventh with respect to mean acute aquatic toxicity risk score (1.83×10^{-3}), and seventh with respect to overall ranking score (6 + 6 + 8 + 7 = 27).

Table 5-7 lists the mean PAH sediment concentrations (mg/kg), mean human carcinogenic risk scores ($\times 10^4$), mean human non-carcinogenic risk scores ($\times 10^2$), mean acute aquatic life risk scores ($\times 10^3$), overall ranking scores and associated rankings for subregions and sediment sampling sites in the Detroit region. Subregions within the Detroit region and sampling sites within the subregions are presented in order of increasing overall ranking score. Sediment sampling sites in the Detroit region are presented in Appendix B, Table B-2A. A map showing sediment sampling site locations in the Detroit River subregions was presented in Figure 3-4.

5.7.1 Detroit River/Tributaries Subregions

Of the 4 reported subregions in the Detroit region, the Detroit River/Tributaries subregion ranks first with respect to mean PAH sediment concentration (3.16 mg/kg), mean human carcinogenic risk score (3.7×10^{-5}), mean human non-carcinogenic risk score (1.94×10^{-2}), mean acute aquatic toxicity risk score (2.13×10^{-3}), and overall ranking score (1 + 1 + 1 + 1 = 4). Of 28 reported sites in the Detroit River/Tributaries subregion, the 8 highest ranking sites with respect to both overall ranking score and mean PAH sediment concentration are ROR 82-02, DTR 82-13, DTR 82-19, DTR 82-08, DTR 82-22, ROR 82-06, DTR 82-29, and ROR 82-07, respectively. The mean PAH sediment concentrations for those sites range from 4.34 to 8.94 mg/kg. Three of the eight highest ranking sites are located in the Rouge River at Zug Island or at and just below the Ford Motor Company Steel producing facilities. The other 5 sites are located in the upper Detroit River between Belle Island and the mouth of the Ecorse River. The third ranking site (DTR 82-19) is located below the National Steel outfall off Zug Island. The fifth ranking site (DTR 82-22) is located at the Slip Fuel Oil Corp.

Most of the 9th through the 28th and lowest ranking sites in the Detroit River/Tributaries subregion are located in the lower Detroit River south of the Ecorse River mouth, in the upper Detroit River north of or adjacent to Belle Island and in the Huron River. However, only 5 of the 28 reported sites

Table 5-7. Summary of Mean PAH Sediment Concentrations, Mean Risk Scores, and Rankings for Subregions and Sampling Sites in the Detroit Region

Subregions and sampling sites	Mean PAH sediment concentration (mg/kg) and (ranking)	Mean carcinogenic risk score ($\times 10^4$) and (ranking)	Mean non-carcinogenic risk score ($\times 10^2$) and (ranking)	Mean acute aquatic toxicity risk score ($\times 10^3$) and (ranking)	Overall ranking score and (ranking)
Detroit Region	**2.74 (6)**	**0.33 (6)**	**1.65 (8)**	**1.83 (7)**	**27 (7)**
Detroit River and Tributaries Subregion	3.16 (1)	0.37 (1)	1.94 (1)	2.13 (1)	4 (1)
ROR 82-02	8.94 (1)	1.03 (2)	5.59 (2)	6.94 (2)	7 (1)
DTR 82-13	8.08 (2)	1.13 (1)	4.13 (5)	4.74 (4)	12 (2)
DTR 82-19	6.94 (3)	0.44 (9)	6.79 (1)	11.6 (1)	14 (3)
DTR 82-08	5.76 (4)	0.76 (3)	2.81 (6)	2.16 (8)	21 (4)
DTR 82-22	5.00 (5)	0.71 (4)	2.42 (7)	1.51 (12)	28 (5)
ROR 82-06	4.71 (6)	0.34 (14)	4.87 (4)	4.14 (5)	29 (6)
DTR 82-29	4.49 (7)	0.62 (5)	2.34 (8)	1.29 (14)	34 (7)
ROR 82-07	4.34 (8)	0.27 (17)	4.95 (3)	2.80 (7)	35 (8)
DTR 82-38	3.99 (9)	0.54 (7)	1.95 (9)	1.76 (10)	35 (9)
DTR 82-32	3.45 (11)	0.33 (15)	1.53 (13)	6.06 (3)	42 (10)
DTR 82-43A	3.98 (10)	0.58 (6)	1.83 (11)	0.93 (18)	45 (11)
DTR 82-56	3.44 (12)	0.44 (9)	0.93 (20)	3.89 (6)	47 (12)
DTR 82-49	3.16 (13)	0.42 (10)	1.17 (16)	1.57 (11)	50 (13)
DTR 82-26	2.81 (16)	0.37 (13)	1.61 (12)	1.43 (13)	54 (14)
DTR 82-45	2.94 (14)	0.46 (8)	1.06 (18)	0.97 (17)	57 (15)
DTR 82-03	1.84 (20)	0.12 (20)	1.90 (10)	1.83 (9)	59 (16)
DTR 82-23	2.88 (15)	0.41 (11)	1.37 (15)	0.92 (19)	60 (17)
DTR 82-25	2.52 (17)	0.38 (12)	1.08 (17)	1.28 (15)	61 (18)
DTR 82-27	2.40 (18)	0.28 (16)	1.44 (14)	1.15 (16)	64 (19)
DTR 82-48	2.35 (19)	0.37 (13)	0.94 (19)	0.55 (21)	72 (20)
DTR 82-52	1.69 (21)	0.14 (19)	1.61 (13)	0.69 (20)	72 (21)
DTR 82-30	1.47 (22)	0.23 (18)	0.52 (22)	0.39 (22)	84 (22)
DTR 82-53	0.63 (23)	0.04 (21)	0.67 (21)	0.33 (23)	88 (23)
DTR 82-01	0.37 (24)	0.03 (22)	0.42 (23)	0.24 (24)	93 (24)

Table 5-7. Summary of Mean PAH Sediment Concentrations, Mean Risk Scores, and Rankings for Subregions and Sampling Sites in the Detroit Region
(continued)

Subregions and sampling sites	Mean PAH sediment concentration (mg/kg) and (ranking)	Mean carcinogenic risk score ($\times 10^4$) and (ranking)	Mean non-carcinogenic risk score ($\times 10^2$) and (ranking)	Mean acute aquatic toxicity risk score ($\times 10^3$) and (ranking)	Overall ranking score and (ranking)
DTR 82-05A	0.17 (25)	0.01 (23)	0.20 (24)	0.15 (25)	97 (25)
HUR 82-01	0.07 (27)	0.00 (24)	0.09 (25)	0.09 (26)	102 (26)
DTR 82-57	0.08 (26)	0.01 (23)	0.06 (26)	0.05 (27)	103 (27)
HUR 82-02	0.04 (28)	0.00 (24)	0.06 (26)	0.06 (28)	105 (28)
St. Clair River/Lake Subregion	1.54 (2)	0.21 (2)	1.01 (2)	0.97 (2)	8 (2)
CLI 81-03	2.45 (1)	0.45 (1)	1.36 (2)	0.78 (3)	7 (1)
CLI 81-05	2.16 (2)	0.27 (2)	1.33 (3)	1.75 (1)	8 (2)
CLI 81-04	1.87 (3)	0.00 (5)	1.65 (1)	1.62 (2)	11 (3)
CLI 81-02	0.80 (4)	0.11 (3)	0.43 (4)	0.77 (4)	15 (4)
CLI 81-01	0.35 (5)	0.03 (4)	0.30 (5)	0.35 (5)	19 (5)
Raisin River Subregion	0.64 (3)	0.09 (3)	0.37 (3)	0.65 (3)	12 (3)
MON 81-05	1.70 (1)	0.26 (1)	0.74 (1)	1.98 (1)	4 (1)
MON 81-04	0.70 (2)	0.11 (2)	0.41 (2)	1.03 (2)	8 (2)
NOAA	0.43 (3)	0.04 (3)	0.34 (3)	0.07 (5)	14 (3)
MON 81-02	0.21 (4)	0.02 (4)	0.20 (4)	0.12 (4)	16 (4)
MON 81-08	0.02 (5)	0.00 (5)	0.00 (5)	0.19 (3)	18 (5)
Lake Erie off Raisin River Subregion	0.19 (4)	0.01 (4)	0.19 (4)	0.05 (4)	16 (4)
NOAA 5KMN	0.38 (1)	0.02 (1)	0.35 (1)	0.09 (1)	4 (1)
NOAA 5KMN	0.24 (2)	0.01 (2)	0.27 (2)	0.06 (3)	9 (2)
NOAA 10KMN	0.21 (3)	0.01 (2)	0.23 (3)	0.07 (2)	10 (3)
NOAA 10KMS	0.07 (4)	0.01 (2)	0.06 (4)	0.01 (4)	14 (4)
NOAA 1KMS	0.06 (5)	0.01 (2)	0.05 (5)	0.01 (4)	16 (5)

in the Detroit River/Tributaries subregion have mean PAH concentrations below 1 mg/kg.

5.7.2 St. Clair River/Lake Subregion

Of the 4 reported subregions in the Detroit region, the St. Clair River/Lake subregion ranks second with respect to mean PAH sediment concentration (1.54 mg/kg), mean carcinogenic risk score (2.1×10^{-5}), mean non-carcinogenic risk score (1.01×10^{-2}), mean acute aquatic toxicity risk score (9.7×10^{-4}), and overall ranking score (2 + 2 + 2 + 2 = 8).

The five reported sites in the St. Clair River/Lake subregion are ranked in the following order with respect to both overall ranking score and PAH sediment concentration: CLI 81-03, CLI 81-05, CLI 81-04, CLI 81-02, and CLI 81-01, respectively. The mean PAH sediment concentrations for those sites are 2.45, 2.16, 1.87, 0.80, and 0.35 mg/kg. The two highest ranking sites are located in the St. Clair River at the Ft. River Bridge and in Lake St. Clair, 30 feet offshore, below the Clinton STP.

5.7.3 Raisin River Subregion

Of the 4 reported subregions in the Detroit region, the Raisin River subregion ranks third with respect to mean PAH sediment concentration (0.64 mg/kg), mean human carcinogenic risk score (9.0×10^{-6}), mean human non-carcinogenic risk score (3.7×10^{-3}), mean acute aquatic toxicity risk score (6.5×10^{-4}), and overall ranking score (3 + 3 + 3 + 3 = 12).

Of the 5 reported sites in the Raisin River subregion, the highest ranking site with respect to both overall ranking score and mean PAH sediment concentration is MON 81-05, which is located near the mouth of the Raisin River at the Ford Motor Co. RO outfall. Site MON 81-05 is the only site in the Raisin River subregion with a mean PAH sediment concentration exceeding 1 mg/kg (1.70 mg/kg). The second ranking site is MON 81-04, which is located downstream of the Monroe STP and has a mean PAH sediment concentration of 0.70 mg/kg.

5.7.4 Lake Erie Off Raisin River Mouth Subregion

Of the 4 reported subregions in the Detroit region, the Lake Erie subregion, off the mouth of the Raisin River, ranks fourth and last with respect to mean PAH sediment concentration (0.19 mg/kg), mean carcinogenic risk score (1.0×10^{-5}), mean non-carcinogenic risk score (1.9×10^{-3}), mean acute aquatic toxicity risk score (5×10^{-5}), and overall ranking score (4 + 4 + 4 + 4 = 16). All 5 of the reported sites in the Lake Erie subregion are within a 10 km arc of the mouth of the Raisin River. The mean PAH sediment concentrations for all 5 sites are less than 1 mg/kg.

5.8 MICHIGAN REGION

Of the 10 regions covered in this report, the Michigan region ranks eighth with respect to mean PAH sediment concentration (2.06 mg/kg), seventh with respect to mean human carcinogenic risk score (2.6×10^{-5}), seventh with respect to mean human non-carcinogenic risk score (1.70×10^{-2}), sixth with respect to mean acute aquatic toxicity risk score (2.07×10^{-3}), and eighth with respect to overall ranking score (8 + 7 + 7 + 6 = 28).

Table 5-8 lists the mean PAH sediment concentrations (mg/kg), mean human carcinogenic risk scores ($\times 10^4$), mean human non-carcinogenic risk scores ($\times 10^2$), mean acute aquatic life risk scores ($\times 10^3$), overall ranking scores and associated rankings (in parenthesis) for subregions and sediment sampling sites within subregions, in order of increasing overall ranking score. Descriptions of the sediment sampling sites in the Michigan region are presented in Appendix B, Table B-6A.

5.8.1 St. Joseph Harbor Subregion

Of the 5 reported subregions in the Michigan region, the St. Joseph Harbor subregion ranks first with respect to mean PAH sediment concentration (4.53 mg/kg), second with respect to mean human carcinogenic risk score (2.4×10^{-5}), first with respect to mean human non-carcinogenic risk score (4.60×10^{-2}), second with respect to mean acute aquatic toxicity risk score (2.46×10^{-3}), and first with respect to overall ranking score (1 + 2 + 1 2 =6).

Table 5-8. Summary of Mean PAH Sediment Concentrations, Mean Risk Scores, and Rankings for Subregions and Sampling Sites in the Michigan Region

Subregions and sampling sites	Mean PAH sediment concentration (mg/kg) and (ranking)	Mean carcinogenic risk score ($\times 10^4$) and (ranking)	Mean non-carcinogenic risk score ($\times 10^2$) and (ranking)	Mean acute aquatic toxicity risk score ($\times 10^3$) and (ranking)	Overall ranking score and (ranking)
Michigan Region	**2.06 (8)**	**0.26 (7)**	**1.70 (7)**	**2.07 (6)**	**28 (8)**
St. Joseph Harbor Region	4.53 (2)	0.24 (2)	4.60 (1)	2.46 (2)	6 (1)
SJH 81-05	9.10 (1)	0.00 (4)	10.1 (1)	7.73 (1)	8 (1)
SJH 81-17	10.1 (2)	0.00 (4)	9.56 (2)	4.22 (3)	10 (2)
SJH 81-02	8.45 (3)	0.00 (4)	7.76 (3)	2.80 (4)	14 (3)
SJH 81-15	6.13 (4)	0.00 (4)	6.46 (4)	4.56 (2)	14 (4)
SJH 81-16	2.95 (5)	0.53 (1)	1.68 (5)	0.88 (6)	17 (5)
SJH 81-20	0.80 (6)	0.00 (4)	0.81 (6)	1.06 (5)	21 (6)
SJH 81-21	0.65 (7)	0.11 (2)	0.42 (7)	0.28 (7)	23 (7)
SJH 81-18	0.49 (8)	0.07 (3)	0.29 (8)	0.19 (8)	27 (8)
Manistee Subregion	2.86 (2)	0.56 (1)	1.39 (2)	4.48 (1)	6 (2)
MST 81-06	9.38 (1)	1.43 (1)	4.12 (1)	5.08 (3)	6 (1)
MST 81-08	4.33 (2)	0.23 (2)	3.04 (2)	14.6 (1)	7 (2)
MST 81-01	2.22 (3)	0.00 (5)	1.18 (3)	9.52 (2)	13 (3)
MST 81-03	1.13 (4)	0.00 (5)	0.69 (4)	3.99 (4)	17 (4)
MST 81-13	0.96 (5)	0.10 (3)	0.67 (5)	1.25 (6)	19 (5)
MST 81-07	0.45 (7)	0.00 (5)	0.23 (7)	1.97 (5)	24 (6)
MST 81-12	0.67 (8)	0.06 (4)	0.55 (6)	0.08 (9)	27 (7)
MST 81-09	0.20 (9)	0.00 (5)	0.11 (8)	0.74 (8)	30 (8)
MST 81-04	0.19 (10)	0.00 (5)	0.11 (8)	1.05 (7)	30 (9)

Table 5-8. Summary of Mean PAH Sediment Concentrations, Mean Risk Scores, and Rankings for Subregions and Sampling Sites in the Michigan Region
(continued)

Subregions and sampling sites	Mean PAH sediment concentration (mg/kg) and (ranking)	Mean carcinogenic risk score ($\times 10^4$) and (ranking)	Mean non-carcinogenic risk score ($\times 10^2$) and (ranking)	Mean acute aquatic toxicity risk score ($\times 10^3$) and (ranking)	Overall ranking score and (ranking)
Manistique Subregion	0.26 (3)	0.03 (3)	0.15 (4)	0.40 (3)	13 (3)
MTQ 81-01	0.63 (1)	0.04 (2)	0.34 (2)	1.62 (1)	6 (1)
MTQ 81-14	0.55 (2)	0.06 (1)	0.39 (1)	0.05 (5)	9 (2)
MTQ 81-10	0.20 (3)	0.03 (3)	0.14 (3)	0.21 (3)	12 (3)
MTQ 81-21	0.10 (4)	0.00 (4)	0.08 (4)	0.21 (3)	15 (4)
MTQ 81-12	0.07 (5)	0.00 (4)	0.00 (6)	0.47 (2)	17 (5)
MTQ 81-17	0.03 (6)	0.00 (4)	0.01 (5)	0.14 (4)	19 (6)
MTQ 81-02	0.01 (7)	0.00 (4)	0.01 (5)	0.00 (6)	22 (7)
Kalamazoo River Subregion	0.17 (4)	0.00 (5)	0.17 (3)	0.15 (5)	17 (4)
KAL 81-07	0.23 (1)	0.00 (1)	0.22 (1)	0.18 (2)	5 (1)
KAL 81-05	0.19 (2)	0.00 (1)	0.20 (2)	0.14 (3)	8 (2)
KAL 81-06	0.06 (4)	0.00 (1)	0.04 (4)	0.21 (1)	10 (3)
KAL 81-02	0.11 (3)	0.00 (1)	0.10 (3)	0.10 (4)	11 (4)
Muskegon/New Buffalo Subregion	0.13 (5)	0.02 (4)	0.10 (5)	0.36 (4)	18 (5)
MUS 81-13	0.31 (1)	0.03 (1)	0.27 (1)	0.80 (1)	4 (1)
MUS 81-01	0.12 (2)	0.02 (2)	0.06 (2)	0.31 (2)	8 (2)
NBH 68-03	0.07 (3)	0.00 (4)	0.05 (3)	0.16 (3)	13 (3)
MUS 81-14	0.06 (4)	0.01 (3)	0.03 (4)	0.13 (4)	15 (4)
NBH 68-01	0.02 (5)	0.00 (4)	0.01 (5)	0.09 (5)	19 (5)

Of the 8 reported sites in the St. Joseph subregion, the five highest ranking sediment sampling sites with respect to overall ranking score are SJH 81-05, SJH 81-17, SJH 81-02, SJH 81-15, and SJH 81-16, respectively. These sites have mean PAH sediment concentrations of 9.20, 10.1, 8.45, 6.13, and 2.95 mg/kg, respectively. The mean PAH sediment concentrations of the 3 lowest ranking sites are less than 1 mg/kg. All 8 sites are located in St. Joseph Harbor except for the highest ranking site, SJH 81-05, which may be located in the St. Joseph River.

5.8.2 Manistee Subregion

Of the 5 reported subregions in the Michigan region, the Manistee subregion ranks second with respect to mean PAH sediment concentration (2.86 mg/kg), first with respect to mean human carcinogenic risk score (5.6×10^{-5}), second with respect to mean human non-carcinogenic risk score (1.39×10^{-2}), first with respect to mean acute aquatic toxicity risk score (4.48×10^{-3}), and second with respect to overall ranking score (2 + 1 + 2 + 1 = 6). (Although the overall ranking score of the Manistee subregion is the same as for the St. Joseph Harbor subregion, it was ranked second based upon its lower mean PAH sediment concentration.)

Of the 9 reported sites in the Manistee subregion, the 4 highest ranking sites with respect to both overall ranking score and mean PAH sediment concentration are MST 81-06, MST 81-08, MST 81-01, and MST 81-03, respectively. The mean PAH sediment concentrations for these sites are 9.38, 4.33, 2.22, and 1.13 mg/kg, respectively. The mean PAH sediment concentrations for the remaining 5 sites are all less than 1 mg/kg.

All 9 reported sites for the Manistee subregion are located in the harbor. Sites MST-81-06, MST 81-08, MST 81-01, and MST 81-03 are located 300m south of Great Lakes Chem. Corp., at Standards Lime and Cement Company, south of Pkg. Corp. of America, and 2000 meters north of Little Manistee River, respectively.

5.8.3 Manistique Subregion

Of the 5 reported subregions in the Michigan region, the Manistique subregion ranks third with respect to mean PAH sediment concentration (0.26 mg/kg), mean human carcinogenic risk score (3.0×10^{-6}), mean acute aquatic toxicity risk score (4.0×10^{-4}), and overall ranking score (3 + 3 + 4 + 3 = 13). It ranks fourth with respect to mean human non-carcinogenic risk score (1.5×10^{-3}).

All of the 7 reported sampling sites in the Manistique subregion are located in Manistique Harbor and all have mean PAH sediment concentrations less than 1 mg/kg.

5.8.4 Kalamazoo River Subregion

Of the 5 reported subregions in the Michigan region, the Kalamazoo River subregion ranks fourth with respect to mean PAH sediment concentration (0.17 mg/kg), fifth with respect to mean human carcinogenic risk score (0.0), third with respect to mean human non-carcinogenic risk score (1.7×10^{-3}), fifth with respect to mean acute aquatic toxicity risk score (1.5×10^{-4}), and fourth with respect to overall ranking score (4 + 5 + 3 + 5 = 17).

All 4 of the reported sites in the Kalamazoo River subregion are located in the Kalamazoo River in West Allegany County. All 4 sites have mean PAH sediment concentrations well below 1.0 mg/kg.

5.8.5 Muskegon/New Buffalo Harbor Subregion

Of the 5 reported subregions in the Michigan region, the Muskegon/New Buffalo Harbor subregion ranks fifth with respect to mean PAH sediment concentration (0.13 mg/kg), fourth with respect to mean human carcinogenic risk score (2.0×10^{-6}), fifth with respect to mean human non-carcinogenic risk score (1.0×10^{-3}), fourth with respect to mean acute aquatic toxicity risk score (3.6×10^{-4}), and fifth with respect to overall ranking score (5 + 4 + 5 + 4 = 8).

All 3 reported sites in the Muskegon area and both reported sites in the New Buffalo area are located in the harbor. The mean PAH sediment concentrations for all 5 sites are well below 1.0 mg/kg.

