Stratus Consulting

Creosote-Treated Wood in Aquatic Environments: Technical Review and Use Recommendations

Prepared for:

Joe Dillon
NOAA Fisheries
Southwest Division
Habitat Conservation Division
777 Sonoma Avenue, Suite 325
Santa Rosa, CA 95404

Prepared by:

Stratus Consulting Inc.
PO Box 4059
Boulder, CO 80306-4059
(303) 381-8000
–with–
Duke University
Durham, NC

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Acronyms and Abbreviations

AAC	alkyl ammonium compound
AET	Apparent Effects Threshold
AHR	aryl hydrocarbon receptor
amu	atomic mass unit
AWPA	American Wood-Preservers' Association
BaP	benzo[*a*]pyrene
BCFs	bioconcentration factors
BMPs	best management practices
BPDE	benzopyrene diol epoxide
CCC	California Coastal Commission
CPF	chlorpyrifos
CRMP	Coastal Resources Management Program
CTL	chlorothalonil
Cu8	oxine copper
CuN, CuNapth	copper naphthanate
CYP	cytochrome P450
DCOI	4,5-dichloro-2-n-octyl-4-isothiozolin-3-one
DMBA	7,12-dimethylbenzanthrace
EECs	estimated environmental concentrations
EFH	Essential Fish Habitat
ER-L	Effects Range – Low
ER-M	Effects Range – Median
EROD	ethoxyresorufin O-deeethylase
ESA	Endangered Species Act
ESUs	Evolutionarily Significant Units
FCA	foci of cellular alteration
FID	flame ionization detector
FIFRA	Federal Insecticide, Fungicide, and Rodenticide Act
GC	gas chromatography
GENEEC	Generic Expected Environmental Concentrations

HAPC	habitat areas of particular concern
HPAH	heavy PAH
IPBC	3-iodo-2-propynyl butyl carbamate
ISQG	Interim Sediment Quality Guideline
ISW	interstitial water
LOEC	lowest observable effects concentration
LPAH	light PAH
MSA	Magnuson-Stevens Act
MW	molecular weight
NAVSTA	Naval Station San Diego
NMFS	National Marine Fisheries Service
NOAA	National Oceanic and Atmospheric Administration
NOEC	no observable effects concentration
NOEL	no observable effects level
NPS	National Park Service
OC	organic carbon
PAHs	polycyclic aromatic hydrocarbons
pcf	pounds per cubic foot
PCP	pentachlorophenol
PEC	pigment-emulsified creosote
PEL	Probable Effect Level
Penta	pentachlorophenol
ppm	parts per million
ppt	parts per thousand
PPZ	propiconazole
RPD	redox potential discontinuity
RQs	risk quotients
SPMDs	semipermeable membrane sampling devices
SPME	solid-phase microextraction
SQGs	sediment quality guidelines
SQuiRTs	Screening Quick Reference Tables

T&E	threatened and endangered
TCDD	2,3,7,8-tetrachlorodibenzo-*p*-dioxin
TEB	tebuconazole
TEL	Toxicity Effect Level
TOC	total organic carbon
TPAH	total PAH
TU	toxic unit
U.S. EPA	U.S. Environmental Protection Agency
USFS	USDA Forest Service
USFWS	U.S. Fish and Wildlife Service
UV	ultraviolet
WHO	World Health Organization
WSF	water-soluble fraction
WWPI	Western Wood Preservers Institute

1. Introduction

1.1 Background and Report Organization

Wood is a common construction material used for bridges, docks, piers, and other submerged and overwater structures. Wood is subject to fungal decay and to attack by wood boring organisms, especially in saltwater and estuarine environments. To reduce the incidence of decay and attack, chemicals are impregnated into wood used for submerged and near-water construction. Wood-treating chemicals, which include a wide array of organic and inorganic chemicals, can leach from the wood into the immediate aquatic environment, potentially harming aquatic biota.

The National Marine Fisheries Service (NMFS) of the National Oceanic and Atmospheric Administration (NOAA) is developing guidance on the use of treated wood in aquatic environments inhabited by NMFS trust resources. NMFS trust resources include commercially important marine species and their habitats, as well as threatened and endangered (T&E) marine species and their habitats. The NMFS provides review and consultation on marine, estuarine, and freshwater construction projects that potentially could impact trust resources. Federal and state agencies and industry have requested guidelines from the NMFS on the use of construction materials, including treated lumber, in aquatic environments in the Pacific coastal region.

The purpose of this report is to assist the NMFS with the development of these guidelines. Data and information are reviewed to evaluate potential hazards to aquatic organisms from treated wood in aquatic environments. The data and information review focused specifically on the Pacific Coast states of California, Oregon, Washington, and Alaska. This report is a companion to "Treated Wood in Aquatic Environments: Technical Review and Use Recommendations" (Stratus Consulting and Paladin Water Quality Consulting, 2005). That report describes water-soluble wood treatments; this report describes creosote treatments. The two reports share a similar introduction and overall structure; however, the other report includes separate chapters about alternative materials and current regulations and best management practices (BMPs) that are covered more briefly in the introduction of this report.

In the following sections, we describe NMFS trust resources, types of oil-borne wood preservatives, the chemical composition of creosote, and creosote policies, regulations, and BMPs.

The remainder of this report is organized as follows. In Chapter 2, we discuss data and information regarding leaching of creosote from treated wood into aquatic environments, and the potential for exposure of aquatic organisms to leached creosote. In Chapter 3, we discuss the

toxicity of the leached creosote compounds to aquatic biota. In Chapter 4, we discuss potential risks to NMFS trust resources, including recommendations to minimize the environmental risks of toxic chemicals in aquatic environments. Literature cited follows Chapter 4.

