



# **ECOLOGICAL RISK ASSESSMENT for ATLANTIC WOOD INDUSTRIES**

**April 1992**

**Michael F. Buchman  
Jay Field  
Alyce Fritz  
Diane Wehner**

**Hazardous Materials Response Division  
National Oceanic and Atmospheric  
Administration  
Seattle, Washington**

**Robert Stuart  
Robert Dexter  
EVS Consultants, Inc.,  
Seattle, Washington**

**[This document has been recreated from the best available photocopy: although there are differences in layout from the original, the content remains essentially the same.]**



UNITED STATES ENVIRONMENTAL PROTECTION AGENCY

Region III

841 Chestnut Building Philadelphia, Pennsylvania 19107

April 13, 1992

FEDERAL EXPRESS

Atlantic Wood Industries, Inc.  
Sugar Refinery Road  
Port Wentworth, GA 31407

Attn: Ross F. Worsham  
Senior Environmental Engineer

Re: Final Ecological Risk Assessment (ERA) for the Atlantic  
Wood Industries (AWI) Superfund Site, Portsmouth, VA

Dear Mr. Worsham:

A final draft ERA report was provided to AWI and Keystone Environmental Resources, Inc. (Keystone) on October 24, 1991. We discussed this document during a December 4, 1991 telephone conversation and written comments from AWI were received by the U.S. Environmental Protection Agency (EPA) on December 19, 1991. For your information, I am enclosing the final ERA report, which was prepared by the National Oceanic and Atmospheric Administration (NOAA) under an Interagency Agreement (IAG) with EPA. All comments from AWI have been incorporated into the final ERA report.

As you know, discussions involving sediment target cleanup levels, which were developed to be protective of aquatic receptors, and associated ecological considerations regarding first operable unit remediation had been included in the final draft ERA. At my request, NOAA has removed the aforementioned information from this document. Target cleanup levels and related discussion are now included in a separate Technical Memorandum, which I will be receiving tomorrow. Upon receipt, I will forward a copy of this Technical Memorandum to AWI and Keystone.

Risk to terrestrial habitats at the AWI site was also discussed in the final draft ERA. This discussion remains, for the most part, unchanged in the final ERA. However, in accordance with the scope of work outlined in the IAG, I have requested that soil target cleanup levels be developed that are protective of terrestrial receptors. Recommended draft target cleanup levels have been prepared and are currently undergoing internal review through EPA's Interagency Biological Technical Assistance Group (BTAG). I anticipate that BTAG will complete its review in approximately one (1) week, after which time I will forward a copy of a second Technical Memorandum to AWI and Keystone.

## CONTENTS

<b>PURPOSE</b> .....	1
<b>SCOPE OF ASSESSMENT</b> .....	1
<b>SITE CHARACTERIZATION</b> .....	2
<b>AQUATIC ECOLOGICAL RISK ASSESSMENT</b> .....	2
<b>POTENTIAL RECEPTORS</b> .....	2
<u>Aquatic Habitats</u> .....	5
<u>Habitats of Special Concern</u> .....	6
<u>Species</u> .....	6
<b>EXPOSURE ASSESSMENT</b> .....	7
<u>Contaminants of Concern</u> .....	8
<u>Sources of Contaminants</u> .....	12
<u>Fate and Transport Analysis</u> .....	13
Polycyclic Aromatic Hydrocarbons.....	13
Pentachlorophenol .....	18
Polychlorinated Dibenzo-p-Dioxins and Dibenzofurans .....	18
Trace Elements .....	19
<u>Exposure Pathways</u> .....	20
<u>Estimated Exposure Levels</u> .....	20
<u>Uncertainty Analysis</u> .....	21
<b>TOXOCITY ASSESSMENT</b> .....	21
<u>Reported Effects of Contaminants of Concern</u> .....	21
Polycyclic Aromatic Hydrocarbons.....	21
Pentachlorophenol .....	26
Polychlorinated Dibenzo-p-Dioxins and Polychlorinated Dibenzofurans.....	26
Trace Elements .....	27
<u>Site-specific Toxicity Studies</u> .....	28
<u>Existing Toxicity-based Criteria and Standards</u> .....	28
<b>RISK CHARACTERIZATION</b> .....	29
<u>Comparison Against Criteria and Standards</u> .....	29
<u>Comparison of Estimated Exposure Levels with Toxicity Data</u> .....	30
<u>Adverse Biological Effects</u> .....	31

<u>Joint Action of Chemical Mixtures</u> .....	32
<b>TERRESTRIAL ECOLOGICAL RISK ASSESSMENT</b> .....	33
<b>POTENTIAL RECEPTORS</b> .....	33
<u>Terrestrial Habitat</u> .....	33
<u>Habitats of Special Concern</u> .....	33
<u>Species</u> .....	33
<b>EXPOSURE ASSESSMENT</b> .....	36
<u>Contaminants of Concern</u> .....	36
<u>Sources of Contaminants</u> .....	36
<u>Fate and Transport Analysis</u> .....	36
<u>Exposure Pathways</u> .....	36
<u>Estimated Exposure Levels</u> .....	36
Ingestion Doses.....	36
Dermal Exposure.....	37
Inhalation Exposure.....	37
<u>Uncertainty Analysis</u> .....	37
<b>TOXICITY ASSESSMENT</b> .....	39
<u>Reported Effects of Contaminants of Concern</u> .....	39
<u>Site-Specific Toxicity Tests</u> .....	42
<u>Existing Toxicity-based Criteria and Standards</u> .....	42
<b>RISK CHARACTERIZATION</b> .....	42
<u>Comparison of Estimated Exposure Levels with Toxicity Data</u> .....	43
<u>Adverse Biological Effects</u> .....	43
<u>Joint Action of Chemical Mixtures</u> .....	43
<b>REFERENCES</b> .....	44

# **ATLANTIC WOOD INDUSTRIES SITE ECOLOGICAL RISK ASSESSMENT**

## **PURPOSE**

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 with respect to releases or potential releases of contaminants from hazardous waste sites. The National Oil and Hazardous Substances Pollution Contingency Plan (NCP) of March 8, 1990, calls for identification and mitigation of the environmental impact of these sites and the selection of remedial actions that protect the environment. In addition, numerous federal and state laws and regulations are potentially “applicable or relevant and appropriate requirements” (ARARs).

The National Oceanic and Atmospheric Administration (NOAA) has entered into an Interagency Agreement (IAG) with the EPA to perform an Environmental Assessment at the Atlantic Wood Industries Superfund Site (Figure 1). In partial fulfillment of the requirements of the IAG, NOAA has prepared an ecological risk assessment report. The primary objective of the ecological risk assessment is to determine whether contaminants released to the environment at the Atlantic Wood site have impacted terrestrial and aquatic habitats.

## **SCOPE OF ASSESSMENT**

The ecological risk assessment for the Atlantic Wood site is based upon information from a variety of sources. Information presented in the ecological risk assessment regarding the site's history, physical description, geology, hydrology, and analytical chemistry were taken directly from the site's remedial investigation (RI) report prepared by Keystone Environmental Resources (KER), Inc. (1990). Aquatic and terrestrial resource information for the Atlantic Wood site and the surrounding vicinity was obtained through state and federal agencies. No site-specific information was available describing aquatic or terrestrial species that actually use the site or in what capacity they use it. Therefore, the resource information is general in nature and represents species that most likely could be found at or near the site.

The ecological risk assessment attempts to answer the following questions:

- What organisms are potentially exposed to contaminants from the site?
- What are the significant routes of exposure?
- To what amounts of each contaminant are organisms potentially exposed?
- What are the potential risks to aquatic and terrestrial species that come in contact with contaminants at the site?

The geographic scope of this ecological risk assessment is limited to the Atlantic Wood property; the associated shoreline of the Elizabeth River from the Jordan Bridge to the southern boundary of the site and extending out into the river to a line that includes the Atlantic Wood dock; and the river inlet between Jordan Bridge and the Atlantic Wood dock (Figure 2).

Although the geographic scope of this assessment is limited to the aforementioned areas, due to the lack of site-specific information, ecological evaluations of the surrounding vicinity have been included to provide representative habitat and species information. Only the primary contaminants (based on concentrations, toxicity, and association with site operations) were considered in this risk assessment. Not all compounds detected at the site were included. Chemical or toxicological site data for some trace contaminants (tar acids or bases) were unavailable, and, so, were not included.

A risk characterization was developed to determine whether contaminants have impacted terrestrial and aquatic habitats. Three approaches were used to describe the levels of soil and sediment contamination that cause significant environmental toxicity. Information from these approaches, plus other environmental studies can be used to develop target cleanup levels intended to protect aquatic resources.

## **SITE CHARACTERIZATION**

Atlantic Wood Industries is an active wood-processing facility that has been operating since 1929. The site is located on the west bank of the Elizabeth River in the industrialized waterfront of Portsmouth, Virginia. The site occupies 19.2 hectares of flat land, with elevations ranging from mean sea level to 3 meters above mean sea level (Figure 1). The eastern half of the site contains active wood-processing facilities and wood storage areas. The western half is used to store treated and untreated wood (Figure 2).

Surface runoff from the site drains into the Elizabeth River *via* three National Pollutant Discharge Elimination System (NPDES)-permitted stormwater outfalls plus the Elm Avenue storm sewer outfall (Figure 2). Runoff from the northeast portion of the site drains *via* the storm sewer outfall and Outfall 002 to a small inlet of the Elizabeth River between the site and the Jordan Bridge. Outfall 001 receives runoff flowing east across the southeast storage area. Outfall 003, located in the northwestern corner of the property, discharges runoff from the western portion of the site into an open ditch that leads to Paradise Creek and eventually discharges to the river (Environmental Strategies Corporation (ESC), 1988). The inlet from the Elizabeth River that runs along the northern boundary of the site also receives direct surface water runoff from the site.

Two groundwater-bearing zones have been identified beneath the Atlantic Wood site, the upper Columbia aquifer, ranging between 5.5 and 7 meters, and the lower Yorktown-Eastover aquifer. A semi-confining unit of clay is located beneath the Columbia aquifer. The Columbia aquifer is considered to be a water table aquifer recharged predominantly from precipitation (KER, 1990).

Within the eastern portion of the site, groundwater flows east towards the Elizabeth River. Groundwater flow in the western portion of the site is radial due to a groundwater mound. Average linear velocity calculated for flow beneath the eastern portion of the site is 27.5 meters per year (KER, 1990). Flow velocity information was not available for the western portion of the site.

## **AQUATIC ECOLOGICAL RISK ASSESSMENT**

### **POTENTIAL RECEPTORS**

Ecological receptors in three types of aquatic habitat have been identified as being at risk due to contaminant migration from the Atlantic Wood site. These are the estuarine communities in the Elizabeth River along the shoreline of the site and in the on-site inlet of the river, five very small and disturbed wetlands onsite, and a nearby freshwater stream (Paradise Creek). Only the first two types of habitats, which occur within the geographical scope of this assessment, will be addressed in this risk assessment. Organisms that are full-time residents of these habitats or that may utilize these areas during migratory periods are considered to be potential receptors of site-related contamination.

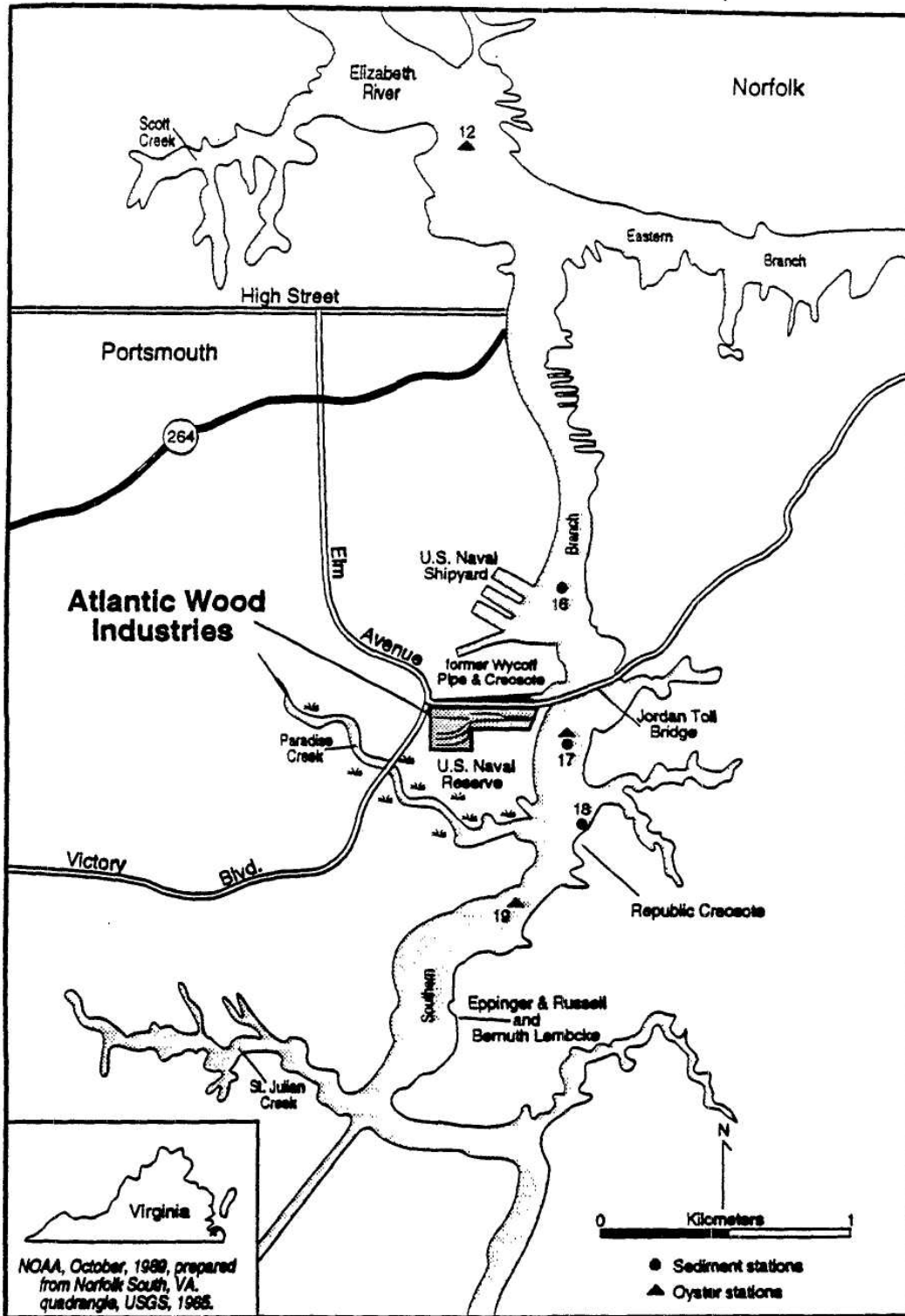


Figure 1. Atlantic Wood Industries Site in Portsmouth, Virginia, showing sediment sampling stations (16, 17, 18) and oyster transplant locations (12, 17, 19) in main river (Hargis *et al.*, 1984; Huggett *et al.*, 1984).

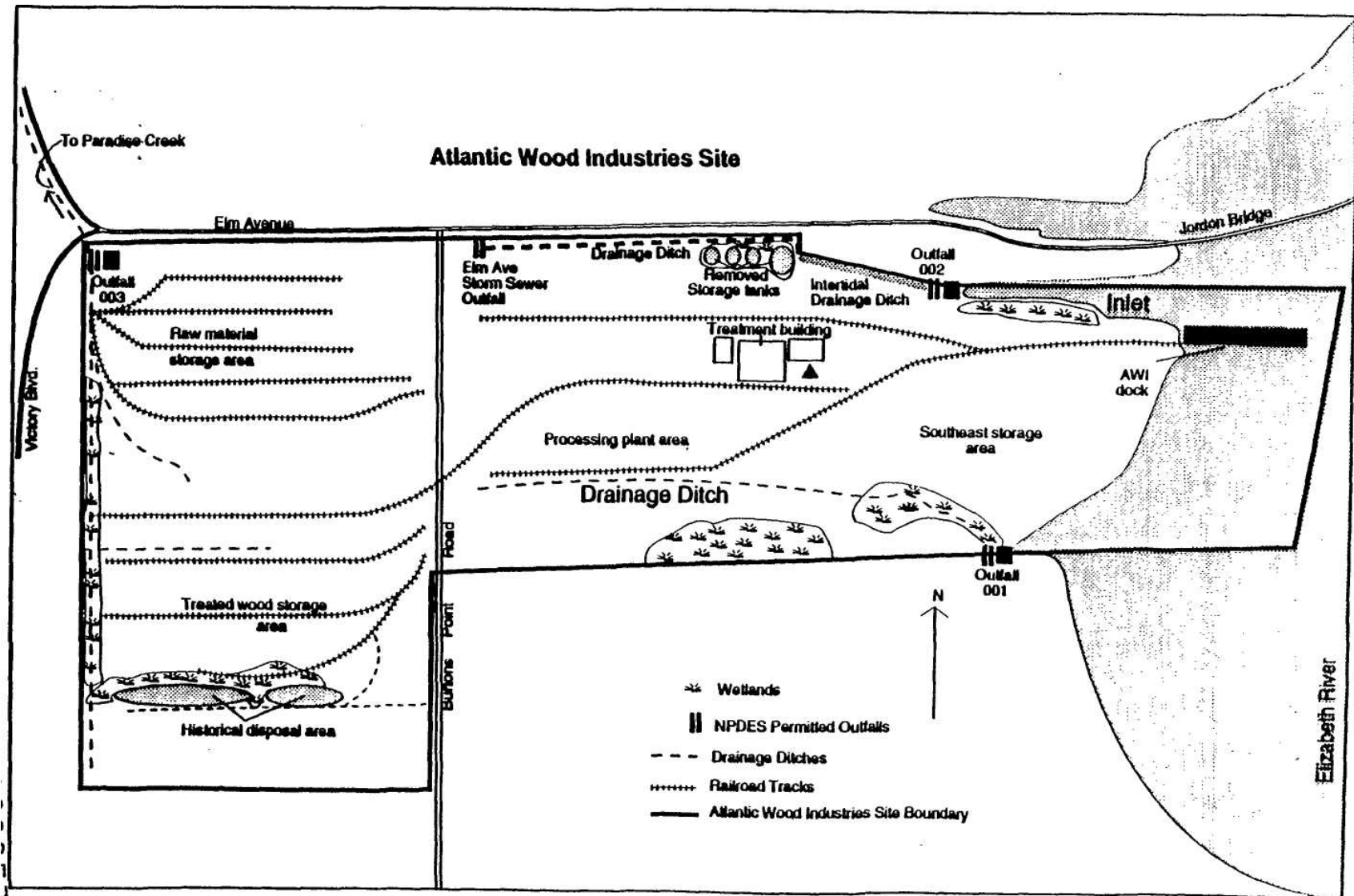


Figure 2. Geographic scope of Ecological Risk Assessment (ESC, 1988).



## Aquatic Habitats

The Elizabeth River is the dominant aquatic habitat impacted by the site. The river system has three main branches that empty into the southern end of Chesapeake Bay. The Atlantic Wood site is located on the west bank of the south branch of the river, approximately 17 kilometers (km) from the river mouth (Figure 1). Tidal influence in the south branch extends approximately 30 km upstream from the river mouth, with salinities in the area of Atlantic Wood ranging from 10 to 22 parts per thousand. Intertidal mudflats occur along the border of the site, especially at the mouth of the inlet and Outfall 001. These mudflats are small and essentially devoid of macrophytes. No biological survey has been conducted to determine the functional viability of the mudflats or river bottom bordering the site.