5.9 LAKE SUPERIOR REGION

Of the 10 regions covered in this report, the Lake Superior region ranks ninth with respect to mean PAH sediment concentration (1.15 mg/kg), mean human carcinogenic risk score (1.0×10^{-5}), mean human non-carcinogenic risk score (9.1×10^{-3}) and overall ranking score (9 + 9 + 9 + 8 = 26). It ranks eighth with respect to mean acute aquatic toxicity risk score.

Table 5-9 lists mean PAH sediment concentrations (mg/kg), mean human carcinogenic risk scores ($\times 10^{4}$), mean human non-carcinogenic risk scores ($\times 10^{2}$), mean acute aquatic toxicity risk scores ($\times 10^{3}$), overall ranking scores and associated rankings (in parenthesis) for subregions and sediment sampling sites in the Lake Superior region.

5.9.1 Ashland Subregion

Of the two reported subregions in the Lake Superior region, the Ashland subregion ranks first with respect to mean PAH sediment concentration (2.75 mg/kg), mean human carcinogenic risk score (1.0×10^{-5}), mean human non-carcinogenic risk score (2.20×10^{-2}), mean acute aquatic toxicity risk score (3.29×10^{-3}) and overall ranking score (1 + 1 + 1 + 1 = 2).

The three reported sediment sampling sites in the Ashland subregion rank in the following order with respect to both overall ranking score and mean PAH sediment concentration: ASH 81-03, ASH 81-01, and ASH 81-05. The mean PAH sediment concentrations for those sites are 6.78, 1.05, and 0.43 mg/kg, respectively.

All three reported sampling sites in the Ashland subregion are in Chequamegon Bay (Lake Superior) just offshore of Ashland, Wisconsin. The highest ranking site (ASH 81-03) is located just off a sewage treatment plant outfall and is also in the vicinity of coal storage and handling facilities. The second ranking site (ASH 81-01) is located off a power plant outfall.

Table 5-9. Summary of Mean PAH Sediment Concentrations, Mean Risk Scores, and Rankings for Subregions and Sampling Sites in the Lake Superior Region

Subregions and sampling sites	Mean PAH sediment concentration (mg/kg) and (ranking)	Mean carcinogenic risk score ($\times 10^4$) and (ranking)	Mean non-carcinogenic risk score ($\times 10^2$) and (ranking)	Mean acute aquatic toxicity risk score ($\times 10^3$) and (ranking)	Overall ranking score and (ranking)
Lake Superior Region	**1.15 (9)**	**0.10 (9)**	**0.91 (9)**	**1.34 (8)**	**35 (9)**
Ashland Subregion	2.75 (1)	0.23 (1)	2.20 (1)	3.29 (1)	4 (1)
ASH 81-03	6.78 (1)	0.62 (1)	5.45 (1)	4.11 (2)	5 (1)
ASH 81-01	1.05 (2)	0.00 (3)	1.09 (2)	5.76 (1)	8 (2)
ASH 81-05	0.43 (3)	0.08 (2)	0.04 (3)	0.00 (3)	11 (3)
Miscellaneous Lake Superior Subregion	0.03 (2)	0.00 (2)	0.03 (2)	0.01 (2)	8 (2)
LAN 81-04	0.11 (1)	0.01 (1)	0.10 (1)	0.02 (1)	4 (1)
Lake Superior	0.05 (2)	0.00 (2)	0.06 (2)	0.01 (2)	8 (2)
LAN 81-03	0.00 (3)	0.00 (2)	0.00 (3)	0.00 (3)	11 (3)
LAN 81-07	0.00 (3)	0.00 (2)	0.00 (3)	0.00 (3)	11 (4)
BRH 81-03	0.00 (3)	0.00 (2)	0.00 (3)	0.00 (3)	11 (5)

5.9.2 Miscellaneous Lake Superior Subregion

Of the two reported subregions in the Lake Superior region, the Miscellaneous Lake Superior subregion ranks second (last) with respect to mean PAH sediment concentration (0.03 mg/k), mean human carcinogenic risk score (0.00), mean human non-carcinogenic risk score (3.0×10^{-4}), mean acute aquatic toxicity risk score (1.0×10^{-5}), and overall ranking score (2 + 2 + 2 + 2 = 8). None of the five reported sampling sites in the Miscellaneous Lake Superior subregion has a mean PAH sediment concentration above 0.11 mg/kg.

5.10 OPEN LAKE MICHIGAN REGION

Of the 10 regions covered in this report, the Open Lake Michigan region ranks tenth and last with respect to mean PAH sediment concentration (0.40 mg/kg), mean human carcinogenic risk score (2×10^{-6}), mean human non-carcinogenic risk score (4.1×10^{-3}), mean acute aquatic toxicity risk score (1.0×10^{4}), and overall ranking score (10 + 10 + 10 + 10 = 40).

Table 5-10 lists mean PAH sediment concentrations (mg/kg), mean human carcinogenic risk scores ($\times 10^{4}$), mean human non-carcinogenic risk scores ($\times 10^{2}$), mean acute aquatic toxicity risk scores ($\times 10^{3}$), overall ranking scores and associated rankings (in parenthesis) for subregions and sediment sampling sites in the Open Lake Michigan region.

Only 1 of the 12 reported sites in the Open Lake Michigan region has a mean PAH sediment concentration exceeding 1 mg/kg (1.29 mg/kg for T3). All of the 12 sites are located in the southern half of Lake Michigan.

Table 5-10. Summary of Mean PAH Sediment Concentrations, Mean Risk Scores, and Rankings for Subregions and Sampling Sites in the Lake Michigan Region

Subregions and sampling sites	Mean PAH sediment concentration (mg/kg) and (ranking)	Mean carcinogenic risk score ($\times 10^4$) and (ranking)	Mean non-carcinogenic risk score ($\times 10^2$) and (ranking)	Mean acute aquatic toxicity risk score ($\times 10^3$) and (ranking)	Overall ranking score and (ranking)
Open Lake Michigan Region	**0.40 (10)**	**0.02 (10)**	**0.41 (10)**	**0.10 (10)**	**40 (10)**
T3	1.29 (1)	0.07 (1)	1.45 (1)	0.35 (1)	4 (1)
T2	0.72 (2)	0.03 (3)	0.82 (2)	0.26 (2)	9 (2)
NOAA 60	0.65 (3)	0.04 (2)	0.63 (4)	0.17 (3)	12 (3)
T5	0.55 (4)	0.03 (3)	0.66 (3)	0.15 (4)	14 (4)
T7	0.49 (5)	0.04 (2)	0.44 (5)	0.09 (5)	17 (5)
T8	0.38 (6)	0.03 (3)	0.33 (7)	0.07 (6)	22 (6)
T11	0.37 (7)	0.03 (3)	0.33 (7)	0.07 (6)	23 (7)
T4	0.34 (8)	0.02 (4)	0.37 (6)	0.09 (5)	23 (8)
NOAA 45	0.11 (9)	0.00 (5)	0.13 (8)	0.03 (7)	29 (9)
T6	0.04 (10)	0.00 (5)	0.04 (9)	0.01 (8)	32 (10)
T1	0.03 (11)	0.00 (5)	0.03 (10)	0.01 (8)	34 (11)
NOAA 24	0.03 (11)	0.00 (5)	0.02 (11)	0.00 (9)	36 (12)

6. CONCLUSIONS AND RECOMMENDATIONS

Chapter 6 is divided into the following 3 sections: Conclusions with Respect to Sources of PAH Contamination in Sediments (6.1), Recommendations with Respect to the Sampling and Analysis of Sediments for PAHs (6.2), and Recommendations with Respect to the Sampling and Analysis of Fish, Shellfish, and Drinking Water for PAHs (6.3).

6.1 CONCLUSIONS WITH RESPECT TO SOURCES OF PAH CONTAMINATION IN SEDIMENT

Table 6-1 combines, in decreasing order, mean PAH sediment concentrations from sites below coke plants and other steel industry outfalls with mean PAH sediment concentrations above 5 mg/kg from all other sites. The type of outfall associated with each site is described in the table. Conclusions with respect to sources of PAHs in sediments are presented below, based on Table 6-1 and Chapter 2.

The highest mean PAH sediment concentrations in the 10 regions considered in this report are generally in the vicinity of coke plants or otherwise unidentified steel industry outfalls (Table 6-1). It can be seen that most of the mean PAH sediment concentrations exceed 5 mg/kg with several exceeding 100 mg/kg. Mean PAH sediment concentrations exceeded 10 mg/kg in at least some of the sites in the vicinity of the following coke plants and/or otherwise unidentified steel industry outfalls:

a. U.S. Steel coke Plant outfalls 002, 005, 007, 010 to the Grand Calumet River in Indiana (still operational but samples taken in 1972)

b. Inland Steel Coke Plant outfall 012 (possibly) to the Indiana Harbor in Indiana (operational)

c. U.S. Steel coke Plant (in Lorain) outfall 002 to the Black River in Ohio (shut down)

d. Youngstown Sheet and Tube Campbell Works Coke Plant outfall to the Mahoning River in Ohio (shut down)

e. LTV (Republic) Steel (in Youngstown) Coke Plant outfall to the Mahoning River in Ohio (shut down)

Table 6-1
Mean PAH Sediment Concentrations Near Coke Ovens or Other Steel Industry Outfalls and Mean PAH Sediment Concentrations Greater Than 5 mg/kg at Sites Which Are Not Near Coke Oven Outfalls

Region	Subregion	Site	Mean PAH Sediment Concentration (mg/kg)	Date	Outfalls
Indiana	Grand Calumet River	10800	2650	1972	U.S. Steel Coke Plant outfalls U.S. Steel Blast Furnace outfall U.S. Steel Sintering Plant outfall
Indiana	Grand Calumet River	4560	1030	1972	U.S. Steel Coke Plant outfalls 002,005,007,010
Indiana	Grand Calumet River	6975	960	1972	U.S. Steel Coke Plant outfalls 002,005,007,010 U.S. Steel Blast Furnace outfalls U.S. Steel Sintering Plant outfalls
Indiana	Indiana Harbor	ACOE	226	1985	Possibly at Inland Steel Coke Plant outfall 012 but exact location not specified
Indiana	Grand Calumet River	3300	150	1972	U.S. Steel Coke Plant outfalls 002,005,007,010
Indiana	Grand Calumet River	12900	133	1972	Downstream of U.S. Steel Bar Plate Mill and Basic Oxygen Furnace outfalls 20,28,30,32
Indiana	Grand Calumet River	C3	97.3	1983	Downstream of U.S. Steel Bar Plate Mill and Basic Oxygen Process Furnace outfall 28,30,32,33
Indiana	Grand Calumet River	26900	84.4	1972	At the Gary POTW outfall Downstream of U.S. Steel Hot Strip Mill and Miscellaneous Finishing Operations outfall 034
Indiana	Grand Calumet River	8700	77.0	1972	Downstream and/or in the vicinity of U.S. Steel Bar Mill Plate and Basic Oxygen Furnace outfalls 28,30,32
Ohio	Cuyahoga River	CUY 82-18	75.7	1982	LTV (Republic) Steel - Coke Plant
Indiana	Indiana Harbor Canal	Indianapolis Blvd.	55.3	1977	Downstream of CF Petroleum outfall
NY/PA Harbors	Dunkirk/Erie	DNK82-06	46.8	1982	At an unidentified outfall beside Main Beach
Ohio Steel	Black River/Lorain	LOR 82-14.9	41.0	1982	U.S. Steel Coke Plant outfall 002
Indiana	Grand Calumet River	500	39.0	--	Just upstream of U.S. Steel Coke Plant outfalls 002,005
Indiana	Indiana Harbor Canal	Columbus Drive	28.7	1977	Downstream of Blau-Knox Foundry and Union Carbide outfalls

Table 6-1 (continued)
Mean PAH Sediment Concentrations Near Coke Ovens or Other Steel Industry Outfalls and Mean PAH Sediment Concentrations Greater Than 5 mg/kg at Sites Which Are Not Near Coke Oven Outfalls

Region	Subregion	Site	Mean PAH Sediment Concentration (mg/kg)	Date	Outfalls
Milwaukee	---	MIL80-15	24.1	1980	In Milwaukee River at Walnut St. but no outfalls identified
Indiana	Indiana Harbor Canal	Forks	22.3	1977	At Forks between Lake George and Grand Calumet Branches and at J&L Steel Flat Rolling outfall
Indiana	Grand Calumet River	100	22.2	--	Just upstream of U.S. Steel Coke Plant outfalls 002,005
Milwaukee		MIL80-23	21.7	1980	In Menomokee River at 25th St. but no outfalls identified
Buffalo	Rush Creek	BUF 81-03	21.1	1981	Location not specified but Rush Creek received wastewater from Bethlehem Steel
Ohio Steel	Black River/Lorain	B-2	21.0	Before 1982	U.S. Steel Coke Plant outfall 002
Ohio Steel	Black River/Lorain	USS-4	21.0	1984	U.S. Steel Coke Plant outfall 002
Buffalo	Scajaquada Creek	N37	20.8	--	At Mouth of Scajaquada Creek but no outfalls identified
Buffalo	Scajaquada Creek	N43	20.2	--	In Scajaquada Creek but no outfalls identified
Milwaukee		MIL80-06	19.9	1980	In Kinnickinnic River at Basin but no outfalls identified
Buffalo	Buffalo Harbor	BUF 81-55	19.2	1981	Mouth of Lackawana Ship Canal (Receives wastewater from Bethlehem Steel)
Indiana	Grand Calumet River	C5	16.5	1983	Downstream of Vulcan outfall. In vicinity of CITGO Petroleum and Explorer Pipeline outfalls
Ohio Steel	Mahoning River	Struthers (Campbell)	16.6	1975	Youngstown Sheet and Tube Campbell Works Coke
Ohio Steel	Mahoning River	Youngstown	15.9	1975	LTV (Republic) Steel - Youngstown Coke Plant outfall
Buffalo	Buffalo Harbor	BUF 81-12	15.4	1981	Located at Buffalo Color but only short distance upstream of Republic Steel and Donner Coke outfall
Indiana	Indiana Harbor Canal	Canal Street	15.3	1977	In the vicinity of a storm sewer overflow, a combined sewer overflow and the American Steel Foundry outfall
Indiana	Grand Calumet River	C4	15.1	1983	Downstream of U.S. Steel Hot Strip Mill and Miscellaneous Finishing Operations outfall 034 downstream of Gary POTW outfall 001
Milwaukee		MIL90-08	15.0	1980	In Kinnickinnic River at Kinnickinnic Ave. but no outfalls identified

Table 6-1 (continued)
Mean PAH Sediment Concentrations Near Coke Ovens or Other Steel Industry Outfalls and Mean PAH Sediment Concentrations Greater Than 5 mg/kg at Sites Which Are Not Near Coke Oven Outfalls

Region	Subregion	Site	Mean PAH Sediment Concentration (mg/kg)	Date	Outfalls
Milwaukee	---	MIL-80-22	15.0	1980	In Menomonee River at Muskego Ave. but no outfalls identified
Ohio Steel	Black River/Lorain	19472(8B)	14.9	1974	U.S. Steel Coke Plant outfall 002
Indiana	Grand Calumet River	C7	14.8	1983	At East Chicago POTW outfall
Buffalo	Two Mile Creek	4-3	13.9	--	In tributary to 2 Mile Creek but no outfalls identified
Milwaukee	---	MIL80-13	13.5	1980	In Milwaukee River at St. Paul Street but no outfalls identified
Buffalo	Buffalo River	BUF81-26	13.3	1981	Located in vicinity of combined sewer
Buffalo	Scajaquada Creek	N42	11.7	--	In Scajaquada Creek but no outfalls identified
Ohio	Ashtabula	AST79-15	11.1	1979	In Fields Brook Creek but no outfalls identified
NY/PA Harbors	Dunkirk/Erie	ERH82-05	10.8	1982	In Erie, PA, but no outfalls identified
Indiana	Grand Calumet River	C2	10.4	1983	Downstream of U.S. Steel Bar Plate Mill and Basic Oxygen Process Furnace outfalls 28,30,32,33
Michigan	St. Joseph Harbor	SJH81-17	10.1	1981	St. Joseph Harbor but no outfalls identified
Buffalo	Two Mile Creek	N31	10.0	--	In the vicinity of Union Carbide-Linde Div. (Metal Finishing)
Buffalo	Buffalo Harbor	3-1 (N9)	9.57	1981	200 Yds. from entrance to Union Ship Canal at Bethlehem Steel
Buffalo	Lower (South) Niagara River	BUF81-31	9.45	1981	Located at mouth of Scajaquada Creek but no outfalls identified
Michigan	St. Joseph Harbor	SJH81-05	9.20	1981	St. Joseph Harbor but no outfalls identified
Ohio Steel	Black River/Lorain	USS-1	9.39	1984	U.S. Steel Coke Plant outfall 002
Michigan	Manistee	MST81-06	9.28	--	Manistee Harbor south of Great Lakes Chemical Corp.
Detroit	Detroit River/Tributaries	ROR 82-02 (Rouge River)	8.94	1982	Rouge River at Zug Island below Ford Motor Company (Coke Plant?)
Buffalo	Lake Erie/Buffalo	U15	8.71	--	Offshore from a Bethlehem Waste Disposal Site and a dredged sediment disposal site
Ohio	Ashtabula	AST79-16	8.70	1979	In Fields Brook Creek but no outfalls identified
Michigan	St. Joseph Harbor	SJH81-02	8.45	1981	St. Joseph Harbor but no outfalls identified
Indiana	Indiana Harbor Canal	508	8.27	--	Downstream of Fork and J&L Steel Flat Rolling outfall

Table 6-1 (continued)
Mean PAH Sediment Concentrations Near Coke Ovens or Other Steel Industry Outfalls and Mean PAH Sediment Concentrations Greater Than 5 mg/kg at Sites Which Are Not Near Coke Oven Outfalls

Region	Subregion	Site	Mean PAH Sediment Concentration (mg/kg)	Date	Outfalls
Detroit	Detroit River/Tributaries	DTR82-13	8.08	1982	In Detroit River between Park David and Belle Island. No outfalls identified
Buffalo	Tonawanda Canal	BUF81-72	7.29	1981	Located at a storm sewer overflow
Indiana	Indiana Harbor Canal	509	7.17	--	At Mouth of Indiana Harbor Canal Downstream of Inland Steel Power House outfall 008 and J&L Steel Blast Furnace outfall 001
Detroit	Detroit River/Tributaries	DTR 82-19	6.94	1982	Below National Steel (Coke Plant?) outfall off Zug Island
Lake Superior	Ashland	ASH81-03	6.78	1981	In Lake Superior offshore to Ashland POTW outfall
Indiana	Grand Calumet River	35600	6.70	1972	Downstream of Vulcan (Secondary Tin) outfall and in vicinity of CITGO Petroleum and Explorer Pipe outfalls
Buffalo	Lower (South) Niagara River	BUF81-45	6.68	1981	Located in Black Rock Canal south of Waste Disposal Site on Squaw Island
Buffalo	Buffalo Harbor	3-3 (N11)	6.42	1981	Mouth of Lackawana Ship Canal (Received wastewater from Bethlehem Steel)
NY/PA Harbors	Dunkirk/Erie	ERH82-03	6.35	1982	In Erie, PA, but no outfalls identified
Michigan	St. Joseph Harbor	SJH81-15	6.13	1981	St. Joseph Harbor but no outfalls identified
Indiana	Indiana Harbor Canal	S13	6.03	--	In Lake George Branch upstream of Petroleum outfall
Indiana	Indiana Harbor Canal	C16	6.00	--	Downstream of Fork and J&L Steel Flat Rolling outfall 001
Milwaukee	---	MIL80-17	5.92	1980	Confluence of Menomonee River and Canal but no outfalls identified
Buffalo	Buffalo Harbor	3-2 (N10)	5.88	1981	Mouth of Union Ship Canal (Received wastewater from Bethlehem Steel)
Detroit	Detroit River/Tributaries	DTR82-08	5.76	1982	Upper Detroit River between Park Davis and Belle Island
Indiana	Indiana Harbor Canal	S17	5.43	--	In Lake George Branch downstream of CF Petroleum
Buffalo	Buffalo Harbor	BUF 81-60	5.21	1981	Mouth of Union Ship Canal (Received wastewater from Bethlehem Steel)
Buffalo	Lake Erie/Buffalo	U10	5.14	--	Offshore from a Bethlehem Steel Waste disposal site and a dredged sediment disposal site

Table 6-1 (continued)
Mean PAH Sediment Concentrations Near Coke Ovens or Other Steel Industry Outfalls and Mean PAH Sediment Concentrations Greater Than 5 mg/kg at Sites Which Are Not Near Coke Oven Outfalls

Region	Subregion	Site	Mean PAH Sediment Concentration (mg/kg)	Date	Outfalls
Ohio	Cuyahoga River	CUY82-08	5.08	1982	At Williams Company outfall
Detroit	Detroit River/Tributaries	DTR82-22	5.00	1982	Detroit River at Slip Fuel Oil Co.
Buffalo	Buffalo Harbor	1-5 (N19)	4.76	1981	Located downstream of Buffalo Color but only a short distance upstream of Republic Steel and Donner Coke outfall
Detroit	Detroit River/Tributaries	DTR 82-06	4.71	1982	Rouge River below Ford Motor Co. (Coke Plant?)
Detroit	Detroit River/Tributaries	DTR 82-07	4.34	1982	Rouge River at Ford Motor Co. (Coke Plant?)
Detroit	Detroit River/Tributaries	DTR 82-26	2.81	1982	Below National Steel outfall
Buffalo	Buffalo Harbor	1-6 (N20)	2.78	1981	Located downstream of Republic Steel and in vicinity of Donner Coke outfall
Ohio	Cuyahoga River	CUY 82-13	2.42	1982	In vicinity of U.S. Steel outfall
Detroit	Detroit River/Tributaries	DTR 82-27	2.40	1982	Below National Steel outfall
Buffalo	Smokes Creek	Sm. Cr. (A)	1.30	Before 1983	Location not specified but Smokes Creek received wastewater from Bethlehem Steel
Ohio Steel	Mahoning River	Warren	0.05	1975	LTV (Republic) Steel - Warren Coke Plant outfall

f. LTV (Republic) Steel (in Cleveland) Coke Plant outfall to the Cuyahoga River (operational)

g. Bethlehem Steel (in Buffalo) outfalls to Rush Creek and the Lackawana Ship Canal (shut down)

h. Donner-Coke (in Buffalo) outfall to Buffalo River (shut down).