1.2 Trust Resources

Under the Magnuson-Stevens Act (MSA), sections 303(a)(7) and 305(b)(2), the NMFS is responsible for managing commercially harvested aquatic species (including several salmonid species) by, among other things, implementing fishery management plans and designating protective Essential Fish Habitat (EFH) areas. The fishery management plans for commercially important species are managed by regional fisheries management councils. The Pacific Fisheries Management Council manages commercially important species for the states of California, Oregon, and Washington. The Northern Pacific Fisheries Management Council manages commercially important species for the state of Alaska.

The fishery management plans must designate both the habitat essential to the commercial species of concern and the threats to their habitat from fishing and non-fishing activities. EFH includes, as defined by Congress, ". . . those waters and substrate necessary to fish for spawning, breeding, feeding, or growth to maturity." EFH guidelines at 50 CFR 600.10 also specifically define substrate as including, ". . . associated biological communities." This is interpreted to mean all organisms (and particularly prey organisms) belonging to the same food web as any of the trust species. Salmonid EFH designated in accordance with the MSA includes all streams, lakes, and other water bodies currently or historically accessible to salmon in Alaska, Washington, Oregon, and California, and includes most Pacific Coast rivers, streams, and estuaries. In addition to EFH, which is geographically broad, NMFS may designate habitat areas of particular concern (HAPC) for the protection of the commercially important species it manages.

Chinook salmon (*Oncorhynchus tshawytscha*), coho salmon (*Oncorhynchus kisutch*), and pink salmon (*Oncorhynchus gorbuscha*) are the three main commercially significant salmon species managed under the MSA by the North Pacific and Pacific Fishery Management Councils. EFH for these species in marine and estuarine areas of the Pacific Coast region extends seaward from the shoreline out to the 200 mile limit of the U.S. Exclusive Economic Zone. Shoreward, salmonid EFH comprises all bodies of water extending inland that were historically accessible to salmon, with the exception of certain barriers and dams that fish cannot pass (PFMC, 2004). Chinook salmon habitat spans from the U.S.-Mexico border to Kotzebue Sound in northwestern Alaska. Coho salmon spawn in tributaries from the San Lorenzo River in Monterey Bay, California, to Point Hope, Alaska, and throughout the Aleutian Islands (PFMC, 2003).

Under the Endangered Species Act (ESA), NMFS' trust resources include T&E aquatic species. In addition to the MSA mandated habitat protections, sections 3(5)(A) and 7 of the ESA require NMFS to conserve the ecosystems on which T&E species depend, to provide a program for the conservation of T&E species, and to ensure that they (and all federal agencies) do not fund, authorize, or carry out any actions that will harm the habitat or jeopardize the continued existence of listed species. To this end, NMFS is authorized to designate "critical habitat" for those species. Under ESA section 7(a)(2), NMFS is responsible for developing guidelines and policies to protect federally listed T&E aquatic organisms and their habitats from pollutants.

There are 1,290 species, subspecies, Distinct Population Segments, and Evolutionarily Significant Units (ESUs) listed under the ESA. Of the aquatic species, the NMFS Office of Protected Resources manages mostly marine and anadromous species. The U.S. Fish and Wildlife Service (USFWS) manages the remainder of the listed species, which are primarily terrestrial and freshwater species. The NMFS Office of Protected Resources manages 61 ESA-listed aquatic species, 43 aquatic species of concern, and approximately 175 marine mammal stocks listed under the Marine Mammal Protection Act. Of the 51 salmonid ESUs, 30 are either listed as threatened or endangered, or are candidates for listing (Table 1.1).

Table 1.1. Status of West Coast salmonid species and ESUs

Species	Evolutionary significant unit (ESU)[a]	Listing status[b]	T/E status
Pink salmon	Even year ESU[c]	NW	
	Odd year ESU[c]	NW	
Coho salmon	Central CA ESU	L	E
	Southern OR/northern CA coasts ESU	L	T
	OR coast ESU	L	T
	Puget Sound/Strait of Georgia ESU	C	
	Lower Columbia River ESU	C	
	Olympic Peninsula ESU	NW	
	Southwest Washington	NW	
Chinook salmon	Sacramento River winter-run ESU	L	E
	Snake River fall-run ESU	L	T
	Snake River spring/summer-run ESU	L	T
	Puget Sound ESU	L	T
	Lower Columbia River ESU	L	T

Table 1.1. Status of West Coast salmonid species and ESUs (cont.)

Species	Evolutionary significant unit (ESU)[a]	Listing status[b]	T/E status
Chinook salmon (cont.)	Upper Willamette River ESU	L	T
	Upper Columbia River spring-run ESU	L	E
	Central Valley spring-run ESU	L	T
	CA coastal ESU	L	T
	Central Valley fall and late fall-run ESU	C	
	Upper Klamath-Trinity rivers ESU	NW	
	OR coast ESU	NW	
	WA coast ESU	NW	
	Mid-Columbia River spring-run ESU	NW	
	Upper Columbia River summer/fall-run ESU	NW	
	Southern OR/northern CA coasts ESU	NW	
	Deschutes River summer/fall-run ESU	NW	
Chum salmon	Hood Canal summer-run ESU	L	T
	Columbia River ESU	L	T
	Puget Sound/Strait of Georgia ESU	NW	
	Pacific Coast ESU	NW	
Sockeye salmon	Snake River ESU	L	E
	Ozette Lake ESU	L	T
	Baker River ESU	NW	
	Okanogan River ESU	NW	
	Lake Wenatchee ESU	NW	
	Quinault Lake ESU	NW	
	Lake Pleasant ESU	NW	
Steelhead	Southern CA ESU	L	E
	South-Central CA coast ESU	L	T
	Central CA coast ESU	L	T
	Upper Columbia River ESU	L	E
	Snake River Basin ESU	L	T
	Lower Columbia River ESU	L	T
	CA Central Valley ESU	L	T
	Upper Willamette ESU	L	T

Table 1.1. Status of West Coast salmonid species and ESUs (cont.)

