a patch test (Mylanvarapu and Sun, 1991). To protect the most sensitive individuals, a cleanup level to 5  $\mu$ g/m<sup>2</sup> on surfaces would have to be achieved (Symms, 1991).

Based on exposure via inhalation, the EPA has classified inhaled chromium (VI) in Group A - human carcinogen (EPA, 1992a). Chronic exposure to chromium-bearing dusts via inhalation has been associated with lung cancer in occupationally exposed workers in a number of studies (ATSDR, 1989b; 1992b). Exposure data have not been sufficient to clearly establish the form(s) of chromium responsible for the increases in lung cancer (ATSDR, 1989b; 1992b). However, it has been generally accepted that chromium (VI) compounds are likely to be the key etiologic agents. This is consistent with the findings that in vitro chromium (VI) compounds can enter cells readily, while chromium (III) compounds are largely excluded (ATSDR, 1989b; 1992b). Chromium (VI) is effective at low concentrations in induction of chromosome aberrations and sister chromatid exchanges (SCEs), gene mutation, and cell transformation (reviewed in Bianchi and Levi, 1985). A few studies have measured increases in chromosome aberrations and SCEs in the peripheral lymphocytes of workers exposed to soluble chromium (VI) compounds (reviewed in ATSDR, 1989b; 1992b).

The EPA has concluded that there is inadequate evidence that chromium compounds are carcinogenic via ingestion (EPA, 1992b). Chronic oral exposure to chromium (VI) compounds did not cause an increased tumor incidence in rats (Mackenzie et al., 1958). This result, combined with the lack of evidence for cancer following oral exposure in humans has led the EPA to the conclusion that chromium (VI) is not likely to be carcinogenic via this route.

1.4.2 Quantitative Description of Health Effects.

The EPA (1984b) based its quantitative risk assessment for inhaled hexavalent chromium on a study by Mancuso (1975). Mancuso's study showed excess risks of lung cancer in workers exposed to chromates between 1931 and 1937 and followed until 1974. Lung cancer risks increased with duration of exposure and with age. Estimates of cumulative exposure

to soluble, insoluble, and total chromium were derived from a single set of industrial hygiene measurements taken in 1949. Smoking habits of the workers were not determined or discussed. For lifetime exposure the "unit risk" was calculated to be  $1.2 \times 10^{-2} (\mu g/m^3)^{-1}$ . Expressed in terms of total intake via inhalation, the cancer potency factor was calculated as 41 (mg/kg-day)<sup>-1</sup> (EPA, 1984b, 1992a).

Confidence in the EPA's unit risk is attenuated by several factors. Although results of studies of chromium exposure are consistent across locations and investigators, and a dose-response relationship has been established, the Mancusco study based its exposure calculations on the assumption the ratio between chromium (III) and chromium (VI) was 6:1. This was the assumed minimum chromium (VI) content and could underestimate risks. The 1949 hygiene data may have underestimated actual exposures which could lead to overestimation of risk. Finally, the study's implicit assumption that smoking rates were similar in the worker and general populations may cause an overestimation of risk, since smoking rates are often higher among industrial workers (EPA, 1992a).

An RfC for inhalation of chromium (VI) has been established by EPA (1992a). The value is based on a human study (occupational) that established a LOAEL of 0.7  $\mu$ g/m<sup>3</sup> for production of atrophy in the nasal mucosa. An uncertainty factor of 300 was applied to convert the LOAEL to a NOAEL to protect sensitive subpopulations and to account for less-than-chronic exposures in the worker population studies. The resulting RfC is 2E-06 mg/m<sup>3</sup>. An RfD of 6E-07 mg/kg-day is generated by multiplying the RfC by the inhalation rate (20 m<sup>3</sup>/day) and dividing by body weight (70 kg).

The interim MCL for total chromium under the National Interim Primary Drinking Water Regulations is 0.05 mg/L. A proposed MCL of 0.12 mg/day is proposed for total chromium, based on an Adjusted Allowable Daily Intake (AADI) of 0.17 mg/L with exposure by other routes (0.10 mg/day via the diet and 0 mg/day via air) factored in (EPA, 1985p).

SUMMARY OF CHROMIUM CRITERIA		SOURCE
EPA Carcinogenic Classification (inhalation of Chromium VI only)	Group A	EPA, 1992a
Maximum Contaminant Level (MCL) (Total Chromium)	0.1 mg/L	EPA, 1992a
Maximum Contaminant Level Goal (MCLG) (Total Chromium)	0.1 mg/L	EPA, 1992a
Inhalation SF	42 (mg/kg-day) <sup>-1</sup>	RBCT, 1993*
Oral RfD (Chromium VI)	0.005 mg/kg-day	EPA, 1992a
Inhalation RfC (Chromium VI)	3.0E-04 µg/m <sup>3</sup>	EPA, 1992a
		· · ·
EPA Drinking Water Health Advisories (HA)		
Lifetime HA	0.1 mg/L	ЕРА, 1991ь
Long-term HA Child Adult	2 mg/L 0.8 mg/L	EPA, 1991b EPA, 1991b
Short-term HA Ten-day HA (Child) One-day HA (Child)	1 mg/L 1 mg/L	EPA, 1991b EPA, 1991b

\* Risk Based Concentration Table, First Quarter 1993.

1.4.3 Fate and Transport.

Most of the chromium in surface water may be present in particulate form as sediment. Some of the particulate chromium would remain as suspended matter and ultimately be deposited in sediments. The exact chemical forms of chromium in surface waters may be present as Cr(VI); a small amount may be present as Cr(III) organic complexes. Hexavalent chromium is the major stable form of chromium in seawater; however, Cr(VI) may be reduced to Cr(III) by organic matter present in water and may eventually deposit in sediments (HSDB, 1992).

Uptake is greater from ultrabasic soils by a factor of 5-40 than on calcarious or silica-based

soils (HSDB, 1992).

### 1.5 3,3'-Dichlorobenzidine.

The Human Health Assessment Group in EPA's Office of Health and Environmental Assessment has evaluated 3,3'-dichlorobenzidine for carcinogenicity. According to their analysis, the weight-of-evidence classifies 3,3'-dichlorobenzidine as a group B2. No human data is available. As a group B2 carcinogen, 3,3'-dichlorobenzidine is considered to be a probable human carcinogen. Dermal exposure to a 3,3'-dichlorobenzidine has caused dermatitis in dye workers (HSDB, 1993). The LD<sub>50</sub> value in rats is in the range of 5000 mg/kg (Patnaik, 1992).

There are no known cases in which 3,3'-dichlorobenzidine has been associated with the occurrence of cancer in humans. However, 3,3'-dichlorobenzidine may have contributed to cases of bladder cancer attributed to benzidine, as both substances may be prepared in the same facility (HSDB, 1993).

In one study, the health records of 59 workers at a dyestuff plant in Great Britain, who were exposed to 3,3'-dichlorobenzidine from 1953 to 1973, were examined and compared to those working with both benzidine and 3,3'-dichlorobenzidine, and to unexposed populations. It was calculated that the 3,3'-dichlorobenzidine process worker was actually exposed to 3,3'-dichlorobenzidine for a maximum of 10 hours per work week. Men whose total 3,3'-dichlorobenzidine exposure was less than 245 hours (six months of full-time work) were excluded from the study, leaving 35 segregated 3,3'-dichlorobenzidine exposure, had no urinary tract tumors, no other tumors, and two deaths from other causes (coronary thrombosis, cerebral hemorrhage). In contrast, among 14 mixed benzidine and 3,3'-dichlorobenzidine, 40% worked with 3,3'-dichlorobenzidine), three men developed tumors of the bladder, and one man developed a carcinoma of the bronchus. One death from coronary thrombosis was noted (HSDB, 1993).

Another study presented the results of a survey of the incidence and type of neoplasms which developed in men exposed to 3,3'-dichlorobenzidine while working in a manufacturing plant in the United States. The results included lung cancer (2 workers), leukemia bone marrow (1 worker), lipoma (6 workers), rectum papilloma (3 workers), sigmoid colon carcinoma (2 workers), prostate carcinoma (1 worker), breast muscle myoblastoma (1 worker), and skin basal cell epithelioma (1 worker). A total of 17 workers of the 207 workers surveyed had developed neoplasms. It was unclear from the HSDB reference whether the potential for exposure to benzidine existed (HSDB, 1993).

A urine cytology surveillance of occupational bladder tumor incidence was evaluated by reviewing the clinical history of 9 bladder tumor cases found in dyestuff plant workers. A bladder tumor surveillance system was organized for workers (179 active and 65 retired) who were exposed to benzidine or beta-naphthylamine in the plant. The urine cytology surveillance was found to be useful for two reasons: first, it detected tumors in five out of six cases surveyed by the system, and second, four cases screened by cytology had tumors curable by transurethral operation, while other cases underwent cystectomy. Calculated average latent periods from the first and last exposure was 26.4 years (SD=6.0) and 14.0 years (SD=6.2) (HSDB, 1993).

Further information (i.e., noncarcinogenic effects) was not available in HSDB or IRIS.

## 1.6 Polychlorinated biphenyls.

Hepatic, dermal, and ocular effects are relatively well-established in case studies of PCB exposure. There are also reports of respiratory, gastrointestinal, hematological, muscular and skeletal, developmental, and neurological effects related to PCB exposure, but the effects can not be positively attributed to PCBs.

Hepatic effects include an increase in serum levels of enzymes and cholesterol, hepatocellular damage, neurosis, and lipid accumulation in humans and animals. Dermal lesions including skin irritation, chloracne, and pigmentation of nails and skin have been

observed in humans following occupational exposure to relatively low-levels of PCB. Eye irritation, burning sensation, conjunctivitis, and eye discharge were also reported by occupationally exposed individuals.

Case studies of exposed workers have reported respiratory effects of tightness in the chest, impaired lung function and upper respiratory tract irritation; gastrointestinal effects of loss of appetite, nausea, epigastric distress and pain and intolerance to fatty foods; and neurological effects of dizziness, headaches, depression and fatigue. Human developmental effects are seen in lower birth weights and a shortened gestational age. Evaluations of blood samples from women who aborted, miscarried, or delivered prematurely showed associations between those effects and concentrations of PCBs (ATSDR, 1992).

Occupational studies suggest possible PCB-related liver, gastrointestinal tract, hematopoietic system, and skin carcinogenicity. In animal studies, PCB exposure caused cancerous liver tumors. PCBs as a group have been classified as probable human carcinogens by IARC and by EPA. These classifications are based on sufficient evidence of carcinogenicity in animals, and as evaluated by IARC, limited evidence of carcinogenicity in humans. NTP has concluded that PCBs are reasonably anticipated to be carcinogenic in humans based on sufficient evidence of carcinogenicity in animals. Because there is insufficient information about which constituents of the PCB mixtures are carcinogenic, it is assumed that PCB mixtures of any composition are potentially carcinogenic. This assumption has uncertainty since it can not be verified with present knowledge (ATSDR, 1992).

## 1.7 Polynuclear aromatic hydrocarbons.

Polycyclic Aromatic Hydrocarbons (PAHs) are generally categorized into two groups: carcinogens and non-carcinogens. Those that have been shown to be carcinogenic to animals by the oral route are: benzo(a)anthracene, benzo(a)pyrene, and dibenzo(a,h)anthracene. Benzo(a)anthracene, benzo(a)pyrene, benzo(b)fluoranthene, benzo(k)fluoranthene, chrysene, dibenzo(a,h)anthracene, indeno(1,2,3-cd)pyrene have been shown to be carcinogenic by the dermal route. For many of the carcinogenic PAHs, it would appear that the site of tumor

induction is generally the point of first contact, i.e., stomach tumors are observed following ingestion, and skin tumors following dermal exposure.

Evidence exists to indicate that certain PAHs are carcinogenic in humans. PAHs express their carcinogenic activity through biotransformation to chemically reactive intermediates which then covalently bind to cellular macromolecules (i.e., DNA) leading to mutation and tumor initiation. The evidence of carcinogenicity in humans comes primarily from occupational studies where workers involved in such processes as coke production, roofing, oil refining or coal gasification are exposed to mixtures containing PAHs (e.g., coal tar, roofing tar, soot, coke oven emissions, soot, and crude oil). PAHs have not been clearly identified as the causative agent, however. Cancer associated with exposure to PAHcontaining mixtures in humans occurs predominantly in the lung and skin following inhalation and dermal exposure, respectively. Some ingestion of PAHs is likely due to swallowing of particles containing PAHs subsequent to mucociliary clearance of these particulates from the lung.

Noncancer adverse health effects associated with noncarcinogenic PAHs (acenaphthene, acenaphthylene, anthracene, fluoranthene, fluorene, phenanthrene, and pyrene) exposure have been observed in animals, but (with the exception of adverse hematological and dermal effects) generally not in humans. Animals studies demonstrate that PAHs tend to affect proliferating tissues such as bone marrow, lymphoid organs, gonads and intestinal epithelium. Thus, although PAHs are distributed extensively throughout the body, their major target organs appear to be the hematopoietic and lymphoid systems in animals.

The lymphoid system, because of its rapidly proliferating tissues, is susceptible to PAHinduced toxicity. The mechanism of action for this effect is most likely inhibition of DNA synthesis. No adverse effects on this system associated with PAH exposure have been reported in humans, but several accounts of lymphoid toxicity in animals were observed. Lymphoid effects in animals from PAH exposure include an increase in reticulum cells, accumulation of iron, reduced lymphoid cells, and dilated lymph sinuses.

The skin is susceptible to PAH-induced toxicity in humans. Regressive verrucae were reported following subchronic application of benzo(a)pyrene to human skin. Although reversible and apparently benign, these changes were seen to represent neoplastic proliferation. Benzo(a)pyrene application also apparently exacerbated skin lesions in patients with pre-existing skin conditions (pemphigus vulgaris and xeroderma pigmentosum). Workers exposed to substances that contain PAHs (e.g., coal tar) experienced chronic dermatitis and hyperkeratosis.

Anthracene has been associated with gastrointestinal toxicity in humans. Humans that consumed laxatives that contained anthracene (anthracene concentration not specified) for prolonged periods were found to have an increased incidence (73.4%) of melanosis of the colon and rectum as compared to those who did not consume anthracene-containing laxatives (36.5%).

## 1.8 References.

## 1.8.1 Antimony.

- Agency for Toxic Substances and Disease Registry (ATSDR). Antimony. ATSDR/U.S. Public Health Service, DRAFT.
- Belyaeva, A. P. The effect of antimony on reproduction. Gig. Truda. Prof. Zabol. 11:32. (Cited in ATSDR, 1990).
- Bio/dynamics. A three month inhalation toxicity study of antimony trioxide in the rat followed by a recovery period. Prepared by Bio/dynamics, Inc. E. Millstone, NJ for the Antimony Oxide Industry Assoc., Washington, D.C. (Cited in ATSDR, 1990).
- Bradley, W.R. and W. G. Frederick. The toxicity of antimony animal studies. Ind. Med. 10:15-22. (Cited in ATSDR, 1990).
- Breiger, H., C. W. Semisch, J. Stasney and D.A. Piatnek. Industrial antimony poisoning. Indust. Med. Health 23:521-523. (Cited in ATSDR, 1990).
- Cooper, D.A., E. P. Pendergrass, A. J. Vorwald, et al. Pneumoconiosis among workers in an antimony industry. Am. J. Roentgenol. Rad. Ther. Nuclear Med. 103:495-508. (Cited in ATSDR, 1990).

Davies, D.J.A. and B.G. Bennett. Summary exposure assessments for copper, vanadium,

antimony In: Exposure Assessments of Environmental Pollutants, vol. 30. Monitoring and Assessment Centre, Chelsea College, University of London, London.

- Felicetti, S. W., R. G. Thomas and R. O. McClellan. Metabolism of two valence states of inhaled antimony in hamsters. Am. Ind. Hyg. Assoc. J. 355:292-300. (Cited in ATSDR, 1990).
- Gerber, G. B., J. Maes and B. Eykens. Transfer of antimony and arsenic to the developing organism. Arch. Toxicol. 49:159-168.
- Groth, D. H., L. E. Stettler, J. R. Burg, et al. Carcinogenic effects of antimony trioxide and antimony ore concentrate in rats. J. Toxicol. Environ. Health 18:607-626. (Cited in ATSDR, 1990).

Industrial Biotest Laboratories (IBTL). Acute toxicity with antimony oxide. OTS206223. (Cited in ATSDR, 1990).

Ludersdorf, R., A. Fuchs, P. Mayer, et al. Biological assessment of exposure to antimony and lead in the glass-producing industry. Int. Arch. Occup. Environ. Health 59:469-474. (Cited in ATSDR, 1990).

Potkonjak, V. and M. Pavlovich. Antimoniosis: A particular form of pneumoconiosis. I. Etiology, clinical and x-ray findings. Int. Arch. Occup. Environ. Health 51:199-207. (Cited in ATSDR, 1990).

Renes, L. E. Antimony poisoning in industry. Arch. Ind. Hyg. 7:99-108.

Rossi, F., R. Acampora, C. Vacca, et al. Prenatal and postnatal antimony exposure in rats: effect on vasomotor reactivity development of pups. Teratogen. Carcinogen. Mutagen. 7:491-496.

Schroeder, H.A., M. Michener and A.P. Nason. Zirconium, niobium, antimony, vanadium and lead in rats: Life-time studies. J. Nutr. 100:59-68.

Stevenson, C. J. Antimony spots. Trans. St. John's Hospital Dermat. Soc. 51:40-42. (Cited in ATSDR, 1990).

U. S. Environmental Protection Agency (EPA). 1985. Chemical, Physical, and Biological Properties of Compounds Present at Hazardous Waste Sites: Antimony. Final Report for the Environmental Protection Agency.

U. S. Environmental Protection Agency (EPA). December 1992. Office of Drinking Water and Health Advisory. Washington, DC.

Watt, W.D. Chronic inhalation toxicity of antimony trioxide: validation for the TLV - progress report-summary of results. OTS206195. (Cited in ATSDR, 1990).

Wong, L. C. K., J. M. Winston, J. Hagensen, et al. Study of carcinogenicity and toxicity of inhaled antimony trioxide, antimony ore concentrate and thallic oxide in rats. NIOSH/U.S. Dept. Health. OTS0511065. (Cited in ATSDR, 1990).

1.8.2 Arsenic.

- Agency for Toxic Substances and Disease Registry (ATSDR). 1989. Toxicological Profile for Arsenic. Agency for Toxic Substances and Disease Registry, U.S. Public Health Service, Atlanta, GA. ATSDR/TP-88/02.
- Agency for Toxic Substances and Disease Registry (ATSDR). Toxicological Profile for Arsenic. United States Department of Health and Human Services, Public Health Service, Atlanta: ATSDR. ATSDR/TP-88/02.
- Armstrong, C.W.; Stroube, R.B.; Rubio, T.; Siudyla, E.A.; Miller, G.B. Outbreak of fatal arsenic poisoning caused by contaminated drinking water. Arch. Environ. Health 39:276-279.
- Axelson, O.; Dahlgren, E.; Jasson, C.-D.; Rehnlund, S.O. Arsenic exposure and mortality: a case-referent study from a Swedish copper smelter. Br. J. Ind. Med. 35:8-15. (Cited in ATSDR, 1989).
- Baroni, C; van Esch, G.J.; Saffiotti, U. Carcinogenesis tests of two inorganic arsenicals. Arch. Environ. Health 7:668-674.
- Baxley, M.N; Hood, R.D.; Vedel, G.C.; Harrison, W.P.; Szczech, G.M. Prenatal toxicity of orally administered sodium arsenite in mice. Bull. Environ. Contam. Toxicol. 26:749-756.
- Beaudoin, A.R. Teratogenicity of sodium arsenate in rats. Teratology 10:153-158. (Cited in U.S. EPA, 1984).
- Blakely, B.R.; Sisodia, C.S.; Mukkur, T.K. The effect of methylmercury, tetrethyl lead, and sodium arsenite on the humoral immune response in the rat. Toxicol. Appl. Pharmocol. 52:245-254.
- Blom, S.; Lagerkvist, B.; Linderholm, H. Arsenic exposure to smelter workers: clinical and neurophysiological studies. Scand. J. Work Environ. Health 11:265-270.
- Borgono, J.M.; Greiber, R. Epidemiological study of arsenism in the city of Antofagasta. In: Trace Substances in Environmental Health, vol. 5. D.C. Hemphill, ed. University of Missouri, Columbia, MS. pp. 13-24.
- Brown, M.M.; Rhyne, B.C.; Goyer, R.A.; Fowler, B.A. Intracellular effects of chronic arsenic administration on renal proximal tubule cells. J. Toxicol. Environ. Health 1:505-514.

Burk, D.; Beaudoin, A.R. Arsenate-induced renal agenesis in rats. Teratology 16:247-260.

- Byron, W.R.; Bierbower, G.W.; Brouwer, J.B.; Hansen, W.H. Pathologic changes in rats and dogs from two-year feeding of sodium arsenite or sodium arsenate. Toxicol. Appl. Pharmacol. 10:132-147.
- Cebrian, M.E.; Albores, A.; Aguilar, M.: Blakely, E. Chronic arsenic poisoning in the north of Mexico. Hum. Toxicol. 2:121-133.
- Crecelius, E.A. Changes in the chemical speciation of arsenic following ingestion by man. Environ. Health Perspect. 19:147-150. (Cited in U.S. EPA, 1984):

Datta, D.V. Arsenic and noncirrhotic portal hypertension. Lancet 1:433.

Eisler, Ronald 1988 Arsenic Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review Contaminant Hazard Reviews Report no. 12, Biological Report 85 (1.12).U.S. Fish and Wildlife Service, Patuxent Wildlife Research Center, Laurel, Maryla pp. 92.

Feldman, R.G.; Niles, C.A.; Kelly-Heyes, M.; Sax, D.S.; Dixon, W.J.; Thompson, D.J.; Landau, E. Peripheral neuropathy in arsenic smelter workers. Neurology 29:939-944.

Ferm, V.H.; Carpenter, S.J. Malformations induced by sodium arsenate. J. Reprod. Fert. 17:199-201.

Fowler, B.A.; Weissberg, J.B. Arsine poisoning. New Eng. J. Med. 291:1171-1174.

Fowler, B.A.; Woods, J.S. The effects of prolonged oral arsenate exposure on liver mitochondria of mice: morphometric and biochemical studies. Toxicol. Appl. Pharmacol. 50:177-187.

Fowler, B.A.; Woods, J.S.; Schiller, C.M. Studies of hepatic mitochondria structure and function: Morphometric and biochemical evaluation of in vivo perturbation by arsenate. Lab. Invest. 42:313-320.

Franzblau, A.; Lilis, R. Acute arsenic intoxication from environmental arsenic exposure. Arch. Environ. Health 44(6):385-390.

Gerber, G. B., J. Maes and B. Eykens. Transfer of antimony and arsenic to the developing organism. Arch. Toxicol. 49:159-168.

Heyman, A.; Pfeiffer, J.B.; Willett, R.W.; Taylor, H.M. Peripheral neuropathy caused by arsenical intoxication. New England J. Med. 254(9): 401-409.

Heywood. R.; Sortwell, R.J. Arsenic intoxication in the rhesus monkey. Toxicol. Lett. 3:137-144.



- Hindmarsh, J.T.; McCurdy, R.F. Clinical and environmental aspects of arsenic toxicity. CRC Crit. Rev. Clin. Lab. Sci. 23:315-347.
- Hindmarsh, J.T.; McLetchie, O.R.; Heffernan, L.P.; Haynie, O.A.; Ellenberger, H.A.; McCurdy, R.F.; Thiebaux, H.J. Electromyographic abnormalities in chronic environmental arsenicalism. J. Anal. Toxicol. 1:270-276.
- Hine, C.H.; Pinto, S.S.; Nelson, K.W. Medical problems associated with arsenic exposure. J. Occup. Med. 19(6):391-396.
- Hood, R.D.; Bishop, S.L. Teratogenic effects of sodium arsenate in mice. Arch. Environ. Health 24:62-65.
- Hood, R.D.; Thacker, G.T.; Patterson, B.L. Effects in the mouse and rat of prenatal exposure to arsenic. Environ. Health Perspect. 19:219-222. (Cited in ATSDR, 1989).
- Huang, Y.Z.; Qian, X.C.; Wang, G.Q.; Xiao, B.-Y.; Ren, D.D.; Feng, Z.Y.; Wu, J.Y.; Xu, R.J.; Zhang, F. Endemic chronic arsenism in Xinjiang. Chin. Med. J. (Eng). 98:219-222.
- Kyle, R.A.; Pease, G.L. Hematologic aspects of arsenic intoxication. New Eng. J. Med. 273(1):18-23.
- Lagerkvist, B.E.A.; Linderholm, H.; Nordberg, G.F. Vasospastic tendency and Raynaud's phenomenon in smelter workers exposed to arsenic. Environ. Res. 39:465-474.
- Landau, E.D.; Thompson, D.J.; Feldman, R.G.; Goble, G.J.; Dixon, W.J. Selected Noncarcinogenic Effects of Industrial Exposure to Inorganic Arsenic. U.S. Environmental Protection Agency, Washington, DC. EPA 659/6-77-018.
- Lee, A.M.; Fraumeni, J.F., Jr. Arsenic and respiratory cancer in man an occupational study. J. Natl. Cancer Inst. 42:1045-1052.
- Lyster, W.R. Arsenic and sex ratio in man. Med. J. Austral. 2:442.
- Mahaffery K.R.; Capar, S.G.; Gladen, B.C.; Fowler, B.A. Concurrent exposure to lead, cadmium, and arsenic: effects of toxicity and tissue metals in the rat. J. Lab. Clin. Med. 98:463-481
- Mizuta, N.; Mizuta, M.; Ita, F.; Ito, T.; Uchida, H.; Watanabe, Y.; Akama, H.; Murakami, T.; Hayashi, F.; Nakamura, K.; Yamaguchi, T.; Mizuia, W.; Ois An outbreak of acute arsenic poisoning caused by arsenic-contaminated soy sauce (shoye). A clinical report of 220 cases. Bull. Yamaguchi Med. Sch. 4:131-150. (Cited in U.S. EPA, 1984).
- Morris, J.S.; Schmid, M.; Newman, S.; Scheuer, P.J.; Sherlock, S. Arsenic and noncirrhotic portal hypertension. Gastroenterology 64:86-94.

- Nagymjtenyi, L.; Selypes, A.; Berencsi, G. Chromosomal aberrations and fetotoxic effects of atmospheric arsenic exposure in mice. J. Appl. Toxicol. 5:61-63. (Cited in ATSDR, 1989).
- Perry, K; Bowler, R.G.; Buckell, H.M.; Druett, H.A.; Schilling, R.S.F. Studies in the incidence of cancer in a factory handling inorganic compounds of arsenic II: clinical and environmental investigations. Br. J. ind. Med. 5:6-15.
- Pershagen, G. Lung cancer mortality among men living near an arsenic-emitting smelter. Am. J. Epidemiol. 122:684-696.
- Pinto, S.S.; McGill, C.M. Arsenic trioxide exposure in industry. Ind. Med. Surg. 22:281-287. (Cited in U.S. EPA, 1984).
- Rozenshstein, I.S. Sanitary toxicological assessment of low concentrations of arsenic trioxide in the atmosphere. Hyg. Sanit. 34:16-22. (Cited in U.S. EPA, 1984).
- Salcedo, J.C.; Portales, A.; Landecho, E.; Diaz, R. Transverse study of a group of patients with vasculopathy from chronic arsenic poisoning in communities of the Francisco de Madero and San Pedro Districts, Coahuila, Mexico. Revista de la Facultad de Medicina de Torreon 12:16. (Cited in ATSDR, 1989).
- Tay, C.H.; Seah, C.S. Arsenic poisoning from anti-asthmatic herbal preparations. Med. J. Aust. 2:424-428.
- Terada, H.; Katsuta, K.; Sasakawa, T.; Saito, T.; Shrota, H.; Fukuchi, K.; Sekiya, E.; Yokoyama, Y.; Hirokawa, S.; Watanabe, G.; Hasegawa, K.; Shina. Clinical observations of chronic toxicosis by arsenic. Nihon Rinsho 18:2394-2403.
- Tseng, W.P.; Chu, H.M.; How, S.W.; Fong, J.M.; Lin, C.S.; Yeh, S. Prevalence of skin cancer in an endemic area of chronic arsenium in Taiwan. J. Natl. Cancer Inst. 40:453-463.
- U. S. Environmental Protection Agency (EPA). Health Assessment Document for Arsenic. Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Research Triangle Park, NC. EPA 600/8-32-021F.
- U. S. Environmental Protection Agency (EPA). Special Report on Ingested Arsenic: Skin Cancer; Nutritional Essentiality. Prepared for the Risk Assessment Forum, U.S. Environmental Protection Agency, Washington, DC. EPA/625/3-87/013.
- United States Air Force (USAF). Arsenic . In: The Installation Restoration Program Toxicology Guide, vol. 5. Wright-Patterson Air Force Base, OH. pp.75-102.
- Valentine, J.L.; Campion, D.S.; Schluchter, M.D.; Massey, F.J. Arsenic effects on human nerve conduction. In: Proceedings of the Fourth International Symposium on Trace Element Metabolism in Man and Animals, J.M. Gawthorne, J.M. Howell, and C.L. White,

eds.

- Viallet, A.; Guillaume, L.; Cote, J.; Legare, A.; Lavoie, P. Presinusoidal portal hypertension following chronic arsenic intoxication. Gastroenterology 62:177.
- Webb, D.R.; Wilson, S.E.; Carter, D.E. Comparative pulmonary toxicity of gallium arsenide, gallium (III).oxide, and arsenic (III).oxide intratracheally instilled into rats. Toxicol. Appl. Pharmacol. 82:405-416.
- Webb, D.R.; Wilson, S.E.; Carter, D.E. Pulmonary clearance and toxicity of respirable gallium arsenide particulates intratracheally instilled into rats. Am. Ind. Hyg. Assoc. J. 48(7):660-667.
- Woods, J.S.; Fowler, B.A. Altered regulation of mammalian hepatic heme biosynthesis and urinary porphyrin excretion during prolonged exposure to sodium arsenate. Toxicol. Appl. Pharmacol. 43:361-371.
- Woods, J.S.; Fowler, B.A. Effects of chronic arsenic exposure on hematopoietic function in adult mammalian liver. Environ. Health Perspect. 19:209-213.
- Wu, M.M.; Kuo, T.L.; Hwang, Y.H.; Chen, C.J. Dose-response relation between arsenic concentration in well water and mortality from cancers and vascular diseases. Am. J. Epidemiol. 130:1123-1132.
- Zaldivar, R. Arsenic contamination of drinking water and foodstuffs causing endemic chronic poisoning. Beitr. Path. 151:384-400.

1.8.3 Beryllium.

- Agency for Toxic Substances and Disease Registry (ATSDR). Toxicological Profile for Beryllium. United States Department of Health and Human Services, Public Health Service. Atlanta: ATSDR. ATSDR/TP-88/07.
- Agency for Toxic Substances and Disease Registry (ATSDR). Toxicological Profile for Beryllium. Prepared by Syracuse Research Corporation under Contract 68-C8-0004. U.S. Public Health Service. ATSDR/TP-88/09.
- Bayliss, D.L. and J.K. Wagoner. Bronchogenic cancer and cardio-respiratory disease mortality among white males employed in a beryllium production facility. OSHA Beryllium hearing, 1977, Exhibit 13.F. (Cited in U.S. EPA, 1987a).
- Bayliss, D.L. and W.S. Lainhart. Mortality patterns in beryllium production workers. Presented at the Am. Ind. Hygiene Assoc. Conf. OSHA Exhibit No. 66, Docket No. H005. (Cited in U.S. EPA, 1987a).

Bayliss, D.L., W.S. Lainhart, L.J. Crally, R. Ligo, H. Ayer and F. Hunter. Mortality patterns

in a group of former beryllium workers. In: Tran. 334rd Ann. Meeting ACGIH, Toronto, Canada. p. 94-107. (Cited in U.S. EPA, 1987a).

- Constantinidis, K. Acute and chronic beryllium disease. Br. J. Clin. Pract. 32: 127-136. (Cited in U.S. EPA, 1987a).
- Crowley, J.F., J.G. Hamilton and K.J. Scott. The metabolism of carrier-free radioberyllium in the rat. J. Biol. Chem. 177: 975-984.
- Freiman, D.G. and H.L. Hardy. Beryllium disease: the relation of pulmonary pathology to clinical course and prognosis based on a study of 130 cases from the U.S. Beryllium Case Registry. Hum. Pathol. 1: 25-44. (Cited in Kriebel et al., 1988b).
- Hall, T.C., C.H. Wood, J.D. Stoeckle and L.B. Tepper. Case data from the Beryllium Registry. Am. Med. Assoc. Arch. Ind. Health 19: 100-103.
- Kriebel, D., J.D. Brain, N.L. Sprince and H. Kazemi. The pulmonary toxicity of beryllium. Am. Rev. Respir Dis. 137: 464-473.
- Kriebel, D., N.L. Sprince, E.A. Eisen, I.A. Greaves, H.A. Feldman and R.E. Greene. Beryllium exposure and pulmonary function: A cross sectional study of beryllium workers. Br. J. Ind. Med. 45: 167-173.
- Mancuso, R.F. Mortality study of beryllium industry workers' occupational lung cancer. Environ. Res. 21: 48-55.
- Mancuso, T.F. Occupational lung cancer among beryllium workers in dusts and disease. In: R. Lemen and J. Dement, Eds. Proc. Conf. Occup. Exp. to Fibrous and Particulate Dust and Their Extension into the Environment. Pathrotox Publishers, Inc. (Cited in USEPA, 1987).
- Mancuso, T.F. Relation of duration of employment and prior illness to respiratory cancer among beryllium workers. Environ. Res. 3: 251-275.
- Meehan, W.R. and L.E. Smyth. Occurrence of beryllium as a trace element in environmental materials. Environ. Sci. Technol. 1: 839-844.
- Nishimura, M. Clinical and experimental studies on acute beryllium disease. Nagoya J. Med. Sci. 18: 17-44. (Cited in U.S. EPA, 1987a).
- Reeves, A.L. The absorption of beryllium from the gastrointestinal tract. Arch. Environ. Health 11: 209-214.
- Reeves, A.L. and A.J. Vorwald. Beryllium carcinogenesis. II. Pulmonary deposition and clearance of inhaled beryllium sulfate in the rat. Cancer Res. 27: 446-451.

- Reeves, A.L. and D. Deitch. Influence of age on the carcinogenic response to beryllium inhalation. In: S. Harishima, Ed. Proc. 16th Internat. Cong. Occup. Health, Tokyo, Japan. Japan Industrial Safety Association, Tokyo, Japan. p. 651-652.
- Schepers, G.W.H., T.M. Durkan, A.B. Delahant and F.T. Creedon. The biological action of inhaled beryllium sulfate: A preliminary chronic toxicity study on rats. Am. Med. Assoc Arch. Ind. Health 15: 32-38.
- Schroeder, H.A. and M. Mitchener Life-term studies in rats; Effects of aluminum, barium, beryllium and tungsten J. Nutr. 105: 421-427.
- Stiefel, T., K. Schulze, H. Zorn and G. Tolg. Toxicokinetic and toxicodynamic studies of beryllium. Arch. Toxicol. 45: 81-92.
- Tepper, L.B., H.L. Hardy and R.J. Chamberlin. Toxicity of Beryllium Compounds. Elsevier, New York, pp. 146-168. (Cited in ATSDR, 1988).
- U. S. Environmental Protection Agency (EPA). Drinking Water Criteria Document for Beryllium. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the office of Drinking Water.
- U. S. Environmental Protection Agency (EPA). Health Assessment Document for Beryllium. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Research Triangle Park, NC. External Review.
- U. S. Environmental Protection Agency (EPA). Health Assessment Document for Beryllium. Office of Health and Environmental Assessment, U.S. EPA. (EPA/600/8-84/026F).
- U. S. Environmental Protection Agency (EPA). Health Effects Assessment for Beryllium and Compounds. Prepared for Office of Solid Waste and Emergency Response by Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cinncinati, OH ECAO-CINN-H108.
- Vorwald A.J., P.C. Pratt and E.J. Urban. The production of pulmonary cancer in albino rats exposed by inhalants to an aerosol of beryllium sulfate. Acta. Unio. Int. Cancrum. 11: 735. (Cited in U.S. EPA, 1987a).
- Vorwald, A.J. Adenocarcinoma in the lung of albino rats exposed to compounds of beryllium. In: Cancer of the Lung - An Evaluation of the Problem. Proc. of the Scientific Session, Annual Meeting, November. Am. Cancer Soc., New York. p. 103-109. (Cited in U.S. EPA, 1987a).
- Vorwald, A.J., A.L. Reeves and E.J. Urban. Experimental beryllium toxicology. In: H.E. Stokinger, Ed. Beryllium Its industrial Hygiene Aspects. Academic Press, New York. pp.

201-234.

Wagoner, J.K., P.F. Infante and D.L. Bayliss. Beryllium: An etiologic agent in the induction of lung cancer, nonneoplastic respiratory disease and heart disease among industrially exposed workers. Environ. Res. 21: 15-34.

1.8.4 Cadmium.

Agency for Toxic Substances and Disease Registry (ATSDR) 1992 Draft Toxicological Profile for Cadmium. U.S. Public Health Service 1992.

Agency for Toxic Substances and Disease Registry (ATSDR) 1989 Toxicological Profile for Cadmium. U.S. Public Health Service 1989.

Agency for Toxic Substances and Disease Registry (ATSDR) Toxicological Profile for Cadmium. United States Department of Health and Human Services, Public Health Service. Atlanta: ATSDR. ATSDR/TP-88/08.

Armstrong BG and Kazantzis G. 1983 The mortality of cadmium workers. Lancet: 1425-1427.

Aughey E, Fell GS, Scott R and Black M. 1984 Histopathology of early effects of oral cadmium in the rat kidney. Environ Health Perspect; 54:153-161.

Barrett, H.M., D.A. Irwin and E. Semmons. Studies on the toxicity of inhaled cadmium. I. The acute toxicity of cadmium oxide by inhalation. J. Ing. Hyg. Toxicol. 29:279. (Cited in ATSDR, 1989)

Beton, D.C., G.S. Andrews, H. J. Davies, L. Howelss and G.F. Smith. Acute cadmium fume poisoning: Five cases with one death from renal necrosis. Br. J. Ind. Med. 23:292. (Cited in ATSDR, 1989)

Dixon RL, Lee IP, and Shering RJ. 1976 Methods to assess reproductive effects of environmental chemicals: Studies of cadmium and boron administered orally. Environ Health Perspect; 13:59-67.

Engstrom B, and Nordberg CF. 1979 Dose-dependence of gastrointestinal absorption and biological half-time of cadmium in mice. Toxicology; 13:215-222.

Exor JH, and Koller LD. 1986 Immunotoxicity of cadmium. In: Foulkes EC, ed. Handbook of Experimental Pharmacology, Vol 80. Berlin: Springer-Verlag.

Friberg LT, Piscator M, Nordberg G. 1974 Cadmium in the Environment, 2nd ed. Cleveland: CRC Press.

Friberg, L. Health hazards in the manufacture of alkaline accumulators with special

reference to chronic cadmium poisoning. Acta Med. Scad. (Suppl 240) 138:1-124. (Cited in ATSDR, 1989)

- Hadley JG, Conklin AW, and Sanders CL. 1980 Rapid solubilization and translocation of 109CdO following pulmonary deposition. Toxicol Appl Pharmacol; 54:156-160.
- Hagino N, and Yoshioka Y. 1961 A study of the etiology of Itai-Itai disease. J Jap Orthoped Assoc; 35:812-815 (in Japanese, as quoted in Friberg, et al, 1974).
- Kipling MD, and Waterhouse JAH. 1967 Cadmium and prostatic carcinoma. Lancet; 1:730.
- Kjellstrom T, Evrin PE, and Rahnster B. 1977 Dose-response analysis of cadmium-induced tubular proteinuria: A study of urinary beta 2-microglobulin excretion among workers in a battery factory. Environ Res; 13:303-317.
- Kopp, SJ. 1986 Cadmium and the cardiovascular system. In: Foulkes EC, ed. Handbook of Experimental Pharmacology, Vol 80. Berlin: Springer-Verlag. 195-280.
- Mahaffey, K.R.; Capar,S.G.; Gladen, B.C.; Fowler, B.A. Concurrent exposure to lead, cadmium, and arsenic: effects of toxicity and tissue metal concentrations in the rat. J. Lab. Clin. Med. 98:463-481
- Nordberg GF, Kjellstrom T, and Nordberg M. 1985 Kinetics and metabolism. In: Friberg C, Elinder G, Kjellstrom T, and Nordberg GF, eds. Cadmium and Health: A Toxicological and Epidemiological Appraisal. Vol I: E
- Takenaka, S., H. Oldiges, H. Konig, D. Hochrainer, and G. Oberdoerster. 1983 Carcinogenicity of cadmium aerosols in Wisher rats. J. Nat'l Cancer Inst. 70: 367-373.

Thun MJ, Schnorr TM, Smith AB, Halperin WE, and Lemen BA. 1985 Mortality among a cohort of U.S. cadmium production workers - an update. JNCI; 74:325-333.

- U. S. Environmental Protection Agency (EPA) 1985 Drinking Water Criteria Document for Cadmium (Final Draft). Office of Drinking Water. Washington, D.C. April 1985. PB86 117934/AS.
- U. S. Environmental Protection Agency (EPA) 1981 Health Assessment Document for Cadmium. Research Triangle Park, NC. EPA 600/8-81-023.
- U. S. Environmental Protection Agency (EPA) 1985 Updated Mutagenicity and Carcinogenicity Assessment Document for Cadmium (May 1 981, EPA 600/B-B1-023). EPA 600/B-83-025F.
- U. S. Environmental Protection Agency (EPA) 1985 Updated Mutagenicity and Carcinogenicity Assessment for Cadmium. Addendum to the Health Assessment Document for Cadmium (May 1981; EPA/600/8-81/023). Office of Health and Environmental

Assessment. Washington, D.C. June 1985. PB86 117934/AS.

United States Air Force (USAF) Cadmium. In: Installation Restoration Program Toxicology Guide, Vol. 5. Harry G. Armstrong Aerospace Medical Research Laboratory, Wright Patterson AFB, OH.

1.8.5 Chromium.

Agency for Toxic Substances and Disease Registry (ATSDR). Draft Toxicological Profile for Chromium. United States Department of Health and Human Services, Public Health Service. Atlanta: ATSDR.

Agency for Toxic Substances and Disease Registry (ATSDR). 1992. Draft Toxicological Profile for Chromium. U.S. Public Health Service 1992.

Agency for Toxic Substances and Disease Registry (ATSDR). Toxicological Profile for Chromium. Prepared by Syracuse Research Corporation under Contract 68-C8-0004. U.S. Public Health Service. ATSDR/TP-88/10.

Agency for Toxic Substances and Disease Registry (ATSDR). 1989. Toxicological Profile for Chromium. U.S. Public Health Service 1989.