According to the City of Portsmouth Tidal Marsh Inventory (July 1989), there are no remaining wetlands along the western shoreline of the south branch of the Elizabeth River. The National Wetland Inventory also conducted a wetland study in this area and found no wetlands within the site boundaries. However, despite these earlier findings, a wetlands delineation study conducted by KER, Inc. (1990) identified five wetlands according to the Federal Manual for Identifying and Delineating Jurisdictional Wetlands. All the wetlands identified are in a disturbed condition and are of very low functional value based: on their small size, low vegetation diversity, scattered vegetation, disturbed soils, and very minimal wildlife usage.

There are basically two types of wetlands found on the site in five different locations (Figure 2). The first type is a reed grass community and the second a saltbush community (groundsel tree). Reedgrass (*Phragmites australis*) is an aggressive, less desirable species with little value to wildlife. It typically invades disturbed marshes and competes with species considered more desirable for their habitat value. Groundsel communities are considered of moderate value for the diversity and bird nesting area they add to the marsh ecosystem.

The five wetland areas can be described as:

- Location 1 - Small, fringing, tidal zone wetland, groundsel tree community, found along the stormwater drainage channel entering the inlet.
- Location 2 - Tidal zone, groundsel tree community, also small patches of saltmarsh cordgrass. Soil is mostly contaminated waste.
- Location 3 - Reedgrass community in small depression along the property border receiving runoff from off-site areas.
- Location 4 - Disturbed, mixed community in constructed depression formed from the removal of tanks from this area. Water flows continuously into this area from the plant's cooling tower, thus creating conditions sufficient to allow the growth of some wetland plants.
- Location 5 - Reedgrass community, partially fringing the Outfall 003 drainage ditch and covering the previous waste fill area on the southwest corner of the site property.

Six small tidal tributaries represent the extent of riparian habitat in off-site areas near the site, including those creeks below the Jordon Bridge (100 meters north of the site) and the Gilmerton Bridge (3.5 km to the south): Paradise, Scuffletown, Jones, Gilligan, St. Julian, and Milldam creeks. Riparian habitats near the site are estuarine emergent marshes and stream banks. The total area of emergent marsh for these six creeks is approximately 52 hectares, with Paradise Creek offering the largest aggregate area of approximately 21 hectares. These wetlands are narrow fringe marshes interspersed along each tributary, often abutting developed uplands. Vegetation is salt-tolerant grasses and sedges with low shrubs along the higher upland edge. Their small size limits the utility of these habitats for wildlife. According to local sources, these wetlands and stream banks are only considered valuable because they are scarce, not because they provide superior habitat (Schwab, personal communication, 1991). Overall value is considered to be further diminished by surface water and sediment contamination in the Elizabeth River

(Schwab, personal communication, 1991). Despite such conditions, these riparian habitats do support small, but stable populations of wildlife, most prominently muskrat (Halbrook, personal communication, 1991).

The site is located between two large riparian/estuarine wetland habitats of high value--Chesapeake Bay, 7 km to the north, and Great Dismal Swamp National Wildlife Refuge, 12 km to the south (U.S. Fish and Wildlife Service (USF&WS), 1980). The Elizabeth River connects these two large habitats, acting as an avian wildlife corridor. The site is also located under the Atlantic Flyway, the primary East Coast avian migratory route. Therefore, in addition to supporting small resident populations of wildlife, the limited habitats on site are also used on an intermittent, but at times moderate, basis by transient wildlife.

### Habitats of Special Concern

Based on the limited information regarding species occurrence near the site, no endangered or threatened species are present and no critical habitats are located near the site. The closest significant habitats are two heron rookeries--great blue heron and yellow-crowned night heron--5 km downstream near the Hampton Roads Army Terminal (USF&WS, 1980).

### Species

Because comprehensive wildlife surveys at or nearby the Atlantic Wood site have been limited, information on the occurrence of species onsite is sporadic. No data were found regarding the presence of aquatic vegetation in the Elizabeth River. The river would be expected to support a normal phytoplankton population, but macrophytes are probably limited to the shoreline wetlands. Data regarding the invertebrate species in the Elizabeth River were largely unavailable. The river would be expected to support a normal estuarine population of zooplankton and benthic infauna, but the identities of such species and their spatial and seasonal distribution have not been established.

Blue crab (*Callinectes sapidus*) and eastern oyster (*Crassostrea virginica*), species of commercial and recreational fishing interest, are known to be present in the Elizabeth River (Table 1). Blue crabs are harvested throughout the river, both recreationally and commercially, from approximately 14.5 km upstream of the site to the river mouth (Gillingham, personal communication, 1989; Travelstead, personal communication, 1989). Blue crabs are distributed differently by sex and life stage, with juveniles and adult males preferring less saline habitat.

Eastern oysters are normally present throughout lower portions of the river, although the extent of their presence near the site has not been established. In addition, the Virginia Division of Shellfish Sanitation enacted a prohibition on the taking of shellfish, other than blue crabs, in the Elizabeth River in April 1982, based on contamination by trace elements, including cadmium, arsenic, chromium, copper, lead, and zinc. This prohibition covers the river and its tributaries from Craney Island at the river mouth, and includes all areas approximately 20 km upstream of the site to Oak Grove, Virginia (Wright, personal communication, 1991).

The south branch of the Elizabeth River provides adult foraging habitat and juvenile nursery habitat for numerous anadromous, catadromous, and estuarine fish species. Species that inhabit the river near the site and their life stages are listed in Table 1. The species in Table 1 have been observed recent samples collected within 2 km of the Atlantic Wood site, or their range is known to include the Atlantic Wood section of the Elizabeth River's south branch. The category of adult "foraging" habitat includes use by resident species and migratory fish that occupy the area on a seasonal basis. Many of these species have been found immediately offshore of the Atlantic Wood site: hogchokers (*Trinectes maculatus*), weakfish (*Cynoscion regalis*), and oyster toadfish (*Opsanus tau*) are residents; while spot (*Leiostomus xanthurus*), and Atlantic croaker (*Micropogonias undulatus*) are present during the spring and summer (Huggett *et al.*, 1987; Moyle and Cech, 1982). Juveniles of these species as well as juvenile Atlantic menhaden (*Brevoortia tyrannus*), bay anchovy (*Anchoa mitchelli*), alewife (*Alosa pseudoharengus*), and blueback

herring (*Alosa aestivalis*) have been sampled in the river, either directly offshore of the site or within 2 km (Virginia Institute of Marine Science (VIMS), 1989).

**Table 1.** Habitat use and fisheries of the south branch of the Elizabeth River within 2 km of Atlantic Wood (Gillingham, personal communication, 1989; Huggett *et al.*, 1987; Austin, personal communication, 1989; VIMS, 1989).

Common Name	Species	Spawning	Habitat		Fisheries	
	Scientific Name		Nursery	Foraging	Sport	Comm.
<u>Anadromous</u>						
Blueback herring	<i>Alosa aestivalis</i>		♦	♦	♦	♦
Alewife	<i>Alosa pseudoharengus</i>		♦	♦	♦	♦
American Shad	<i>Alosa sapidissima</i>			♦	♦	♦
Striped bass	<i>Morone saxatilis</i>			♦	♦	♦
<u>Catadromous</u>						
American eel	<i>Anguilla rostrata</i>			♦	♦	♦
<u>Estuarine</u>						
Bay anchovy	<i>Anchoa mitchelli</i>		♦	♦		
Blue crab	<i>Callinectes sapidus</i>		♦	♦	♦	♦
Eastern oyster*	<i>Crassostrea virginica</i>	♦	♦	♦	♦	♦
Weakfish	<i>Cynoscion regalis</i>		♦	♦	♦	♦
Mummichog	<i>Fundulus heteroclitus</i>	♦	♦	♦		
Striped killifish	<i>Fundulus majalis</i>	♦	♦	♦		
Atlantic silverside	<i>Menidia menidia</i>	♦	♦	♦		
Hogchocker	<i>Trinectes maculatus</i>	♦	♦	♦	♦	
Oyster toadfish	<i>Opsanus tau</i>	♦	♦	♦		
<u>Marine</u>						
Atlantic menhaden	<i>Brevoortia tyrannus</i>		♦	♦	♦	♦
Spot	<i>Leiostomus xanthurus</i>		♦	♦	♦	♦
Atlantic croaker	<i>Micropogonias undulatus</i>		♦	♦	♦	♦
Spotted hake	<i>Urophycia regia</i>			♦		♦

\* A 1982 shellfish advisory still in effect prohibits the taking of shellfish, other than blue crab, in the Elizabeth River.

## EXPOSURE ASSESSMENT

To estimate the exposure that aquatic species have received or may receive from contaminants from the Atlantic Wood site requires an assessment that includes identifying each of the following:

- which contaminants that have been detected on site are of prime ecological concern?
- what are the sources of these contaminants?
- how are the contaminants being physically transported through the environment and chemically altered?
- what are the actual pathways and levels at which contaminants may migrate to habitats and receptors of concern?

Ideally, this assessment would produce two estimates for each of the primary contaminants for potential receptors within the habitats of concern: 1) an estimate of the maximum possible exposure level, and 2) an estimate of the most-likely exposure level. Finally, the assumptions and uncertainties associated with these estimates must be made explicit so that subsequent risk assessments reflect the appropriate level of probability.

## Contaminants of Concern

Contaminants of concern at Atlantic Wood were identified based on four criteria: prevalence, mobility, persistence, and toxicity. Contaminant data is from the RI (KER, 1990), which includes samples collected from soil, groundwater and sediment.

The contaminants of primary concern at the Atlantic Wood site are the polycyclic aromatic hydrocarbons (PAHs) associated with creosote, pentachlorophenol (PCP), polychlorinated dibenzo-p-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs), arsenic, copper, and zinc. Substantial data demonstrate that PAHs are persistent compounds that can accumulate to high levels in many invertebrate organisms and are toxic to most species at low concentrations. PCP and the associated chlorinated dioxins and furans also have been shown to be toxic to most biological species. Because creosote (PAHs), PCP, PCDDs, and the trace elements of concern are all known to be acutely toxic to aquatic and terrestrial species and because most of the toxicological information available describes acute toxicity, mortality will be the primary endpoint of concern for this risk assessment. Since PAHs are also known to induce a number of mutagenic effects, cancer, and histopathological changes in certain organisms, histopathological endpoints will be evaluated for the PAHs. Based on information gained from other regions regarding reproductive impairment due to PAH exposure, reproductive endpoints will also be considered.

Elevated levels of PAHs and PCP were found in soil, in on-site groundwater, and in sediments of the drainage ditches and inlet. Elevated levels of arsenic, copper and zinc were detected in on-site soils and in sediments of the drainage ditches (Table 2). Additionally, a number of PCDDs and PCDFs were detected in soil samples (Table 2) (KER, 1990). Not all samples (*e.g.*, inlet and river sediments) were analyzed for these additional analytes, and the detection limits were too high for some samples to evaluate their occurrence relative to toxic levels. Therefore, the distribution pattern and their risk to environmental receptors for PCDDs, PCDFs and trace elements cannot be discerned at this time. If information on additional contaminants of concern is generated, an update to this risk assessment could be conducted.

Creosote, a wood preservative used at the site, appears to be the primary source of PAHs at the Atlantic Wood site. Coal tar creosote is a distillate of bituminous coal, whose properties vary depending upon the source of the coal tar. Creosote is typically composed of 85 to 90 percent PAHs, as well as about 10 percent tar acids (phenols and creosols) and 5 percent tar bases (N-, S, and O-heterocyclics) (Table 3) (Chemical Information Systems (CIS), Inc., 1986; Mueller *et al.*, 1989). Because PAHs represent the majority of creosote constituents, and because it is anticipated that the scope of remediation will be driven by these particular constituents, tar acids and bases will not be considered further. Although a number of different PAHs are present in creosote, many of the analyses performed at the Atlantic Wood site measured only selected PAHs. For simplicity, total PAHs ( $\Sigma$ PAHs) will be used here to represent the sum of the individual PAHs that were measured and reported. Most likely, there are other PAHs present that were not reported, and these would not be reflected in the  $\Sigma$ PAH figure.

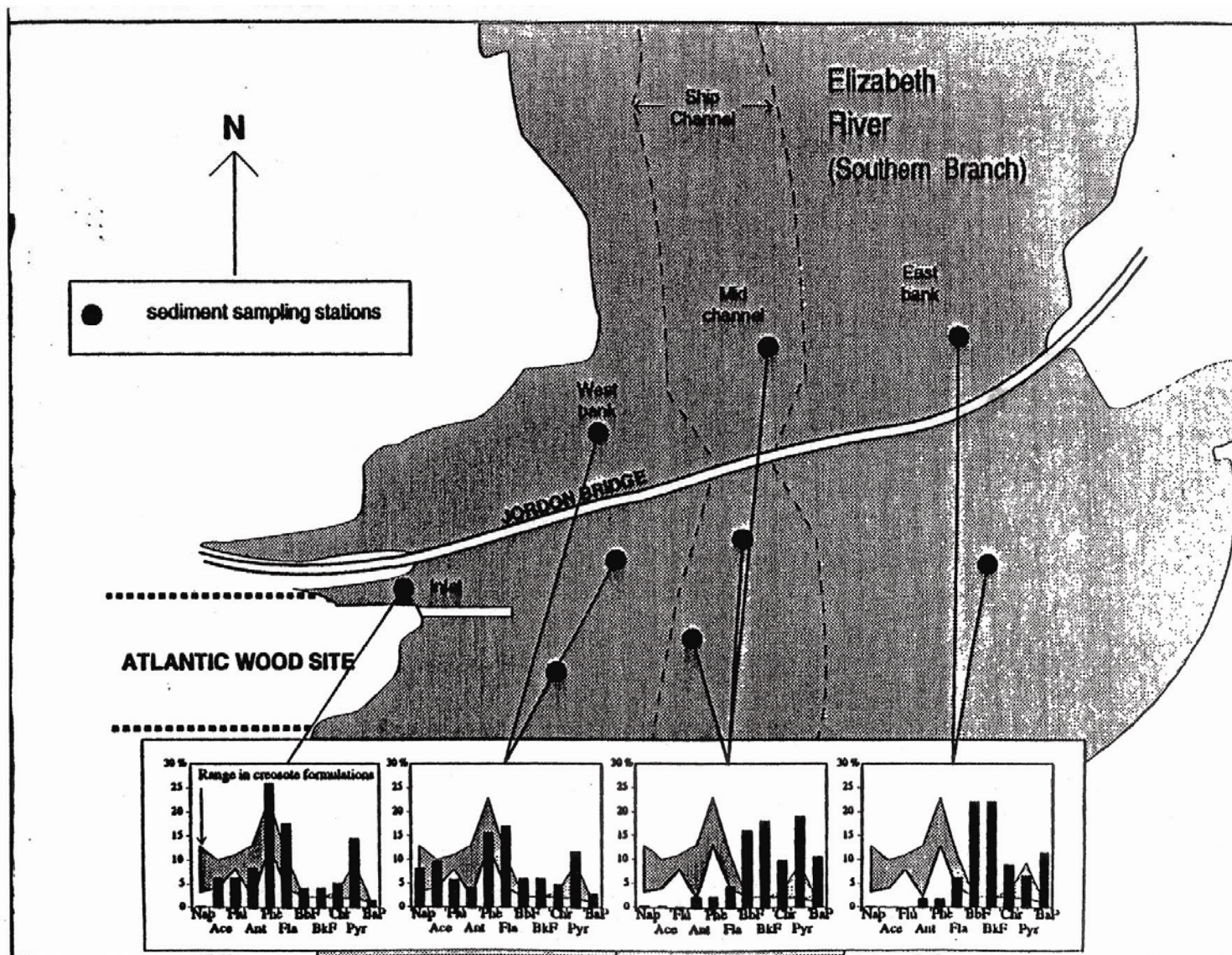
**Table 2.** Concentrations (mg/l or mg/kg) of the contaminants of concern and their frequency of detection in groundwater, surface soils, ditch sediments, inlet sediments and Elizabeth River sediments (KER, 1990).

Chemicals	Groundwater			Surface Soil			Ditch Sediments			Inlet Sediments			Elizabeth River		
	Mm	Max	Freq.	Min	Max	Freq.	Min	Max	Freq.	Min	Max	Freq.	Min	Max	Freq.
<u>PAHs</u>															
Acenaphthene	<0.01	17000	10/30	0.05	940	74/87	<0.33	19	6/12	25	2400	5/5	0.15	120	5/9
Acenaphthylene	<0.01	1000	S/30	0.05	42	77/87	0.47	6.3	12/12	5.7	87	5/5	0.12	1.2	7/9
Anthracene	<0.01	4000	8/30	0.20	2200	80/87	1.6	560	12/12	14	3200	5/5	0.1	52	9/9
Benzo(a)anthracene	<0.01	0.26	6/30	0.06	890	84/87	38	290	12/12	33	880	5/5	0.32	36	9/9
Benzo(a)pyrene	<0.01	1800	S/30	0.06	840	84/87	3.4	210	12/12	27	630	5/5	0.5	21	9/9
Benzo(b)fluoranthene	<0.01	2800	S/30	0.11	1600	84/87	5.2	240	12/12	28	1600	5/5	1	39	9/9
Benzo(g,h,i)perylene	<0.01	310	S/30	0.49	300	82/87	1.8	77	12/12	9	190	5/5	0.18	4.5	9/9
Benzo(k)fluoranthene	<0.01	2800	S/30	0.05	1600	85/87	62	310	12/12	24	1600	5/5	1	39	9/9
Chrysene	<0.01	32000	6/30	0.07	1100	84/87	4.9	320	12/12	44	2000	5/5	0.49	42	9/9
Dibenz(a,h)anthracene	<0.01	140	1/30	0.05	130	78/87	0.42	35	12/12	6.4	110	5/5	0.1	2.5	9/9
Fluoranthene	<0.01	18000	8/30	0.17	4800	84/87	7.3	540	12/12	150	6800	5/5	0.19	160	5/5
Fluorene	<0.01	13000	10/30	0.04	1100	73/87	0.18	37	12/12	7.9	2400	5/5	0.11	77	5/9
Indeno(1,2,3-c,d)pyrene	<0.01	3600	3/30	0.18	380	82/87	1.8	84	12/12	9	210	5/5	0.2	4.4	9/9
Naphthalene	<0.01	64000	11/30	0.10	1900	73/87	0.17	8.5	12/12	6.2	120	5/5	0.075	140	7/9
Phenanthrene	<0.01	36000	8/30	0.04	3300	84/87	1.3	320	12/12	36	10000	5/5	0.1	210	9/9
Pyrene	<0.01	12000	8/30	0.17	2100	83/86	7.9	480	12/12	86	5600	5/5	0.57	110	9/9
Total PAH	<0.01	2.6 X 10 <sup>5</sup>	16/30	2	13254	67/87	S3	3,116	12/12	511	38437	5/5	5	1059	9/9
PCP	0.004	860	10/30	0.11	970	49/85	0.42	12	10/11	<250	<250	0/5	<8.9	<8.9	0/9
<u>PCDDs/PCDFs</u> (ng/l or µg/kg)															
2,2,7,8-TCDD (TEF)	40.01	0.54	6/8	0.02	12.77	17/19	NA	NA	NA	NA	NA	NA	NA	NA	NA
<u>Trace Elements</u>															
Arsenic	0.002	0.676	23/28	4	495	32/32	31.2	374	11/11	NA	NA	NA	NA	NA	NA
Copper	0.003	1.99	23/28	S	9780	32/32	95.1	1350	11/11	NA	NA	NA	NA	NA	NA
Zinc	0.007	9.5	25/28	40	20400	77/77	291	1890	11/11	NA	NA	NA	NA	NA	NA
NA: Not analyzed															
TEF: 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCOO) toxicity equivalency factor.															