Species	Evolutionary significant unit (ESU)[a]	Listing status[b]	T/E status
Steelhead (cont.)	Middle Columbia River ESU	L	T
	Northern CA ESU	L	T
	OR coast ESU	C	
	Southwest WA ESU	NW	
	Olympic Peninsula ESU	NW	
	Puget Sound ESU	NW	
	Klamath Mountains Province ESU	NW	

a. L = listed, C = candidate, NW = not warranted.
b. E = endangered, T = threatened.
c. Managed by NMFS every other year (jointly with Canada).
Source: NOAA, 2005.

1.3 Types of Oil-borne Wood Preservatives

Treated wood pilings, timbers, and other wooden lumber have been used in marine construction in the United States for more than a hundred years (Lebow and Tippie, 2001). Although some woods are more naturally resistant to deterioration, wood construction materials exposed to water must be preserved with chemicals to prevent deterioration and eventual destruction by marine borers such as crustaceans (gribbles, *Limnaria* spp.), mollusks (boring clams, *Teredo* or *Bankia* spp.), and other wood degrading organisms, including fungi. To protect wood from these organisms, preservative formulations must be toxic to the wood-degrading organisms.

Oil-borne wood treatments include creosote, creosote mixed with coal tar or petroleum, and other preservatives such as pentachlorophenol (PCP) and copper naphthanate (CuN) (Table 1.2) (Hutton and Samis, 2000; AWPA, 2003). Creosote is the most commonly used wood preservative worldwide, and comprises nearly 15% of the total volume of wood treatment preservatives used in the United States (Crawford et al., 2000). PCP is not resistant to marine borers, and therefore is only recommended for pilings in freshwater or in saltwater splash zones. CuN currently is not recommended for use in either freshwater or saltwater. The remainder of the P8 preservatives listed in Table 1.2 are used so infrequently that they are not listed in the BMPs for oil-borne preservatives (Hutton and Samis, 2000; Lebow and Tippie, 2001; WWPI, 2002b). Therefore, in this report we confine our analysis of oil-borne preservatives to creosote.

Table 1.2. Coal-derived and oil-borne wood preservatives in the United States

Type of preservative	AWPA standard	Components
Creosote	P1/P13	Coal tar distillate
Creosote solution	P2	Mixture of creosote and coal tar
Creosote-petroleum solution	P3	Mixture of creosote and petroleum, comprising at least 50% creosote
Other oil-borne preservatives	P8	Pentachlorophenol (PCP or Penta) Copper naphthenate (CuN or CuNapth) Oxine copper (copper-8-quinolinolate or Cu8) Alkyl ammonium compound (AAC) 4,5-dichloro-2-n-octyl-4-isothiozolin-3-one (DCOI) 3-iodo-2-propynyl butyl carbamate (IPBC) Chlorothalonil (CTL) Tebuconazole (TEB) Propiconazole (PPZ) Chlorpyrifos (CPF)

Sources: AWPA, 2003; Dickey, 2003.

The American Wood-Preservers' Association (AWPA) currently approves only the P1/P13 creosote standard (AWPA, 2003). The term creosote in this report refers specifically to the AWPA-recognized P1/P13 creosote standard. Australia has a standard for pigment-emulsified creosote (PEC), which it claims does not leach from treated wood (Crawford et al., 2000). Because this compound is not available in the United States, we have excluded it from our analysis.

1.4 Creosote Composition

Creosote is a distillate of coal tar, and its chemical composition varies depending on the source of the coal tar and the distillation conditions and fraction removed. The World Health Organization (WHO, 2004) concluded that there might be 1,000 compounds present in a typical coal tar creosote mixture, though most of them are present in minute quantities. Creosote compounds can be distributed among several chemical classes, including polycyclic aromatic hydrocarbons (PAHs), alkyl-PAHs, tar acids/phenolics, tar bases/N-heterocyclics (quinolines and carbazoles), S-heterocyclics (thiophenes), O-heterocyclics/furans (dibenzofuran), and aromatic amines (such as aniline). A detailed discussion of the physical properties and chemical structures of these compounds is beyond the scope of this document. See WHO (2004) and Eisler (2000) for more details.

Several studies have included summaries of creosote compositions, including Ingram et al. (1982), Cooper (1991), U.S. EPA (2003b), and WHO (2004). WHO (2004) includes creosote compositional analyses from eight separate studies, including creosotes from the United States, Britain, Germany, and the former Soviet Union.

Table 1.3 contains summary statistics for the more prominent chemical compounds in creosote from each of the above sources. On average, the compounds shown in Table 1.3 comprise roughly two-thirds of creosote. The remaining one-third includes hundreds of other compounds, each comprising less than 1% of the total mixture.