Anderson, RA. 1981. Nutritional role of chromium. Sci Total Environ. 17:13-29.

Baetjer, A.M., J.F. Lowney, H. Stefee and V. Budacz. Effect of chromium on incidence of lung tumors in mice and rats. Arch. Ind. Health 20: 124-135.

Bianchi, B. and Levis, AG. 1985. Mechanisms of chromium genotoxicity. In: Merian, E., Fre, RW., Hard, W., and Schlatter, E., eds. Carcinogenic and Mutagenic Metal Compounds: Environmental and Analytical Chemistry and Biological Effects. London: Gordon et al.

Bragt, P.C. and E.A. van Dura. Toxicokinetics of hexavalent chromium in the rat after intratracheal administration of chromates of different solubilities. Ann. Occup. Hyg. 27: 315-322.

Burrows, D. In: Burrows, D. Ed., Chromium: Metabolism and Toxicity CRC Press, Inc., Boca Raton, FL. p. 137-163.

Danielsson, G.R.G., E. Hassoun and L. Dencker. Embryotoxicity of chromium: Distribution in pregnant mice and effects on embryonic cells in vitro. Arch. Toxicol. 51: 233-245.

Donaldsonm, RM., and Barreras, RF. 1966. Intestinal absorption of trace quantities of chromium. J Lab Clin Med, 68:484-493.

Eisler, Ronald. 1986. Chromium hazards to fish, wildlife and invertebrates: A synoptic

A7-39

review U.S. Department of the Interior, Fish and Wildlife Service Biological Report 85(1.8) January.

- Glaser, U., D. Hochrainer, H. Kloppel and H. Oldiges. Carcinogenicity of sodium dichromate and chromium (VI/III) oxide aerosols inhaled by male Wistar rats. Toxicology 42: 219-232.
- Guthrie, B.E. The nutritional role of chromium. In: Langard, S., Ed. Biological and Environmental Aspects of Chromium. Elsevier Biomedical Press, Amsterdam, p. 117-148.
- Hamilton, J.W. and K.E. Wetterhahn. Chromium. In: Handbook on Toxicity of Inorganic Compounds, eds. H.G Seiler, H. Sigel and A. Sigel. pp. 239-250. New York: Marcel Dekker, Inc.
- Hayes, R.B. Carcinogenic effects of chromium. In: Langard, S., Ed. Biological and Environmental Aspects of Chromium. Elsevier Biomedical Press, Amsterdam, p. 221-247.
- Hertal, RF. 1986. Sources of exposure and biological effects of chromium. In: O'Neill IK, Schuller P, and Fishbein L, eds. Environmental Carcinogens: Selected Methods of Analysis, Vol 8. IARC Scientific Publ. No. 71. Lyon, France: World Health Organization.
- Hertal, RF. 1986. Sources of exposure and biological effects of chromium. In: O'Neill IK, Schuller P, and Fishbein L, eds. Environmental Carcinogens: Selected Methods of Lyon, France: World Health Organization. 64-77.
- Iijima, S., N. Matsumoto, and C.-C. Lu. Transfer of chromic chloride to embryonic mice and changes in the embryonic mouse neuroepithelium. Toxicology 26: 257-265.
- Ivankovic, S. and R. Preussmann. Absence of toxic and carcinogenic effects after administrations of high doses of chromic oxide pigment in subacute and long-term feeding experiments in rats. Fd. Cosmet. Toxicol. 13: 347-351.
- Korallus, U. 1986. Biological activity of chromium (VI)- against chromium (III) compounds: New aspects of biological monitoring. In: Serrone DM, ed. Chromium Symposium 1986: An Update. Pittsburgh, PA: Industrial Health Foundation, Inc. 210-230.
- Langard, S., and Norseth, T. 1986. Chromium. In: Friberg L, Nordberg GF, and Vouk VB, eds. Handbook on Toxicology of Metals, Vol II. Amsterdam, the Netherlands: Elsevier.
- Langard, S. Absorption, transport and excretion of chromium in an and animals. In: S. Langard, Ed. Biological and Environmental Aspects of Chromium. Elsevier Biomedical Press, Amsterdam, p. 149-169.
- Langard, S. The carcinogenicity of chromium compounds in man and animals. In: Burrows, C., Ed. Chromium: Metabolism and Toxicity. CRC Press, Inc., Boca Raton, FL, pp. 13-30.

COTTMAN AVENUE SITE: BASELINE HEALTH RISK ASSESSMENT: (1/27/94)

- Leonard, A. and R.R. Lauwerys. Carcinogenicity and mutagenicity of chromium. Mutat. Res. 76: 227-239.
- Lindberg R, and Hendenstierna G. 1983. Chromeplating: Symptoms finding in the upper airways and effects on lung function. Arch Environ Health; 38:367-374.
- Love, A.H.G. Chromium biological and analytical considerations. In: Burrows, D., Ed. Chromium: Metabolism and Toxicity. CRC Press, Inc., Boca Raton, FL, p. 1.
- MacKenzie RD, Byerrum RV, Decker CF, Hoppert CA, and Longham FL. 1958 Chronic toxicity studies II. Hexavalent and trivalent chromium administered in drinking water to rats. Arch Ind Health; 18:232-234.
- Mancuso, T.F. and W.C. Hueper. Occupational cancer and other health hazards in a chromate plant: A medical appraisal. I. Lung cancer in chromate workers. Ind. Med. Surg. 20: 358-363.
- Matsumoto, N., S. Iijima and H. Katsunuma. Placental transfer of chromic chloride and its teratogenic potential in embryonic mice. J. Toxicol. Sci. 2: 1-13.
- Mylanvarapu VB, and Sun T-J. 1991. Chromium contact dermatitis: A health-based risk assessment approach. The Toxicologist; 11:194 (abstract).
- Nettesheim, P., M.G. Hanna, Jr., D.G. Doherty, R.F. Newell and A. Hellman. Effect of calcium chromate dust, influenza virus, and 100 R whole-body X-radiation on lung tumor incidence in mice. J. Natl. Cancer Inst. 47: 1129-1138.
- Ogawa E. 1976. Experimental study on absorption, distribution and excretion of trivalent and hexavalent chromes. Japanese J Pharmacol; 26:92.
- Pedersen, N.B. The effects of chromium on the skin. S. Langard, Ed. Biological and Environmental Aspects of Chromium. Elsevier Biomedical Press, Amsterdam, pp. 249-275.
- Samitz MH, and Shrager J. 1966. Patch test reactions of hexavalent and trivalent chromium compounds. Arch Dermatol; 93:304-306.
- Schroeder, H.A., J.J. Balassa and I.H. Tipton. Abnormal trace metals in man: chromium. J. Chron. Dis 15: 941. (Cited in U.S. Air Force, 1990).
- Steffee, C.H. and A.M. Baetjer. Histopathologic effects of chromate chemicals. Report of studies in rabbits, guinea pigs, rats, and mice. Arch. Environ. Health 11: 66-75.
- Stern, R.M. Chromium compounds; production and occupational exposure. In: Langard, S., Ed. Biological and Environmental Aspects of Chromium. Elsevier, New York, pp. 7-47.

Symms KG. 1991. A health assessment of chromium residues following cleanup of a large



dichromate spill at a public facility. The Toxicologist; 11:194 (abstract).

- U. S. Environmental Protection Agency (EPA) 1980. Ambient Water Quality for Chromium. Office of Water Regulations and Standards. Washington, D.C. EPA 440/5-80-035.
- U. S. Environmental Protection Agency (EPA) 1984. Draft Health Advisory for Chromium. Office of Drinking Water. Washington, D.C. September 30 draft.
- U. S. Environmental Protection Agency (EPA) 1992. Hazardous Substances Database (HSDB).
- U. S. Environmental Protection Agency (EPA) 1984. Health Assessment Document for Chromium. Environmental Criteria and Assessment Office. Research Triangle Park, NC. EPA 600/8-83-014F.
- U. S. Environmental Protection Agency (EPA) 1984. Health Effects Assessment for Hexavalent Chromium. Environmental Criteria and Assessment Office. Cincinnati, OH. EPA 540/1-86-019.
- U. S. Environmental Protection Agency (EPA) 1984. Health Effects Assessment for Trivalent Chromium. Environmental Criteria and Assessment Office. Cincinnati, OH. EPA 540/1-86-035.
- United States Air Force (USAF) Chromium. In: The Installation Restoration Program Toxicology Guide. Wright-Patterson Air Force Base, OH, pp. 72-1 to 72-81.
- Wahlberg, J.E. and E. Skog. 1965. Percutaneous absorption of trivalent and hexavalent chromium. Arch. Dermatol. 92:315-318.
- Weigand H.J., H. Ottenwaelder, and H.M. Bolt. 1984. The reduction of chromium (IV) to chromium (III) by glutathioene: An intracellular redox pathway in the metabolism of the carcinogen chromate. Toxicol. 33:341-348.

1.8.6 3,3'-Dichlorobenzidine.

- Patnaik, Pradyot, Ph.D. 1992. <u>A Comprehensive Guide to the Hazardous Properties of Chemical Substances</u>. Van Nostrand Reinhold. New York, New York.
- U.S. Environmental Protection Agency (EPA). 1993. Hazardous Substances Databank (HSDB). Online October 26, 1993.
- U.S. Environmental Protection Agency (EPA). 1993. Integrated Risk Information System (IRIS) Database. Online October 28, 1993.

1.8.7 Polychlorinated biphenyls.

Agency for Toxic Substances and Disease Registry (ATSDR), 1992. Toxicological Profile for Polychlorinated Biphenyls (PCBs). U.S. Public Health Service. Draft for Public Comment.

1.8.8 Polynuclear aromatic hydrocarbons.

Agency for Toxic Substances and Disease Registry (ATSDR). 1990f. Toxicological Profile for Polycyclic Aromatic Hydrocarbons (PAHs). U.S. Public Health Service. Draft for Public Comment.

# Appendix D.2

# US EPA (Terrestrial) Ecological Risk Assessment

6696-403/revmarks/haz/drifs7 AR300844



United States Environmental Protection Agency Region III Philadelphia, PA 19107-4431

## ECOLOGICAL RISK ASSESSMENT

FOR

METAL BANK

MARCH 13, 1994

Prepared by:

Robert S. Davis, Biologist Technical Support Section Superfund Program Branch Hazardous Waste Management Division



#### METAL BANK (COTTMAN AVE.)

ECOLOGICAL RISK ASSESSMENT: Terrestrial areas and mudflats

FINAL: 3/15/94

CONTACT: Robert S. Davis, Biologist Technical Support Section (3HW13) EPA Region III 841 Chestnut Bldg. Phila., Pa. 19107

Phone: 215/597-3155

Introduction:

The objective of Ecological Risk Assessment (ERA) is to answer the question: "is there actual/potential ... ecological risk as a result of exposure to site-associated stressors?"?. The Ecological Risk Assessment is a formal part of the Remedial Investigation and is based upon technical information contained in it. Specific RI sections are not referenced.

There are three general approaches used in ERA, referred to as 'tiers'. Tier 1 involves identification of stressors (or contaminants of concern); ecological characterization of the site; fate & transport, identification of receptors, and receptor impacts. Tier 2 (tier 1 leads to tier 2) involves the uncertainties due to data gaps, data quality imperfections, variability in data (due to limits of study and time & space considerations -- when, where, and how sampling was done, methodologies, location of sampling points, etc.), and assumptions used in all aspects of the work (including selection of methodologies, analyses, etc. See footnote 1, below.).

Tier 3 involves site-specific sampling to refine exposure assessment, receptor characterization, and ecological effects characterization, ecological <u>surveys</u> (as opposed to mere characterizations) and in site toxicity testing to refine exposure/ risk (see footnote 1, below).

This ERA uses a conservative approach closer to a Tier 1 & 2 assessment than a Tier 3, since in-depth, site-site specific surveys were not carried out. The RI report did not include the kinds of information and details needed for a Tier 3 assessment. EPA Region III believes that the time and funds required to carry out the in-depth surveys, bioassays, and thorough chemical analyses of

<sup>&</sup>lt;sup>1</sup> See: <u>Framework for Ecological Risk Assessment.</u> 1992. [EPA/630/R-92/001] & <u>Risk Assessment Guidance for Superfund</u>: <u>Yol</u> <u>II Environmental Evaluation Manual (Interim Final</u>). 1989. EPA 540/1-89/001.

all media to complete a rigorous ERA may be unwarranted. Sufficient information is available through the RI effort to satisfy the needs of the quotient approach risk assessment. As a result, this conservative approach is an efficient and ecologically protective approach.

The approach involves the following 10 section items:

- 1) Problem definition.
- 2) Source characterization & exposure pathways.
- 3) Exposure assessment.
- 4) Ecological receptor characterization.
- 5) Ecological effects characterization.
- 6) Risk characterization.
- 7) Limitations.
- 8) Interpretation.
- 9) Risk Assessment (Conclusions).
- 10) Recommendations

#### Problem Definition:

Problem definition is intended to identify the areas of greatest concern and the goals are to characterize contamination and ecosystem(s), habitats, and the physiological responses of ecological receptors. This is followed by identification of the potential for impacts to specific species (if available or if possible to calculate). The assessment of the potential for risk, all media, except air, are included.

Actual species specific impacts are not included as the RI did not include such work. As a result, the bases for the risk assessment are ambient water quality criteria (chronic values) and literature-derived values. Species specific assessments are often difficult to consider since the efforts needed to carry out such studies relative to the site and receptors are both time consuming and costly. Where appropriate information gathered for the assessment of human health risks is also used.

The ERA is based upon the most conservative Environmental Effects Quotient (EEQ) defined as the chronic toxicity value, derived from literature, AWQC or other sources, divided into the reported environmental concentration (EEQ = Reported Concentration / Criterion). The RI falls short with respect to impact endpoints, making it dif-

ficult or impossible to select site-specific organisms to serve as subjects in the risk assessment. The only reasonable approach is to evaluate the potential for impact by using habitat and media values reported in the RI as the basis.

In general, EEQ's exceeding 1.0 indicate risk potential. Using this screening level approach, no specific receptor organism will be used. Rather, the occurrence of contaminants in media will be Any EEQ that exceeds 10 is considered an extreme the focus. potential for  $risk^2$ .

Stressors of serious nature identified at the site are: aluminum, cadmium, chloride, chromium (VI), copper, lead, mercury, zinc, 2butanone (methylethyl ketone), Di-n-butylphthalate, DDT-related compounds, Endosulfan, Heptachlor, PCBs, and several PAHs.

High levels of aluminum and iron are often discounted since their elevated levels may be attributable to soil sources. In this case, the levels of both in groundwater are at levels that would be considered in the toxic range, but are difficult to distinguish from soil background levels. In this case, it is assumed that the source is on-site soils rather than the wastes at the site and, therefore, these two contaminants are being ignored for this risk assessment.

The results of soils analyses from samples taken inside the  $court^{L}$ yard show PCBs (reported as Aroclor-1242) at levels considered to be of negligible concern for ecological receptors.

Soils sampled outside the courtyard show high levels of aluminum (which has been preferentially eliminated), copper, lead, 2-butanone, and Di-n-butylphthalate. These are contaminants at levels that are potentially of ecological concern.

The mudflats near the site show several organic compounds at levels that are of potential ecological concern: 4,4-DDD, benzo(a) anthracene, benzo(a)pyrene, chrysene, dibenz(a,h)anthracene, fluoranthene, phenenthrene, and pyrene. Dioxins and furans may also be of concern at the site, but are not included in this assessment. The aquatic risk assessment, however, has included them and concern on the part of the reader may be at least partially satisfied on a reading of that document.

Sediment samples from mudflats beyond the site show levels of aroclor-1248, 4,4-DDD, and 4,4-DDE at levels that are of potential risk to ecological resources.

The literature and file notes used in this risk assessment can be accessed at the Region III office and library. The sediment associated with riprap show the following contaminants at levels that are considered to be potentially harmful to ecological resources: Aroclor 1260, acenaphthene, anthracene, benzo(a)anthracene, benzo(a)pyrene, chrysene, dibenz(a,h)anthracene, fluoranthene, fluorene, 2-methylnaphthalene, naphthalene, and phenenthrene.

Groundwater along with the courtyard soils are the only media contaminated with metals. However, the groundwater is much more contaminated with metals than is the courtyard. Groundwater is contaminated with cadmium, chromium, copper, lead, mercury, and zinc (both aluminum and iron contaminants have been preferentially eliminated). The maximum reported for selenium also shows levels that are above those of potential risk to ecological resources. Organic contaminants include endosulfan, heptachlor, and phenanthrene. Chloride has also been identified in quantities that exceed threshold levels of potential ecological risk.

#### Source Characterization & Exposure Pathways:

The report entitled "Background Data Report for the Metal Bank/ Cottman Avenue RI/FS, by HMM Assoc., Inc. Sept. 1993 (BDR) describes the site and surroundings as well as the uses of the site, pathways, and the ecological/geological features. Please refer to that report for specific information on this issue.

Contaminants have been released into the groundwater as a nonaqueous phase liquid and have moved via the groundwater, mainly, to the mudflat and river. Soils are also a pathway by which ecological receptors can encounter contaminants.

The contamination picture is straight forward, as all of the contaminants are associated with the site and appear in more than one medium. Section 1.2 of the above referenced report, BDR, describes the character of the basic materials of which the site is comprised and the questions raised regarding attributable sources. It appears that no question is raised regarding the source of PCB, but some question exists as to the source of PAHs. However, if the site is comprised of "urban brown", a material that may contain asphalt among other kinds of materials, the site is likely to be the source of either all or at least the substantial percentage of, contaminants reported in the sampling analyses.

#### Exposure Assessment:

Terrestrial and avian species are exposed via direct contact and incidental ingestion of the contaminants of concern. Exposure occurs throughout the food chain when aquatic systems are under consideration, for those organisms that allocate portions of their time to media in both habitat settings. Ingestion, direct contact, and the food chain are the means of exposure.

All organisms from the base of the food chain (heterotrophic and autotrophic plants, oligochaetes, insect instars, crayfish) through the top are subject to exposure. In addition, avian and nonaquatic foragers and predators are exposed, both as members of the food chain and as water consumers.

Observation of ecological effects has been limited to reconnaissance without benefit of any tissue analyses or bioassays (either acute or chronic). Without this level of information, a specific target organism ecological risk assessment is impossible, therefore, the best recourse is a conservative approach using an environmental effects quotient (EEQ) based on the 95% upper confidence limits (UL). Exposure assessment cannot be verified without a much more specific sampling effort than was carried out. That is, sampling of tissues of a variety of organisms (as was done for the aquatic portion of the risk assessment done by NOAA) would justify a risk assessment focused upon specific organisms in the ecosystem. Since this level of study was not carried out for the terrestrial or mudflat areas, a species specific risk assessment is out of the question and is not justified.

The main routes of exposure are soil, sediments of the mudflats and riprap areas, and groundwater. Neither groundwater discharge points nor the air pathways are considered. Groundwater seeps would be expected to occur in the periphery of the site, i.e., in the steep banks along the Delaware, especially during periods of heavy and lengthy precipitation events. (The aquatic ecological risk assessment carried out by NOAA should be consulted for additional specifics regarding seeps.) This would be most notable in the riprap area, but none was reported in the RI and, in fact, was not a major concern of the RI. The systematic investigation of the air pathway was also not a focus of the RI. Therefore, only the soil and sediments of the mudflats and riprap areas along with ground-water are the pathways of major interest. The groundwater is included because it was studied as an integral part of the remedial investigation and because it carries contaminants in high quanti- ties and can be expected to discharge both to the river and along the periphery of the site.

Exposure appears to be general in all media mentioned above and not likely to be limited to any time of the year. Given the lack of site specific data on the site, it is not appropriate to assume any surrogate species can represent the extent and degree of ecological exposure. Therefore, this assessment uses the most conservative media contamination numbers in establishing a basis for assessing the potential for impacts.

#### Ecological Receptor Characterization:

Ecological receptor characterization is de-emphasized in this risk assessment because the level of detail in the RI is insufficient to support the higher levels of risk assessment. To carry out a tier

3 risk assessment, for example, more site-specific studies would be needed than provided in the RI and a phased study to determine the stressors carried by pathways to various media would be required. A list of species found at the site can be found in the appended tables covering those species found in terrestrial and wetland areas.

#### Ecological Effects Characterization:

The ecological effects of the site-related contaminants of concern for this ERA are developed from both published criteria documents and literature reviews. The information developed was then used in calculating environmental effects quotients (EEQ). Little if any uncertainty exists with regard to the ecological potentials of the contaminants, as both literature and the water quality criteria development publications show that a great deal of research has been done to document adverse impacts.

The contaminant, 2-Butanone (methylethyl ketone), found in subsoils outside the courtyard, is mainly of interest as a contaminant of groundwater. Soil degradation of this contaminant is unknown, but a portion is lost to the atmosphere and some leaches into groundwater. Adsorption onto soil particles is not well understood.

While the maximum values for soils (see Table 6) show elevated levels of copper, lead, and Di-n-butylphthalate, these contaminants are not considered in the risk assessment because the 95 UL values appear to be realistically low.

The sediment in the mudflats contains one chlorinated hydrocarbon, 4,4-DDD, and 7 PAH contaminants at levels considered to be potentially hazardous to ecological receptors. The chlorinated hydrocarbon, 4,4-DDD, has a high BCF and, therefore, a high potential for bioaccumulation as well as magnification through the food chain. Its impacts upon birds and amphibian eggs are well known and need not be enumerated here. Implications of the contaminant in the food chain are also inherent in light of its insecticidal properties.

PCBs have also been a major focal point of the investigations and are one, if not the main, reason for the site's listing on the NPL. While they are of minor concern for surface soils outside the courtyard area, they are of major concern in the remainder of the area and play a major role in the assessments.

The PAH compounds listed in Table 7 are all potentially damaging to ecological receptors. They are of particular concern to macroinvertebrates found in depositional areas, such as those represented by the rip-rap and mudflat areas.

PAHs cause tumors, and hyperplastic disease, but these conditions are generally not of great concern to macroinvertebrates receptors, due to short life span. But other qualities of these compounds are of interest. For example, the higher molecular weight PAHs are more bioaccumulative than the lower; they are accumulated in aquatic organisms from water, sediment, and diet; lower forms of aquatic life tend to bioaccumulate PAHs more than higher forms because of limited degradative capabilities (mussels, scallops, snails, etc.); PAHs have a long half-life in sediments and comparably long in soils; PAHs can cause skin darkening, hyperplasia, skin papillomas, coarsening and localized pigmentation in brown bullhead.

Invertebrate terrestrial receptors are equally as susceptible to PAHs as aquatic invertebrates. The literature search carried out for this assessment failed to discover sub-lethal impacts to vertebrates that might result from bioaccumulation in predatory species that prey on such organisms. It is presumed that impacts are comparable.

### Risk Characterization:

A list of ecological receptors exposed via all pathways has not been completed for this site, therefore, the types of effects, extent and severity of contamination cannot be calculated. For example, the types of habitats have been characterized, but not evaluated for the impacts from contamination using bioassays tissue residue evaluations, productivity, fecundity, population dynamics, etc. Without this level of information, the exposure of the ecological receptors cannot be evaluated in any way other than using the EEQ approach. This approach does not specify endpoints because, again, the appropriate level of investigation was not carried out at the site. No environmental testing other than presence/absence of contamination was carried out. No productivity tests or food chain tests, were carried out. the absence of these kinds of tools render difficulty with characterizing risk beyond the media level.

Some other factors, however, are available. As an example, seasonal variations in precipitation (and subsequent infiltration) are expected, resulting in cyclical increases in contaminants released from the primary source to receptors and secondary sources. Specifically, there would be an expected increase in leachate and the flow of groundwater, and consequent discharge to the offsite streams, and mudflat areas. Seasonal increases in the release and migration of contaminants may correspond with the species migration and breeding activity. Contaminated standing water may become attractive to migratory birds, or may attract amphibians, but impacts are not expected to be influenced by diurnal/nocturnal cycles of receptors.

Exposure to contaminants is both direct through ingestion, incidental contact, etc. or indirect through the food chain. Ex-

posure is not limited to any class or kind of organisms, as both flora and fauna are receptors.

With regard for surface water contaminants, exposure to terrestrial and avian species is possible in areas of mudflats standing or 'ponded' water onsite in the neighborhood of the mudflats and riprap sediment. Exposure is likely through ingestion of water and suspended particulates, as well as incidental ingestion of sediment.

Amphibian species are exposed in the same areas and, possibly, through any ponding that may result from ponding of water from seeps. Also of concern is exposure to species using the area either opportunistically or seasonally.

Exposure is possible to species which reside or forage on the site property or predators preying on species living and foraging on the site; exposure also via direct contact and incidental ingestion, an exposure route also considered. Plants can also be impacted by uptake of contaminants via their root systems and at the same time act as secondary or tertiary sources to fauna that utilize both roots and above ground parts as forage.

Amphibian species residing in mudflats and sediment areas in seasonally inundated areas are exposed when contaminated soil runoff brings contaminants with it. The sediment in runoff constitutes a pathway and is a secondary source and pathway for both aquatic plants and organisms found in the mudflats.

Groundwater contaminants can also impact aquatic and semi-aquatic species. Exposure is likely through the ingestion of water and suspended particulates, as well as incidental ingestion of sediments. A broad range of terrestrial, amphibian, and aquatic life routinely come into contact with contamination via this route of exposure.

Leachate Contaminants may affect a broad range of organisms, from terrestrial plants and animals through aquatic. The range of plants that invade such areas range from microscopic through trees and can be those that are obligate uplands through obligate wetlands species. In addition to plants, a wide variety of fauna describing a range similar to plants may also find leachate an attractive area. For example, seeps in the rip-rap and contaminants in the sediment of the mudflats are habitat for insects, making it an attractive area, as well, for insect predators, e.g., the eastern king bird. Exposure is also possible through ingestion of water at leachate and seep outfalls and discharge points.

Plants that invade such areas may have an attraction for foraging species and root-eating animals may be especially drawn to such areas.

As an example, soil gas can adversely impact soil organisms such as earthworms that are foundational in the food chain. Many animals, especially birds, use this organism as a basic food item. This risk assessment does not cover this route of contamination because of the lack of data in the RI and the paucity of information in the refereed literature.

Since nothing was done in regard to ecological surveys or insitu toxic effects for the flora and fauna identified in the site habitats, it is inappropriate to select any individual species for expression of a potential for risk. As a result, the environmental effects quotient is used in an effort to describe the potential for risk to organisms found in the habitats on site.

### Limitations:

The following data gaps and limitations have been identified:

- Terrestrial surveys have not been conducted on or around the site. Thus, information regarding habitat descriptions for the site and surrounding areas, and identification of terrestrial receptor species located on the site, are confined to characterizations only.
- From the habitat/receptor view point, reference areas<sup>1</sup>
   were not selected and described.
- Chemical data are the sole basis for the ecological risk and as such the conservative approach may necessarily ignore specific ecological impacts or lack of impacts.
- The EEQ does not take endangered species into consideration in the hope that the conservative approach will be adequately protective.
- Surrogate receptors were not used nor were extrapolations made to on-site receptors. On the other hand, this may not be drawback, as the conservative approach will protect the habitat of the most sensitive species.
- Basing discussions and findings on either EEQ or the 95%
   UL is founded upon the risk assessor's view of the relative toxicity of the various contaminants.
- Background soils were located in New Jersey and may not be representative of the area where the site is located.
- In most cases, the data shows generally an adequate sample population, but different sampling activitie: demonstrated different detection limits. The result is that some contaminants appear twice for the same media at

differing quantities. This risk assessment used the highest levels reported for calculating the 95% UL.

- The number of samples collected for some contaminants made use of the 95% UL unrealistic, as the calculated figure exceeds the maximum. In these cases, the maximum was used.
- The lack of congener-specific toxicity data makes any conclusions uncertain with regard to specific PCB congeners.
- The toxicity of PAHs in soils is not complete. The same can be said for furans and dioxins. Furthermore, the breakdown products of all these contaminants cloud the picture even more.

#### Interpretation:

The following discussion is an interpretation of the risk. As stated above, a conservative approach is used because of the lack of information on individual species and the fact that no tests were done on-site to establish toxicity to ecological receptors found in any of the habitats or media. In addition, the conservative approach considers any EEQ above 1 as representative of a potential for risk. The contaminants are discussed below in light of the hazards represented by the levels found in the particular media discussed.

#### Soils:

The values derived for courtyard soils are EEQs based upon the most conservative criteria available, primarily the lowest acute toxicity available; little information was found beyond laboratory rats and only one citation for plants. The only contaminant of concern in soil is Aroclor 1242; the LD<sub>(50)</sub> for rats was used in calculating the EEQ. Some information is available indicating impacts to ostrich ferns at 26 ppm.

Soils from outside the courtyard are contaminated with copper, lead, methylethyl ketone, and a phthalate. The calculated EEQs are all above the threshold of one for the maximum levels reported, but since the EEQs used here are based upon the 95% UL, none but methylethyl ketone is used.

#### Groundwater and leachate:

Since the RI did not include any information regarding groundwater discharge points (other than possibly that seeps are assumed to be groundwater discharge points), the conservative approach assumes that the worst case prevails and that impacts to water quality and sediment in the rip-rap sediment and the mudflats can be adverse

(as a result of groundwater discharge through seeps, springs, etc.). Organisms that use these seeps opportunistically may be subject to contaminant concentrations up to thousands of times higher than the toxicity reference dose. Predators would be comparably exposed via the food chain.

It is assumed that the rip-rap and mudflat sediment is contaminated with groundwater expressed as leachate. Since leachate is associated with seeps from groundwater sources, it can be assumed that the contaminants in groundwater will eventually be found in leachate and surface waters as well. Seep areas are regularly used by a broad range of ecological receptors that feed and propagate at such locations. All the contaminants of concern are potential sources of risk to those found in the habitat niches found at seeps and springs.

The metals identified in groundwater are present at levels that exceed the chronic toxicity AWQC. Assuming no attenuation, any seeps in the area from the site's groundwater will impact ecological receptors through either direct contact or indirectly through the food chain. The metals considered individually show exceedance of threshold risk levels.

The organic contaminants of groundwater show the same trends. Organic contaminants that are in the range of potential risk are the chlorinated hydrocarbons endosulfan and heptachlor and the PAH, phenanthrene.

Sediment:

The sediments of the mudflats (also covered in the NOAA efforts, but supplemented here) and the rip-rap area are known to be contaminated with PAHs. Contamination of these areas is serious due to the potential for impact to benthic and intertidal zone organisms found there that play a crucial role in the food chain. In addition, many opportunistic birds and mammals use the area on either a diurnal or seasonal basis. The investigation covered two areas of mudflats separated by an artificially determined dividing line. The two areas are called 'mudflats near the site' and 'mudflats beyond the site'. The area near the site shows higher levels of PAHs than the area beyond the site while the area beyond the site shows no PAHs, but elevated levels of PCB. Both areas are contaminated with chlorinated hydrocarbons associated with DDT.

Ecoreceptors (including a full range of species from macroinvertebrates through avian and mammalian receptors) are exposed to several PAHs and a chlorinated hydrocarbon. A concern arises over the impacts of PAHs on macroinvertebrates, as they have no known mechanism for degradation of them. These organisms are a highly important part of the food chain for such terrestrial receptors a raccoons, muskrats, herons (and other avian predators), as well as amphibians.

Sediments such as those found in the two mudflats areas are generally associated with zones of deposition where contaminants may accumulate to very high levels. Such areas can be either continuing or secondary sources of contamination to streams for several years even after the primary source is cleaned up. In addition, they serve as quiet areas that some organisms seek for refuge or feeding and reproduction, thus acting as areas where exposure is proportionately elevated above other areas associated with the site.

Surface Water:

The ecological risk assessment for surface water is assumed to be adequately covered in the NOAA effort, entitled "Draft Aquatic Ecological Risk Assessment for Metal Bank of America/Cottman Avenue NPL Site."

<u>Risk Assessment</u> (Conclusions):

On the individual contaminant basis, the ecological risk assessment shows that the potential exists for impact to ecological receptors. This assessment, based upon literature-derived values and calculations of EEQ concludes that the site is a source of contamination at levels that pose a potential risk to ecological receptors. These receptors are the resident flora and fauna as well as any migratory fauna using these media for habitat and opportunistic resting/feeding and propagation.

Some risk assessment procedures (see RAGS I) include the additive affects calculations. While literature references for the mix of contaminants and impacts to specific organisms on and near the site are unavailable, the risk assessor believes it is appropriate to include the potential for additive risk to the risk assessment. This is done by adding the EEQ values without consideration for proportionate impact levels, i.e., the values are added according to the formula:

Total Risk = Risk  $(COC^{6})$  + Risk  $(COC^{b})$  + Risk  $(COC^{c})$  + ...

Where COC is 'contaminant of concern' and ", ", etc. is specific contaminant level used the risk assessment, i.e., the 95% UL.

The calculations are carried out for each medium discussed below. If the additive risk exceeds 10 (except for chlorinated hydrocarbons -- due to their high BCF and food chain implications) the risk is considered to be potentially high. All calculations use the 95% UL levels found in the appropriate tables attached.

#### Ground Water:

No specific data is offered by the RI for discharges from groundwater to the streams, but the RI shows elevated site-related contaminants in the areas where seeps are expected, in the vicinity of riprap sediment sampling stations. (Aluminum and iron are not included because they are considered to be derived from the natural soils of the area.)

Contaminants	EEQ	Contaminants	EEQ
Cđ	3.61E+00	DDT	2.69E+01
Pb	1.15E+02	DDE	4.87E+02
Hg	2.51E+02	Endosulfan	4.91E+00
cī	1.25E+00	Phenanthrene	4.43E+00
Cr(VI)	2.69E+01	Heptachlor	4.26E+01
Cu	7.47E+00	_	
Zn	6.66E+00		

The potential for risk from any of these contaminants is recognized as high for ecological receptors.

Summing the EEQ values shows an additive risk of nearly 1000, exclusive of aluminum and iron. Therefore, the additive risk is above the factor of 10 and the potential for risk exists.

Soil:

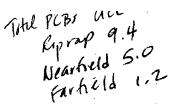
The potential additive risk for soils inside the courtyard is not calculated because no contaminant exceeds an EEQ of one. The soils outside the courtyard, however, are contaminated with methylethyl ketone at a level exceeding an EEQ of one, but that is the only contaminant exceeding this risk level, at 9.66 (an acute value). In addition, the risk is associated with soils deep in the profile and would likely be an exposure route only for deeply rooted vegetation.

#### Surface Water:

The additive potential for risk is also not calculated because the ecological risk assessment for this habitat was carried out by NOAA.

#### Mudflats:

Mudflats beyond the site are contaminated with PCB (Aroclor - 1248), and DDT-related contaminants. The only EEQ calculated is for the acutely toxic level.



# Contaminants EEQ

	PCB	13.04	(Aroclor - 1248)
.1.	4,4-DDD	114.77	•
234	4,4-DDD 4,4-DDE	32.50	•

The botential for additive risk in this area is calculated to be over 150. This is based upon the calculated risk to benthic fauna found in the sediment and not terrestrial receptors. The logic here is that impacts upon the lowest members of the food chain will be visited upon predators higher in the food chain. The loss of feeding opportunities (due to the reduction in productivity of the system) is a result of the contamination and, therefore, and ecological impact.

14

Mudflats near the site are contaminated with a variety of PAHs and a DDT-related compound. As with the other mudflats area, the only EEQ calculated is for the acute values.

Contaminants	EEQ	Contaminants	EEQ
4,4-DDD	235.0	Dibenz(a,h)anthracene	38.11
Benzo(a) anthracene	2.78	Fluoranthene	2.0
Benzo(a)pyrene	2.94	Phenanthrene	2.44
Chrysene	1.70	Pyrene	3.14

The potential additive risk is nearly 300.

Rip-rap sediments:

457

One PCB mixture and several PAHs are identified at risk levels greater than one.

Contaminants	EEQ 1.88E+02	Contaminants	EEQ
Aroclor-1260	111E+02-	Dibenz(a,h)anth'cene	2.90E+02
Acenaphthene	1.60E+03	Fluoranthene	4.62E+02
Anthracene	2.91E+03	Fluorene	6.75E+03
Benzo(a) an'cene	9.50E+02	2-me'naphathalene	1.67E+03
Benso(a)pyrene	4.19E+02	Naphthalene	7.06E+02
Chrysene	5.10E+02	Phenanthrene	2.43E+03

The potential for additive risk approaches 20,000.

Conclusion:

A clear potential is demonstrated by the above calculations for all media used in the risk assessment. It is also clear from the assessment that Rip-rap sediment, and mudflats near the site show the greatest potential for immediate risk on the basis that, a wide

AR300859

distribution of contamination to ecoreceptors is possible. In the case of rip-rap sediment contamination, it is considered to be serious from the view point that stationary flora are subject to impacts and resident as well as opportunistic fauna easily contact the medium.

It may be argued that groundwater risk is exaggerated because the RI fails to identify discharge points. But it was not readily apparent in the RI that either discharge points or attenuation have been demonstrated and in fact results from analyses of the rip-rap sediment (the most likely source is groundwater) show elevated levels of site related contaminants.

It is concluded that all media used in this risk assessment show a potential for risk as a result of the contamination associated with the site.

#### Recommendation:

The remediation plans should include consideration of all media for clean up, except surface soil unless removal of the in-place storage tank located in the open filed is carried out. All media except the surface soil outside the courtyard are contaminated to levels that are of potential risk and control of the sources of this contamination is needed. If tank removal is planned, then a dedicated assessment of the release of contaminants along with the restoration of the area should be planned as well. Both source control as well as remediation of contaminated media should be the subjects of the feasibility study.

- 1). Surface soils outside the courtyard should receive remedial attention only if soils are disturbed at a result of either remediation or construction activities. If remediation causes the subsurface soils to become accessible to terrestrial organisms, a 'spot-check' risk assessment should be completed.
- 2.) 'Hot spot' remediation along the river periphery should alleviate the potential for risks in this 'hot-spot' area. Prior to remediation, intensive sampling should be carried out to assure that remediation will remove or otherwise isolate all potentially toxic contamination.

If, on the other hand, either 'hot spot' removal or remediation is not carried out, then intensive monitoring (both chemical and biological) should be designed. This monitoring program should involve indiginous, opportunistic, and seasonal organisms.

•

.

METAL BANK - COTTMAN AVENUE

ECOLOGICAL EFFECTS QUOTIENT TABLES



. . .

#### TABLE 1 METAL BANK - COTTMAN AVENUE INSIDE COURTYARD SURFACE SOILS

COMPARISON WITH ECOTOXICOLOGICAL VALUES CONTAMINANT EEC (A) ACUTE EEQ OF POTENTIAL CONCERN (ppm) (ppm) EEQ ACUTE(C)				
UPPER 95% CONFIDENCE INTERVAL VALUES FOR SOILS COMPARISON WITH ECOTOXICOLOGICAL VALUES				

(A) EEC - Expected Environmental Concentration
 (B) Toxicological effect criteria concentration; appropriate chronic values not reported.
 (C) EEQ - Ecological Effects Quotient = EEC/Toxicological Effect Criteria Concentration

<sup>(1)</sup> Acute dermal LD<sub>(50)</sub> for rats (USEPA, 1980).

#### TABLE 2 METAL BANK - COTTMAN AVENUE INSIDE COURTYARD SURFACE SOILS

MAXIMUM VALUES FOR SOILS COMPARISON WITH ECOTOXICOLOGICAL VALUES				
CONTAMINANT OF POTENTIAL CONCERNEEC (^)ACUTE VALUE (II) 				
Aroclor - 1242 70.000 8650 <sup>(1)</sup> 8.09E - 03				

(A) EEC - Expected Environmental Concentration

(5) Toxicological effect criteria concentration; appropriate chronic values not reported.

(C) EEQ - Ecological Effects Quotient = EEC/Toxicological Effect Criteria Concentration

AR300863

(1) Acute dermal LD<sub>(50)</sub> for rats (USEPA, 1980).

#### TABLE 3 METAL BANK - COTTMAN AVENUE OUTSIDE COURTYARD SURFACE SOILS

UPPER 95% CONFIDENCE INTERVAL VALUES FOR SOILS COMPARISON WITH ECOTOXICOLOGICAL VALUES				
CONTAMINANT OF POTENTIAL CONCERN	EEC <sup>(A)</sup> (ppm)	ACUTE VALUE (*) (ppm)	EEQ ACUTE <sup>(C)</sup>	
Aluminum Arsenic Beryllium Cobait Mercury Nickel Zinc Fluoranthene	18.396 0.006 0.001 0.018 0.0002 0.025 0.103 0.073	2 <sup>(1)</sup> 50 <sup>(1)</sup> 50 <sup>(7)</sup> 255 <sup>(7)</sup> 250 <sup>(4)</sup> 80 <sup>(7)</sup> 500 <sup>(3)</sup> 3180 <sup>(4)</sup>	9.20E+00 1.20E-04 2.00E-05 7.16E-04 7.04E-07 3.11E-04 2.06E-04 2.30E-05	

(A) EEC - Expected Environmental Concentration

(\*) Toxicological effect criteria concentration; appropriate chronic values not reported.

(5) EEQ - Ecological Effects Quotient = EEC/Toxicological Effect Criteria Concentration

<sup>(1)</sup> Growth supression noted in very sensitive plants (USEPA, 1983).

- (7) Corresponds to the soil accumulation levels when the upper limit of recommended concentration in irrigation water is applied; level at which phytotoxic effects are noted (USEPA, 1983).
- (3) Lowest concentration at which phytotoxic effects are noted (USEPA, 1983).
- (4) Reduced yield of oat (Avena sativa) (USEPA, 1983).
- <sup>(5)</sup> Level at which yield reduction was noted in select species (USEPA, 1983).
- (9) Acute dermal LD<sub>(30)</sub> for rabbits (USEPA, 1960).

#### TABLE 4 METAL BANK - COTTMAN AVENUE OUTSIDE COURTYARD SURFACE SOILS

MAXIMUM VALUES FOR SOILS COMPARISON WITH ECOTOXICOLOGICAL VALUES				
CONTAMINANT OF POTENTIAL CONCERNEEC (A) (ppm)ACUTE 				
Aluminum Arsenic Beryllium Cobalt Mercury Nickel Zinc Fluoranthene	20.6000 0.0068 0.0011 0.0198 0.0002 0.0272 0.1415 2.7000	2 <sup>(1)</sup> 50 <sup>(1)</sup> 50 <sup>(2)</sup> 25 <sup>(3)</sup> 250 <sup>(4)</sup> 80 <sup>(3)</sup> 500 <sup>(3)</sup> 3180 <sup>(9)</sup>	1.03E+01 1.36E-04 2.20E-05 7.92E-04 8.80E-07 3.40E-04 2.83E-04 8.49E-04	

(A) EEC - Expected Environmental Concentration

(\*) Toxicological effect criteria concentration; appropriate chronic values not reported.