**Table 3.** Composition of creosote (CIS, 1986; Mueller *et al.*, 1989).

Chemical	Percent Composition
<u>PAHs</u>	85-90
Acenaphthene	9
Anthracene	2
Benzo[fluoranthenes	2
Biphenyl	0.8
Carbazole	2
Chrysene	3
Dibenzofuran	5
Dimethylnaphthalenes	2
Fluoranthane	10
Fluorene	10
Methylanthracenes	4
Methylfluorenes	3
1-Methylnaphthalene	0.9
2-Methylnaphthalene	1.2
Methylphenanthrenes	3
Naphthalene	3
Phenanthrene	21
Pyrene	8.5
<u>Tar Acids</u>	10
<u>Tar Bases</u>	5

The PAH composition of inlet sediments is similar to that found in creosote (Figure 3). To compare the PAH composition of inlet sediments to that of creosote, only those PAHs detected in inlet sediments that are also reported to be components of creosote (CIS, 1986; Mueller *et al.*, 1989) were evaluated. The concentrations of the 11 selected PAHs in each sediment sample were summed and the percentage composition for each PAH calculated to determine the relative contribution of each PAH compound to the summed total. This was done for each of the four sampling locations. The range in percentage composition of these same selected PAHs in different creosote formulations was also calculated. In Figure 3, the percentage composition of each of the PAHs at each sampling location is represented by a vertical bar. The range of the selected PAHs in creosote is represented by the shaded area. The relative levels of the lighter and more volatile PAHs (naphthalene, acenaphthene, fluorene, and anthracene) are generally below what would be expected in creosote, but this may be due to volatilization and metabolism of these more labile compounds. There appears to be an increase in the relative concentrations of a number of the heavier and more persistent PAHs above that expected in creosote, which is most likely due in part, to the loss of the lighter fraction. For instance, a dense nonaqueous-phase liquid (DNAPL) sample consisted of 24.6, 13.8, and 6.9 percent of naphthalene, phenanthrene, and fluoranthene, respectively. (This sample was collected from the monitoring well nearest the inlet which happened to have the thickest layer of DNAPL). If it is assumed that the DNAPL is due primarily to parent creosote used at the site, then weathering of the creosote in soils and sediments most likely would have resulted in the loss of naphthalene with a resultant increase in the relative concentrations of the less volatile components.



**Figure 3.** Average proportional PAH composition (% of  $\Sigma$ PAH) of sediment samples (vertical bars) from the Inlet and Elizabeth River compared to the range of composition in different creosote formulations.

The PAH composition of sediments collected in the Elizabeth River near the site changed as the distance from the site increased (Figure 3). Three sediment samples were collected in a track across the Elizabeth River offshore from the site--at the western bank, mid-channel, and the eastern bank. The relative concentrations from each of three sampling stations across the transect were averaged and these values are represented in Figure 3. Sediment samples collected on the western shore of the Elizabeth River closest to the site had a PAH composition similar to creosote, while samples collected from mid-channel and the eastern shore had PAH compositions showing less resemblance to creosote. The PAH composition of mid-channel and eastern shore sediment samples are disproportionately low in the lighter PAHs while the heavier PAHs, particularly benzo(b)- and benzo(k)fluoranthene and pyrene are disproportionately high. It is possible that the mid-channel and eastern shore samples could be due to creosote that has been in the environment longer and has thus weathered for a longer period than that in the inlet and adjacent nearshore areas. It is also possible that there may be a different source of these PAHs than the Atlantic Wood site. Potential sources include, or may have included, wood preserving sites depicted in Figure 1.

Another widely used wood preservative and sapstain control agent used at the site is PCP. As a wood preservative, it is carried in either a petroleum solvent or a water-soluble sodium salt. Commercial formulations of PCP contain a variety of chemicals present as impurities (Table 4). Impurities in commercial PCP include lower chlorinated phenols and the condensation products of two chlorinated phenol molecules, PCDDs and PCDFs (Firestone *et al.*, 1972; Jensen and Renberg, 1972). Concentrations of the dioxin and furan impurities have been reported to range from 2 to 2,500 µg/g in technical grade PCP (Johnson *et al.*, 1973). Although the most toxic dioxin congener (2,3,7,8-tetrachlorodibenzo-p-dioxin) has not been detected in technical grade PCP produced in North America, the presence of other highly toxic PCDDs and PCDFs has been documented (Eisler, 1989).

**Table 4.** Composition of commercial pentachlorophenol (Firestone *et al.*, 1972).

Chemical	Percent Composition
Pentachlorophenol	35-90
Tetrachlorophenol	4-8
Trichlorophenol	<0.1
Higher chlorophenols	24
Caustic insolubles	1

### Sources of Contaminants

The sources of primary contaminants at the Atlantic Wood site can be associated with current or past site activities and the raw materials used in the treatment process. Creosote and PCP, both wood preservatives used by the facility, are the major raw materials from which on-site contaminants originated. A special formulation of creosote and PCP (creo-penta) was used from the late 1950s to the early 1960s. PCP was also used at the site from 1972 to 1985, and its use was briefly resumed in spring 1991. Creosote had been used at the site since the 1950s. However, it should be noted that all wood treatment operations were suspended on August 6, 1991. Although timber treated with chromated copper arsenate was, and continues to be, stored on the site, this compound was never employed in wood treatment operations at Atlantic Wood (ESC, 1988).

Creosote was originally stored in four above-ground storage tanks located along the south side of Elm Avenue. Tank 1 held 3.3 million liters and the remaining three each held 1.7 million liters. Before their removal in 1985 through 1986, these tanks contained creosote and creosote contaminated water, which were leaking into the storm sewer system. Since 1975, creosote being used for treatment has been stored in smaller tanks located in the central part of the site (ESC, 1988).



Prior to 1972, the waste preservative left from the wood treatment process was stored at the southwest corner of the property in the “historic disposal area.” From 1972 to 1983 the “historic disposal area” was used to hold cuttings from the processed wood. The area was backfilled in 1983 (ESC, 1988). Additional information regarding past waste management practices is discussed in the RI (ESG, 1988; KER, 1992).

Based on the results of sampling conducted during the RI, areas surrounding the treatment buildings contain the most heavily PAH-contaminated soils (Figure 4). Since these areas are nearest the river, it may represent the greatest source of contaminated runoff to wetlands of the intertidal drainage ditch, inlet, and river. Sampling of wetland sediments in the drainage ditch and inlet (Figure 5), and the Elizabeth River (Figure 6) have documented extensive PAH contamination of these areas (KER, 1990) and confirm transport of contaminants to these habitats. When grain size is accounted for, samples taken from the five stations in the inlet indicate a decreasing gradient in PAH content. The head of the inlet is dominated by sand and gravel with 100 percent product saturation (*i.e.*, creosote) of the pore spaces (KER, 1990). At sampling sites nearer to the mouth of the inlet, the sediment texture changes to clayey sand and then sandy clay and there is only residual-to-heavy product saturation of pore spaces. However, these finer-grained sediments allow more surface area for adsorption of PAHs, and therefore greater apparent concentrations than gravelly samples.

In addition to PAHs, other contaminants were detected at the Atlantic Wood Site. Sampling across the site indicates widespread contamination of soils and sediments by PCP (Figures 4 and 5), although high detection limits associated with some samples preclude formulation of conclusions regarding relative source contributions to aquatic habitats. PCDDs and PCDFs were detected in soil (Figure 4) and can most likely be attributed to the use of PCP, which is known to contain these substances as impurities (Firestone *et al.*, 1972; Jensen and Renberg, 1972; ESC, 1988).

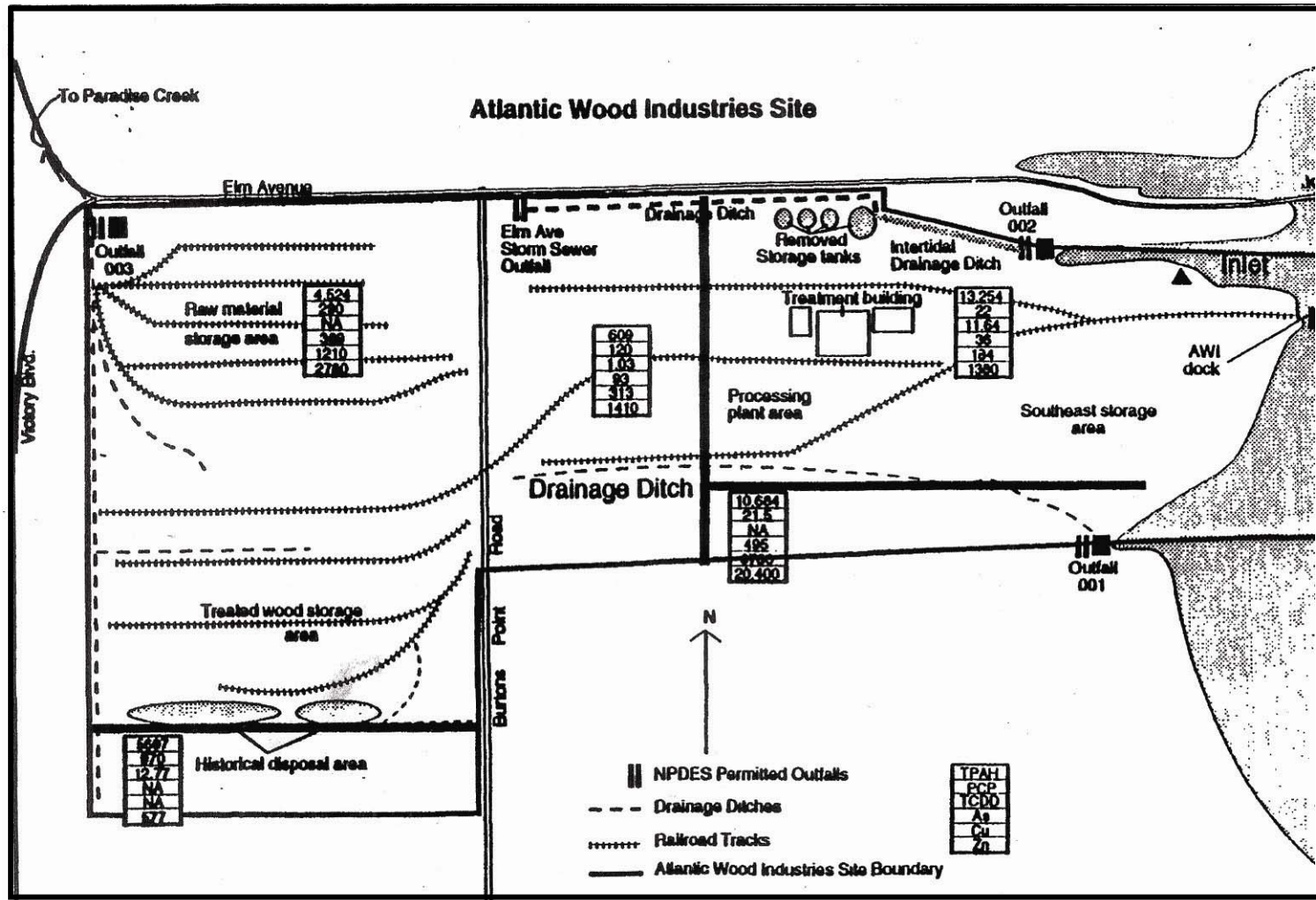
Elevated levels of arsenic, copper, and zinc were also detected in soils and sediments of drainage ditches (Figures 4 and 5). The arsenic and copper may have been derived from treated wood that was stored on the site. The occurrence of zinc has not been associated with the site. The occurrence of zinc has not been associated with current or past operational activities at the site (KER, 1992). The presence of zinc, and its possible association with off-site sources/activities, is currently being investigated.

Analysis of ground water samples collected during the RI indicated the presence of PAHs and PCP in the surficial aquifer throughout the site (Figure 7). Moreover, PCDDs/PCDFs, arsenic, copper, and zinc were also detected in the surficial aquifer at elevated levels. As previously stated, DNAPL was also observed in several ground water monitoring wells and may serve as significant source of contamination in ground water. The presence of dissolved contaminants and DNAPL indicates that habitats of concern may be impacted.

### Fate and Transport Analysis

#### **Polycyclic Aromatic Hydrocarbons**

The primary source of PAHs at the Atlantic Wood site is coal tar creosote used in the wood treatment process. The fate and transport of creosote are largely dependent upon the physical and chemical properties of its components, but its behavior in the environment is complicated due to the complex mixture of hundreds of organic compounds. Creosote has been observed to separate into three fractions when mixed with seawater--floating, sinking, and dissolved (Merrill and Wade, 1985). The sinking fraction was reported to have a composition similar to the original sample, suggesting that this fraction may be transported relatively intact to sediments. Some relatively insoluble components of creosote may become soluble because many of the compounds occurring in creosote probably act as co-solvents to enhance the aqueous solubility of the less soluble component (Merrill and Wade, 1985).



**Figure 4.** Maximum concentrations of total PAHs; PCP; 2,3,7,8-TCDD (TEFs); arsenic, copper, and zinc in surface soil. Concentrations are in mg/kg, except for TCDD, which is in µg/kg.

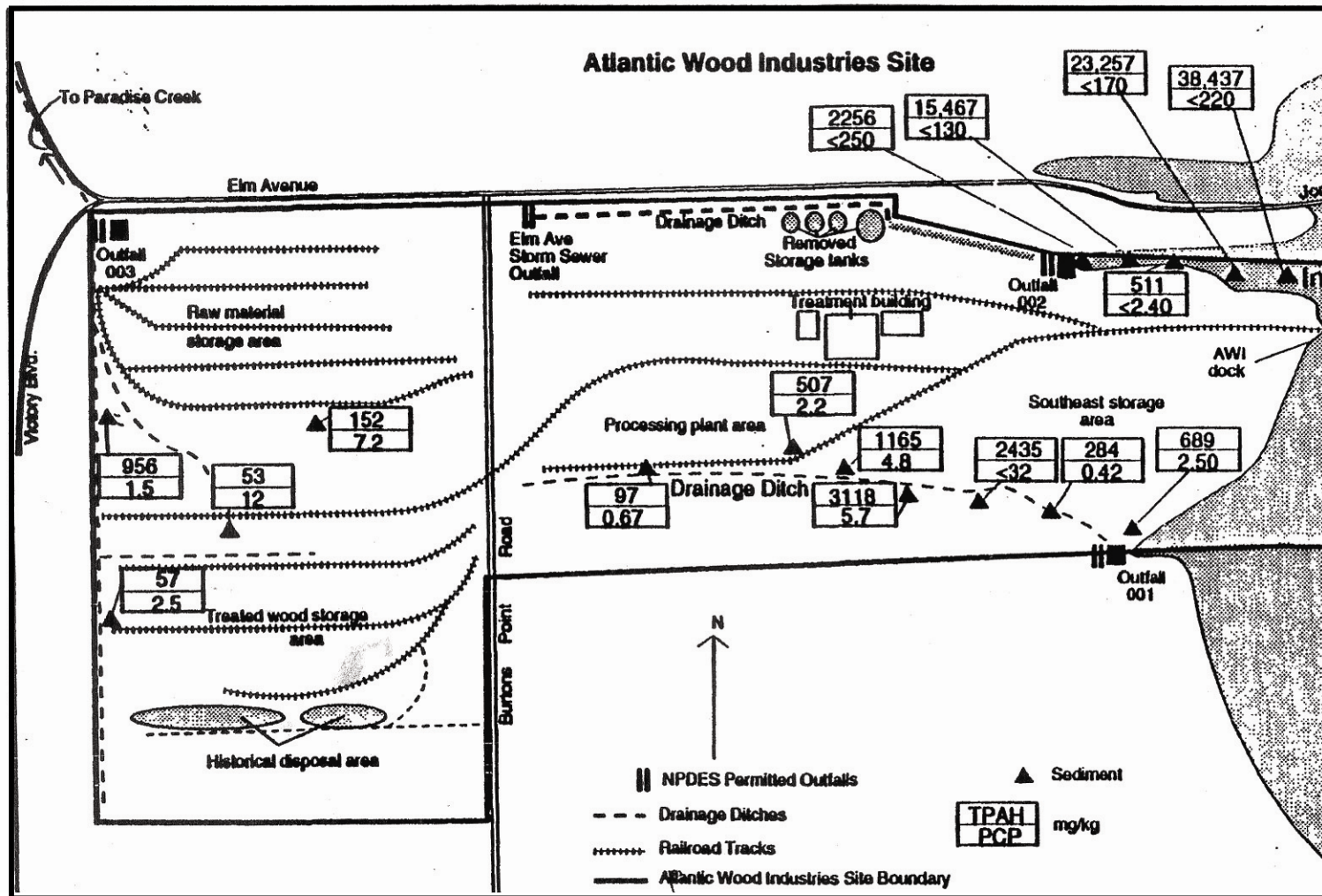
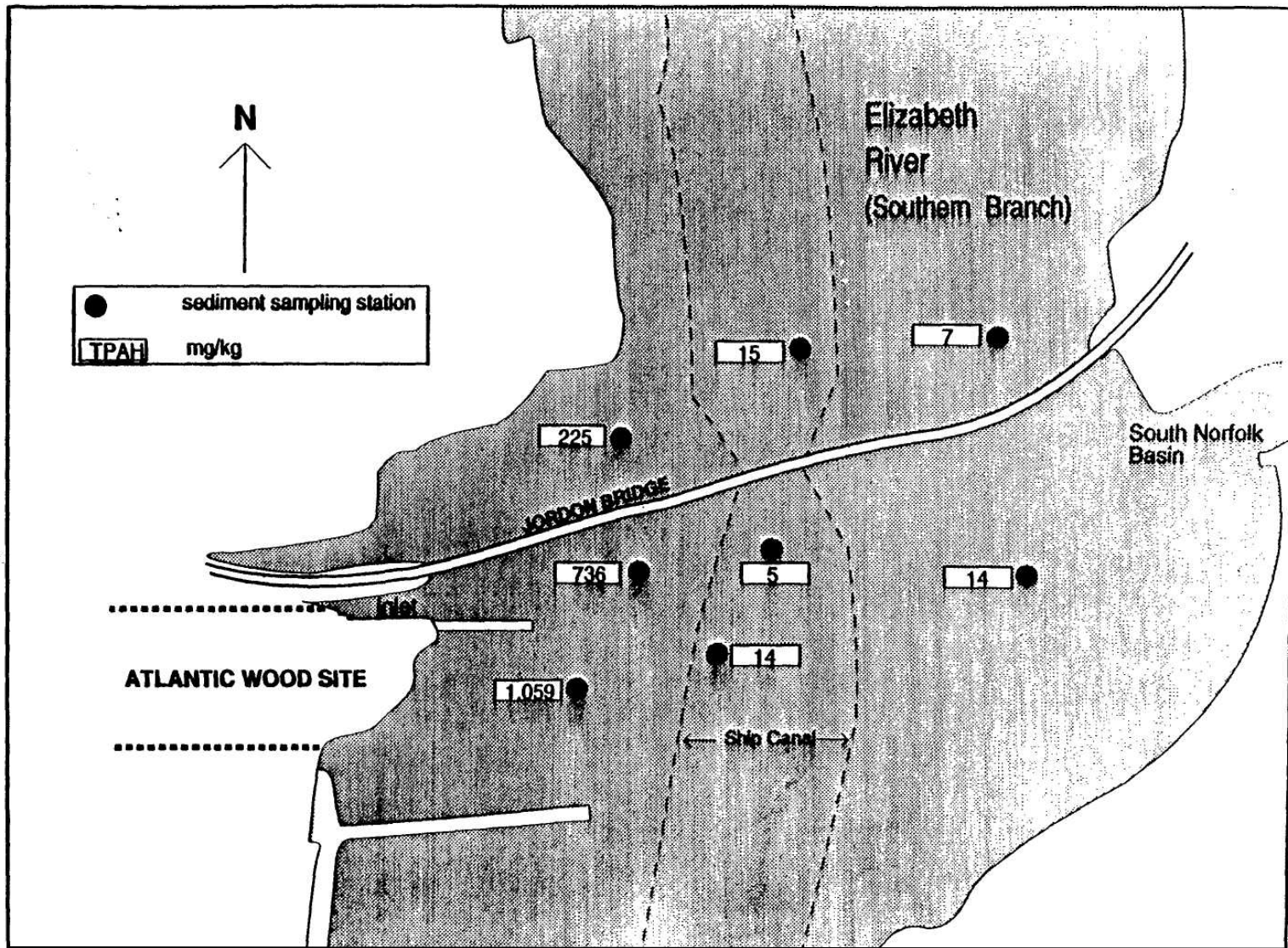


Figure 5. Sediment sampling results at Atlantic Wood Industries (ESC 1988; Keystone Environmental Resources, 1990).