Mean PAH sediment concentrations were below 10 mg/kg at all of the reported sites in the vicinity of the following coke plant or otherwise unidentified steel industry outfalls:

a. LTV (Republic) Steel (in Warren) outfall to the Mahoning River

b. Ford Motor Company Coke Plant (in Detroit) outfall to the Rouge River

c. National Steel Coke Plant (in Detroit) outfall to the Detroit River.

Although mean PAH sediment concentrations at sampling sites in the vicinity of coke plant outfalls are generally greater, there were numerous sites not identified as being in the vicinity of coke oven outfalls which had mean PAH sediment concentrations exceeding 5 mg/kg (Table 6-1). Such sites included the following:

a. Numerous sites in the Grand Calumet River in the vicinity of blast furnace, sintering plant, bar mill plate, basic oxygen process furnace, miscellaneous finishing operation and other non-coke plant steel industry outfalls

b. Several sites in the Indiana Harbor Canal in the vicinity of steel industry blast furnace or flat rolling operation outfalls

c. Several sites in the vicinity of Petroleum handling and storage facilities including ones in the Grand Calumet River, Indiana Harbor Canal, and Detroit River

d. Several sites in the vicinity of storm or combined sewer outfalls or overflows including ones in the Indiana Harbor Canal, Buffalo River and Tonawanda Canal (Niagara River)

e. Several sites in the vicinity of POTW outfalls including ones in the vicinity of the Gary and East Chigaco POTW outfalls to the Grand Calumet River and the Ashland POTW outfall to Lake Superior

f. Sites in the vicinity of waste disposal sites and dredged sediment sites including one adjacent to a dredged disposal site along the Black River in Lorain and two sites in Lake Erie off a Bethlehem Steel Waste disposal site and a dredged sediment disposal site in Buffalo

g. Sites in the vicinity of miscellaneous outfalls including the Blau-Knox Foundry, American Steel Foundry, and Union Carbide (unidentified) outfalls to the Indiana Harbor Canal, the Vulcan (Secondary Tin) outfall to the Grand Calumet River, the Williams Company (unidentified) outfall to the Cuyahoga River, the Union Carbide-Linde Division (Metal Farbrication and Finishing of Cryogenic Hardware) outfall to Two Mile Creek in Buffalo and the Great Lake Chemical Corp. outfall to Manistee Harbor (Lake Michigan)

h. Numerous sites at which no outfalls were identified including 6 in the Milwaukee region, 2 in the Ashtabula subregion of the Ohio region, 3 in the Dunkirk/Erie subregion of the NY/PA Harbors region, 3 in the Scajaquada Creek subregion of the Buffalo region, 1 in the Two Mile Creek subregion of Buffalo and 4 in the St. Joseph Harbor subregion of Michigan.

Based on the PAH effluent data and discussion presented in Chapter 2, elevated (but probably much lower than at coke oven) PAH sediment concentrations may possibly also occur at or below outfalls from the following industrial categories and/or subcategories:

a. Wood Preserving facilities using creosote as a preservative

b. Oily wastewater outfalls from Metal Finishing facilities

c. Aluminum Forming facilities

d. Aluminum Casting Subcategory of the Foundry Industry and possible other subcategories not yet examined

e. Secondary Copper and Primary Aluminum (particularly those using Soderberg electrode refining) Subcategories of the Non-Ferrous Metals Industry and possibly other subcategories not yet examined

f. Petroleum Refining facilities involved in coke and coke by-product production and in the production of other high boiling fraction petroleum products.

The mean PAH sediment concentrations at sites in the open Great Lakes or in harbor areas of the Great Lakes not recieving direct industrial discharges are generally less than 2 mg/kg, even in areas where the tributaries are heavily polluted with PAHs. Examples of these sites include the following: 3 of the 4 reported sites in the Indiana Harbor, both reported sites in the Black River/Lorain Harbor, one of 2 reported sites in Lake Michigan off the Milwaukee Estuary, both reported sites in Ashtabula Harbor, all 3 reported sites in Toledo Harbor, all 4 reported sites in the Conneaut Harbor, both reported

sites in the Fairpoint Harbor, 7 reported sites in Buffalo Harbor away from the contaminated Lackawana and Union Ship Canals, numerous sites (except 4) in Lake Erie off Buffalo, 3 of 4 reported sites in Lake St. Clair, all 5 reported sites in Lake Erie off the mouth of the Raisin River, all 12 reported sites in the Open Lake Michigan region, and 5 of 6 reported sites in Lake Superior and Lake Superior harbors.

PAH sediment concentrations appear to generally decrease rapidly with longitudinal distance from a polluting outfall. PAHs generally partition into the sediment and suspended solids to a far greater extent than the water column. Therefore, they are generally found in elevated levels in sediments at or just below polluting outfalls and in downstream areas of sediment deposition. In the Black River at Lorain, concentrations of PAHs in sediments at different transverse locations, but at the same longitudinal distance downstream from the coke oven outfall, varied greatly, apparently due to differences in sediment deposition rates (ERG 1984).

6.2 RECOMMENDATIONS WITH RESPECT TO THE SAMPLING AND ANALYSIS OF SEDIMENTS FOR PAHS IN REGION V OF EPA

Recommendations with respect to the selection of sediment sampling sites in Region V of EPA are as follows:

1. Operating Coke Plants. The highest mean PAH sediment concentrations (e.g., >10 mg/kg) and associated risk scores are generally at or just below coke oven outfalls. Therefore, to determine maximum PAH sediment concentrations in Region V, sediment samples should be taken for PAH analysis at and just below operational coke plant outfalls.

Coke plants are associated with steel manufacturing and with some petroleum refineries. Tables A-3A and A-3B of Appendix A list the Blast Furnace/Steel Mills and Petroleum Refineries, respectively, in EPA Region V which have NPDES permits. Some but not all of those facilities have coke plants. Amendola (1985) listed 20 operational and closed steel industry coke plants in Region V (Appendix D).

Since it is probably not practical to sample sediments at and below all of the operational coke plant outfalls in Region V, priority should be given

to sampling sites in the vicinity of those coke plant outfalls which are predicted to be responsible for the greatest PAH loadings. In the usual absence of the PAH effluent data necessary to compute PAH loadings, the coke plants with the greatest mean wastewater volumes discharged per day should be selected for study. In the absence of daily wastewater discharge volume per day data, the coke plants with the largest production volumes should be selected for study. Coke plant production volumes are proprietary information and therefore cannot be included in this report. However, for the purpose of selecting sampling sites for study, EPA Region V can review coke oven production volumes for both steel industry and petroleum refinery coke plants with the Office of Solid Waste of EPA in Washington, D.C.

Historically, one of the greatest sources of PAHs to sediments in Region V has been the U.S. Steel coke Plant outfalls to the Grand Calumet River. Although there is some data on PAHs in sediments in the vicinity of the U.S. Steel coke plant outfalls to the Grand Calumet River, most of it is for samples taken in 1972. Furthermore, the 1972 samples were only analyzed for benzo[a]pyrene, anthracene, and phenanthrene. Therefore, sediments at and below the still-operational U.S. Steel coke plant outfalls should be resampled and analyzed for a wider range of PAHs (see comment below). Further, additional samples of sediments at and below the LTV (Republic) Steel outfall to the Cuyahoga River should be taken since the mean PAH concentration at the one reported site in that area (CU 81-18) is extremely high (75.7 mg/kg). Also, sediment samples should be taken at the Inland Steel Coke Plant outfall 012 to the Indiana Harbor. In addition to the coke plant recommended above, EPA Region V staff have recommended the following operational coke plants for possible study: Interlake and LTV Steel of Chicago, which discharge to the Calumet River; New Boston Coke, which discharges to the Ohio River; and National Steel (Granite City), and Allied Chemical, National Steel and Ford Motor Company, all of Detroit, which all discharge to the Rouge River.

2. Non-Operating Coke Plants. To determine the rate at which PAH levels will decrease in sediments no longer exposed to high PAH loadings, it would be valuable to sample sediments at and below one or more coke plants which have shut down and for which a substantial amount of historical PAH data exist. A good candidate for such a study would be the U.S. Steel coke plant outfall 002

to the Black River at Lorain, especially since there do not appear to be any major PAH sources within several miles upstream of the outfall. Of course, rates of PAH degradation and removal are site specific. Therefore, it would be beneficial to examine several different types of coke oven sediment sites if possible, before any generalizations should be attempted.

3. Non-Coke Plant Steel Industry. Much of the PAH sediment concentration data for the Grand Calumet River and the Indiana Harbor Canal indicate that elevated levels of PAHs in sediment (but lower than those at coke oven outfalls) may occur at and below various non-coke plant steel industry outfalls. However, the level of PAH contamination of sediments and water by coke oven outfalls to the Grand Calumet River is extremely high and sites at or below other types of steel industry outfalls are also further downstream of the coke plant outfalls. Therefore, it is difficult to determine whether the PAH contamination of sediments at or below various non-coke plant steel industry outfalls to the Grand Calumet River and Indiana Harbor Canal are due primarily to those outfalls or to the coke plant outfalls further upstream. Furthermore, there is very little evidence available from other regions to indicate whether steel industry outfalls other than those from coke plants contribute significantly to PAH loading. Nevertheless, PAH concentrations in effluents from blast furnaces and sintering plant processes along with the range of wastewater volumes discharged indicate that blast furnace and sintering plant outfalls may contribute significantly to PAH loadings in some cases (see Chapter 2). Therefore, to determine whether various other types of steel industry outfalls contribute significantly to sediment contamination, sites should be sampled at or below various types of non-coke plant steel industry outfalls located far enough upstream of any coke plant outfalls to insure that no contamination from the coke plant outfalls occur.

4. Other (Non-Steel) Industries. As discussed in Section 6.1, elevated mean PAH concentrations (e.g. >5 mg/kg) but generally lower than at or below coke plant outfalls have been observed in sediments at and below various types of outfalls other than those from steel industry facilities including outfalls from petroleum handling and storage facilities, storm and combined sewers, POTWs, a couple of foundries, a secondary tin manufacturer, a cryogenic hardware fabricating and finishing facility, and a chemical company. In addition,

elevated PAH levels are present at many sites at which no outfalls are identified, and have also been observed in sediments adjacent to dredged sediment disposal sites and a steel industry waste disposal site. Data on the concentrations of PAHs in industrial wastewater suggests that elevated levels of PAHs may also sometimes occur in sediments at or below outfalls from wood preserving facilities which use creosote, metal finishing facilities, aluminum forming facilities, the Aluminum Casting Subcategory of the Foundry Industry, the Primary Aluminum and Secondary Copper Subcategories of the Non-Ferrous Metals Industry, and petroleum refineries.

Based on the available data in this report it is difficult to identify those types of non-steel industry outfalls which should recieve priority for study. However, it may be possible to obtain additional information on the concentrations of PAHs in effluents from the various types of outfalls and on typical volumes discharged which would allow for computations and comparisons of PAH loadings from the various types of outfalls. In addition, an attempt should be made to identify outfalls in the vicinity of the numerous sites for which outfalls are not identified. Such information would be useful in deciding upon which type of outfalls should receive priority for study.

In the absence of sufficient data to make firm recommendations as to the type of non-steel industry outfalls which should receive priority, it is tentatively recommended that EPA assign priority to determing the PAH loadings from outfalls related to non-point sources such as storm and combined sewer outfalls and from non-point sources such as direct surface runoff, leakage from petroleum storage facilities and ship engines, leaching and erosion from sediment dredging piles, leaching from waste disposal sites, and atmospheric deposition. The reason is that, theoretically, the contribution of point sources to overall PAH loadings should rapidly decrease compared to those of non-point sources as the level of regulatory control over point sources continues to increase. Even if PAHs are not specifically limited in such controls, limitations on suspended solids, oil, and grease should effectively lower the quantities of PAHs discharged by most point sources.

5. Confirmation Sampling. The previous recommendations refer primarily to the selection of sediment sampling sites within a given region. The selections of regions for analysis should depend primarily upon the presence of

operational coke oven facilities but secondarily upon the extent of industrial and urban development since there is evidence that elevated levels of PAHs can occur in sediments in industrialized and heavily developed areas that do not have coke plants. Areas in EPA Region V which are heavily industrialized and also have operating coke facilities include the Grand Calumet River/ Indiana Harbor Canal/Indiana Harbor region, Chicago, Cleveland, Indianapolis, Toledo, and Detroit. Although a substantial amount of data is available for the Grand Calumet River/Indiana Harbor Canal region, much of it is for samples taken in the 1970's. Therefore, most of that region should be resampled. Additional data may not have to be obtained for the Detroit region, but additional effort should be made to identify outfalls and other points of pollution for a number of the reported sites in the Detroit region. No PAH data were found for either Chicago or Indianapolis and only limited data was found for Cleveland in the Cuyahoga River and for Toledo.

Amendola (1985) lists no steel industry coke plants in either Milwaukee or St. Joseph but elevated levels of PAHs have been reported for a number of sites in both these areas. However, before additional sampling is done in these areas, an attempt should be made to identify possible sources of pollution for sites for which PAH data are already available. The Youngstown/ Warren area is also heavily industrialized but 3 of the 4 previously operating coke plants have been shut down and most of the PAHs from the operating coke facility in Warren reportedly settle out in a swampy area before they reach the Mahoning area. However, because the Youngstown/Warren area is heavily industrialized and has historically received PAHs (some of which may still remain in sediments) from coke oven outfalls, that area should be considered for sampling as well.

6. Sampling Locations. Another recommendation on sediment sampling addresses sampling location and depth, with respect to suspected sources of pollution. As previously discussed, it appears that a large proportion of PAHs discharged to the aquatic environment are sorbed to suspended solids (which rapidly settle out) or are rapidly absorbed from solution by sediments upon discharge. Therefore, a large amount of the PAHs discharged to the aquatic environment settle out in the immediate vicinity of the outfall and at the first major depositional area downstream of the outfall. Therefore, once an

outfall has been identified as possibly being a significant source of PAHs to the environment, sediment samples should be taken immediately below the outfall, midway between the outfall and the first major sediment deposition area downstream of the outfall, and in the first major depositional area downstream of the outfall. If the suspected PAH source is a non-point source (e.g., dredged sediment disposal pile), samples should be taken at the downstream edge of the source, midway between the downstream edge of the source and the first major depositional area downstream of the source, and in the first major depositional area downstream of the source.

The ERG (1984) study of sediments downstream of the USS Steel Coke oven outfall 002 to the Black River in Lorain indicated that PAH concentrations in sediment samples taken at different transverse locations at the same longitudinal distance downstream from an outfall may vary widely. Therefore, samples at several different transverse locations for a given longitudinal distance downstream should be taken and composited before analysis of mean PAH concentrations in sediments at that distance downstream is attempted. If maximum PAH concentrations are desired, the transverse location at which the greatest sediment deposition occurs should be selected for the sampling.

The study by GLNPO (1985) in the Milwaukee region indicated that maximum PAH sediment concentrations sometimes occur in core fractions beneath the surface core fraction (e.g., 30-60 cm or 60-90 cm fractions instead of the 0-30 cm surface fraction). This may be especially true in regions with high sediment deposition rates and where levels of PAH loadings to the aquatic system have decreased in recent years. Obviously, if maximum PAH concentrations in the sediment are desired, a core of sufficient depth should be taken, such that a core fraction with maximum PAH concentrations can be observed other than the lowest core fraction obtained. In the Milwaukee study, core depths of 100 cm were sufficient in most but not all cases. If mean PAH sediment concentrations over the entire depth of the contaminated sediment zone are desired, cores must be taken to a depth at which no significant PAH contamination is observed. In the Milwaukee study, that would have required taking core samples well in excess of 100 cm at all of the sites.

Obviously, it would require a great deal of sampling if a knowledge of PAH sediment concentrations as a function of depth was desired at each site.

However, for purposes of estimating the potential effects of PAH sediment contamination on the aquatic environment, it is probably sufficient and perhaps even desirable to sample to depths of no greater than 30 cm since the greatest interaction between the water column and benthic organisms with sediments probably occur in the upper 30 cm or 1 foot of sediment.

7. Sample Analysis. The last recommendation on sediment sampling and analysis concerns the kinds of PAHs for which sediment samples should be analyzed. As can be seen from Appendix C, Table C-1, there are no obvious patterns in the concentrations of PAHs in sediments with respect to molecular weight or number of rings. Furthermore, PAHs with the higher molecular weight and numbers of rings PAHs appear to generally be more toxic to humans, yet PAHs with lower molecular weights and number of rings appear to be more acutely toxic to aquatic organisms. Therefore, sediments should ideally be analyzed for representatives of 2, 3, 4, 5, and at least 6 ring PAHs. The PAHs listed in Table 1-1 are among those most frequently analyzed for in the environment, include both carcinogenic and non-carcinogenic PAHs, and include at least 2 representatives each of 2, 3, 4, 5, and 6-ring PAHs. Therefore, in most cases, an analysis of sediments for the PAHs listed in Table 1-1 should be sufficient to characterize PAH contamination in general unless other PAHs are known or suspected to be at high levels due to the nature of the sources involved. Methylated derivatives of some but not all of the PAHs listed in Table 1-1 appear to be more toxic and therefore might be included in some analyses. In addition, 7,12-dimethyldibenzo[a,h]anthracene and methycholanthrene (which along with benzo[a]anthracene, benzo[a]pyrene, and dibenzo[a,h]anthracene have been conclusively shown to cause malignant tumor formation in laboratory animals) should probably be included in analyses.

6.3 RECOMMENDATIONS WITH RESPECT TO THE SAMPLING AND ANALYSIS OF OTHER MEDIA

Since sediments are the primary reservoirs for PAHs in the aquatic environment, measurement of PAH concentrations in sediment samples is a good indicator of the extent to which an aquatic system has received PAH inputs. However, in estimating the risks to human and aquatic life associated with PAH contamination of aquatic systems, determinations of PAH concentrations in edible aquatic organisms, drinking water and in the water column are of much greater value.

6.3.1 Estimating Carcinogenic Risks to Human Life Associated with the PAH Contamination of Aquatic Systems

An equation for essentially estimating the carcinogenic risk (XC_{1j}) to humans associated with the concentration of a given PAH (j) in sediment at a given sampling site (i) was presented in Chapter 3 (Equation 3-1, without the 10^4 term). Equation 3-1 is based on the assumptions listed in Chapter 3 and on the additional assumption that the aquatic organisms consumed all remain in the immediate vicinity of the given site long enough to reach steady state and are then caught in that general area. None of the assumptions listed in Chapter 3 (except possibly assuming that the PAH contribution of drinking water is negligible compared to that of the edible aquatic organisms), nor the additional assumption discussed above are fully realistic for computing carcinogenic risk. Therefore, equation 3-1 should only be used for computing relative site rankings and not for computing absolute risks. In addition, equation 3-1 should only be used in the absence of actual levels of PAHs in edible aquatic organisms caught from the water body of concern.

The total lifetime carcinogenic risk to an individual person due to the consumption of contaminated aquatic organisms and drinking water from a given water body is given by:

$$CR = \sum_j (UCR_j) \frac{(6.5 \times 10^{-3} \text{ kg/day}) (\overline{CF_j}) + (2L) (\overline{CDW_j})}{(70 \text{ kg})} \qquad (6\text{-}1)$$

where

CR = total lifetime (70 year) carcinogenic risk to an individual person due to the consumption of 6.5×10^{-3} kg/day of contaminated fish and 2 liters per day of contaminated drinking water from the same given water body (unitless fraction)

UCR_j = unit carcinogenic risk for carcinogenic PAH j (mg/kg/day $^{-1}$)

$\overline{CF_j}$ = mean concentration of carcinogenic PAH j in edible fish caught from the water body of concern over a 70 year period (mg/kg fish)

$\overline{CDW_j}$ = mean concentration of carcinogenic PAH j in drinking water derived from the given water body of concern over a 70 year period (mg/L)

70 kg = assumed body mass of an adult male.

The sum is over all carcinogenic PAHs present at significant levels in either the edible aquatic organisms or in drinking water. Although the values of $\bar{\bar{CF}}_j$ and $\bar{\bar{\bar{CDW}}}_j$ over the next 70 year period are unknown, current values of the concentrations in fish (CF_j) and in drinking water (CDW_j) can be substituted for $\bar{\bar{CF}}j$ and $\bar{\bar{\bar{CDW}}}_j$ in equation 6-1 to give worst case estimates of carcinogenic risks assuming that PAH levels in fish and drinking water will remain the same or decrease over the next 70 year period.

The use of equation 6-1 to estimate the total lifetime carcinogenic risks to individuals from consuming PAH contaminated fish and drinking water from a given water body obviously requires water body specific data on the concentrations of carcinogenic PAHs in both those media. However, since PAHs have low aqueous solubilities, are susceptible to degradation by chlorination and tend to partition to a much greater extent into suspended solids (which are generally efficiently removed by water treatment facilites) than in water, the contribution of drinking water to the overall PAH input to an individual is probably generally negligible compared to that of PAH contaminated fish. Therefore, if an estimate of carcinogenic risks associated with the PAH contamination of a given water body is desired, priority should be given to the determination of carcinogenic PAH levels in fish and shellfish caught from the given water body.

It was stated in Section 6.2 that analysis of sediments for the PAHs listed in Table 1-1 was probably more than sufficient for determing the relative PAH contamination of different aquatic systems. However, if a reasonable estimate of total carcinogenic risks are to be made, carcinogenic PAHs in addition to those listed in Table 1-1 should be included in the analysis of fish and shellfish tissues. At the very least, the 5 PAHs which have been conclusively shown to induce malignant tumors in test animals after oral administration should be included in the analysis. Three of the 5 PAHs (benzo[a]pyrene, benzo[a]anthracene and dibenzo[a,h]anthracene) are listed in Table 1-1 but the other 2 PAHs (7,12-dimethylbenzo[a]anthracene and methlcholanthrene) are not listed in Table 1-1 but should also be included in any analysis of fish tissue or drinking water.