(9) EEQ - Ecological Effects Quotient = EEC/Toxicological Effect Criteria Concentration

<sup>(1)</sup> Growth supression noted in very sensitive plants (USEPA, 1983).

(3) Corresponds to the soil accumulation levels when the upper limit of recommended concentration in irrigation water is applied; level at which phytotoxic effects are noted (USEPA, 1983).

- <sup>(3)</sup> Lowest concentration at which phytotoxic effects are noted (USEPA, 1983).
- (4) Reduced yield of oat (Avena sativa) (USEPA, 1983).
- (5) Level at which yield reduction was noted in select species (USEPA, 1983).
- (6) Acute dermal LD<sub>(50)</sub> for rabbits (USEPA, 1980).

#### TABLE 5 METAL BANK - COTTMAN AVENUE OUTSIDE COURTYARD SUBSURFACE SOILS

UPPER 95% CONFIDENCE INTERVAL VALUES FOR SOILS COMPARISON WITH ECOTOXICOLOGICAL VALUES				
CONTAMINANT OF POTENTIAL	EEC (A)	ACUTE VALUE (5)	EEQ ACUTE <sup>(C)</sup>	
CONCERN	(ppm)	(mqq)		
Aluminum	15.4390	2 (1)	7.72E+00	
Arsenic	0.0114	50 <sup>(1)</sup>	2.27E-04	
Beryllium	0.0017	50 <sup>(2)</sup>	3.35E-05	
Cadmium	0.0092	1 (3)	9.17E-03	
Chromium	0.4949	30 <sup>(4)</sup>	1.65E-02	
Cobalt	0.0845	25 <sup>(3)</sup>	3.38E-03	
Copper	15.1327	70 (3)	2.16E-01	
Lead	30.4373	200 <sup>(3)</sup>	1.52E-01	
Manganese	1.3570	65 <sup>(3)</sup>	2.09E-02	
Mercury	0.0016	<sup>(3)</sup> 250	6.58E-06	
Nickel	0.2049	80 ()	2.56E-03	
Zinc	2.3672	500 <sup>(3)</sup>	4.73E-03	
Acetone	7.8007	20000 (7)	3.90E-04	
Arocior-1242	219.4475	8650 <sup>(8)</sup>	2.54E-02	
2-Butanone	96.5833	10 🕅	9.66E+00	
4,4'-DDT	0.5561	300 <sup>(19)</sup>	1.85E-03	
Di-n-butylphthalate	10.4427	20 (11)	5.22E-01	
Endosulfan I	0.0585	35000 <sup>(12)</sup>	1.67E-06	
Ethylbenzene	200.1648	15415 <sup>(L3)</sup>	1.30E-02	
Fluoranthene	73.8464	3180 (14)	2.32E-02	

(A) EEC - Expected Environmental Concentration

(B) Toxicological effect criteria concentration; appropriate chronic values not reported.

(C) EEQ - Ecological Effects Quotient = EEC/Toxicological Effect Criteria Concentration

(1) Growth supression noted in very sensitive plants (USEPA, 1983).

(2) Corresponds to the soil accumulation levels when the upper limit of recommended concentration in irrigation water is applied; level at which phytotoxic effects are noted (USEPA, 1983).

AR300866

- (3) Lowest concentration at which phytotoxic effects are noted (USEPA, 1983)
- (\*) Lowest acute dermai LD<sub>50</sub> value reported for rabbits (ATSDR, 1991).

(9) Reduced yield of oat (Avena sativa) (USEPA, 1983).

(9) Level at which yield reduction was noted in select species (USEPA, 1983).

(7) Acute dermal LD<sub>(39)</sub> for rabbits (Little, 1987).

- (6) Acute dermal LD (30) for rats (USEPA, 1980).
- (7) Acute dermai LD<sub>30</sub> for rabbits (ATSDR, 1990).
- (10) Acute dermai LD<sub>5</sub> for rabbits (Cameron and Burgess, 1945).
- (11) Acute dermal LD<sub>(39)</sub> for rabbits (USEPA, 1980).
- <sup>(12)</sup> Lowest reported level at which phytotoxic effects are noted (WHO, 1984).
- (13) Acute dermal LD<sub>(S9)</sub> for rabbits (ATSDR, 1990).
- (14) Acute dermai LD<sub>(Sen</sub> for rabbits (USEPA, 1980).

#### TABLE 6 METAL BANK – COTTMAN AVENUE OUTSIDE COURTYARD SUBSURFACE SOILS

MAXIMUM VALUES FOR SOILS COMPARISON WITH ECOTOXICOLOGICAL VALUES				
CONTAMINANT OF POTENTIAL CONCERN	EEC (^) (ppm),	ACUTE VALUE <sup>(B)</sup> (ppm)	EEQ ACUTE <sup>(C)</sup>	
			1	
Aluminum	37.5000	2 (1)	1.88E+01	
Arsenic	0.0211	50 <sup>(1)</sup>	4.22E-04	
Beryllium	0.0082	50 (2)	1.63E-04	
Cadmium	0.0310	1 (3)	3.10E-02	
Chromium	2.7300	· 30 <sup>(4)</sup>	9.10E-02	
Cobait	0.5670	25 <sup>(3)</sup>	2.27E-02	
Copper	88.3000	70 <sup>(3)</sup>	1.26E+00	
Lead	227.0000	200 (3)	1.13E+00	
Manganese	5.0100	65 <sup>(3)</sup>	7.71E-02	
Mercury	0.0105	<sup>(3)</sup> 250	4.20E-05	
Nickel	0.7050	80 (9)	8.81E-03	
Zinc	11.8000	500 (3)	2.36E-02	
Acetone	6.0000	20000 (7)	3.00E-04	
Arocior-1242	503.0000	8650 (4)	5.82E-02	
2-Butanone	74.0000	10 (7)	7.40E+00	
4,4'-DDT	0.9500	300 (10)	3.17E-03	
Di-n-butylphthalate	23.5000	20 (11)	1.18E+00	
Endosulfan I	0.2280	35000 (12)	6.51E-06	
	150,0000	15415 <sup>(L3)</sup>	9.73E-03	
Ethylbenzene		3180 (14)		
Fluoranthene	220.0000	3100 (4)	6.92E-02	

(A) EEC – Expected Environmental Concentration

<sup>(B)</sup> Toxicological effect criteria concentration; appropriate chronic values not reported.

(C) EEQ - Ecological Effects Quotient = EEC/Toxicological Effect Criteria Concentration

<sup>(1)</sup> Growth supression noted in very sensitive plants (USEPA, 1983).

(2) Corresponds to the soil accumulation levels when the upper limit of recommended concentration in irrigation water is applied; level at which phytotoxic effects are noted (USEPA, 1983).

- (3) Lowest concentration at which phytotoxic effects are noted (USEPA, 1983)
- (4) Lowest acute dermal LD<sub>ge</sub> value reported for rabbits (ATSDR, 1991).
- (5) Reduced yield of oat (Avena sativa) (USEPA, 1983).
- (9) Level at which yield reduction was noted in select species (USEPA, 1983).
- Acute dermal LD<sub>(S0)</sub> for rabbits (Little, 1987).
- (5) Acute dermal LD<sub>(50)</sub> for rats (USEPA, 1980).
- (\*) Acute dermai LD<sub>30</sub> for rabbits (ATSDR, 1990).
- (10) Acute dermal LD<sub>30</sub> for rabbits (Cameron and Burgess, 1945).
- (11) Acute dermal LD<sub>(SP)</sub> for rabbits (USEPA, 1980).
- <sup>(12)</sup> Lowest reported level at which phytotoxic effects are noted (WHO, 1984).
- (13) Acute dermal LD<sub>(SP)</sub> for rabbits (ATSDR, 1990).
- <sup>(14)</sup> Acute dermal LD<sub>(59)</sub> for rabbits (USEPA, 1980).

# TABLE 7 METAL BANK – COTTMAN AVENUE MUDFLATS NEAR THE SITE

UPPER 95% CONFIDENCE INTERVAL VALUES FOR SEDIMENT COMPARISON WITH ECOTOXICOLOGICAL VALUES				
CONTAMINANT OF POTENTIAL CONCERN	EEC <sup>(A)</sup> (ppb)	ACUTE VALUE <sup>(B)</sup> (ppb)	EEQ ACUTE <sup>(C)</sup>	
4,4–DDD Benzo(a)anthracene Benzo(a)pyrene Chrysene Dibenz(a,h)anthracene Fluoranthene Phenanthrene Pyrene	365 470 4575 640 27481,177 4209 680 22022,286 32491,200 12,24950 13,7651,100	2 <sup>(1)</sup> 230 <sup>(1)</sup> 400 <sup>(1)</sup> 400 <sup>(1)</sup> 60 <sup>(1)</sup> 600 <sup>(1)</sup> 225 <sup>(1)</sup> 350 <sup>(1)</sup>	235.00 2.78 2.94 1.70 38.11 2.00 2.44 3.14	182.5 19.9 7.12 10.52 36.7 13.8 54.44 30.76

(A) EEC – Expected Environmental Concentration

(B) Toxicological effect criteria concentration.

(C) EEQ - Ecological Effects Quotient = EEC/Toxicological Effect Criteria Concentration

<sup>(1)</sup> NOAA ER-L, NOAA 1990.

356.77

Nearfield PCBs = 100 (5000/50) (45

# TABLE 8 METAL BANK – COTTMAN AVENUE MUDFLATS NEAR THE SITE

MAXIMUM VALUES FOR SEDIMENT COMPARISON WITH ECOTOXICOLOGICAL VALUES				
CONTAMINANT OF POTENTIAL CONCERNEEC (A) (ppb)ACUTE 				
4,4–DDD Benzo(a)anthracene Benzo(a)pyrene Chrysene Dibenz(a,h)anthracene Fluoranthene Phenanthrene Pyrene	470 640 540 680 7,000 1,200 550 1,100	2 (1) 230 (1) 400 (1) 400 (1) 600 (1) 600 (1) 225 (1) 350 (1)	235.00 2.78 1.35 1.70 116.67 2.00 2.44 3.14	

(A) EEC – Expected Environmental Concentration

(B) Toxicological effect criteria concentration.

(C) EEQ - Ecological Effects Quotient = EEC/Toxicological Effect Criteria Concentration

<sup>(1)</sup> NOAA ER-L, NOAA 1990.

#### TABLE 9 METAL BANK - COTTMAN AVENUE MUDFLATS BEYOND SITE

UPPER 95% CONFIDENCE INTERVAL VALUES FOR SEDIMENT COMPARISON WITH ECOTOXICOLOGICAL VALUES						
CONTAMINANT OF POTENTIAL CONCERN	EEC <sup>(4)</sup> (ppb)	ACUTE VALUE ( <sup>a</sup> ) (ppb)	CHRONIC VALUE <sup>(3)</sup> (ppb)	EEQ ACUTE <sup>(6)</sup>	EEQ CHRONIC <sup>(O)</sup>	
Beryllium	0.826	130 (1,2)	5 (1,2)	0.01	0.16	
Cadmium	1.64	5,000 (3)		0.00		
Chromium	35.8	80,000 <sup>(3)</sup>		0.00		
Copper	68.1	70,000 <sup>(3)</sup>		0.00		
Lead	160	35,000 (3)		0.00		
Mercury	0.554	150 (3)		0.00		
Nickel	22.6	:0,000 <sup>(3)</sup>		0.00		
Zinc	367	120,000 (3)		24 13.04		
Aroclor-1248	1200 652	50 (3.4)				
4,4-DDD	212-229.5	2 <sup>(3)</sup>	·	106114.77		
4,4-DDE	2-08 65	2 <sup>(3)</sup>		104 32.50		

(A) EEC – Expected Environmental Concentration

(\*) Toxicological effect criteria concentration.

(C) EEQ - Ecological Effects Quotient = EEC/Toxicological Effect Criteria Concentration

(1) The concentrations of a chemical in the sediment and water column are assumed to be in equilibrium and the concentrations of chemicals noted for sediment are reflective of chemical concentrations in interstitial spaces. Thus, sedument analytical data have been directly compared to published aquatic criteria and ecotoxicological values. This approach was utilized as it would be protective of sessile benthic invertebrates which would be in direct contact with and exposed to contaminants found in both the sediment and the water column.

(2) IRIS lowest effect level (LEL) value.

(3) NOAA ER-L, NOAA 1990.

(4) Value for Total PCBs.

AR300870

234

#### TABLE 10 METAL BANK - COTTMAN AVENUE MUDFLATS BEYOND SITE

MAXIMUM VALUES FOR SEDIMENT COMPARISON WITH ECOTOXICOLOGICAL VALUES						
CONTAMINANT OF POTENTIAL CONCERN	EEC (A) (ppb)	ACUTE VALUE ( <sup>a</sup> ) (ppb)	CHRONIC VALUE ( <sup>III)</sup> (ppb)	EEQ ACUTE <sup>(C)</sup>	EEQ CHRONIC <sup>(C)</sup>	
Beryllium Cadmium Chromium Copper Lead Mercury Nickel Zinc Aroclor-1248 4,4-DDD 4,4-DDE	0.826 1.64 35.8 68.1 160 0.554 22.6 367 1,200 310 65	130 ( <sup>1,2</sup> ) 5,000 ( <sup>3</sup> ) 80,000 ( <sup>3</sup> ) 70,000 ( <sup>3</sup> ) 35,000 ( <sup>3</sup> ) 150 ( <sup>3</sup> ) 30,000 ( <sup>3</sup> ) 120,000 ( <sup>3</sup> ) 50 ( <sup>3,4</sup> ) 2 ( <sup>3</sup> ) 2 ( <sup>3</sup> )	5 (1.2)         	0.01 0.00 0.00 0.00 0.00 0.00 0.00 24.00 155.00 32.50	0.16	

(A) EEC - Expected Environmental Concentration

(\*) Toxicological effect criteria concentration.

(9) EEQ - Ecological Effects Quotient = EEC/Toxicological Effect Criteria Concentration

<sup>1)</sup> The concentrations of a chemical in the sediment and water column are assumed to be in equilibrium and the concentrations of chemicals noted for sediment are reflective of chemical concentrations in interstitial spaces. Thus, sedument analytical data have been directly compared to published aquatic criteria and ecotoxicological values. This approach was utilized as it would be protective of sessile benthic invertebrates which would be in direct contact with and exposed to contaminants found in both the sediment and the water column.

AR300871

<sup>(2)</sup> IRIS lowest effect level (LEL) value.

<sup>(3)</sup> NOAA ER-L, NOAA 1990.

(4) Value for Total PCBs.

#### TABLE 11 METAL BANK - COTTMAN AVENUE RIVER SEDIMENTS

UPPER 95% CONFIDENCE INTERVAL VALUES FOR SEDIMENT COMPARISON WITH ECOTOXICOLOGICAL VALUES						
CONTAMINANT OF POTENTIAL CONCERN	EEC <sup>(4)</sup> (ppb)	ACUTE VALUE ( <sup>a</sup> ) (ppb)	CHRONIC VALUE (#) (ppb)	EEQ ACUTE <sup>(5)</sup>	EEQ CHRONIC <sup>(9)</sup>	
Beryllium	1.347	130 <sup>(L,2)</sup>	(تـبا) ج	0.01	0.25	
Mercury	0.459	150 (3)		0.00		
Aroclor-1248	1,541	50 <sup>(3,4)</sup>		30.82		
Aroclor-1254	2484.144	50 (3.4)		49.68		
4,4-DDD	242	2 (3)		120.86		
4,4-DDE	243	2 (3)		121.38		
Acenaphthene	7,825	150 <sup>(3)</sup>		52.17		
Anthracene	7,887	85 <sup>(3)</sup>		92.79		
Benzo(a)anthracene	10,039	230 <sup>(3)</sup>		43.65		
Benzo(a)pyrene	7,161	400 <sup>(3)</sup>		17.90		
Chrysene	8,967	400 <sup>(3)</sup>		22.42		
Dibenz(a,h)anthracene	758	60 <sup>(3)</sup>		12.63		
Di-n-octylphthalate	2,452	6 (1,5)		396.79		
Fluoranthene	23,391	600 <sup>(3)</sup>		38.99		
Fluorene	12,745	35 <sup>(3)</sup>		364.13	·	
2-methyinaphthalene	8,263	65 <sup>(3)</sup>		127.13		
Naphthalene	1,199	340 (3)	]	3. <b>53</b>		
Phenanthrene	40,044	225 (3)		177.97		
Pyrene	29,814	350 <sup>(3)</sup>		85.18		

(A) EEC - Expected Environmental Concentration

(\*) Toxicological effect criteria concentration.

(C) EEQ - Ecological Effects Quotient = EEC/Toxicological Effect Criteria Concentration

(1) The concentrations of a chemical in the sediment and water column are assumed to be in equilibrium and the concentrations of chemicals noted for sediment are reflective of chemical concentrations in interstitial spaces. Thus, sedument analytical data have been directly compared to published aquatic criteria and ecotoxicological values. This approach was utilized as it would be protective of sessile benthic invertebrates which would be in direct contact with and exposed to contaminants found in both the sediment and the water column.

(2) IRIS lowest effect level (LEL) value.

- (3) NOAA ER-L, NOAA 1990.
- (4) Value for Total PCBs.

(5) Lowest reported LC<sub>30</sub> for fish (7-day LC<sub>30</sub> for Redear sunfish) (Birge, 1980).

#### TABLE 12 METAL BANK - COTTMAN AVENUE RIVER SEDIMENTS

MAXIMUM VALUES FOR SEDIMENT COMPARISON WITH ECOTOXICOLOGICAL VALUES						
CONTAMINANT OF POTENTIAL CONCERN	EEC (4) (ppb)	ACUTE VALUE ( <sup>a)</sup> (ppb)	CHRONIC VALUE (#) (ppb)	EEQ ACUTE <sup>(9)</sup>	EEQ CHRONIC <sup>(9)</sup>	
Beryllium Mercury Aroclor-1248 Aroclor-1254 4,4-DDD 4,4-DDE Acenaphthene Anthracene Benzo(a)anthracene Benzo(a)pyrene Chrysene Dibenz(a,h)anthracene Dibenz(a,h)anthracene Di-n-octylphthalate Fluorene 2-methylnaphthalene	0.50 0.220 2,300 2,595 315 315 9,200 9,100 11,000 7,900 10,000 750 2,150 26,000 15,000 9,800	130 (1.2) 150 (3) 50 (3.9) 2 (3) 2 (3) 150 (3) 85 (3) 230 (3) 400 (3) 400 (3) 60 (3) 6 (1.5) 600 (3) 35 (3) 85 (3)	5 (1,2)          -	0.00 0.00 46.00 51.90 157.50 61.33 107.06 47.83 19.75 25.00 12.50 347.90 43.33 428.57 150.77	0.09	
Naphthalene Phenanthrene Pyrene	1,200 46,000 34,000	340 <sup>(3)</sup> 225 <sup>(3)</sup> 350 <sup>(3)</sup>		3.53 204.44 97.14		

(A) EEC - Expected Environmental Concentration

(B) Toxicological effect criteria concentration.

(C) EEQ - Ecological Effects Quotient = EEC/Toxicological Effect Criteria Concentration

(1) The concentrations of a chemical in the sediment and water column are assumed to be in equilibrium and the concentrations of chemicals noted for sediment are reflective of chemical concentrations in interstitial spaces. Thus, sedument analytical data have been directly compared to published aquatic criteria and ecotoxicological values. This approach was utilized as it would be protective of sessile benthic invertebrates which would be in direct contact with and exposed to contaminants found in both the sediment and the water column.

AR300873

(2) IRIS lowest effect level (LEL) value.

() NOAA ER-L, NOAA 1990.

(4) Value for Total PCBs.

(5) Lowest reported LC<sub>30</sub> for fish (7-day LC<sub>30</sub> for Redear sunfish) (Birge, 1980).

#### TABLE 13 METAL BANK - COTTMAN AVENUE RIVER SEDIMENTS - LESS THAN 6"

UPPER 95% CONFIDENCE INTERVAL VALUES FOR SEDIMENT COMPARISON WITH ECOTOXICOLOGICAL VALUES						
CONTAMINANT OF POTENTIAL CONCERN	EEC <sup>(A)</sup> (ppb)	ACUTE VALUE (®) (ppb)	CHRONIC VALUE (P) (ppb)	EEQ ACUTE <sup>(C)</sup>	EEQ CHRONIC <sup>(9)</sup>	
Beryllium	1.35	130.00 <sup>(1,2)</sup>	5.30 (1.2)	1.04E-02	2.54E-01	
Mercury	0.46	150.00 (3)		3.06E-03		
Aroclor-1248	2154.00	50.00 (3.4)		4.31E+01		
Aroclor-1260	2369.70	50.00 (3.4)		4.74E+01		
4.4-DDD	241.71	2.00 (5)		1.21E+02		
4.4-DDE	242.76	2.00 (5)		1.21E+02		
Acenaphthene	7825.37	150.00 (3)		5.22E+01		
Anthracene	7887.03	85.00 (3)		9,28E+01		
Benzo(a)anthracene	10038.58	230.00 (3)		4.36E+01		
Benzo(a)pyrene	7160.99	400.00 (5)		1.79E+01		
Chrysene	8967.22	400.00 (5)		2.24E+01		
Dibenz(a,h)anthracene	757.72	60.00 <sup>(3)</sup>		1.26E+01		
Fluoranthene	3400.00	600.00 <sup>(3)</sup>		5.67E+00		
Fluorene	690.00	35.00 (3)		1.97E+01		
2-methyinaphthalene	8263.21	65,00 <sup>(3)</sup>		1.27E+02	l '	
Naphthalene	1198.93	340.00 (5)		3.53E+00	1	
henanthrene	40043.65	225.00 (3)		1.78E+02	·	
Pyrene	29814.41	350.00 (3)	[	8.52E+01		

(A) EEC - Expected Environmental Concentration

(F) Toxicological effect criteria concentration.

(C) EEQ - Ecological Effects Quotient = EEC/Toxicological Effect Criteria Concentration

(1) The concentrations of a chemical in the sediment and water column are assumed to be in equilibrium and the concentrations of chemicals noted for sediment are reflective of chemical concentrations in interstitial spaces. Thus, sedument analytical data have been directly compared to published aquatic criteria and ecotoxicological values. This approach was utilized as it would be protective of sessile benthic invertebrates which would be in direct contact with and exposed to contaminants found in both the sediment and the water column.

(7) IRIS lowest effect level (LEL) value.

(3) NOAA ER-L, NOAA 1990.

(4) Value for Total PCBs.

### TABLE 14 METAL BANK - COTTMAN AVENUE RIVER SEDIMENTS - LESS THAN 6"

### MAXIMUM VALUES FOR SEDIMENT COMPARISON WITH ECOTOXICOLOGICAL VALUES

CONTAMINANT OF POTENTIAL	EEC (4)	ACUTE VALUE (**)	CHRONIC VALUE (*)	EEQ ACUTE <sup>(C)</sup>	EEQ CHRONIC <sup>(9)</sup>
CONCERN	(ppb)	(ppb)	(ppb)		
Beryllium	0.50	130.00 <sup>(1,2)</sup>	5.30 <sup>(1,2)</sup>	3.85E-03	9.43E-02
Mercury	0.22	150.00 <sup>(3)</sup>	1	1.47E-03	~
Aroclor-1248	4600.00	50.00 (3,4)	· ]	9.20E+01	)
Aroclor-1260	3150.00	50.00 (3,4)		6.30E+01	
4,4-DDD	315.00	2.00 (5)	]	1.58E+02	
4,4-DDE	315.00	2.00 (3)		1.58E+02	
Acenaphthene	9200.00	150.00 (3)		6.13E+01	
Anthracene	9100.00	85.00 <sup>(5)</sup>	· ]	1.07E+02	
Benzo(a)anthracene	11000.00	230.00 <sup>(5)</sup>		4.78E+01	
Benzo(a)pyrene	7900.00	400.00 <sup>(5)</sup>	·	1.98E+01	
Chrysene	10000.00	400.00 (5)	]	2.50E+01	
Dibenz(a,h)anthracene	750.00	60.00 <sup>(3)</sup>		1.25E+01	
Fluoranthene	3400.00	600.00 <sup>(5)</sup>		5.67E+00	
Fluorene	690.00	35.00 (5)		1.97E+01	·
2-methyinaphthalene	9600.00	65.00 <sup>(3)</sup>		1.51E+02	
Naphthalene	1200.00	340.00 (3)		3.53E+00	
Phenanthrene	45000.00	225.00 (3)		2.04E+02	
Pyrene	34000.00	350.00 <sup>(3)</sup>		9.71E+01	

(A) EEC - Expected Environmental Concentration

(\*) Toxicological effect criteria concentration.

(9) EEQ - Ecological Effects Quotient = EEC/Toxicological Effect Criteria Concentration

(1) The concentrations of a chemical in the sediment and water column are assumed to be in equilibrium and the concentrations of chemicals noted for sediment are reflective of chemical concentrations in interstitial spaces. Thus, sedument analytical data have been directly compared to published aquatic criteria and ecotoxicological values. This approach was utilized as it would be protective of sessile benthic invertebrates which would be in direct contact with and exposed to contaminants found in both the sediment and the water column.

(2) IRIS lowest effect level (LEL) value.

(3) NOAA ER-L, NOAA 1990.

(4) Value for Total PCBs.

# TABLE 15 METAL BANK – COTTMAN AVENUE RIPRAP SEDIMENTS – LESS THAN 6

UPPER 95% CONFIL COMPARISON		VALUES FOR SEDI	
CONTAMINANT OF POTENTIAL CONCERN	EEC <sup>(A)</sup> (ppb)	ACUTE VALUE <sup>(B)</sup> (ppb)	EEQ ACUTE <sup>(C)</sup>
Aroclor – 1260 Acenaphthene Anthracene Benzo(a)anthracene Benzo(a)pyrene Chrysene Dibenz(a,h)anthracene Fluoranthene Fluorene 2 – methylnaphthalene Naphthalene Phenanthrene	5566.28 239973.25 247283.23 218556.61 167439.70 204109.17 17383.75 276970.22 236318.23 108393.26 239973.25 547214.50	$\begin{array}{c} 50.00 \ (^{1,2)} \\ 150.00 \ (^{1)} \\ 85.00 \ (^{1)} \\ 230.00 \ (^{1)} \\ 400.00 \ (^{1)} \\ 400.00 \ (^{1)} \\ 60.00 \ (^{1)} \\ 600.00 \ (^{1)} \\ 35.00 \ (^{1)} \\ 65.00 \ (^{1)} \\ 340.00 \ (^{1)} \\ 225.00 \ (^{1)} \end{array}$	1.11E+02 1.60E+03 2.91E+03 9.50E+02 4.19E+02 5.10E+02 2.90E+02 4.62E+02 6.75E+03 1.67E+03 7.06E+02 2.43E+03

(A) EEC – Expected Environmental Concentration

(B) Toxicological effect criteria concentration.

(C) EEQ - Ecological Effects Quotient = EEC/Toxicological Effect Criteria Concentration

<sup>(1)</sup> NOAA ER-L, NOAA 1990.

<sup>(2)</sup> Value for Total PCBs.

# TABLE 16 METAL BANK - COTTMAN AVENUE **RIPRAP SEDIMENTS - LESS THAN 6**\*

MAXIMUM VALUES FOR SEDIMENT COMPARISON WITH ECOTOXICOLOGICAL VALUES							
CONTAMINANT OF POTENTIAL CONCERN	EEC <sup>(A)</sup> (ppb)	ACUTE VALUE <sup>(B)</sup> (ppb)	EEQ ACUTE <sup>(C)</sup>				
Aroclor – 1260 Acenaphthene Anthracene Benzo(a)anthracene Benzo(a)pyrene Chrysene Dibenz(a,h)anthracene Fluoranthene Fluorene Methylnaphthalene,2– Naphthalene Phenanthrene	6500.00 67000.00 69000.00 60000.00 46000.00 56000.00 6100.00 76000.00 66000.00 31000.00 67000.00 150000.00	$\begin{array}{c} 50.00 \ (1,2) \\ 150.00 \ (1) \\ 85.00 \ (1) \\ 230.00 \ (1) \\ 400.00 \ (1) \\ 400.00 \ (1) \\ 60.00 \ (1) \\ 600.00 \ (1) \\ 35.00 \ (1) \\ 65.00 \ (1) \\ 340.00 \ (1) \\ 225.00 \ (1) \end{array}$	1.30E+02 4.47E+02 8.12E+02 2.61E+02 1.15E+02 1.40E+02 1.02E+02 1.27E+02 1.89E+03 4.77E+02 1.97E+02 6.67E+02				

(A) EEC - Expected Environmental Concentration
 (B) Toxicological effect criteria concentration.
 (C) EEQ - Ecological Effects Quotient = EEC/Toxicological Effect Criteria Concentration

<sup>(1)</sup> NOAA ER-L, NOAA 1990.

<sup>(2)</sup> Value for Total PCBs.

#### TABLE 17 METAL BANK - COTTMAN AVENUE

CONTAMINANT OF POTENTIAL	EEC (A)	ACUTE CRITERIA (*)	CHRONIC CRITERIA	EEQ ACUTE	EEQ CHRONIC
CONCERN	(ug/l}	(ug/l)	(ug/l)		
Acenaphthene	11.6270	1700.00 (1)	520.00 <sup>(1)</sup>	6.84E-03	2.24E-02
Aluminum	15483.4600		87.00		1.78E+02
Arsenic(III)	24.6080	360.00	190.00	6.84E-02	1.30E-01
Benzene	18.3130	5300.00 (1)	1	3.46E-03	
Beryllium	2.2350	130.00 (1)	5. <b>30</b> <sup>(1)</sup>	1.72E-02	4.22E-01
Cadmium	4.0960	3.92 <sup>(3)</sup>	1.15 (3)	1.04E+00	3.61E+00
Chloride	287329.1560	860000.00	230000.00	3.34E-01	1.25E+00
Chromium(VI)	82.1530	16.00	11.00	5.13E+00	7.47E+00
Copper	317.7760	17.78 <sup>(3)</sup>	11.82 <sup>(3)</sup>	1.79E+01	2.69E+01
DDT	0.4870	1.10	0.001	4.43E-01	4.87E+02
DDE (DDT metabolite)	5.0340	1050.00 (1)		4.79E-03	
Dimethyl phenol, 2,4–	171.3520	2120.00 (1)		8.08E-02	
Endosulfan	0.2750	0.22	0.06	1.25E+00	4.91E+00
Ethylbenzene	211.2590	\$2000.00 <sup>(1)</sup>	~-	6.60E-03	
Fluoranthene	17.7660	3980.00 (1)		4.46E-03	
Heptachlor	0.1620	0.52	0.004	5.12E-01	4.26E+01
Iron	62355.7850		, 1000.00		6.24E+01
Lead ·	365.7080	81.65 <sup>(3)</sup>	5.18 <sup>(3)</sup>	4.48E+00	1.15E+02
Mercury	3.0170	2.40	0.01	1.26E+00	2.51E+0
Naphthalene	75.1570	2300.00 (1)	620.00 <sup>(1)</sup>	3.27E-02	1.21E-
Nickel	69.0160	1418.24 <sup>(3)</sup>	1 <b>57.67</b> <sup>(3)</sup>	4.87E-02 ·	4.38E-
Phenanthrene	27.9390	<b>30.00</b> <sup>(2)</sup>	6.30 <sup>(2)</sup>	9.31E-01	4.43E+00
Phenol	57.1800	10200.00 (1)	2560.00 <sup>(1)</sup>	5.61E-03	2.23E-02
Selenium	2.4290	20.00	5.00	1.21E-01	4.86E-01
Tetrachloroethylene	56.4280	5280.00 <sup>(1)</sup>		1.07E-02	6.72E-02
Zinc	708.4010	117.02 (3)	105.99 (3)	6.05E+00	6.68E+00

(A) EEC - Expected Environmental Concentration

(B) Toxicological effect criteria concentration. Values are EPA Water Quality Criteria for the protection of aquatic life and their uses.

(1) Insufficient data to develop criteria. Value presented is the LO.E.L-Lowest Observed Effect Level.

(2) Proposed criterion

(3) Hardness dependent criteria (100 mg/L CaCO<sub>3</sub> used)

(4) pH dependent criteria (7.8 pH used)

### TABLE 18 METAL BANK - COTTMAN AVENUE

MAXIMUM VALUES FOR GROUNDWATER COMPARISON WITH EPA WATER QUALITY CRITERIA (FRESHWATER)						
CONTAMINANT OF POTENTIAL CONCERN	EEC (^) (ug/l)	ACUTE CRITERIA (*) (ug/l)	CHRONIC CRITERIA (11) (ug/1)	EEQ ACUTE	EEQ CHRONIC	
· · · · · · · · · · · · · · · · · · ·			,			
Acenaphthene	38.0000	1700.00 (1)	520.00 <sup>(1)</sup>	2.24E-02	7.31E-02	
Aluminum	60400.0000		87.00	·	6.94E+02	
Arsenic(III)	184.5000	360.00	190.00	5.13E-01	9.71E-01	
Benzene	78.0000	5300.00 <sup>(1)</sup>		1.47E-02		
Beryllium	4.3000	130.00 (1)	5.30 <sup>(1)</sup>	3.31E-02	8.11E-01	
Cadmium	9.9000	<b>3.92</b> <sup>(3)</sup>	1.13 <sup>(3)</sup>	2.52E+00	8.73E+00	
Chioride	781000.0000	860000.00	230000.00	9.08E-01	3.40E+00	
Chromium(VI)	288.0000	16.00	11.00	1.80E+01	2.62E+01	
Copper	1160.0000	17.73 (3)	11.82 (3)	6.54E+01	9.81E+01	
DDT	3.5500	1.10	0.001	3.23E+00	3.55E+03	
DDE (DDT metabolite)	59.0000	1050.00 (1)		5.62E-02		
Dimethyl phenol, 2,4-	880.0000	2120.00 (1)	·	4.15E-01		
Endosulfan	3.5500	0.22	0.06	1.61E+01	6.34E+01	
Ethylbenzene	1100.0000	32000.00 (1)		3.44E-02		
Fluoranthene	61.0000	3980.00 (1)		1.53E-02		
Heptachlor	1.8000	0.52	0.004	3.46E+00	4.74E+02	
Iron	389000.0000		1000.00		3.89E+02	
Lead	1380.0000	81.65 (3)	3.18 (3)	1.69E+01	4.34E+02	
Mercury	22.2000	2.40	0.01	9.25E+00	1.85E+03	
Naphthalene	310.0000	2300.00 (1)	620.00 <sup>(1)</sup>	1.35E-01	5.00E-01	
Nickel	172.0000	1418.24 (3)	157.67 (3)	1.21E-01	1.09E+00	
Phenanthrene	96.0000	<b>30.00</b> <sup>(2)</sup>	6.30 (2)	3.20E+00	1.52E+01	
Phenoi	290.0000	10200.00 (1)	2560.00 (1)	2.84E-02	1.13E-01	
Selenium	7.5000	20.00	5.00	3.75E-01	1.50E+00	
Tetrachioroethylene	125.0000	5280.00 <sup>(1)</sup>	840.00 <sup>(1)</sup>	2.37E-02	1.49E-01	
Zinc	2200.0000	117.02 (3)	105.99 (3)	1.88E+01	2.08E+01	

(A) EEC - Expected Environmental Concentration

(8) Toxicological effect criteria concentration. Values are EPA Water Quality Criteria for the protection of aquatic life and their uses.

(1) Insufficient data to develop criteria. Value presented is the L.O.E.L.-Lowest Observed Effect Level.

(2) Proposed criterion

(3) Hardness dependent criteria (100 mg/L CaCO<sub>3</sub> used)

(4) pH dependent criteria (7.8 pH used)

.

•

. . . 1

.

.

## SPECIES LIST.

# SPECIES LIST

## Terrestrial Flora

Horseweed (Conyza canadensis) Plantain (Plantago spp.) Common Mullein (Verbascum thapsus) Senna (Cassia spp.) Vetch (Vicia spp.) Orchard Grass (Dactylis glomerata) Hawthorn Oueen Anne's Lace (Daucus carota) Poplar (Populus spp.) Honeysuckle (Lonicera spp.) . Pokeweed (Phytolacca americana) Goldenrod (Solidago spp.) Aster (Aster spp.) Dock (Rumex spp.) Reed Canary Grass (Phalaris arundinacea) Sycamore (Platanus occidentalis) Red Maple (Acer rubrum) Silver Maple (Acer saccharinum) Black Willow (Salix nigra) Buckwheat (?) Reed (Phragmites communis) Black Locust (Robinia pseudoacacia) Staghorn Sumac (Rhus typhina) Mulberry (Morus spp.) Black Cherry (Prunus serotina) Tree-of-heaven (Ailanthus altissima) Ash (Fraxinus spp.) Norway Maple (Acer platanoides) Nightshade (Solanum dulcamara) Bush-Clover (Lespedeza spp.)

# Terrestrial Fauna

Eastern Cottontail (Sylvilagus floridanus) Double-crested Cormorant (Phalacrocorax auritus) Great Cormorant (Phalacorocorax) Canada Goose (Branta canadensis) Black Duck (Anas rubripes) Sharp-shinned Hawk (Accipiter striatus) Killdeer (Charadrius vociferus) Spotted Sandpiper (Actitus macularia) Great Black-backed Gull (Larus marinus) Herring Gull (Larus argentatus) Ring-bill Gull (Larus delawarensis) Laughing Gull (Larus atricilla) Foster's Tern (Sterna forsteri) Rock Dove (Columba livia) Downy Woodpecker (Picoides pubescens) European Starling (Sturnus vulgaris) Northern Mockingbird (Mimus polyglottos) Northern Cardinal (Cardinalis cardinalis) Song Sparrow (Melospiza melodia) Dark-eyed Junco (Junco hyemalis) House Finch (Carpodacus mexicanus) American Goldfinch (Spinus tristus) House Sparrow (Passer domesticus) Barn Swallow (Hirundo rustica) Common Crow (Corvus brachyrhynchos) Ringneck Pheasant (Phasianus colchicus) Mallard (Anas platyrhynchos) American Kestrel (Falco sparverius) Yellow-shafted Flicker (Colaptes auratus) Rough-Winged Swallow (Stelgidopteryx ruficollis) American Crow (Corvus brachyhynchos) Common Yellow Throat (Geothlypis trichas) Red-winged Blackbird (Agelaius phoeniceus) Brown-headed Cowbird (Molothrus ater)

Common Grackle (Ouiscalus quiscula) Northern Oriole (Icterus galbula) Mourning Dove (Zenaida macroura) American Goldfinch (Carduelis tristis) Great Blue Heron (Ardea herodias) Canvasback (Aythya valisineria) Ring-necked Duck (Aythra collaris) Common Goldeneye (Bucephala clangula) Bufflehead (Bucephala albeola) Red-tailed Hawk (Buteo jamaicensis) Great Horned Owl (Bubo virginianus) Belted Kingfisher (Megaceryle alcyon) Hairy Woodpecker (Picolides villosus) Blue Jay (Cyanocitta cristata) Black-capped Chickadee (Parus atricapillus) Carolina Chickadee (Parus carolinensis) Brown Creeper (Certhia familiaris) Tufted Titmouse (Parus bicolor) White-breasted Nuthatch (Sitta carolinensis) Carolina Wren (Thyrothorus ludovicianus) Cedar Waxwing (Bombycilla cedrorum) Golden-crowned Kinglet (Regulus satrapa) White-throated Sparrow (Zonotrichia albicollis) Chipping Sparrow (Spizella passerina) Belted Kingfisher (Megaceryle alcyon) Gray Catbird (Duyetella carolinensis) American Robin (Turdus migratorius)

# Aquatic species

Peltandra spp. Potamogeton spp. snails amphipods flatworms freshwater clams (Corbiculae spp.) scuds (Gammarus spp.)

COMPOUNDS FOR WHICH APPROPRIATE ECOTOXICOLOGICAL DATA WAS UNAVAILABLE Positively identified compounds for which ecotoxicological data appropriate for evaluating sediments have not been identified:

Barium Calcium Cobalt Magnesium Manganese Potassium

Acenaphthylene Benzo(b) fluoranthene Benzo(k) fluoranthene Benzo(g,h,i)perylene Benzo(g,g,1)perylene Bis(2-ethylhexyl)phthalate Carbazol Dibenzofuran 2,4-Dichlorophenol 1,2,3,4,7,8,9-HPCDF Indeno(1,2,3-cd)pyrene 4-Methylphenol OCDF Total HPCDD Total HPCDF Total HXCDD Total HXCDF Total PECDD Total PECDF Total TCDD Total TCDF



Positively identified compounds for which ecotoxicological data appropriate for evaluating soils have not been identified:

Antimony Barium Calcium Iron Magnesium Potassium Selenium Silver Sodium Thallium Vanadium

Acenaphthene Acenaphthylene Aldrin Anthracene Arochlor 1221 Arochlor 1232 Arochlor 1248 Arochlor 1254 Arochlor 1260 Benzo (a) anthracene Benzo(b) fluoranthene Benzo(k) fluoranthene Benzoic Acid Benzo(g,h,i)perylene Benzo(a)pyrene Bis(2-chloroethyl)ether Bis(2-ethylhexyl)phthalate Butylbenzylphthalate Carbon disulfide Crysene 4,4'-DDD 4,4'-DDE dibenz (a, h) anthracene 3,3-dichlorobenzidine diethylphthalate Fluorene 2-Hexanone Methoxychlor 4-Methyl-2-Pentanone Naphthalene Phenanthrene Pyrene Toluene Xylene

For .

<u>.</u>\*r

.

.

• •

· .

# REFERENCES

SEDIMENT REFERENCES

Birge, W.J. et al; Effects of Polychlorinated Biphenyl Compounds and Proposed PCB-Replacement Products on Embryo-Larval Stages of Fish and Amphibians #118 (1978) as cited by USEPA; Ambient Water Quality Criteria Doc: Phthalate Esters p. B-18 (1980) EPA 440/5-80-067.

National Oceanic and Atmospheric Administration. The Potential for Biological Effects of Sediment-Sorbed Contaminants Tested in the National Status and Trends Program. NOAA Technical Memorandum NOS OMA 52; March 1990.

#### SOIL REFERENCES

Agency for Toxic Substances and Disease Registry, 1990. Toxicological Profile for 2-Butanone. Draft. USPHS/USEPA. October 1990.

Agency for Toxic Substances and Disease Registry, 1990. Toxicological Profile for Ethylbenzene. USPHS/USEPA. December 1990.

Agency for Toxic Substances and Disease Registry. 1991. Toxicological Profile for Chromium (Draft). USPHS/USEPA. October 1991.

Cameron and Burgess, 1945. The Toxicity of DDT. British Medical Journal 1:865-871.

Little, Arthur D., Inc. 1987. The Installation Restoration Program Toxicology Guide. Volume 3. Cambridge, MA. June 1987.

U.S. Environmental Protection Agency. 1980. Ambient Water Quality Document: Phthalate Esters. EPA 440/5-80-067. 1980.