**Figure 6.** Sediment sampling stations in the Elizabeth River adjacent to the Atlantic Wood site (Keystone Environmental Resources, 1990).

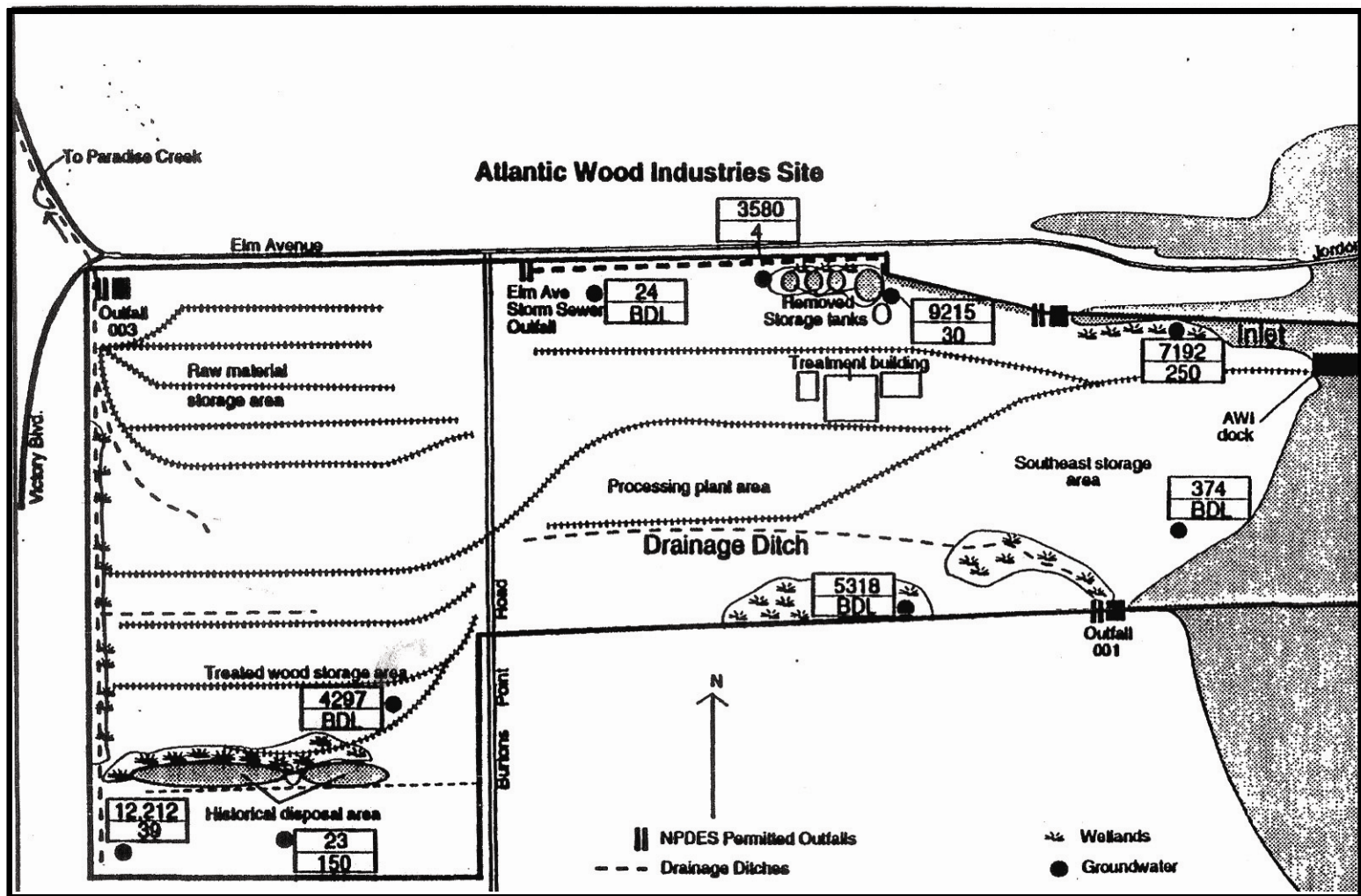


Figure 7. Groundwater sampling results at Atlantic Wood Industries (ESC, 1988; Keystone Environmental Resources, 1990).

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 most of the major component creosote. Thus, the PAH component of creosote discharged onto surface soils and to the surface waters sorb to the soils and sediments near the location of discharge. Once in the sediments, creosote is subjected to burial, resuspension, and degradation. In addition, creosote-contaminated soils and sediments can be transported by erosion and deposited in depositional areas away from the original source. As a result, PAHs can be found at considerable distances from their source, as fine particles containing PAHs and other creosote components are transported by prevailing water currents (PTI, 1988b). Water column exposures have been demonstrated to be significant as well (Mothershead and Hale, 1991).

The lower molecular weight PAHs (three aromatic rings or less) predominate in the PAH assemblage of creosote and, because of their greater aqueous solubility and degradability, will tend to be lost more rapidly in the weathering process. Thus, in weathered creosote the less labile, higher molecular weight PAHs will predominate. The more polar components of creosote (*i.e.*, the tar acids and bases) are more soluble than PAHs of comparable molecular weight and can be expected to be preferentially lost during weathering.

Although PAHs are readily absorbed by biota, they can be metabolized by the mixed-function oxidase (MFO) enzyme system and eliminated from most vertebrates, and, to a lesser extent, from invertebrate species. Thus, fish exposed to PAHs would not be expected to accumulate significant levels of PAHs in their tissues, while bivalves and some other invertebrate species could accumulate high levels (Varanasi *et al.*, 1985). Mothershead and Hale (1991) reported PAH bioaccumulation in newly molted blue crabs exposed in the vicinity of Atlantic Wood was three times higher than in intermolts. Since newly molted blue crab are regarded as a seafood delicacy, elevated body burdens represent a significant exposure pathway and may be a human health concern. Though accumulation in vertebrate organisms is usually short term and not considered an important fate reservoir, it too can be a significant exposure pathway. More importantly, in process of PAH metabolism, reactive intermediates are produced which are capable of initiating lipid peroxidation or binding to DNA. Reactions with DNA can lead to gene alterations and neoplasia (*e.g.*, cancer). Thus, in converting PAHs to more water soluble forms, non-carcinogenic or pro-carcinogenic compounds can be converted to carcinogens.

Microbial degradation and biotransformation are probably the ultimate fate processes for PAHs. The available data suggest PAHs with high molecular weights are degraded and metabolized by microbes and multicellular organisms more slowly than lower-weight compounds. Biodegradation probably occurs more slowly in aquatic systems than in soil (Clement Associates, 1985).

### **Pentachlorophenol**

PCP, though more soluble than PAHs, does tend to sorb to soils and sediments. In the aquatic environment, PCP may be present in a dissolved form, sorbed to suspended matter or bottom sediments, or sorbed by aquatic organisms. PCP is a neutral organic compound in aqueous solutions of pH less than 5, but ionizes as the pH rises. The degree of dissociation will determine the solubility and degree of adsorption to suspended matter and sediments in the aquatic system. PCP is generally considered to be moderately persistent in the aquatic environment (EPA, 1980).

Uptake of PCP by organisms typically is fairly quick, but PCP can also be metabolized and excreted quickly as well. In steady-state conditions (*i.e.*, long-term exposures), depuration rates are usually lower than uptake resulting in a net accumulation. A wide range of bioaccumulation values for PCP in fish and bivalves have been measured.

### **Polychlorinated Dibenzo-p-Dioxins and Dibenzofurans**

The PCDDs and PCDFs have very similar structures and are composed of two-ring structures linked by oxygen. There are 135 possible isomers of PCDDs and PCDFs each, depending on chlorine substitutions on the rings. Because of their high octanol-water partition coefficients, PCDDs and PCDFs tend to be

highly sorbed to particulates, sediments, and biota. Their low vapor pressures mean they are unlikely to volatilize in the atmosphere. PCDDs and PCDFs do not readily undergo photodegradation unless solvents are present that will act as hydrogen donors during reductive dechlorination. Due to their tendency to be sorbed to particulate material and their environmental persistence, PCDDs and PCDFs can be transported through wind erosion or through surface water runoff. In aquatic environments, these substances can be transported to depositional areas by suspended particulate matter.

Some microbial degradation of PCDDs has been reported, but it appears to be limited and highly dependent on the isomers present. The half-life in soil has been found to range from 130 days to several years (Eisler, 1986). Uptake by biota is rapid, and bioaccumulation from PCDD-contaminated freshwater and marine sediments has been well documented (Kuehl *et al.*, 1986; Kuehl *et al.*, 1987; EPA, 1990; Rubenstein *et al.*, 1990). The ability to metabolize PCDDs and PCDFs with low chlorine substitution via MFO activity has been demonstrated in some species of fish and birds but not in bivalves (Konasewich *et al.*, 1982). Sedimentation and bioaccumulation seem to be the most significant environmental fate for these compounds.

### **Trace Elements**

Arsenic has four different oxidation states, and chemical speciation is important in determining arsenic's distribution and mobility. The  $+3$  and  $+5$  valence states, as well as organic complexation, are the most important forms of arsenic in the environment. Arsenic is generally quite mobile and sorption to sediment can be an important fate process. In the aquatic environment volatilization is important when biological activity or highly reducing conditions produce arsine or methylarsenics (Clement Associates, 1985).

Copper has two oxidation states,  $+1$  (cuprous) and  $+2$  (cupric). Cuprous copper is unstable in aerated water over the pH range of most natural waters (6 to 8 standard units) and oxidizes to the cupric state. Several processes determine the fate of copper in the aquatic environment: formation of complexes, especially with humic substances; sorption to hydrous metal oxides, clays, and organic materials; and bioaccumulation. In waters with soluble organic material, copper can form complexes with organic ligands, thus favoring prolonged dispersion of copper in solution. The presence of organic acids can also lead to the mobilization of copper from the sediments to solution. Sorption processes are quite efficient in scavenging dissolved copper and in controlling its mobility in natural unpolluted waters. The levels of copper able to remain in solution are directly dependent on water chemistry. Generally, ionic copper is more soluble in low pH waters and less soluble in high pH waters (Clement Associates, 1985).

As an essential nutrient, copper is accumulated by plants and animals, although apparently it is not generally biomagnified. Because copper is strongly bioaccumulated and because biogenic ligands play an important role in completing copper, biological activity is a major factor in determining the distribution and occurrence of copper in the ecosystem (Clement Associates, 1985).

Because many copper compounds and complexes are readily soluble, copper is among the more mobile of trace elements. The major process that limits the environmental mobility of copper is adsorption to organic matter and clays (Clement Associates, 1985).

Zinc can occur in both suspended particulates and dissolved forms. Dissolved zinc may occur as the free (hydrated) zinc ion or as dissolved complexes and compounds with varying degrees of stability and toxicity. The predominant fate of zinc in aerobic aquatic systems is sorption of the divalent cation by hydrous iron and manganese oxides, clay minerals, and organic material. Concentrations of zinc in suspended and bed sediments always exceed concentrations in ambient water. Zinc tends to be more readily sorbed at higher pH than lower pH, and tends to be desorbed from sediments as salinity increases (Clement Associates, 1985).

Atmospheric transport of zinc is also possible. However, except near major sources such as smelters, zinc concentrations in air are relatively low and fairly constant (Clement Associates 1985).

Because it is an essential nutrient, zinc is strongly bioaccumulated even in the absence of abnormally high ambient concentrations. Zinc does not appear to biomagnify through the food web however. Although zinc is actively bioaccumulated in aquatic systems, biota appear to represent a relatively minor sink compared to the sediments (Clement Associates, 1985).

### Exposure Pathways

Environmental sampling at the site and in the Elizabeth River confirms significant pathways and migration of PAHs and PCP to sediments in the wetlands of the intertidal drainage ditch, inlet, and the nearshore areas along the river bank (Figures 4 and 5). Besides confirming pathways, sediment residue data indicate an extreme level of contamination within these habitats. Sediments are thus a primary source of contamination to aquatic resources both directly and indirectly through desorption to overlying water column. Aquatic species can be exposed to contamination from the Atlantic Wood site through contact with contaminated sediments during residence or temporary visits to the inlet and nearshore areas of the site. Species residing in or on the sediments, or feeding on benthic or infaunal organisms, are expected to receive the greatest exposure contamination from the site. In addition to ingestion or contact with contaminated sediment by demersal species, contaminated sediments represent a significant source and pathway to pelagic aquatic species using wetlands of the intertidal drainage ditch, the other wetlands on site, or the river inlet. These organisms may be exposed to aqueous contaminants that have leached from contaminated sediment, or come from outfall effluents and surface runoff.

### Estimated Exposure Levels

Estimates of exposures are based on existing knowledge of the levels of contamination across the site derived from the RI sampling. The contaminants of concern, their concentrations, and frequency of detection are summarized by media type in Table 2. Figures 3 through 6 show some of the groundwater, surface soil, and sediment sampling locations with the highest concentrations of the contaminants of concern.

Two estimates for sediment exposure levels of PAHs will be used for assessing risk to aquatic species. The maximum value detected in sediments of the inlet, on-site wetlands, or Elizabeth River adjacent to Atlantic Wood, will be used as the maximum sediment exposure level. This value of 38,437 mg/kg  $\Sigma$ PAH occurs near the mouth of the inlet and will be used to indicate a “worst-case scenario.” Because organisms would not be continuously exposed to this maximum possible level, it is recognized that this maximum is not likely to be the long-term exposure concentration for resident organisms. Therefore, the median of these samples, 1,008 mg/kg, will be used as a “more-likely” sediment exposure level. This median was derived from 18 values that include the samples shown in Figure 5, the sample nearest Atlantic Wood shoreline in Figure 6 (1,059 mg/kg), and a surficial sample of Huggett (1985) collected adjacent to the Atlantic Wood site (1,100 mg/kg). These data points, though from different sources, were obtained by similar analytical techniques and all represent observations of contaminant levels in the nearshore sediments of the Elizabeth River within the geographic scope of this assessment.

Although PCP was reported in soil samples collected near the Elizabeth River at concentrations as high as 970 mg/kg and in wetland sediments of an on-site drainage ditch at a concentration of 12 mg/kg, no PCP was reported in sediments collected from the inlet or the Elizabeth River adjacent to the site (KER, 1990). However, it should be noted that the detection limits for PCP in inlet sediments and sediments of the Elizabeth River (250 and 8.9 mg/kg, respectively) were relatively high. Given such elevated detection limits, it is quite possible that PCP could be present at significant concentrations in the sediments of these areas. In the absence of riverine sediment values, concentrations in soil samples will be used as surrogates. The maximum level of PCP in soils near the inlet, 67 mg/kg, will be used as the “high” exposure level to determine risk potential. The median for all soil samples, 15 mg/kg, will be used as the “more-likely” exposure level.

Since metals were not measured in all samples, estimates of sediment exposure levels for metals will be



based on ten samples from drainage ditches. The median and maximum, respectively, are 135 and 364 mg/kg for arsenic; 376 and 1,350 mg/kg for copper; and 729 and 1,890 mg/kg for zinc.

### Uncertainty Analysis

In performing the aquatic exposure assessment for the Atlantic Wood site, uncertainties arose from two main sources. First, those uncertainties associated with the distribution, transport, and fate of compounds in the environment. Second, those uncertainties associated in the estimation of chemical intakes resulting from contact by a receptor with a particular medium (KER, 1990).

As discussed in the RI for the Atlantic Wood site (KER, 1990), samples were not collected randomly throughout the site, but were instead focused on areas suspected to have elevated levels of site constituents. Therefore, the resulting soil and sediment data were biased toward elevated concentrations. Thus, exposures estimated from these data would also be biased because potentially exposed species would most likely not spend the majority of their time in just those regions with the highest soil or sediment concentrations.

To estimate an intake, certain assumptions must be made about exposure events, exposure duration, and the corresponding assimilation of constituents by the receptor. Because very little is known about which species use the site, in what capacity, and to what extent, there is a great degree of uncertainty associated with the estimated doses. In the extreme dose scenario, it is highly unlikely that most species utilizing the site would feed exclusively on the site and only in those areas where they would be exposed to the highest contaminant levels.

## **TOXICITY ASSESSMENT**

This section summarizes what is known about the sediment toxicity of the contaminants of concern at the Atlantic Wood site to aquatic species. Information on toxicity has been derived from literature reports of bioassays with the chemicals of concern, from regional ambient toxicity surveys, and by evaluation of toxicity-based criteria and standards. Although this assessment of toxicity is limited to the contaminants of concern *through* sediment exposure, it is recognized that the toxicity of additional contaminants present at the site (*e.g.*, tar acids and bases) may also impart significant adverse effects.

### Reported Effects of Contaminants of Concern

#### **Polycyclic Aromatic Hydrocarbons**

PAHs vary substantially in their toxicity to, and bioaccumulation by, aquatic species. PAHs can have adverse impacts on all biological levels of measure--from enzymatic and immunological changes, through tissue damage, to mortality and community alteration. The potential impact of PAHs will be discussed for all of these levels of biological organization, with particular emphasis on impacts on species reported from within the site's region.

#### *Bioaccumulation*

In most fish, PAHs are rapidly metabolized and excreted so that levels in edible tissue are generally low (Varanasi *et al.*, 1989). Invertebrates, especially mollusks, do not metabolize PAHs as efficiently and may accumulate high concentrations of PAHs in tissue or may retain detectable levels for a period of days to weeks (Mix, 1979; Eisler, 1987). Presence of PAHs in such species is a clear indication of bioavailability and recent exposure.

Oysters transplanted from relatively "clean" areas to locations throughout the Elizabeth River were used to determine the bioavailability of PAHs. Huggett and co-workers found that the uptake rate of PAHs was highest at the station nearest the Atlantic Wood facility (Huggett *et al.*, 1987) where sediment  $\Sigma$ PAHs were approximately 30 mg/kg. Tissue residues of  $\Sigma$ PAH were also greatest at the sites off Atlantic Wood

and just downstream, and reached levels twice as high as any other stations. Except for a station near a historical spill of pure creosote, sediment concentrations of PAHs were the highest in sediments off Atlantic Wood or just downstream of the site. Elevated levels of PAHs in the tissues of oysters have also been associated with reduced condition index<sup>1</sup>.