Finally, it should be again noted that benzo[a]pyrene is the only PAH for which a unit carcinogenic risk factor (UCR) has been determined. Therefore, to use equation 6-1 to compute carcinogenic risks associated with the PAH contamination of aquatic systems, UCRs for at least the carcinogenic PAHs in Table 1-1, as well as 7, 12-dimethylbenzo[a]anthracene and methylcholanthrene should be determined.

6.3.2 Estimating Non-Carcinogenic Risks to Human Life Associated with the PAH Contamination of Aquatic Systems

An equation for essentially estimating the non-carcinogenic risk (XN_{ij}) to humans associated with the concentration of a given PAH j in sediment at a given sampling site i was presented in Chapter 3 (Equation 3-2 without the 10^2 term). Equation 3-2 is based on the same 4 assumptions (the 3 listed in Chapter 3 and the one listed above) used to derive equation 3-1. Therefore, equation 3-2, like equation 3-1, should only be used for computing relative site ranking and not for actual risk computations. In addition, it should only be used in the absence of data on the concentrations of PAHs in edible aquatic organisms caught from the water body of concern.

The non-carcinogenic risk (which is not really a risk but a ratio of estimated dose to the ADI) to an individual due to the consumption of a given non-carcinogenic PAH j in contaminated fish and drinking water from a given water body is given by:

$$NCR_j = \frac{(6.5 \times 10^{-3} \text{ kg/day})\ (CF_j) + (2L)\ (CDW_j)}{(ADI_j)\ (70 \text{ kg})} \qquad (6\text{-}2)$$

where

NCR_j = non-carcinogenic risk (ratio) to an individual due to the consumption of a given non-carcinogenic PAH j in contaminated fish and drinking water from the water body of concern

ADI_j = acceptable daily intake of non-carcinogenic PAH j (mg/kg/day)

CDW_j = mean concentration of non-carcinogenic PAH j in drinking water derived from the water body of concern.

The rest of the parameters are the same as defined for equation 6-1.

Unlike carcinogenic risks, non-carcinogenic risks (ratios) are generally not summed on different PAHs because the effects of different PAHs are generally different. However, non-carcinogenic risks (ratios) are sometimes summed over different PAHs to give a "hazard index". If that is desired, equation 6-2 can be modified to include a summation over all non-carcinogenic PAHs.

Finally, it should be again noted that ADIs have only been determined for fluoranthrene and naphthalene and need to be determined for a number of other PAHs before more extensive non-carcinogenic risk computations using equation 6-2 can be performed.

6.3.3 Estimating Risks to Aquatic Life Associated with the PAH Contamination of Aquatic Systems

An equation for essentially calculating acute aquatic toxicity risks (Y_{ij}) associated with the concentration of a given PAH j in sediment at a given sampling site i was presented in Chapter 3 (Equation 3-3, without the 10^3 term). However, some of the same assumptions used in deriving equations 3-1 and 3-2 were also used in deriving equation 3-3. Therefore, equation 3-3, like equations 3-1 and 3-2, should only be used for computing relative site rankings and not for actual risk computations. In addition, it should only be used in the absence of data on the concentrations of PAHs in the water column.

The acute or chronic risks (which is not really a risk but a ratio of PAH concentration in water to acute or chronic water quality criteria) to aquatic organisms due to the mean concentration of PAH j in the water column is given by:

$$W_j = \frac{CW_j}{(AACRI_j \text{ or } CACRI_j)} \qquad (6\text{-}3)$$

where

W_j = acute or chronic risk (ratios) to aquatic organisms due to the mean concentration of PAH j in the water column

CW_j = mean concentration of PAH j in the water column

$AACRI_j$ = acute water quality criteria for PAH j for the protection of aquatic life

$CACRI_j$ = chronic water quality criteria for PAH j for the protection of aquatic life.

Acute and chronic aquatic life risks (ratios) are generally like non-carcinogenic risks (ratios), but are not summed over different PAHs because different PAHs exert different effects on the same and different organisms. However, just like human non-carcinogenic risks, acute and chronic aquatic life risks can be summed over different PAHs to give a "hazard index". If that is desired, equation 6-3 can be modified to include a summation over all $PAHs_J$.

The use of equation 6-3 to compute acute or chronic risks to aquatic life requires not only data on the concentrations of PAHs in the water column but also the determination of acute and chronic water quality criteria for various PAHs for the protection of aquatic life. Unfortunatly, as previously stated, no acute or chronic water quality criteria have as yet been established for any of the PAHs. This needs to be done before any reasonable aquatic life risk estimates can be computed.

In closing, it should again be emphasized that the evidence that PAHs cause significant increases in the incidence of tumor formation in fish populations is based on field studies of aquatic systems which are heavily contaminated with other potential carcinogens as well. Evidence that PAHs alone, even at extremely high levels, can cause increases in the incidence of tumor formation is scant and virtually non-existent for levels of PAHs that organisms could typically be expected to be exposed to in even highly contaminated aquatic systems. Therefore, in addition to additional field studies of the type conducted by Black (1983) and Bauman et al. (1982), studies should be conducted to determine if PAHs alone at levels which could reasonably be expected to be encountered in highly contaminated aquatic systems can increase the incidences of tumor formation or other adverse effects in aquatic organisms.

7. SUMMARY

Polycyclic aromatic hydrocarbons (PAHs) are complex organic molecules formed through incomplete "burning" (pyrolysis) of organic matter. Literally hundreds of separately-recognized PAHs are known, yet complete toxicological information is available for only a very few. The available toxicological data have demonstrated the ability of at least 40 to 50 PAHs to induce cancer in laboratory studies, however none are regulated in drinking water (i.e.; no drinking water standards exist for PAHs under the SDWA). In addition, while at least three PAHs have been shown to exert acute toxic effects on aquatic species, ambient water quality criteria under the CWA exist for PAHs only as a class, and individually for only acenapthene, naphthalene, chlorinated naphthalene and fluoranthene.

This report has been prepared to characterize the nature and extent of polycyclic aromatic hydrocarbon (PAH) contamination of sediments in USEPA Region V and the Great Lakes basin. This characterization includes calculation of potential risks to human health and aquatic life, and cross-comparison of various sediment sampling sites based on calculated risk scores. Risk characterizations are based on currently available information on PAH mobility in the aquatic environment and toxicological effects on human health and aquatic life. Because this information is quite limited, the assignment of risks has involved a number of assumptions. These assumptions are discussed in the text.

Evidence of PAHs causing significant increases in the incidences of tumor formation in fish populations is drawn from field studies of aquatic systems which are also heavily contaminated with other potential carcinogens. Evidence that PAHs alone can cause increases in the incidence of tumor formation is inadequate, particularly for PAH levels that organisms could typically be expected to be exposed to, in even highly contaminated aquatic systems. Field studies should be conducted to determine the degree to which PAHs alone increase the incidences of tumor formation and/or other adverse effects in aquatic organisms.

This report is based on available PAH data, supplied by various USEPA sources. Geographically, these data include sites in USEPA Region V and the Great Lakes Basin.

7.1 BACKGROUND INFORMATION ON PAHs

This review of background information on PAHs covers the following topics: physical, chemical and toxicological properties of PAHs; sources of PAHs to the aquatic environment; environmental fate and transport of PAHs; PAH toxicity to humans and other mammals; and PAH toxicity to aquatic life.

7.1.1 Physical, Chemical and Toxicological Properties

Structures and various physical, chemical, and toxicological properties of PAHs that are frequently analyzed for in the environment are listed in Table 1-1. Structures and physical/chemical properties of PAHs were taken from the pulished literature, or were calculated from formulae or procedures available in the literature. This includes primarily octanol/water partition coefficients (K_{OC}) and bioconcentration factors (BCF).

Values of toxicological parameters for most PAHs have not yet been determined. Benzo[a]pyrene is reported to have a unit carcinogenic risk factor (UCR) of 11.5 kg/day/mg but no other UCRs for PAHs could be found in the literature. The only available acceptable daily intakes (ADI) are for fluoranthene (5.9×10^{-3} mg/kg/day) and naphthalene (0.26 mg/kg/day). No acute or chronic water quality criteria for the protection of aquatic life have been established for any of the PAHs. Concentrations of acenaphthene, napthalene, and fluoranthene as low as 1.7 mg/L, 2.3 mg/L, and 4.0 mg/L, respectively have been reported to cause acute toxic effects in some aquatic organisms.

For the purpose of scoring and ranking regions, subregions, sediment sampling sites and PAHs according to potential risks to human and aquatic life, the following assumptions were made in assigning values of UCRs, ADIs and acute aquatic criteria to the PAHs in Table 1-1:

1. PAHs were assumed to be either carcinogens or non-carcinogens based upon available evidence for carcinogenicity.

2. "Carcinogenic" PAHs were all assigned UCR values of 11.5 kg/day/mg which is the reported UCR for benzo[a]pyrene.

3. "Non-carcinogenic" PAHs other than naphthalene were assigned ADI values of 5.9×10^{-3} mg/kg/day which is the reported ADI value for fluoranthene. Naphthalene has a reported ADI value of 0.26 mg/kg/day.

4. No acute aquatic life criteria have been established for any of the PAHs. However, PAHs other than fluorathene and naphthalene were assumed to have acute aquatic life criteria of 1.7 mg/L based on the no-effect level for acenaphthene. Acute aquatic life criteria for naphthalene and fluoranthene were assumed to be 2.3 mg/L and 4.0 mg/L, respectively, based on no-effect levels.

7.1.2 Sources to the Aquatic Environment

PAHs are formed by incomplete combustion (pyrolysis) of organic materials; naturally occuring diagenesis of sedimentary organic material, which produces fossil fuels; and by plant and microbe biosynthesis.

Potentially major point sources of PAH pollution to the aquatic environment include effluents from the following:

- Coke and coke byproduct production facilities
- Iron and steel blast furnace and sintering operations
- Wood preserving facilities using creosote
- Metal finishing rinsing operations
- Aluminum-forming facilities
- Facilities using high temperature furnances such as metal smelters and foundries (particulary those using a Soderberg electrode which consists of anthracite, coketar pitch and anthracene)
- Oil refineries
- Organic chemical manufacturers which use solid or liquid hydrocarbon feedstock.

Potentially major non-point sources of PAH pollution to the aquatic environment include the following:

- Petroleum and petroleum product spills and leaks during transport or storage
- Surface runoff from roads and contaminated soil
- Leaching from hazardous waste sites, including dredged sediment piles
- Discharges from boat motors and ship engines
- Atmospheric deposition of PAH contaminated particulates

Potentially major sources of PAH contaminated particulates to the atmosphere include emissions from coal and oil burning electric power plants, coal and

wood burning heat furnances, refuse burning, coke and coke byproducts production, oil refineries, and internal combustion engines.

7.1.3 Fate and Transport

In general, much more of the total PAH in aquatic systems is bound to sediment or suspended solids than is dissolved in the water column. Sediment/water partitioning of PAHs generally increases with decreasing aqueous solubility and increasing molecular weight, number of rings, octanol/water partition coefficient and sediment organic content. The primary transport mechanism for PAHs in aquatic systems appears to be by transport of PAH contaminated suspended solids. Volatilization under turbulent conditions and from shallow water bodies may remove substantial amounts of the lower molecular weight and ring number PAHs dissolved in the water column within several days. However, for most PAHs under most conditions, volatilization over periods well exceeding 1 month may be required to remove substantial proportions of the dissolved PAH from water.

PAHs are resistant to hydrolysis but some PAHs, such as benzo[a]anthracene and benz[a]pyrene, may be suspectible to chemical oxidation by photochemically produced reactive species. Photolysis half-lives for many PAHs are less than 24 hours under optimal conditions but increase substantially with increasing water depth, water turbidity, humic content and sorption to sediments. Some PAHs appear to be susceptible to at least aerobic biodegradation but rates of biodegradation greatly decrease with increasing molecular weights and number of rings. Although predicted bioconcentration factors based on the octanol/ water partition coefficient are moderately high for many PAHs (e.g., $>10^3$ for PAHs with 4 or more rings), the actual values for fish may generally be substantially lower due to metabolism of the PAHs.

Sediment/water equilibrium partition coefficients (K_D) for the adsorption of different PAHs to the same sediment type generally increase with decreasing aqueous solubility and increasing molecular weight, number of rings and octanol/water partition coefficient (K_{OC}). However, the concentration of various PAHs in sediments do not generally follow any particular order with respect to molecular weight and numbers of rings.

Sediment/water equilibrium partition coefficients for the adsorption of the same PAH to different sediments will generally increase with increasing

organic content of the sediment. However, the organic coefficient (which is the ratio of the sediment/water partition coefficient to the organic fraction of the sediment) should remain relatively constant for the same PAH absorbed to different sediments.

The relatively low aqueous solubilities and relatively high K_{OC} values for most PAHs indicate that a much larger proportion of the total PAH in an aquatic system will generally be adsorbed to sediments and suspended solids than will be in solution in the water column. This is particularly true for PAHs with 4 or more rings and for sediments with high organic carbon.

The relatively high sediment/water partitioning for most PAHs suggest that the primary mechanism of transport for those PAHs in aquatic systems is by transport of PAH contaminated sediment. This had led to speculation that PAH concentrations in sediments may be higher in areas of suspended solids deposition (e.g. areas of reduced current and turbulence) than in other areas of aquatic systems.

Volatilization half-lives for PAHs in water generally increase with increasing molecular weights and number of rings due to the corresponding decrease in Henry's constant and diffusion constants in air and water. Volatilization half-lives for a given PAH from water decrease with increasing turbulence in either the air or water.

With the exception of 2 and 3 ring compounds under turbulent conditions and in shallow water, it appears that PAH volatilization half-lives are too long for volatilization alone to prevent substantial proportions of dissolved PAH from being transported many km downstream from the point of input. However, over time periods exceeding one month, volatilization alone could possibly account for a substantial decrease in dissolved PAH concentrations, at least for PAHs with no more than 5 rings.

PAHs do not readily undergo nucleophillic aromatic substitution and therefore are not predicted or known to undergo hydrolysis in natural waters.

Major naturally occuring oxidizing species in natural waters are thought to include the peroxy free radical RO_2 and singlet oxygen $^1O_2$ which are generated photochemically. It appears that at least some PAHs may be susceptible to oxidation in natural water by photochemically produced oxidants, whereas others (such as naphthalene and phenanthrene) are not. PAHs appear to be

readily oxidized to quinones by both chlorine and ozone during the treatment of drinking water. Half-lives for many PAHs at exposure concentrations of ozone and chlorine typically used in water treatment are generally less than one hour.

Most PAHs appear to be susceptible to rapid rates of direct aqueous photolysis under optimal conditions (e.g. dissolved in shallow clean water, exposed to midday sunlight or to intense sun lamps). However, rates of aqueous photolysis decrease with increasing water depth, turbidity, humic concentrations, and PAH adsorption to sediments. Therefore, under actual field conditions, aqueous photolysis half-lives may be much longer than those reported under optimal conditions.

Biodegradation rates are generally much greater for PAHs with no more than 3 rings than for PAHs with 4 or more rings. In addition, biodegradation rates are generally greater in continuously contaminated water or sediment than in previously uncontaminated water or sediment. Biodegradation half-lives for PAHs in previously uncontaminated sediment may be 10 to 400 times longer than the half-lives of the same PAHs in previously contaminated sediments. Long acclimation times may be required before bacteria are able to degrade PAHs. Biodegradation rates are reported to be much greater under aerobic conditions than anaerobic conditions and may be generally greater for PAHs adsorbed to sediment than for PAHs dissolved in the water column.

The rate at which aquatic organisms can metabolize and/or excrete PAHs appears to generally follow the order: fish > crustaceans >> bivalve mollusks >>> microalgae. The ability of the higher aquatic organisms to metabolize and/or excrete PAHs rapidly is reflected in the relatively short times required to reach steady state PAH tissue concentration.

Bivalve mollusks do not readily metabolize PAHs and have generally lower excretion rates. Therefore, they may bioaccumulate PAHs to a greater extent than most fish or crustaceans. Nevertheless, PAH contaminated mollusks appear to be able to remove most of the PAH in their tissue (presumably primarily by excretion) within a few days to several weeks after being placed in uncontaminated water. Microalgae reportedly bioaccumulate and retain PAHs to a greater extent than the higher organisms due possibly to irreversible binding of the PAHs to the cell wall.

Steady state bioconcentration factors for the uptake of different PAHs by aquatic organisms from water generally increase with increasing molecular weight, number of rings, and octanol/water partition coefficient. However, the concentrations of various PAHs in the tissues of aquatic organisms do not generally follow any order with respect to molecular weight or number of rings. Although the bioconcentration factor generally increases with increasing molecular weight, the concentration of the higher molecular weight PAHs in water are frequently less than those of the lower molecular weight PAHs due to lower aqueous solubility and/or loading rates. Other reasons may include non-steady state conditions and contributions of PAHs from other media such as food and sediment.

The octanol/water partition coefficient is generally a good indicator of a chemical's partitioning between the lipid containing tissues of aquatic organisms and water. In organisms which remove PAH's primarily through excretion, bioconcentration factors increase with increasing octanol/water partition coefficients. In species which remove PAH's primarily by metabolism, bioconcentration factors increase with increasing molecular weight and number of rings, which may be due to a corresponding decrease in the ability of the organism to metabolize PAHs with high molecular weights and number of rings.

Bioconcentration factors derived as the ratio of PAH concentrations in organisms to those in food or sediment may be substantially lower than those based on the ratio of PAH concentration in organisms to those in water. However, since PAH concentrations in sediment are generally much greater than those in water, a substantial accumulation of PAH in organisms from the sediment may sometimes occur.

The extent and mechanism of PAH accumulation in an aquatic organism depends primarily on whether the organism is benthic. Benthic organisms may accumulate PAHs from sediment by direct physical contact or through respiration in interstitial water or water near the sediments which have been contaminated by deadsorption from the sediment. Non-benthic organisms probably accumulate PAHs primarily during respiration in contaminated water. However, in most cases, the concentrations of PAH in the general water column will be are less than PAH concentrations in interstitial water or water near the sediments.

7.1.4 PAH Toxicity to Humans and Test Mammals

Various PAHs have long been suspected of being carcinogenic to humans based on their relatively high concentrations in media known to be carcinogenic. Numerous studies have shown that various PAHs can induce malignant tumors in laboratory animals through dermal exposure or subcutaneous injection. Oral administration of a limited number of PAHs have been shown to induce stomach tumors in laboratory animals, but the concentrations of PAH required is generally quite high (>30 ppm). Only benzo[a]anthracene, benzo-[a]pyrene, dibenzo[a,h]anthracene, 7,12-dimethylbenzo[a]anthracene, and methylcholanthrene have been conclusively shown to induce malignant tumors after oral administration. The most frequently observed effects of the oral administration of these PAHs were leukemia, forestomach tumors, hepatoma, pulmonary adenoma, and mammary tumors. The only PAH for which a unit carcinogenic risk (UCR) has been established is benzo[a]pyrene (11.5 kg/day/ mg).

There are very few data concerning the non-carcinogenic effects of PAHs. Several PAHs are known or suspected skin and/or mucous membrane irritants to humans and/or laboratory animals. PAH effects the skin, causes loss of body weight, blood alterations, and mild morphological damage to the kidneys and liver. Acceptable daily intakes have only been established for fluoranthene and naphthalene.

Data on the acute toxicity of PAHs to freshwater aquatic organisms are extremely limited. Acenaphthene, fluorathene, napthalene, benzo[a]anthracene, 1-methylnapthalene, 2-methylnapthalene, phenanthrene, anthracene and pyrene have been shown to elicit toxic responses in various aquatic organisms in laboratory studies.

Data on the acute toxicity of PAHs to saltwater organisms are a little more extensive and show that such organisms are generally more sensitive to PAHs than similar types of freshwater organisms. Data on the acute toxicity of PAHs to both freshwater and saltwater organisms indicates that the acute toxicity of PAHs with 4 or less rings generally increases with increasing molecular weight and number of rings. However, PAHs with 5 or more rings are generally not acutely toxic, perhaps at least partly due to their low aqueous solubility.

Data on chronic toxicity of PAHs to aquatic organisms are extremely limited. A chronic value of 620 ug/L has been reported for fathead minnows exposed to naphthalene in an embryo-larval test. No other chronic values for freshwater species were reported in the various PAH water quality criteria documents. Reported effects of 2 and 3 ring PAHs on primarily saltwater organisms exposed to sublethal concentrations included retarded larval development for mud crabs; reduction in offspring for marine copepods; impaired movement in blue crabs; lesion development in oysters; histological changes in the livers of mullets; and gill hyperplasia and gill filament hemorrhages in mummihog fish.

There is some circumstantial evidence that PAHs in heavily contaminated aquatic systems may induce the formation of malignant and/or benign epidermal and/or liver tumors in bottom feeding fish. This evidence comes from studies of brown bullheads and freshwater drum in multiple locations.

Although field studies indicate that PAHs in heavily polluted aquatic systems may induce the formation of tumors in fish, the evidence is only circumstantial since other toxic and potentially carcinogenic organics and heavy metals are also present. Unfortunately, there is very little direct evidence for or against the postulate that PAHs can induce tumor formation in fish. The limited evidence available suggests that PAHs do not readily induce tumor formation in fish, but may do so in some fish if they are exposed to high concentrations of PAH over extended time periods.

7.2 SOURCES OF PAHs TO USEPA REGION V AND THE GREAT LAKES AREA

PAH contamination in USEPA Region V and the Great Lakes area originates from both point and nonpoint sources. Industries considered to be the major point source contributors are the following: Timber Products Processing; Iron and Steel Manufacturing; Metal Finishing; Aluminum Forming; Foundries; Non-Ferrous Metals Manufacturing; and Petroleum Refining. Major nonpoint sources of PAHs include: atmospheric deposition; petroleum product spills and leakage; runoff from roads and contaminated soils; and runoff from hazardous waste disposal sites.

The USEPA classifies effluent from the point source industries into subcategories based on a variety of factors such as manufacturing processes, materials used in various processes, and predominant components of wastes from

processing steps. Data is provided on PAH concentrations in effluents from specific industrial subcategories and on the effluent from all subcategories combined, for each industry. For some industries, data on both raw and treated effluent was available for various subcategories and/or for the industrial effluent as a whole.