Table 1.3. Summary statistics for major compounds in creosote (by percent)

Class	Compound	n	Max	Min	Mean
PAHs	Phenanthrene	9	21.0	6.7	13.3
	Naphthalene	10	15.5	1.3	9.1
	Acenaphthene	10	14.7	3.1	8.4
	Fluorene	10	10.0	3.1	6.3
	2-methylnaphthalene	9	12.0	1.2	5.6
	Fluoranthene	9	10.0	2.3	5.3
	1-methylnaphthalene	8	14.5	0.9	4.4
	Pyrene	10	8.5	1.1	4.3
	Anthracene	8	8.2	0.8	3.3
	Chrysene	9	6.1	0.1	1.9
Phenolics	Phenol	3	0.6	0.2	0.3
	Cresols	3	2.3	0.3	1.2
O-heterocyclics/furans	Dibenzofuran	9	7.5	1.1	4.7
N-heterocyclics	Quinoline	6	2.0	0.6	1.0
	Carbazole	6	3.9	0.2	1.6
S-heterocyclics	Benzothiophene	4	0.5	0.3	0.4

Sources: Ingram et al., 1982; Cooper, 1991; U.S. EPA, 2003b; WHO, 2004.

The data in Table 1.3 show a wide range in composition for many compounds, depending on the source of the creosote. Xiao et al. (2002), and the U.S. Environmental Protection Agency (U.S. EPA, 2003b) cite separate studies that list creosote composition as typically 85% PAHs, 10% phenolic compounds, and 5% heterocyclics. However, of the 10 creosote compositions that are included in Table 1.3, the greatest total of phenolic compounds (sum of phenol, 2,4-dimethylphenol, and cresols) is 3.5% (WHO, 2004).

PAHs are by far the most common compounds in creosote (Table 1.3). In addition to the PAHs listed in Table 1.3, high molecular weight PAHs such as benzo[*a*]pyrene (BaP), benz[a]anthracene, and benzo[b]fluoranthene are some of the more common compounds in creosote not listed in Table 1.3 (U.S. EPA, 2003b; WHO, 2004).

Based on creosote industry data, U.S. EPA (2003b) lists the top 17 aromatic hydrocarbons typically in creosote. These include the 10 PAHs in Table 1.3 [which comprise 80% of creosote in the U.S. EPA (2003b) example], plus biphenyl, 2,3-dimethyl naphthalene, 2,6-dimethyl naphthalene, 2-methyl anthracene, anthraquinone, 2,3-benzo(b)fluorene, and BaP. The U.S. EPA notes that 16 of the 17 compounds are on the U.S. EPA's List of Priority Pollutants, pursuant to the Clean Water Act.

The U.S. EPA has classified seven PAHs as Group B2 – probable human carcinogens: BaP, benz[a]anthracene, chrysene, benzo[b]fluoranthene, benzo[k]fluoranthene, dibenz[a,h]anthracene, and indeno[1,2,3-cd]pyrene. Most of these have been identified in creosote, including BaP, the most studied PAH in terms of carcinogenicity (WHO, 2004). In addition, of U.S. EPA's 31 priority chemicals, eight are PAHs: acenaphthene, acenaphthylene, anthracene, benzo[g,h,i]perylene, fluorene, naphthalene, phenanthrene, and pyrene (U.S. EPA, 2005).

Many studies of PAHs and creosote compounds in aquatic and marine environments distinguish between light PAHs (LPAHs) and heavy PAHs (HPAHs). Generally, PAHs with two or three aromatic rings are denoted LPAH, while PAHs containing greater than or equal to four rings are termed HPAH. All U.S. EPA Group B2 probable human carcinogens are HPAH, while all but two (benzo[g,h,i]perylene and pyrene) of the eight U.S. EPA priority chemical PAHs are LPAH. This distinction becomes important in later discussions of creosote-related environmental fates and toxicities. When considering the environmental impacts and toxicity of leached creosote in this report, we often will refer to the contaminants in leachate as "PAHs" for simplicity, though we in fact mean "PAHs, phenolics, heterocyclics, and other contaminants."

1.5 Creosote Regulations and Policies

Both the European Union and the United Kingdom have banned all nonprofessional use of creosote (HSE, 2005). The U.S. EPA published a risk assessment, described below, that will be used by the agency to decide whether creosote will be re-registered as a pesticide. Meanwhile, in the absence of federal guidelines, many states and local agencies have implemented their own regulations. A thorough review of creosote regulations and policies is beyond the scope of this document. However, Table 1.4 provides some examples of creosote regulations and policies that have been enacted in the past few years and demonstrates the consistency of approaches toward creosote use in aquatic environments.

Table 1.4. Examples of creosote regulations and policies as of summer 2005

Management entity	Regulation/action	Source
Washington State Ferries	Washington State Ferries concluded that creosote-treated timbers were significantly degrading water quality in Puget Sound, and they commenced a large-scale project to replace all creosote timbers in the ferry system. This project has been ongoing since 2000. By 2012, Washington State Ferries will have replaced over 15 million board-feet of creosote timbers in Puget Sound.	1
Port of Port Angeles, Washington	In 2004, Port Angeles instituted prohibitions on the installation of creosote-treated timbers in waters under their jurisdiction.	2
Oregon Dept. of Environmental Quality, State Marine Board	BMPs for recreational boating facilities state that creosote-treated wood should be avoided, and existing creosote-treated wood pilings should be removed.	3
California Coastal Commission (CCC)	The CCC originally recommended that creosote-treated pilings be wrapped in plastic to prevent leaching of creosote into water. After discovering that the plastic wrap tears readily, they recommended that plastic pilings should be used.	4
U.S. Army Corps of Engineers, Los Angeles District	Standard permit conditions restricting the use of creosote-treated wood, and requiring maintained isolation of creosote via plastic wrappings.	5
Delaware Dept. of Natural Resources and Environmental Control	Delaware banned creosote-treated timbers for boat docks in the early 1990s.	6
New York State legislature	In 2004, the legislature passed S04975 to phase out the manufacture, sale, and use of creosote in the state. Gov. Pataki vetoed the bill.	7, 8
Rhode Island Coastal Resources Management Program (CRMP)	CRMP "Red Book" of regulations specifies that no residential docks, piers, or floats may be constructed of creosote-treated timbers, and creosote may not be used as a wood preservative on wetland boardwalks.	9

1. WSDOT, 2005.
2. PPF, 2004.
3. Oregon DEQ, 2002.
4. CCC, 2003.
5. Personal communication, D.J. Castanon, U.S. Army Corps of Engineers, Los Angeles District, November 30, 2004.
6. Delaware Department of Natural Resources and Environmental Control, 1992.
7. Pesticide.Net, 2004.
8. Online Lawyer Source, 2004.
9. RI CRMC, 2005.