PAHs are metabolized and transformed by a family of MFO enzymes that includes the enzymes cytochrome P-450, P-450E, and ethoxyresorufin O-deethylase (EROD). These proteins are sensitive to environmental levels of xenobiotics and have been established as reliable indicators of exposure to xenobiotics, including PAHs, in feral fish populations. One study of spot collected from the Elizabeth River found levels of total cytochrome P-450, P-450E, and EROD elevated in intestine and liver microsomes (Van Veld *et al.*, 1990). Fish were collected over a PAH concentration gradient in the sediments that ranged from 0.009 to 96 mg/kg dry weight (see Figure 8 for sampling locations). Intestinal P-450E was near the lower limits of detection in fish collected at the relatively clean sites but was elevated 80- to 100-fold in fish collected from contaminated sites. Intestinal EROD activity exhibited a similar trend. Liver P-450E and associated EROD activity was detectable in all samples and was induced approximately 8-fold at the most heavily contaminated sites (Table 5).

### *Immune*

Other studies report additional sublethal and subcellular effects due to PAH exposure. Hogchoker from the Elizabeth River, where sediments contained approximately 100 rag/kg  $\Sigma$ PAH, were found to have reduced macrophage activities (Weeks and Warinner, 1984). Further studies indicated that the macrophages had diminished chemotactic capability (the ability to migrate toward a stimulus). This depression on the cellular immune system could result in increased susceptibility to disease (Weeks and Warinner, 1986; Weeks *et al.*, 1986). Studies of oyster hemocytes, which are believed to play an important role in defense against pathogens, have also shown depressions on the cellular immune systems.

Another study of PAH effects on immune systems may add to an understanding of tumor development in fish due to PAH exposure. Faisal *et al.* (in press) reported significant alterations of the *in vitro* tumorigenic activity of kidney and spleen leukocytes that defend against cancer. Mummichog (*Fundulus heteroclitus*) were collected from two sites heavily contaminated with PAHs in the Elizabeth River, plus a reference site. One class of cells that should spontaneously recognize their targets was unable to do so in fish collected from areas of sediment containing 2,200 and 61 mg/kg  $\Sigma$ PAH. A second class of defense cells that require prior exposure to the tumor cell antigen was found to be active, indicating an ongoing oncogenic process in these fish. Fish from the reference site with 3 mg/kg total sediment PAHs, displayed neither of the oncogenic indications observed in the Elizabeth River.

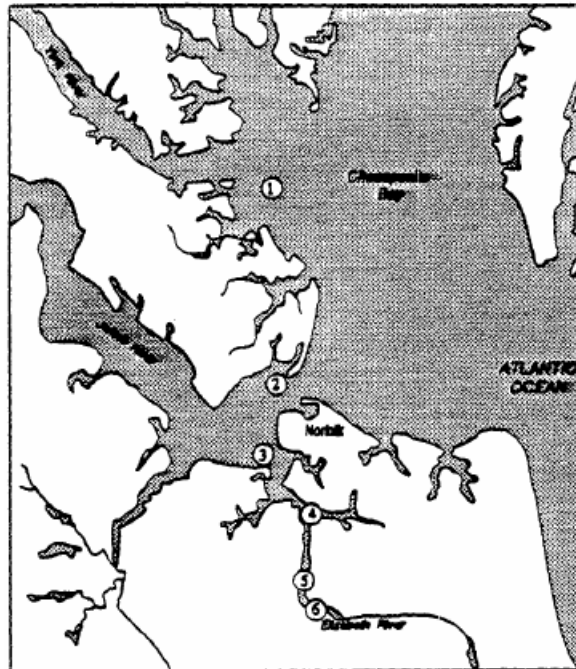
---

<sup>1</sup> Reduced condition index is a measure of tissue health as indicated by lipid content.

**Table 5.** Total sediment PAH levels (mg/kg) and MFO activity in spot intestine and liver in fish collected from the lower Chesapeake Bay and Elizabeth River (Van Veld *et al.*, 1990). Refer to Figure 8 for station locations.

Parameter	Station					
	1	2	3	4	5 <sup>a</sup>	6
Total PAH <sup>1</sup>	0.009	0.055	3.1	16.0	96.0	43.0
<u>EROD</u> <sup>2</sup>						
Gut	47±20*	105±58*	915±320**	1550±1068**	985±319* **	1053±594**
Liver	230±100	267±52	652±207**	981±678**	1894±813**	1291±682**
<u>P450E</u> <sup>3</sup>						
Gut	<0.001*	0.015±0.001*	0.079 ±0.004*	0.0109	0.131 ±0.880*	0.088 ±0.041*
Liver	0.012±0.003	0.014±0.003	0.042	0.042 ±0.004**	0.280	0.200
<u>Cyt. P-450</u> <sup>4</sup>						
Gut	0.43±0.05*	0.47±0.15*	0.67	0.76±0.22* **	0.57±0.18*	0.53±0.15*
Liver	0.78±0.15	1.01±0.18	1.00±0.28	1.14±0.31**	1.45±0.25**	1.14±0.09**

a: Adjacent to the Atlantic Wood site  
1: Individual PAHs were not identified  
2: Values are pmoles resorufin produced per mg of microsomal protein (mean ± SD; N=7)  
3: Values are nmoles of P-450E equivalents per mg of microsomal protein (mean ± SD; N=3)  
4: Values are nmoles of cytochrome P-450 per mg of microsomal protein (mean ± SD; N=7)  
\*: Significantly different from corresponding liver value P<0.05  
\*\*: Significantly different from value determined for reference site (station 1) P<0.05



**Figure 8.** Sampling sites for total sediment PAH levels (rag/kg) and MFO activity in spot collected from the lower Chesapeake Bay and Elizabeth River (Van Veld *et al.*, 1990).

Tumors and other tissue abnormalities have been observed in resident Elizabeth River fish. Abnormalities observed included fin erosion in hogchoker (*Trinectes maculatus*) and toadfish, plus cataracts in spot, weakfish, and croaker (Table 6; Huggett *et al.*, 1987). Prevalences of these conditions increased at stations with sediment PAHs greater than approximately 16 mg/kg. High prevalences of idiopathic hepatic lesions have also been found in mummichog collected in the Elizabeth River adjacent to the Atlantic Wood site. Grossly visible hepatic lesions occurred in 93 percent of the fish from this location where sediment contained 2,200 mg/kg ΣPAH. Hepatic lesions were not detected in fish from two less contaminated sites where total sediment PAHs were 3 and 61 mg/kg (Table 7; Vogelbein *et al.*, 1990).

The association between PAHs and liver lesions has been made in other regions of the country as well. For example, studies of English sole (*Parophrys vetulus*) from a creosote-contaminated harbor in Puget Sound, Washington indicate statistically significant correlations between the sediment concentrations of PAHs and the prevalence of hepatic neoplasms and other liver lesions. Similar correlations are also shown between the concentrations of PAH metabolites in the bile (Malins *et al.*, 1987). In addition, high levels of PAHs in stomach contents suggest that the consumption of contaminated benthic invertebrates is an important exposure pathway (Malins *et al.*, 1985).

**Table 6.** Total sediment PAH concentrations (mg/kg dry weight), biomass, number, and percentage of fish showing gross abnormalities at various sites along the Elizabeth River (Huggett *et al.*, 1987).

Parameter	River Kilometers from Atlantic Wood Site						
	Downstream					Adjacent	Up-stream
	10.5	8.5	6.5	4.5	2	0	-2
Total PAH <sup>1</sup>	1.10	5.00	5.10	11.50	15.82	24.4	154.4
Total number of fish	1,335	1,200	1,125	705	380	320	245
Biomass (kg)	34	26	21	13	9	7	4
<u>Fin Erosion (% incidence)</u>							
Hogchoker	0.7	0	0	0.4	1.4	5.5	4.3
Toadfish	0	0	11.0	5	0	11.5	30.1
<u>Cataracts (% incidence)</u>							
Spot	0	0	0.1	0	3.0	0.8	9.6
Weakfish	0.2	0	0	0.8	1.0	1.8	3.5
Croaker	3.3	1.4	1.5	2.2	4.5	7.9	15.8

<sup>1</sup> Total PAH is the sum of the PAHs listed by Huggett *et al.* which were common to those measured at the Atlantic Wood site and includes phenanthrene, fluoranthene, pyrene, benzo(a)pyrene, chrysene, the benzo(a)- and benzo(b)fluoranthene, benz(a)anthracene, indeno(c,d-1,2,3)pyrene, and benzo(g,h,i)perylene.

**Table 7.** Total sediment PAH concentrations and lesion prevalences for mummichog from three sampling locations in Chesapeake Bay (Vogelbein *et al.*, 1990).

Lesion Types	Percentage of Fish with Lesions		
	Ware River (N=15)	Scuffletown Creek (N=30)*	Elizabeth River (N=60)**
Total sediment PAHs (mg/kg dry wt) <sup>1</sup>	3	61	2200
Foci of cellular alteration	0	0	73.3
Cholangiolar proliferation	0	0	11.7
Hepatocellular adenoma	0	0	1.77
Early hepatocellular carcinoma	0	0	13.3
Advanced hepatocellular carcinoma	0	0	20.0
Total hepatocellular neoplasms	0	0	35.0
* : Tributary to the Elizabeth River directly across from the Atlantic Wood site			
** : Adjacent to the Atlantic Wood site			
<sup>1</sup> Total PAH as measured by Vogelbein <i>et al.</i> Individual PAHs were not identified.			

Studies from other regions have also documented the potential for PAH exposure to disrupt the reproductive cycle of fish. Another study of English sole from Eagle Harbor, Washington provided evidence of reduced reproductive success in association with high levels of sediment PAHs (Johnson *et al.*, 1988). Females from highly contaminated sites were found to be less likely to undergo gonadal maturation and to have lower mean levels of plasma estradiol (a steroid sex hormone) than those from cleaner sites. Both these conditions were highly correlated with aryl hydrocarbon hydroxylase (AHH) activity, and were also in concordance with general levels of PAH metabolites in the bile. Similar findings for starry flounder (*Platichthys stellatus*) from San Francisco Bay, California have been reported. Females collected from contaminated sites exhibited higher AHH activity, lower levels of plasma steroids, lower levels of fertilization success, and decreased normal embryologic developmental success (Long and Buchman, 1989).

#### *Mortality*

At greater concentrations, PAHs are also capable of causing acute mortality. One laboratory study in which the freshwater amphipod, *Diporeia* sp., was exposed to sediments dosed with a PAH mixture reported mortality at ΣPAH concentrations in the range of 100 mg/kg (dry weight) after 26 days of exposure (Landrum *et al.*, 1991). The rate of PAH accumulation was also examined and found to be dependent upon the sediment PAH concentration. At high exposures, uptake was greater than that predicted through measured partitioning between interstitial water and sediment.

Alden and Butt (1987) conducted sediment toxicity bioassays on sediments from 16 stations in the Elizabeth River using the grass shrimp *Palaemonetes pugio*. Few biological effects were observed in sediments with PAH concentrations in the range of 0.081 to 11 mg/kg. However, sediments from the south branch of the Elizabeth River near the Atlantic Wood site with PAH concentrations in the range of 5.8 to 65.5 mg/kg were reported to produce rapidly lethal and sublethal effects.

Bioassays exposing juvenile spot to creosote-contaminated sediments from the south branch of the Elizabeth River (near Republic Creosote Co.) demonstrate that contact with sediment and/or the water associated with these contaminated sediments may result in acute death or serious sublethal effects. Skin lesions and mortality were observed in some of the test fish after only 8 days of exposure to sediment containing approximately 2,500 mg/kg ΣPAH. After 28 days, there was over 50 percent mortality. All the test fish exposed to the contaminated sediments showed sublethal abnormalities (skin lesions, fin erosion, gill erosion, reduced hematocrit level, pancreatic and liver alterations, and lack of weight gain). These same abnormalities were observed in several to all test fish that were exposed only to the effluent from

the test tank containing the contaminated sediment (Hargis *et al.*, 1984; Roberts *et al.*, 1989). Some of the same adverse effects have been observed in feral fish collected from the Elizabeth River near the site.

If PAH exposure imparts acute mortality to individual organisms, a cumulative impact on overall community structure would be also expected. A study of benthic macroinvertebrate assemblages in Elizabeth River sediments concluded that the vertical distribution of organisms was affected in the areas of highest PAH contamination. In areas of high hydrocarbon contamination, few organisms were found below a depth of 5 cm (Diaz, 1985). Other field experiments testing the colonization of creosote-contaminated sediment by benthic invertebrates indicated that polychaetes and crustaceans were significantly affected at creosote concentrations greater than 177 mg/kg. Mollusks, however, were apparently more resistant to the effects of creosote contamination, showing effects only at concentrations greater than 844 mg/kg (Tagatz *et al.*, 1983).

Fish distributions along the Elizabeth River have also exhibited adverse community impacts. Huggett *et al.* (1987) reported reduced total biomass, reduced total numbers of individuals, and reduced abundance of selected species (Table 6). These factors, as well as the increased prevalence of several gross abnormalities noted above, correlated with PAH contamination in the sediments and proximity to the wood treatment facilities. The largest shift in fish assemblages seem to occur when sediment PAH concentrations were greater than approximately 12 mg/kg.

### **Pentachlorophenol**

No studies were found relating sediment concentrations of PCP to toxicity in aquatic organisms. However, a number of studies have established the aqueous toxicity of PCP to both freshwater and marine species (Choudhury *et al.*, 1986). PCP affects energy metabolism and increases oxygen consumption. Collectively, its effects would lead to reduced availability of energy for growth, reproduction and reduced ability to avoid predators or to catch prey. Acute toxicity values (LC<sub>50</sub>) for sensitive marine and freshwater fish species are less than 100 µg/l. Abnormal development of oyster larvae was reported at levels of 40 mg/l, with acute toxicity at 77 mg/l (USF&WS, 1989). Reproductive impacts have been reported for a variety of sensitive species at levels ranging from 15 to 100 mg/l (USF&WS, 1989). Results from a study with an experimental ecosystem showed that low levels of PCP (15.8 µg/l) led to a reduction in numbers of individuals and species in a marine benthic community, with mollusks showing the most sensitivity (Tagatz *et al.*, 1978).

Fish may bioconcentrate PCP to levels 10,000 times the concentration in water (Eisler, 1989). Measurements of PCP levels in six species of fish representing two trophic levels from Lake Ontario suggested that PCP accumulation in fish is primarily through direct uptake from water rather than accumulation through the food chain (Niimi and Choy, 1983).

### **Polychlorinated Dibenzo-p-Dioxins and Polychlorinated Dibenzofurans**

PCDDs or PCDFs were not reported in drainage ditch sediments or sediments of the inlet. However, due to high detection limits, the presence and spatial occurrence of the chemicals cannot be evaluated. Both compounds were detected in on-site soil and groundwater and could be of concern in nearshore habitats. Dioxins and dibenzofurans, particularly 2,3,7,8-TCDD and 2,3,7,8-TCDF, are known to be toxic to aquatic organisms at extremely low concentrations. Very little information is available on the toxicity of other dioxin congeners to aquatic organisms, but some are also known to be toxic at very low levels. Dioxins and furans are environmentally stable and have a strong tendency to accumulate in lipid tissues of aquatic organisms. Bioavailability of 2,3,7,8-TCDD in freshwater and marine sediments to aquatic organisms has been demonstrated (Kuehl *et al.*, 1986; Rubenstein *et al.*, 1990). Effects associated with dioxins and furans include lethality, carcinogenicity, mutagenicity, teratogenicity, reproductive impairment, immunotoxicological, and histopathological abnormalities (Eisler, 1986). Toxicity is related to duration of exposure as well as concentration (EPA, 1990). The toxic effects following even short-term TCDD exposure are commonly delayed 30 to 80 days. Fish species appear to be more sensitive than other

aquatic organisms. Developing embryos are the most sensitive life stage to dioxin effects (Cooper, 1989; Spitzbergen *et al.*, 1991; Walker *et al.*, 1991).

The U.S. Food and Drug Administration (FDA) has issued a health advisory for concentrations of 2,3,7,8-TCDD over 50 pg/g (parts per trillion) and suggested that concentrations less than 25 pg/g in fish tissue should not cause serious human health risk (EPA, 1984). Ambient water quality criteria (AWQC) for the protection of aquatic organisms are available for freshwater only (EPA 1984). Acute AWQC was not determined due to the lack of sufficient toxicity data on TCDD. The “chronic” AWQC value (final residue value) of 10 picograms per liter for 2,3,7,8-TCDD, based on a bioconcentration factor of 5,000, is the concentration estimated to result in average levels in edible fish tissue that exceed the PDA advisory levels (EPA, 1984). However, a recent EPA report concluded that levels of 0.038 pg/l for 2,3,7,8-TCDD (or TCDD-equivalents) and 0.41 pg/l for 2,3,7,8-TCDF would be expected to result in toxicity in some aquatic species (EPA, 1990).

### **Trace Elements**

The toxicity of arsenicals to aquatic life is modified by numerous biological and abiotic factors. Arsenic is carcinogenic and teratogenic in humans and other mammals. Both acute mortality and sublethal effects have been observed in fish and invertebrates. Acute toxicity has been shown to be affected by water temperature, pH, Eh, organic content, phosphate concentration, suspended solids, and presence of other substances and toxicants, as well as arsenic speciation and duration of exposure (Eisler, 1988). Early developmental stages are the most sensitive to arsenic toxicity. Some aquatic species have been reported to be adversely affected at water concentrations of 19 to 48 µg/l, or 120 mg arsenic/kg in the diet, or tissue residues of 1.3 to 5 mg arsenic/kg fresh weight (Eisler, 1988).

Arsenic has been shown to bioconcentrate in organisms but is not biomagnified in the food chain. Bioconcentration factors for inorganic arsenic in most aquatic invertebrates and fish exposed for 21 to 30 days did not exceed 17 (Eisler, 1988).

Information on the toxicity of sediment-associated arsenic is scarce (Long and Morgan, 1990). Data from Puget Sound, Washington indicate that arsenic levels of 63 mg/kg are moderately toxic to amphipods: nearly one-fourth of the sampled oyster larvae developed abnormally following exposure to sediment containing levels of 59 mg/kg. Benthic community structure was altered when sediment concentrations were above 57 mg/kg. Unfortunately, no spiked sediment bioassays or laboratory studies were available to confirm impact levels suggested by these field observations.

The toxicity of copper to aquatic life has been shown to be related primarily to activity of the cupric (+2) ion, and possibly to some hydroxy complexes (EPA, 1985). The cupric ion is highly reactive and forms moderate to strong complexes and precipitates with many inorganic and organic constituents of natural waters. It is readily sorbed onto surfaces of suspended solids. The proportion of copper present as the free cupric ion is generally low and may be less than one percent in waters where complexation predominates. Most organic and inorganic copper complexes and precipitates appear to be much less toxic than free cupric ion and tend to reduce toxicity attributable to total copper (EPA, 1985).

As an essential nutrient, copper is accumulated by plants and animals, although it is not generally biomagnified. Among saltwater species, the highest bioaccumulation factors are those for the bivalve mollusks. Oysters can bioaccumulate copper up to 28,200 times ambient levels without significant mortality (Clement Associates, 1985).

Several observations on sediment toxicity of copper are available. Phelps *et al.* (1983) reported that burrowing time in littleneck clams, *Protothaca staminea*, was increased when copper concentrations in marine sediments were above 17.8 mg/kg dry sediment. Burrowing times of *Macoma* were significantly longer at concentrations of 67 mg/kg (McGreer, 1979). Levels above 19.5 mg/kg were found to be toxic to *Hyalella* (in Long and Morgan, 1990), while significant mortality to *Daphnia* has been reported at levels of 68 mg/kg (Qasim *et al.*, 1980). Significant mortality to amphipods and abnormal development of

bivalve larvae have occurred in San Francisco sediments with copper levels above 64 and 76 mg/kg, respectively.