The mean of the mean PAH concentrations in raw wastewater is much greater for the Timber Products Processing, Iron and Steel Manufacturing, and Metal Finishing Industries than for the other industrial categories. Treatment processes for the Timber Processing Industry are considered inefficient, decreasing PAH concentrations by only approximately 50%. The Iron and Steel Manufacturing Industry has the second highest concentration of PAHs in raw effluent, however, treatment processes remove about 90% of the PAH material. Raw effluent from the Metal Finishing Industry as a whole ranks third highest based on total concentration of PAHs and mean of the means of concentration for specific PAHs. Despite the comparatively high effluent treatment efficiency for the Iron and Steel Manufacturing Industry (90%), total daily PAH loadings from this source are much higher for this industrial category than for the Timber Products Processing and Metal Finishing Industries, due to the much higher total wastewater volumes generated.

In Region V, very high PAH concentrations are often found in sediments near coke oven outfalls. However, BPT treatment processes in the Iron and Steel Manufacturing industry as a whole have now reduced average PAH concentrations in treated effluents to less than 10% of the concentrations in raw wastewater. Within several subcategories of this industry, considerable variability exists in efficiency of treatment processes, and a subcategory with comparatively low PAH concentrations in raw wastewater may exhibit relatively high PAH concentrations (and environmental loadings) in treated effluent.

Data from the Aluminum Forming Industry was anomalous in that the total and mean of mean PAHs concentrations were an order of magnitude higher in treated effluent than raw effluent. This industry ranked sixth highest for PAH concentrations in raw effluent and second highest for concentrations in treated effluent. The increase was due to increases of anthracene and phenanthrene in treated waste. It is possible that data were reported erroneously.

Raw effluent from the Foundry Industry is somewhat higher than that of the Aluminum Forming Industry, being ranked fifth highest based on mean of means PAH concentrations. Treated effluent is ranked fourth.

Raw effluent from the Non-ferrous Metals Industry is comparable to that of the Foundry Industry, but treated effluent has a substantially lower concentration, based on available data.

Concentrations of PAHs in raw and treated effluent from the Petroleum Refining Industry were surprisingly low, considering that the processes involved are known to produce significant quantities of PAHs. It is suggested that sampling of effluent was not representative, given the variety of processes involved in refining and the complexity of the facilities performing these processes.

Contributions from nonpoint sources, unlike point sources, are compared on a qualitative basis. The major nonpoint source of PAHs to surface waters is via atmospheric deposition. Tributaries to the Great Lakes may contribute severely contaminated suspended solids, but such inputs are predicted to equal only 10-25% of the contribution from atmospheric particulates. Spills and leakage of petroleum products are another major nonpoint source of PAHs to the Great Lakes. Nonpoint sources of lesser magnitude include dredged sediment piles, runoff and leakage from hazardous waste disposal sites, and runoff from roads and contaminated soils.

7.3 PAH SEDIMENT CONCENTRATIONS AND ASSOCIATED RISK SCORES

For the purpose of determining relative levels of PAH sediment contamination and associated risks, the Great Lakes area has been divided into 10 geographic regions, with associated subregions. The regions and subregions are described below:

Great Lakes Regions/Subregions

- Indiana region - 35 sampling sites in Lake Michigan harbors and tributaries in Indiana (three subregions: Indiana Harbor, Indiana Harbor Canal, and Grand Calumet River)
- Detroit region - 43 sampling sites (four subregions: Detroit River and Tributaries, St. Clair River and Lake, Raisin River, and Lake Erie off Raisin River)
- Milwaukee region - 12 sampling sites (no subregions)

- Ohio Steel region - 23 sampling sites in the Black and Mahoning Rivers in Ohio (two subregions: Black River at Lorrain, and Mahoning River)

- Ohio region - 42 sampling sites in Lake Erie harbors and tributaries in Ohio (five subregions: Toledo, Cuyahoga River at Cleveland, Fairpoint Harbor, Astabula, Conneaut Harbor)

- Michigan region - 33 sampling sites in Lake Michigan harbors and tributaries in Michigan (five subregions: Manistique, Manistee, Muskegon and New Buffalo Harbor, Kalamazoo River, and St. Joseph Harbor)

- Open Lake Michigan region - 12 sampling sites well offshore in Lake Michigan (no subregions)

- Lake Superior region - 8 sampling sites in Lake Superior harbors and tributaries (two subregions: Ashland, and Miscellaneous)

- Buffalo region - 151 sampling sites in the Niagara River system (nine subregions: Upper Niagara River and Chippawa Canal, Tonawanda Canal, Two Mile Creek, Lower Niagara River, Scajaquada Creek, Buffalo River, Buffalo Harbor, Lake Erie near Buffalo, and Rush and Smokes Creek)

- New York/Pennsylvania region - 25 sampling sites in New York and Pennsylvania harbors (three subregions: Dunkirk/Erie, Rochester, and Oswego/Olcott).

Data were then collected and collated from the 384 individual reported sampling sites within these regions/subregions.

From the PAH sediment concentrations associated with each sampling site, human carcinogenic, human non-carcinogenic and acute aquatic toxicity risk scores were calculated. These risk scores were calculated individually for each specific sampling site. The scores for each site were derived as means of the individual risk scores calculated for each of the 1 to 14 PAHs reported for each site.

Human carcinogenic, human non-carcinogenic, and acute aquatic toxicity risk scores are summarized in Table 7-1. This table is subdivided according to the regions/subregions identified above (although risk scores were calculated individually for each of the 384 sampling sites, these individual scores are not included in Table 7-1). In Table 7-1, only mean risk scores are presented.

Table 7-1. Summary of Mean PAH Sediment Concentrations, Mean Risk Scores, and Rankings for Regions and Subregions

Region, subregion	Sediment concentration (mg/kg) and (ranking)	Carcinogenic risk score ($\times 10^4$) and (ranking)	Non-carcinogenic risk score ($\times 10^2$) and (ranking)	Acute aquatic toxicity risk score ($\times 10^3$) and (ranking)	Overall ranking score and (ranking)
Indiana Region	**51.5 (1)**	**1.22 (1)**	**68.6 (1)**	**98.1 (1)**	**4 (1)**
Grand Calumet River Subregion	88.6 (1)	2.02 (1)	150 (1)	75.7 (2)	5 (1)
Indiana Harbor Subregion	61.4 (2)	1.36 (2)	31.1 (2)	98.1 (1)	7 (2)
Indiana Harbor Canal Subregion	8.40 (3)	0.43 (3)	9.16 (3)	5.07 (3)	12 (3)
Ohio Steel Region	**11.8 (2)**	**0.55 (3)**	**15.2 (2)**	**7.92 (3)**	**10 (2)**
Black River/Lorain Subregion	11.9 (1)	0.55 (1)	15.5 (1)	7.39 (2)	5 (1)
Mahoning River Subregion	10.8 (3)	0.00[a] (2)	12.8 (2)	14.5 (1)	7 (2)
Milwaukee Region	**10.7 (3)**	**0.84 (2)**	**8.65 (3)**	**3.62 (4)**	**12 (3)**
Ohio Region	**5.81 (4)**	**0.42 (5)**	**4.07 (4)**	**15.5 (2)**	**15 (4)**
Cuyahoga River Subregion	15.2 (1)	0.58 (1)	12.1 (1)	55.1 (1)	4 (1)
Ashtabula Subregion	3.44 (2)	0.26 (3)	3.12 (2)	1.80 (3)	10 (2)
Toledo Subregion	2.41 (3)	0.43 (2)	1.06 (3)	2.01 (2)	10 (3)
Conneaut Subregion	0.81 (4)	0.09 (4)	0.71 (4)	0.34 (4)	16 (4)

Table 7-1. Summary of Mean PAH Sediment Concentrations, Mean Risk Scores, and Rankings for Regions and Subregions (continued)

Region, subregion	Sediment concentration (mg/kg) and (ranking)	Carcinogenic risk score ($\times 10^4$) and (ranking)	Non-carcinogenic risk score ($\times 10^2$) and (ranking)	Acute aquatic toxicity risk score ($\times 10^3$) and (ranking)	Overall ranking score and (ranking)
Fairport Harbor Subregion	0.55 (5)	0.07 (5)	0.40 (5)	0.17 (5)	20 (5)
New York/Pennsylvania Harbors Region	**3.00 (5)**	**0.45 (4)**	**2.19 (6)**	**1.06 (9)**	**24 (5)**
Dunkirk/Erie Subregion	28.8 (1)	2.46 (1)	22.8 (1)	8.69 (1)	4 (1)
Rochester Subregion	0.26 (2)	0.05 (2)	0.13 (2)	0.11 (3)	9 (2)
Oswego/Olcott Subregion	0.22 (3)	0.04 (3)	0.13 (3)	0.16 (2)	11 (3)
Buffalo Region	**2.64 (7)**	**0.19 (8)**	**2.92 (5)**	**2.16 (5)**	**25 (6)**
Rush Creek/Smokes Creek Subregion	10.81 (1)	0.67 (2)	7.92 (1)	3.69 (2)	6 (1)
Scajaquada Creek Subregion	8.38 (2)	0.70 (1)	7.25 (2)	6.35 (1)	6 (2)
Buffalo Harbor Subregion	4.22 (4)	0.33 (4)	5.37 (4)	3.19 (3)	15 (3)
Two Mile Creek Subregion	4.43 (3)	0.16 (6)	5.51 (3)	2.79 (5)	17 (4)
Lower Niagara River Subregion	3.07 (5)	0.46 (3)	3.57 (5)	1.44 (6)	19 (5)
Buffalo River Subregion	2.81 (6)	0.15 (7)	2.32 (6)	3.17 (4)	23 (6)
Tonawanda Canal Subregion	1.34 (7)	0.17 (5)	1.58 (7)	0.81 (8)	27 (7)
Lake Erie/Buffalo Subregion	0.93 (8)	0.04 (8)	1.20 (8)	1.34 (7)	31 (8)
Upper Niagara River/Chippawa Canal Subregion	0.09 (9)	0.00 (9)	0.13 (27)	0.02 (9)	36 (9)

Table 7-1. Summary of Mean PAH Sediment Concentrations, Mean Risk Scores, and Rankings for Regions and Subregions (continued)

Region, subregion	Sediment concentration (mg/kg) and (ranking)	Carcinogenic risk score ($\times 10^4$) and (ranking)	Non-carcinogenic risk score ($\times 10^2$) and (ranking)	Acute aquatic toxicity risk score ($\times 10^3$) and (ranking)	Overall ranking score and (ranking)
Detroit Region	**2.74 (6)**	**0.33 (6)**	**1.65 (8)**	**1.83 (7)**	**27 (7)**
Detroit River and Tributaries Subregion	3.16 (1)	0.37 (1)	1.94 (1)	2.13 (1)	4 (1)
St. Clair River/Lake Subregion	1.54 (2)	0.21 (2)	1.01 (2)	0.97 (2)	8 (2)
Raisin River Subregion	0.64 (3)	0.09 (3)	0.37 (3)	0.65 (3)	12 (3)
Lake Erie off Raisin River Subregion	0.19 (4)	0.01 (4)	0.19 (4)	0.05 (4)	16 (4)
Michigan Region	**2.06 (8)**	**0.26 (7)**	**1.70 (7)**	**2.07 (6)**	**28 (8)**
St. Joseph Harbor Region	4.53 (2)	0.24 (2)	4.60 (1)	2.46 (2)	6 (1)
Manistee Subregion	2.86 (2)	0.56 (1)	1.39 (2)	4.48 (1)	6 (2)
Manistique Subregion	0.26 (3)	0.03 (3)	0.15 (4)	0.40 (3)	13 (3)
Kalamazoo River Subregion	0.17 (4)	0.00 (5)	0.17 (3)	0.15 (5)	17 (4)
Muskegon/New Buffalo Subregion	0.13 (5)	0.02 (4)	0.10 (5)	0.36 (4)	18 (5)
Lake Superior Region	**1.15 (9)**	**0.10 (9)**	**0.91 (9)**	**1.34 (8)**	**35 (9)**

Table 7-1. Summary of Mean PAH Sediment Concentrations, Mean Risk Scores, and Rankings for Regions and Subregions (continued)

Region, subregion	Sediment concentration (mg/kg) and (ranking)	Carcinogenic risk score ($\times 10^4$) and (ranking)	Non-carcinogenic risk score ($\times 10^2$) and (ranking)	Acute aquatic toxicity risk score ($\times 10^3$) and (ranking)	Overall ranking score and (ranking)
Ashland Subregion	2.75 (1)	0.23 (1)	2.20 (1)	3.29 (1)	4 (1)
Miscellaneous Lake Superior Subregion	0.03 (2)	0.00 (2)	0.03 (2)	0.01 (2)	8 (2)
Open Lake Michigan Region	**0.40 (10)**	**0.02 (10)**	**0.41 (10)**	**0.10 (10)**	**40 (10)**

[a]No analysis for carcinogenic PAHs were performed on samples taken from the Mahoning River.

Table 7-1 also includes relative ranking numbers for regions and subregions, given in the Table in parentheses. The ranking numbers are specific to each hierarchical level of geographic classification. That is, the ranking number for any given subregion in Table 7-1 are relative to only the other subregions within the same region.

Regional and subregional rankings provided in Table 7-1 are arranged against four categories:

- o Sediment PAH concentration,
- o Carcinogenic risk score,
- o Non-carcinogenic risk score, and
- o Acute aquatic toxicity risk score.

Therefore, any particular subregion/region may be assigned a different relative ranking order in each of the four ranking categories.

Finally, an overall ranking score is provided in the extreme right hand column in Table 7-1. These overall ranking scores result from the accumulated, individual ranking scores of each region/subregion for the four categories (sediment concentration, carcinogenic risk, non-carcinogenic risk and acute aquatic toxicity risk). The overall ranking scores allow comparison of subregions within the same region, and all regions.

In developing the data used in preparing Table 7-1, it was seen that the highest mean human carcinogenic risk scores, averaged over the individual risk scores of each PAH reported from that sampling site, were calculated from 13 sites. These sites were recognized as exhibiting risk scores of >2.0 X 10,000. These sites were clustered around the Indiana Region (9 of the 13 sites) and, more specifically, around the Grand Calumet River Subregion (6 of the 13 sites).

Similarly, the highest mean human non-carcinogenic risk scores, averaged over the individual risk scores of each PAH reported from that sampling site, were calculated from 11 sites. These sites were recognized as exhibiting risk scores of >50 X 100. These sites were again clustered around the Indiana Region (9 of the 11 sites) and, more specifically, around the Grand Calumet River Subregion (7 of the 11 sites).

Finally, the highest mean acute aquatic toxicity risk scores, averaged over the individual risk scores of each PAH reported from that sampling site, were calculated from 15 sites. These sites were recognized in Table 7-1 as exhibiting risk scores of >25 X 1,000. These sites were again clustered around the Indiana Region (9 of the 15 sites) and, more specifically, around the Grand Calumet River Subregion (7 of the 15 sites).

7.3.1 Calculation of Human Carcinogenic Risk Scores

To calculate risk scores, PAHs were first classified as carcinogenic or non-carcinogenic to humans, based on available data. Human carcinogenic risk scores were then calculated for each carcinogenic PAH, at each sampling site, as products of the unit carcinogenic risk (UCR) factor times the daily dose of carcinogenic PAH due to the consumption of contaminated fish. These scores were derived using the following assumptions:

- Humans are exposed to PAHs in the aquatic system only through the consumption of contaminated fish
- Fish are at steady state with respect to PAH tissue concentrations and become contaminated with PAHs only by uptake from the water column
- The concentrations of PAHs in the water column are equal to those that would be present in interstitial water at equilibrium with the PAH contaminated sediment.

The last assumption is primarily responsible for computed human carcinogenic risk scores probably being several orders of magnitude greater than actual risks. The concentrations of PAHs in the water column even close to the sediments are much smaller than those that would be present in interstitial water at equilibrium with the sediment. However, without knowledge of the hydrodynamic characteristics of a water body, it is not possible to estimate the concentration of PAHs in the water column as a function of distance from, and PAH concentrations in, the sediment. Even if the hydrodynamic characteristics were known, such estimates would require modeling.

Since the only PAH for which a UCR has been established is benzo[a]pyrene, the UCRs of the other PAHs had to be assumed to be equal to the UCR for benzo[a]pyrene (11.5 day/kg/mg). Therefore, since the computed human carcinogenic risk scores are proportional to the UCR, they were positively biased for PAHs less carcinogenic than benzo[a]pyrene and negatively biased for PAHs more carcinogenic than benzo[a]pyrene.

7.3.2 Calculation of Human Non-Carcinogenic Risk Scores

Human non-carcinogenic risk scores (x 10^2) were calculated for each non-carcinogenic PAH sediment concentration or mean concentration at each sediment sampling site as the ratios of the daily doses of non-carcinogenic PAHs, due to the consumption of contaminated fish, to the acceptable daily intakes (ADIs) of these PAHs. The non-carcinogenic risk equation was derived using the same assumptions as used in the carcinogenic risk equation. Again, the assumption that PAH concentrations in the water column are equal to those that would be present in interstitial water in equilibrium with the sediment is primarily responsible for human non-carcinogenic risk scores probably being much higher than actual risks.

The ADIs for all of the non-carcinogenic PAHs except naphthalene were assumed to be equal to that of fluoranthene. Therefore, since the computed non-carcinogenic risk scores are inversely proportional to the ADI, they are positively biased for non-carcinogens less toxic than fluoranthene and negatively biased for non-carcinogens more toxic than fluoranthene.

7.3.3 Calculation of Acute Aquatic Toxicity Risk Scores

Acute aquatic toxicity risk scores (x 10^3) were calculated for each PAH sediment concentration or mean concentration at each sediment sampling site as the ratios of PAH concentrations in the water column to acute aquatic criteria, assuming that the PAH concentrations in the water column are equal to those that would be present in interstitial water in equilibrium with the sediment. That assumption may again make acute aquatic toxicity risk scores much higher than actual risks.

There have been no acute aquatic life criteria developed for any of the PAHs. Therefore, all of the PAHs except fluoranthene and naphthalene were assumed to have acute aquatic life criteria of 1.7 mg/L based on a no effect level of acenaphthene on bluegills. Acute aquatic criteria for napthalene and fluoranthene were assumed to be 2.3 mg/L and 4.0 mg/L, respectively, based on no effect levels. Therefore, since the acute aquatic life risk scores are inversely proportional to the assumed acute aquatic criteria, they will be positively biased for PAHs less toxic than acenaphthene (other than naphthalene and fluoranthene) and negatively biased for PAHs more toxic than acenaphthene.

7.4 DISCUSSION AND SUMMARY OF PAH SEDIMENT CONCENTRATIONS, ASSOCIATED RISK SCORES AND RANKINGS

The overall ranking score of a given region or subregion is equal to the sum of its individual rankings in each of the 4 categories of means (mean PAH sediment concentration, mean human carcinogenic risk score, mean human non-carcinogenic risk score, and mean acute aquatic toxicity risk score). The overall ranking scores are given in the extreme right hand column of Table 7-1.

A discussion of PAH sediment concentrations, associated risk scores and rankings is provided in the following pages, as organized by regions. The regions are discussed in order of increasing overall ranking score (decreasing overall risk). Subregions within a region are also discussed in order of increasing overall ranking scores (decreasing overall risk).

Although risk scores for individual sampling sites are not included in Table 7-1, they were integral to computation of the mean scores reported in this table and are considered in the following discussions.

7.4.1 Indiana Region

Of the 10 regions discussed in this report, the Indiana region ranks first, with the highest mean PAH sediment concentration (51.5 mg/ kg), the highest mean human carcinogenic risk score (1.22×10^{-4}), the highest mean human non-carcinogenic risk score (0.686), the highest mean acute aquatic toxicity risk score (9.81×10^{-2}), and the lowest overall ranking score (sum of regional rankings = 1 + 1 + 1 + 1 = 4). The Indiana region is a heavily industrialized steel producing area with numerous outfalls from various steel and petrochemical industry processes located along the East Branch of the Grand Calumet River, the Indiana Harbor Canal, and Indiana Harbor.

7.4.1.1 Grand Calumet River Subregion

Of 3 reported subregions in the Indiana region, the Grand Calumet River subregion has the highest mean PAH sediment concentration (88.6 mg/kg), the highest mean human carcinogenic risk score (2.02×10^{-4}), the highest mean human non-carcinogenic risk score (1.50), and the lowest overall ranking score

(sum of subregional rankings = 1 + 1 + 1 + 2 = 5). It ranks second with respect to mean acute aquatic toxicity risk score (7.56×10^{-2}).

Of 19 reported sediment sampling sites in the Grand Calumet River subregion, the 1st through 4th ranking sites are located downstream of U.S. Steel coke plant, sintering plant, and blast furnace outfalls. Therefore, the four highest ranking sampling sites in the highest ranking subregion of the highest ranking region are located at or just below coke plant, sintering plant, and blast furnace outfalls. However, these sites were sampled in 1972 and may not reflect current conditions. Of the five U.S. Steel outfalls implicated, only the blast furnace outfall (# 017) was described as discharging any process wastewater in 1983. The other four outfalls were described as discharging only cooling (non-contact) water.

The 5th through 8th ranking sites in the Grand Calumet River subregion are located within two miles downstream of five U.S. Steel outfalls. Discharges from these outfalls in 1983 included cooling and/or process water from bar plate mills, basic oxygen steel making furnaces, an atmospheric gas plant, a treatment plant, miscellaneous finishing operations, and hot strip mill recycling processes.

The rankings of the sampling sites in the Grand Calumet River subregion can be summarized as follows. The four highest ranking sites are located between river miles 12 and 13.4 in the vicinity of several U.S. Steel coke plant, sintering plant, and blast furnace outfalls. However, data for the four highest ranking sites were reported in 1972 and may not reflect current conditions. The 5th through 8th ranking sites are located between river mile 8.6 and 11.5, at or downstream of several other types of U.S. Steel outfalls and/or the Gary POTW outfall. The 9th through 15th ranking sites are located throughout the Grand Calumet River subregion. Two of the four lowest ranking sites are located in the West Branch of the Grand Calumet River. The other two lowest ranking sites are located upstream of any known industrial point source discharger.

7.4.1.2 Indiana Harbor Subregion

Of 3 reported subregions in the Indiana region, the Indiana Harbor subregion has the highest mean acute aquatic toxicity risk score (9.81×10^{-3}) and the second highest mean PAH sediment concentration (61.4 mg/kg), mean

human carcinogenic risk score (1.36×10^{-4}), and mean human non-carcinogenic risk score (0.311). This subregion has the lowest overall ranking score (2 + 2 + 2 + 1 = 7).