U.S. Environmental Protection Agency. 1980. Ambient Water Quality Document: Polychlorinated Biphenyls. EPA 440/5-80-068. 1980.

U.S. Environmental Protection Agency. 1983. Hazardous Waste Land Treatment (Revised Edition). Office of Solid Waste and Emergency Response. SW-874. Washington, D.C.

World Health Organization. 1984. Environmental Health Criteria: Endosulfan.



.

• •

. . . .

Appendix D.3

6696-403/revmarks/haz/drifs7 AR300893

N.O.A.A. Final Draft Aquatic Ecological Risk Assessment



U.S. DEPARTMENT OF COMMERCE National Oceanic and Atmospheric Administration Office of Ocean Resources Conservation and Assessment Hazardous Materials Response and Assessment Division Coastal Resources Coordination Branch 7600 Sand Point Way N.E. — Bin C15700 Seattle, Washington 98115

March 15, 1994

Cesar Lee U.S. EPA Region III 841 Chestnut Street Philadelphia, PA 19107

Dear Mr. Lee:

Enclosed are two copies of the Final Draft Aquatic Ecological Risk Assessment for the Metal Bank Cottman Avenue NPL site.

An additional copy was sent directly to HMM Associates.

Sincerely,

L. Jay Field

cc: Peter Swinick, HMM Associates, Inc.



### AQUATIC ECOLOGICAL RISK ASSESSMENT

FOR

### METAL BANK OF AMERICA / COTTMAN AVENUE NPL SITE

FINAL DRAFT

Prepared For:

U.S. Environmental Protection Agency Region III Philadelphia, PA

### Prepared By:

National Oceanic and Atmospheric Administration Office of Ocean Resources Conservation and Assessment Hazardous Materials Response and Assessment Division Coastal Resources Coordination Branch 7600 Sand Point Way N.E. — Bin C15700 Seattle, Washington 98115

March 15, 1994

i

AR300896

## CONTENTS

Lis Ac	st of Figu st of Table knowledg ecutive S	les gments	iii iv v vi
1	INTRO 1.1	ODUCTION Organization of the Aquatic Ecological Risk Assessment	
2	Habita 2.1 2.2 2.3	ats and Aquatic Receptors.Aquatic Species Using the Delaware RiverReceptors of Concern.Life Histories of Selected Fish Species in the Delaware River2.3.1Shortnose Sturgeon (Acipenser brevirostrum)2.3.2White Perch (Morone americana)2.3.3Channel Catfish (Ictalurus punctatus)2.3.4Eastern Silvery Minnow (Hybognathus regius)	2-1 2-6 2-6 2-7 2-8 2-9
3	CONT 3.1 3.2 3.3 3.4	FAMINANTS       OF       CONCERN.         Selection Criteria for Contaminants of Concern       Contaminants of Concern in Groundwater and Surface Water         Contaminants of Concern in Non-Aqueous Phase Liquid (NAPI Contaminants of Concern in Sediments	<b>3-</b> 1 <b>3-</b> 2 L). <b>3-</b> 4
	4.1 4.1. 4.1. 4.1. 4.1. 4.1. 4.1. 4.2 4.3 4.3 4.3 4.3 4.3	1.2Polynuclear aromatic hydrocarbons.1.3DDT and Metabolites1.4Phthalates1.5Cadmium.4.1.2Summary of Exposure Pathways.Receptor PopulationsExposure-Point Concentrations4.3.1PCBs1.1PCBs in Groundwater and Surface Water1.2Exposure to PCBs in NAPL.1.3Exposure to PCBs in Sediments4.3.2PAHs.2.1Exposure to PAHs in NAPL.2.2Exposure to PAHs in Sediments	4-1 4-2 4-2 4-5 4-5 4-5 4-6 4-6 4-6 4-7 4-7 4-7 4-7 4-7 4-12 4-12 4-15 4-16
	4.3	<ul><li>Exposure to PAHs in Sediments</li><li>4.3.3 Phthalates, DDTs, and Cadmium</li></ul>	

,

# CONTENTS (cont.)

	4.3.3	.1	Exposure to DEHP in NAPL4-17
	4.3.3.2		Exposure to Phthalates, DDTs, and Cadmium in
	Sedir	nents	17
		4.3.4	PCB Tissue Residue Levels in Invertebrates and Fish4-20
	4.3.4	.1	<i>Corbicula</i>
	4.3.4	.2	Silvery minnow and channel catfish4-21
	4.3.4	.3	Delaware River data
	4.4	Upstrea	m and Downstream Transport of COCs from MBA4-27
	4.5		inty Analysis4-27
		4.5.1	General
,		4.5.2	Surface Water Concentrations4-28
		4.5.3	NAPL Concentrations4-29
		4.5.4	Sediment Concentrations
		4.5.5	Tissue Concentrations4-30
5	TOXIC	ITY A	ASSESSMENT5-1
	5.1.	Toxicit	y Reference Concentrations for Water 5-1
		5.1.1	Toxicity Reference Concentrations for Surface Water 5-1
		5.1.2	Toxicity Reference Concentrations for NAPL 5-2
	5.2.	-	y Reference Concentrations for Sediment
X.		5.2.1	
		5.2.2	
		5.2.3	DDT and Metabolites5-5
		5.2.4	Phthalates5-5
		5.2.5	
	5.3.		y Reference Concentrations for PCBs in Tissues
	5.4		ainty Analysis5-10
		5.4.1	General
	5.4.		Use of toxicity data5-10
	5.4.	1.2	Use of total PCBs5-11
		5.4.2	Surface Water Toxicity
		5.4.3	Sediment Toxicity
		5.4.4	Toxicity Associated with PCB Tissue Residues5-12
C		<b>ΥΤΑΓΣΑ</b>	
6			CTERIZATION
	6.1	6.1.1	Exposure
			Exposure to Surface Water
	6.2	6.1.2 Expos	Exposure to COCs in NAPL
	0.2	Expos 6.2.1	ure to COCs in Sediments
			Total PCB in Sediment
		6.2.2	I Otal FARI IN Sediment



7

AR300898

iii

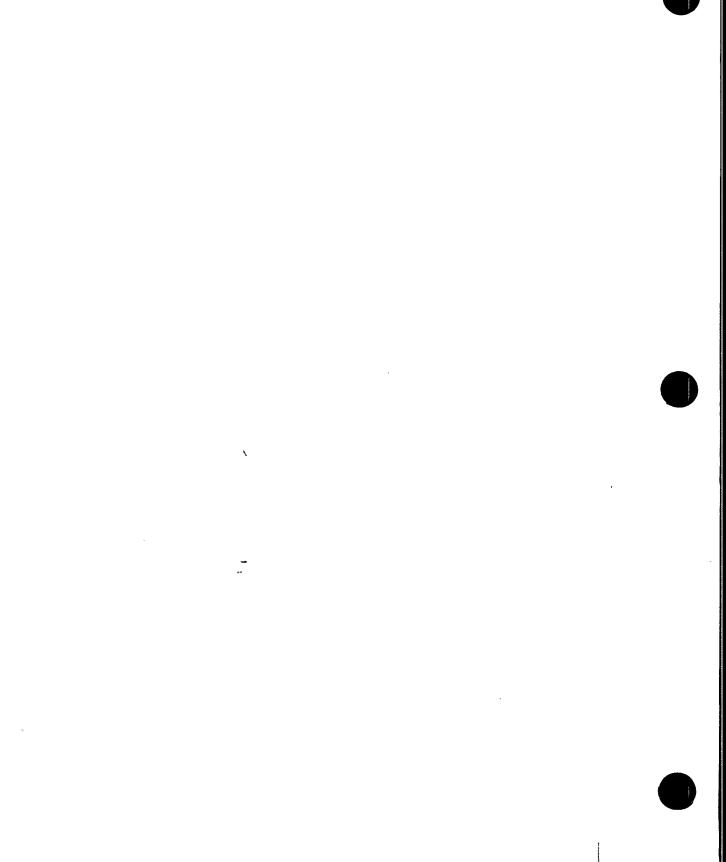


## CONTENTS (cont.)

	6.2.3	Phthalates	6-4
	6.2.4	DDT and Metabolites	6-4
	6.2.5	Cadmium	6-5
	6.2.6	Benthic Community Survey	6 <b>-</b> 6
6.3	PCBs	in Tissues	6-7
6.4	Uncert	tainty Analysis	6-8
	6.4.1	General	6-8
	6.4.2	Surface Water Risk Characterization	6-9
	6.4.3	NAPL Risk Characterization	6-9
	6.4.4	Sediment Risk Characterization	6-10
	6.4.5	Tissue Residue Risk Characterization	6-10
6.5	Concl	usions	<b>6-</b> 12
REFER	RENCES		7-1







AR300900

### LIST OF FIGURES

- Figure 4-1. Sampling locations for sediments, clam tissues, surface water, and groundwater seeps (Weston 1978; Dodd 1992; HMM 1992).
- Figure 4-2. 1993 sediment sampling locations (HMM 1993).
- Figure 4-3. Average total PCB concentrations for the western riprap and nearfield mudflat stations plotted against their distance from riprap station MF-107 (distance = distance along riprap + perpendicular distance from riprap). The sloping line represents the regression line for just the riprap stations (R<sup>2</sup>=0.96).
- Figure 4-4. Fish sampling locations on the Delaware River (Menzie-Cura 1993a).
- Figure 4-5. PCB concentrations (mg/kg wet weight) in channel catfish collected in the Delaware River above and below the Metal Bank Site (river kilometer 175) (DRBC 1988 and unpublished; Greene 1991; USFWS 1991; EPA 1992; HNIM 1993).
- Figure 4-6. PCB concentrations (mg/kg lipid) in channel catfish collected in the Delaware River above and below the Metal Bank Site (river kilometer 175) (DRBC 1988 and unpublished; Greene 1991; USFWS 1991; EPA 1992; HMM 1993).
- Figure 5-1. Cumulative percentile plot of the tissue concentrations of PCBs (mg/kg, wet weight) associated with adverse effects in fish species.

## LIST OF TABLES

- Table 2-1. List of major species and their habitat use of the Delaware River in the vicinity of the Metal Bank site (Kaufmann, personal communication 1993).
- Table 2-2. Fish species collected from three zones of the Delaware River estuary during a three year inventory beginning in July 1984 (DRBC 1987).
- Table 3-1. Concentrations and frequency above detection limits (DL) of the potential contaminants of concern in groundwater, surface water ( $\mu g/l$ ) and non-aqueous phase liquid (NAPL) (mg/l) at the Metal Bank site (HMM 1993b) compared to the chronic AWQC for the protection of freshwater organisms (EPA, 1992b).
- Table 3-2. Concentrations of the potential organic and inorganic contaminants of concern reported in mudflat, riprap, and Delaware River sediments (HMM 1993b) compared to their respective ER-L, ER-M, or AET concentrations (PTI 1988; Long and Morgan 1991; Long and MacDonald 1992).
- Table 4-1. Summary of bioaccumulation factors (ratio of the concentration of PCB in the organism to that in the sediment) from PCB-contaminated sediments.
- Table 4-2. Summary of early analytical data from sampling at Metal Bank of America (Weston 1978).
- Table 4-3. Mean and upper 95% confidence limit (CL) concentrations (mg/kg) of total PCBs in the sediments near Metal Bank site normalized to dry weight and TOC.
- Table 4-4. Estimated concentrations (mg/kg)of four PAHs in NAPL and their respective AWQC values.
- Table 4-5. Mean and upper 95% (CL) concentrations (mg/kg) of total PAHs in the sediments near the Metal Bank site normalized to dry weight and TOC.
- Table 4-6. Mean and upper 95 percent CL concentrations (mg/kg) of DEHP, DBP, and DOP in the sediments near the Metal Bank site normalized to dry weight and TOC.
- Table 4-7. Mean and upper 95% (CL) concentrations (µg/kg) of 4,4'-DDT, 4,4'-DDE and 4,4'-DDD in the sediments near the Metal Bank site normalized to dry weight and TOC.
- Table 4-8. Total PCB concentrations in sediments and tissues (based on congener analysis<br/>(Hermanson 1992)) and bioaccumulation factors (BAFs, tissue lipid normalized<br/>PCB/sediment TOC normalized PCB concentrations) for *Corbicula* from five stations<br/>in the mudflats of the Metal Bank site.
- Table 4-9. Total PCB concentrations (sum of Aroclors 1242 + 1260) in the tissues of channel catfish and silvery minnow collected near the Metal Bank site (HMM 1993).
- Table 4-10. Summary of PCB concentrations in muscle tissue of channel catfish and white perch collected in the Delaware River from 1987 through 1992. Concentrations are listed by wet weight (mg/kg) and as lipid normalized (mg/kg lipid).
- Table 5-1. MATC and chronic values for PCBs (µg/l) based on exposure for life-cycle, partial life-cycle, or early-life stage tests (from EPA 1980).



March 1994

MB Aquatic ERA

AR300902

vii

Table 5-2. Tissue concentrations of PCBs (mg/kg, wet weight) associated with adverse effects in freshwater and marine fish species.

Table 5-3. Tissue concentrations of PCBs (mg/kg, wet weight) associated with miscellaneous effects in freshwater fish species.

Table 6-1. Hazard quotients for exposure to water-borne PCBs in the Delaware River under low-flow and average discharge conditions for aquatic organisms and shortnose sturgeon.

- Table 6.2. Hazard quotients for sediment exposure based on toxicity reference values (TRV) for<br/>contaminants of concern (COC) in sediment and the mean and upper 95 percent<br/>confidence limit of sediment COC concentrations.
- Table 6-3. Total score, density of organisms, total PCB concentrations in sediments (mg/kg, dry weight (DW) and TOC normalized) and distance from Metal Bank site boundary for mudflat stations. Total score is based on qualitative benthic survey data. (HMM 1993a and 1993b).
- Table 6-4. Hazard quotients for tissue residue concentrations of PCBs in channel catfish and silvery minnow collected from the Delaware River vicinity of the Metal Bank site.

,



· ·

March 1994

AR3009

### Acknowledgments

Funding for this ecological risk assessment was provided by U.S. Environmental Protection Agency, Region III, Hazardous Waste Management Division under Interagency Agreement Number DW13943516-01-3.

Menzie-Cura and Associates (for the PRP Group) provided useful summaries of life history information on selected fish species in the Delaware River.

Two meetings were held between technical representatives of the PRP Group and NOAA: the first meeting in Seattle in July 1993, discussed the general approach and methodology to ecological risk assessment at the Metal Bank site. At the second meeting, in Seattle on February 4, 1994, the PRP Group's comments on the draft aquatic ecological assessment were discussed. Both of these meetings provided useful exchange of ideas and discussion of different technical approaches. The PRP Group also provided extensive written comments on the initial draft risk assessment.

## **Executive Summary**

This document presents an ecological risk assessment for aquatic resources near the Metal Bank of America (MBA), Cottman Avenue site, located on the Delaware River in Philadelphia, Pennsylvania.

### Receptors

The habitats of primary concern include surface waters, tideflats, and associated bottom substrates of the Delaware River. The Delaware River is a freshwater tidal system near the Metal Bank site, providing resident and seasonal habitat for numerous species of anadromous, catadromous, estuarine, and freshwater fish.

The shortnose sturgeon, a Federal- and State of Pennsylvania-designated endangered species, is a receptor of primary importance. Shortnose sturgeon live their entire life cycle in the Delaware River and are known to be present in the river reach that includes the site during summer after spawning in upstream areas.

Other fish species considered in the assessment include channel catfish, silvery minnows, and white perch. Important considerations in selecting other receptors of concern included the need to evaluate species that represented different trophic levels and food-web pathways, species that were likely to be present near the site for at least part of the spring-summer-fall feeding period, and the availability of data.

Channel catfish serve as a representative of a benthic freshwater fish species that feed on a variety of prey types and are likely to live in the river near the site for some time. Channel catfish are also used as a surrogate species for the shortnose sturgeon. Silvery minnow are an important forage fish species that feed in shallow water areas, such as the mudflat next to the site. White perch are an anadromous fish species that is abundant near the site and recreationally important.

Benthic invertebrates, such as Asiatic clams (*Corbicula fluminea*), are present in the mudflat and Delaware River next to the site. The invertebrates were considered as a single group for<sup>-</sup> the purposes of this assessment. Benthic invertebrates may provide an important exposure pathway for higher trophic level fish and wildlife species that feed in the intertidal and nearshore Delaware River habitats.

### **Contaminants of Concern**

PCBs were identified as the contaminants of primary concern because of their presence at elevated concentrations in groundwater, non-aqueous phase layer (NAPL), and sediment. Other contaminants were screened for consideration in the risk assessment using chronic ambient water quality criteria (AWQC) for surface water and groundwater and Effects-Range Low (ER-L) values determined by Long and MacDonald (1992) for sediment. Other contaminants of concern included polycyclic aromatic hydrocarbons (PAHs), phthalates, DDT and its metabolites DDE and DDD, and cadmium.

### **Exposure Pathways**

Exposure-pathways included surface water, direct exposure to NAPL, and contaminated sediment in the mudflat next to the site and the Delaware River. The accumulation of PCBs in the tissues of fish species was considered as a means to integrate all the exposure pathways near the site.

Exposure-point concentrations in surface water of the Delaware River from groundwater discharge were estimated from dilution factors calculated using a 15-meter dilution zone and river-flow volumes for average- and low-flow discharges.

Exposure-point concentrations for NAPL were estimated from the concentrations of PCBs, PAH, and phthalates in one sample collected from monitoring well (MW)-6 in 1991 and historical values for PCBs. The volume of NAPL currently discharging into the intertidal and nearshore areas of the Delaware River is unknown and may be small. Subsequent attempts to sample or locate this layer have been unsuccessful. The existence of a NAPL in the subsurface of the riprap area has been observed but not sampled directly. No active NAPL seeps on the surface of the riprap or mudflat were observed during the 1993 sampling. Groundwater seeps, which have been observed recently, have resulted in "oily sheens" on the surface as opposed to a distinct oil layer. Thus, it appears that any direct seepage of oil to the surface of the riprap or mudflat is probably highly localized, if it occurs to any significant extent.

Mean (arithmetic) and upper 95 percent confidence limit values were determined for sediment concentrations, using one-half detection limit for values below detection if at least one sample had concentrations above the detection limit for that contaminant. Because the pattern of sediment contamination for PCBs, PAHs, and the phthalate DEHP showed a clear decrease in concentration with distance from the site boundary (Tables 4-3, 4-5, and 4-6), sediment samples for these contaminants were grouped into three separate zones for risk characterization:



samples from the riprap area; samples from the mudflat and Delaware River within 30 meters of the site boundary; and samples from the mudflat and Delaware River greater than 30 meters from the site boundary. No similar gradient was apparent in sediment concentrations of DDTs or cadmium; therefore, the risk evaluation for these contaminants was based on the combined values for all sediment samples.

Tissue residue PCB concentrations in fish species integrate all the above exposure pathways, since fish accumulate most of their PCB body burden through the food web. Whole body concentrations in silvery minnow and channel catfish collected in the fall of 1993 from the Delaware River next to the site were used to estimate concentrations in other fish species.

### Toxicity

Toxicity reference values were determined for exposure to surface water, sediment, and the tissue residue concentrations (for PCBs only). For surface water, toxicity reference values were based on chronic AWQC for the protection of freshwater organisms. For PCBs, the toxicity reference value was based on maximum acceptable toxicant concentrations (MATC) for fathead minnow. Because of the endangered species status of the shortnose sturgeon, the limited amount of available chronic toxicity data for fish species, and the lack of species-specific toxicity information, a safety factor of 100 was applied to the toxicity reference value for sturgeon.

Toxicity reference values for NAPL were based on AWQC acute toxicity values for PCBs, PAH, and the phthalate DEHP.

Toxicity reference values for sediment were based on Long and MacDonald (1992) ERL and ERM values, or Apparent Effects Threshold (AET) values if ER-L or ER-M values were not available. As suggested by Long et al. (1994), adverse effects were considered unlikely at concentrations below the ER-L and probable at concentrations above the ER-M.

Toxicity reference values for tissue residue PCB concentrations were derived from a compilation of published studies linking adverse effects to PCB tissue concentrations in a variety of fish species. The tenth percentile of the lowest observed effect levels and the median effect level were 0.2 and 7.0 mg/kg (wet weight), respectively.



### **Risk Characterization**

The risk characterizations were based on the toxicity quotient approach, using exposure point concentrations for each media and toxicity reference values.

Surface water exposure from groundwater discharge is reduced rapidly with dilution from Delaware River. Only PCBs were considered a contaminant of concern for surface water. Any exposure to PCB concentrations exceeding toxicity reference values would be confined to a 15-meter-wide band of the river next to the site. The risk of adverse effects is expected to be low. The highly conservative toxicity reference value for the shortnose sturgeon exceeded the estimated mean exposure concentration for PCBs in the dilution zone under low flow conditions by less than a factor of two, indicating possible risk. However, it is highly unlikely that individual shortnose sturgeon would remain in the exposure area long enough to receive chronic exposure.

Any exposure to NAPL, if it occurs, is likely to be highly localized in the immediate vicinity of the riprap area. However, because measured concentrations of PCBs, PAHs, and phthalate in NAPL exceeded toxicity reference concentrations by as much as five orders of magnitude, toxic effects to exposed organisms are highly probable. Any discharge of NAPL would also contribute to PCB accumulation in nearshore organisms and food webs.

The primary route of exposure for benthic organisms in the nearshore areas of the Delaware River and mudflat near MBA is through contact with sediments (and/or sediment interstitial water). Mean sediment concentrations of PCBs, PAHs, and the phthalates bis (2-ethylhexyl) phthalate (DEHP), di-n-butyl phthalate (DBP) and di-n-octyl phthalate (DOP) greatly exceeded probable effects levels (ER-M or high AET) in the riprap area, indicating that adverse effects to benthic invertebrates exposed to these contaminated sediments are highly likely. Concentrations of PAHs and phthalates decreased in a steep gradient away from the site, resulting in hazard quotient values that were one or less in the mudflat and Delaware River greater than 30 meters from the site boundary. Hazard quotients for PCBs exceeded 1 for all three zones, ranging from over 400 in the riprap to less than 5 in the outer zone. No spatial pattern was observed in the concentrations of DDT, DDD, DDE, and cadmium, based on limited sampling. Samples with detectable concentrations exceeded probable effects levels (ER-M or high AET) by about an order of magnitude.

Because of limited sampling of Delaware River sediment, the extent of PCB contamination was not adequately defined and could extend both downstream and upstream of the site, although

concentrations appear to decline rapidly with distance from the site boundary. Limited data are available for contaminants other than PCBs and PAHs. Problems with data quality (high detection limits for PCBs and other contaminants) further increase the uncertainty in the exposure-point concentrations and the size of the exposure area. The minimum exposure area includes intertidal and nearshore sediment in the Delaware River next to the site. The lack of consistent increase in incidence of effects with increasing PCB concentrations in the data used to derive the toxicity reference value adds an additional source of uncertainty to the risk characterization.

The limited information available in the literature suggests that benthic invertebrates can accumulate high tissue concentrations of PCBs without adverse effects, although invertebrates may experience direct toxicity from exposure to contaminated sediments. Tissue PCB concentrations in Asiatic clams in the mudflat, however, demonstrate that PCBs in the area are bioavailable and are likely to be accumulated in benthic food webs. The bioaccumulation factor (BAF) (lipid-normalized tissue to organic carbon-normalized sediment ratio) observed for Asiatic clams was consistent with literature values. Congener-specific PCB analysis showed very similar patterns for the mudflat sediment (and one groundwater sample) and clam tissue residues, suggesting that PCBs from the site were a probable source for the PCBs in the clam tissue.

Mean whole body PCB concentrations in tissue of silvery minnows and channel catfish collected in the Delaware River next to the site exceeded the tenth percentile of the lowest observed adverse effect concentrations associated with tissue residues in fish species, but not the median value. The silvery minnow is considered representative of non-migratory forage fish species that feed in the immediate nearshore area. Exposure to site-related PCBs in this species is likely. Channel catfish are probably less restricted in their movements than the silvery minnow, so the degree of exposure to site-related contaminants is less certain. Based on the mean PCB concentration in the samples collected more than 30 m from the site boundary, the observed BAF for silvery minnow was comparable to literature values, while the BAF for channel catfish was less than expected for similar trophic level fish species reported in the literature. The fact that the silvery minnows have a larger BAF than the channel catfish further suggests that they are exposed on average to sediments containing higher levels of PCBs than are the channel catfish. Utilizing these species as representative of other fish species with similar extent of exposure to the nearshore PCB-contaminated sediments immediately adjacent to the Metal Bank site or to nearshore food webs, the results suggest a potential risk of reproductive effects in sensitive fish species.

### March 1994

xv

AR300910

Channel catfish were also used as a surrogate species for estimating the exposure to site-related contaminants and the accumulation of PCBs in the endangered shortnose sturgeon. Considerable uncertainty exists in estimating the extent of exposure for the sturgeon, and nothing is known about its relative sensitivity to adverse effects from accumulated PCBs. Although they occupy a lower trophic level than the channel catfish, the shortnose sturgeon may be particularly prone to accumulating and transferring high concentrations of PCBs to their developing offspring (considered the most sensitive toxic endpoint for PCBs to fish) due to their benthic feeding habit, longevity, late age of sexual maturity, and high lipid content of their eggs. Thus, the potential risk to shortnose sturgeon resulting from accumulation of PCBs for all exposure pathways near the site may be greater than for other fish species because of these life-history characteristics.

## **1** INTRODUCTION

The Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA), as amended by the Superfund Amendments and Reauthorization Act of 1986 (SARA), calls upon the U.S. Environmental Protection Agency (EPA) to protect human health and the environment from releases or potential releases of contaminants from hazardous waste sites. The proposed revision of the National Contingency Plan (NCP) calls for identifying and mitigating the environmental impact of these sites and selecting remedial actions that are protective of the environment. In addition, numerous Federal and State laws and regulations are potentially "applicable or relevant and appropriate requirements" (ARARs). Compliance with these laws and regulations may require evaluating a site's ecological impacts and the measures needed to mitigate those impacts.

This document presents an ecological risk assessment for the aquatic component at the Metal Bank of America (MBA) Superfund site. The primary objective of the aquatic ecological risk assessment is to provide a qualitative and quantitative assessment of the potential risks to aquatic receptor species in the Delaware River and the mudflats next to the site. Specific goals of the assessment are to:

- identify receptor species in the Delaware River and mudflats next to the site that may be exposed to site-related contaminants;
- identify pathways of exposure by which aquatic receptor species may be exposed to site-related contaminants;
- estimate exposure-point concentrations;
- collect information on the toxic effects of the chemicals of concern and select endpoints of concern;
- characterize risks to aquatic receptor species associated with exposure-point concentrations;
- discuss uncertainties associated with the assessment; and
- discuss ecological significance of the findings.

### 1.1 Organization of the Aquatic Ecological Risk Assessment

The remainder of the aquatic ecological risk assessment is organized in the following sections:

• Section 2: <u>Habitats and Aquatic Species of the Delaware River</u> - This section lists the habitats and the major aquatic species that use the Delaware River near the site.

1-1

MB Aquatic ERA

1-2

- Section 3: <u>Contaminants of Concern</u> This section lists the contaminants of concern in surface waters, sediments, and aquatic biota of the Delaware River and mudflats.
- Section 4: <u>Exposure Assessment</u> This section discusses exposure pathways and estimates exposure-point concentrations for the contaminants of concern in surface water, sediments, and tissues.
- Section 5: <u>Toxicity Assessment</u> This section discusses the toxicity of the contaminants of concern and derives toxicity reference concentrations.
- Section 6: <u>Risk Characterization:</u> This section presents the qualitative and quantitative characterization of risk.

March 1994

#### MB Aquatic ERA

### 2 HABITATS AND AQUATIC RECEPTORS

The Delaware River provides important habitat for fishes, migratory waterfowl, and shore birds. Habitats of primary concern include surface waters, tideflats, and associated bottom substrates of the Delaware River.

Near the site, the river is characterized as a low gradient, tidal freshwater system. The Delaware River maintains a variable flow velocity dependent on the tide and freshwater discharge and a tidal amplitude of 0.5 to 2.0 meters. The river is approximately 1.0 km wide at the site and has a mean depth of 4.5 meters with the greatest depth (14 meters) centrally located in a dredged shipping channel. Salinities in the Delaware River near the site commonly range from 0 to 3 parts per trillion (ppt) and fluctuate throughout the year depending on rainfall, saltwater intrusion, and urban runoff (Kaufmann, personal communication 1993). Seven tributaries enter the river within 15 river km of the site. Substrate composition at this reach of the river is primarily silty sand, gravel, and mud. The water quality of the Delaware River, particularly dissolved oxygen concentration, has improved substantially since municipal waste treatment was required by the City of Philadelphia in 1985 (Lupine, personal communication 1993). The site is approximately 174 kilometers upstream from Delaware Bay (USGS 1976).

### 2.1 Aquatic Species Using the Delaware River

The Delaware River has historically been identified as a spawning site for over 60 species of fish (Daiber 1988). Near the site, the river supports diverse and abundant natural resources (Table 1; Kaufmann, personal communication 1993; Miller, personal communication 1993; Lupine, personal communication 1992; Daiber 1988). Numerous species migrate close to the site and live in the area for extended periods during sensitive life stages. Eight species of anadromous fish use the Delaware River as a migratory corridor. Species of special interest due to their commercial importance or abundance in the area are alewife, American eel, American shad, blueback herring, blue crab, striped bass, and white perch. The reach of the Delaware River near the site also supports populations of the federally endangered shortnose sturgeon and the New Jersey state-protected Atlantic sturgeon (O'Herron, personal communication 1993; Kaufmann, personal communication 1993). A fish inventory study conducted by the Pennsylvania Fish Commission from 1984 through 1986 identified a total of 53 species of fish in Zones 2, 3, and 4 of the Delaware River. Of the 53 species identified, 36 occurred in Zone 3, the stretch of the Delaware River that includes the MBA site (Table 2-2).

AR300914

# Table 2-1. List of major species and their habitat use of the Delaware River near the Metal Bank site (Kaufmann, personal communication 1993).

	Species		Habitat	Fisheries		
· -				Adult		
Common Name	Scientific Name	Spawning	Nursery	Forage	Comm.	Recr.
ANADROMOUS /CAT	ADROMOUS SPECIES					
Shortnose sturgeon	Acipenser brevirostrum		•	•		
Atlantic sturgeon	Acipenser oxyrhynchus		•	٠		
Blueback herring	Alosa aestivalis		٠	•	1	•
Alewife	Alosa pseudoharengus		*	٠	1	٠
American shad	Alosa sapidissima	•	٠	٠	1	•
American eel <sup>2</sup>	Anguilla rostrata		٠	•		•
White perch <sup>2</sup>	Morone americana	•		•		•
Striped Bass	Morone saxatilis	•	٠	•		•
Sea lamprey	Petromyzon marinus			•		
- · · ,						
ESTUARINE SPECIES	ι i					
Atlantic menhaden	Brevoortia tyrannus		• .	<u>م</u> .		
Banded killifish	Fundulus diaphanus	•	•	•		
Mummichog	Fundulus heteroclitus	•	•	•		
Hogchoker	Trinectes maculatus		•	•	,	
FRESHWATER SPECI	ES					
White catfish	Ictalurus catus	•	•	•		•
Brown bullhead	Ictalurus nebulosus	•	٠	•		•
Channel catfish <sup>2</sup>	Ictalurus punctatus	•	•	٠		•
Largemouth bass	Micropterus salmoides	•		<b>ب</b> .		•
Yellow perch	Perca flavescens			•		<u> </u>
-					1	
INVERTEBRATE SPE	CIES					
Blue crab	Callinectus sapidus		٠	٠		•

 Commonly of more species country in effect on human consumption of these species caught in the Delaware River due to high levels of PCBs and chlordane.

# Table 2-2. Fish species collected from three zones of the Delaware River estuary during a three-year inventory beginning in July 1984 (DRBC 1987).

Species		Zone		Total				
-	2							
Blueback herring	22405 (93%)	3933	2075	28413				
Bay anchovy	.0	24	6790	6814				
White perch	2664	1969	1318	5951				
Silvery minnow	225 (87%)	1334 (93%)	2042	3601				
Spottail shiner	347 (95%)	100 (93%)	938	1385				
Banded killifish	373 (94%)	332 (94%)	504	1209				
Pumpkinseed sunfish	480	194	13	687				
nland silverside	142	145 (99%)	275	562				
Alewife	62 (99%)	367	78	507				
Mummichog	96 (97%)	240 (96%)	169	505				
American shad	136	66	175	377				
Channel catfish	177	139	57	373				
Blue crab	29 (99%)	22	281	332				
Tessellated darter	120 (98%)	30 (96%)	162	312				
Bluegill	275	15	12	302				
Gizzard shad	169 (90%)	12 (97%)	71	252				
Atlantic menhaden	0	17	219	232				
Hogchoker	4	47	182	233				
Brown bullhead	115	45	40 (99%)	200				
Redbreast sunfish	190	43 2	0	192				
Largemouth bass	177	7	0	192				
White catfish	81 (99%)	42	25	148				
	0	3	120	148				
Grass shrimp	24	16	59	99				
Striped bass	0	2	89	91				
Spot	66	13	1	79				
Black crappie		2		50				
Spotfin shiner	46 (99%)	$\overset{2}{0}$	2 (99%)	38				
Smallmouth bass	38	=		35				
Carp	15 (83%)	8 (90%)	12 (99%)	34				
White sucker	27 (80%) 24	4 (97%) 0	3	27				
Yellow perch		5 (90%)	4 (94%)	24				
American eel	15 (92%)	3 (90%)	3	18				
Tiger muskellunge	12	3	0	18				
White crappie Swallowtail shiner	0	15	0	15				
		1 (99%)	1	15				
Golden shiner Green sunfish	13 (90%) 13	0	0	13				
	0	1 .	8 '	9				
Naked goby Atlantic Croaker	0	0	8	8				
	5-	0	0	5				
Shortnose sturgeon Atlantic needlefish	5_0	0	4	4				
	0	0		4				
Walleye Book boos		U U	0	-				
Rock bass	4	0	0	4				
Fallfish Chain nickers)	2 (98%)	0	0	2				
Chain pickerel		1	0	2				
Redfin pickerel		0	0	1				
Atlantic sturgeon	1(0707)	0	0	1				
Goldfish	1(97%)	0	0	1				
Bowfin		0	0	1,				
Smallmouth flounder	0	0		1				
Striped mullet	0	0	1	I.				
Bluntnose minnow		0	0	1				
Margined madtom		0	0	1				
Individuals	28596	9159	15744	53499				

(e.g., 93% means that individuals were counted at 93% of the sites sampled and observed but not counted at 7% of the sites sampled). If no percentage is specified, the value is based on 100% of the sites sampled. Table does not include 3 species that were observed but not counted at 1% of sites.

### MB Aquatic ERA

Blueback herring is one of the most abundant finfish found in the Delaware River system. This species is an important component of the forage base for larger predators such as striped bass (Kaufmann, personal communication 1993; Soldo, personal communication 1992; Daiber 1988; Delaware River Basin Commission [DRBC] 1988). Spawning blueback herring are present throughout the estuary into the tidal freshwater zone up to Chester Island, approximately 38 km downstream from the site. Juveniles are known to frequent reaches of the river upstream from Philadelphia. Alewife move into the vicinity of the site from March to August and congregate in reaches approximately 13 km downstream from the site. This species occurs in greatest abundance during May and early July. Juvenile alewife are commonly present in the same area through October (Kaufmann, personal communication 1993; Soldo, personal communication 1992; Daiber 1988).

The Delaware River shortnose sturgeon population is estimated at 10,000 individuals. This species uses the reach near the site as a forage area and a migratory corridor on a year-round basis. Shortnose sturgeon tend to be found in the deeper central channels of the river; however, field investigations have tracked individuals by radio telemetry into nearshore habitats similar to those associated with the site. Shortnose sturgeon may periodically use these shallow water habitats for foraging during adult and juvenile stages of its life history. Spawning activities typically occur in surface waters of the Delaware River near Trenton, approximately 45 km upstream of the site (O'Herron, personal communication 1993). Shortnose sturgeon were caught approximately 11 km downstream and 16 km upstream from the site in 1987 (DRBC 1988).

Atlantic sturgeon occur in lesser numbers and primarily use surface waters near the site for juvenile rearing. Adult Atlantic sturgeon enter the Delaware River estuary and congregate near Pea Patch Island, approximately 75 km downstream from the site, in late April and early May for spawning. Following their emergence from gravel, young-of-the-year Atlantic sturgeon migrate into non-tidal portions of the Delaware River upstream from the site near Trenton (O'Herron, personal communication 1993).

The semi-anadromous white perch and striped bass live throughout the estuary. White perch typically migrate upstream past the site to spawn; while some striped bass use surface waters near the site for spawning in late-April and early May (Kaufmann, personal communication 1993; Daiber 1988). The run size of American shad in the Delaware River has steadily increased over the past 10 years to approximately 800,000 individuals (Miller, personal communication 1993). Adults are commonly present during April and May with some spawning beginning near the Memorial Bridge, approximately 15 km downstream from the

site. However, the majority of shad spawning activity has been recorded farther upstream beyond tidal effects. Following spawning, the adult shad leave the estuary for marine environments by the end of June. Out-migrating juveniles are present throughout the lower Delaware River basin, with the largest numbers present near the site during August and September (Kaufmann, personal communication 1993; Soldo, personal communication 1992; Daiber 1988).

Blue crabs are present during spring, summer, and fall and are reportedly abundant. Male blue crabs are more tolerant to low salinity than are females and likely predominate in this reach of the Delaware River. Adult and juvenile blue crab are likely to use the intertidal habitats near the site for foraging (Kaufmann, personal communication 1993; Soldo, personal communication 1992).

The Delaware Estuary is the world's largest freshwater port (DRBC 1988). High levels of marine traffic near the site limit commercial fishing activity. Except for small harvests of blue crab, American shad, and blueback herring, minimal commercial fishing occurs in the reach of the Delaware River near the site. Most commercial fishing occurs approximately 85 km south of the site where the river begins to widen into Delaware Bay and brackish conditions predominate (Lupine, personal communication 1992).

A significant sport fishing effort occurs in the Delaware River. Species of recreational importance include striped bass, American shad, herring, catfish, largemouth bass, white perch, yellow perch, American eel, and blue crab (Lupine, personal communication 1992). Blue crabs are harvested near the site by recreational crabbers from March through November (Kaufmann, personal communication 1993; Soldo, personal communication 1992; DRBC 1988). Fishermen use a boat launching ramp located at the yacht harbor directly downstream of the site, and fishing tournaments for largemouth bass are based out of this ramp. A very popular location for shore-based fishing is located approximately 1.6 km upstream of the site at the mouth of the Pennypack River (Kaufman personal communication 1989).

The Pennsylvania Bureau of Water Quality currently has an advisory on the human consumption of several dominant species that are fished for recreational purposes in the Delaware River due to excessive tissue concentrations of PCBs and chlordane. The advisory covers the area from Burlington Island to Tinicum Island, and includes the portion of the river of the Metal Bank site. White perch, channel catfish, and American eel are among the resources included in the advisory (Soldo, personal communication 1992; Kaufmann, personal communication 1993).

### 2.2 Receptors of Concern

The habitats of primary concern include surface waters, tideflats, and associated bottom substrates of the Delaware River. The Delaware River is a freshwater tidal system near the Metal Bank site, providing resident and seasonal habitat for numerous species of anadromous, catadromous, estuarine, and freshwater fish.

The shortnose sturgeon, a Federal and State of Pennsylvania designated endangered species, is a receptor of primary importance. Shortnose sturgeon live their entire life cycle in the Delaware River and are known to be present in the river reach that includes the site during summer after spawning in upstream areas.

Other fish species considered in the assessment include channel catfish, silvery minnows, and white perch. Important considerations in selecting other receptors of concern included the need to evaluate species that represented different trophic levels and food web pathways, species that were likely to be present near the site for at least part of the spring-summer-fall feeding period, and the availability of data.

Channel catfish serve as a representative of a benthic freshwater fish species that feed on a variety of prey types and are likely to be resident in the river next to the site for some time. Channel catfish are also used as a surrogate species for the shortnose sturgeon. Silvery minnows are an important forage fish species that feed in shallow water areas, such as the mudflat next to the site. White perch are an anadromous fish species that is abundant near the site and recreationally important.

Benthic invertebrates, such as Asiatic clams (*Corbicula fluminea*), are present in the mudflat and Delaware River next to the site. The invertebrates were considered as a single group for the purposes of this assessment. Benthic invertebrates may provide an important exposure pathway for higher trophic-level fish and wildlife species that feed in the intertidal and nearshore Delaware River habitats.

### 2.3 Life Histories of Selected Fish Species in the Delaware River

The following section, much of which was directly excerpted from Menzie-Cura (1993b), presents life history information for selected fish species present in the Delaware River. The profiles focus on the movements, preferred habitats, and feeding ecology of these species in order to understand their habitats and potential exposures in the Delaware River as they relate to the MBA site.

### 2.3.1 Shortnose Sturgeon (Acipenser brevirostrum)

The shortnose sturgeon is the only federally designated endangered fish near the Metal Bank site. Sturgeon populations within the Delaware River are thought to have declined in number due to overfishing and slaughter of fish caught in shad nets. In addition, shortnose sturgeon experience slow growth and late maturation. The historic area of low levels of dissolved oxygen in the Delaware River may have also hindered their recovery several years ago, although this no longer seems a limiting factor (Schick, personal communication, as cited in MenIe-Cura, 1993b). Presently, the adult population in the Delaware River is estimated between 6,000 to 14,000 shortnose sturgeon (Hastings, et al. 1987 as cited in Menzie-Cura, 1993b).

Delaware River shortnose sturgeon generally use the river from Lambertville into the Philadelphia area in a cyclic pattern (O'Herron, et al. 1993 as cited in Menzie-Cura, 1993b). They overwinter in a dense, sedentary aggregation from Burlington Island to Duck Island. The channel area off Duck Island (approximately 32 km upstream of the Metal Bank site), in particular, appears to be an important overwintering site (Brundage 1988; O'Herron, et al. 1993 as cited in Menzie-Cura, 1993b). From late March to April, spawning sturgeon are found in aggregations between Scudders Falls and Trenton Rapids, approximately 49 km upstream of the Metal Bank site. The post-spawning males and females move rapidly downstream into the Philadelphia area from April to May. During this period, some individuals have been captured as far downstream as Delaware Bay. Some sturgeon remain in the Philadelphia area throughout the summer, many return upriver within a few weeks, while others gradually move upriver over the course of the summer. By November the aggregation of shortnose sturgeon has again formed near Duck Island (O'Herron, et al. 1993 as cited in Menzie-Cura, 1993b).