Zinc may be found in several chemical forms in natural waters. The dominant fate for zinc is sorption to sediments, but this process is influenced by pH, salinity, and the presence of organic complexes. Reported acute toxicity values for saltwater fish range from 2,730 to 83,000 µg/l and from 166 to 55,000 µg/l for saltwater invertebrates. Zinc produces chronic toxicity in the mysid shrimp (*Mysidopsis* sp.) at 166 µg/l. Adult mummichog exposed to 43,000 µg/l of zinc for 192 hours showed no adverse effects; whereas, a concentration of 157,000 µg/l resulted in 100 percent mortality (EPA, 1987).

Zinc is another essential nutrient and is bioregulated by most species. Bioconcentration factors in edible portions of aquatic organisms range from 43 for the soft-shell clam to 16,700 for the oyster (Clement Associates, 1985).

In a spiked-sediment bioassay with the marine amphipod *Rhepoxynius abronius*, the 10-day LC<sub>50</sub> for zinc was 276 mg/kg (Swartz *et al.*, 1988). In Puget Sound, field-collected sediments were always toxic to *R. abronius* at zinc concentrations greater than 960 mg/kg (n=287) and resulted in a significant decrease in the abundance of one or more major macroinvertebrate taxa (polychaetes, mollusks, crustaceans, or total benthic infauna) at concentrations greater than 410 mg/kg (n=20).

### Site-Specific Toxicity Studies

The only direct measure of toxicity of site-specific contaminants migrating from the Atlantic Wood site are the result of biomonitoring requirements for Atlantic Wood's NPDES permit. The NPDES permit specifies that 48-hour static acute bioassays be performed annually with effluent samples from outfalls #001, #002, and #003 to determine the acute mortality due to exposure to outfall effluents. Bioassays conducted on effluent samples collected in 1986 from two of the Atlantic Wood stormwater outfalls showed acute toxicity to mysid shrimp *Mysidopsis bahia* at effluent concentrations of 47 and 37 percent (*i.e.*, 37 % effluent and 64% diluent water) for Outfalls 001 and 002, respectively (ESC, 1988). No toxicity was observed in tests with sheepshead minnows (*Cyprinodon variegatus*) and mysid shrimp exposed to 1991 samples (Zieske, 1991). Data for other years (1984 to 1989) were not provided.

### Existing Toxicity-Based Criteria and Standards

The only criteria and standards applicable to the Atlantic Wood site are the EPA AWQC for the protection of aquatic organisms and the site-specific NPDES effluent permits. Applicable AWQC for marine surface waters are presented in Table 8.

**Table 8.** AWQC for the protection of marine organisms for the chemicals of concern at the Atlantic Wood site (EPA, 1986g).

Chemicals of Concern	Marine AWQC (µg/l)	
	Acute	Chronic
<u>PAHs</u>		
Acenaphthene	970*	710*
Fluoranthene	40*	16*
Naphthalene	2,350*	NA
PAH	300*	NA
Pentachlorophenol	13	7.9*
<u>Trace Elements</u>		
Arsenic (trivalent)	69	36
Arsenic (pentavalent)	2319*	
Copper	2.9	2.9
Zinc	95	86
* Insufficient data to develop criteria. Value presented is the lowest observed affect level. NA: Not Available		



## RISK CHARACTERIZATION

In its present condition and use, the Atlantic Wood site presents a continuing threat to aquatic habitats based on examinations of potential pathways for contaminant migration, the toxicity hazard due to site-related contaminants, and the estimated exposure levels. The south branch of the Elizabeth River, located immediately adjacent to the site, is of particular concern since it has received contamination through direct releases of creosote to the river. Since contamination in soil, groundwater, and sediment onsite represents a reservoir of source contamination, there is the continued potential for site-related contaminants to be transported off-site through surface water runoff, water discharge, and fugitive dust emissions from wind erosion.

Since many of the contaminants of concern at Atlantic Wood will likely end up adsorbed to sediments, benthic and epibenthic organisms that have frequent contact with sediments will be most at risk. This includes benthic fish and invertebrates as well as those species that feed on benthic prey such as blue crab, weakfish, spot, and croaker. However, given the extreme levels of contamination, any organism that lives near the Atlantic Wood site may be at potential risk.

### Comparison Against Criteria and Standards

Based upon comparisons with EPA's AWQC for the protection of aquatic organisms (Table 8), it is probable that aquatic species present in the inlet and along the shoreline of the site are at risk to acute and chronic toxicity due to exposures of site-related contaminants. Maximum levels of ΣPAHs detected in groundwater exceed the AWQC concentrations by three orders of magnitude. Maximum levels of PCP exceed AWQC by nearly two orders of magnitude. Given these extreme values, it is likely that AWQC may be exceeded for these compounds even after groundwater are diluted upon discharge to surface waters<sup>2</sup>. No sampling of surface waters has been conducted to verify this hypothesis.

No criteria are presently available to evaluate the hazard of sediment contamination that are comparable to the AWQC. For the purposes of this risk characterization, the data compiled by concentrations of contaminants of concern at the Atlantic Wood site that would be predictive of adverse biological impacts. Because no information was presented by Long and Morgan for PCP, the Apparent Effects Threshold (AET) values (PTI, 1988a) have been presented for this compound. Only the maximum concentrations of the contaminants of concern will be used.

Based on the comparisons of the concentrations of the contaminants of concern in sediments with the values compiled by Long and Morgan (1990), there is a significant potential for impact to aquatic species inhabiting on-site wetland areas, the inlet, and nearshore areas of the Elizabeth River adjacent to the site (Table 9). Impacts could occur at both the maximum estimated exposure level and the "more-likely" (median) estimated exposure level. Because some of the data compiled by Long and Morgan (1990) includes the results of chronic bioassays, it is probable that both chronic and acute effects could be observed as a result of exposure of aquatic organisms to contaminated sediments from the Atlantic Wood site.

---

<sup>2</sup> A model of predicted exposure levels upon discharge of contaminated groundwater to the river was conducted and included as Appendix L in the RI. However, groundwater flow rates and contamination levels were not fully characterized; actual river flow data for calculation of dilution factors were not available; and, tidal flushing and contributory surface water sources were not accommodated. Since no ground-truthing of this model has been conducted, the model will not be incorporated in this assessment.

**Table 9.** Comparison of the sediment concentrations (mg/kg) of the contaminants of concern to the data compiled by Long and Morgan (1990) and AETs (PTI, 1988a).

Chemical	Sediment Concentration		Long & Morgan Data			AET
	Median	Max	ER-L	ER-M	OEAT	
Total PAH	1008	38,437	4	35	22	140
PCP	15	67	ND	ND	ND	360
<u>Trace Elements</u>						
Arsenic	N/D	374	33	85	50	57
Copper	N/D	1,350	70	390	300	390
Zinc	N/D	1,890	120	270	260	410

### Comparison of Estimated Exposure Levels with Toxicity Data

Sediment creosote toxicity data have been reported for laboratory and field studies, including field studies near the Atlantic Wood facility, using benthic macroinvertebrates and fish. In all the studies, creosote concentrations were expressed as ΣPAHs. The ΣPAH concentrations of these studies are compared to the maximum ΣPAH concentration in sediments of the inlet at the Atlantic Wood site (Table 10). The toxicity data cited in the literature reviewed is based upon the PAH composition of the sediments used in those studies. These data may differ from the PAH composition of sediments found at the Atlantic Wood site. However, for comparison purposes, it is assumed that the PAH compositions of the sediments are similar enough to allow meaningful comparison. Based on the highest ΣPAH concentration of 38,437 mg/kg in inlet sediments, the toxicity information indicates that there is a substantial risk to aquatic organisms in the inlet and nearshore areas of the Elizabeth River adjacent to the Atlantic Wood site from exposure to site-related contaminants. Based upon the median value of 1,008 mg/kg, substantial risk to aquatic organisms is still indicated by the data in Table 10.

The toxicity data show that a wide range of toxic responses could result from the levels of contamination at the site. Lethal toxicity could result from brief exposures to areas where extremely high concentrations of contaminants exist. Decreased species abundance and diversity may be expected, especially for benthic macroinvertebrate populations in direct contact with sediments, but also among the fish community. Sublethal impacts, such as decreased reproductive success, immunosuppression, tissue damage, neoplasia, and reduced scope for growth, could result from long-term exposure to contaminated sediments in this stretch of the river.

Some of these sublethal impacts, immunosuppression, for example, have been shown to be reversible upon cessation of the toxic stress. The levels of PAH exposure at which there would be no observable adverse impacts have not been determined, however.

**Table 10.** Comparison of sediment PAH toxicity data from the literature with the maximum concentration of ΣPAH (mg/kg) in sediments from the inlet of 38,437 mg/kg.

Species	Total PAH	Effect	Reference
<b>Invertebrates</b>			
Grass shrimp	5.8-65.5	Lethal and sublethal effects	Alden & Butt, 1987
Diporeia	100	Mortality	Landrum <i>et al.</i> , 1991
Benthic invertebrates	≥177	Reduced colonization by polychaetes and crustaceans; reduced biomass	Tagatz <i>et al.</i> , 1983; Diaz, 1985
	>844	Reduced colonization by mollusks	Tagatz <i>et al.</i> , 1983
<b>Fish</b>			
Spot	3	Induced MFO enzymes	Van Veld <i>et al.</i> , 1990
Various species in the Elizabeth River	11.5	Reduced biomass	Huggett <i>et al.</i> , 1987
Spot, weakfish, croaker	15.82	Cataracts	Huggett <i>et al.</i> , 1987
Mummichog	61	Immunosuppression	Faisal <i>et al.</i> , in press
Hogchoker	100	Immunosuppression	Weeks & Warinner, 1984
Toadfish	154	Fin erosion	Huggett <i>et al.</i> , 1987
Mummichog	2200	Presence of neoplasia	Vogelbein <i>et al.</i> , 1990

### Adverse Biological Effects

No specific studies were conducted at the Atlantic Wood site that relate on-site contaminant concentrations in sediments to adverse effects on aquatic organisms. However, a number of studies were conducted in the Elizabeth River next to the site that have related sediment concentrations of PAHs to probable effects on aquatic organisms within the river. The results of these studies and the PAH levels at which effects were associated have already been discussed in the Toxicity Assessment section and summarized in Table 10. Only the types of effects observed will be reviewed here.

Blue crabs collected in the south branch of the Elizabeth River just below and just above the Atlantic Wood site were found to have elevated levels of PAHs in their muscle (up to 3,200 ppb) and hepatopancreas (up to 13,000 ppb) (Graves, 1989). The PAH composition in tissues of blue crab collected from the majority of the sites in the Elizabeth River system was predominantly low molecular weight alkylated PAHs, such as alkyl naphthalenes. The pattern of PAH contamination in crab collected from the Elizabeth River in the vicinity of the Atlantic Wood site changed to one more comparable with that of the sediments in this area, showing an increase in unsubstituted PAHs. Spot from the area were found to have induced MFO enzyme systems (Van Veld *et al.*, 1990). These observations confirm the bioavailability, bioaccumulation, and bioresponse of resident organisms to existing PAH exposures adjacent to Atlantic Wood.

Macrophage activity in hogchoker from the vicinity were in a depressed state (Weeks and Warinner, 1984). This depression of the cellular immune system could result in increased susceptibility to disease. There are also reports of alterations in cancer defense mechanisms (Faisal *et al.*, in press), indicating an ongoing oncogenic process in fish from the area of the Atlantic Wood site.

Tumors and other indications of tissue damage (fin erosion, cataracts, gill hyperplasia) have been observed in fish collected near the site (Huggett *et al.*, 1987). Grossly visible hepatic lesions occurred in 93 percent of the mummichog collected next to Atlantic Wood (Vogelbein *et al.*, 1990).

Sediments from the Elizabeth River near the Atlantic Wood site were reported to rapidly produce lethal and sublethal effects in grass shrimp (Alden and Butt, 1987).

Huggett *et al.* (1987) reported that the total number and biomass of several species of fish, including hogchoker, toadfish, spot, weakfish, and croaker decreased from the mouth of the Elizabeth River to a station 2 km above the Atlantic Wood site. This decrease was associated with an increasing concentration

of PAHs in the sediments of the river. Additionally, higher incidences of fin erosion and cataracts in fish were also associated with increasing sediment PAH concentration.

These observations encompass a full range in levels of biological organization, from subcellular to community parameters, and document existing adverse impacts due to PAH exposure. Based upon comparisons of the maximum and “more likely” exposure estimates with the reported concentrations associated with these impacts, sediments along the shoreline of the Atlantic Wood site may cause a wide variety of acute and chronic effects on aquatic organisms.

### Joint Action of Chemical Mixtures

Potential receptors at the Atlantic Wood site are exposed to a diverse mixture of chemicals. The interactions among these chemicals may either increase or decrease the overall combined effects, or there may be no interaction at all. Another outcome of mixtures is that a compound that may not be particularly toxic by itself could become highly toxic in the presence of certain other chemicals. Predicting all the possible interactions among the vast array of contaminants present onsite is not feasible; however, some general predictive indications from existing studies are available.

As an approximation, chemicals that work by similar modes of toxic action typically will have a combined impact that is equal to the addition of all the individual impacts (an additive impact) when exposures are at high concentrations. For instance, exposure to two compounds each at LC<sub>10</sub> concentration would give the same response as an LC<sub>20</sub> concentration to either compound singularly. As exposure levels decrease, observations seem to indicate either the chemicals impart less joint action and approach no interaction, or our ability to detect interactions are surpassed. Chemicals of differing toxic modes may have a less-than-additive effect, a more-than-additive effect, or may be antagonistic. Predicting which outcome occurs for specific sets of chemicals based on chemical properties or structures seems to have had limited success, and must be based at this point on empirical observation.

There are two studies that supply empirical observations for contaminants of concern at the Atlantic Wood site. Landrum *et al.* (1991) studied the impact of PAH mixtures and Verma *et al.* (1981) has reported results with PCP. Landrum *et al.* (1991) exposed *Diporeia* (a burrowing amphipod) for up to 26 days to a mixture of PAHs with radio-labeled tracers at four molar concentrations and measured mortality and selected toxicokinetics. Their limited preliminary data suggests that the combined toxicity of a mixture of individual PAH compounds is additive (*i.e.*, the sum of toxicity for each of the individual compounds). Verma *et al.* (1981) studied joint action of PCP, phenol, and dinitrophenol. Tests were made with all possible permutations of holding one or two chemical concentrations fixed at an LC<sub>0</sub>, while testing the LC<sub>50</sub> of the other. All paired combinations were found to be more-than-additive (the combined toxicity was greater than that predicted by adding the two individual toxicities). Most three-way combinations were more-than-additive, one was additive, and two combinations were antagonistic. The two combinations that were antagonistic involved PCP held constant, while phenol or dinitrophenol were varied.

Given the extreme levels of contamination observed in the inlet and shoreline at Atlantic Wood, exposures within these habitats may be considered as “acute” (equivalent or greater than those used in short-term lethality bioassays). They are also within the range used in the studies cited above. Data from these studies would then be clearly applicable to the exposure situations in these habitats. Results of these studies provide general indications that the overall toxicity of mixtures of PAHs, and mixtures of PCP with selected other contaminants, is the sum of the toxicities of all the individual compounds added together. Risk characterizations, which are determined for individual compounds, would therefore be considered only partial risks and underestimates of the total, overall risk of exposure to mixtures of contaminants.

# TERRESTRIAL ECOLOGICAL RISK ASSESSMENT

## POTENTIAL RECEPTORS

The potential terrestrial receptors are those species that live within, or may transit through, the limited terrestrial habitat within the Atlantic Wood site.

### Terrestrial Habitats

The following represents a general characterization of the terrestrial habitat and fauna (mammalian, avian, reptilian/amphibian) near the Atlantic Wood site.

The site, located along the banks of the south branch of the Elizabeth River, is situated in an urbanized industrial complex. Overall habitat near the site is marginal to poor. Fences around the periphery of the site limit access for large mammals.

Surrounding land use near the site is industrial and residential. Upland habitat and open space have been reduced to intermittent patches of deciduous forest and managed recreational or residential lands (playgrounds, golf courses, residential landscaping). Habitat providing a suitable home range (cover, food, water, migration corridors, and edge) for large mammals, particularly predators, is not available. Numerous highways, roads, and railways below the site and north of Great Dismal Swamp form a physical barrier for mammals, less so for reptiles and amphibians, and are of no consequence to birds. While not impassable, these features are an effective form of mortality on migratory mammal populations (Keel, personal communication, 1991). For this reason black bear, white tail deer, red fox, river otter, mink, and bobcat are rarely observed outside Great Dismal Swamp near Portsmouth, Virginia. Surrounding upland habitat may still be utilized by songbirds (swallows, yellowthroats, etc.) for nesting and foraging. Also, it is probable that some waterfowl species may graze on available lawns and turf fields (Schwab, personal communication, 1991).

### Habitats of Special Concern

There are no significant or critical habitats located near the site. The closest critical habitats are two heron rookeries, great blue heron and yellow-crowned night heron, 5 km downstream near the Hampton Roads Army Terminal (USF&WS, 1980).

### Species

The site itself is largely devoid of natural vegetation, but the wetland areas on the site or in nearby off-site areas contain reed grass or saltbush communities, which are generally at least partially disturbed.

No data were found describing the invertebrate species present at the site or in the wetlands. It would be expected that normal soil and wetland invertebrate species would be supported in areas not occupied by construction or paving, and not grossly contaminated.

Available data on reptilian and amphibian species near the site are summarized in Table 11. Data were available only from quite general sources. The species listed in Table 11 are presumed to be present at the site based upon broad survey data. The many-lined salamander, *Stereochilus marginatus*, is a rare species that occurs in St. Julian Creek but has no state or federal legal status (Virginia Department of Conservation and Recreation, 1991).

Mammals are limited near the site, but those present probably reside near the site for long durations, increasing their exposure to contaminants. This may be particularly true for species that burrow into the stream banks for shelter. The mammalian species near the site are typical of those species adapted to, and found near, urban environments. Muskrat, raccoon, marsh rabbit, and Norway rat are known to breed, raise young, and feed as adults near or on the site. Opossum, meadow vole, and marsh rice rat are also

likely to breed near or on the site. It is suspected that feral cats are also present (Halbrook, personal communication, 1991). These populations are stable, probably because of limited available habitat rather than predation by carnivores. Recent studies indicated that muskrats taken from Paradise Creek were stressed (as indicated by elevated numbers of parasites, significantly higher concentrations of cadmium, copper, and aluminum in their tissue, and lower body and liver weights), but did not identify conclusive causes that could be linked with activities at the Atlantic Wood site (Halbrook, personal communication, 1991).

Survey data indicate that waterfowl, wading birds, shorebirds, raptors, and songbirds may be found utilizing the site during different parts of the year (Table 11). Waterfowl are considered the predominant users of areas near the site. Nesting areas or rookeries near the site are extremely unlikely, especially with the abundance of superior habitat to the north and south. Utilization by avian species is primarily roosting and adult forage.

Common waterfowl species are dabblers, black duck, mallard, and ruddy duck. These species feed primarily on submerged and emergent aquatic vegetation near the shore of the river and in the adjacent wetlands. Use by waterfowl is greatest during annual migrations (Beccasio, 1980).

Wading birds most typically found are great blue heron, black-crowned night heron, yellow-crowned night heron, clapper rail, snowy egret, and great egret. It is possible that these species (except clapper rail) may roost periodically on piers, wharfs, and other waterfront structures. Great blue heron and yellow-crowned night heron are State-protected species. These latter two species typically feed on marine invertebrates, reptiles, amphibians, and fish in shallow waters (Beccasio, 1980).

**Table 11.** Preliminary listing of terrestrial fauna utilizing habitat in the general vicinity of the Southern Branch of the Elizabeth River (Beccasio, 1980; USFWS, 1980; Niering, 1987; Keel, personal communication, 1991; Schwab, personal communication, 1991; Virginia Department of Conservation and Recreation, 1991).