Of four reported sampling sites in the Indiana Harbor subregion, the highest ranking site (ACOE) has by far the highest mean PAH concentration (226 mg/kg) and associated risk scores of the four sampled sites in Indiana Harbor and has the fourth highest mean PAH concentration of all the sites in the Indiana region. Unfortunately, the exact location of this site within Indiana Harbor was not specified.

The second highest ranking site in the Indiana Harbor subregion (LTI) is located west of the entrance to the Canal, in the vicinity of several Inland Steel outfalls. Discharges from those outfalls include blast furnace blow downs, coke plant cooling water, hearth furnace cooling water, and treated blast furnace process water. The lowest ranking sites in the Indiana Harbor subregion (S11 and LTI 2) are located at the entrance to Indiana Harbor from Lake Michigan. Both sites have mean PAH sediment concentrations less than 1 mg/kg.

The ranking of the Indiana Harbor subregion ahead of the Indiana Harbor Canal subregion is due to the mean PAH sediment concentration and associated risk scores at only one site, whose location is not specified. The mean PAH concentrations and associated risk scores of the other three reported sites in the Indiana Harbor subregion are lower than for most of the other reported sites in both the Grand Calumet River and Indiana Harbor subregions. Although the number of sites for which data are reported is too low to make definite conclusions, the relatively low mean PAH sediment concentrations at the two sites at the entrance to the harbor from Lake Michigan may indicate that very little of the PAH contamination from the Grand Calumet River, Indiana Harbor Canal, and Indiana Harbor subregions is entering Lake Michigan via the water column.

7.4.1.3 Indiana Harbor Canal Subregion

Of 3 reported subregions in the Indiana region, the Indiana Harbor Canal subregion ranks third with respect to mean PAH sediment concentration (8.40 mg/kg), mean carcinogenic risk score (4.3×10^{-5}), mean non-carcinogenic risk score (0.092), mean acute aquatic toxicity risk score (5.07×10^{-3}), and over-

all ranking score (3 + 3 + 3 + 3 = 12). However, the mean PAH sediment concentration and associated risk scores for most of the 12 reported sites in the Indiana Harbor Canal subregion are substantially greater than those for three of the four reported sites in the Indiana Harbor subregion.

In summary, the four highest ranking sites in the Indiana Harbor Canal subregion are all located within one mile of the fork where the Indiana Harbor Canal divides into the Lake George and Grand Calumet branches (river mile 2.1). Furthermore, each of the three segments of the Indiana Harbor Canal is represented by one of the four highest ranking sites, with one site located at the Fork, one site in the Lake George Branch, one site in the Grand Calumet Branch, and one site in the Indiana Harbor Canal downstream of the Fork. However, all of the four highest ranking sites were sampled in 1977. PAH levels in sediment samples taken at similar sites in 1980 and 1983 were much lower, even though the U.S. Steel Plant was still operating. Therefore, there may have been substantial reductions in the PAH levels present in the effluents since 1977.

7.4.2 Ohio Steel Region

Of 10 regions discussed in this report, the Ohio Steel region ranks second with respect to mean PAH sediment concentration (11.8 mg/kg), third with respect to mean human carcinogenic risk score (5.5×10^{-3}), second with respect to mean human non-carcinogenic risk score (0.152), third with respect to mean acute aquatic toxicity risk score, and second with respect to overall ranking score (2 + 3 + 2 + 3 = 10).

The Ohio Steel region is divided into the Black River/Lorain and Mahoning River subregions. The Ohio Steel region was separated from the Ohio region because most of the PAH data in the Black River/Lorain subregion and all of the PAH data in the Mahoning River subregion are for sites in the vicinity of steel industry outfalls. Some of the PAH data in the Cuyahoga River subregion of the Ohio region were also for sites located in the vicinity of steel industry outfalls. Therefore, the Cuyahoga subregion, which is the highest ranking subregion of the Ohio region, could be considered with the Ohio Steel region.

7.4.2.1 Black River/Lorain Subregion

Of the two reported subregions in the Ohio Steel region, the Black River/Lorain subregion ranks first, with the highest mean sediment concentration (11.9 mg/kg), the highest mean carcinogenic risk score (5.5×10^{-5}), the highest mean non-carcinogenic risk score (0.155), and the lowest overall ranking score (1 + 1 + 1 + 2 = 5). It ranks second with respect to mean aquatic toxicity risk score (7.39×10^{-3}).

Based on data from 20 reported sampling sites, it appears that the major source of PAHs to the Black River/Lorain subregion was the U.S. Steel coke plant outfall 002. However, it also appears that most of the PAH discharged at U.S. Steel outfall 002 is deposited in the immediate vicinity of the outfall (e.g., within 500 feet) and does not appear to affect Lake Erie. Other moderate sources of PAH to the Black River/ Lorain subregion may include a sediment dredging disposal site, U.S. Steel outfalls 003 and/or 004, and the Ashland Oil outfall. The PAH concentrations in sediment samples taken in Lake Erie off the mouth of the Black River, at the mouth of the Black River, and upstream of U.S. Steel outfall 002 were relatively low (e.g., $\leq$ 1 mg/kg) compared to those in samples taken from most other sites in the Black River/ Lorain subregion.

Several sites were sampled in 1974 and may not reflect current conditions. The U.S. Steel coke plant in Lorain has reportedly been shut down.

7.4.2.2 Mahoning River Subregion

Of the 2 reported subregions in the Ohio Steel region, the Mahoning River subregion ranks second (last) with respect to mean PAH sediment concentration (10.8 mg/kg), mean human carcinogenic risk score (0.0), mean non-carcinogenic risk score (0.128) and overall ranking score (2 + 2 + 2 + 1 = 7). It ranks first with respect to mean acute aquatic toxicity risk score (1.45×10^{-2}).

The computed mean carcinogenic risk score for the Mahoning River subregion is 0.0 because none of the 7 PAHs identified in the Mahoning River study are considered to be carcinogenic. However, other PAHs were present (but were not identified) which could possibly be carcinogenic.

Three reported sediment sampling sites in the Mahoning River subregion are located below coke oven outfalls. However, these sites were all sampled

in 1975 and may therefore not reflect current conditions. Although the Republic (LTV) Steel/Warren Plant is still operating, the Republic (LTV) Steel/Youngstown Plant has reportedly been shut down. No information on the current status of the Youngstown Sheet and Tube-Campbell Works (Struthers) was available.

7.4.3 Milwaukee Region

Of the 10 regions discussed in this report, the Milwaukee region ranks third with respect to mean PAH concentration (10.7 mg/kg), second with respect to mean human carcinogenic risk score (8.4×10^{-5}), third with respect to mean human non-carcinogenic risk score (8.65×10^{-2}), fourth with respect to mean aquatic toxicity risk score and third with respect to overall ranking score (3 + 2 + 3 + 4 = 12). The Milwaukee region is not divided into subregions.

The PAH contamination of sediments appears to be fairly widespread in the Milwaukee region. Of 12 reported sediment samplings sites in the Milwaukee region, the first and sixth ranking sites (with respect to overall ranking score) are in the Milwaukee River, the second and fifth ranking sites are in the Menomonee River and the third and fourth ranking sites are in the Kinnickinnic River. Oil and grease concentrations exceed 10 g/kg of sediment at the 6 highest ranking sites and at 8 of the 12 reported sites overall.

7.4.4 Ohio Region

Of 10 regions covered in this report, the Ohio region ranks fourth with respect to mean PAH sediment concentration (5.81 mg/kg), fifth with respect to mean human carcinogenic risk score (4.2×10^{-5}), fourth with respect to mean human non-carcinogenic risk score (4.07×10^{-2}), second with respect to mean acute aquatic toxicity risk score (1.55×10^{-2}) and fourth with respect to overall ranking score (4 + 5 + 4 + 2 = 15). The second-place ranking of the Ohio region with respect to mean acute aquatic toxicity score is due primarily to extremely high concentrations of naphthalene, acenaphthylene, fluorene, and phenanthrene in sediment samples taken from a single site (CUY 82-18) in the Cuyahoga River below an LTV (Republic) Steel outfall.

7.4.4.1 Cuyahoga River Subregion

Of the 5 reported subregions in the Ohio region, the Cuyahoga River subregion ranks first with respect to mean PAH sediment concentration (15.2 mg/kg), mean human carcinogenic risk score (6.8×10^{-5}), mean non-carcinogenic risk score (1.21×10^{-2}), acute aquatic toxicity risk score (5.51×10^{-2}), and overall ranking score (1 + 1 + 1 + 1 = 4).

The mean PAH sediment concentration and associated mean risk scores are much higher for the Cuyahoga River subregion than for any other subregion in the Ohio region, and exceed those for the Black River/Lorain and Mahoning River subregions of the Ohio Steel region. The relatively high mean PAH sediment concentration and associated risk scores for the Cuyahoga River subregion is primarily due to the high mean PAH sediment concentration (75.7 mg/kg) at one sediment sampling site (CUY 82-18) which is located at an LTV (Republic) Steel outfall. Therefore, it may be more appropriate to transfer the Cuyahoga River subregion from the Ohio region to the Ohio Steel region.

7.4.4.2 Ashtabula Subregion

Of 5 reported subregions in the Ohio region, the Ashtabula subregion ranks second with respect to mean PAH sediment concentration (3.44 mg/kg), third with respect to mean human carcinogenic risk score (2.6×10^{-5}), second with respect to mean human non-carcinogenic risk score (3.12×10^{-2}), third with respect to the acute aquatic toxicity risk score (1.80×10^{-3}) and second with respect to overall ranking score.

Of 12 reported sampling sites in the Ashtabula subregion, the highest five sites, with respect to both the lowest overall ranking score and the highest mean PAH sediment concentration, are all located in the Ashtabula River. The sixth and eighth ranking sites are located in the harbor. The three lowest ranking sites (10th through 12th) all have mean PAH sediment concentrations less than 1 mg/kg.

7.4.4.3 Toledo Subregion

Of 5 reported subregions in the Ohio region, the Toledo subregion ranks third with respect to mean PAH sediment concentration (2.41 mg/ kg), mean human carcinogenic risk score (4.3×10^{-5}), and mean human non-carcinogenic

risk score (1.06×10^{-2}), and ranks second with respect to mean acute aquatic toxicity risk score (2.01×10^{-3}). The Toledo subregion ranks third with respect to overall ranking score.

7.4.4.4 Conneaut Subregion

Of 5 reported subregions in the Ohio region, the Conneaut subregion ranks fourth with respect to mean PAH sediment concentration (0.81 mg/kg), mean human carcinogenic risk score (9×10^{-6}), mean human non-carcinogenic risk score (7.1×10^{-3}), mean acute aquatic toxicity risk score (3.4×10^{-4}), and overall ranking score. Only one of the 4 reported sampling sites in the Conneaut subregion has a mean PAH sediment concentration exceeding 1 mg/kg (1.04 mg/kg for CON 82-02). However, all 4 sites are located in the Harbor. Therefore, no information is available on the levels of PAHs in the Conneaut River.

7.4.4.5 Fairpoint Harbor Subregion

Of 5 reported subregions in the Ohio region, the Fairpoint Harbor subregion ranks fifth with respect to mean PAH sediment concentration (0.55 mg/kg), mean human carcinogenic risk score (7×10^{-5}), mean human non-carcinogenic risk score (4.0×10^{-3}), mean acute aquatic toxicity risk score (2.0×10^{-2}) and overall ranking score. Only one of the 6 reported sampling sites in the Fairpoint Harbor subregion exceeds 1 mg/kg (1.04 mg/kg for FPH 82-02). From the latitudes and longitudes given in STORET, it appears that 3 of the 6 sampling sites may be in the Grand River.

7.4.5 New York/Pennsylvania Region

Of 10 regions covered in this report, the NY/PA region ranks fifth with respect to mean PAH concentration (3.00 mg/kg), fourth with respect to mean human carcinogenic risk score (4.5×10^{-5}), sixth with respect to mean human non-carcinogenic risk score (2.19×10^{-2}), ninth with respect to mean acute aquatic toxicity risk score (1.06×10^{-3}) and fifth with respect to overall ranking score (5 + 4 + 6 + 9 = 24).

7.4.5.1 Dunkirk/Erie Subregion

Of 3 reported subregions in the NY/PA region, the Dunkirk/Erie subregion ranks first with respect to mean PAH sediment concentration (28.0 mg/kg), mean human carcinogenic risk score (2.46×10^{-4}), mean human non-carcinogenic risk score (0.228), mean acute aquatic toxicity risk score (8.69×10^{-3}) and overall ranking score (1 + 1 + 1 + 1 = 4). The mean PAH sediment concentrations and associated mean risk scores are much higher for the Dunkirk/Erie subregion than for the other two subregions (Rochester and Oswego/Olcott). The mean sediment concentrations for the 5 reported sampling sites in the Dunkirk/Erie subregion are higher than for any of the other reported sites in the NY/PA region. The highest ranking site (DNK 82-06) is located off Dunkirk in Lake Erie at an unidentified outfall beside the main beach. The second highest ranking site (ERH 82-05) is in the Erie area but its specific location is not described in STORET.

7.4.5.2 Rochester Subregion

Of 3 reported subregions in the NY/PA region, the Rochester subregion ranks second with respect to mean PAH sediment concentration (0.26 mg/kg), mean human carcinogenic risk score (5.0×10^{-6}), mean human non-carcinogenic risk score (1.3×10^{-3}), and overall ranking score (2 + 2 + 2 + 3 + = 9). It ranks third (last) with respect to mean acute aquatic toxicity risk score (1.10×10^{-4}). None of the 13 reported sediment sampling sites in the Rochester subregion have mean PAH sediment concentrations exceeding 1 mg/kg even though some of the sites are located downstream of various Kodak outfalls. All 13 sites are located in the Genessee River. The highest ranking site (lowest overall ranking score) in the subregion (ROC 81-02) is located near a storm sewer.

7.4.5.3 Oswego/Olcott Subregion

Of 3 reported subregions in the NY/PA region, the Oswego/Olcott subregion ranks third and last with respect to mean PAH sediment concentration (0.22 mg/kg), mean human carcinogenic risk score (4.0×10^{-6}), mean human non-carcinogenic risk score (1.3×10^{-3}), and overall ranking score (3 + 3 + 3 + 2 = 11). It ranks second with respect to mean acute aquatic toxicity risk score (1.6×10^{-4}). Of 7 reported sampling sites in the Oswego/Olcott subregion,

the highest ranking site (lowest overall ranking score) is located in a swampy area which drains to Wine Creek, in Oswego, and is the only site in the Oswego/Olcott region with a mean PAH sediment concentration exceeding 1 mg/kg (1.29 mg/kg). The second highest ranking site in the subregion, and the highest ranking site in the Olcott area, is located near the mouth of 18 Mile Creek.

7.4.6 Buffalo Region

Of the 10 regions covered in this report, the Buffalo region ranks seventh with respect to mean PAH sediment concentration (2.64 mg/kg), eighth with respect to mean human carcinogenic risk score (1.9×10^{-5}), fifth with respect to mean human non-carcinogenic risk score (2.9×10^{-2}), fifth with respect to mean acute aquatic toxicity risk score (2.16×10^{-3}), and sixth with respect to overall ranking score (7 + 8 + 5 + 5 = 25).

7.4.6.1 Rush Creek/Smokes Creek Subregion

Of 9 reported subregions in the Buffalo region, the Rush Creek/Smokes Creek subregion ranks first with respect to mean PAH sediment concentration (10.8 mg/kg), second with respect to mean human carcinogenic risk score (6.7×10^{-5}), first with respect to mean human non-carcinogenic risk score (7.92×10^{-2}), second with respect to mean acute aquatic toxicity risk score (3.69×10^{-3}) and first with respect to overall ranking score (1 + 2 + 1 + 2 = 6).

The Rush Creek/Smokes Creek subregion consists of only one sediment sampling site in Rush Creek and one sediment sampling site in Smokes Creek. Smokes Creek empties into Lake Erie just south of Buffalo Harbor. Rush Creek empties into Lake Erie just south of Rush Creek.

The Rush Creek site which has the highest mean PAH sediment concentration of any reported site in the entire Buffalo region (2.1 mg/kg). The mean PAH sediment concentration for the Smokes Creek site is 1.30 mg/kg. Unfortunatly, specific locations for the Rush Creek and Smokes Creek sites were not described. However, Bethlehem Steel outfalls discharge to both Rush and Smokes Creeks.

7.4.6.2 Scajaquada Creek Subregion

Of 9 reported subregions in the Buffalo region, the Scajaquada Creek subregion ranks second with respect to mean PAH sediment concentration (8.38 mg/kg), first with respect to mean carcinogenic risk score (7.0×10^{-5}), second with respect to mean human non-carcinogenic risk score (7.25×10^{-2}), first with respect to mean acute aquatic toxicity risk score (6.35×10^{-3}), and second with respect to overall ranking score (2 + 1 + 2 + 1 = 6). (Its overall ranking score is actually identical to that of the Rush Creek/Smokes Creek subregion, but it is ranked below that subregion due to a lower mean PAH sediment concentration.) Scajaquada Creek empties into the lower Niagara River/Black Rock Canal north of the Buffalo River.

The mean PAH sediment concentrations for two of the seven sites reported for this subregion are the second and third highest, respectively, of all reported sites in the entire Buffalo region.

Although the Scajaquada creek subregion ranks second among the 9 subregions in the Buffalo region, no descriptions of the sampling sites were available. Furthermore, none of the currently-recognized, potentially significant point and non-point pollution sources for the Niagara River System are located along Scajaquada Creek.

7.4.6.3 Buffalo Harbor Subregion

The Buffalo Harbor subregion ranks fourth with respect to mean PAH sediment concentration (4.22 mg/kg), mean human carcinogenic risk score (3.3×10^{-5}), and mean human non-carcinogenic risk score (5.37×10^{-2}). It ranks third with respect to mean acute aquatic toxicity risk score (3.19×10^{-3}) and overall ranking score (4 + 4 + 4 + 3 = 15).

Of 19 reported sites in the Buffalo Harbor subregion, the five highest ranking sites, with respect to both overall ranking scores and mean PAH sediment concentrations, are located at the mouths of either the Lackawana or Unionship Canals where they empty into the south end of the harbor. The mouths (entrances) to both canals are in the vicinity of Bethlehem Steel.

7.4.6.4 Two Mile Creek Subregion

Of 9 subregions in the Buffalo region, the Two Mile Creek subregion ranks third with respect to mean PAH sediment concentration (4.43 mg/kg), sixth with respect to mean human carcinogenic risk score (1.6×10^{-5}), third with respect to mean human non-carcinogenic risk score (5.51×10^{-2}), fifth with respect to mean acute aquatic toxicity risk score (2.79×10^{-3}), and fourth with respect to overall ranking score (4 + 4 + 4 + 3 = 15). Of 9 reported sites in the Two Mile Creek subregion, the highest ranking site, with respect to overall ranking score, is located in the vicinity of Union Carbide Corp., Linde Div., which manufactures and fabricates cyrogenic hardware.

7.4.6.5 Lower Niagara River/Black Rock Canal Subregion

Of 9 subregions in the Buffalo region, the Lower Niagara River/Black Rock Canal subregion ranks fifth with respect to mean PAH sediment concentration (3.07 mg/kg), third with respect to mean human carcinogenic risk score (4.6×10^{-5}), fifth with respect to mean human non-carcinogenic risk score (3.57×10^{-2}), sixth with respect to mean acute aquatic toxicity risk score (1.44×10^{-3}), and fifth with respect to overall ranking score (5 + 3 + 5 + 6 = 18).

Of 15 reported sites in the Lower Niagara River subregion, the 3 highest ranking sites, with respect to both overall ranking score and mean PAH sediment concentration, are located at the mouth of Scajaquada Creek and in Black Rock Canal, south of Squaw Island. There are no significant point sources listed in the vicinity of the Scajaquada Creek site (BUF-81-31), however there is a waste disposal site on Squaw Island which contains foundry sand, incinerator residues, trace oils, resins, and municipal wastes.

7.4.6.6 Buffalo River Subregion

Of 9 subregions in the Buffalo region, the Buffalo River subregion ranks sixth with respect to mean PAH sediment concentration (2.81 mg/kg), seventh with respect to mean human carcinogenic risk score (1.5×10^{-5}), sixth with respect to mean human non-carcinogenic risk score (2.32×10^{-2}), fourth with respect to mean acute aquatic toxicity risk score (3.17×10^{-3}), and sixth with respect to overall ranking score (6 + 7 + 6 + 4 = 23). Of 31 reported sites in the Buffalo River subregion, 4 of the 6 highest ranking sites, with respect to overall ranking score, are located at or near outfalls of Republic Steel, Donner-Coke, Buffalo Color, and a combined sewer.

Although the Buffalo River subregion contains a few sites with relatively high levels of PAH contamination, its overall ranking with respect to the various criteria is relatively low, compared to a number of other subregions in the Buffalo region, due to the relatively low PAH levels in sites upstream of the Buffalo Color, Republic Steel, and Donner-Coke outfalls and in sites near the mouth of the Buffalo River subregion, 13 of which have mean PAH sediment concentrations below 1 mg/kg.

7.4.6.7 Tonawanda Creek Subregion

Of 9 subregions in the Buffalo region, the Tonawanda Canal ranks seventh with respect to mean PAH concentration (1.34 mg/kg), fifth with respect to mean human carcinogenic risk score (1.7×10^{-5}), seventh with respect to mean human non-carcinogenic risk score (1.58×10^{-2}), eighth with respect to mean acute aquatic toxicity risk score (8.1×10^{-4}), and seventh with respect to overall ranking score (7 + 5 + 7 + 8 = 27). Of 31 reported sites in the Tonawanda Canal subregion, the 5 highest ranking sites, with respect to both overall ranking score and mean PAH sediment concentration, are located at storm sewer overflows, the Chevrolet outfall, at the mouth of Two Mile Creek (downstream of the Tonawanda WWTP) and at the Hooker Chemical outfall. Twenty of the 31 reported sites in the Tonawanda Canal subregion had mean PAH sediment concentrations less than 1 mg/kg or had non-detectable levels of PAH.