Shortnose sturgeon feed indiscriminately upon bottom organisms. Food items of juvenile and adult fish include polychaete worms, mollusks, crustaceans, aquatic insects, and small bottomdwelling fishes (Gilbert 1989). Adult shortnose sturgeon feed off benthic or plant surfaces. They generally feed in shallow water at night or when turbidity is high. Shortnose sturgeon appear to prefer feeding in freshwater with shallow, muddy bottoms and abundant macrophytes. In the summer, adults feed in deeper water, possibly in response to water temperature, in areas of little or no current. Larval and juvenile shortnose sturgeon appear to prefer deep channels. Juveniles may prefer a sand-mud or gravel-mud substrate.

A study conducted on the Connecticut River (Buckley and Kynard 1985; as cited by Gilbert 1989) suggests water velocity and depth may be more critical than substrate in determining

#### MB Aquatic ERA

spawning locations. Spawning typically occurs in deep areas where water velocities are between 0.4 and 1.3 meters per second (m/s) and the substrate is a combination of gravel, rubble and cobble. Female sturgeon in Connecticut begin spawning between ages 8 to 12, and males slightly earlier (Gilbert 1989). The interval between successive spawnings by the same individual sturgeon may range from 4 to 12 years. Shortnose sturgeon are a long-lived species, with oldest reported ages from individuals from northern rivers ranging between 34 and 67 years.

The release of the eggs in the proper water velocity may be critical to egg deposition and survival. If the water velocity is too great, the eggs may not adhere to the substrate before losing their adhesiveness. If the velocity is too low, the eggs may clump and result in increased mortality from respiratory stress, fungus growth, or potential egg predation.

### 2.3.2 White Perch (Morone americana) excerpted from Menzie-Cura, 1993b

White perch is an important fisheries species and is present near the site. White perch thrive in a variety of habitats. They are found throughout the Delaware River (from the mouth north to the Musconetcong River); however, they are uncommon in the lower part of the estuary. Most of the tidal tributaries of the Delaware River contain white perch populations; some may support discrete populations.

The average lifespan of white perch is 5 to 7 years. In the Delaware River, the maximum age is 8 to 10 years. Males reach sexual maturity between 2 and 3 years; females between 2 and 4 years.

In the Delaware River, items most commonly eaten by white perch are the aquatic invertebrates *Neomysis americana*, *Gammarus*, and copepods. Adults may also eat other fish. Young-of-the-year eat microplankton, and as they grow eat aquatic insects. White perch move nearshore at night to feed, returning to deeper water at dawn.

White perch are anadromous and migrate upstream in the spring to spawn in fresh water and oligohaline areas, returning to deeper brackish water in the fall and winter. White perch spawn at water temperatures of 16- to 24-degrees C (average of 21°C). In the Delaware River, this occurs from late March to early June when the fish move into spawning areas upstream of Newbold Island to as far north as Lambertville. This range is approximately 10 to 32 km upstream of the MBA site. Spawning also occurs in tributary creeks.

Spawning usually takes one to two weeks, and the female does not release all her eggs at once. Spawning usually occurs in shallow water with gravel shoals or sparse submerged vegetation. Males and females release sperm and eggs together. The eggs then become attached to vegetation and rocks. The number of eggs released can vary from 20,000 to greater than 300,000.

The incubation period for the eggs is inversely related to temperature. The hatched larvae are planktonic and can drift downstream or up and downstream with the tide. Development from the larval to juvenile stage takes approximately six weeks. During this time, the young white perch move to shallow water nursery areas where they spend the summer. In the fall, they leave the tributary and nursery areas for deeper more saline waters. Many of the young-of-the-year overwinter north of Salem, New Jersey, 65 km downstream of Philadelphia.

### 2.3.3 Channel Catfish (Ictalurus punctatus)

The reported home range and movement of channel catfish vary by investigator. McCammon (1956) as cited in Menzie-Cura, 1993b, reported highly restricted movements by most catfish tagged in the lower Colorado River. However, Hubley (1963) as cited in Menzie-Cura, 1993b, observed that only 24 percent of tagged channel catfish were characterized as "local" (within 3.2 km of release site) in the upper Mississippi River. Hubley observed one catfish travel 180 km over a 36-day period. The differences in movement may be in response to available habitat and food.

Young-of-the-year channel catfish feed predominantly on plankton and aquatic insects. The adults are opportunistic feeders; their diet includes: terrestrial and aquatic insects, detrital and plant material, crayfish, and mollusks. Fish may be a significant part of the diet for large catfish.

Feeding is conducted usually at night through vision and chemosenses. The channel catfish may feed both off the bottom and in the water column, although the former is more characteristic. They typically move into the shoreline at night to feed. Optimum riverine conditions have been described as locations with warm temperatures and a diversity of velocities; depths; structural features such as debris, logs, cavities, and boulders that provide cover. The largest populations of channel catfish in the Delaware were found in moderate- to slow-flowing pools and backeddies over bottoms of gravel, rubble, and bedrock (Mihursky 1962 as cited in Menzie-Cura, 1993b).

2-9

#### MB Aquatic ERA

AR300922

Channel catfish probably spawn in the Delaware River from May through July, as evidenced by the occurrence of small young. Most, if not all, spawning occurs in nontidal fresh water since small young (less than 30 millimeters [mm]) were extremely scarce in the tidal waters of the Delaware (Wang and Kernehan, 1979 as cited in Menzie-Cura, 1993b).

Spawning is greatly inhibited if suitable nesting cover is unavailable. Dark and secluded areas are required for nesting. Males build and guard the nests in a variety of protective covers. In large rivers such as the Delaware, channel catfish are likely to move into shallow, flooded areas to spawn. Newly hatched fry remain at the nest for seven to eight days, then they move to shallow arms containing cover.

### 2.3.4 Eastern Silvery Minnow (Hybognathus regius) (from Menzie-Cura, 1993b)

The silvery minnow is primarily a year-round resident of the tidal freshwater portion of the Delaware estuary, although some specimens have been taken from salinities as high as 8.3 ppt. Young and adults were found in the shore zones of tidal creeks and mainstem of the Delaware River (Walton and Patrick 1973 as cited in Menzie-Cura, 1993b); only larger individuals were found in deeper channel waters (Wang and Kernehan 1979 as cited in Menzie-Cura, 1993b).

Spawning occurs in shallow inshore waters usually over sand and gravel in tidal creeks. The eggs are nonadhesive. Approximately two weeks after hatching, the young congregate into small schools near shore among emergent vegetation. Fast growing individuals may mature at age 1 (Raney 1942). In the Delaware River estuary, large schools of adults are found on spawning grounds in the upstream areas or tidal creeks during April and May at water temperatures of 12 to 20 °C (Wang and Kernehan 1979 as cited in Menzie-Cura, 1993b).

The total length of individuals in September is approximately 41 to 71 mm at age 0, 64 to 89 mm at age 1, and 76 to 97 mm at age 2 (Raney 1942 as cited in Menzie-Cura, 1993b).

Examinations of stomach contents indicate that the diet of the silvery minnow consists primarily of bottom ooze and algae (Flemer and Woolcott 1966 as cited in Menzie-Cura, 1993b).

2-10

## **3 CONTAMINANTS OF CONCERN**

The initial step in a site-specific ecological risk assessment is to identify the potential contaminants of concern (COC) to be evaluated in the assessment. This section presents the methodology used to accomplish this step. Including a chemical as a potential COC does not imply that the chemical is posing a risk to ecological receptors in the Delaware River and the mudflat next to the site, but only that it is being considered as part of the evaluation process.

### 3.1 Selection Criteria for Contaminants of Concern

COCs for this risk assessment were selected by evaluating the data from the Metal Bank site for sufficiency and by comparing the data to existing criteria relating to aquatic environmental health. Contaminant levels in water were compared to freshwater chronic ambient water quality criteria (AWQC; EPA 1992b). Because aquatic organisms would not be directly exposed to groundwater, contaminant concentrations were reduced by an appropriate dilution factor, based on groundwater and river flow, prior to this comparison.

There is very little information regarding the toxicity of contaminated sediments. No criteria similar to the AWQC are available. Long and Morgan (1991) assembled data from a large number of different studies on the concentrations of inorganic and organic contaminants in sediments associated with a variety of adverse effect endpoints. The studies included the results from synoptically collected sediment chemistry and biological data, spiked sediment bioassays, interim equilibrium partitioning (EP) values, and Apparent Effects Threshold (AET) values. Using the effects concentrations observed or predicted by these various methods, the lower tenth percentile (Effects Range-Low or ER-L) and the median concentrations (Effects Range-Median or ER-M) were calculated. After the initial effort, additional studies were added to the Long and Morgan database resulting in changes to some of the ER-Ls and ER-Ms (Long and MacDonald 1992). The ER-L and ER-M concentrations were proposed as weight-of-evidence based guidelines for determining contaminant-specific sediment concentrations below which adverse biological effects would be unlikely (ER-L) and above which effects would be probable (ER-M; Long and Morgan 1991; Long and MacDonald 1992).

The AET approach to developing sediment criteria was developed by EPA Region 10 for Puget Sound (Washington) and is currently the basis for sediment quality standards for the State of Washington. The AET approach relates the chemical concentrations in field-collected sediments to at least one biological indicator of injury (e.g., sediment bioassays or altered benthic infauna abundance) to determine the concentration of the contaminant above which biological effects were *always* observed (PTI 1988).

It should be noted that, by definition, the AET is a less conservative estimator of risk than the ER-M because above the AET, effects were *always* observed; above the ER-M, effects are probable but not assured. The exact degree of probability varies with each contaminant.

### **MBAquatic ERA**

A tiered approach was used to select the COCs. Analytes were eliminated from further consideration as a COC if:

- There was insufficient data to make an evaluation.
- An analyte was not detected in the medium being evaluated, or was detected only once.
- In groundwater: following dilution by the Delaware River, the maximum analyte concentration would result in surface water concentrations less than the chronic AWQC for the protection of freshwater organisms (EPA 1992b).
- In surface water: the maximum analyte concentration was less than the chronic AWQC for the protection of freshwater organisms (EPA 1992b).
- In sediment: the maximum analyte concentration was less than the ER-L (Long and MacDonald 1992).

### 3.2 Contaminants of Concern in Groundwater and Surface Water

Within the scope of this aquatic ecological risk assessment, groundwater becomes a concern when it enters the Delaware River and becomes surface water. Therefore, prior to evaluation as potential COCs, the maximum concentration for each analyte (Table 3-1) was divided by a dilution factor representing the estimated groundwater discharge rate and low-flow conditions in the Delaware River. Comparison of the estimated concentrations of the COCs in surface water, based on groundwater input, to the freshwater chronic AWQCs (EPA 1992b) indicates that none of the COCs exceed their respective chronic AWQC. Based on this analysis the contribution of site related groundwater to surface water exposure to polynuclear aromatic hydrocarbons (PAHs), 4,4'-DDD, 4,4'-DDE, bis (2-ethylhexyl) phthalate (also known as di (2-ethylhexyl) phthalate or DEHP), butylbenzyl phthalate, diethyl phthalate, dimethyl phthalate, di-n-butyl phthalate (DBP), di-n-octyl phthalate (DOP), arsenic, cadmium, chromium, copper, lead, mercury, silver, and zinc is expected to be negligible due to concentrations of these COCs in groundwater and their dilution upon entering the Delaware River. While total polychlorinated biphenyls (PCB) concentrations were also well below their chronic AWQC concentration, because they are of major concern at the site it was thought necessary to evaluate their contribution from groundwater. Therefore PCBs will be treated as a COC in groundwater.

Because surface water analysis was restricted to PCBs and pesticides from pooled water in the mudflats, surface water concentrations for potential organic and inorganic contaminants were estimated based on dilution of groundwater concentrations (see above). Based on these calculated concentrations and the measured concentrations for PCBs and pesticides (the vast majority were below detection) only total PCBs will be considered (Table 3-1).

### MB Aquatic ERA

r

Table 3-1.Concentrations and frequency above detection limits (DL) of the potential contaminants of concern in groundwater, surface water ( $\mu g/l$ ) and non-aqueous phase liquid (NAPL) (mg/l) at the Metal Bank site (HMM 1993b) compared to the chronic AWQC for the protection of freshwater organisms (EPA, 1992b).

	Gre	oundwa	iter			ater Se	ep		Sur	face W	ater			
Chemical	Min <sup>.</sup>	Max	Freq	Min	Max	Freq	Freq	Min	Max	Freq	Freq	Oil	AWQC	NAPL
	μg/l	μg/l	Above DL	μg/l	μg/l	Above DL	Above AWQC	μg/l	μg/l	Above DL	Above AWQC	μg/1	μg/l	mg/l
Total PCBs	<0.5- <1	26	3/30	<0.5- <1	3.7	1/3	1/1	<0.5- <1	ND	0/4		<36- <71	0.014	1100
Total PAHs	6	2100	22/35	NT	NT			NT	NT			NT	NA	87
Acenapthene	1 (<10)	110	16/35										520*	<750
Fluoranthene	2 (<10)	200	19/35						]				3980*	<750
Naphthalene	1 (<10)	1500	17/35										620*	<750
Phenanthrene	2 (<10)	240	17/35										6.3p	<750
Pyrene	2 (<10)	160	20/35										NA	87
Pesticides										1				
DDT	<0.1- <0.5	0.52	2/16	<0.1- <0.2	ND	0/3		<0.1	ND	0/4		, <7.1	0.001	<48
DDE	<0.1- <0.5	59	9/16	<0.1	2.3	1/3	0/1	<0.1	0.02	1/4	0/1	<7.1	1050*	<48
DDD	<0.1- <0.5	23	8/16	<0.1	0.68	1/3	1/1	<0.1	ND	0/4		<7.1	0.6*	<48
Phthalates Bis (2-ethylhexyl) phthalate	0.13 (<10)	240	21/36	NT	NT			NT	NT			NT	360p	590
Butylbenzyl phthalate	1 (<10)	8	16/36	-									3*c	
Diethyl phthalate	7	22	6/36										3*c	
Dimethyl phthalate	<10	ND	0/36										3*c	
Di-n-butyl phthalate	1	11	9/36										3*c	
Di-n-octyl phthalate	0.014	4	4/36										3*c	

### Table 3-1. Continued.

Groundwater					Groundwater Seep				Sur					
Chemical	Min	Max	Freq	Min	Max	Freq	Freq	Min	Max	Freq	Freq	Oil	AWQC	NAPL
	11 11	11.0/1	Above DL	11 0/1	110/1	Above DL	Above AWQC	u σ/1		Above DL	Above AWQC	11.0/1		
	μg/l	µg/l	12	μg/l	µg/l		Ange	µg/l	µg/l	11	And	_μg/l	μg/l	mg/l
Trace Elements				NT	NT			NT	NT			NT		
Arsenic	0.9 (<10)	370	27/38										190	<0.18
Cadmium	<2.8- <5	11	6/16										1.1+	NT
Chromium	<4- <10	290	26/28										11	NT
Copper	9.6 (<25)	1200	26/27			1							12+	NT
Lead	ົ<3໌	1400	25/35										3.2+	NT
Mercury	<0.2	22	17/44										0.012	<0.1
Nickel	<10- <40	172	22/32										160+	NT
Silver	<2.3- <10	ND	0/31										0.12	NT
Zinc	3.2 (<20)	2200	41/41										110+	NT

NT: Not tested

NA: No criterion available

Less than value in () indicates a detection limit higher than the lowest reported detected concentration p: Proposed

c: Value for chemical class

: Insufficient data to develop criteria, the listed concentration is the acute lowest observed effect level

+: Hardness dependent criteria (based on 100 mg CaCO<sub>3</sub>/l)

### 3.3 Contaminants of Concern in Non-Aqueous Phase Liquid (NAPL)

The NAPL layer at the site is of concern as a potential continuing source of contamination for adjacent sediments and surface water if it is permitted to migrate off the site. A sample of the NAPL layer was taken from Monitoring Well (MW) 6 during October 1991 and analyzed for total PCBs; total PAHs; DEHP; 4,4'-DDT; 4,4'-DDE; and 4,4'-DDD. Total PCBs, a single PAH (18 individual PAHS were analyzed for), and DEHP were detected in high concentrations. While the remaining individual PAHs; phthalates; 4,4'-DDT; 4,4'-DDE; and 4,4'-DDD; 4,4'-DDE; and 4,4'-DDD were all below the detection limits, the detection limits were extremely high. Because of the excessively high detection limits and lack of a method for estimating the concentrations of the other phthalates; 4,4'-DDT; 4,4'-DDE; and 4,4'-DDD, they were excluded from consideration as COCs in the NAPL. Therefore, the COCs in the NAPL layer that will be addressed in this risk assessment are total PCBs, total PAHs, and DEHP.

### 3.4 Contaminants of Concern in Sediments

The potential COCs for the Delaware River, mudflat, and riprap sediments are PCBs, PAHs, polychlorodibenzo-p-dioxins (PCDDs), polychlorodibenzofurans (PCDFs), DDT, phthalates and trace elements (Table 3-2).

Two riprap sediment samples were analyzed for PCDDs and PCDFs (Table 3-2). The 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) toxicity equivalency (TEQ) concentration for each PCDD and PCDF were calculated (EPA 1989a) and summed to produce a maximum total TEQ of 0.303 ng/kg. The TEQ was below the concentration of 60 ng/kg in sediment reported by EPA (1993) to present low risk to fish. PCDDs and PCDFs were eliminated as potential COCs.

Total PCBs, total PAHs, 4,4'-DDE and 4,4'-DDD were found at elevated concentrations in numerous sediment samples, while 4,4'-DDT was only detected in one of 21 samples. However, the single detection of 4,4'-DDT exceeded both the ER-L (1  $\mu$ g/kg) and the ER-M (7  $\mu$ g/kg), and the detection limits for the other samples were extremely high (19-4000  $\mu$ g/kg). Because of the high detection limits, there is no justification in considering that because 4,4'-DDT was not detected it was not present at elevated concentrations. The phthalates, DEHP, DBP and DOP were detected in several sediment samples in concentrations above their respective AET concentrations. Therefore, the organic COCs in sediment that will be addressed in this risk assessment are: total PCBs; total PAHs; 4,4'-DDT; 4,4'-DDE; 4,4'-DDE; 4,4'-DDD; DEHP; DBP; and DOP.

Only seven stations (two in the river and five in the mudflat) had sediment samples analyzed for the trace elements arsenic, cadmium, chromium, copper, lead, mercury, nickel, silver, and zinc. Two river-station samples (US1 and US2) and one mudflat station (DS1 with a duplicate) sample from the June 1991 sampling round (Round 1) were analyzed for trace elements. All trace elements, except silver, were detected in all four samples; silver was detected in only one sample. Samples from four mudflat stations (MF3, MF9, MF10 and MF11 with a duplicate) collected during the October 1991 sampling round (Round 2) were analyzed for trace elements. Only cadmium was reported as being above detection; however, the detection limits for each trace element exceeded their respective ER-Ls and often their ER-Ms. Because these excessively high detection limits resulted in all of the trace elements, with the exception of cadmium, being detected only at the extremes of the study area (US1 &2, DS1) there is no way to evaluate the potential risk of trace elements in the intervening area nor does the data permit relating trace element concentrations to the Metal Bank site. Therefore, cadmium will be the only trace element to be treated as a COC in sediments for this risk assessment. This does not mean that other trace elements do not pose a risk it just means their risk cannot be evaluated.

Table 3-2. Concentrations of the potential organic and inorganic contaminants of concern reported in mudflat, riprap, and Delaware River sediments (HMM 1993b) compared to their respective ER-L, ER-M, or AET concentrations (PTI 1988; Long and Morgan 1991; Long and MacDonald 1992).

	1993 Sampling Data						1991	Screening Values				
Chemical	Min	Max	Freq Above Dl	Freq Above	Freq Above ER-M or AET	Min		Freq Above Dl	Freq Above	Freq Above ER-M or AET	ER-L	ER-M /AET
Organic Chemicals			<u>~~ \</u>					~1				
Total PCBs (mg/kg)	<0.25- <8.1	16	9/11	9/9	6/9	<0.095 -<6.3	14	22/44	22/22	21/22	0.023	0.18/
Total PAHs (mg/kg)	0.62	240	15/15	11/15	5/15	0.46	920	8/8	5/8	.2/8	4	45/
Pesticides (µg/kg) DDT	NT	NT	NT			<19-	16	1/40	. /1			- /
DDE						<4000 <19-	16	1/40	1/1	1/1 '	1	7/
						<630 <19-	500	13/40	13/13	11/13	2.2	27/
DDD						<630	470	14/40	14/14	14/14	2	20/
PCDD/PCDF (ng/kg) 2,3,7,8-TCDD Total TCDD Total TCDD Total PeCDD Total HxCDD OCDD 2,3,7,8-TCDF Total TCDF Total TCDF Total PeCDF Total HxCDF Total HpCDF OCDF TEQ Phthalates (mg/kg)	NT	NT	NT			<0.015 0.01 <0.042 0.11 0.18 1.602 0.061 0.12 0.5 0.4 0.59 0.249 0.009	ND 1.826 0.06 0.52 1.91 4.33 0.403 1.11 6.94 15.08 22.83 6.14 0.303	0/2 2/2 1/2 2/2 2/2 2/2 2/2 2/2 2/2 2/2 2	NA NA NA NA NA NA NA NA	NA NA NA NA NA NA NA NA NA NA O/2	NA	60*
Bis(2-ethylhexyl) phthalate	0.067 (<0.45- <5.1)	1900	14/17	-	6/14	0.19 <0.6-<5	8.5	6/10	ŇA	2/6		/1.3
Butylbenzyl phthalate	<0.41- <8	0.11	1/17		1/1	<0.41- <5	ND	0/10	NA			/0.063
Diethylphthalate	<0.41- <8	ND	0/17			<0.41-	ND	0/10	NA			/0.2
Dimethylphthalate	<0.41- <8	ND	0/17			<0.41-<5	ND	0/10	NA			/0.071
Di-n- butylphthalate	0.15 (<0.41- <8)	2.5	2/17		1/2	0.054 (<0.6- <5)	0.098	4/10	NA	0/4		/1.4
Di-n- octylphthalate	-0.28 (<0.45- <8)	130	4/17		1/4	0.19 (<0.48- <5)	0.64	2/10	NA	0/2		/6.2

3-6

#### Table 3-2. continued.

		1993 Sampling Data				1991 Sampling Data				1	Screening Va	
Chemical	Min	Max	Freq Above Dl		Freq Above ER-M or AET	Min	Max	Freq Above Dl		Freq Above ER-M or AET	ER-L	ER-M /AET
<u>Trace Elements</u> (mg/kg)	ΝΓ	NT	NT	NT	NT							
Arsenic	`					1.1 (11- <59)	290	4/9	1/4	1/4	8.2	70/
Cadmium						(<1.55 -<26)	330	6/8	6/6	5/6	1.2	9.6/
Chromium						21 (<23- <120)	36	4/9	0/4	0/4	81	370/
Copper						(<11- <59)	695	4/9	4/4	0/4	34	270
Lead						<110- <590	2030	4/9	4/4	2/4	47	223/
Mercury						0.13 (<23- <120)	0.55	4/9	3/4	0/4	0.15	0.71
Nickel						20 (<110- <590)	35	4/9	3/4	0/4	21	52/
Silver						<0.69- <59	490	1/9	1/1	1/1	1	3.7/
Zinc	,					201 (<230- <1200)	1110	4/9	4/4	1/4	150	410

NT: Not tested

NA: No ER-L, ER-M, or AET have been developed for PCDDs and PCDFs

Polychlorinated dibenzo-p-dioxins PCDD:

PCDF: Polychlorinated dibenzofurans

TCDD: TetraCDD

PentaCDD PeCDD:

HexaCDD HxCDD:

HpCDD: HeptaCDD

OCDD: OctaCDD

TEQ: 2,3,7,8-TCDD toxic equivalency concentration (EPA 1989a) \* Value reported by EPA as a low risk to fish (EPA 1993)

••

4

## **EXPOSURE ASSESSMENT**

This section determines the routes and magnitudes of exposure to the COCs present at or migrating from the Metal Bank site. Results of this exposure assessment are combined with chemical-specific toxicity information developed in the toxicity assessment to characterize risks to ecological receptors. In general, concentrations will be reported to two significant figures.

The key components of the exposure assessment include:

- Identifying potential exposure pathways; and
- Estimating exposure-point concentrations and identifying important exposure contact areas.

#### 4.1 Exposure Pathways at Metal Bank of America

Transformer salvage operations were conducted at the MBA site between 1968 and 1973. Transformers were disassembled on a concrete pad and PCB-contaminated oil from the transformers was drained into a 37,850-liter underground storage tank (BCM 1988). Spills of oil and rupture of the underground storage tank contaminated soils and groundwater on the site. It is estimated that between 44,000 and 175,000 liters of PCB-contaminated oil infiltrated groundwater beneath the site (ICF Clement 1987). NAPL was detected in monitoring wells as early as 1977 with a PCB concentration of 1,080 mg/kg and as late as 1991 with a PCB concentration of 1,090 mg/kg (Weston 1978; HMM 1992). Subsequently, contaminated groundwater and oil product seeped into the intertidal and nearshore areas of the Delaware River, contaminating sediments and surface water (BCM 1988). Oil seeps discharging from the site to the Delaware River were reported as early as 1972 (HMM 1993). A sample collected from an oil slick on the Delaware River in 1973 was reported to have a PCB concentration of 800 mg/kg (Weston 1978).

An oil recovery and treatment system operated at the site from 1983 until April 1989 and recovered approximately 16,000 liters of oil by January 1987 (ICF Clement 1987; CDM 1989). In 1989 EPA determined that, though the system as designed had collected all recoverable oil, underground oil was still present at the site (CDM 1989).

The elevated concentrations of PAHs, phthalate esters, and the trace elements copper, lead, mercury, and zinc reported in soil and groundwater from the site may be attributable to combustion products from the burning of the insulation on electrical wire as part of the metal refabrication activities from 1968 to 1972. (HMM 1993).

The primary pathways of transport of site-related contaminants from the MBA site to the mudflat and Delaware River are through discharge of contaminated groundwater and NAPL.

### 4.1.1 Fate and Transport Analysis of COCs at MBA

This section discusses the fate of COCs from MBA after their discharge to the environment. The chemical and physical characteristics of the COCs as well as those of the environment in which they exist will determine their action in the environment as well as their availability to receptor organisms.

## 4.1.1.1 PCBs

As a result of their highly lipophilic nature and low water solubility, PCBs are generally found to have low concentrations in water and relatively high concentrations in sediments. Individual PCB congeners have different physical and chemical properties based on the degree of chlorination and position of chlorine substitution, although differences in the degree of chlorination are usually more significant (Phillips 1986). Vapor pressure and water solubility are inversely related to degree of chlorination (Erickson 1985; Phillips 1986). Octanol-water partition coefficients, which are often used as estimators of the potential for bioconcentration, are highest for PCB congeners with the highest degree of chlorination. Solubilities and octanol-water partition coefficients range over several orders of magnitude. Due to their higher water solubility, lower-chlorinated PCBs may show greater dispersion from a point source, while the higher-chlorinated components of a PCB mixture may remain in the sediments closer to the source (Phillips 1986).

The mobility of PCBs in sediment is also a function of the chlorine substitution pattern and degree of chlorination and is generally quite low, particularly for the higher chlorinated biphenyls (Fisher et al. 1983). As a result of this low mobility, without disturbance of the sediment or bioaccumulation, even low rates of sedimentation may prevent PCBs in the sediment from reaching the overlying water via diffusion (Fisher et al. 1983). The measurement of PCB concentrations in sediment is complicated by their non-uniform distribution on both vertical and horizontal scales.

PCB concentrations are also affected by physical characteristics of the sediment such as grain size and total organic carbon content (Pavlou and Dexter 1979; Lynch and Johnson 1982). Fine sediments generally contain higher concentrations of PCBs than coarser sediments, probably as a result of larger surface area on the finer sediments (Phillips 1986). The amount

of PCBs sorbed to sediments is also a function of the total organic carbon content of the sediment (Chou and Griffin 1986; Sawhney 1986).

The persistence of PCBs in the environment is a result of their general resistance to degradation. The rate of degradation of PCB congeners by bacteria decreased with increasing degree of chlorination (Furukawa 1986); other structural characteristics of the individual PCBs also affected susceptibility to microbial degradation to a lesser extent. Photochemical degradation, via reductive dechlorination, is also known to occur in aquatic environments; the higher chlorinated PCBs appear to be most susceptible to this process (Sawhney 1986). In addition, reductive dechlorination of the higher chlorinated PCB congeners by anaerobic bacteria in sediments has been reported (Brown et al. 1987). The overall significance of the different degradation pathways in terms of reduction of environmental PCB concentrations is not clear at this time.

PCBs in sediments can provide a significant source of contamination for aquatic organisms (Larsson 1984 and 1986; Lake et al. 1990; Connolly 1991; Ankley et al. 1992; Pruell et al. 1993). Bioaccumulation of PCBs from contaminated sediments may result from uptake from the interstitial or overlying water (via respiration), direct dermal sorption, ingestion of sediment, or indirectly through the food web (Rubenstein et al. 1984; Swartz and Lee 1980). The availability of PCBs in sediments to aquatic organisms depends on the concentrations of the specific PCBs present, physical properties of the sediment, environmental factors, and characteristics of the organisms. The presence of other contaminants may also influence the availability of PCBs (Shaw and Connell 1982). In higher trophic-level fish species, PCB exposure through the food web may serve as the major source of observed tissue concentrations (Thomann and Connolly 1984; Oliver and Niimi 1988).

The empirical relationship between PCB concentration in sediments and the resulting levels in resident biota has been investigated in a number of studies (Table 4-1). Bioaccumulation factors (BAF; biota [PCB]/sediment [PCB]) in freshwater invertebrates ranged from 1 to about 24, whereas BAFs in field-collected freshwater fish ranged from 0.13 to as high as 30 based on data normalized for lipids and TOC (biota [ $\mu$ g PCB/g lipid]/sediment [ $\mu$ g PCB/g TOC]; MacDonald et al. 1993). The BAF appears to increase with each increase in trophic level (MacDonald et al. 1993).

Organism	Location	BAF (Range)	Source
Invertebrates			
Chironomid larvae	Artificial ponds	4.2	Larsson 1984
	Field	2.9	
Oligochaete	Niagara River	3	Fox et al. 1983
Clam	Laboratory	2.4	Tatem 1982
	Field	6.1 (2.7-10.4)*	MacDonald et al. 1993**
Corbicula	Field	1.6 (0.52-4.13)*	Metal Bank
Crayfish	Field	11 (2.0-23.7)*	MacDonald et al. 1993
Prawn	Laboratory	1.1	Tatem 1982
Zooplankton	Field	5.0 (1.0-9.1)*	MacDonald et al. 1993
<u>Fish</u>			
Bluntnose minnow	Field	6.5 (1.6-13.8)*	MacDonald et al. 1993
Golden shiner	Field	2.7 (0.13-7.3)*	MacDonald et al. 1993
Silvery minnow	Field -	0.35 (0.07-0.58)*	Metal Bank <sup>†</sup>
Silvery minnow	Field	0.92 (0.19-1.5)*	Metal Bank <sup>††</sup>
Channel catfish	Field	0.25 (0.07-0.73)*	Metal Bank <sup>†</sup>
Channel catfish	Field	0.66 (0.18-1.9)*	Metal Bank <sup>††</sup>
Lake trout	Field	6.9 (3.8-10.7)*	MacDonald et al. 1993
Perch	Laboratory	2.7(1.5-3.9)*	Seelye et al. 1982
Smallmouth bass	Field	7.9 (3.8-15.5)*	MacDonald et al. 1993
Yellow perch	Field	14.2 (4.4-30)*	MacDonald et al. 1993

Table 4-1. Summary of bioaccumulation factors (ratio of the concentration of PCB in the organism to that in the sediment) from PCB-contaminated sediments.

\* Biota PCB concentration (lipid-based)-to-sediment PCB concentration (organic carbon- based).

\*\* MacDonald et al. (1993) refer to these ratios as BSF (biota sediment factor) rather than BAF.

<sup>†</sup> Based on nearfield PCB concentrations

<sup>††</sup> Based on farfield PCB concentrations (see text).



#### 4.1.1.2 Polynuclear aromatic hydrocarbons

Because of their relatively low water solubility, most PAHs are strongly adsorbed to particulate matter in aquatic systems and can accumulate in sediments and biota (Clement Associates 1985). As a result, PAHs can be found at considerable distances from their source, with fine particles containing PAHs transported by prevailing water currents (PTI 1988b).

PAHs are relatively insoluble in water, but the dissolved portion may undergo rapid, direct photolysis. Singlet oxygen is the oxidant, and quinones are the products in these reactions. Oxidation by chlorine and ozone may be an important fate process when these oxidants are available in sufficient concentrations (Clement Associates 1985).

Although PAHs are rapidly bioaccumulated, they are also quickly metabolized and eliminated from most organisms (shellfish are a known exception). Bioaccumulation, especially in vertebrate organisms, is usually short term, so it is not considered an important fate process in multicellular organisms. Biodegradation and biotransformation are probably the ultimate fate processes for PAHs. The available data suggest that the PAHs with high molecular weights are degraded slowly by microbes and readily metabolized by multicellular organisms. Biodegradation probably occurs more slowly in aquatic systems than in soil (Clement Associates 1985).

Because of the relatively low aqueous solubility of PAHs and their strong affinity for particulate matter in aquatic systems, sediments are the primary reservoir for PAHs. Once in the sediments, PAHs are subject to burial, resuspension, and degradation. The lowermolecular weight PAHs (three aromatic rings or less), because of their greater aqueous solubility and degradability, will tend to be lost in the weathering process so that the less labile, higher-molecular weight PAHs tend to predominate in sediments.

#### 4.1.1.3 DDT and Metabolites

4,4'-DDD (DDD) and 4,4'-DDE (DDE) are metabolites of 4,4'-DDT, a chlorinated hydrocarbon pesticide. DDD and DDE are highly persistent in the environment because of their low vapor pressure and resistance to degradation and photooxidation. Soils and sediments act as a primary reservoir for DDD and DDE. Estimates of the half-life for DDT biodegradation in soil range from 2 to >15 years (Clement 1992) These compounds are highly lipophilic, resulting in accumulation in tissue of aquatic organisms and biomagnification through the aquatic food web, so that the highest residue concentrations are often found in carnivorous predators at the top of the food chain. (Clement Associates 1985).

Sorption and bioaccumulation are the most important transport processes for DDT and its metabolites. Although it occurs slowly, the ultimate fate process for DDT and DDD is biotransformation to form bis (2-chlorophenyl) methanone. For DDE, direct photolysis is the most important ultimate fate process in the environment, although biotransformation may also be important (Clement Associates 1985).

### 4.1.1.4 Phthalates

Relatively little information is available on specific phthalate esters, but the environmental fate and transport of DEHP, DBP, and DOP can be largely inferred from data for phthalate esters as a group. Of the phthalate esters, DEHP is the most thoroughly studied. All three probably hydrolyze in surface waters, but at such slow rates that this process would not be significant under most conditions. Photolysis, volatilization, and oxidation do not appear to be important environmental fate processes, especially in aquatic systems (Clement Associates 1985).

Adsorption onto suspended solids and particulate matter, and complexation with natural organic matter are probably the most important environmental transport processes for DEHP, DBP and DOP. The high-log octanol/water partition coefficients for DEHP, DBP, and DOP (5.3, 5.2 and 9.2, respectively) suggest that these substances would readily adsorb onto particulate matter high in organic carbon. This contention is supported by the fact that phthalate esters are commonly found in freshwater and marine sediment samples. DEHP, DOP and, probably, DBP readily interact with fulvic acid present in humic substances in water and sediment, forming complexes that are very soluble in water (Clement Associates 1985).

Bioaccumulation by aquatic organisms is considered an important fate process for the phthalate esters. Biodegradation is also an important fate process in aquatic systems. DEHP, DBP, and DOP are biodegraded under most environmental conditions, and they can be metabolized by multicellular organisms. Long-term bioaccumulation and biomagnification are not likely to occur (Clement Associates 1985).

### 4.1.1.5 Cadmium

Cadmium, like most naturally occurring metals, is insoluble in water, however, its chloride and sulphate salts are readily soluble. cadmium exists in the aquatic environment as a divalent cation (+2 valence), and as the free dissolved cation it is readily bioavailable. The concentration of dissolved cadmium, and therefore its bioavailability is regulated in aerobic systems by the divalent cation's sorption to hydrous iron and manganese oxides, clay minerals, and organic matter. The efficiency of these materials in removing cadmium from solution

varies according to their compositions and concentrations; the pH and salinity of the water; the concentrations of complexing ligands; and the concentration of cadmium (Eisler 1985). Concentrations of cadmium in suspended and bed sediments always exceed concentrations in ambient water. In reducing environments, precipitation of cadmium sulfide limits the mobility of cadmium (DiToro et al. 1990). While the principal source of bioavailability of cadmium is the dissolved cation in surface and pore water, cadmium can also become available from ingested sediment particles, to which it is bound, by the reduced pH levels in the guts of some organisms (Green et al. 1993).

#### 4.1.2 Summary of Exposure Pathways.

Routes of exposure for PCBs that were identified for aquatic organisms in the Delaware River next to MBA include direct contact with NAPL, contaminated sediments and surface water, and ingestion of contaminated prey species with subsequent transfer through the food web. Because of the limited data available for the non-PCB contaminants of concern, direct exposure to the contaminated sediments and NAPL were the only pathways of exposure evaluated for PAHs and phthalates. Direct exposure to the contaminated sediments was the only pathway of exposure evaluated for DDT and its metabolites and cadmium.

### 4.2 Receptor Populations

Aquatic species in the Delaware River are the primary receptor species that are potentially impacted by COCs discharging to habitats in the river. Of particular concern are benthic and epibenthic species that spend a substantial portion of their life histories feeding and living in and on the sediments, which act as a sink for many contaminants. The Asiatic clam, channel catfish, and shortnose sturgeon are examples of benthic and epibenthic species that are found near MBA. Of special concern in the Delaware River is the federally-endangered shortnose sturgeon.

### 4.3 Exposure-Point Concentrations

Exposure point concentrations have been calculated for sediment, surface water, and NAPL. Figures 4-1 and 4-2 show the sampling station locations for the various rounds of sampling from which data was used to make these calculations. Of the three scenarios, exposure to contaminated sediment is expected to predominate in terms of duration and extent of exposure. Exposure to oil seeps could result in localized exposures to relatively high concentrations of PCBs or PAHs of short duration for mobile species such as fish, but less mobile species may

## AR300936

experience extended periods of contact with high concentrations of PCBs in the oil. Exposure by accumulation through the ingestion of contaminated food is discussed, but, because direct measurements are available for the concentrations of PCBs in receptor organisms, this route will not be quantified.

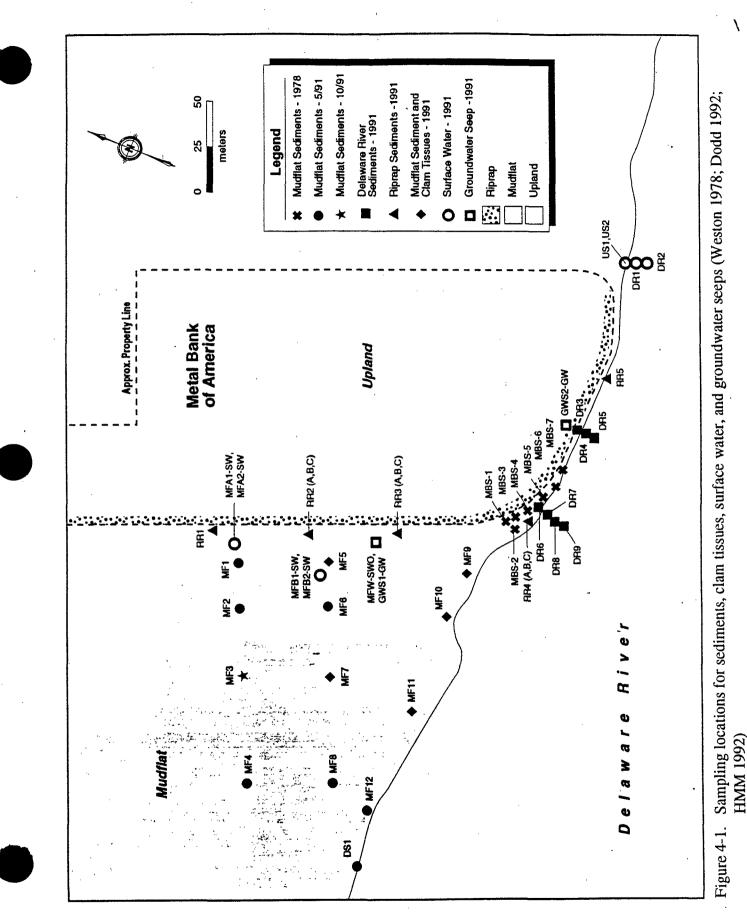
## 4.3.1 *PCBs*

PCBs were detected in samples from all environmental media tested (groundwater, groundwater seeps, NAPL and sediments) with the exception of surface water. No PCBs were detected in four surface water samples. However, the detection limits were relatively high, about two orders of magnitude above the AWQC (EPA 1992b) for PCBs.

#### 4.3.1.1 PCBs in Groundwater and Surface Water

Only limited data are available regarding the concentrations of PCBs in surface water at MBA. PCBs have been reported in oil slicks from the Delaware River as early as 1972 and from oil slicks in the mudflat since 1977 (Table 4-2). The Pennsylvania Department of Environmental Resources sampled water in the Delaware River 1.6 km below MBA in 1974 and 1975 and reported concentrations of PCBs as high as  $0.33 \ \mu g/l$  (Cottman Avenue PRP Group 1992). None of the site investigation studies have examined PCBs in Delaware River surface water. Surface water samples collected from pooled water on the mudflat at ebb tide in 1991 were reported to contain no PCBs, but elevated detection limits (up to 71  $\mu g/l$ ) precluded detection of low concentrations of PCBs in those samples (Table 3-1). A sample from a groundwater seep collected along the riprap wall bordering the mudflat in 1991 was reported to contain 3.7  $\mu g/l$  of PCBs (Table 3-1).

Concentrations of PCBs in the water column of the Delaware River resulting from groundwater discharge are expected to be negligible. The estimated groundwater discharge to the Delaware River is between 0.45 and 1.22 liters/second (l/s; HMM 1993). According to the Delaware River Basin Commission, the average discharge of the Delaware River is between 311,500 and 339,800 l/s. In all but the most extreme drought conditions, a minimum flow of approximately 85,000 l/s is maintained to prevent the migration upstream of salt water to a point where it threatens public groundwater supply (Swinick 1993).