Common Name	Scientific Name	Breeding	Juvenile	Adult Forage
<u>Reptiles/ Amphibians</u>				
Two-lined salamander	<i>Eurycea bislineata</i>			♦
Mud turtle	<i>Kinosternon subrubrum</i>	♦	♦	♦
Brown water snake	<i>Nerodia taxispilota</i>	♦	♦	♦
Northern water snake	<i>Nerodia sipedon</i>	♦	♦	♦
Mud salamander	<i>Pseudotriton montanus</i>			♦
Pickerel frog	<i>Rana palustris</i>	♦	♦	♦
Many-lined salamander	<i>Stereochilus marginatus</i>	♦	♦	♦
<u>Birds*</u>				
Red-winged blackbird	<i>Agelaius phoeniceus</i>	♦	♦	♦
Mallard duck	<i>Anas platyrhynchos</i>		♦	♦
Black duck	<i>Anas rubripes</i>		♦	♦
Great blue heron	<i>Ardes herodias</i>			♦
Great egret	<i>Casmerodius albus</i>			♦
Marsh wren	<i>Cistothorus palustris</i>	♦	♦	♦
Peregrine falcon	<i>Falco peregrinus</i>			♦
Bald eagle	<i>Haliaeetus leucocephalus</i>			♦
Herring gull	<i>Larus argentatus</i>			♦
Black-backed gull	<i>Larus marinus</i>			♦
Snowy egret	<i>Leucophox thula</i>			♦
Swamp sparrow	<i>Melospiza georgiana</i>	♦	♦	♦
Black-crowned night heron	<i>Nycticorax nycticorax</i>			♦
Yellow-crowned night heron	<i>Nycticorax violaceus</i>			♦
Ruddy duck	<i>Oxyura jamaicensis</i>			♦
Osprey	<i>Pandion haliaetus</i>			♦
Double-crested cormorant	<i>Phalacrocorax auritus</i>			♦
Clapper rail	<i>Rallus longirostris</i>			♦
<u>Mammals</u>				
Opossum	<i>Didelphis marsupialis</i>			♦
Meadow vole	<i>Microtus pennsylvanicus</i>		♦	♦
Muskrat	<i>Ondatra zibethica</i>	♦	♦	♦
Marsh rice rat	<i>Oryzomys palustris</i>	♦	♦	♦
Raccoon	<i>Procyon lotor</i>		♦	♦
Norway rat	<i>Rattus norvegicus</i>	♦	♦	♦
Marsh rabbit	<i>Sylvilagus palustris</i>		♦	♦
Gray fox	<i>Urocyon cinereoargenteus</i>			♦
* Habitat use by avian species is temporary, primarily for roosting and foraging, and variable depending on seasonal migrations. It is probable that songbirds may be nesting near the site, but doubtful that any waterfowl, wading birds, shorebirds, or raptors are also nesting.				

Three raptors, all listed as federally protected, periodically use habitat near the site. Peregrine falcon and osprey are the most common, and an occasional bald eagle has been observed. The utilization by these species is thought to be infrequent (Schwab, personal communication 1991).

Typical shorebirds include great black-backed gull, herring gull, and double-crested cormorant. Red-winged blackbird, swamp sparrow, and marsh wren are species most likely found near the site.

## **EXPOSURE ASSESSMENT**

### Contaminants of Concern

Identification of the contaminants of concern for the Atlantic Wood site is based on findings in the RI (KER, 1990). PAHs, PCP, PCDDs, PCDFs, arsenic, copper, and zinc have already been identified in the Aquatic Ecological Risk section. Screening approaches for contaminants of concern will not be discussed further in this section.

### Sources of Contaminants

The pattern of soil contamination is described in the Aquatic Ecological Risk section and is depicted in Figure 4. Sources will not be discussed further in this section.

### Fate and Transport Analysis

The environmental behavior of the contaminants of concern has already been discussed in the Aquatic Ecological Risk section and will not be discussed further here.

### Exposure Pathways

The primary sources of exposure to the contaminants of concern are through surface soils and inhalation. Ingestion and dermal absorption are pathways considered from contact with contaminated soils. Inhalation is an additional pathway of concern for burrowing animals. An exposure estimate was made for a representative mammal, bird, and amphibian through the oral exposure route. Two exposure scenarios were assumed: a realistic (low) exposure and an extreme exposure.

### Estimated Exposure Levels

In selecting representative target species, no information was found regarding the morphometrics and dietary intake of species found near Atlantic Wood. Wildlife exposures were based on information for species found in areas supporting forest vegetation (Table 12). The methodology used to determine the exposures is the same as that presented in the environmental impact statements prepared by the U.S. Forest Service (USFS) (1988). Representative species were selected based on their probable similarity in size and dietary intake to species that might be found at the Atlantic Wood site.

### **Ingestion Doses**

Each representative species was assumed to feed on contaminated food items at the site. These dietary amounts are listed in Table 12. In the realistic exposure scenario, each species is assumed to consume a portion of its daily intake in contaminated food items based on its body size. The percentage of food contaminated (PFC) is based on the following formula:

$$\text{PFC} = 100 \times (1/\text{BW})^{0.2},$$

where BW is body weight (USFS, 1988). In the extreme case, each species' entire daily intake is assumed to consist of contaminated items.

In addition to ingestion of contaminants through diet, birds and mammals can also ingest contaminants through preening of feathers and grooming of fur. The amount of contaminant ingested through this route was not calculated.



## **Dermal Exposure**

Although dermal exposures were not calculated, it should be noted that this is another route of contaminant uptake. Dermal exposures are assumed to come primarily from contact with surface soils for mammals and birds, and from contact with surface soils and sediments for amphibians. Fur, feathers, and scales afford varying degrees of protection against dermal exposure. It is likely that amphibians would receive a larger dose of a contaminant when exposed to the same concentration as a mammal or bird because the moist, glandular skin of the amphibian serves to a large extent as a respiratory organ and is much more permeable than the skin of other animal classes (USFS, 1988).

## **Inhalation Exposure**

At the Atlantic Wood site, contaminated airborne particulate matter resulting from wind erosion or construction practices would be the most likely scenario for inhalation exposure. As with any exposure route, the dose received would be a function of concentration, frequency, and duration of exposure. With inhalation exposure, particulate size is another factor that must be considered when estimating the dose an animal receives.

Contamination is known to be present in the surface and subsurface soils throughout the upland portions of the site. In addition, soils and sediments in wetland areas of the site are also contaminated. Reptiles and mammals can be exposed to contamination from the site through the ingestion of food organisms that are contaminated and through the direct ingestion of contaminated soil on food and from cleaning activities.

Birds can be exposed primarily through the ingestion of contaminated food from upland, wetland, and surface water habitats. Because of the extent of resource use, surface water-dependent birds (ducks and shorebirds) are considered to be most at risk.

Four representative species were selected for the Atlantic Wood site: one omnivorous mammal, the cotton rat; two avian species, the kingfisher, a piscivore and the eastern bluebird, an omnivore; and one insectivorous amphibian, the woodhouse toad. Because no morphometric or food consumption data were found for species that may actually be present at the site, the species listed above were selected based on their probable similarity to species found on site. The morphometric and food consumption data for the selected species were used to calculate estimated daily doses from ingestion of contaminated food items.

Two exposure scenarios were assumed, low and extreme exposures. In the low-dose scenario, the percentage of food contaminated was calculated using the percentage of food contaminated (PFC) equation, which is representative of a more realistic situation (USFS, 1988). In the extreme exposure, 100 percent of the diet is assumed to be contaminated. In each case, the maximum concentration of contaminant found in on-site soil was used as the exposure concentration. In the cases of the kingfisher and the woodhouse toad, the maximum concentration of ΣPAH found in inlet sediments was used in calculating the estimated dose because these species are more likely to come in contact with sediments than the other representative species.

Estimated daily doses (EDD) of contaminants were calculated as follows:

$$\text{EDD} = \frac{(\text{Contaminant Concentration in mg/kg}) (\text{Daily food Intake in g/dy})}{(\text{Body Weight in g})}$$

The estimated daily doses are summarized in Table 12.

## **Uncertainty Analysis**

There is a level of uncertainty associated with any approach employed to determine potential risk to potential receptors posed by exposure to contaminants. This uncertainty usually stems from lack of knowledge of the true exposure regimes, lack of information on the health risk posed by a contaminant to the specific receptors of concern, or lack of detailed information on the biology of potential receptors. The overall, cumulative risk posed by exposure to multiple contaminants, either simultaneously or

sequentially, also carries a great deal of uncertainty with it due to the lack of data on multiple exposure toxicity. In this risk assessment, the uncertainty of estimating exposure levels is due to a lack of site-specific data regarding the use of the Atlantic Wood site by terrestrial species. The concentrations used in calculating the estimated daily doses are those found in soil and sediments rather than in the actual food items selected by the representative species. In foraging, it is likely that animals will ingest a certain percentage of contaminated soil, but this probably represents only an incidental amount in most cases.

The PFC equation only takes into account the body weight of the animal for estimating the percentage of food that is contaminated and does not account other factors such as residence time within a particular area or the proportion of the daily food intake that might be consumed within a given location. This factor leads to potential biases in determining exposure estimates. For instance, it is recognized that one would expect birds to have a larger home range and thus consume proportionately less food from a given area than a small mammal, and thus birds have smaller exposure levels. This subjective assumption is not reflected in the estimated exposure levels calculated by the PFC equation (Table 12). Because the equation is quite general and relies only on one parameter, it is limited in its application. However, because so little is known about the specific species that use the Atlantic Wood site and the behaviors of those species, it is necessary to base estimates on the data that are available. Because the PFC equation is objective, and does not arbitrarily specify the percentage of food consumed on site by each target species, this method chosen over more subjective approaches to estimating exposure.

The estimated daily doses for the low-dose scenario in the cotton rat and the woodhouse toad, though conservative, probably represent a more realistic situation. Small species of mammals and amphibians tend to have smaller home ranges and will spend more time in a given area than larger species (USFS, 1988). For this reason, small mammals and amphibians at the Atlantic Wood site are more likely to spend substantial portions of their lives on the site and consume a greater percentage of their diet there.

From the limited information available, it does not appear that the site is used extensively by avian species. Though the site is located along the corridor of the Atlantic Flyway, it is doubtful the site is a preferred habitat for bird species. Some incidental exposure could occur, but the estimated daily dose for the low exposure scenario is most likely excessively conservative and overestimates the actual exposure that avian species would encounter at the site.

**Table 12.** Representative wildlife, their associated biological parameters, and the estimated dose (mg/kg bw/day) of contaminant received through ingestion (does not include dose received from grooming and preening activities) (USFS, 1988).

Species	Chemical <sup>1</sup>	Body weight (g)	Daily intake (g)	Percentage of food contaminated		Low Dose <sup>2</sup>	Extreme Dose <sup>2</sup>
				Low	Extreme		
<b>MAMMALIAN</b> Cotton rat (omnivorous)	Total PAH	156	45	36	100	1,376	3,322
	PCP					100	280
	TCDD					0.001	0.004
	Arsenic					51	143
	Copper					1,015	2,321
	Zinc					2,118	5,885
<b>AVIAN</b> Kingfisher (piscivorous)	Total PAH	250	60	33	100	3,044	9,225
	PCP					77	233
	TCDD					0.001	0.003
	Arsenic					39	119
Eastern bluebird (omnivorous)	Total PAH	29	6	51	100	1,398	2,741
	PCP					100	200
	TCOO					0.001	0.003
	Arsenic					52	102
<b>AMPHIBIAN</b> Woodhouse toad (insectivorous)	Total PAH	22	5	54	100	4,717	8,736

1 The concentration of chemical used in calculating dose was the highest surface soil concentration found on site. For the kingfisher and the woodhouse toad, the highest ΣPAH concentration found in on-site sediments was used in calculating the estimated daily dose because these species are more likely to come in contact with sediments than the other representative species.

2 Estimated daily doses were calculated only for those chemicals for which toxicity information was available for the species listed.

## TOXICITY ASSESSMENT

This section summarizes the toxicity of the contaminants of concern at the Atlantic Wood site to terrestrial species. For the purposes of this study, terrestrial species will include mammals, birds, amphibians, and reptiles.

### Reported Effects of Contaminants of Concern

The terrestrial toxicity assessment summarizes the findings of laboratory studies that indicated the toxicity to wildlife of the contaminants of concern found in surface soils and sediments at the Atlantic Wood site. The term *wildlife*, as used here, refers to mammals, birds, reptiles, and amphibians. In most cases, laboratory studies of domestic animals have been used because of a lack of studies specifically on wildlife. The results of domestic animal studies are considered to be representative of the effects that would occur in similar species in the wild. Toxicity information for each of the contaminants of concern

has been tabularized (Tables 13 through 18) to include data for mammalian, avian, reptilian, and amphibian species. The toxicity information has been subdivided into acute and chronic studies.<sup>3</sup>

**Table 13.** Summary of reported levels for PAH toxicity in wildlife (EPA, 1986d; Eisler, 1987).

Animal Class	Exposure	Dose	Effects
<b>MAMMALIAN</b> Acute Chronic	20-250 mg/kg diet	50-2,000 mg/kg bw 80-100 mg/kg bw 4x10 <sup>-5</sup> - 3,300 mg/kg bw Not reported	LD-50 Reproductive impairment Carcinogenesis Carcinogenesis
<b>AVIAN</b> Acute Chronic		1,261 mg/kg bw 4,000 mg/kg bw 0.002 µg/egg	LC-50 No effect Teratogenic effects
<b>REPTILIAN &amp; AMPHIBIAN</b> Acute Chronic		40 mg/kg bw 1.5 mg (implanted into abdominal cavity)	Induction of mixed-function oxidase activity Production of lymphosarcoma

**Table 14.** Summary of reported levels for PCP toxicity in wildlife (EPA, 1986c; Eisler, 1989)

Animal Class	Exposure	Dose	Effects
<b>MAMMALIAN</b> <u>Acute</u> Laboratory rodents Eastern chipmunk	20-500 mg/kg feed	27-300 mg/kg bw 138 mg/kg bw	LD-50 LD-50
<u>Chronic</u> Laboratory rodents		1.2-30 mg/kg bw/day	Disruption of liver enzyme activity and reduced weight gain after 8-month exposure Teratogenic
<b>AVIAN</b> <u>Acute</u> Mallard & ring-necked pheasant Japanese quail	200 mg/kg diet	13 mg/kg bw/day	
<u>Chronic</u> Domestic chicken	5,139 mg/kg diet	380-504 mg/kg bw	LD-50
<b>AMPHIBIAN &amp; REPTILIAN</b>	1-1,000 mg/kg diet No studies were found	Not reported Not reported	LC-50 Liver abnormalities

<sup>3</sup> Designations of “acute” and “chronic” doses employed are reproduced as stated in the original references.

**Table 15.** Summary of reported levels for 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) toxicity in wildlife (Eisler, 1986; EPA, 1986e).

Animal Class	Exposure	Dose	Effects
<b>MAMMALIAN</b> <u>Acute</u> Laboratory rodents		0.6-284 µg/kg 0.01 -3.57 µg/kg/day	LD-50 Decreased body weight toxic hepatitis & porphyria after 4 to 13 weeks exposure
		0.01-3 µg/kg/day	Teratogenic effects after maternal exposure during days
		0.1 µg/kg/day	Carcinogenesis
<b>AVIAN</b> <u>Acute</u> Quail, dove, mallard & chicken		15-810 µg/kg/day	LD-50
<u>Chronic</u> Chicken		1 -10 µg/kg/day of mixed PCDDs	Death Liver enlargement & necrosis Pericardial, subcutaneous & peritoneal edema
<b>REPTILIAN &amp; AMPHIBIAN</b>	No studies were found		

**Table 16.** Summary of reported levels for arsenic (inorganic) toxicity to wildlife (Eisler, 1988).

Animal Class	Exposure	Dose	Effects
<b>MAMMALIAN</b> <u>Acute</u> Various mammalian species		35-100 mg/kg bw (calcium arsenite) =13-38 mg As/kg bw 1-25 mg/kg bw (sodium arsenite) =0.6-14.5 mg As/kg bw 3-250 mg/kg bw (As trioxide) =2.3-190 mg As/kg bw	LD-50  Lethal  Lethal
<u>Chronic</u> Mouse	5 mg/kg diet for 3 generations (sodium arsenite) = 2.9 mg As/kg diet	Not reported	Reduced litter size
<b>AVIAN</b> <u>Acute</u> Mallard, quail & pheasant		47.6-386 mg/kg bw (sodium arsenite) =28-224 mg As/kg bw	LD-50
Mallard	1,000 mg/kg diet (sodium arsenite) =580 mg As/kg diet	Not reported	LD-50
<u>Chronic</u> <b>REPTILIAN &amp; AMPHIBIAN</b>	No studies were found No studies were found		

**Table 17.** Summary of reported levels for copper toxicity to wildlife (EPA, 1986b).

Animal Class	Exposure	Dose	Effects
<b>MAMMALIAN</b> <u>Acute</u>	No studies were found		
<u>Chronic</u> Laboratory rodents	5,000 mg/kg diet (Cu acetate) = 1,600 mg Cu/kg diet 3,000-4,000 mg/kg diet (Cu sulfate) =1,195-1,593 mg Cu/kg diet	Not reported  155-207 mg/kg bw/day	Accumulation in liver & kidney  Embryonic malformations
<b>AVIAN</b>	No studies were found		
<b>REPTILIAN &amp; AMPHIBIAN</b>	No studies were found		

**Table 18.** Summary of reported levels for zinc toxicity to wildlife (EPA, 1986f)A

Animal Class	Exposure	Dose	Effects
<b>MAMMALIAN</b> <u>Acute</u>	No studies were found		
<u>Chronic</u> Laboratory rodents	5,000 ppm Zn in drinking water 30,000 ppm in diet (Zn sulfate) =12,150 ppm Zn 5,000-10,000 ppm Zn in diet	850 mg/kg bw/day  Approx. 950-1,880 mg/kg bw/day  250-500 mg/kg bw/day	No observed effect  Decreased food intake Retarded growth Hematologic abnormalities Mortality Reproductive impairment Reduced growth
<b>AVIAN</b>	No studies were found		
<b>REPTILIAN &amp; AMPHIBIAN</b>	No studies were found		

### Site-Specific Toxicity Tests

No site-specific bioassays have been conducted with terrestrial species.

### Existing Toxicity-Based Criteria and Standards

There are currently no existing toxicity-based criteria or standards for soils.

## **RISK CHARACTERIZATION**

As with the aquatic species, insufficient data are available regarding the presence of terrestrial species near the site to allow a quantitative risk assessment to be performed. In addition, no toxicity data are available for species that have been identified near the site. Because of these limitations, the risk assessment will be limited to a more general consideration of potential toxicity to mammals (primarily rodents), birds, reptiles, and amphibians.

In selecting representative target species, no information was found regarding the morphometrics and dietary intake of species found in the vicinity of Atlantic Wood. Species selection was based on a list of species presented in an environmental impact statement prepared by the USFS (1988) which listed

mammalian, avian, reptilian, and amphibian species; their estimated body weights; and their estimated daily dietary intake. Representative species were selected based on their probable similarity in habitat, size, and dietary habits to species that might be found at the Atlantic Wood site.

#### Comparison of Estimated Exposure Levels with Toxicity Data

In this section, the estimated daily doses of the chemicals of concern received by representative target species through ingestion are compared to laboratory toxicity data (ingestion only) (Table 19). Both chronic and acute laboratory toxicity data were used for comparison to the estimated daily doses. Daily doses due to inhalation and dermal exposures were not calculated because there is insufficient data to make such calculations and because the estimated daily doses calculated due to ingestion most likely overestimate the doses being received by receptor species using the site.