In 2003, the U.S. EPA performed a data review and risk assessment on creosote as part of the creosote re-registration process (U.S. EPA, 2003a). The studies they reviewed did not meet the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) guidelines, specific creosote compositions were not described in the studies, and many of the studies examined the fate of specific PAHs rather than creosote as a whole. Based on these limited data, the U.S. EPA calculated risk quotients (RQs) for acute and chronic effects on fish using their Generic Expected Environmental Concentrations (GENEEC) computer model to create estimated environmental concentrations (EECs) of creosote compounds. The U.S. EPA's (2003a) conclusions were as follows:

> The Agency has concluded that risk to birds and terrestrial mammals is probably minimal, due to lack of exposure and the ability of these organisms to avoid creosote. Risk to terrestrial plants would also be considered minimal due to lack of exposure. However, risk to freshwater and marine/estuarine aquatic organisms is harder to quantitate using these data. Certainly there will be some exposure due to leaching from the treated wood into the aquatic environment; however, determining the amount of exposure and the amount of toxicity due to this exposure is difficult using the data at hand. The RQ values calculated with the available data do not demonstrate a concern for acute effects on aquatic organisms or chronic effects on freshwater fish. However, the EECs were calculated for the component PAHs, while the aquatic toxicity data were generated using whole creosote. The available data found in the open literature were not adequate to supply the information needed to assess chronic effects to freshwater invertebrates or to marine/estuarine aquatic organisms. It is not possible, therefore, to determine the chronic risk creosote may present to freshwater invertebrates and marine/estuarine aquatic organisms, including endangered species. However, the data indicate that creosote does not exceed the level of concern for acute toxicity to fish and aquatic invertebrates or for chronic toxicity to freshwater fish.

According to the U.S. EPA re-registration schedule, the U.S. EPA is expected to make a decision on the re-registration of creosote by June 2006. As detailed in Chapters 2-4 of this report, the conditions under which toxic constituents of creosote can reach marine resources are common enough that U.S. EPA's conclusions may benefit from greater emphasis on the known transport and toxicity of toxic constituents, rather than on the uncertainties. U.S. EPA's description of some of the uncertainties is valid, but might better fit in a standard characterization of uncertainties in a more definitive finding regarding the risks that creosote constituents pose to elements of the marine environment.

1.6 Creosote BMPs

The AWPA and the Western Wood Preservers Institute (WWPI) maintain BMPs for creosote-treated wood (WWPI and Canadian Institute of Treated Wood, 1996; WWPI, 2002b; AWPA, 2003). Most other sources of creosote treatment BMPs (e.g., Hutton and Samis, 2000; Lebow and Tippie, 2001; WDNR, 2002) reference the AWPA and/or the WWPI. The BMPs are quite detailed, with different BMPs for different tree species and different creosote mixtures. These BMPs are readily available from AWPA and WWPI and therefore will not be described here. For the purposes of this report, we attempt to call attention to laboratory and field leaching studies where BMPs are not followed. Most researches specify when they are not following BMPs in their leaching tests, particularly as they pertain to creosote retention in the timber. However, verifying that every leaching study that we reviewed followed BMPs was not possible.

2. Models of PAH Leaching from Treated Wood and Environmental Exposure

In this chapter we review and evaluate models that have been developed to predict the leaching of creosote constituents, primarily PAHs, from creosote-treated wood and the resultant concentrations in the environment. The rate and amount of PAHs that leach from treated wood is a key component in the evaluation of the potential effects of creosote-treated wood on aquatic biota, and much study has been conducted in this area. Nearly all studies of leaching from creosote-treated wood have been conducted in the laboratory under controlled conditions. The leaching models that have been developed predict PAH leaching under such controlled conditions. Estimating the environmental concentrations that result from the leaching is a second component in evaluating potential effects on aquatic biota. Few field and laboratory studies address this component, but two transport models have been developed to predict concentrations of PAHs in surface water and sediments around creosote-treated piling, based on modeled leaching rates.

Our review focuses primarily on PAHs, but where their leaching characteristics have been studied, N-heterocycles are also discussed. In Section 2.1, we discuss factors that affect PAH leaching rates from creosote-treated wood. In Section 2.2, we discuss leaching models that have been developed and applied, in Section 2.3, we discuss predictions of environmental concentrations of PAHs resulting from the use of creosote-treated wood, and in Section 2.4, we present conclusions.

2.1 Factors that Affect PAH Leaching from Treated Wood

The rate at which PAHs leach from treated wood is a complex function of many factors, including the nature of the wood, the treatment solution and method, and various environmental variables. In this section, information on factors that affect PAH leaching rates from treated wood is presented and summarized as a prelude to the description of the PAH leaching models contained in Section 2.2. In Section 2.1.1, specific laboratory and field studies on PAH leaching rates and the variables that can affect them are presented. In Section 2.1.2, review articles and other more general information on factors that affect PAH leaching from treated wood are summarized.