MB Aquatic ERA

AR300938

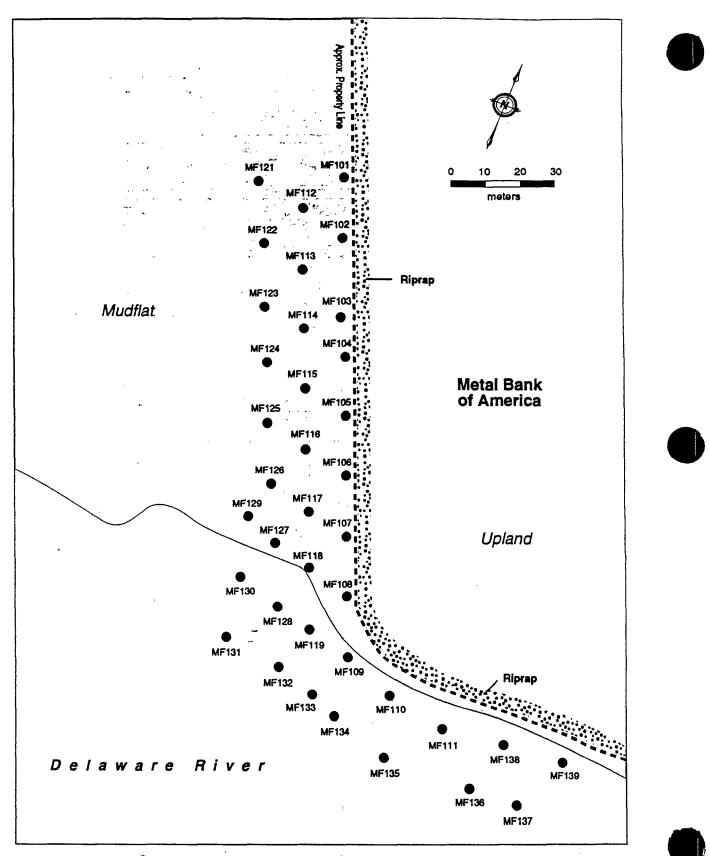


Figure 4-2. 1993 sediment sampling locations (HMM 1993a).

4-10

AR300939.

MB Aquatic ERA

L

For the purposes of estimating dilution of groundwater discharge, it was assumed that dilution would occur in a 15-meter wide envelope of the river surrounding the site. Since the Delaware River is approximately 900-meter wide near MBA, this represents 1.7 percent of the river width. In calculating dilution, for simplicity, it was assumed that the section of the river bed will hold the same portion of the river flow (Swinick 1993).

For estimating dilution, average and low-flow discharge conditions in the Delaware River will be used. The volume of the dilution water during average discharge conditions would be:

#### 0.017 (311,500 l/s) = 5,295 l/s

The dilution factors for average and maximum groundwater discharges would be 11,688 and 4350, respectively. Under low-flow conditions with a minimum river discharge of 85,000 l/s and average groundwater discharge of 0.45 l/s, the dilution factor would be 3,125 (Swinick 1993). It is expected that low-flow conditions would occur only occasionally and that average conditions would prevail most of the time.

Final estimated exposure-point concentrations for use in the risk characterization are based on contributions from PCBs discharged to the Delaware River via the groundwater and from NAPL seeps. Groundwater data collected in 1991 and 1992 reported detectable concentrations of PCBs from a total of two wells (out of approximately 15 wells sampled during each episode), monitoring wells MW-6 and MW-7 in the 1991 sampling episode and MW-6 in 1992 (HMM 1991 and 1992). The highest concentration of total PCBs ( $26 \mu g/l$  measured as Aroclors 1242 and 1260) was detected in MW-6 in 1991. If it is assumed that the other Aroclors were present at half of their respective detection limits, then the total PCB concentration in MW-6 would be 27  $\mu$ g/l. Mean and upper 95 percent confidence limit concentrations were calculated for total PCBs by combining the 1991 and 1992 groundwater data. Samples in which PCBs were reported below the detection limit were treated as if PCB was present at half the reported detection limit. The mean and upper 95 percent confidence limit concentrations for PCBs in groundwater are 4.18 and 6.17  $\mu$ g/l, respectively. Under average discharge conditions in the Delaware River, a dilution factor of 5,295 would result in PCB concentrations in the Delaware River ranging between 0.79 and 1.2 ng/l. Based on the groundwater dilution factor of 3125 for low flow conditions in the Delaware River (Section 4.3.1), the corresponding estimated concentrations of total PCBs in the water column of the Delaware River adjacent to the site would be between 1.3 and 2.0 ng/l. These values would represent the maximum expected concentrations of PCBs contributed to Delaware River water, in the immediate vicinity of the site, from site groundwater and are considered to be extremely

AR300940

Date	Location	Media Sampled	Concentration (ppm)
1972 1977	Delaware River Delaware River	Oil Slick Oil Slick	800 980
	On site	NAPL	1100-1600
	Mudflats	Sediment	620
•	Intertidal Area	Oil on Water	1100
1978	On-site	NAPL	780-1600
	On-site	Groundwater	ND-0.21
	Delaware River	Sediment	0.54-32

Table 4-2.	Summary of early	analytical data	from sampling	at Metal Bank of America
	(Weston 1978).	•		

conservative estimates. The resulting final concentrations of PCBs in Delaware River water is unknown due to lack of data on background concentrations in the river.

## 4.3.1.2 Exposure to PCBs in NAPL

During the most recent round of sediment sampling, while no NAPL was observed seeping from the site and attempts to sample the NAPL from groundwater were unsuccessful, a subsurface NAPL layer was observed in the lower riprap during low tide. As the tide rises, raising the groundwater elevation, this NAPL layer may be brought to the surface, making direct exposure to biota a possibility. Direct exposure to the NAPL, and the associated extremely high concentrations of PCBs, would be expected to occur in only localized areas, presenting the greatest risk to species with reduced mobility in the area of release. Earlier analytical data reported total PCB concentration in the NAPL ranging from 780 to 1,600 mg/l (mean = 1,200 mg/l) (Table 4-2) (Weston 1978), while the most recent data report the total PCB concentration to be 1,100 mg/l (Table 3-1) in the one sample of NAPL collected from monitoring well MW-6 (HMM 1992). Based on the recent data, it appears that the PCB concentration in the NAPL has changed little, if at all. Therefore, a conservative exposure point concentration of 1,200 mg/l will be used, a concentration that will be assumed to be representative of the PCB concentration in NAPL that may be currently released from the site.

## 4.3.1.3 Exposure to PCBs in Sediments

PCBs have been reported in sediments from the mudflat as early as 1977 and from the Delaware River as early as 1978 (Table 4-2; Weston 1978). The most recent sampling episodes (1991 and 1993) found PCBs in sediments from the mudflat and Delaware River

MB Aquatic ERA

(HMM 1992 and 1993). These data are summarized in Table 3-2. Sampling of Delaware River sediments has been confined to the mudflat and nearshore area immediately adjacent to the MBA site (Figures 4-1 and 4-2). The majority of the sediment samples collected in the mudflat and river during the 1991 and 1993 sampling episodes had detectable concentrations of PCBs, with concentrations as high as 16 mg/kg (Table 3-2). Because of the restricted area of sampling in the Delaware River (all river sampling stations were within about 40 meters of the site), the precise extent of PCB contamination in the river originating from the site has not been determined.

One of the most likely routes of exposure for fish and aquatic invertebrates using the Delaware River and the mudflat is through contact with PCB-contaminated sediments. Asiatic clams collected from the mudflat in 1992 and the sediments in which they were buried had elevated concentrations of PCBs, suggesting that the clams accumulated PCBs from the sediments. Several studies have demonstrated that benthic invertebrates, including the Asiatic clam; fish; and zooplankton accumulated PCBs when exposed to PCB-contaminated sediments (McLeese et al. 1980; Larsson 1986; Tatem 1986).

Calculation of an exposure-point concentration for sediment was based on sediment data collected in 1991 and 1993. After examination of the data it was decided to divide it into three areas: the riprap, nearfield mudflat and river, and farfield mudflat. The reasons for this division were two-fold: (1) there appeared to be a trend of decreasing PCB concentrations with increasing distance from the site; (2) all the reported river data and the majority of the mudflat data were from within 30 meters of the site; therefore, any overall mean would be biased by the near-site data and would not be representative of the mudflat as a whole. The 30-meter dividing line between nearfield and farfield was somewhat arbitrary; it included almost all of the 1993 mudflat and river data (except MF-129 and MF-130) and the first row of mudflat stations from 1991 in the nearfield. The 30-meter dividing line roughly corresponded to the drainage swale in the mudflat. To calculate the mean sediment concentration for the three areas (Table 4-3), sediment data were combined based on sampling station location, not station designation. and included all data from 1991 and 1993 sampling (including that generated by congener analysis). Where concentrations were reported as below detection then half the detection limit was used in the calculations. All other values used were as reported, whether or not they had qualifications associated with them. The quality of the available sediment data from the Delaware River was compromised by high detection limits for individual Aroclor mixtures. Of 23 sediment samples collected in the Delaware River, the quantification limits for Aroclors 1248 and 1254 in 11 of the samples in which no PCBs were detected ranged between 0.74 and 6.3 mg/kg.

## AR300942

Hansen, D.J., J.I. Lowe, A.J. Wilson, Jr., and P.D. Wilson. 1971. Chronic toxicity, uptake and retention of Aroclor 1254 in two estuarine fishes. *Bulletin Environmental Contamination* and Toxicology 6: 113-119.

Hansen, D.J., S.C. Schimmel, and J. Forester. 1974. Aroclor 1254 in eggs of sheepshead minnows: effect on fertilization success and survival of embryos and fry. Proc. Southeastern Assoc. Game Fish. Comm. pp. 805-812.

Hansen, D.J., S.C. Schimmel, and J.Forester. 1975. Effects of Aroclor 1016 on embryos, fry, juveniles, and adults of sheepshead minnows (*Cyprinodon variegatus*). *Transactions. American Fisheries Society 3*: 584-588.

Hansen, L.G. 1987. Food chain modification of the composition and toxicity of polychlorinated biphenyl (PCB) residues. *Rev. Environ. Toxicol.* 3: 149-212.

Hansen, L.G, W.B. Wiekhorst, and J. Simon. 1976. Effects of dietary Aroclor 1242 on channel catfish (*Ictalurus punctatus*) and the selective accumulation of PCB components. J. Fish. Res. Board Can. 33: 1343-1352.

Hasler, A. and W. Wisby. 1958. The return of displaced Largemouth Bass and Greeen Sunfish to a "home" area. *Ecology* 39(2)::289-293.

Hermanson, M. 1992. Memo on PCB congener analysis. Academy of Natural Sciences.

HMM Associates, Inc. 1992. Final draft report: Cottman Ave. NPL Site, data summary document. Prepared for the Cottman Ave. PRP Committee. 15 pp. + appendices.

HMM Associates, Inc. 1993a. Background data report for the Metal Bank/Cottman Avenue RI/FS. Prepared for the Cottman Avenue PRP Group.

HMM Associates, Inc. 1993b. Validated analytical data package: 1993 sampling program, Metal Bank/Cottman Ave. NPL Site, Philadelphia, PA.

HMM Associates, Inc. 1994. Letter to Cesar Lee: Comments on US EPA Draft Aquatic Ecological Risk Assessment for the Metal Bank/Cottman Avenue NPL site. January 14, 1994.

Hogan, J.W. and J.L. Brauhn. 1975. Abnormal rainbow trout fy from eggs containing high residues of a PCB (Aroclor 1242). *Progr. Fish. Cult.* 37:: 229-230.

Holm, G., L. Norrgren, T. Andersson, and A. Thuren. 1993. Effects of exposure to food contaminated with PBDE, PCN or PCB on reproduction, liver morphology and cytochrome P450 activity in the three-spined stickleback, *Gastersoteus aculeatus*. Aquatic Toxicology 27: 33-50.

Howard, D.E. & R. D. Evans. 1993. Acid-volatile sulfide (AVS) in a seasonally anoxic mesotrophic lake: seasonal and spatial changes in sediment AVS. *Environmental Toxicology and Chemistry* 12:1051-1057.

Hubley, R. 1963. Movement of tagged Channel Catfish in the Upper Mississippi River. *Transactions. American Fisheries Society* 92:165-168. Huggett, R.J., M.E. Bender, and M.A. Unger. 1987. Polynuclear aromatic hydrocarbons in the Elizabeth River, Virginia. In: Dickson, K.L., A.W. Maki, and W.A. Brungs (eds.). *Fate and Effects of Sediment-Bound Chemicals in Aquatic Systems*. Proceedings of the Sixth Pellston Workshop, Florissant, CO, August 12-17, 1984. Soc. Environ. Toxicol. Chem., Spec. Pub. Ser. pp 327-341.

ICF-Clement Associates. 1987. Final task 3 report. Endangerment assessment, feasibility evalulation, Metal Bank site. Philadelphia, PA. Phildelphia, PA: Prepared for the U.S. Environmental Protection Agency.

Ito, Y. 1973. Studies on the influence of PCB on aquatic organisms—III. Relationship between the intake of PCB and its accumulation in various tissues of carp. *Bull. Jap. Soc. Sci. Fisheries.* 39: 1139-1143.

Kannan, N., S. Tanabe and R. Tatsukawa. 1988. Toxic potential of non-ortho and monoortho coplanar PCBs in commercial PCB preparations: "2,3,7,8-T4 CDD toxicity equivalence factors approach". *Bulletin. Environmental Contamination and Toxicology* 41: 267-276.

Kaufmann, M. 1993. Personal Communication. Area Fisheries Manager, Lower Delaware drainage and lower Susquehanna drainage, Pennsylvania Fish Commission. Revere, PA. March 8.

Kemp, P.F. and R.C. Swartz. 1986. Response of the Phoxocephalid amphipod, *Rhepoxynius abronius*, to a small oil spill in Yaquina Bay, Oregon. *Estuaries* 9(4B) 340-347.

Lake, J.L., N.I. Rubinstein, H. Lee II, C.A. Lake, J. Heltshe, and S. Pavignano. 1990. Equilibrium partitioning and bioaccumulation of sediment-associated contaminants by infaunal organisms. *Envrionmental Toxicology and Chemistry* 9: 1095-1106.

Landrum, P.F., B.J. Eadie and W.R. Faust. 1991. Toxicokinetics and toxicity of a mixture of sediment-associated polycyclic aromaitc hydrocarbons to the amphipod *Diporeia* sp. *Envrionmental Toxicology and Chemistry* 10: 35-46.

Larsson, P. 1984. Transport of PCBs from aquatic to terrestrial environments by emerging chironomids. *Environmental Pollution 34A*: 283-289.

Larsson, P. 1986. Zooplankton and fish accumulate chlorinated hydrocarbons from contaminated sediments. *Canadian Journal of Fisheries and Aquatic Sciences* 43: 1463-1466.

Larrson, P. and A. Thurén. 1987. Di-2-ethylhexylphthalate inhibits the hatching of frog eggs and is bioaccumulated by tadpoles. *Environmental Toxicology and Chemistry* 6: 417-422.

Lidman, U., L. Forlin, O. Molander, and G. Axelson. 1976. Induction of the drug metabolizing system in rainbow trout (Salmo gairdnerii) liver by polychlorinated biphenyls (PCBs). Acta pharmacol. et toxicol. 39: 262-272.

Long, E.R. and L.G. Morgan. 1991. The potential for biological effects of sediment-sorbed contaminants tested in the National Status and Trends Program. NOAA Technical Memorandum NOS OMA 52. Seattle: United States Department of Commerce, National Oceanic and Atmospheric Administration. 175 pp. + appendices.

Long, E.R. and D.D. MacDonald. 1992. National Status and Trends Program Approach. In: Sediment Classification Methods Compendium. EPA 823-R-92-006.

## AR300944

Long, E.R., D.D. MacDonald, S.L. Smith, and F.D. Calder. 1994. Incidence of adverse biological effects within ranges of chemical concentrations in marine and estuarine sediments. Environmental Management. In Press.

. .. . . .

Lupine, A. 1993 and 1992. Personal communication. Fisheries Biologist. Division of Freshwater Fisheries, Department of Environmental Protection. Lebanon, NJ. March 12 and March 13.

Lynch, T.R. and H.E. Johnson. 1982. Availability of hexachlorobiphenyl isomer to benthic amphipods from experimentally contaminated sediments. pp. 273-287. In: Aquatic Toxicology and Hazard Assessment: Fifth Conference. ASTM STP 766. J.G. Pearson, R.B. Foster, and W.E. Bishop (eds.). Philadelphia, PA: American Society of Testing and Materials.

Mac, M.J. and C.C. Edsall. 1991. Environmental contaminants and the reproductive success of lake trout in the Great Lakes: an epidemiological approach. *Journal of Toxicology and Environmental Health* 33: 375-394.

Mac, M.J. and J.G. Seelye. 1981. Patterns of PCB accumulation by fry of lake trout. *Bulletin. Environmental Contamination and Toxicology* 27: 368-375.

MacDonald, C.R., C.D. Metcalfe, G.C. Balch, and T.L. Metcalfe. 1993. Distribution of PCB congeners in seven lake systems: interactions between sediment and food-web transport. *Environmental Contamination and Toxicology* 12: 1991-2003.

Mayer, F.L., P.M. Mehrle, and H.O. Sanders. 1977. Residue dynamics and biological effects of polychlorinated biphenyls in aquatic organisms. *Archives Environmental Contamination 5*: 501-511.

McCammon, G.W. 1956. A tagging experiment with channel catfish (*Ictalurus punctatus*) in the Lower Colorado River. *California Fish and Game 42*:323-335

McFarland, V.A. and J.U. Clarke. 1989. Environmental occurrence, abundance, and potential toxicity of polychlorinated biphenyl congeners: considerations for a congener-specific analysis. *Environmental Health Perspectives* 81: 225-239.

Mearns, A. J., R. C. Swartz, J. M. Cummins, P. A. Dinnel, P. Plesha, and P.M. Chapman. 1986. Inter-laboratory comparison of a sediment toxicity test using the marine amphipod, Rhepoxynius abronius. *Marine Environmental Research 19*: 13-37.

Melancon, M.J., and J.J. Lech. 1983. Dose-effect relationship for induction of hepatic monooxygenase activity in rainbow trout and carp by Aroclor 1254. *Aquatic Toxicology 4*: 51-61.

Melancon, M.J., K.A. Turnquist, and J.J. Lech. 1989. Relation of hepatic monooxygenase activity to tissue PCBs in rainbow trout (*Salmo gairdneri*) injected with [<sup>14</sup>C]PCBs. *Environmental Toxicology and Chemistry* 8: 777-782.

Menzie-Cura & Associates, Inc. 1993a. Memo: Figure 1. General Sampling Area for Fish. September 16, 1993.

Menzie-Cura & Associates, Inc. 1993b. Memo: Life histories of selected fish species in the Delaware River. October 1, 1993. 11 p.

Menzie-Cura & Associates, Inc. 1993c. Memo: PCB body burden for the shortnose sturgeon. October 13, 1993.

Mihursky, J.A. 1962. Fishes of the Middle Lenapewihtuck (Delaware River) Basin. Doctoral Dissertation. Lehigh University. University Microfilms, Inc. No. 63-2628. Ann Arbor, MI.

Miller, J. 1993. Personal communication. Coordinator. Delaware River Anadromous Fisheries Project, Delaware River Basin Commission. Trenton, NJ. March 17.

Monod, G. 1985. Egg mortality of Lake Geneva charr (Salvelinus alpinus) contaminated by PCB and DDT derivatives. Bulletin. *Environmental Contamination and Toxicology* 35: 531-536

Nebeker, A.V. and F.A. Puglisi. 1974. Effect of polychlorinated biphenyls (PCB's) on survival and reproduction of *Daphnia*, *Gammarus*, and *Tanytarsus*. *Transactions*. *American Fisheries Society 103*: 722-728.

Nebeker, A.V., G.S. Schuytema, W.L. Griffis, J.A. Barbitta, and L.A. Carey. 1989. Effect of sediment organic carbon on survival of *Hyalella azteca* exposed to DDT and endrin. Environ. Toxicol. Chem. 8: 705-718.

Nestel, H. and J. Budd. 1975. Chronic oral exposure of rainbow trout (Salmo gairdneri) to a polychlorinated biphenyl (Aroclor 1254): pathological effects. Can. J. Comp. Med. 39: 209-215.

O'Herron, J. 1993. Personal Communication. Fisheries Biologist. Tom Lloyd Associates. Burlington, NJ. March 9.

O'Herron, J., K. Able, and R. Hastings. 1993. Movements of Shortnose Sturgeon (Actipenser brevirostrum) in the Delaware River. Estuaries 16(2):235-240.

Oliver, B. G. and A. J. Niimi. 1988. Trophodynamic analysis of polychlorinated biphenyl congeners and other chlorinated hydrocarbons in the Lake Ontario ecosystem. *Environmental Science and Technology* 22: 388-397.

Olla, B., V. Estelle, R.C. Swartz, G.Braun, and A. Studholme. 1988. Responses of polychaetes to cadmium-contaminated sedimenta: comparison of uptake and behavior. *Environmental Toxicology and Chemistry* 7: 587-592.

Parkinson, A. and S. Safe. 1987. Mammalian biologic and toxic effects of PCBs. Environ. Toxin. Ser. 1: 49-75.

Pavlou, S.P. and R.N. Dexter. 1979. Distribution of polychlorinated biphenyls (PCB) in estuarine ecosystems. Testing the concept of equilibrium partitioning in the marine environment. *Environmental Science and Technology 13*: 65-71.

Phillips, D.J.H. 1986. Use of organisms to quantify PCBs in marine and estuarine environments. In: J.S. Waid (ed.).*PCBs and the Environment*. Boca Raton, FL: CRC Press, Inc. pp. 127-182.

Pruell, R.J., N.I. Rubinstein, B.K. Taplin, J.A. LiVolsi, and R.D. Bowen. 1993. Accumulation of polychlorinated organic contaminants from sediment by three benthic marine species. Arch. *Environmental Contamination and Toxicology* 24: 290-297.

PTI Environmental Services. 1988. Briefing report to the EPA Science Advisory Board: the Apparent Effects Threshold Approach. Seattle: U.S. Environmental Protection Agency, Region 10, Office of Puget Sound.

Raney, E.C. 1942. Propagation of the silvery minnow (Hybognathus nuchalis) in ponds. Transactions. American Fisheries Society 71:215-218.

Ray. S., B.M. Jessop, J. Coffin, and D.A. Swetnam. 1984. Mercury and polychlorinated biphenyls in striped bass (*Morone saxatilis*) from two Nova Scotia rivers. *Water, Air, Soil Pollution 21*: 15-23.

Robinson, A.M., J.O. Lamberson, F.A. Cole and R.C. Swartz. 1988. Effects of culture conditions on the sensitivity of a phoxocephalid amphipod, *Rhepoxynius abronius*, to cadmium in sediment. *Environmental Toxicology and Chemistry* 7: 953-959.

Rubinstein, N.I, W.T. Gilliam, and N.R. Gregory. 1984. Dietary accumulation of PCBs from a contaminated sediment source by a demersal fish (*Leiostomus xanthurus*). Aquatic Toxicology 5: 331-342.

Safe, S. 1984. Polychlorinated biphenyls (PCBs) and polybrominated biphenyls (PBBs): biochemistry, toxicology, and mechanism of action. CRC Crit. Rev. Toxicol. 13:319-393.

Sawhney, B.L. 1986. Chemistry and properties of PCBs in relation to environmental effects. In: J.S. Waid (ed.), *PCBs and the Environment*. 1: 47-65. Boca Raton, FL: CRC Press, Inc.

Scott, W.B. and E.J. Crossman. 1973. Freshwater fishes of Canada. Fisheries Research Board of Canada. Ottawa, Canada.

Seelye, J.G., R.J. Hesselberg and M.J. Mac. 1982. Accumulation by fish of contaminants released from dreged sediments. *Environmental Science and Technology* 16:459-464.

Shaw, G.R. and D.W. Connell. 1982. Factors influencing concentrations of polychlorinated biphenyls in organisms from an estuarine ecosystem. *Aust. J. Mar. Freshw. Res.* 33: 1057-1070.

Sloan, R., M. Brown, R. Brandt, and C. Barnes. 1985. Hudson River PCB relationships between resident fish, water and sediment. *Northeast. Environ. Sci.* 3: 137-151.

Smith, L.M., T.R. Schwartz, and K. Feltz. 1990. Determination and occurrence of AHHactive polychlorinated biphenyls, 2,3,7,8-tetrachloro-p-dioxin and 2,3,7,8tetrachlorodibenzofuran in Lake michigan sediment and biota. The question of their relative toxicological significance. *Chemosphere 21*: 1063-1085.

Soldo, J. 1992. Personal Communication. Fisheries Biologist, Pennsylvania Fish Commission. Revere, PA. August 3.

Stephan, C.E., D.I. Mount, D.J. Hansen, J.H. Gentile, G.A. Chapman, and W.H. Brungs. 1986. Guidelines for deriving numerical national water quality criteria for the protection of aquatic organisms and their uses. (NTIS PB85-227049). Washington, D.C.: U.S. Environmental Protection Agency, Office of Water Regulations and Standards, Criteria and Standards Division.

Suter, G.W., II. 1993. Ecological Risk Assessment. Chelsea, MI: Lewis Publishers. 538 pp.

Spies, R.B., D.W. Rice, Jr., P.A. Montagna, and R.R. Ireland. 1985. Reproductive success, xenobiotic contaminants and hepatic mixed-function oxidase (MFO) activity in *Platichthys stellatus* populations from San Francisco Bay. *Marine Environmental Research 17*: 117-121.

Swartz, R.C. and H. Lee. 1980. Biological processes affecting the distribution of pollutants in marine sediments. Part I: accumulation, trophic transfer, biodegradation and migration. In: *Contaminants and Sediments. Vol II.* R.A. Baker (ed.). Ann Arbor, MI: Ann Arbor Science Publishers. pp. 533-553.

Swartz, R.C., G.R. Ditsworth, D.W. Schults, and J.O. Lamberson. 1985. Sediment toxicity to a marine infaunal amphipod: cadmium and its interaction with sewage sludge. Mar. Environ. Res. 18: 133-153.

Swartz, R.C., P.F. Kemp, D.W. Schults, and J.O. Lamberson. 1988. Effects of mixtures of sediment contaminants on the marine infaunal amphipod, *Rhepoxynius abronius*. *Environmental Toxicology and Chemistry* 7: 1013-1020.

Swinick, P.P. 1993. Letter to Mr. Roy Smith, U.S. EPA Region III, regarding Cottman Avenue Project Information for Human Health Risk Assessment. Wayne, PA: HMM Associates, Inc. 2 pp.

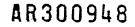
Tatem, H.E. 1982. Bioaccumulation of PCB by freshwater prawns and clams exposed to contaminated dredged material. Engineering and Scientific Research at Waterways Experiment Station, Vol O-82-3. Vicksburg, MS: U.S. Army Corps of Engineers, Waterways Experiment Station. pp. 1-6.

Tatem, H.E. 1986. Bioaccumulation of polychlorinated biphenyls and metals from contaminated sediment by freshwater prawns, *Macrobrachium rosenbergii* and clams, *Corbicula fluminea. Archives of Environmental Contamination\_and Toxicology 15*: 171-183.

Thomann, R.V. and J.P. Connolly. 1984. Model of PCB in the Lake Michigan lake trout food chain. *Environmental Science and Technology 18*: 65-71.

U.S. ACOE. 1988. Relationship between tissue residues and reproductive success of fathead minnows. Environmental Effects of Dredging, Technical Notes EEDP-01-13. Vicksburg, MS: U.S. Army Corps of Engineers, Waterway Experiment Station. 9 pp.

U.S. EPA. 1980. Ambient water quality criteria for polychlorinated biphenyls. Washington, D.C.: U.S. Environmental Protection Agency, Office of Water Regulations and Standards, Criteria and Standards Division.



U.S. EPA. 1988. Review of the Draft drinking water criteria document for polychlorinated biphenyls (PCBs). U.S. Environmental Protection Agency, Science Advisory Board, Environmental Health Committee. SAB-EHC-88-019.

U.S. EPA. 1989a. Interim procedures for estimating risks associated with exposures to mixtures of chlorinated dibenzo-{rho}-dioxins and -dibenzofurnas (CDDs and CDFs) and 1989 update. EPA/625/3-89/016. Washington, DC: U.S. Environmental Protection Agency, Risk Assessment Forum. 98 pp.

U.S. EPA. 1989b. Risk assessment guidance for Superfund. Volume 1: human health evaluation manual (part A). Interim final. EPA/540-1-89-002. Washington, D.C.: U.S. Environmental Protection Agency, Office of Emergency and Remedial Response.

U.S. EPA. 1989c. Risk assessment guidance for Superfund. Volume 2: environmental evaluation manual. Interim final. EPA/540-1-89-001. Washington, D.C.: U.S. Environmental Protection Agency, Office of Emergency and Remedial Response.

U.S. EPA. 1992a. National study of chemical residues in fish. Volume I. EPA 823-R-92-008a. Washington, D.C.: U.S. Environmental Protection Agency, Office of Science and Technology. 166 pp. + appendices.

U.S. EPA. 1992b. Quality criteria for water. EPA 440/5-92-001. Washington, D.C.: U.S. Environmental Protection Agency, Office of Water Regulations and Standards.

U.S. Geological Survey. 1976. Wilmington: DE, NJ, PA, MD. 1:250 Series. Reston, VA: USGS.

U.S.EPA. 1993. Interim report on data and methods for assessment of 2,3,7,8tetrachlorodibenzo-p-dioxin risks to aquatic life and associated wildlife. EPA/600/R-93/055. Washington, D.C.: U.S. Environmental Protection Agency, Office of Research and Development.

U.S. Fish and Wildlife Service. 1991. Concentrations of organochlorines and trace elements infish and blue crabs from the Delaware River, Easton to Deepwater. Pennsylvania Field Office Special Project Report 93-5 (Study I.D. No. 90-5-057). State College, PA: U.S. Fish and Wildlife Service.

Von Westernhagen, H., H. Rosenthal, V. Dethlefsen, W. Ernst, U. Harms, and P.D. Hansen. 1981. Bioaccumulating substances and reproductive success in Baltic flounder, *Platichthys flesus*. Aquatic Toxicology 1: 85-99.

Walton, T.E. and R. Patrick. 1973. Delaware Estuary System, Environmental Impacts and Socio-Economic Effects. Delaware River Estuary Marsh Survey. National Science Foundation RANN Program. 172 pp.

Wang, J.C.S. and R.J. Kernehan. 1979. Fishes of the Delaware estuaries, a guide to the early life histories. Townson MD: E.A. Communications, Ecological Analysts. 410 pp.

Weston, R.F., Inc. 1978. Hydrogeologic evaluation of a subsurface oil spill at the Metals Bank of America, Inc. Disposal Site, Philadelphia, PA: Prepared for the United States Coast Guard Marine Environmental Unit, Gloucester, NJ.

7-11

AR300950

Word, J.Q., J.A. Ward, L.M. Franklin, V.I. Cullinan and S.L. Kiesser. 1987. Evaluation of the equilibrium partitioning theory for estimating the toxicity of the nonpolar organic compound DDT to the sediment dwelling amphipod *Rhepoxynius abronius*. Battelle Washington Environmental Program Office. Report prepared for Criteria and Standards Division, U.S. Environmental Protection Agency. 60 pp.

AR300951

Exposure point concentrations for PCBs in sediment are based on total PCBs. The patterns of PCBs in sediment were most frequently measured as Aroclors 1248, 1254, and 1260. When detectable concentrations were determined for one or more of these Aroclors in a single sample, the concentrations were summed to arrive at the total PCB concentration in a sample. Since Aroclors 1016, 1221, 1232, and 1242 were not detected in any of the sediment samples, total PCB concentrations were calculated by summing the concentrations of Aroclors 1248, 1254, and 1260. Samples in which PCBs were not reported above the detection limit were treated as if the PCBs were present at half of the detection limit and this concentration was included in calculating the total PCB concentration of a sample. For the samples that underwent analysis for individual PCB congeners, the total PCB value was calculated by the lab and did not represent the sum of the individual congeners reported.

Four exposure point concentrations were calculated for sediments in each of the three areas: the arithmetic mean and the upper 95 percent confidence limit normalized to dry weight and TOC. TOC concentrations affect the bioavailability of PCBs and it is the TOC normalized concentrations that are used in BAF calculations. The resulting mean and upper 95 percent confidence limit concentrations for total PCBs in sediments are presented in Table 4-3. All sets of values show a clear trend in decreasing PCB concentrations with increasing distance from the site.

The limited extent of the highest concentrations of PCBs in sediments is further demonstrated by an examining the average total PCB concentrations of the individual riprap stations along the western edge of the site and the nearfield mudflat stations. These concentrations are plotted against the distance of the individual stations from the station with the highest total PCB concentration,MF-107 (distance from MF-107 = distance measured parallel to the western site boundary + the distance measured from and perpendicular to the site boundary). Total PCB

Table 4-3.	Mean and upper 95 percent confidence limit (CL) concentrations
	(mg/kg) of total PCBs in the sediments near the Metal Bank site
	normalized to dry weight and total organic compound (TOC).

	Dry Weight Normalized		TOC Normalized		
Area	Mean	Upper 95 percent CL	Mean	Upper 95percent CL	Number of Samples
Riprap	5.9	9.4	150	240	13
Nearfield (< 30 meters)	3.8	5.0	79	110	24
Farfield (> 30 meters)	0.87	1.2	30	44	16

concentrations appear to drop off rapidly towards the Delaware River but more gradually away from the river with respect to MF-107 (Figure 4-3). When a regression analysis was conducted only on those stations increasingly farther from both the river and MF-107 (stations: MF-107, RR-3, MF-124, RR-2, MF-5, MF-101, RR-1, and MF-1) there was a strong negative correlation between total PCB concentration and distance with an R<sup>2</sup> of 0.71. When the analysis was conducted on only the corresponding riprap stations (stations: MF-107, RR-3, RR-2, MF-101 and RR-1) the R<sup>2</sup> increased to 0.96 (Figure 4-3). This pattern of decreasing concentrations resembles that of a point source gradient.

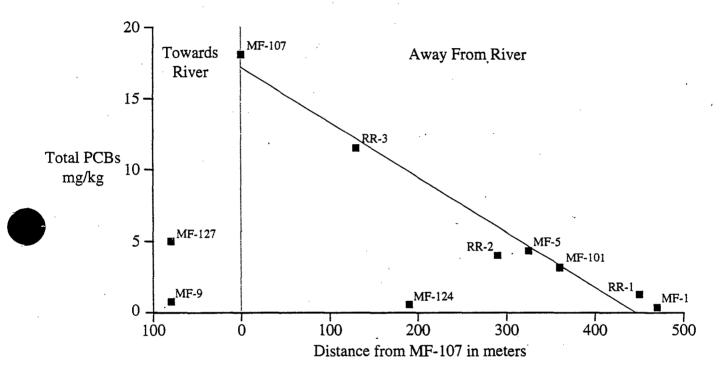


Figure 4-3. Average total PCB concentrations for the western riprap and nearfield mudflat stations plot against their distance from riprap station MF-107 (distance = distance along riprap + perpendicular distance from riprap). The sloping line represents the regression line for just the riprap stations ( $R^2$ =0.96).

4.3.2 PAHs

After dilution, the contribution of PAHs to surface water was considered to be negligible so exposure to PAHs was only evaluated for NAPL and sediments.

#### 4.3.2.1 Exposure to PAHs in NAPL

As discussed above, aquatic organisms in the Delaware River and in the mudflat could potentially experience direct, localized contact with NAPL adjacent to the site. Only one individual PAH (pyrene) was above its detection limit in the single NAPL sample (MW-6)

analyzed. The reported pyrene concentration was 87 mg/l, while the detection limits for all the other individual PAHs was 750 mg/l. Because of this extremely high detection limit, it was decided that a total PAH concentration based on half the detection limit would be unrealistically high. Therefore, another method was devised to determine the concentrations of the individual PAHs in the NAPL before summing them. The ratios of each individual PAH concentration to the pyrene concentration was calculated for the two groundwater samples with the highest number of detectable PAHs (MW-1 and MW-1D had only four non-detected PAHs). The ratios for each individual PAH were averaged and the average ratio for each PAH was multiplied by the concentration of pyrene in the NAPL. Since pyrene is one of the less soluble PAHs, the resulting values would tend to err on the high side, resulting in a conservative but reasonable estimate (based on the available data) of the individual PAH concentrations in the NAPL. The resulting concentration of total PAHs in the NAPL was 1,200 mg/l. The estimated concentrations for the individual PAHs with AWQC are shown in Table 4-4.

Table 4-4.	Estimated concentrations (mg/kg)of four PAHs in
	NAPL and their respective AWQC values (* indicates
	an LOEL, p-indicates proposed).

РАН	Estimated Concentration	AWQC
acenapthene	43	520*
fluoranthene	73	3980*
napthalene	590	620*
phenanthrene	134	6.3p

## 4.3.2.2 Exposure to PAHs in Sediments

Calculating an exposure-point concentration for sediment was based on sediment data collected in 1991 and 1993. After examining the PAH data, it was decided to divide it into the same three areas as those for PCBs: the riprap, nearfield mudflat and river, and farfield mudflat. The calculation of the mean sediment concentrations for the three areas (Table 4-5) used the combined PAH data from 1991 and 1993 based on sampling station location, not station designation. Four exposure-point concentrations were calculated for sediments in each of the three areas: the arithmetic mean and the upper 95 percent confidence limit normalized to dry weight and TOC. The resulting mean and upper 95 percent confidence limit concentrations for total PAHs in sediments are presented in Table 4-5. All sets of values show a clear trend in decreasing PAH concentrations with increasing distance from the site.

	Dry Weight Normalized		TOC nor		
Area	Mean	Upper 95% CL	Mean	Upper 95% CL	Number of Samples
Riprap	310	980	3900	9400	.4
Nearfield (< 30 meters)	52	88	1100	1800	15
Farfield (> 30 meters)	4.1	4.3	200	410	4

Table 4-5. Mean and upper 95 percent CL concentrations (mg/kg) of total PAHs in the sediments near the Metal Bank site normalized to dry weight and TOC.

#### 4.3.3 Phthalates, DDTs, and Cadmium

The remaining COCs (DEHP, DBP, DOP, DDT, DDD, DDE and cadmium), with the exception of DEHP, will only be evaluated with regard to potential risks associated with sediments. Potential risks due to DEHP will be evaluated with respect to NAPL and sediments.

#### 4.3.3.1 Exposure to DEHP in NAPL

DEHP was the only phthalate with a concentration above the detection limits in the single sample of NAPL analyzed. Therefore the exposure point concentration for DEHP in NAPL is as reported (HMM 1993b) 590 mg/l.

4.3.3.2 Exposure to Phthalates, DDTs, and Cadmium in Sediments Sediment exposure-point concentrations for the three phthalates of concern (DEHP, DBP, and DOP) were calculated in the same manner as the calculations for PCBs and PAHs. Table 4-6 presents these exposure-point concentrations showing a similar trend of decreasing concentrations with increasing distance from the site. However, because of the reduced sample size involved, these numbers must be viewed with caution.

4-17

Because of the high number below detection limit values and the lack of any apparent pattern of pesticide concentrations, exposure concentrations for 4,4'-DDT; 4,4'-DDE; and 4,4'-DDD were calculated based on the entire sediment data set (i.e., the data set was not subdivided by proximity to the site). The same was true for cadmium because of the very limited size (six samples) of the data set and the lack of any apparent pattern. Table 4-7 gives the exposure point concentrations for 4,4'-DDT; 4,4'-DDE; and 4,4'-DDD as a mean and an upper 95 percent confidence limit normalized for dry weight and TOC. Since TOC has little effect on cadmium concentrations or availability, exposure values were only calculated on a dry weight basis. The resulting mean concentration was 73 mg/kg, while the upper 95 percent confidence limit was 173 mg/kg.

Table 4-6. Mean and upper 95 percent CL concentrations (mg/kg) of DEHP, DBP, and DOP in the sediments near the Metal Bank site normalized to dry weight and TOC.

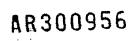
DEHP (Bis (2-ethylhexyl) pthalate)							
	Dry W Norma		TOC nor	·			
Area	Mean	Upper 95% CL	Mean	Upper 95% CL	Number of Samples		
Riprap	290	810	3100	8500	7		
Nearfield (< 30 meters)	2.5	5.0	66	120	13		
Farfield (> 30 meters)	0.28	0.53	13	33	2		

## DBP (Di-n-butyl pthalate)

	Dry Weight Normalized		TOC normalized		
Area	Mean	Upper 95% CL	Mean	Upper 95% CL	Number of Samples
Riprap	2.0	2.8	42	65	7
Nearfield (< 30 meters)	0.45	0.67	13	18	13
Farfield (> 30 meters)	0.16	0.65	9.0	51	2

## DOP (Di-n-octyl pthalate)

	Dry Weight Normalized		TOC normalized		
Area	Mean	Upper 95% CL	Mean	Upper 95% CL	Number of Samples
Riprap	20	56	220	590	7
Nearfield (< 30 meters)	0.97	1.6	24	40	13
Farfield (> 30 meters)	0.25	0.34	12	37	2



	Dry W Norma	eight llized	TOC nor		
Chemical	Mean	Upper 95% CL	Mean	Upper 95% CL	Number of Samples
4,4'-DDT	110	131	2500	3200	40
4,4'-DDE	140	170	3300	4000	40
4,4'-DDD	140	170	3500	4400	40

Table 4-7.	Mean and upper 95 percent CL concentrations (µg/kg) of 4,4'-DDT;
	4,4'-DDE; and 4,4'-DDD in the sediments near the Metal Bank site
	normalized to dry weight and TOC.

## 4.3.4 PCB Tissue Residue Levels in Invertebrates and Fish

Data on PCB concentrations in invertebrate and fish tissue near the site integrates all exposure pathways. While tissue residue levels in sessile invertebrates such as *Corbicula* can be estimated from sediment concentrations using an EP approach, higher trophic level species (e.g., fish) accumulate a substantial percentage of their PCB body burdens from food.

To estimate PCB body burdens in fish from a food web model requires considerable information, much of which is unavailable. A more direct approach, using tissue-to-sediment ratios derived from matching tissue and sediment data from other areas, allows estimation of tissue concentrations in fish from observed sediment concentrations based on available tissue data and knowledge of the biology of the target species.

## 4.3.4.1 Corbicula

Asiatic clams (*Corbicula fluminea*) were collected from five stations in the mudflat in the fall of 1992. PCB concentrations were determined in whole-body composites by congener-specific analysis (44 individual congeners or congener pairs) and by standard Aroclor analysis (EPA CLP method 8080; **citation**). Based on the congener analysis the total PCB concentrations ranged from 0.2 to 1.0 mg/kg wet weight and 17.4 to 75.8 mg/kg lipid (Table 4-8; Hermanson 1992). When the lipid weight values are compared to their respective TOC normalized sediment values the BAFs range from 0.5 to 4.1 with an average of 1.6. The range of BAFs for benthic infauna reported in the literature is between 1 and 10 with the majority slightly higher than 1 (MacDonald et al. 1993). Total PCBs determined by the Aroclor method were all below detection; however, the detection limits ranged between 2 and 5.2 mg/kg.

Table 4-8. Total PCB concentrations in sediments and tissues (based on congener analy	
(Hermanson 1992)) and bioaccumulation factors (BAFs, tissue lipid normalized	zed
PCB/sediment TOC normalized PCB concentrations) for Corbicula from five station	ons
in the mudflats of the Metal Bank site.	