Based on the low-dose scenario of the estimated daily doses calculated for the chemicals of concern at the Atlantic Wood site, mammalian, avian, and amphibian species onsite are at risk due to exposure to these chemicals. Mammalian species could suffer chronic and acute toxicity from ingestion of PAHs, PCP, PCDDs and PCDFs, and arsenic. Concentrations of copper and zinc are high enough so that mammals could suffer chronic toxicity from long-term exposure to these substances.

Avian species are potentially at risk from both chronic and acute toxicity from exposures to the PAHs, PCDDs and PCDFs, and arsenic. No toxicity data were available describing the toxicity of copper and zinc to avian species, so the risk of these substances to avian species cannot be addressed.

Amphibian species could receive both chronic and acute doses of the PAHs. No toxicity data were found describing the toxicity of the other chemicals of concern to either amphibians or reptiles, so the risk to these species associated with exposure to the other chemicals will not be addressed.

#### Adverse Biological Effects

Very limited information was available describing habitats on the site. A wetland delineation study conducted by KER, Inc. (1990) reported five wetland areas on the site described as being in a disturbed condition and of very low ecological importance. No information was available describing presence or absence of key species, population assessments of key species, or community indices. No studies have been conducted relating contaminant concentrations at the site with adverse effects in terrestrial species.

#### Joint Action of Chemical Mixtures

Joint action of chemicals mixtures has already been discussed in the Aquatic Ecology Risk section. Such action will not be discussed further in this section.

**Table 19.** Comparison of laboratory toxicity data with the estimated daily doses that the selected target species could receive at the Atlantic Wood site through exposure to site soils or sediments (Eisler 1986, 1987, 1988, 1989; EPA 1986 b,c,d,e,f). All concentrations are expressed as mg/kg body weight unless otherwise noted.

Representative Species & Chemical	Laboratory Toxicity Data		Estimated Daily Doses	
	Acute	Chronic	Low Dose	Extreme Dose
<b>MAMMALIAN</b>				
<u>Cotton Rat</u>				
Total PAH	50-2,000	4x10 <sup>-5</sup> -3,300	1,376	3,822
PCP	27-300	1.2-30	100	280
TCDD	0.0006-0.284	(1-357)x10 <sup>-5</sup>	0.001	0.004
Arsenic	0.6-190	2.9 mg/kg diet	51	143
Copper	No data	155-207	1,015	2,821
Zinc	No data	250-1,880	2,118	5,885
<b>AVIAN</b>				
<u>Kingfisher</u>				
Total PAH*	1,261	4,000	3,044	9,225
PCP	380-504	1-1,000 mg/kg diet	77	233
TCDD	0.015-0.81	0.001-0.01	0.001	0.003
Arsenic	48-386		39	119
<u>Eastern Bluebird</u>				
Total PAH	1,261	4,000	1,398	2,741
PCP	380-504	1-1,000 mg/kg diet	100	200
TCDD	0.015-0.81	0.001-0.01	0.001	0.003
Arsenic	48-386	No data	52	102
<b>AMPHIBIAN</b>				
<u>Woodhouse Toad</u>				
Total PAH*	No data	40	4,717	8,736

\* For these species, the highest sediment concentration of ΣPAHs was used in calculating the estimated daily dose.

## REFERENCES

Alden, R.W., and A.J. Butt. 1987. Statistical classification of the toxicity and polynuclear aromatic hydrocarbon contamination of sediments from a highly industrialized seaport. Environmental Toxicology and Chemistry 6: 673-684.

Beccasio, A.D., G.H. Weissberg, A.E. Redfield, R.L. Frew, W.M. Levitan, J.E. Smith, and R.E. Godwin. 1980. Atlantic coast ecological inventory user's guide and information base. Washington, D.C.: U.S. Fish and Wildlife Service, Biological Services Program.

Choudhury, H., J. Coleman, C.T. Rosa, and J.F. Stara. 1986. Pentachlorophenol: health and environmental effects profile. Toxicology and Industrial Health 2: 483-574.

Chemical Information Systems, Inc. (CIS). 1986. Oil and Hazardous Materials/Technical Assistance Data System.

Clement Associates. 1985. Chemical, Physical, and Biological Properties of Compounds present at Hazardous Waste Sites. Washington, D.C.: U.S. Environmental Protection Agency.

Diaz, R.J. 1985. Macrobenthos of the Elizabeth River in relation to hydrocarbon contamination of the sediments. Final Report to the Coastal Energy Impact Program. Virginia Institute of Marine Science,



College of William and Mary. Gloucester Point, VA.

Eisler, R. 1986. Dioxin hazards to fish, wildlife, and invertebrates: a synoptic review. U.S. Fish and Wildlife Service Biological Report 85 (1.8). 37 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 (1.11). 81 pp.

Eisler, R. 1988. Arsenic hazards to fish, wildlife, and invertebrates: a synoptic review. U.S. Fish and Wildlife Service Biological Report 85 (1.12). 92 pp.

Eisler, R. 1989. Pentachlorophenol Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review. U.S. Fish and Wildlife Service Biological Report 85 (1.17). 72 pp.

ESC. 1988. Remedial Investigation/Feasibility Study Work Plan and Appendices for the Atlantic Wood Industries, Inc. Site. Vienna, VA: Environmental Strategies Corporation.

Faisal, M., B.A. Weeks, W.K. Vogelbein, and R.J. Huggett. In press. Evidence of aberration of the natural cytotoxic cell activity in *Fundulus heteroclitus* (Pisces: Cyprinodontidae) from the Elizabeth River, Virginia. Virginia Institute of Marine Science, School of Marine Science, College of William and Mary, Gloucester Point, VA.

Firestone, D., J. Riss, N.C. Brown, R.P. Barron, and J.N. Daraico. 1972. Determination of polychlorinated dibenzo-p-dioxins and related compounds in commercial chlorophenols. Journal Association of Official Analytical Chemists 5: 85-92.

Gillingham, Lewis. Virginia marine resources commission, Newport News, Virginia, personal communication, October 5, 1989.

Graves, J. 1989. Elizabeth River Long-Terra Monitoring Program - Phase 1, 1989: Analysis of organic pollutants in sediments and blue crab (*Callinectes sapidus*) tissues. Final Report to the Virginia State Water Control Board, Richmond, VA.

Halbrook, Richard S. 1991. Wildlife biologist, Oak Ridge National Laboratory, Oak Ridge, TN. Personal communication, March 5, 1991.

Hargis, W.J., M.H. Roberts, and D.E. Zwerner. 1984. Effects of contaminated sediments and sediment-exposed effluent water on estuarine fish: acute toxicity. Marine Environmental Research 14: 337-354.

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. Maid, 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.

Jensen, S. and L. Renberg. 1973. Chlorinated dimers present in several technical chlorophenols used as fungicides. Environmental Health Perspect 5: 37-39.

Johnson, R.L., P.J. Gehring, R.J. Kociba and B.A. Schwetz. 1973. Chlorinated dibenzodioxin and pentachlorophenol. Environmental Health Perspect 5: 171-175.

Johnson, L.L., E. Casillas, T.K. Collier, B.B. McCain, and U. Varanasi. 1988. Contaminant effects on ovarian maturation in English sole (*Parophrys vetulus*) from Puget Sound, Washington. Canadian Journal of Fisheries and Aquatic Sciences 45: 2133-2146.

Keel, Ralph. 1991. Staff biologist, Great Dismal Swamp National Wildlife Refuge, Suffolk, VA. Personal communication, February 28, 1991.

Keystone Environmental Resources, Inc. (KER). 1990. Remedial Investigation Report of Atlantic Wood Industries, Inc., Portsmouth, Virginia Site. Prepared for Atlantic Wood Industries, Inc., Savannah, GA.

- Konasewich, D.E., P.M. Chapman, E. Gerencher, G. Vigers, N. Treloar. 1982. Effects, pathways, processes, and transformation of Puget Sound contaminants of concern. NOAA Technical Memorandum OMPA-20. Boulder, CO. National Oceanic and Atmospheric Administration. 357 pp.
- Kuehl, D.W., P.M. Cook, and A.R. Batterman. 1986. Uptake and depuration studies of PCDDs and PCDFs in freshwater fish. Chemosphere 15: 2023-2026.
- Kuehl, D.W., P.M. Cook, A.R. Batterman, D. Lothenbach and B.C. Butterworth. 1987. Bioavailability of polychlorinated dibenzo-p-dioxins and dibenzofurans from contaminated Wisconsin River sediment to carp. Chemosphere 16: 667-679.
- Landrum, P.P., B.J. Eadie and W.R. Faust. 1991. Toxicokinetics and toxicity of a mixture of sediment-associated polycyclic aromatic hydrocarbons to the amphipod *Diporeia* sp. Environmental Toxicology and Chemistry 10: 35-46.
- Long, E.R. and M.F. Buchman. 1989. An evaluation of candidate measures of biological effects for the National Status and Trends Program. NOAA Technical Memorandum NOS OMA 45. Seattle, WA. National Oceanic and Atmospheric Administration. 105 pp.
- Long, E.R. and L.G. Morgan. 1990. The Potential for Biological Effects of Sediment-sorbed Contaminants Tested in the National Status and Trends Program. NOAA Technical Memorandum NOS OMA 52. Seattle, WA. National Oceanic and Atmospheric Administration. 175 pp. + appendices.
- Malins, D.C., M.M. Krahn, M.S. Myers, L.D. Rhodes, D.W. Brown, C.A. Krone, B.B. McCain, and S.L. Chan. 1985. Toxic chemicals in sediments and biota from a creosote-polluted harbor: relationships with hepatic neoplasms and other hepatic lesions in English sole. Carcinogenesis 10: 1463-1469.
- Malins, D.C., B.B. McCain, M.S. Myers, D.W. Brown, M.M. Krahn, W.T. Roubal, M.H. Schiewe, J.T. Landahl, and S.L. Chan. 1987. Field and laboratory studies of the etiology of liver neoplasms in marine fish from Puget Sound. Environmental Health Perspectives 71: 5-16.
- McGreer, E.R. 1979. Sublethal effects of heavy metal contaminated sediments on the bivalve *Macoma balthica* (L.). Marine Pollution Bulletin 10(9): 259-262.
- Merrill, E.G. and T.L. Wade. 1985. Carbonized coal products as a source of aroma hydrocarbons to sediments from a highly industrialized estuary. Environmental Science and Technology 19: 597-603.
- Mix, M.C. 1979. Chemical carcinogens in bivalve mollusks from Oregon estuaries. EPA-600/3-79-034. Gulf Breeze, FL: U.S. Environmental Protection Agency. 33 pp.
- Mothershead, R.F. II, R.C. Hale. Influence of Ecdysis on the accumulation of polycyclic aromatic hydrocarbons in field exposed blue crabs (*Callinectes sapidus*). Gloucester Point, VA. Virginia Institute of Marine Science, College of William and Mary. 24 pp.
- Moyle, P.B. and J.J. Cech, Jr. 1982. Fishes: An introduction to Ichthyology. Prentice-Hall, Inc. New Jersey.
- Mueller, J.G., P.J. Chapman, and P.H. Pritchard. 1989. Creosote-contaminated sites: their potential for bioremediation. Environmental Science and Technology 23: 1197-1201.
- Niering, William A. 1987. Wetlands. The Audubon Society Nature Guides. Alfred A. Knopf, Inc., New York.
- Niimi, A.J. and C.Y. Choy. 1983. Laboratory and field analysis of pentachlorophenol (PCP) accumulation by salmonids. Water Research 17: 1791-1795.
- Phelps, H.L., J.T. Hardy, W.H. Pearson, and C.W. Apts. 1983. Clam burrowing behaviour: inhibition by Cu-enriched sediment. Marine Pollution Bulletin 14(12): 452-455.
- PTI. 1988a. Preliminary Literature Review of the Aquatic Transport, Fate, and Effects of Creosote; and

- Recommendations for Chemical and Biological Studies. Tech. Memo. 88-4. Seattle, Washington: National Oceanic and Atmospheric Administration.
- PTI. 1988b. The briefing report to the Environmental Protection Agency Science Advisory Board: The Apparent Effects Threshold approach. Washington, D.C.: Environmental Protection Agency.
- Qasim, S.R., A.T. Armstrong, J. Corn, and B.L. Fordan. 1980. Quality of water and bottom sediments in the Trinity River. Water Resources Bulletin 16(3): 522-531.
- Roberts, M.H., Jr., W.J. Hargis, Jr., C.J. Strobel, and P.P. DeUsle. 1989. Acute toxicity of PAH contaminated sediments to the estuarine fish, *Leiostomus xanthurus*. Bulletin of Environmental Contamination and Toxicology 42:142-149.
- Rubinstein, N.I., R.J. Pruell, B.K. Taplin, J.A. LiVolsi, and C.B. Norwood. 1990. Bioavailability of 2,3,7,8-TCDD, 2,3,7,8-TCDF and PCBs to marine benthos from Passaic River sediments. Chemosphere 20: 1097-1102.
- Schwab, Donald. 1991. Field biologist, Virginia Department of Game and Inland Fisheries, Wildlife Division. Suffolk, VA. Personal communication, February 27, 1991.
- Swartz, R.C., P.P. 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.
- Tagatz, M.E., J.M. Ivey, and M. Tobia. 1978. Effects of Dovicide G-ST on development of experimental estuarine macrobenthic communities. In: K.R. Rao, (ed.). Pentachlorophenol: chemistry, pharmacology, and environmental toxicology. pp 157-163.
- Spitzbergen *et al.*, 1991 (Page 27).
- 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.
- Tagatz, M.E., J.M. Ivey, and M. Tobia. 1978. Effects of Dovicide G-ST on development of experimental estuarine macrobenthic communities. In: K.R. Rao, (ed.). Pentachlorophenol: chemistry, pharmacology, and environmental toxicology. pp 157-163.
- Tagatz, M.E., G.R. Plaia, C.H. Deans, and E.M. Lores. 1983. Toxicity of creosote-contaminated sediment to field- and laboratory-colonized estuarine benthic communities. Environmental Toxicology and Chemistry 2: 441-450.
- Travelstead, J. 1989. Associate Commissioner for Fisheries Management, Virginia Marine Resources Commission, Personal communication, October 18, 1989.
- U.S. EPA. 1980. Ambient water quality criteria for pentachlorophenol. Washington, D.C.: U.S. Environmental Protection Agency, Office of Water Regulations and Standards, Criteria and Standards Division. EPA 440/5-80-065.
- U.S. EPA. 1985. Ambient water quality criteria for Cu - 1984. Washington, DC: U.S. Environmental Protection Agency, Office of Water Regulations and Standards, Criteria and Standards Division. EPA 440/5-84-031.
- U.S. EPA. 1986b. Health effects assessment for Cu. EPA/540/1-86-025. Cincinnati, OH: U.S. Environmental Protection Agency, Office of Research and Development, Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office.
- U.S. EPA. 1986c. Health effects assessment for pentachlorophenol. EPA/540/1-86-043. Cincinnati, OH: U.S. Environmental Protection Agency, Office of Research and Development, Office of Health and

Environmental Assessment, Environmental Criteria and Assessment Office.

U.S. EPA. 1986d. Health effects assessment for polycyclic aromatic hydrocarbons (PAHs). EPA/540/1-86-013. Cincinnati, OH: U.S. Environmental Protection Agency, Office of Research and Development, Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office.

U.S. EPA. 1986e. Health effects assessment for 2,3,7,8-TCDD. EPA/540/1-86/044. Cincinnati, OH: U.S. Environmental Protection Agency, Office of Research and Development, Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office.

U.S. EPA. 1986f. Health effects assessment for Zn (and compounds). EPA/540/1-86-048. Cincinnati, OH: U.S. Environmental Protection Agency, Office of Research and Development, Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office.

U.S. EPA. 1986g. Quality Criteria for Water. 440/5-86-001. Washington, D.C.: U.S. Environmental Protection Agency, Office of Water Regulations and Standards, Criteria and Standards Division.

U.S. EPA. 1990. Background document to the integrated risk assessment for dioxins and furans from chlorine bleaching in pulp and paper mills. Washington, D.C.: U.S. Environmental Protection Agency, Office of Toxic Substances. EPA 560/5-90-014.

U.S. Fish and Wildlife Service. 1980. Atlantic Coast Ecological Inventory Map. Norfolk, VA. 36076-A1-E1-250.

U.S. Fish and Wildlife Service. 1989.

U.S. Forest Service. 1988. Final Environmental Impact Statement: Vegetation Management in the Coastal Plain/Piedmont, Volume II. Appendix A: Risk assessment for the use of herbicides in the southern region, United States Department of Agriculture, Forest Service.

Van Veld, P.A., D.J. Westbrook, B.R. Woodin, R.C. Hale, C.L. Smith, R.J. Huggett and J.L. Stegeman. 1990. Induced cytochrome *P*-450 in intestine and liver of spot (*Leiostomus xanthurus*) from a polycyclic aromatic hydrocarbon contaminated environment. Aquatic Toxicology 17: 119-132.

Varanasi, U., W.L. Reichert, J.E. Stein, D.W. Brown, and H.R. Sanborn. 1985. Bioavailability and biotransformation of aromatic hydrocarbons in benthic organisms exposed to sediment from an urban estuary. Environmental Science and Technology 19: 836-841.

Varanasi, U., J.E. Stein, and M. Nishiraoto. 1989. Biotransformation and disposition of polycyclic aromatic hydrocarbons (PAH) in fish. In: U. Varanasi (ed.), *Metabolism of polycyclic aromatic hydrocarbons in the aquatic environment*, p. 93-149. CRC Press, Inc., Boca Raton, FL.

Verma, S., S. Rani, and R. Dalela. 1981. Synergism, antagonism, and additivity of phenol, pentachlorophenol, and dinitrophenol to a fish (*Notopterus notopterus*). Archives of Environmental Contamination and Toxicology 10: 365-370.

Virginia Department of Conservation and Recreation, 1991. Letter communication with Kennedy H Clark, Environmental Review Coordinator. March 6, 1991. Richmond, VA.

Virginia Institute of Marine Sciences (VIMS). 1989. Trawl survey database for juvenile fishes: 1975-1985, Gloucester Point, VA.

Vogelbein, W.K., J.W. Fournie, P.A. Van Veld, and R.J. Huggett. 1990. Hepatic neoplasms in the mummichog *Fundulus heteroclitus* from a creosote-contaminated site. Cancer Research 50: 5978-5986.

Walker *et al.*, 1991 (Page 27)

Weeks, B.A. and J.E. Warinner. 1984. Effects of toxic chemicals on macrophage phagocytosis in two estuarine fishes. Marine Environmental Research 14: 327-335.

Weeks, B.A. and J.E. Warinner. 1986. Functional evaluation of macrophages in fish from a polluted estuary. Vetinary Immunology and Immunopathology 12: 313-320.

Weeks, B.A., J.E. Warinner, P.L. Mason, and D.S. McGinnis. 1986. Influence of toxic chemicals on the chemotactic response of fish macrophages. Journal of Fish Biology 28: 653-658.

Wright, M., Sanitarian Manager. Virginia Department of Shellfish Sanitation, Richmond, VA, Personal communication, January 10, 1991.

Zieske, G. 1991. Atlantic Wood Industries, Inc. Portsmouth, Virginia NPDES Biomonitoring Program, #VA0004189, Outfalls #001, #002, and #002, 48-hour static acute definitive toxicity bioassays using *Mysidopsis bahia* and *Cyprinodon variegatus* August 21-23m 1991. Presented Savannah Laboratories and Environmental Services, Inc. Gainesville, FL. Alvarez, Lehman and Associates, Inc. 45 pp.