2.1.1 Laboratory observations of PAH leaching from creosote-treated wood immersed in water

Ingram et al. (1982)

Study description

Ingram et al. (1982) measured leaching of creosote from dual-treated southern pine pilings immersed in freshwater and seawater. The authors quantified the effects of water temperature, piling age, and exposure time on leaching rates.

The test pilings were treated to a preservative retention of 354 to 378 kg/m^3, and then aged for 6 months in open air. Leaching from the recently treated pilings was compared to leaching from dual-treated pilings that had been in seawater for approximately 12 years off Key West, Florida. Preservative retention in the aged sections ranged from 442 to 596 kg/m^3. Before testing, the freshly cut ends of all piling sections were coated with epoxy resin.

Water concentrations of 16 PAHs and dibenzofuran were measured. The tests were conducted in large (300 gallon steel tanks) and small (4 liter glass beakers) vessels. In both tests, the water in test vessels was stirred continuously. The focus of the small vessel studies was to compare leaching from recently treated wood in freshwater (distilled water), aged wood in freshwater, and aged wood in seawater. Water temperatures were held at 20ºC, 30ºC, or 40ºC. Test duration was 30 days. For the large vessel studies, pilings were placed in 200 to 250 gallons of seawater for 12-21 days. Water temperature was controlled to between 18ºC and 21ºC. Two-liter samples were removed daily, and analyzed by gas chromatography.

Temperature effects on leaching

Leaching rates in freshwater and saltwater increased with increasing water temperature (Table 2.1), with slopes of 1.5 and 1.7, respectively (Figure 2.1). The rates presented in Table 2.1 are an average of leaching over the first three days following immersion. Vertical bars in Figure 2.1 show the range of the leaching rates measured. At each temperature tested, total PAH leaching (the sum of all compounds measured) was higher in freshwater than in saltwater. Ranges of water concentrations are reported in the paper for each of the 16 PAHs plus dibenzofuran, and leaching was generally more rapid for the more water soluble compounds.

Table 2.1. Temperature and salinity effects on PAH leaching rates

Temperature (ºC)	Salinity (ppt)	Minimum (µg/cm^2 per d)	Maximum (µg/cm^2 per d)
20	0	26.6	35.9
30	0	39.4	56.9
40	0	52.5	70.2
20	30	7.94	7.94
30	30	14.7	27.7
40	30	36.6	47.9

Source: Ingram et al., 1982.

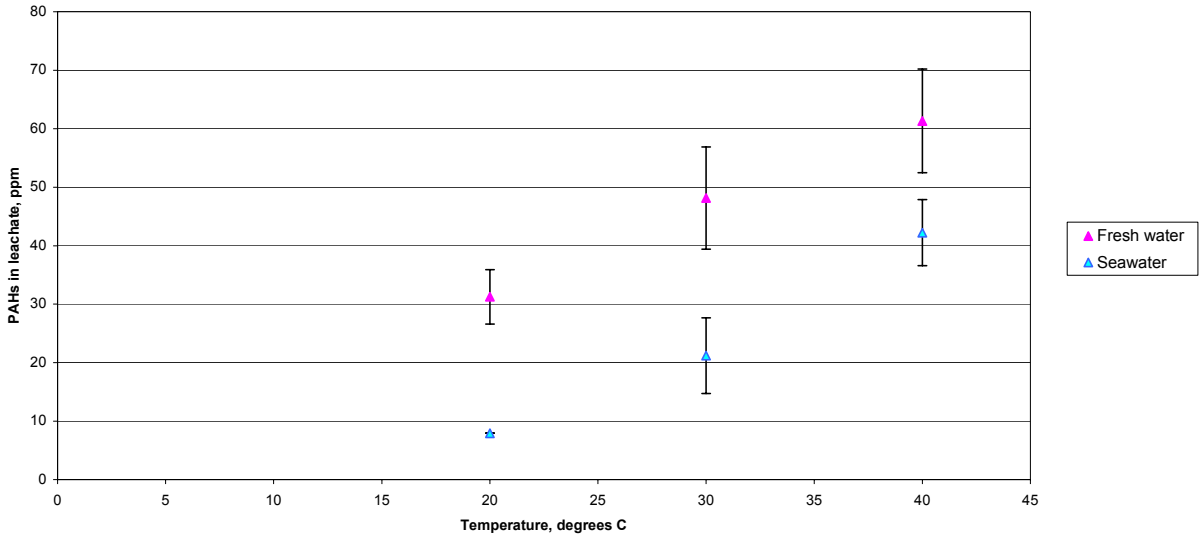

Figure 2.1. Effects of temperature and salinity on leaching.

Source: Ingram et al., 1982.

Aging effects on leaching

Leaching of PAHs to seawater over 30 days was greater from recently treated pilings than from aged pilings. Final concentrations of PAHs in seawater were approximately 750 µg/L for the aged wood and 1,000 µg/L for the recently treated wood (as estimated from graphs presented in Ingram et al., 1982). The 12 years of field installation in seawater appeared to have reduced leaching rates by only about 25%. This reduction is smaller than most model predictions suggest (see Section 2.3). Since the sample ends were sealed, leaching from the freshly cut portions should not have been a factor in the leaching rates. However, the aged and recently treated samples probably differed in other respects, including creosote formulation, initial retention, treatment method, post-treatment processing and storage, wood density, and possibly, wood species. The results may also be influenced by the test method: the water was stirred but not replaced, so increasing concentrations in the water over time might have limited leaching and diminished differences between the sample types.