7.4.6.8 Lake Erie Subregion

Of 9 subregions in the Buffalo region, the Lake Erie subregion ranks eighth with respect to mean PAH sediment concentration (0.93 mg/kg), mean human carcinogenic risk score (4×10^{-6}), mean human non-carcinogenic risk score (1.20×10^{-2}), and overall ranking score (8 + 8 + 8 + 7 = 31). It ranks seventh with respect to mean acute aquatic toxicity risk score (1.34×10^{-3}). Of 33 reported sites in the Lake Erie subregion, the 4 highest ranking sites, with respect to both overall ranking score and mean PAH sediment concentration, are in Lake Erie north of the mouth of Smokes Creek and offshore from the Bethlehem Steel waste disposal site and a sediment dredging disposal site. Of the 29 remaining sites, both north and south of the mouth of Smokes Creek, 25 have mean PAH sediment concentrations below 1 mg/kg.

7.4.6.9 Upper Niagara River/Chippawa Canal Subregion

Of 9 subregions in the Buffalo region, the Upper Niagara River/Chippawa Canal subregion ranks ninth with respect to mean PAH sediment concentration (0.09 mg/kg), mean carcinogenic risk score (0.00), mean non-carcinogenic risk score (1.3×10^{-3}), mean acute aquatic toxicity risk score (2×10^{-5}), and overall ranking score (9 + 9 + 9 + 9 = 36). Both of the 2 reported sampling sites in the Chippawa Canal and both of the 2 reported sampling sites in the Upper Niagara River have mean PAH sediment concentrations below 1 mg/kg.

7.4.7 Detroit Region

Of 10 regions covered in this report, the Detroit region ranks sixth with respect to mean PAH sediment concentration (2.74 mg/kg) and mean human carcinogenic risk score (3.3×10^{-5}), eighth with respect to mean human non-carcinogenic risk score (1.65×10^{-2}), seventh with respect to mean acute aquatic toxicity risk score (1.83×10^{-3}), and seventh with respect to overall ranking score (6 + 6 + 8 + 7 = 27).

7.4.7.1 Detroit River/Tributaries Subregion

Of 4 reported subregions in the Detroit region, the Detroit River/ Tributaries subregion ranks first with respect to mean PAH sediment concentration (3.16 mg/kg), mean human carcinogenic risk score (3.7×10^{-5}), mean human non-carcinogenic risk score (1.94×10^{-2}), mean acute aquatic toxicity risk score (2.13×10^{-3}), and overall ranking score (1 + 1 + 1 + 1 = 4). Of 28 reported sites in the Detroit River/Tributaries subregion, the 8 highest ranking sites, with respect to both overall ranking score and mean PAH sediment concentration, are located in the Rouge River (3 of the 8) at Zug Island, near the National Steel outfall or the Ford Motor Company Steel producing facilities, or in the upper Detroit River (the other 5 sites) between Belle Island and the mouth of the Ecorse River. The fifth ranking site (DTR 82-22) is located at the Slip Fuel Oil Corp. Only 5 of the 28 reported sites in the Detroit River/Tributaries subregion have mean PAH concentrations below 1 mg/kg.

7.4.7.2 St. Clair River/Lake Subregion

Of 4 reported subregions in the Detroit region, the St. Clair River/ Lake subregion ranks second with respect to mean PAH sediment concentration (1.54 mg/kg), mean carcinogenic risk score (2.1×10^{-5}), mean non-carcinogenic risk score (1.01×10^{-2}), mean acute aquatic toxicity risk score (9.7×10^{-4}), and overall ranking score (2 + 2 + 2 + 2 = 8). The two highest ranking sites in the subregion are located in the St. Clair River at the Ft. River Bridge and in Lake St. Clair, 30 feet offshore, below the Clinton STP.

7.4.7.3 Raisin River Subregion

Of 4 reported subregions in the Detroit region, the Raisin River subregion ranks third with respect to mean PAH sediment concentration (0.64 mg/kg), mean human carcinogenic risk score (9.0×10^{-6}), mean human non-carcinogenic risk score (3.7×10^{-3}), mean acute aquatic toxicity risk score (6.5×10^{-4}), and overall ranking score (3 + 3 + 3 + 3 = 12).

Of 5 reported sites in the Raisin River subregion, the highest ranking site, with respect to both overall ranking score and mean PAH sediment concentration, is located near the mouth of the Raisin River at the Ford Motor Co. RO outfall. This site (MON 81-05) is the only site in the Raisin River subregion with a mean PAH sediment concentration exceeding 1 mg/kg (1.70 mg/kg). The second ranking site is located downstream of the Monroe STP and has a mean PAH sediment concentration of 0.70 mg/kg.

7.4.7.4 Lake Erie Subregion

Of 4 reported subregions in the Detroit region, the Lake Erie subregion, off the mouth of the Raisin River, ranks fourth and last with respect to mean PAH sediment concentration (0.19 mg/kg), mean carcinogenic risk score (1.0×10^{-5}), mean non-carcinogenic risk score (1.9×10^{-3}), mean acute aquatic toxicity risk score (5×10^{-5}), and overall ranking score (4 + 4 + 4 + 4 = 16). All 5 of the reported sites in the Lake Erie subregion are within a 10 km arc of the mouth of the Raisin River. The mean PAH sediment concentrations for all 5 sites are less than 1 mg/kg.

7.4.8 Michigan Region

Of 10 regions covered in this report, the Michigan region ranks eighth with respect to mean PAH sediment concentration (2.06 mg/kg), seventh with respect to mean human carcinogenic risk score (2.6×10^{-5}), seventh with respect to mean human non-carcinogenic risk score (1.70×10^{-2}), sixth with respect to mean acute aquatic toxicity risk score (2.07×10^{-3}), and eighth with respect to overall ranking score (8 + 7 + 7 + 6 = 28).

7.4.8.1 St. Joseph Harbor Subregion

Of 5 reported subregions in the Michigan region, the St. Joseph Harbor subregion ranks first with respect to mean PAH sediment concentration (4.53 mg/kg), second with respect to mean human carcinogenic risk score (2.4×10^{-5}), first with respect to mean human non-carcinogenic risk score (4.60×10^{-2}), second with respect to mean acute aquatic toxicity risk score (2.46×10^{-3}), and first with respect to overall ranking score (1 + 2 + 1 + 2 =6).

7.4.8.2 Manistee Subregion

Of 5 reported subregions in the Michigan region, the Manistee subregion ranks second with respect to mean PAH sediment concentration (2.86 mg/kg), first with respect to mean human carcinogenic risk score (5.6×10^{-5}), second with respect to mean human non-carcinogenic risk score (5.6×10^{-5}), first with respect to mean acute aquatic toxicity risk score (4.48×10^{-3}), and second with respect to overall ranking score (2 + 1 + 2 + 1 = 6). (Although the overall ranking score of the Manistee subregion is the same as for the St. Joseph Harbor subregion, it was ranked second based upon its lower mean PAH sediment concentration.)

Of 9 reported sites in the Manistee subregion, the 4 highest ranking sites, with respect to both overall ranking score and mean PAH sediment concentration, are located in the harbor, at or near the Great Lakes Chem. Corp., Standards Lime and Cement Company, Pkg. Corp. of America, and 2000 meters north of the Little Manistee River.

7.4.8.3 Manistique Subregion

Of 5 reported subregions in the Michigan region, the Manistique subregion ranks third with respect to mean PAH sediment concentration (0.26 mg/kg), mean human carcinogenic risk score (3.0×10^{-6}), mean acute aquatic toxicity risk score (4.0×10^{-4}), and overall ranking score (3 + 3 + 4 + 3 = 13). It ranks fourth with respect to mean human non-carcinogenic risk score (1.5×10^{-3}). All of the 7 reported sampling sites in the Manistique subregion are located in Manistique Harbor and all have mean PAH sediment concentrations less than 1 mg/kg.

7.4.8.4 Kalamazoo River Subregion

Of 5 reported subregions in the Michigan region, the Kalamazoo River subregion ranks fourth with respect to mean PAH sediment concentration (0.17 mg/kg), fifth with respect to mean human carcinogenic risk score (0.0), third with respect to mean human non-carcinogenic risk score (1.7×10^{-3}), fifth with respect to mean acute aquatic toxicity risk score (1.5×10^{-4}), and fourth with respect to overall ranking score (4 + 5 + 3 + 5 = 17). All 4 of the reported sites in the Kalamazoo River subregion are located in the Kalamazoo River in West Allegany County. All 4 sites have mean PAH sediment concentrations well below 1.0 mg/kg.

7.4.8.5 Muskegon/New Buffalo Harbor Subregion

Of the 5 reported subregions in the Michigan region, the Muskegon/New Buffalo Harbor subregion ranks fifth with respect to mean PAH sediment concentration (0.13 mg/kg), fourth with respect to mean human carcinogenic risk score (2.0×10^{-6}), fifth with respect to mean human non-carcinogenic risk score (1.0×10^{-3}). fourth with respect to mean acute aquatic toxicity risk score (3.6×10^{-4}), and fifth with respect to overall ranking score (5 + 4 + 5 + 4 = 8). The mean PAH sediment concentrations for all 5 reported sites are well below 1.0 mg/kg.

7.4.9 Lake Superior Region

Of the 10 regions covered by this report, the Lake Superior region ranks ninth with respect to mean PAH sediment concentration (1.15 mg/kg), mean carcinogenic risk score (1.0×10^{-5}), mean non-carcinogenic risk score (9.1 x

10^{-3}) and overall ranking score (9 + 9 + 9 + 8 = 26). It ranks eighth with respect to mean acute aquatic toxicity risk score.

7.4.9.1 Ashland Subregion

Of 2 reported subregions in the Lake Superior region, the Ashland subregion ranks first with respect to mean PAH sediment concentration (2.75 mg/kg), mean carcinogenic risk score (1.0×10^{-5}), mean non-carcinogenic risk score (2.20×10^{-2}), mean acute aquatic toxicity risk score (3.29×10^{-3}), and overall ranking score (1 + 1 + 1 + 1 = 2). All 3 reported sampling sites in the Ashland subregion are in Chequamegon Bay (Lake Superior) just offshore of Ashland, Wisconsin. The highest ranking site (ASH 81-03) is located just off a sewage treatment plant outfall and is also in the vicinity of coal storage and handling facilities. The second ranking site (ASH 81-01) is located off a power plant outfall.

7.4.9.2 Miscellaneous Lake Superior Subregion

Of the 2 reported subregions in the Lake Superior region, the Miscellaneous Lake Superior subregion ranks second (last) with respect to mean PAH sediment concentration (0.03 mg/k), mean carcinogenic risk score (0.00), mean non-carcinogenic risk score (3.0×10^{-4}), mean acute aquatic toxicity risk score (1.0×10^{-5}), and overall ranking score (2 + 2 + 2 + 2 = 8). None of the five reported sampling sites in the Miscellaneous Lake Superior subregion has a mean PAH sediment concentration above 0.11 mg/kg.

7.4.10 Open Lake Michigan Region

Of the 10 regions covered in this report, the Open Lake Michigan region ranks tenth and last with respect to mean PAH sediment concentration (0.40 mg/kg), mean human carcinogenic risk score (2×10^{-6}), mean human non-carcinogenic risk score (4.1×10^{-3}), mean acute aquatic toxicity risk score (1.0×10^{4}), and overall ranking score (10 + 10 + 10 + 10 = 40). Only 1 of the 12 reported sites in the Open Lake Michigan region has a mean PAH sediment concentration exceeding 1 mg/kg (1.29 mg/kg for T3). All of the 12 sites are located in the southern half of Lake Michigan.

7.5 CONCLUSIONS AND RECOMMENDATIONS

Topics addressed in this portion of the Summary include the following: Conclusions with Respect to Sources of PAH Contamination in Sediments (7.5.1), Recommendations with Respect to the Sampling and Analysis of Sediments for PAHs (7.5.2), and Recommendations with Respect to Risk Formulae and the Sampling of Other Media (7.5.3).

7.5.1 Conclusions with Respect to Sources of PAH Contamination in Sediment

The highest mean PAH sediment concentrations in the 10 regions considered in this report are generally in the vicinity of coke plants or otherwise unidentified steel industry outfalls. Most of the mean PAH sediment concentrations at these sites exceed 5 mg/kg with several exceeding 100 mg/kg. Mean PAH sediment concentrations exceeded 10 mg/kg in at least some of the sites in the vicinity of the following coke plants and/or otherwise unidentified steel industry outfalls:

- U.S. Steel Coke Plant outfalls 002, 005, 007, 010 to the Grand Calumet River in Indiana (still operational but samples taken in 1972)
- Possibly Inland Steel Coke Plant outfall 012 to the Indiana Harbor in Indiana (operational)
- U.S. Steel coke Plant (in Lorain) outfall 002 to the Black River in Ohio (shut down)
- Youngstown Sheet and Tube Campbell Works Coke Plant outfall to the Mahoning River in Ohio (shut down)
- LTV (Republic) Steel (in Youngstown) Coke Plant outfall to the Mahoning River in Ohio (shut down)
- LTV (Republic) Steel (in Cleveland) Coke Plant outfall to the Cuyahoga River (operational)
- Bethlehem Steel (in Buffalo) outfalls to Rush Creek and the Lackawana Ship Canal (shut down)
- Donner-Coke (in Buffalo) outfall to Buffalo River (shut down).

Although mean PAH sediment concentrations at sampling sites in the vicinity of coke plant outfalls are generally greater than at other sites, there were numerous sites not identified as being in the vicinity of coke oven outfalls which had mean PAH sediment concentrations exceeding 5 mg/kg. Such sites included the following:

- Numerous sites in the Grand Calumet River in the vicinity of blast furnace, sintering plant, bar mill plate, basic oxygen process furnace, miscellaneous finishing operation and other non-coke plant steel industry outfalls

- Several sites in the Indiana Harbor Canal in the vicinity of steel industry blast furnace or flat rolling operation outfalls

- Several sites in the vicinity of Petroleum handling and storage facilities including ones in the Grand Calumet River, Indiana Harbor Canal, and Detroit River

- Several sites in the vicinity of storm or combined sewer outfalls or overflows including ones in the Indiana Harbor Canal, Buffalo River and Tonawanda Canal (Niagara River)

- Several sites in the vicinity of POTW outfalls including ones in the vicinity of the Gary and East Chigaco POTW outfalls to the Grand Calumet River and the Ashland POTW outfall to Lake Superior

- Sites in the vicinity of waste disposal sites and dredged sediment sites including one adjacent to a dredged disposal site along the Black River in Lorain and two sites in Lake Erie off a Bethlehem Steel waste disposal site and a dredged sediment disposal site in Buffalo

- Sites in the vicinity of miscellaneous outfalls including the Blau-Knox Foundry, American Steel Foundry, and Union Carbide (unidentified) outfalls to the Indiana Harbor Canal, the Vulcan (Secondary Tin) outfall to the Grand Calumet River, the Williams Company (unidentified) outfall to the Cuyahoga River, the Union Carbide-Linde Division (Metal Fabrication and Finishing of Cryogenic Hardware) outfall to Two Mile Creek in Buffalo and the Great Lake Chemical Corp. outfall to Manistee Harbor (Lake Michigan)

- Numerous sites at which no outfalls were identified including 6 in the Milwaukee region, 2 in the Ashtabula subregion of the Ohio region, 3 in the Dunkirk/Erie subregion of the NY/PA Harbors region, 3 in the Scajaquada Creek subregion of the Buffalo region, 1 in the Two Mile Creek subregion of the Buffalo region and 4 in the St. Joseph Harbor subregion of the Michigan region.

Based on the PAH effluent data, elevated (but probably much lower than at coke oven) PAH sediment concentrations may possibly also occur at or below outfalls from the following industrial categories and/or subcategories:

- Wood Preserving facilities using creosote as a preservative

- Oily wastewater outfalls from Metal Finishing facilities

- Aluminum Forming facilities

- o Aluminum Casting Subcategory of the Foundry Industry and possible other subcategories not yet examined
- o Secondary Copper and Primary Aluminum (particularly those using Soderberg electrode refining) Subcategories of the Non-Ferrous Metals Industry and possibly other subcategories not yet examined
- o Petroleum Refining facilities involved in coke and coke by-product production and in the production of other high boiling fraction petroleum products.

The mean PAH sediment concentrations at sites in the open Great Lakes or in harbor areas of the Great Lakes not recieving direct industrial discharges are generally less than 2 mg/kg, even in areas where the upstream tributaries are heavily polluted with PAHs. Because relatively "clean" sediment samples were often collected in harbor or lake sites downstream of areas of heavily PAH contaminated sediments, it is observed that PAHs settle out and deposit within a short distance of outfalls. Examples of this phenomenon include the following:

- o Three of the four reported sites in the Indiana Harbor
- o Both reported sites in the Black River/Lorain Harbor
- o One of 2 reported sites in Lake Michigan off the Milwaukee Estuary
- o Both reported sites in Ashtabula Harbor
- o All 3 reported sites in Toledo Harbor
- o All 4 reported sites in the Conneaut Harbor
- o Both reported sites in the Fairpoint Harbor
- o 7 reported sites in Buffalo Harbor away from the contaminated Lackawana and Union Ship Canals
- o Numerous sites in Lake Erie off Buffalo
- o 3 of 4 reported sites in Lake St. Clair
- o All 5 reported sites in Lake Erie off the mouth of the Raisin River
- o All 12 reported sites in the Open Lake Michigan region, and
- o 5 of 6 reported sites in Lake Superior and Lake Superior harbors.

PAH sediment concentrations appear to generally decrease rapidly with longitudinal distance from a polluting outfall. Because PAHs generally parti-

tion into the sediment and suspended solids to a far greater extent than the water column, they are generally found in elevated levels in sediments at or just below polluting outfalls and in downstream areas of sediment deposition. However, in the Black River at Lorain, concentrations of PAHs in sediments at different transverse locations, but at the same longitudinal distance downstream from the coke oven outfall, varied greatly, apparently due to differences in sediment deposition rates.

In addition to those sites associated with steel industry outfalls, there are a number of relatively high ranking sites within the Buffalo region which appear to be associated with other types of pollution sources. For example, the highest ranking site in the Two Mile Creek subregion is in the vicinity of the Union Carbide Corp., Linde Div. Two of the highest ranking sites in the Lower Niagara River/Black Rock Canal subregion are located south of a waste disposal site on Squaw Island. Several of the 6 highest ranking sites in the Buffalo River subregion are at or just below the Buffalo Color outfall. The 2 highest ranking sites in the Tonawanda Canal subregion are in the vicinity of storm sewer overflows. The 4 highest ranking sites in the Lake Erie subregion are offshore of a Bethlehem Steel waste disposal site and a dredged sediment disposal site. However, while sources other than the steel industry may contribute to PAH pollution, it should be pointed out that none of the mean PAH sediment concentrations for any of the above sites are nearly as high as those for sites in the Indiana, Ohio Steel and Ohio regions associated with steel coke oven outfalls. Further, the highest mean PAH concentration in the Buffalo region was associated with 2 Bethlehem Steel outfalls to Rush Creek.

A comparison of steel industry PAH loading rates versus non-steel industry rates in the Buffalo region was provided in an NRTC report. In this report, the NRTC estimated PAH loadings to the Niagara River system from a variety of point sources based on mean wastewater discharge flows and PAH concentrations in effluents. Of an estimated mean PAH loading of 17.4 kg/day to the Niagara River System in 1981, the following industries were estimated to contribute the daily PAH loadings indicated in parenthesis: Donner-Coke (14.9 kg/day), Bethlehem Steel (1.1 kg/day), Buffalo Sewer Authority WWTP (0.7 kg/day), Union Carbide Linde Div. (0.4 kg/day), and 0.3 kg/day combined total from 3 separate WWTP's. Therefore, despite the relatively high PAH concentrations in sediment at and just downstream from the Buffalo Color outfall (see

preceding paragraph), Buffalo Color is not estimated to contribute to PAH loading based on PAH effluent data. The PAH concentrations in the sediments at and just downstream of the Buffalo Color outfall may be due to some upstream mixing and dispersion from the Donner-Coke outfall. Both Donner-Coke and Bethlehem Steel ceased operations in 1982 and 1983, respectively.

7.5.2 Recommendations with Respect to the Sampling and Analysis of Sediments for PAHs in Region V Of USEPA

The results of the 1984 ERG study of Black River (Ohio Steel region) sediments indicate that care must be taken in sampling sediments for PAH contamination. PAH concentrations in sediments in the ERG study not only decreased rapidly with longitudinal distance from U.S. Steel outfall 002, but also varied widely between different transverse locations at the same longitudinal distances downstream. Therefore, based on the results of the ERG study, it appears that sediment samples should be taken in the immediate vicinity of the outfalls of suspected PAH dischargers and at the first major sediment deposition area downstream. Also, samples should be taken at different transverse locations at the same longitudinal site and either analyzed separately or composited before analysis.

PAH sediment concentrations for various PAHs at nine sampling sites in the Milwaukee region are actually means of PAH concentrations for various depth fractions of the cores that were taken. PAH concentrations varied with depth. Some maximum PAH concentrations occured in the 30-60 cm and 60-90 cm core fractions as well as in the surface 0-30 cm core fractions. Although the occurrence of maximum PAH concentrations in some sub-surface core fractions may be due in part to some downward PAH transport and/or greater rates of degradation and dissolution near the surface, it may also be due to the more recent deposition of lesser PAH-contaminated sediment overlying the older, greater PAH-contaminated sediment.

The variability of PAH concentrations with depth indicates the importance of specifying core depth and core fraction when reporting PAH concentration in sediments. PAH concentrations in the surface core fractions (e.g., 0-30 cm) are probably more indicative of risks to benthic organisms and humans than PAH concentrations in the lower core fractions, or PAH concentrations averaged over all core fractions. However, a knowledge of the PAH concentrations as a

fraction of depth in the sediment, in addition to a knowledge of PAH concentrations as a function of time, may be of value in determining trends in the PAH contamination of the sediment.

Recommendations with respect to the selection of sediment sampling sites are provided in the following discussions.

7.5.2.1 Operating Coke Plants

The highest mean PAH sediment concentrations (e.g., >10 mg/kg) and associated risk scores are generally at or just below coke oven outfalls (associated with steel manufacturing and with some petroleum refineries). Therefore, to determine maximum PAH sediment concentrations, sediment samples should be taken for PAH analysis at and just below operational coke plant outfalls. Twenty operational and closed steel industry coke plants are listed in USEPA Region V.

