SUMMARY

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

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

November 1987

EPA 905-S-87-001

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.

TABLE OF CONTENTS

TABLE OF CONTENTS

LIST OF TABLES

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.

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.

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. 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_{OW}) 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. 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.

Table 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. 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).

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.

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.

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 benzo[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_{OW}). 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_{ow} 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 less than PAH concentrations in interstitial water or water near the sediments.

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 affects 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 rin_- 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.

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 are 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 were 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.

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)

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

- o 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)

- o 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)

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

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

- o 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)

- o 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.

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 (i.e.; PAH contribution from drinking water sources is comparatively insignificant)
- 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.

One additional assumption in this approach for estimating the carcinogenic risk to humans is 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 preceding assumptions (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 completely realistic for computing carcinogenic risk. Therefore, this approach should only be used for computing relative site rankings and not for computing true risks. In addition, this equation should only be used in the absence of data on actual levels of PAHs in edible aquatic organisms caught from the water body of concern.

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.

This approach for estimating the non-carcinogenic risk to humans is based on the same assumptions used in the human carcinogenic risk approach (Section 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.

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.

Because some of the same assumptions used in deriving equations for human carcinogenic and non-carcinogenic risks were also used in deriving acute aquatic toxicity risk scores, 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.

Human carcinogenic, human non-carcinogenic, and acute aquatic toxicity risk scores are summarized in Table 2. 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 2). In Table 2, only mean risk scores are presented.

Table 2 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 2 are relative to only the other subregions within the same region.

Regional and subregional rankings provided in Table 2 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.

Table 2. 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 2. 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 2. 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 2. 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.

Finally, an overall ranking score is provided in the extreme right hand column in Table 2. 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 2, 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 2 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).

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 2.

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 2, they were integral to computation of the mean scores reported in this table and are considered in the following discussions.

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 petrochemicals industry processes located along the East Branch of the Grand Calumet River, the Indiana Harbor Canal, and Indiana Harbor.

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.

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 second 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 (926 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.

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 overall 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.

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.

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 out-

fall (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.

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.

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.

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.

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.

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.

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.

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.

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.

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).

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.

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.

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.

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).

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.

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 avail-

able. Furthermore, none of the currently-recognized, potentially significant point and non-point pollution sources for the Niagara River System are located along Scajaquada Creek.

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.

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.

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.

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.

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.

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.

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.

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).

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.

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.

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.

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.

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).

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).

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.

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.

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.

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.

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 (9.1×10^{-3}) and overall ranking score (9 + 9 + 9 + 8 = 26). It ranks eighth with respect to mean acute aquatic toxicity risk score.

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.

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.

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.

5. CONCLUSIONS AND RECOMMENDATIONS

Topics addressed in this portion of the Executive Summary include the following: Conclusions with Respect to Sources of PAH Contamination in Sediments (5.1), Recommendations with Respect to the Sampling and Analysis of Sediments for PAHs (5.2), and Recommendations with Respect to Risk Formulae and the Sampling of Other Media (5.3).

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)

- o LTV (Republic) Steel (in Youngstown) Coke Plant outfall to the Mahoning River in Ohio (shut down)
- o LTV (Republic) Steel (in Cleveland) Coke Plant outfall to the Cuyahoga River (operational)
- o Bethlehem Steel (in Buffalo) outfalls to Rush Creek and the Lackawana Ship Canal (shut down)
- o 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:

- o 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
- o Several sites in the Indiana Harbor Canal in the vicinity of steel industry blast furnace or flat rolling operation outfalls
- o Several sites in the vicinity of Petroleum handling and storage facilities including ones in the Grand Calumet River, Indiana Harbor Canal, and Detroit River
- o 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)
- o 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
- o 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
- o 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
- Aluminum Casting Subcategory of the Foundry Industry and possible other subcategories not yet examined
- 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
- 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:

- Three of the four reported sites in the Indiana Harbor
- Both reported sites in the Black River/Lorain Harbor
- One of 2 reported sites in Lake ...chigan 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 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. Because PAHs generally partition 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.

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.

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).

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.

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.

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 manufactur-

er, 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.

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.

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.

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 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 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.

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.

5.3.1 Human Carcinogenic Risk

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 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 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 3.1 to compute carcinogenic risks, UCRs for at least the carcinogenic PAHs in Table 1, as well as 7, 12-dimethylbenzo[a]anthracene and methylcholanthrene should be determined.

5.3.2 Human Non-Carcinogenic Risk

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.

5.3.3 Acute Aquatic Toxicity Risk

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.