PAH concentrations over time

Ingram et al. (1982) observed a decline in the concentration of PAHs in the large test tank over the 288-hour run of their "long-term" leaching experiment. In this study, concentrations leached from recently treated wood peaked at 432 ppb after 72 hours, and declined to 156 ppb after 288 hours. PAH concentrations in the tank containing aged wood declined even more. The aged samples might have contained microbes that degrade PAHs. Therefore, the reported PAH leaching rates might underestimate either the total amount of leaching or the leaching rate. Furthermore, the tanks and beakers were not sealed, and the loss of volatile PAHs such as naphthalenes cannot be ruled out.

Kang et al. (2003)

Study description

Kang et al. (2003) determined leaching rates of individual PAHs at two flow rates (1.2 cm/sec, and 3.3 cm/sec). Samples of Douglas fir lumber were treated with P1/P13 creosote to a retention of 12 pcf, in compliance with the WWPI's BMPs. Freshly cut ends were sealed with epoxy. The samples were completely immersed in carbon-filtered tap water held at approximately 12°C. Water samples for PAH analysis were collected periodically throughout the 14 day test.

Effect of flow rate on leaching

Initial loss rates at 3.3 cm/sec were at least double, and, often, substantially more than double, the loss rates for the same compounds at 1.2 cm/sec.

Variations between individual PAHs

Leaching rates at the two flow rates over the 14 day test were reported for naphthalene, acenaphthene, fluorene, phenanthrene, anthracene, fluoranthene, and pyrene. As observed in several other studies, phenanthrene appeared to leach at a higher rate than predicted by its water solubility, but the remaining compounds tended to leach in a more predictable manner based on water solubilities.

Non-detected compounds

HPAH compounds were not detected in the leachate at any time during the test. Either these compounds did not leach from the wood, or they were not detected because of some deficiency of the analytical method, or they were lost through volatilization, biodegradation, sorption to organic matter, or other route. HPAH compounds are routinely detected in similar tests, so it is unlikely that they did not leach from the samples in this test. Because of their low vapor pressures, it is also unlikely that they were lost though volatilization.

Naphthalene was lost at a high rate initially, but concentrations declined to below detection after four days. No PAHs were detected after seven days. The decline and lack of detection may reflect biodegradation, as suggested in Ingram et al. (1982), or methodological problems, as suggested above.

Becker et al. (2001)

Study description

Becker et al. (2001) compared leaching of creosote compounds in three water preparations. Samples comprised 5 mm diameter borings of *Pina nigra*, cut to 10 mm lengths. Samples (10 g each) were immersed in 100 mL of deionized filtered water, deionized water buffered to pH 4.7, or a solution containing humic substances. The water was stirred continuously during the 120 hour test. Water changes were performed at 24 and 48 hours.

Effect of water chemistry on leaching

The leaching rates of most of the creosote compounds measured were highest in deionized water. Leaching rates of N-heterocyclic compounds were greater than leaching rates of PAHs. The loss rates of N-heterocycles declined rapidly with time, but still exceeded the loss rates of the PAHs by approximately an order of magnitude at the end of the study. N-heterocycles are susceptible to protonation, unlike homocyclic PAHs. Even partial protonation tends to increase the water solubility of a compound, making it more available for leaching (Schwarzenbach, 1993, as cited in Becker et al., 2001), and particularly in low pH water.

Although this study showed high leaching of N-heterocycles (in apparent contrast to other study results), most of the other researchers did not analyze for these compounds. In addition, because this study used small wood samples with a higher surface area to volume ratio, which increases the amount of leaching per volume, the results are not directly comparable to studies performed with larger samples of wood.

Xiao et al. (2000)

Study description

Xiao et al. (2000) treated Douglas fir samples with P1/P13 creosote in accordance with WWPI's BMPs. Freshly cut ends were sealed with epoxy, and the samples were wrapped in plastic to reduce volatilization losses between treatment and leaching in deionized water. An antimicrobial compound was added to the test water to prevent loss of creosote constituents through biodegradation. The tests were run at 35ºC. The water was replaced after each 72 hour test run. Wood samples were subjected to three consecutive test runs, and water concentrations were averaged among the runs at each time point. Water samples were collected regularly for analysis of four PAHs plus dibenzofuran (acenaphthene, fluorene, phenanthrene, and fluoranthene) using solid-phase microextraction (SPME) fibers analyzed by gas chromatography with a flame ionization detector (GC/FID).

PAH leaching rates over time

All components except phenanthrene leached at linearly declining (though different, based on their relative water solubilities) rates over the first eight hours. Phenanthrene leached at a higher rate than predicted by its solubility. With this exception, its leaching behavior was similar to that of the other PAHs.

Chemical concentrations appeared to approach a steady state at about 24 hours. The time between sample collection and analysis increased during the course of the study, and recovery rates were reduced by 8 to 14% per day of delay in analysis. Therefore, the apparent steady state might have been a function of increasingly poor compound recovery in samples collected at later intervals. Also, averaging water concentrations across three consecutive runs might have reduced the reported average concentrations since concentrations decreased with each subsequent round of leaching.

Xiao et al. (2002)

Study description

In a subsequent experiment, Xiao et al. (2002) treated Douglas fir samples with P1/P13 creosote to a retention of 12 pcf in accordance with WWPI's BMPs. Freshly cut ends were sealed with

epoxy, and the samples were wrapped in plastic to reduce volatilization losses between treatment and leaching in deionized water. An antimicrobial compound was added to the water to prevent loss of creosote constituents through biodegradation. Test conditions included three temperatures (5ºC, 20ºC, and 35ºC) and three water flow rates (0 cm/sec, 4 cm/sec, and 8 cm/sec). Tanks were sealed and airspaces minimized to reduce volatilization of PAHs. The water was replaced after each 72 hour test run. Water samples were collected regularly for analysis of PAHs.