	MF-5	MF-7	MF-9	MF-10	MF-11	Mean
PCBs in Sediment						
Total PCBs mg/kg	7.16	0.67	1.26	0.92	1.16	
Percent TOC	4.9	3.7	7.0	2.4	2.8	
Total PCBs mg/kg TOC	146.2	18.0	18.0	38.3	41.4	
Corbicula tissue		i.				
Total PCBs mg/kg ww	1.03	0.23	0.95	0.70	0.80	
Percent Lipid	1.35	1.32	1.29	1.27	1.68	'
Total PCBs mg/kg lipid	75.8	17.4	74.2	54.8	47.8	
PCBs Tissue Lipid/PCBs Sediment TOC	0.52	0.97	4.13	1.43	1.15	1.64

#### 4.3.4.2 Silvery minnow and channel catfish

Thirteen Eastern silvery minnows (*Hybognathus regius*) and 19 channel catfish were collected in the Delaware River adjacent to the site (Figure 4-4) in late September 1993. The silvery minnows were reported to have whole body total PCB concentrations (measured as Aroclors 1242 + 1260) ranging from 0.55 to 2.8 mg/kg wet weight (5.7 to 46 mg/kg lipid; Table 4-9). Channel catfish collected from the Delaware River adjacent to the site had whole body tissue concentrations ranging from 1.1 to 4.0 mg/kg wet weight (5.4 to 57.5 mg/kg lipid) and fillet concentrations from 0.4 to 1.5 mg/kg wet weight (12.0 to 84.7 mg/kg lipid; Table 4-9; HMM 1993b). The overall average lipid-normalized mean concentration for catfish (whole body and fillet samples combined) of 28.2 mg/kg-lipid was comparable to the 27.5 mg/kg-lipid observed for the silvery minnow.

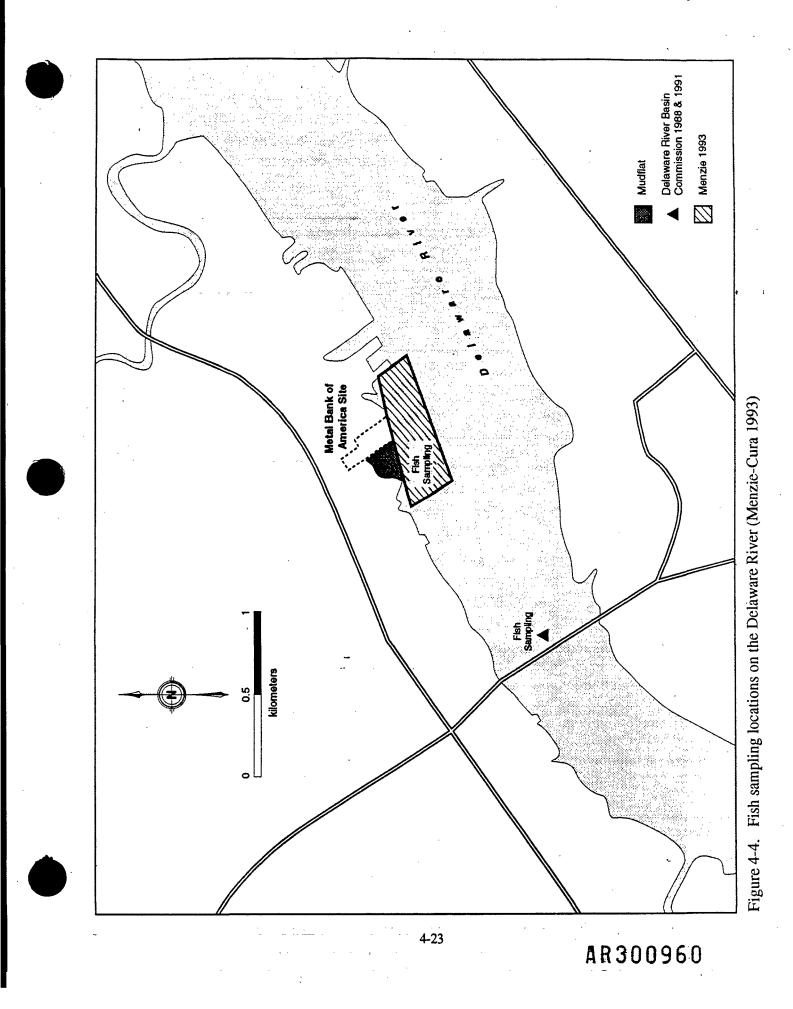
Based on the estimated TOC normalized mean PCB sediment concentration for the nearfield (79 mg/kg TOC) for the Metal Bank site, the BAFs for whole body channel catfish ranged from 0.07 to 0.73 with a mean BAF of 0.25. BAFs for silvery minnows ranged from 0.07 to 0.58 with a mean BAF of 0.35. However, when the BAFs are calculated based on the farfield TOC normalized mean sediment concentration (30 mg/kg), the catfish BAFs range from 0.18 to 1.9 with a mean of 0.66, and the BAFs for minnows range from 0.19 to 1.5 with a mean of 0.92. When compared to BAFs reported in the literature (Table 4-1), the nearfield values

appear low for both catfish and minnows; while the farfield BAFs are in the expected range for a lower trophic level fish species such as the silvery minnow, but still appear low for the catfish. These results suggest that the minnow, which has a relatively small range of movement, are exposed, on average, to sediments containing the mean farfield concetration of PCBs. While the channel catfish, with its larger range of movement, are exposed, on average, to sediments containing less than the mean farfield concentration of PCBs. Furthermore, it is generally recognized that PCBs biomagnify resulting in increasing BAFs with increasing trophic level (MacDonald et al. 1993). The fact that the silvery minnows have a larger BAF than the channel catfish further suggests that they are exposed on average to sediments containing higher levels of PCBs than are the channel catfish.

### 4.3.4.3 Delaware River data

The presence of PCBs in Asiatic clams from the mudflat indicates the potential for transfer of PCBs through the food web in aquatic organisms of the Delaware River, especially for those species that feed predominantly on benthic or epibenthic organisms. A number of studies have demonstrated PCB contamination in fish throughout the main stem of the Delaware River (DRBC 1988; USFWS 1991; EPA 1992a).

PCBs have been detected in fish tissues in numerous studies at limited locations throughout the Delaware River system, some from as early as 1969 (Cottman Avenue PRP Group 1992). Recent data (1987 to 1992) have shown PCBs to be present in fish collected from all locations studied, ranging from 122 km above the site to 160 km below the site in Delaware Bay. Of the species sampled, the channel catfish (*Ictalurs punctatus*) and the white perch (*Morone americana*) were sampled most frequently and with the most consistency. Table 4-10 summarizes PCB data for these species for the years 1987 through 1992 and for the area 122 km above MBA to 64 km below the site. Figures 4-5 and 4-6 depict PCB concentrations on a wet weight and lipid normalized basis, respectively, in the tissues of channel catfish collected at locations above and below the site.



MB Aquatic ERA

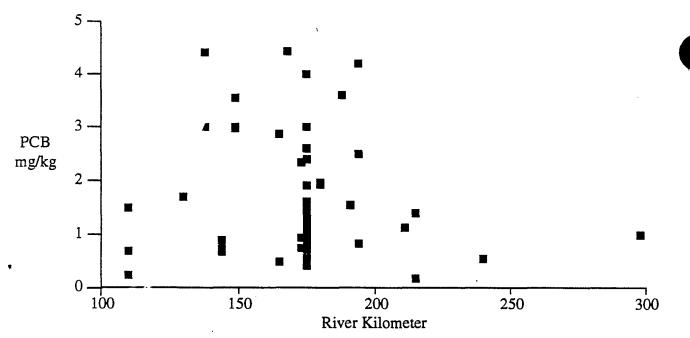


Figure 4-5. PCB concentrations (mg/kg wet weight) in channel catfish collected in the Delaware River above and below the Metal Bank site (river kilometer 175; DRBC 1988 and unpublished; Greene 1991; USFWS 1991; EPA 1992; HMM 1993b).

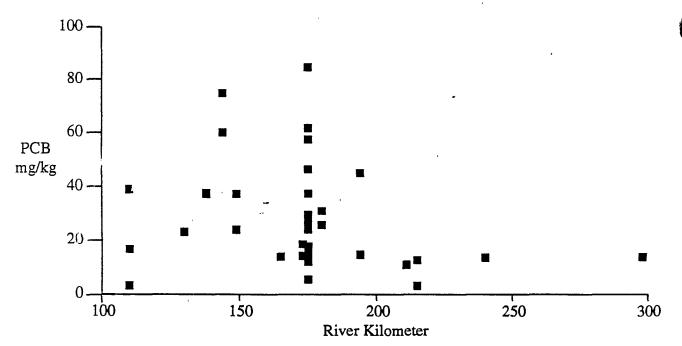


Figure 4-6. Lipid normalized PCB concentrations (mg/kg lipid) in channel catfish collected in the Delaware River above and below the Metal Bank site (river kilometer 175; DRBC 1988 and unpublished; Greene 1991; USFWS 1991; EPA 1992; HMM 1993b).

4-24



Total PCB concentrations (sum of Aroclors 1242 + 1260) in the tissues of channel catfish and silvery minnow collected near the Metal Bank site (HMM 1993). Table 4-9. · - ,..

-....

Species Sample Type	Sample No.	Length	Weight	Sex	Lipid	Total PCBs	Lipid Normalized
		cm	oz		%	µg/g ww	µg/g lipid
Channel catfish							
Whole Body	1	40	19	Μ	20.1	1.08	5.37
Whole Body	2	43	24	Μ	7.8	1.38	17.7
Whole Body	3	44	24	Μ	2.8	1.61	57.5
Whole Body	4	45	24	F	13.5	4.00	29.6
Whole Body	5	45	25	Μ	- 11.9	1.91	16.1
Whole Body	6	42	26	Μ	22.2	1.21	5.45
Whole Body	17	46	35	F	18.0	2.40	13.3
Whole Body	18	42	30	F	16.5	2.60	15.8
Whole Body	19	41	24	Μ	19.2	3.00	15.6
Fillet	7	48	29	Μ	1.7	0.79	46.5
Fillet	8	51	· 49	F	2.4	1.48	61.7
Fillet	9	40	20	Μ	7.7	0.92	11.9
Fillet	10	38	17	Μ	3.6	1.35	37.5
Fillet	11	52	51	Μ	8.3	1.04	12.5
Fillet	12	45	31	Μ	3.0	0.87	29.0
Fillet	13	38	23	M	1.7	0.41	24.1
Fillet	14	38	26	F	5.3	1.43	27.0
Fillet	15	48	33	F	0.85	0.72	84.7
Fillet	16	43	28	Μ	2.3	0.56	24.3
Silvery minnow	,						
Whole Body	1	10	<1		8.8	2.40	27.3
Whole Body	2	11	<1		2.3	1.05	45.7
Whole Body	3	10 ·	<1		9.7	0.55	5.67
Whole Body	4	10	<1			1.52	
Whole Body	5.	11	<1		9.3	2.10	22.6
Whole Body	6	10	<1		8.5	2.70	31.8
Whole Body	7	10	<1		6.5	1.71	26.3
Whole Body	8	11	<1		.6.7	2.00	29.9
Whole Body	9	10	<1		6.8	1.50	22.1
Whole Body	10	10	<1		7.8	2.80	35.9
Whole Body	11	9.5	<1		11:3	2.40	21.2
Whole Body	12	9	<1		5.7	1.62	28.4
Whole Body	13	10	<1		7.1	2.40	33.8

AR300962-25

Table 4-10. Summary of PCB concentrations in muscle tissue of channel catfish and white perch collected in the Delaware River from 1987 through 1992. Concentrations are listed by wet weight (mg/kg) and as lipid normalized (mg/kg lipid).

Disco	Channel Car	-Ci-h	White Deeph		Collection	
River	Channel Cat		White Perch		1	ł
Kilometer	Wet	Lipid	Wet	Lipid	Year	Reference
	Weight	Norm.	Weight	Norm.		
298	0.99	13.75	NA	NA	1990	USFWS 1991
240	0.55	13.55	NA	NA	1990	USFWS 1991
215	1.40	12.61	0.95	50.53	1990	USFWS 1991
	0.17	3.1	NA	NA	1989	Greene 1991
211	1.13	10.9	NA	NA	1989	Greene 1991
194	4.20	NA	0.41	NA	1987	DRBC 1988
	0.83	14.6	NA	NA	1989	Greene 1991
	2.5	45	3.84	36.5	1990	USFWS 1991
191	1.55	NA	<0.1	NA	1987	DRBC 1988
188	3.60	NA	0.285	NA	1987	DRBC 1988
180	1.96	25.8	NA	NA	1987	EPA 1992a
	1.93	31.1	NA	NA	1989	Greene 1991
175 (MBA)	0.41-4.00*	5.37-84.71	NS	NS	1993	HMM 1993
173	2.34	NA	1.12	NA	1987	DRBC 1988
	0.75	14.1	0.59	28.1	1991	DRBC unpublished
	0.94	18.6	NA	NA	1991	DRBC unpublished
168	4.43	NA	0.74	NA	1987	DRBC 1988
165	2.86	NA	0.99	NA	1987	DRBC 1988
	0.49	13.8	0.77	20.4	1990	USFWS 1991
160	NA	NA	1.73	NA	1987	DRBC 1988
154	NA	NA	1.2	NA	1987	DRBC 1988
149	3.54	NA	1.19	NA	1987	DRBC 1988
	2.97**	24**	NA	NA	1987	EPA 1992a
	3.00	37.4	NA	NA	1989	Greene 1991
144	0.73	60	1.2	41.4	1991	DRBC unpublished
	0.89	74.8	NA	NA	1991	DRBC unpublished
	0.68	NA	1.51	NA	1992	DRBC unpublished
	NA	NA	1.25	NA	1992	DRBC unpublished
138	NA	NA	1.41	NA	1987	EPA 1992a
	4.41	37.7*	NA	NA	1987	EPA 1992a
	3.0	37.4	0.86	38.2	1989	Greene 1991
130	1.7	23.2	0.84	30.7	1990	USFWS 1991
110	1.5	16.7	0.87	25	1990	USFWS 1991
	0.23	3.1	0.11	8.9	1991	DRBC, unpublished
	0.23	3.2	NA	NA	1991	DRBC, unpublished
	0.69	39	0.62	44	. 1992	DRBC, unpublished
L	NA	NA	0.67	41	1992	DRBC, unpublished

NA: No data available

NS: Not sampled

\* Concentrations based on fillets and whole body

\*\* Concentration is based on whole body rather than on only muscle tissue



Tissue samples from ten shortnose sturgeon, a federally listed endangered species, collected in 1982 at a location approximately 32 km upstream of MBA had PCB concentrations ranging from 0.016 to 0.14 mg/kg wet weight (mean: 0.07; Menzie-Cura 1993c).

## 4.4 Upstream and Downstream Transport of COCs from MBA

Contaminants of concern entering the Delaware River from MBA will most likely be transported downstream from MBA due to current flow past the site. However, because the stretch of the Delaware River along which MBA is located is tidally influenced, it is also possible that upstream transport of COCs may occur during flood tide conditions. During the summer of 1974 a dye study was conducted at Philadelphia's Northeast Wastewater Pollution Control Plant (NEWPCP), located approximately 6.5 km below MBA on the Delaware River. The dye study involved a four-day release through the NEWPCP effluent. Dye was detected more than eight km upstream of the plant discharge, although at concentrations less than where the dye entered the river at NEWPCP. Dye was reported to be detectable at a distance of 24 km downstream of NEWPCP (Ambrose 1986).

The results of the dye study indicate that COCs from MBA may be transported upstream as far as eight km. However, in the dye study the dye was dissolved in the water column. The majority of the PCBs discharging from MBA are most likely sorbed to suspended particulate matter in the water column, with only a fraction of the discharge being dissolved in the water column. Some of the particulate-bound PCBs will settle out of the water column into the sediments.

4.5 Uncertainty Analysis

4.5.1 General

There are basically two types of uncertainty, analytical and non-analytical. Analytical uncertainty is the uncertainty resulting from the reliability and accuracy of the analytical methods employed in data collection and analysis. To a certain degree analytical uncertainty can be quantified by performing replicate measurements, by determining the method detection or quantification limits, in general by good QA/QC procedures. Non-analytical uncertainty results from the type of data collected, for example, the location of sampling stations, the type of organism sampled, or the chemicals analyzed for, as well as human error. Non-analytical uncertainty can be only poorly quantified, if at all.

AR3009644-27

Exposure consists of two components: the concentration (or dose) of a chemical to which an organism is exposed and the duration of exposure. No data were available describing how fish and aquatic invertebrates in the Delaware River use the river near the site and therefore, the possible duration of exposure that might be expected for these organisms is unknown. This uncertainty discussion will be limited to the uncertainty in determining the exposure concentrations.

Uncertainty in the PCB concentrations are the result of the Aroclor methods of analysis that do not take into account the differences between the composition of the PCBs found in environmental samples and commercial Aroclor mixtures. These differences are the result of physicochemical and biological processes that differentially affect individual congeners (Safe et al. 1987). The solubility and volatility of PCB congeners range over several orders of magnitude and greatly affect the partitioning between sediment-water and water-air interfaces. Analysis by the Aroclor method and reporting the results as total PCBs do not take into account this differential behavior of the individual congeners. Resulting errors in PCB concentrations derived by Aroclor analysis can be greater than 200 percent in either direction (Eganhouse and Gossett 1991). Also the reporting of PCB concentrations as total PCBs does not indicate the relative concentration of individual congeners.

### 4.5.2 Surface Water Concentrations

No samples of Delaware River water were analyzed; therefore, nothing can be said about the actual concentrations of COCs in Delaware River surface water. Four samples from pooled water in the mudflats and three samples from groundwater seeps were the only surface water samples collected. The only contaminants tested for in these samples were PCBs and DDTs. Out of the seven samples, PCBs were detected once and 4,4' DDE was detected twice (Table 3-1). Because of this very limited database, concentrations of PCBs in surface water in a 15 m band adjacent to the site were estimated from groundwater concentrations. Only data from the two wells (MW-6 and MW-7) where PCBs were detected were used. These two wells are the closest wells to the riprap/mudflat area with the highest PCB concentrations and where groundwater seeps have been observed. If this is the only area of groundwater concentrations just before discharge; however, if groundwater discharges elsewhere around the site, the overall contributions of PCBs to the river from the groundwater might be expected to be lower than indicated by these values.

1

Other contributing factors to the uncertainty of the surface water exposure point concentrations for PCBs include the partial estimates of groundwater concentrations, because non-detects were factored into the calculations by using one-half of the detection limits, and the estimates of groundwater and river flow rates that were the basis for the dilution factors. Because of the conservative nature of most of these estimates, the PCB concentration in the groundwater discharge may be overestimated.

#### 4.5.3 NAPL Concentrations

The extent and even the existence of a NAPL on site is uncertian. The single NAPL sample analyzed was from MW-6 in 1991; since then, while attempts have been made, no sampleable NAPL has been found in connection with the groundwater. During the most recent round of sediment sampling, while no NAPL was seen seeping from the site, a subsurface NAPL layer was observed in the lower riprap during low tide.

The suite of chemicals analyzed for in the single NAPL sample from 1991 included: PCBs, PAHs, DDTs, pthalates and only two metals (arsenic and mercury) (HMM 1993b). While the reported value for PCBs in NAPL might be considered reasonably accurate because it agrees with earlier reported values (Weston 1978) detection limits for many of the other contaminants tested for were exceedingly high. Only one PAH (pyrene) and one pthalate (DEHP) was detected; the detection limit for the others was 750 mg/l (Table 3-1). Because of these high detection limits, the concentrations of the non-detected PAHs had to be estimated based on there concentrations in groundwater relative to pyrene. Different PAHs have different solubilities so the relative concentrations in groundwater would be expected to be different than the relative concentrations in NAPL. However, since pyrene has a relatively low water solubility compared to other PAHs, the estimated PAH concentrations are expected to err on the high side. A similar calculation could not be performed on the non-detected pthalates due to insufficient data. DDTs had detection limits so high (48 mg/l) that no conclusions could be drawn about their presence in NAPL.

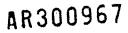
### 4.5.4 Sediment Concentrations

The exposure-point concentrations estimated for sediment of the Delaware River are based on limited data collected over a restricted area of the mudflat and adjacent nearshore Delaware River. Much of the Delaware River analytical data for PCB concentrations in sediments were of poor quality due to high detection limits, possibly caused by matrix interference. High and variable detection limits were also a problem for DDTs and trace elements in the

mudflats (Table 3-2). The exposure-point concentrations for sediments calculated from these data may under- or over-estimate the actual concentrations aquatic organisms may be exposed to in areas outside those sampled. Furthermore, nothing is known of the bioavailability of PCBs in sediments of the mudflat and Delaware River, but PCBs were detected in Asiatic clams collected from PCB contaminated sediments of the mudflats. Silvery minnows and channel catfish collected near the site also contained PCBs in their tissues indicating that PCBs are available to biota using the mudflat and Delaware River.

## 4.5.5 Tissue Concentrations

As mentioned in Section 4.5.1, biological processes alter the pattern of congener distributions in tissue residue samples. Differences between uptake/depuration, metabolism, and efficiency of assimilation of individual congeners may result in large differences between the PCB mixtures in samples and commercial Aroclor mixtures, and these difference would be expected to increase with increasing trophic level. For example, an analysis of NOAA Status and Trends data from the Gulf of Mexico indicated that oysters preferentially accumulated penta-, hexa-, and tetrachlorobiphenyls compared to sediment PCBs (Sericano et al. 1990). The differential accumulation of the highly toxic coplanar PCBs in aquatic biota, which results in an enrichment of coplanar PCB concentrations in biota, also has been reported (Kannan et al. 1989; Smith et al. 1990). Therefore, the analysis of tissue samples by the Aroclor method is prone to even more error than that for water and sediment samples, and the results indicate little about the potential toxicity of the concentrations. Furthermore, since PCBs were analyzed for by individual congener analysis in clam (Corbicula) tissue, while Aroclor analysis was used for the fish tissue sampled, the comparability of the resulting data is questionable.



# 5 TOXICITY ASSESSMENT

To assess the risks to receptor populations due to exposure to contaminants in the mudflat and the Delaware River near MBA, we need to determine toxicity criteria or guidelines against which the environmental concentrations can be compared. As described in Section 4, we will evaluate three exposure pathways to assess the potential effects of contaminants of concern on aquatic organisms. These three routes will require identifying appropriate toxicity reference concentrations for water, sediment, and tissue accumulation (PCBs only). Because no toxicity testing or other direct assessment of biological effects was conducted for any of the potential exposure media, the toxicity assessment will be based entirely on a review of published information on the toxicity of PCBs and other contaminants of concern to aquatic organisms.

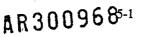
### 5.1. Toxicity Reference Concentrations for Water

5.1.1 Toxicity Reference Concentrations for Surface Water

PCBs are the only identified contaminant of concern for surface water exposure at the MBA site. AWQC, which were derived to be protective for most aquatic organisms in an area of exposure, provide appropriate toxicity reference concentrations for most substances. However, the AWQCs for PCBs are based on "Final Residue Values," which were calculated to protect wildlife and human consumption of aquatic organisms contaminated with PCBs and are not based on toxicity to aquatic organisms. Nevertheless, the data compiled by EPA on the toxicity of PCBs in developing the AWQC can be used to determine appropriate toxicity guidelines for the risk characterization for water exposure.

The majority of the information on the toxicity of PCBs to aquatic organisms resulting from exposure to PCBs in water has been generated from laboratory tests exposing animals to PCBs dissolved in water. Many of the published studies were reviewed in the AWQC document (EPA 1980). The mean acute toxicity values for different freshwater and marine invertebrate and fish species reviewed in the AWQC document ranged from 2.0 to 283  $\mu$ g/l.

Toxicity reference levels for effects of PCBs in water were derived from chronic limits values in the AWQC document for PCBs (Table 5-1; EPA 1980). These concentrations represent the range of no observed adverse effect level (NOAEL) to lowest observed adverse effect level (LOAEL) concentrations that were reported in chronic exposures (full or partial life-cycle or early life stage tests) for organisms exposed to a variety of commercial Aroclor PCB mixtures. These chronic limits are often referred to as maximum acceptable toxicant concentrations



(MATC). The lowest chronic limit is the highest test concentration in an acceptable chronic test that did not cause an unacceptable amount of adverse effect on any of the specified biological measurements, and below which no tested concentrations caused an unacceptable effect (Stephan et al. 1986). The lowest of these MATC values for freshwater organisms is 0.1  $\mu$ g/l, the no effect concentration for second generation fathead minnows exposed to Aroclor 1248 for 30 days (DeFoe et al. 1978). Exposure concentrations of 0.4  $\mu$ g/l and higher resulted in increased mortality of the larvae (most mortality occurred soon after hatching) as well as decreased growth of the survivors after 30 days.

The application of a MATC value of  $0.1 \,\mu g/l$  for PCBs, which was derived from a laboratory toxicity test on a single species, to species within different genera and in different classes under natural conditions results in some uncertainty. Although a concentration of  $0.1 \,\mu g/l$  may be protective of the majority of biota in the Delaware River in the vicinity of MBA, it may not be protective of sensitive species. The use of interspecies extrapolation factors in ecological risk assessment is a way to account for variability in sensitivity between species when extrapolating toxicity values to a species with no available data. Of particular concern in the Delaware River is protection of the endangered shortnose sturgeon. Calabrese and Baldwin (1993) recommend an uncertainty factor of 100 when comparing between species within different orders of the same class. Both the fathead minnow and shortnose sturgeon are in the same class (Osteichthyes), but are in different orders: fathead minnow, *Cypriniformes*; shortnose sturgeon, *Acipenseriformes*. Based on these considerations, a toxicity reference concentration of  $0.1 \,\mu g/l$  will be used for water exposure for all organisms except the endangered species, shortnose sturgeon. For the sturgeon,  $0.001 \,\mu g/l$  will be used as the toxicity reference threshold for PCBs in water.

## 5.1.2 Toxicity Reference Concentrations for NAPL

Freshwater AWQC acute toxicity values or lowest observed effect levels, if AWQC are unavailable, will be used for toxicity reference values for evaluating risk due to direct exposure to NAPL. For PCBs, the acute toxicity value is 2.0  $\mu$ g/l. For acenaphthene, fluoranthene, and naphthalene the lowest observed effects level values are 1,700, 3,980, and 2,300  $\mu$ g/l, respectively. For phenanthrene and DEHP, the proposed acute toxicity values are 30 and 400  $\mu$ g/l, respectively.

# Table 5-1. MATC and chronic values for PCBs (µg/l) based on exposure for life-cycle, partial life-cycle, or early-life stage tests (from EPA 1980).

Species	Type of PCB (Aroclor)	MATC (Chronic Limits) (µg/l)	Chronic Value (µg/l)
nvertebrates			
Cladoceran	1248	2.5 - 7.5	4.3
(Daphnia magna)	1254	1.2 - 3.5	2.1
\	1254	10=24	15
Scud (Gammarus pseudolimnaeus)	1248	2.2 - 5.1	3.3
Midge ( <i>Tanytarsus dissimilis</i> )	1254	0.5 - 1.2	0.8
Fish			
Brook trout			
(Salvelinus fontinalis)	1254	0.7 - 1.5	1.0
Fathead minnow	1248	0.1 - 0.4	0.2
(Pimephales promela)	1254	1.8 - 4.6	2.9
<	1260	1.3 - 4.0	2.3
Sheepshead minnow (saltwater) ( <i>Cyprinodon variegatus</i> )	1254	0.06 - 0.16	0.098

# 5.2. Toxicity Reference Concentrations for Sediment

The ER-L and ER-M concentrations of Long and MacDonald (1992) will be used as toxicity reference concentrations for characterizing the potential risk to receptors exposed to contaminated sediments in the mudflat and the Delaware River. The percent incidence of adverse effects below the ER-L value for a chemical in the studies reviewed by Long and MacDonald indicate the relative protectiveness of the ER-L value. Similarly, the percent incidence of adverse effects at concentrations exceeding the ER-M value indicates the reliability of the ER-M in predicting adverse effects (Long et al. 1994). If ER-L and ER-M values are not available for the contaminant of concern, the lowest and highest AET values will be used. AET values, derived from effects data from Puget Sound sediments, are defined as the concentration above which effects were always observed for a given endpoint. Because effects may be

AR300970

observed at concentrations below an AET value, AETs are not considered to be highly protective and are usually more comparable to ER-M rather than ER-L values.

# 5.2.1 PCBs

The ER-L and ER-M concentrations for PCBs are 0.023 and 0.18 mg/kg dry weight, respectively (Long and MacDonald 1992). Of the studies reviewed by Long and MacDonald, less than 20 percent reported adverse effects at PCB concentrations below the ER-L and over 50 percent at PCB concentrations greater than the ER-M.

Limited data are available from laboratory spiked-sediment bioassays on sediment-associated PCBs with aquatic organisms. Significant mortality was reported for the meiobenthic copepod *Microarthridion littorale* exposed to Aroclor 1254-contaminated sediments at concentrations of 83 mg/kg, whereas lower concentrations (4 mg/kg) resulted in impaired reproduction (DiPinto et al. 1993). Swartz et al. (1988) determined that a lethal concentration (LC)<sub>50</sub> of 10.8 mg/kg for the marine amphipod *Rhepoxynius abronius* exposed to Aroclor 1254-contaminated sediment.

# 5.2.2 PAH

PAHs vary substantially in their toxicity to and bioaccumulation by aquatic organisms. In general, toxicity increases as molecular weight increases and with increasing alkyl substitution on the aromatic ring. In most fish, PAHs are rapidly metabolized and excreted so that concentrations found in edible tissue are generally low. Invertebrates such as molluscs do not metabolize PAHs as efficiently and may accumulate high levels of PAHs in tissue (Eisler 1987).

A laboratory study in which the freshwater amphipod *Diporeia* sp. was exposed to sediments dosed with a mixture of PAHs reported mortality at total PAH concentrations in the range of 100 mg/kg (dry weight) after 26 days of exposure (Landrum et al. 1991). The study also reported that the PAH action appears to be additive, with no evidence of synergism or antagonism. The rate of PAH accumulation depended upon the concentration of PAHs in the sediment and was not predictable through measured partitioning between interstitial water and sediment particles.

Estuarine fish collected from PAH-contaminated areas in the Elizabeth River (Virginia) showed reduced abundance (reduced total biomass, total numbers of individuals, and abundance of selected species) and increased prevalence of several gross abnormalities that correlated with PAH contamination in the sediments (24.4 to 154.4 mg/kg). Abnormalities observed included

fin erosion in hogchoker and toadfish, and cataracts in spot, gray trout, and croaker (Huggett et al. 1987).

The ER-L and ER-M concentrations of 4.0 and 44.8 mg/kg (Long and MacDonald 1992) will be used as sediment reference concentrations for total PAHs. Below the ER-L concentration 14.3 percent of the studies reviewed by Long and MacDonald reported adverse effects and 85 percent at concentrations above the ER-M.

## 5.2.3 DDT and Metabolites

The ER-L and ER-M concentrations of 1 and 7  $\mu$ g/kg will be used as sediment reference concentrations for p,p'-DDT (Long and Morgan 1991).

Spiked sediment bioassays with the freshwater amphipod *Hyalella azteca* resulted in an LC50 value for DDT of 11 mg/kg at 3 percent organic carbon (Nebeker et al. 1989). LC<sub>50</sub> values for spiked sediment bioassays with the marine amphipod *Rhepoxynius abronius*, ranged from 11.2-125.1  $\mu$ g/kg at 1 percent organic carbon (Word et al. 1987).

The ER-L and ER-M concentrations of 2.2 and 27  $\mu$ g/kg will be used as sediment reference concentrations for p,p'-DDE (Long and MacDonald 1992). Below the ER-L concentration, 5 percent of the studies reviewed by Long and MacDonald reported adverse effects and 50 percent at concentrations above the ER-M.

The ER-L and ER-M concentrations of 2.0 and 20  $\mu$ g/kg will be used as sediment reference concentrations for p,p'-DDD (Long and Morgan 1991).

The limited amount of data available and the fact that the incidence of effects did not show a consistent increase with increasing sediment concentration for any of the DDTs results in low confidence in these values.

#### 5.2.4 Phthalates

Very little information is available on the sediment toxicity of phthalate esters. In a laboratory study, the number of successfully hatched frog eggs exposed for 60 days to sediments spiked with DEHP was reported to decline with increasing concentrations of DEHP. Only 50 percent of the eggs hatched when exposed to a concentration of 150  $\mu$ g/g of DEHP (Larsson and Thurén 1987).

Since no ER-L or ER-M concentrations are available for any of the phthalate esters, the upper and lower AET concentrations (PTI, 1988) for DEHP, DBP and DOP will be used as reference concentrations. The AET concentrations are 1.3 and 3.1 mg/kg (dry weight) for DEHP, 1.4 and 5.1 mg/kg for DBP, and 0.4 and 6.2 mg/kg for DOP.

## 5.2.5 Cadmium

The ER-L and ER-M concentrations of 1.2 and 9.6 mg/kg will be used as sediment reference concentrations for cadmium (Long and MacDonald 1992). Below the ER-L concentration 6.6 percent of the studies reviewed by Long and MacDonald reported adverse effects and 65.7 percent at concentrations above the ER-M. The ER-L and ER-M values for cadmium must be used with caution because there are many factors, in addition to concentration, that can affect the bioavailability and toxicity of cadmium. In particular, cadmium bioavailability has been shown to be strongly influenced by the concentration of acid volatile sulfides (AVS) present in the sediment (DiToro et al. 1990; DiToro et al. 1992; Ankley et al. 1991). Excluding other factors, the higher the concentration of AVS the more cadmium is bound with a corresponding reduction in its apparent toxicity. AVS concentrations are highly variable both temporally and spatially (Howard and Evans 1993). Therefore, even if cadmium concentrations stayed relatively constant, cadmium toxicity could vary with varying AVS concentrations.

Several investigators have conducted 10-day bioassays with sediment spiked with cadmium using the amphipod *Rhepoxynius abronius*; LC<sub>50</sub> values range from 6.9 to 11.5 mg/kg (Swartz et al. 1985; Kemp et al. 1986; Mearns et al. 1986; Robinson et al. 1988). No mortality or behavioral affects were observed in the polychaete *Nereis virens* in a 28-day bioassay at 20 mg/kg (Olla et al. 1988).

# 5.3. Toxicity Reference Concentrations for PCBs in Tissues

As is the case for sediments, no criteria exist for protective tissue concentrations of chemicals in aquatic organisms. A number of field and laboratory studies provide evidence of chronic sublethal effects on aquatic organisms at low PCB concentrations in tissues (Table 5-2). Of particular note are the studies reporting chronic toxicity, primarily reproductive effects, to several fish species at PCB tissue concentrations of less than 1 mg/kg wet weight and as low as 0.1 mg/kg wet weight. Other studies (e.g., Monod 1985; Mac and Schwartz 1992) have reported a significant relationship between PCB concentrations in tissue and reproductive

į.

endpoints in fish but were not included in the table because an lowest observed effect concentration could not be determined from the data available.

Table 5-2.	Tissue concentrations of PCBs (mg/kg) associated with adverse effects i	in
	freshwater and marine fish species.	

SPECIES	TISSUE	PCB TYPE	EXPOSURE	EFFECT CONC. (mg/kg)	EFFECT ENDPOINT	REFERENCE
Baltic flounder	Ovaries	Total PCB	FIELD	0.12	Reduced viable hatch	Von Westernhagen et al. 1981
Starry flounder	Eggs	Total PCB	FIELD	0.2	Reduced reproductive success; MFO induction	Spies et al. 1985
Lake trout	Eggs	Total PCB (as 1254)	LAB	0.31	Reduced egg hatchability and fry survival	Mac and Edsall 1991
Coho salmon	Liver	1254	LAB	1.1	Mortality and delayed saltwater adaptation	Folmar et al. 1982
Rainbow trout	Whole body	1254	LAB	1.3	Kidney nephrosis	Nestel and Budd 1975
Striped bass	Gonads	1254	FIELD	1.4	Reproductive failure	Ray et al. 1984
Rainbow trout	Eggs	1242	LAB	2.7	Larval mortality and abnormality	Hogan and Brauhn 1975
Chinook salmon	Eggs	Total PCB	FIELD	3.5	Hatching success	Ankley et al. 1991
Lake trout	Whole body	1254	LAB	4.5	Larval mortality	Mac and Seelye 1981
Sheepshead minnow	Eggs	1254	LAB	7	Post-hatch survival	Hansen et al. 1974
Winter flounder	Eggs	1254	FIELD	7.1	Reduced growth in length and weight	Black et al. 1988
Fathead minnow	Unknown	1254	LAB	13.7	Reduced fecundity	ACOE 1988
Rainbow trout	Liver	Clophen A50	LAB	31	Liver enlargement	Lidman et al. 1976
Spot	Whole body	1254	LAB	46	Mortality	Hansen et al. 1971
Cyprinid minnow	Whole body	Clophen A50	LAB	170	Inhibition of reproductive development	Bengtsson 1980
Sheepshead minnow	Whole body	1016	LAB	200	Fry mortality	Hansen et al. 1975
3-spined stickleback	Liver	Clophen A50	LAB	289	Reduced spawning success	Holm et al. 1993
Fathead minnow	Unknown	1254	LAB	429	Reduced spawning	Nebeker et al. 1974
Fathead minnow	Unknown	1242	LAB	436	Reduced egg hatchability	Nebeker et al. 1974

Aquatic invertebrates appear to be much less sensitive than fish and other vertebrate species to effects of PCBs. Concentrations of PCBs in fish tissues in the range of 0.1 to 1 mg/kg wet

AR300974 5-7

weight have been reported to result in impaired reproductive success, whereas tissue concentrations in *Daphnia magna* as high as 130 mg/kg dry weight (approximately 13 mg/kg wet weight) have been reported to have no effect (Dillon et al. 1990).

To evaluate the risk presented by tissue concentrations of PCBs in tissues of fish collected from the Delaware River near the MBA site and estimated for other species, an approach was followed similar to that used by Long and Morgan (1991) for sediments. A cumulative frequency plot of fish tissue PCB concentrations associated with adverse effects was prepared from the fish data presented in Table 5-2 (Figure 5-1). The studies included both laboratory and field data with a variety of endpoints measured. The values presented are the lowest observed effect values. Table 5-3 lists effect values for other effects endpoints such as enzyme induction, which may not necessarily represent "adverse" effects. Enzyme induction by itself, though associated with the toxicity of chlorinated hydrocarbons, is not generally considered to be an "adverse" effect. The data in Table 5-3 provides additional evidence of the effects of PCBs on fish species at tissue concentrations less than 1 mg/kg.

SPECIES	TISSUE	PCB <sub>.</sub> TYPE	LAB EXPOSURE	EFFECT CONC. (mg/kg)		REFERENCE
Carp	Liver	1254	Single injection	0.1	Induced enzyme activity	Melancon and Lech 1983
Carp	Muscle	1248	Food exposure for 7 or 20 d	0.1	Blood glucose content & hematocrit values elevated; enzyme activities increased significantly.	Ito 1973
Rainbow trout	Liver	1254	Single injection	0.2	Induced enzyme activity	Melancon and Lech 1983
Rainbow trout	Muscle	1254	Injection	0.25	Induced enzyme activity	Melancon et al. 1989
Coho salmon	Thyroid	1254	Food exposure for up to 260 d.	0.28	Stimulated thyroid activity	Mayer et al. 1977
Channel catfish	Thyroid	1254	Food exposure for up to 193 d.	0.3	Stimulated thyroid activity	Mayer et al. 1977
Rainbow trout	Liver	1254	Single injection	0.32	Induced enzyme activity	Melancon et al. 1989
Bluegill	Muscle	1242	Lab study	0.6	Inhibits oligomycin- insensitive Mg++ ATPase	Desalah et al. 1972
Brook trout	Muscle & (Liver)	1254	Food exposure for 18 d	39	Induced enzyme activity	Addison et al. 1978
Channel catfish	Edible carcass	1242	Food exposure for 20 wks	62.2	Weight loss & liver hypertrophy	Hansen et al. 1976

 Table 5-3.
 Tissue concentrations of PCBs (mg/kg) associated with miscellaneous effects in freshwater fish species.



Figure 5-1 illustrates the wide range of concentrations of PCBs in tissues that have been associated with adverse effects. It should be noted that the PCB concentrations included in the table were from a variety of different tissues (i.e., eggs, liver, muscle, whole body). Although differences in tissue concentrations within the same fish can often be standardized by lipid normalization (Parkerton et al. 1993), the lack of lipid data for many of the studies precluded that approach. Impacts on reproductive success are sensitive indicators of toxicity and can have potential population-level effects. Because of the uncertainties in these data,  $0.2 \mu g/kg$  wet weight of tissue (the lower tenth percentile of the tissue effects concentrations listed in Table 5-2b) and the median concentration of 5.8 mg/kg will be used in this risk assessment as the toxicity reference concentration for PCBs in tissues for all fish species.

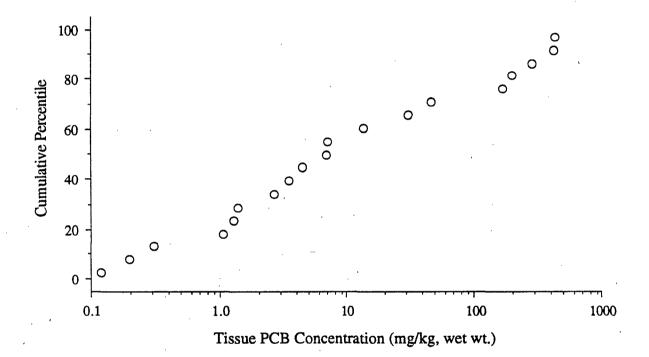


Figure 5-1. Cumulative percentile plot of the tissue concentrations of PCBs (mg/kg, wet weight) associated with adverse effects in fish species.

# 5.4 Uncertainty Analysis

5.4.1 General

5.4.1.1 Use of toxicity data

Extrapolating from data gathered from literature sources or from laboratory tests can be complicated by a number of factors:

- Data usually exist for only relatively few representative species exposed to single chemicals under laboratory conditions. Currently, no widely accepted protocols are available to evaluate the complex chemical, physical, and biological interactions that occur under natural conditions.
- Interspecies extrapolation from laboratory data generated for species other than the important receptor species is required. The data available suggest a wide range of sensitivity to PCBs between species.
- Few studies have examined the toxicity of simultaneous exposure to multiple contaminants. In the event such data are available, it is highly unlikely that the specific conditions and chemicals present at the site of concern would be duplicated in a laboratory setting.
- Most literature information on the toxicity of chemicals is based on fixed-time exposures of less than ten days, frequently 48 to 96 hours. These tests do not consider exposure to critical life stages over one or more generations of a population.