Since it is probably not practical to sample sediments at and below all of the operational coke plant outfalls in Region V, priority should be given to sampling sites in the vicinity of those coke plants with the greatest mean wastewater volumes discharged per day or coke plants with the largest production volumes.

Historically, one of the greatest sources of PAHs to sediments in Region V have been the U.S. Steel coke Plant outfalls to the Grand Calumet River. Although there is some data on PAHs in sediments in the vicinity of the U.S. Steel coke plant outfalls to the Grand Calumet River, most of it is for samples taken in 1972, which were only analyzed for benzo[a]pyrene, anthracene, and phenanthrene. Therefore, sediments at and below the still operational U.S. Steel coke plant outfalls should be resampled and analyzed for a wider range of PAHs. Additional samples of sediments at and below the LTV (Republic) Steel outfall to the Cuyahoga River should also be taken since the mean PAH concentration at the one reported site in that area (CU 81-18) is extremely high (75.7 mg/kg).

7.5.2.2 Non-Operating Coke Plants

To determine the rate at which PAH levels will decrease in sediments no longer exposed to large PAH loadings, it would be valuable to sample sediments

at and below one or more coke plants which have shut down and for which a substantial amount of historical PAH data exists. A good study candidate would be the U.S. Steel coke plant outfall 002 to the Black River at Lorain, since there do not appear to be any major PAH sources within several miles upstream of the outfall.

7.5.2.3 Non-Coke Plant Steel Industry

Much of the PAH sediment concentration data for the Grand Calumet River and the Indiana Harbor Canal indicates that elevated levels of PAHs in sediment (but lower than those at coke oven outfalls) may occur at and below various non-coke plant steel industry outfalls. PAH concentrations in effluents from blast furnaces and sintering plant processes indicate that these outfalls may contribute significantly to PAH loadings in some cases. Therefore, to determine whether various other types of steel industry outfalls contribute significantly to sediment contamination, samples should be collected from sites at or below various types of non-coke plant steel industry outfalls, but located far enough upstream of any coke plant outfalls to insure that no contamination from the coke plant outfalls occur.

7.5.2.4 Other (Non-Steel) Industries

Elevated mean PAH concentrations (e.g. >5 mg/kg), but generally lower than at or below coke plant outfalls, have been observed in sediments at and below various types of outfalls other than those from steel industry facilities including outfalls from petroleum handling and storage facilities, storm and combined sewers, POTWs, a couple of foundries, a secondary tin manufacturer, a cryogenic hardware fabricating and finishing facility, and a chemical company. In addition, elevated levels of PAHs are present at many sites at which no outfalls are identified, and have also been observed in sediments adjacent to dredged sediment disposal sites and a steel industry waste disposal site. Data on the concentrations of PAHs in industrial wastewater suggests that elevated levels of PAHs may also sometimes occur in sediments at or below outfalls from wood preserving facilities which use creosote, metal finishing facilities, aluminum forming facilities, the Aluminum Casting Subcategory of the Foundry Industry, the Primary Aluminum and Secondary Copper Subcategories of the Non-Ferrous Metals Industry, and petroleum refineries.

7.5.2.5 Confirmation Sampling

There is evidence that elevated levels of PAHs can occur in sediments in industrialized and heavily developed areas that do not have coke plants. Areas in USEPA Region V which are heavily industrialized and also have operating coke facilities include the Grand Calumet River/Indiana Harbor Canal/ Indiana Harbor, Chicago, Cleveland, Indianapolis, Toledo, and Detroit. Although a substantial amount of data is available for the Grand Calumet River/ Indiana Harbor Canal area, much of it is for samples taken in the 1970's. Therefore, most of that area (which exhibited the highest PAH risk scores in Region V) should be resampled. No PAH data were found for either Chicago or Indianapolis and only limited data were found for Cleveland (in the Cuyahoga River) and for Toledo.

7.5.2.6 Sampling Locations

PAH sediment sampling should carefully consider both sampling location and depth. Because a large proportion of PAHs discharged to the aquatic environment are sorbed to suspend soilds (which rapidly settle out), or are rapidly absorbed from solution by sediments upon discharge, a large amount of PAHs settle out in the immediate vicinity of the outfall and at the first major depositional area downstream of the outfall. Therefore, sediment samples should be taken immediately below the outfall, midway between the outfall and the first major sediment deposition area downstream of the outfall, and in the first major depositional area downstream of the outfall.

A study of sediments downstream of the USS Steel Coke oven outfall 002 to the Black River in Lorain indicated that PAH concentrations in sediment samples taken at different transverse locations at the same longitudinal distance downstream from an outfall may vary widely. Therefore, samples at several different transverse locations for a given longitudinal distance downstream should be taken and composited before analysis if mean PAH concentrations in sediments at that distance downstream are desired.

A Study in the Milwaukee region indicated that maximum PAH sediment concentrations sometimes occur in core fractions beneath the surface core fraction. This may be especially true in regions with high sediment deposition rates and where levels of PAH loadings to the aquatic system have decreased in

recent years. If mean PAH sediment concentrations over the entire depth of the contaminated sediment zone are desired, cores must be taken to a depth at which no significant PAH contamination is observed. In the Milwaukee study, that would have required taking core samples well in excess of 100 cm at all of the sites. However, for most purposes, sampling should be conducted to depths of no greater than 30 cm since the greatest interaction between the water column and benthic organisms with sediments probably occur in the upper 30 cm or 1 foot of sediment.

7.5.2.7 Sample Analysis

Although the higher molecular weight and number of rings PAHs appear to generally be more toxic to humans, the lower molecular weight and number of rings PAHs appear to be at least more acutely toxic to aquatic organisms. Therefore, sediments should ideally be analyzed for representatives of 2, 3, 4, 5, and at least 6 ring PAHs. In most cases, an analysis of sediments for the PAHs listed in Table 1-1 should be sufficient to characterize PAH contamination in general unless other PAHs are known or suspected to be at high levels due to the nature of the sources involved. Methylated derivatives of some but not all of the PAHs listed in Table 1-1 appear to be more toxic and therefore might be included in some analyses. In addition, 7,12-dimethyldibenzo[a,h]anthracene and methycholanthrene (which along with benzo[a]anthracene, benzo[a]pyrene, and dibenzo[a,h]anthracene have been conclusively shown to cause malignant tumor formation in laboratory animals) should probably be included in the analyses.

7.5.3 Recommendations with Respect to Risk Formulae and the Sampling of Other Media

Because sediments are the primary reservoir for PAHs in the aquatic environment, measurements of PAH concentrations in sediment samples provide an estimation of the extent to which an aquatic system has received PAH inputs. However, in estimating the risks to human and aquatic life, determinations of PAH concentrations in edible aquatic organisms, drinking water and in the water column are of much greater value.

7.5.3.1 Human Carcinogenic Risk

An approach for estimating the carcinogenic risk to humans associated with the concentration of a given PAH in sediment at a given sampling site was presented in Section 7.3.1. That approach is based on the assumptions listed in Section 7.3.1 and on the additional assumption that the aquatic organisms consumed all remain in the immediate vicinity of the given site long enough to reach steady state and are then caught in that general area. None of the assumptions listed in Section 7.3 (except possibly assuming that the PAH contribution of drinking water is negligible compared to that of the edible aquatic organisms), nor the additional assumption discussed above are realistic for computing carcinogenic risk. Therefore, the approach discussed in Section 7.3.1 should only be used for computing relative site rankings and not for computing risks. In addition, this equation should only be used in the absence of actual levels of PAHs in edible aquatic organisms caught from the water body of concern.

Equations are available to compute the total lifetime carcinogenic risk to an individual person due to the consumption of contaminated aquatic organisms and drinking water from a given water body. However, since PAHs have low aqueous solubilities, are susceptible to degradation by chlorination and tend to partition to a much greater extent into suspended solids (which are generally efficiently removed by water treatment facilites) than in water, the contribution of drinking water to the overall PAH input to an individual is probably generally negligible compared to that of PAH contaminated fish. Therefore, if an estimate of carcinogenic risks associated with the PAH contamination of a given water body is desired, priority should be given to the determination of carcinogenic PAH levels in fish and shellfish caught from the given water body.

If reasonable estimates of total human carcinogenic risks are to be made, carcinogenic PAHs in addition to those listed in Table 1-1 should be included in the analysis of fish and shellfish tissues. At the very least, the 5 PAHs which have been conclusively shown to induce malignant tumors in test animals after oral administration should be included in the analysis. Three of those 5 PAHs (benzo[a]pyrene, benzo[a]anthracene and dibenzo[a,h]anthracene) are not listed in Table 1-1 but should also be included in any analysis of fish tissue or drinking water. Finally, it should be again noted that benzo[a]pyrene is

the only PAH for which a unit carcinogenic risk factor (UCR) has been determined. Therefore, to use the approach discussed in Section 7.3.1 to compute carcinogenic risks, UCRs for at least the carcinogenic PAHs in Table 1-1, as well as 7, 12-dimethylbenzo[a]anthracene and methylcholanthrene should be determined.

7.5.3.2 Human Non-Carcinogenic Risk

An approach for estimating the non-carcinogenic risk to humans associated with the concentration of a given PAH in sediment at a given sampling site was discussed in Section 7.3.2. However, this approach is based on the same assumptions used in the human carcinogenic risk approach (Section 7.3.1). Therefore, this approach, like the preceeding, should only be used for computing relative site ranking and not for actual risk computations. In addition, it should only be used in the absence of data on the concentrations of PAHs in edible aquatic organisms caught from the water body of concern.

Equations are available to compute the non-carcinogenic risk (which is not really a risk but a ratio of estimated dose to the ADI) to an individual due to the consumption of a given non-carcinogenic PAH in contaminated fish and drinking water from a given water body. Unlike carcinogenic risks, non-carcinogenic risks (ratios) are generally not summed on different PAHs because the effects of different PAHs are generally different. However, non-carcinogenic risks (ratios) are sometimes summed over different PAHs to give a "hazard index". It should be again noted that ADIs have only been determined for fluoranthrene and naphthalene and need to be determined for a number of other PAHs before more extensive non-carcinogenic risk computations can be performed.

7.5.3.3 Acute Aquatic Toxicity Risk

An approach for calculating acute aquatic toxicity risks associated with the concentration of a given PAH in sediment at a given sampling site was discussed in Section 7.3.3. However, some of the same assumptions used in deriving equations for human carcinogenic and non-carcinogenic risks were also used in deriving the acute aquatic toxicity risk approach. Therefore, this approach, like the preceeding two, should only be used for computing relative site rankings and not for actual risk computations. In addition, it should

only be used in the absence of data on the concentrations of PAHs in the water column.

Equations are available to compute the acute or chronic risks (which is not really a risk but a ratio of PAH concentration in water to acute or chronic water quality criteria) to aquatic organisms due to the mean concentration of a given PAH in the water column. Acute and chronic aquatic life risks (ratios) are generally like non-carcinogenic risks (ratios), but are not summed over different PAHs because different PAHs exert different effects on the same and different organisms. However, just like human non-carcinogenic risks, acute and chronic aquatic life risks can be summed over different PAHs to give a "hazard index".

Computation of acute or chronic risks to aquatic life requires not only data on the concentrations of PAHs in the water column but also the determination of acute and chronic water quality criteria for various PAHs for the protection of aquatic life. Unfortunatly, as previously stated, no acute or chronic water quality criteria have as yet been established for any of the PAHs. This needs to be done before any reasonable aquatic life risk estimates can be computed.

The risk assessment, scoring and comparative ranking procedures used in this report are based on a number of assumptions, however this procedure represents the most reliable approach currently applicable given the general lack of toxicological information and site-specific physico-chemical data. It is hoped that expanding and continued use of this approach will result in laboratory and field research efforts focused on refining the current approach, particularly as applies to narrowing the "error bars" on certain of the assumptions used. Given the cost and complexity of toxic waste remedial investigations and subsequent clean-up programs, it is essential that reliable, quantifiable risk management procedures be developed. This is especially critical in light of the delays inherent in stabilizing or reversing incidents of environmental contamination, and the continued human exposure to these contaminants resulting from such delays.

REFERENCES

ACOE. 1985. U.S. Army Corps of Engineers. Result of the analysis of a 1985 composite sample of Indiana Harbor sediment. Provided to SAIC by Jan Miller of the Detroit District of the U.S. Army Corps of Engineers.

Amendola GA. 1985. PAHs in River Sediments, Memorandum of March 16, 1985 to Howard Zar of Region V Office, U.S. Environmental Protection Agency, Chicago, IL.

ATEC. 1983. Aqua Tech Environmental Consultants, Inc.. Analysis of Sediment from Lorain Harbor. Prepared for the Buffalo District of the U.S. Army Corps of Engineers. Technical Report #G0128-01.

Bauman PC, Smith WD, Rubick M. 1982. Hepatic tumor rates and polynuclear aromatic hudrocarbon levels in two populations of brown bullhead (Icataburis Nebulosis). In: Polynuclear Aromatic Hydrocarbons: 6th International Symposium on Physical and Biological Chemistry (Cooke MW, Dennis AJ, Fisher GL, eds.). Battelle Press, Columbus, Ohio. pp. 93-102.

Black JJ. 1983. Field and laboratory studies of environmental carcinogensis in Niagara River fish. J. Great Lakes Res. 9:326-334.

Black J, Fox H, Black P, Bock F. 1985. Carcinogenic effects of river sediments. Preprint sent to Proc. 5th Water Chlorination Conference.

Clements Associates. 1985. Chemical, physical, and biological properties of compounds present at hazardous waste sites. Prepared for U.S. Environmental Protection Agency.

Eadie BJ, Faust W, Gardner WS, Nalipa T. 1981. Polycyclic hydrocarbons in sediments and associated benthos in Lake Erie. Chemosphere 11(2):185-191.

Eadie BJ, Landrum PF, Faust W. 1982. Polycyclic aromatic hydrocarbons in sediments, pore water, and the amphipod Pontoporeia hoyi from Lake Michigan. Chemosphere 11(9):847-858.

Eadie BJ. 1984. Distribution of polycyclic aromatic hydrocarbons in the great Lakes. In: Toxic Contaminants in the Great Lakes (Nriagu JO, Simmons MS, eds.). John Wiley and Sons, Inc. pp. 195-211

ERG. 1984. Environmental Research Group, Sampling methods for sediment collection. Black River, Lorraine, Ohio. Prepared for United States Steel Corporation, Lorraine, Ohio.

Grice HD, Clegg DJ, Coffin DE, Lo M, Middleton EJ, Sandi E, Scott PM, Sen NP, Smith BL, Withey JR. Carcinogens in food. In: Carcinogens in Industry and the Environment (Sontag JM, ed.). Marcel Decker, Inc. pp. 439-518.

HydroQual. 1984. Grand Calumet River wasteload allocation study. Prepared for Indiana State Board of Health, Indianapolis, Indiana. Project Number 1SBH0010.

Lavoie EJ, Hecht SS. 1981. Chemical carcinogens in vitro metabolism and activation. In: Hazard Assessment of Chemicals. Current Developments. Volume I (Saxena J, Fisher F, eds.). Academic Press. pp. 156-233.

LTI. 1984. Lemno-Tech, Inc. Field methodology and results for Indiana Harbor, Indiana. Prepared for the Detroit District of the U.S. Army Corps of Engineers.

Lyman WJ, Reehl WF, Rosenblatt DH (eds.). 1982. Handbook of Chemical Property Estimation Methods. Environmental Behavior of Organic Compounds. McGraw-Hill Book Company.

Neff JM. 1979. Polycyclic Aromatic Hydrocarbons in the Aquatic Environment. Sources, Fates, and Biological Effects. Applied Science Publishers Ltd., London.

Neff JM. 1985. Polycyclic aromatic hydrocarbons. In: Fundamentals of Aquatic Toxicology (Rand GM, Petrocelli SR, eds.). Hemisphere Publishing Corporation. pp 416-454.

NRTC. 1984. Niagara River Toxics Committee. Report of the Niagara River Toxics Committee.

SAIC. 1984. Science Applications International Corp. Report on existing standards, toxicity data and mobility data for PAHs. Prepared for the Office of Solid Waste, U.S. Environmental Protection Agency, Washington, DC.

Salee MA. 1984. Revised health and environmental effects data for Appendix VII constituents. Memorandum to Kenneth Schuster, Chief Land Disposal Branch, Office of Solid Waste and Emergency Response, U.S. Environmental Protection Agency, Washington, DC.

Smith JH, Maliey WR, Bohonos N, Holt BR, Lee SS, Chou TW, Bomberger DC, Mill T. Environmental pathways of selected chemicals in freshwater systems. Part II. Laboratory Studies. U.S. Environmental Protection Agency, Athens, G.A. EPA-600/7-78-074.

Southworth GR. 1979. The role of volatilization in removing polycyclic hydrocarbons from aquatic environments. Bull. Environ. Contamin. Toxicol. 21:507-14.

STORET. 1985. The STORET data base.

USEPA. 1974. U.S. Environmental Protection Agency. Brass HJ, Elbert WC, Fuge MA, Glick EM, Lington AW. United River Survey: Analysis for hexane organic extractables and polynuclear aromatic hydrocarbons. Office of Enforcement and General Counsel, U.S. Environmental Protection Agency, Cinncinnati, Ohio.

USEPA. 1977. U.S. Environmental Protection Agency. Amendola GA, Schregardus DR, Harris WH, Moloney ME. Mahoning River Waste Load Allocation Study. Prepared for the Ohio Environmental Protection Agency. Eastern District Office, Region V, U.S. Environmental Protection Agency, Fairview Park, Ohio.

USEPA. 1979. U.S. Environmental Protection Agency. Water-related environment fate of 129 priority pollutants. Volume II. Office of Water Planning and Standards, Office of Water and Waste Management, U.S. Environmental Protection Agency, Washington, DC. EPA 440/4-79-029b.

USEPA. 1980a. U.S. Environmental Protection Agency. Ambient water quality cirteria for polunuclear aromatic hydrocarbons. Office of Water Regulations and Standards, Criteria and Standards Division, U.S. Environmental Protection Agency, Washington, DC. EPA 440/5-80-069.

USEPA. 1980b. U.S. Environmental Protection Agency. Ambient water quality criteria for acenaphthalene. Office of Water Regulations and Standards, Criteria and Standards Division, U.S. Environmental Protection Agency, Washington, DC. EPA 440/5-80-015.

USEPA. 1980c. U.S. Environmental Protection Agency. Ambient water quality criteria for fluoranthene. Office of Water Regulations and Standards Criteria and Standards Division, U.S. Environmental Protection Agency, Washington, DC. EPA 440/5-80-049.

USEPA. 1980d. U.S. Environmental Protection Agency. Ambient water quality criteria for naphthalene. Office of Water Regulations and Standards, Criteria and Standards Division, U.S. Environmental Protection Agency, Washington, DC. EPA 440/5-80-059.

USEPA. 1980e. U.S. Environmental Protection Agency. Treatability manual. Volume I. Treatability data. Office of Research and Development, U.S. Environmental Protection Agency, Washington, DC. EPA 600/8-80-042a.

USEPA. 1980f. U.S. Environmental Protection Agency. Treatability manual. Volume II. Industrial desriptions. Office of Research and Development, U.S. Environmental Protection Agency, Washington, DC. EPA 600/8-80-042b.

USEPA. 1981a. U.S. Environmental Protection Agency. Aquatic fate data for organic priority pollutants. Office of Water Regulations and Standards, U.S. Environmental Protection Agency, Washington, DC. EPA 440/4-81-014.

USEPA. 1981b. U.S. Environmental Protection Agency. Development document for effluent limitations guidelines and standards for the Timber Products Point Source Category. Final effluent guidelines. Effluent Guidelines Division, U.S. Environmental Protection Agency, Washington, DC. EPA 440/1-81-023.

USEPA. 1981c. U.S. Environmental Protection Agency. Industrial residuals manual. Volume II. Overview of categorical industries. Office of Water Enforcement and Permits, Washington, DC.

USEPA. 1982. U.S. Environmental Protection Agency. Environmental-regulatory review of Grand Calumet River and Indiana Harbor Canal. Great Lakes National Program Office, U.S. Environmental Protection Agency, Chicago, IL.

USEPA. 1983a. U.S. Environmental Protection Agency. Revision to Treatability manual. Volume II. Industrial descriptions. Office of Research and Development, U.S. Environmental Protection Agency, Washington, DC. EPA 600/2-82-001b.

USEPA. 1983b. U.S. Environmental Protection Agency. Development document for effluent limitations guidelines and standards for the Non-Ferrous Metals Point Source Category. Volume II. Effluent Guidelines Division, U.S. Environmental Protection Agency, Washington, DC. EPA 440/1-83-019b.

USEPA. 1984a. U.S. Environmental Protection Agency. Great Lakes National Program Office. Harbor Sediment Program. Lake Superior 1981: Ashland, Wisconsin; Black River, Michigan; L'Anse, Michigan. Great Lakes National Program Office, U.S. Environmental Protection Agency, Chicago, IL. EPA 905/4-84-003.

USEPA. 1984b. U.S. Environmental Protection Agency, 1981 Buffalo New York Area Sediment Survey (BASS). Great Lakes National Program Office, U.S. Environmental Protection Agency, Chigaco, IL. EPA 905/3-84-001.

USEPA. 1985a. U.S. Environmental Protection Agency. Data on PAH concentrations in sediment samples taken from the Detroit River and its tributaries. From a draft report being prepared by the Great Lakes National Program Office, U.S. Environmental Protection Agency, Chicago, IL.

USEPA. 1985b. U.S. Environmental Protection Agency. Data on PAH concentrations in sediment samples taken from the Milwaukee Estuary. From a draft report being prepared by the Great Lakes Natural Program Office, U.S. Environmental Protection Agency, Chicago, IL.

USEPA. 1985c. U.S. Environmental Protection Agency. Investigation of polycyclic aromatic hydrocarbon discharges to water in the vicinity of Buffalo, New York, Great Lakes National Program Office, U.S. Environmental Protection Agency, Chicago, IL. EPA 905/4-85-002.

Woo YT, Arcos JC. 1981. Environmental chemicals. In: Carcinogens in Industry and the Environment (Sontag JM, ed.). Marcel Dekker, Inc. pp. 167-282.