A wide variety of environmental parameters can influence both the nature and extent of effects of a contaminant on receptor organisms. These factors interact with each other, with contaminants, and with receptor organisms to affect the toxicity of the contaminant through a number of mechanisms:

- chemically changing the contaminant to make it more or less toxic;
- affecting the bioavailability of the contaminant; and
- making the organism more or less tolerant to the contaminant.

Among the many environmental factors that can affect the toxicity of a contaminant in the environment are temperature, pH, salinity, water hardness, and soil or sediment composition (e.g., TOC).



### 5.4.1.2 Use of total PCBs

Reporting environmental PCB concentrations as Aroclor equivalents or total PCBs provides limited information about the potential toxicity of the mixture of congeners in a sample, since the toxicity of individual congeners varies considerably (EPA 1988b; McFarland and Clarke 1989). The coplanar PCBs —a group of PCB congeners that are similar in structure and biological activity to the highly toxic polychlorinated dioxins and dibenzofurans (Safe 1984) are considered to be the most toxic PCBs. Although these coplanar PCBs are found at relatively low concentrations in the commercial Aroclor mixtures, several have been identified as important components of PCB tissue residues in aquatic biota and may be preferentially accumulated, particularly by higher-trophic-level organisms (Safe 1984; Hansen 1987; Kannan et al. 1988; Smith et al. 1990). Thus, PCBs in higher-trophic-level organisms may be more biologically active than the commercial mixtures (Parkinson and Safe 1987; Smith et al. 1990).

Most of the available PCB data from the MBA site and the Delaware River were derived from Aroclor determinations. Congener specific PCB analysis was conducted on a small number of samples (five *Corbicula* tissue samples, seven sediment samples, and one groundwater sample). About 40 individual congeners or congener pairs were quantified, but several of the most toxic coplanar PCBs were not quantified in this analysis.

## 5.4.2 Surface Water Toxicity

Most of the water toxicity data are derived from laboratory studies using commercial PCB mixtures. Because of the differences between commercial (e.g., Aroclor) PCB mixtures and environmental PCBs, the toxicity may be underestimated. For example, PCB-contaminated oil that was salvaged from used transformers, as at the Metal Bank site, may have higher PCDF concentrations than the original Aroclor mixture (EPA 1988b).

Considerable uncertainty is associated with the toxicity reference values for exposure of aquatic species and the shortnose sturgeon to PCBs in surface water, primarily due to the lack of adequate chronic toxicity data. The toxicity reference value for PCBs in surface water were based on a limited number of chronic (life-cycle or early life stage) tests and exposure to different Aroclor mixtures. The NOAEL concentrations for fish species showed over an order of magnitude difference between species, ranging from 0.1-5.4  $\mu$ g/l. The lowest MATC value was from a life-cycle study with fathead minnow, a species not considered to be highly sensitive. Because of the complete lack of information on the relative toxicity of PCBs to shortnose sturgeon compared to other species, a 100-fold safety factor was applied to the

# AR300978

5-11

toxicity reference value for other aquatic organisms. This was based on inter-species extrapolation and the need to protect individuals of an endangered species population. Consequently, the value used for the sturgeon may be overly protective.

## 5.4.3 Sediment Toxicity

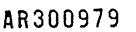
The uncertainty associated with the use of ER-L and ER-M concentrations as toxicity reference values for sediment exposures has both general and chemical specific aspects. The ER-L and ER-M values for PCBs and DDTs have a higher degree of uncertainty, since the relationship between sediment PCB and DDT (and metabolites) concentrations and the incidence of adverse biological effects was weaker than for most other contaminants evaluated (Long et al. 1993).

ER-L and ER-M values, although derived from a number of different studies, are based predominantly on acute toxicity test endpoints. Consequently, concentrations less than the ER-L would be unlikely to result in acute toxicity to most species of aquatic organisms, but may not be protective for chronic effects. However, if the incidence of effects for a contaminant is high (i.e., greater than 75 percent) for concentrations exceeding the ER-M, adverse effects would be expected to occur at concentrations exceeding the ER-M.

Most of the data are derived from field studies matching sediment chemistry and biological effects measurements that may be complicated by the presence of multiple contaminants. Toxic effect levels for a specific chemical determined from studies with high concentrations of several contaminants may tend to overestimate the toxicity of individual contaminants. This may be part of the explanation for the lack of a strong positive relationship between the concentration of a contaminant and the incidence of effects, such as described for PCBs and DDTs above.

## 5.4.4 Toxicity Associated with PCB Tissue Residues

The database for determining a fish tissue residue PCB effects concentration has a number of limitations: 1) Few studies were available that evaluated the post-hatch survival of larvae from eggs contaminated with PCBs via maternal transfer, which is most likely the chronic endpoint of most concern for PCBs. 2) Toxicity observed in field studies may have involved the effects of other contaminants as well as PCBs. 3) Laboratory studies used a variety of commercial PCB mixtures that may not accurately reflect the toxicity of environmental samples, which may be particularly important for higher trophic level fish species. 4) Concentrations of PCBs were measured in different tissues in different studies, and without lipid data for most studies, it was impossible to lipid-normalize the tissue concentrations, which might have provided



5-13

AR300980



greater consistency between studies. 5) Differences in methods of PCB analysis probably resulted in undetermined but significant inconsistencies between the different studies.

Reproductive and early life stage effects (e.g., reduced larval survival) from contamination of the eggs is one of the most sensitive toxicity endpoints for PCBs, as is the case for dioxin and dibenzofurans. The most sensitive endpoint for fish species to PCBs, as for dioxin and dibenzofurans, is most likely the post-hatch survival of larvae from eggs contaminated with PCBs via maternal transfer. Maternal transfer of PCBs from female fish to their offspring is known to occur (Ankley et al. 1991). Because of the lipophilic behavior of PCBs and the high lipid content in eggs of many fish species, maternal transfer of accumulated PCBs may be an important source of reproductive toxicity. Consequently, PCB concentrations in the ovaries of mature female fish may be the most useful measurement for estimating potential reproductive effects in the species of concern.

The tissue residue concentrations in fish species associated with adverse effects range over several orders of magnitude, implying a high degree of inter-species differences in sensitivity to the effects of PCBs. Very few studies were conducted that examined the endpoint of most concern. No information was available on the relative sensitivity of important receptor species, including the shortnose sturgeon, to the reproductive toxicity of PCBs.

# 6 **RISK CHARACTERIZATION**

The risk characterization is the summary output of the risk analysis. The principal component of the risk characterization is the integration of the results of the exposure assessment and toxicity assessment to obtain an estimate of the level of effects that will result from the exposure (Suter 1993). As defined in the U.S. EPA guidance for evaluating environmental risks at hazardous waste sites under CERCLA (EPA 1989 b,c), unless the risk assessment can be strictly limited to comparisons with existing ecological quality criteria, characterization of ecological risk should consist of a weight-of-evidence evaluation. Ecological risk assessments are seldom probabilistic in nature, since the probability of an adverse effect is difficult to quantify. Hence, the risk component of the assessment is usually defined by either the presence of an adverse impact based on actual measurements, the likelihood of an impact based on extrapolation from field and laboratory measurements, or the scientific literature. Field and laboratory measurements of impacts at the site provide site-specific information designed to reduce the uncertainty in predicting impacts to other habitats and receptor organisms.

The only information available for the weight-of-evidence approach at MBA are analytical chemistry data for sediment, surface water, and tissues of fish and invertebrates from the mudflat and Delaware River. With the exception of a qualitative benthic community survey conducted in the mudflat next to the site, no ecological surveys or direct effects assessments were performed to support the ERA. Therefore, the toxicity quotient approach (EPA 1989a) will be used to assess the significance of concentrations of chemicals in environmental media and tissues from the mudflat and Delaware River. The method compares the exposure-point concentrations developed in Section 4 to the toxicity reference concentrations developed in Section 5. Potential risk to receptor organisms is presumed to exist if the exposure-point concentration/toxicity reference concentrations (i.e., the value of the ratio "exposure concentration/toxicity reference concentration" is greater than one). However, it is also necessary to evaluate the frequency and duration of exposure to receptor organisms to fully characterize risk.

## 6.1 Water Exposure

## 6.1.1 Exposure to Surface Water

As noted in Section 4, a toxicity reference concentration for PCBs in water of 0.1  $\mu$ g/l was selected as appropriate for most aquatic organisms within the Delaware River. Because of the endangered species status of the shortnose sturgeon and the necessity of extrapolating between

different taxonomic orders, a more conservative reference concentration of 0.001 µg/l was selected to be used for the sturgeon. Hazard quotients were calculated for shortnose sturgeon and other aquatic organisms in a 15-meter band of the Delaware River next to the MBA site under average- and low-flow discharge conditions of the Delaware River. Under low-flow conditions, which represents a worst-case scenario, PCB concentrations in this area of the Delaware River were estimated to be between 1.34 and 1.97 ng/l, considerably below the toxicity reference concentration of 100 ng/l (Table 6-1). Therefore, most aquatic species in the Delaware River are not at risk from exposure to waterborne PCBs contributed by way of the groundwater from MBA during either average- or low-flow conditions of the river.

The average and upper 95 percent confidence limits estimated exposure-point concentrations for low-flow conditions and the upper 95 percent confidence limit for average-flow conditions slightly exceeds the toxicity reference concentrations used for the sturgeon. However, the toxicity reference concentration is based on life-cycle tests that presume chronic exposure (e.g., 240-day life-cycle exposure) of adults and larvae to the test concentration. While it is known that some adult shortnose sturgeon are found in the section of the river including the MBA site for extended periods during summer feeding following spawning (O'Herron et al. 1993), it is highly unlikely that any individual sturgeon would spend extended periods in the 15-meter band of the river next to the MBA site.

#### 6.1.2 Exposure to COCs in NAPL

The contaminant concentrations of a distinct NAPL were derived from the 1991 groundwater sampling from station MW-6. Subsequent attempts to sample or locate this layer have been unsuccessful. The existence of a NAPL in the subsurface of the riprap area has been observed but not sampled directly. No active NAPL seeps on the surface of the riprap or mudflat were observed during the 1993 sampling. Groundwater seeps, which have been observed recently, have resulted in "oily sheens" on the surface as opposed to a distinct oil layer. Thus, it appears that any direct seepage of oil to the surface of the riprap or mudflat is probably highly localized, if it occurs to any significant extent.

Organisms inhabiting the mudflat and the Delaware River immediately adjacent to the site may experience localized exposure to NAPL with high concentrations of PCBs. Species with limited mobility, such as clams, that occupy the mudflat may experience long-term exposure to elevated PCB concentrations in NAPL discharging to the mudflat. The PCB concentration in NAPL at monitoring well MW-6 was 1,090 mg/l in 1991. This concentration exceeds by a factor of 0.5 million the reference concentration of 2  $\mu$ g/l for PCBs for most aquatic

organisms. Estimated concentrations of individual PAHs exceeded toxicity reference concentrations by factors ranging from 20 to 4,500. The concentration of 590,000  $\mu$ g/l for DEHP in NAPL is nearly 1,500 times the toxicity reference concentration. Localized exposure to the high concentrations of PCBs, PAHs, and phthalates in any NAPL seep is likely to result in acute toxicity to organisms located in the path of these seeps.

Table 6-1. Hazard quotients for exposure to water-borne PCBs in the Delaware River under low-flow and average-discharge conditions for aquatic organisms and shortnose sturgeon.

	Hazard Quotients						
Delaware River Flow Conditions	Mean -	UL <sub>0.95</sub>					
Aquatic Organisms							
Average flow	(0.79 ng/l)/100 ng/l = 0.0079	(1.2 ng/l)/100 ng/l = 0.012					
Low flow	(1.34 ng/l)/100 ng/l = 0.0134	(1.97 ng/l)/100 ng/l = 0.0197					
Shortnose Sturgeon							
Average flow	(0.79 ng/l)/1 ng/l = 0.79	(1.2 ng/l)/1 ng/l = 1.2					
Low flow	(1.34 ng/l)/1 ng/l = 1.34	(1.97 ng/l)/1 ng/l = 1.97					

# 6.2 Exposure to COCs in Sediments

The primary route of exposure for benthic organisms in the nearshore areas of the Delaware River and mudflat near MBA is through contact with sediments (or sediment interstitial water). Based on comparisons the mean and upper 95 percent confidence limit concentrations of PCBs in sediments of the mudflat and Delaware River to the toxicity reference values discussed in Chapter 5, hazard quotients were calculated for exposure of receptor organisms to contaminated sediments near MBA as follows:

Hazard Quotient = Mean (or UL0.95) PCB Concentration in Sediments/TRV

The calculated hazard quotients are summarized in Table 6-2. The most protective of the hazard quotients, which was calculated by dividing the  $UL_{0.95}$  by the ER-L (or the lowest AET if no ER-L are available), represents the worst-case risk estimate associated with exposure to sediments; the least protective hazard quotient was calculated using the mean sediment concentration and the ER-M.

Because the pattern of sediment contamination for PCBs, PAHs, and the phthalate DEHP showed a clear decrease in concentration with distance from the site boundary (Tables 4-3. 4-5. and 4-6), sediment samples for these contaminants were grouped into three separate zones for risk characterization: samples from the riprap area; mudflat and Delaware River samples within 30 meters of the site boundary; and mudflat and Delaware River samples greater than 30 meters from the site boundary. No similar gradient was apparent in sediment concentrations of DDT and metabolites or cadmium; therefore, the risk evaluation for these contaminants will be based on the combined values for all sediment samples.

## 6.2.1 Total PCB in Sediment

Average sediment PCB concentrations ranged from almost 6 mg/kg in the riprap to less 1 mg/kg in samples collected from more than 30 meters beyond the site boundary. All PCB samples exceeded the ER-L value and 27 of 31 samples above detection limits exceeded the ER-M. Hazard quotients for PCBs exceeded 1 for all three zones, ranging from over 400 in the riprap to less than 5 in the outer zone (Table 6-2).

### 6.2.2 Total PAH in Sediment

Hazard quotient values for total PAH showed a steep gradient from the site, ranging from 7 to 246 in the riprap, 1.2 to 22 in the nearfield zone, and 0.1 to 1.1 in the farfield zone (Table 6-2).

### 6.2.3 *Phthalates*

Concentrations of phthalates in the sediment also were much higher in the riprap area than in the mudflat sediments. Hazard quotient values for DEHP were much higher in the riprap (93-623) than in the nearfield samples (1.0-3.8), but were less than 1 in the farfield samples (Table 6-2). Hazard quotient values for DBP exceeded 1 only for the low AET in the riprap area, with a maximum value of 2.0. Hazard quotient values for DOP ranged from 3.2 to 140 in the riprap area, 0.2 to 4.0 in the nearfield samples, and less than 1 in the farfield samples.

#### 6.2.4 DDT and Metabolites

Sediment concentrations of DDT, DDE, and DDD showed no clear pattern. Hazard quotients were similar for DDT and metabolites, ranging from 15 to 131 for DDT, 5 to 76 for DDE, and 7 to 84 for DDD. Almost all samples that had detectable values exceeded the ER-M value (Table 6-2).

Table 6.2. Hazard quotients for sediment exposure based on toxicity reference values (TRV) for COC in sediment and the mean and upper 95 percent confidence limit of sediment COC concentrations.

COC	TRV	RIPRAP					>30 METERS			
		Mean	UL 0.95	Mean	UL 0.95	Mean	UL 0.95			
PCBs ER-L ER-M		260.8 32.9	414.3 52.2	168.1 21.2	220.6 27.8	38.2 4.8				
Total PAH ER-L ER-M		77.3 6.9	245.8 21.8	13.0 1.2	.22.0 2.0	1.0 0.1				
DEHP AET-Low AET-High		220.8 92.6	623.1 261.3	- - 2.3 1.0	3.8 1.6	0.2 0.1				
DBP AET-Low AET-High		1.4 0.4	2.0 0.5	0.3 0.1	0.5 0.1	0.1 0.0	1			
DOP AET-Low AET-High	1	50.0 3.2	140.0 9.0	2.4 0.2	4.0 0.3	0.6 0.0	5 1			
		All Samples	Combined							
	(()	Mean	UL 0.95	-						
DDT ER-L ER-M		106.0 15.1	131.0 18.7							
DDE ER-L ER-M			75.9 6.2							
DDD ER-I ER-N										
Cadmium ER-I ER-N										

## 6.2.5 Cadmium

Hazard quotient values for cadmium ranged from 7.6 to 144 (Table 6-2). Of the six samples with concentrations above detection limits, five exceeded the ER-M value.



### 6.2.6 Benthic Community Survey

In July 1991 the Philadelphia Academy of Natural Sciences conducted a benthic macroinvertebrate survey of the mudflat next to the Metal Bank site (HMM 1993a). Qualitative sampling was conducted at mudflat stations MF-1 through MF-12, while more quantitative sampling was conducted at six of the stations (MF-1, -3, -4, -6, -10, and -11). The Academy assigned the following descriptions to the sampling results for each taxa: not found, rare, occasional, or abundant. To better compare the 12 stations, the subjective descriptors used by the Academy for each taxa were given numerical values from 0 to 3 respectively. These numerical values were summed for each station and the stations ranked by the total scores (Table 6-3). Greater abundance of an individual taxa or the number of different taxa would result in a higher station score.

Table 6-3.	Total score, density of organisms, total PCB concentrations in
	sediments (mg/kg, dry weight (DW), and TOC normalized) and
	distance from the MB site boundary for mudflat stations. Total
•	score is based on qualitative benthic survey data. (HMM 1993a
	and 1993b)

Station ID	Total Score	Density/ 1000 cm3	Total PCB mg/kg DW	Total PCB mg/kg TOC	Meters from Site
MF-1	6	1614	365	10429	21
MF-6	6	1203	198	5809	45
MF-5	7		4080	1 <b>43662</b>	21
MF-2	10		350	6533	46
MF-8	10		745	23281	143
MF-9	11		290	4545	27
MF-7	12		315	11842	85
MF-11	12	643	2300	44487	104
MF-3	13	1300	1220	26638	85
MF-4	13	1193	470	13128	143
MF-10	13	.1204	1080	105882	52
MF-12	18	•	1490	60324	158

A comparison of the total PCB concentrations (normalized to dry weight or TOC) in the sediments at the station and the station score, based on the May 1991 sediment sampling (HMM 1993b), showed no apparent correlation. This is not surprising since the samples for

MB Aquatic ERA

chemical analysis and benthic organisms were taken at different times; thus, the reported PCB concentrations may not have been representative of the sediments from which the organisms were sampled. No relationship was apparent between total density of organisms at a station and either PCB concentrations or distance from the site; although, total density was determined for only a limited number of stations. There was, however, a correlation between increasing distance from the western site boundary and higher station scores, based on the qualitative benthic survey. These results suggest that productivity in the mudflat in the immediate vicinity of the Metal Bank site may be reduced.

# 6.3 PCBs in Tissues

Concentrations of PCBs in the tissues of fish species represent an integration of all exposure pathways. PCBs were found in the tissues of *Corbicula*, channel catfish, and silvery minnows collected from the Delaware River next to the MBA site (Tables 4-8, 4-9, and 4-10). Because invertebrates appear to be much less sensitive to PCB tissue residues than fish species, the PCB concentrations in the *Corbicula* were not evaluated for potential toxicity due to tissue residue effects. To estimate the potential risk to channel catfish and silvery minnows, and indirectly to other fish species including the shortnose sturgeon, the mean and upper 95 percent confidence limit PCB tissue concentration of whole body samples (2.1 and 2.9 mg/kg, wet weight) measured in catfish collected from the Delaware River next to MBA were compared to the toxicity reference concentrations for tissue (0.2 and 7.0 mg/kg, wet weight) (Table 6-4).

Similar comparisons were made for the silvery minnow collected at the site. Silvery minnows most likely forage in shallow water at high tide and thus may be feeding directly over the mudflat and nearshore areas next to the site where exposure to site-related contaminants would be highest. In addition, silvery minnows are likely to have a more restricted feeding area than larger, more mobile species such as the catfish and sturgeon. Silvery minnows collected in the Delaware River next to the site in 1993 had a mean whole body PCB concentration of 1.9 mg/kg wet weight.

The calculated hazard quotients, based on the tenth percentile tissue residue risk reference value exceeded 1 by an order of magnitude for both the channel catfish and silvery minnow (Table 6-4). The hazard quotients based on the fiftieth percentile value, however, were less than 0.5 for both species. About one-third of the 19 studies listed in Table 5-2 reported adverse effects associated with fish tissue PCB concentrations less than the mean tissue concentrations for channel catfish and silvery minnow. These results indicate potential risk to

both fish species from elevated PCB concentrations in their tissues and by implication, to any sensitive fish species with comparable or greater exposure.

Table 6-4.	Hazard que									
	catfish and	silvery	minnow	collecte	d from	the	Delaware	River	near the	Metal
	Bank site.		· ·	• •	-			-		

	Hazard Quotients					
Species	Mean	UL <sub>0.95</sub>				
Channel Catfish	2.1 mg/kg	2.87 mg/kg				
10 percentile (0.2 mg/kg)	10.5	- 14.3				
Median (7.0 mg/kȝ)	0.3	0.4				
Silvery Minnows	1.9 mg/kg	2.23 mg/kg				
10 percentile	9.5	11.2				
median	0.27	0.32				



Channel catfish were selected as an indicator species for the endangered shortnose sturgeon because of their similar habitat needs and feeding strategies. If one assumes a lipid content of 20 percent in the ovaries of mature female sturgeon and applies the mean lipid normalized concentration for the channel catfish (28.2 mg/kg lipid), the equivalent wet weight concentration in the ovaries of female shortnose sturgeon would be 5.6 mg/kg. This level exceeds the concentration associated with adverse effects in 9 of the 19 studies included in Table 5-2. Based on the assumption that shortnose sturgeon would have a comparable exposure to PCBs, and thus tissue PCB concentrations equivalent to those of the channel catfish collected next to the site, shortnose sturgeon may be adversely affected by PCB contamination.

# 6.4 Uncertainty Analysis

6.4.1 General

Relatively little information was available describing the ecosystem in the Delaware River and mudflat next to the site. The frequency and duration of exposure of specific life stages of species of concern are key elements in determining the degree of risk associated with site-related contaminants. The lack of information regarding species use of the areas near MBA increases the level of uncertainty in the risk characterization.

AR300988

6-8

MB Aquatic ERA

Risk to aquatic organisms in the Delaware River due to exposure to contaminants of concern from MBA may result from simultaneous exposures to multiple contaminants in sediments, water, and food. The risk characterization assessed the risk due to exposure to individual contaminants within a given medium and did not consider simultaneous exposure to multiple contaminants in water, sediment, and prey. Furthermore, background concentrations of contaminants in Delaware River water were not considered when assessing exposure, so that species within the river may be exposed to higher concentrations than those presented in this assessment.

## 6.4.2 Surface Water Risk Characterization

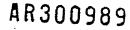
There is a high degree of uncertainty associated with the risk characterization for surface water due to the number of assumptions involved in the exposure and the toxicity assessments. The exposure-point concentrations for PCBs in surface water were derived from limited groundwater monitoring data from two monitoring wells with measured PCB concentrations above detection limits. Using data from these wells alone probably overestimates the overall average groundwater discharge concentration from the site.

The toxicity reference values for aquatic organisms were derived from the lowest chronic limit value reported in the AWQC document (EPA 1980). The limited amount of chronic toxicity data available for the effects of PCBs on fish species make it difficult to determine whether the toxicity reference value would protect most fish species. To take a conservative approach to estimating risk for the endangered shortnose sturgeon, a safety factor of 100 was applied to account for the inter-species extrapolation, the lack of information on the sensitivity of the sturgeon to PCBs, and the need to protect individuals of an endangered species population.

The exposure area was limited to a 15-meter-wide band of the Delaware River next to the site. Very little information is available on the distribution of shortnose sturgeon, and although it is known that individual sturgeon may remain within 5 km of the site during the summer feeding period, the duration and frequency of exposure to the area of concern are not likely to be for extended periods. As a consequence of the conservative nature of most of these assumptions, the characterization of risk due to surface water probably overestimates the actual risk to shortnose sturgeon and other aquatic species.

## 6.4.3 NAPL Risk Characterization

The major uncertainty for the NAPL risk characterization concerns the extent of exposure to NAPL. No recent, direct evidence of active NAPL seeps is available, and recent efforts to



collect samples of NAPL from the groundwater monitoring well where NAPL had been sampled in 1991 were unsuccessful. The level of exceedance of toxicity reference values based on acute toxicity by the concentrations of PCBs, PAHs, and phthalates in previously collected NAPL samples suggests that any exposure to NAPL would result in acute toxicity to benthic organisms.

## 6.4.4 Sediment Risk Characterization

The lack of data on the extent of sediment PCB contamination in the Delaware River near the site, including both downstream and upstream areas that could reasonably be assumed to have been impacted by releases from the site, makes for uncertainty in determining the exposure area and exposure-point concentrations. This is compounded by problems with data quality (i.e., high detection limits and samples of relatively coarse-grained sediment) for some of the existing information.

The uncertainty associated with the use of ER-L and ER-M values for estimating risk varies, to some extent, with the individual contaminants of concern. For PCBs and the DDTs, the lack of a consistent pattern of increasing incidence of effects with increasing concentration, indicates a higher degree of uncertainty in predicting adverse effects for these contaminants. For example, the incidence of effects observed at PCB concentrations exceeding the ER-M was 51 percent compared to 85 percent for total PAH (Long et al. 1994).

The benthic community assessment provides some evidence of reduced abundance and/or diversity at mudflat locations closest to the site boundary, which is consistent with observed pattern of sediment contamination.

#### 6.4.5 Tissue Residue Risk Characterization

The major sources of uncertainty for the tissue residue risk characterization are the determination of the toxicity reference values for PCBs and inter-species extrapolation to estimate the potential exposure to species of concern.

An important assumption in the evaluation of risk was that toxicity information developed for one species would be directly applicable to species of concern in the Delaware River. In particular, the risk to shortnose sturgeon was assessed based entirely upon toxicity data for other aquatic species. Because of the endangered status of the shortnose sturgeon, a conservative approach was taken in assessing risk to this species; however, there is substantial uncertainty as to whether the risk characterization under- or over-estimated the actual risk

MB Aquatic ERA

presented to this species due to exposure to site-related contaminants in the mudflat and Delaware River.

Another major source of uncertainty was the selection of the channel catfish as a surrogate species for the shortnose sturgeon, which was based upon assumed general similarities in habitat use and trophic level. Thus, it was assumed that the exposure to the shortnose sturgeon would be comparable to the exposure to the channel catfish, as measured by the tissue PCB concentrations in the catfish. Differences in trophic level, life span, age at sexual maturity, and migratory behavior between the two species are variables that can affect exposure. The shortnose sturgeon may be particularly vulnerable to the accumulation of PCBs due to their benthic feeding habit, their longevity, and relatively late age of maturity. Shortnose sturgeon are known to live as long as 67 years (Gilbert 1989). The fact that female sturgeon may spawn only once every several years (Gilbert 1989) may be an additional factor leading to increased accumulation of PCBs, since transfer of maternal PCBs to their eggs is thought to be an important mechanism in reducing the PCB body burden in female fish. Large channel catfish may feed on fish to a greater extent than shortnose sturgeon (Menzie-Cura 1993b), which indicates that catfish occupy a higher trophic level than the sturgeon and consequently would be expected to have a higher bioaccumulation factor. Both species spawn in the non-tidal freshwater section of the Delaware River and are known to have seasonal movement patterns. However, it is not known how long the catfish collected near the Metal Bank site had been in the area.

The size of the channel catfish collected in the immediate site vicinity indicates that they were young adult fish. Since PCB concentrations generally show an increase with age (Sloan et al. 1985; Connell 1987), the levels observed in the catfish used in this risk assessment may underestimate the concentrations in older individuals.

No data were available on shortnose sturgeon tissue PCB concentrations from the Delaware River near the site, but limited data were available on tissue PCB concentrations from six shortnose sturgeon collected in 1982 from the Delaware River 32 km upstream from the Metal Bank site (New Jersey Department of Environmental Protection data, provided by Menzie-Cura 1993c). These fish had PCB concentrations ranging from less than 0.05 mg/kg in fillets to 0.14 mg/kg in the ovary of one fish. However, most of these fish were collected in July when some other adult sturgeon are known to remain in the tidal portion of the river that includes the Metal Bank site (O'Herron et al. 1993).

AR300991

6-11



### 6.5 Conclusions

Surface water exposure from groundwater discharge is reduced rapidly with dilution from the Delaware River. Only PCBs were considered a COC for surface water. Any exposure to PCB concentrations exceeding toxicity reference values would be confined to a 15-meter-wide band of the river next to the site. The risk of adverse effects is expected to be low. The highly conservative toxicity reference value for the shortnose sturgeon exceeded the estimated mean exposure concentration for PCBs in the dilution zone under low-flow conditions by less than a factor of two, indicating possible risk. However, it is highly unlikely that individual shortnose sturgeon would remain in the exposure area long enough to receive chronic exposure.

Any exposure to NAPL, if it occurs, is likely to be highly localized in the immediate vicinity of the riprap area. However, because measured concentrations of PCBs, PAHs and phthalate in NAPL exceeded toxicity reference concentrations by as much as five orders of magnitude, toxic effects to exposed organisms are highly probable. Any discharge of NAPL would also contribute to PCB accumulation in nearshore organisms and food webs.

The primary route of exposure for benthic organisms in the nearshore areas of the Delaware River and mudflat near MBA is through contact with sediments (and/or sediment interstitial water). The highest levels of PCB and PAH contamination in sediments is restricted to a relatively small area immediately adjacent to the site. Mean sediment concentrations of PCBs, PAHs, and the phthalates bis (2-ethylhexyl) phthalate (DEHP), di-n-butyl phthalate (DBP) and di-n-octyl phthalate (DOP) greatly exceeded probable effects levels (ER-M or high AET) in the riprap area, indicating that adverse effects to benthic invertebrates exposed to these contaminated sediments are highly likely. Concentrations of PAHs and phthalates decreased in a steep gradient away from the site, resulting in hazard quotient values that were one or less in the mudflat and Delaware River farther than 30 meters from the site boundary. Hazard quotients for PCBs exceeded 1 for all three zones, ranging from over 400 in the riprap to less than 5 in the outer zone. No spatial pattern was observed in the concentrations of DDT, DDD, DDE, and cadmium, based on limited sampling. Samples with detectable concentrations exceeded probable effects levels (ER-M or high AET) by about an order of magnitude.

Because of limited sampling of Delaware River sediment, the extent of PCB contamination was not adequately defined and could extend both downstream and upstream of the site, although concentrations appear to decline rapidly with distance from the site boundary. Limited data are available for contaminants other than PCBs and PAHs. Problems with data quality (high detection limits for PCBs and other contaminants) further increase the uncertainty in the

MB Aquatic ERA

exposure-point concentrations and the size of the exposure area. The minimum exposure area includes intertidal and nearshore sediment in the Delaware River next to the site. The lack of consistent increase in incidence of effects with increasing PCB concentrations in the data used to derive the toxicity reference value adds an additional source of uncertainty to the risk characterization.

The limited information available in the literature suggests that benthic invertebrates can accumulate high tissue concentrations of PCBs without adverse effects, although invertebrates may experience direct toxicity from exposure to contaminated sediments. Tissue PCB concentrations in Asiatic clams in the mudflat, however, demonstrate that PCBs in the area are bioavailable and are likely to be accumulated in benthic feod webs. The BAF (lipid-normalized tissue to organic carbon-normalized sediment ratio) observed for Asiatic clams was consistent with literature values. Congener-specific PCB analysis showed very similar patterns for the mudflat sediment (and one groundwater sample) and clam tissue residues, suggesting that PCBs from the site were a probable source for the PCBs in the clam tissue.

Mean whole body PCB concentrations in tissue of silvery minnows and channel catfish collected in the Delaware River next to the site exceeded the tenth percentile of the lowest observed adverse effect concentrations associated with tissue residues in fish species, but not the median value. The silvery minnow is considered to be representative of non-migratory forage fish species that feed in the immediate nearshore area. Exposure to site-related PCBs in this species is likely. Channel catfish are probably less restricted in their movements than the silvery minnow, so the degree of exposure to site-related contaminants is less certain. Based on the mean PCB concentration in the samples collected more than 30 meters from the site boundary, the observed BAF for silvery minnow was comparable to literature values, while the BAF for channel catfish was less than expected for similar trophic-level fish species reported in the literature. The fact that the silvery minnows have a larger BAF than the channel catfish further suggests that they are exposed on average to sediments containing higher levels of PCBs than are the channel catfish. Using these species as representative of other fish species with similar exposure to the nearshore PCB-contaminated sediments immediately adjacent to the Metal Bank site or to nearshore food webs, the results suggest a potential risk of reproductive effects in sensitive fish species.

Considerable uncertainty exists in estimating the extent of exposure for the sturgeon, and nothing is known about its relative sensitivity to adverse effects from accumulated PCBs. Channel catfish were also used as a surrogate species for estimating the accumulation of PCBs in the endangered shortnose sturgeon. Although they occupy a lower trophic level than the

AR300993 6-13

### MB Aquatic ERA

March 1994

channel catfish, the shortnose sturgeon may be particularly prone to accumulating and transferring high concentrations of PCBs to their developing offspring (considered the most sensitive toxic endpoint for PCBs to fish) due to their benthic feeding habit, longevity, late age of sexual maturity, and high lipid content of their eggs. Thus, the potential risk to shortnose sturgeon resulting from accumulation of PCBs from all exposure pathways near the site may be greater than for other fish species.

# 7 **REFERENCES**

Addison, R.F., M.E. Zinck, and D.E. Willis. 1978. Induction of hepatic mixed-function oxidase (MFO) enzymes in trout (*Salvelinus fontinalis*) by feeding Aroclor 1254 or 3-methylcholanthrene. *Comp. Biochem. Physiol.* 61C:323-325.

Ambrose, R.B., Jr. 1987. Modeling volatile organics in the Delaware River. Journal. Environmental Engineering 113:703-721.

Ankley, G.T., G.L. Phipps, E.N. Leonard, D.A. Benoit, V.R. Mattson, P.A. Kosian, A.M. Cotter, J.R. Dierkes, D.J. Hansen, and J.D. Mahoney. 1991. Acid-volatile sulfide as a factor mediating cadmium and nickel bioavailability in contamianted sediments. *Environmental Toxicology and Chemistry 10*: 1299-1307.

Ankley, G.T., D.E. Tillitt, J.P. Giesy, P.D. Jones, and D.A. Verbrugge. 1991. Bioassayderived 2,3,7,8-tetrachlorodibenzo-p-dioxin equivalents in PCB-containing extracts from the flesh and eggs of Lake Michigan Chinook salmon (*Oncorhynchus tschawytscha*) and possible implications for reproduction. *Canadian Journal of Fish and Aquatic Science* 48:1685-1690

Ankley, G.T., P.M. Cook, A.R. Carlson, D.J. Call, J.A. Swenson, H.F. Corcoran, and R.A. Hoke. 1992. Bioaccumulation of PCBs from sediments by oligochaetes and fishes: comparison of laboratory and field studies. *Canadian Journal Aquatic Science* 49: 2080-2085.

BCM Engineers, Inc. 1988. Draft work plan for remedial investigation/feasibility study. Cottman Avenue Site, Philadelphia, PA. Prepared for the Cottman Avenue Interim PRP Group.

Bengtsson, B.E. 1980. Long-term effects of PCB (Clophen A50) on growth, reproduction and swimming performance in the minnow, *Phoxinus phoxinus*. *Water Research 14*: 681-687.

Black, D.E., D.K. Phelps, and R.L. Lapan. 1988. The effect of inherited contamination on egg and larval winter flounder, *Pseudopleuronectes americanus*. *Marine Environmental Research* 25: 45-62.

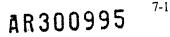
Brown, J.F. Jr., R.E. Wagner, H. Feng, D.L. Bedard, M.J. Brennan, J.C. Carnahan and R.J. May. 1987. Environmental dechlorination of PCBs. *Environmental Toxicology and Chemistry* 6: 579-593.

Brundage, H. 1988. Radio tracking studies of Shortnose Sturgeon (*Acipenser brevirostrum*) in the Delaware River for the Merill Creek Reservoir Project. Middletown, Delaware:.V.J. Schuler Associates, Inc.

Calabrese, E.J. and L.A. Baldwin. 1993. Performing Ecological Risk Assessments. Chelsea, Michigan: Lewis Publishers. 257 pp.

Camp, Dresser, and McKee, Inc. (CDM). 1989. Field activity report, Metal Bank Site. Philadelphia: Prepared for the U.S. Environmental Protection Agency.

Chou, S.F.J. and R.A. Griffin. 1986. Sediment quality critiera from the sediment quality triad: An example. *Environmental Toxicology and Chemistry* 5: 965-976.



Clement Associates. 1985. Chemical, physical, and biological properties of compounds present at hazardous waste sites. Washington, D.C.: Final report to the U.S. Environmental Protection Agency.

Clement International Corporation. 1992. Toxicological profile for DDT, DDE, and DDD (Draft). Atlanta, GA: Prepared for the U.S. Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry. 131 pp. + appendices.

Connell, D.W. 1987. Age to PCB concentration relationship with the striped bass (*Morone saxatilis*) in the Hudson River and Long Island Sound. *Chemosphere 16*: 1469-1474.

Connolly, J. P. 1991. Application of a food chain model to PCB contamination of the lobster and winter flounder food chains in New Bedford Harbor. *Environmental Science and Technology* 25: 760-770.

Cottman Avenue PRP Group. 1992. Preliminary draft appendix to RI report: Presentation of fish, sediment, and water data obtained in the Delaware River estuary by federal and state agencies.

Daiber, F.C. 1988. Finfish resources of the Delaware River Estuary. In: S.K. Mujumar, E. W. Miller, and E.L. Sage (ED.) *Ecology and Restoration of the Delaware River Basin*. Pennsylvania Academy of Science.

DeFoe, D.L., G.D. Veith, and R.W. Carlson. 1978. Effects of Aroclor 1248 and 1260 on the fathead minnow (*Pimephales promelas*). Journal Fisheries Research Board of Canada 35: 997-1002.

Delaware River Basin Commission (DRBC). 1987. Fish population study. DEL USA Project Element 9. Delaware Estuary Use Attainability Project. West Trenton, NJ: Delware River Basin Commission. 4 pp.

Delaware River Basin Commission (DRBC). 1988. Fish health and contamination study. DEL USA Project Element 10. Delaware Estuary Use Attainability Project. West Trenton, NJ: Delware River Basin Commission,

Delaware River Basin Commission (DRBC). 1993. Unpublished fish tissue residue data for PCBs in fish collected from the Delaware River: 1991-1992.

Desaiah, D., L.K. Cutkomp, H.H. Yap, and R.B. Koch. 1972. Inhibition of oligomycinsensitive and -insensitive magnesium adenosine triphosphatase activity in fish by polychlorinated biphenyls. Biochem. Pharmacol. 21: 857-865.

DiPinto, L.M., B.C. Coull, and T.G. Chandler. 1993. Lethal and sublethal effects of the sediment-associated PCB Aroclor 1254 on a meiobenthic copepod. Environ. Toxicol. Chem. 12: 1909-1918.

DiToro, D.M., J.D. Mahony, D.J. Hansen, K.J. Scott, M.B. Hicks, S.M. Mayr & M.S. Redmond. 1990. Toxicity of cadmium in sediments: the role of acid volatile sulfide. Enviro. Tox. & Chem. 9:12, pp. 1487-1502.

DiToro, D.M., J.D. Mahoney, D.J.Hansen, K.J. Scott, A.R. Carlson & G.T. Ankley. 1992. Acid volatile sulfide predicts the acute toxicity of cadmium and nickel in sediments. *Environ. Sci. Tech.* 26: 96-101.

AR300996



7-2

Dodd, J.A. 1992. Letter to C. Lee, RPM, U.S. Environmental Protection Agency, Philadelphia, PA (laboratory analytical results for PCBs in sediments and clams from the mudflat area adjacent to the Metal Bank of America NPL site). Environmental scientist, U.S. Environmental Protection Agency, Program Support Section, Annapolis, MD: 29 October.

Eganhouse, R.P., and R.W. Gossett. 1991. Sources and magnitude of bias associated with determination of polychlorinated biphenyls in environmental samples. *Anal. Chem.63*: 2130-2137.

Eisler, R. 1985. Cadmium hazards to fish, wildlife, and invertebrates: a synoptic review. U.S. Fish and Wildlife Service Biological Report 85(1.2). 46 pp.

Eisler, R. 1987. Polycyclic aromatic hydrocarbon hazards to fish, wildlife, and invertebrates: a synoptic review. U.S. Fish and Wildlife Service Biological Report 85(1.7). 72 pp.

Erickson, M.D. 1985. Analytical Chemistry of PCBs. Ann Arbor, MI: Ann Arbor Science Publishers. 508 pp.

Fisher, J.B., R.L. Petty, and W.Lick. 1983. Release of polychlorinated biphenyls from contaminated lake sediments: flux and apparent diffusivities of four individual PCBs. *Environmental Pollution* 5B: 121-132.

Flemer, D.A. and W.S. Woolcott. 1966. Food habits and distribution of the fishes of Tackahoe Creek, Virginia, with special emphasis on the bluegill, *Lepomis m. macrochirus* Rafinesque. Chesapeake Sci. 7:75-89.

Folmar, L.C., W.W. Dickhoff, W.S. Zaugg, and H.O. Hodgins. 1982. The effects of Aroclor 1254 and No. 2 fuel oil on smoltification and sea-water adaptation of coho salmon (*Oncorhynchus kisutch*). Aquatic Toxicology 2: 291-299.

Fox, M.E., J.H. Carey, and B.G. Oliver. 1983. Compartmental distribution of organochlorine contaminants in the Niagra River and the western basin of Lake Ontario. *Journal. Great Lakes Research* 9: 287-294.

Furukawa, K. 1986. Modifications of PCBs by bacteria and other microoranisms. pp. 89-100. In: *PCBs and the Environment*. J.S. Waid (ed.) Boca Raton, FL: CRC Press, Inc.

Gerking, G. 1958. The restricted movement of fish populations. Biol Rev. 34:221-242.

Gilbert, C.R. 1989. Species profile: life histories and environmental requirements of coastal fishes and invertebrates (Mid-Atlantic Bight). Atlantic and shortnose sturgeons. U.S. Fish and Wildlife Service Biological Report 82(11.122). U.S. Army Corps of Engineers TR EL-82-4. 28 pp.

Green, A.S., G.T. Chandler & E.R. Blood. 1993. Aqueous-, pore-water-, and sedimentphase cadmium: toxicity relationships for a meiobenthic copepod. *Enviro. Tox. & Chem.* 12:8. pp 1497-1506.

Greene, R.W. 1991. Summary and assessment of polychlorinated biphenyls (PCBs) in fish and shellfish from the Delaware Estuary. Dover, DE: Delaware Department of Natural Resources and Environmental Conservation, Division of Water Resources.