Variations between individual PAHs

During the test, there was variable detection of naphthalene, 1-methylnaphthalene, and 2-methylnaphthalene, including non-detects. Those chemicals were excluded from further analysis. Concentrations of the same 4 remaining PAHs, plus dibenzofuran, in water were determined as in the previous study, by sampling with SPME (fibers), followed by GC/FID analysis. This method avoided delays in sample analysis. A high initial leaching rate was followed by an apparent decrease in leaching rate. Leaching of the remaining individual components was comparable to leaching reported by Ingram et al. (1982).

Effect of temperature and flow rate on leaching

Leaching rates of the sum of the PAHs measured increased with temperature and flow rate (Figure 2.2). The effect of temperature depended on flow rate; temperature had a smaller effect in still water, but an increasing effect with increasing flow rate. Leaching was greatest in warm, turbulent water.

Bestari et al. (1998a)

Study description

Bestari et al. (1998a) designed an outdoor freshwater mesocosm study to mimic field leaching conditions as closely as practical. Treated Douglas fir pilings (retention rate not specified) were suspended in 12,000 liter mesocosms containing sediment, rooted and floating macrophytes, fish, phytoplankton, zooplankton, and benthic invertebrates. The pilings did not contact the sediment. Water column and sediment PAH concentrations were analyzed for 15 priority-pollutant PAHs over 16 weeks.

Leaching rates of individual PAHs

The authors estimated a leaching rate of 50 $\mu g/cm^2$ per day, leached primarily from the outer 1 mm of the piling surface. They found no differences in the relative amounts of individual PAH compounds remaining in the piling. Bestari et al. interpreted this to mean that all PAH components either leached at an equal rate, or that some type of degradation process was removing the remaining compounds in proportion to the compounds that leached.

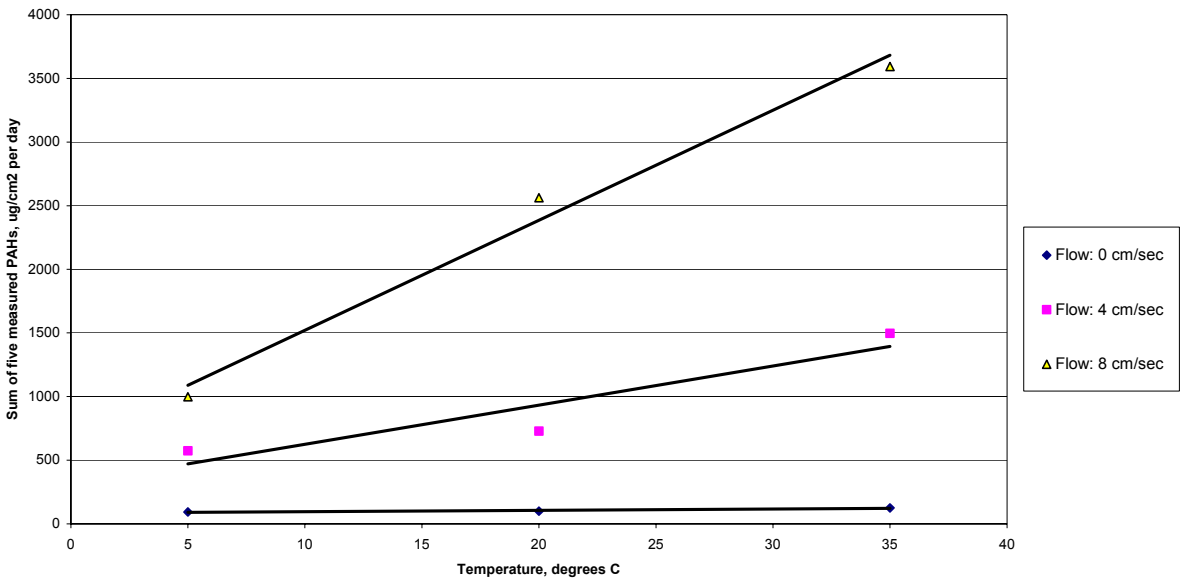

Figure 2.2. Effect of flow rate and temperature on total PAH leaching.

Source: Xiao et al., 2002.

Rao and Kuppusamy (1992)

Rao and Kuppusamy (1992) conducted field leaching tests of tropical wood species treated with a creosote formulation and method that differ from those currently recommended for use in the United States. Two relevant findings of this study are that leaching from samples treated to the same retention varied strongly both by species and within species tested.

2.1.2 Field observations of PAH leaching from creosote-treated wood subjected to natural or simulated rainfall

Whiticar et al. (1994)

Study description

Whiticar et al. (1994) subjected untreated and creosote-treated poles and timbers to natural and simulated rainfall. The treated wood was treated to retentions of either 166 kg/m^3 or 198 kg/m^3. The leachate was collected and analyzed for 18 PAHs, phenols, and total organic carbon (TOC). The document reports the sum of the measured PAH concentrations, and concentrations of phenanthrene, naphthalene, benzo[a]pyrene, phenols, and TOC.

Concentrations of individual PAHs

The results of this study cannot be used to quantify leaching rates because of the design, but the relative concentrations of the individual PAHs leached from the treated wood are of interest (Table 2.2). More phenanthrene was present in the leachate than any other PAH, even though more naphthalene was present in the creosote, and the water solubility of phenanthrene is lower than that of naphthalene. This suggests that either there was less naphthalene than phenanthrene in the whole creosote before treatment, as reported by Lorenz and Gjovik (1972, as cited in Ingram et al., 1982), or that the naphthalene in the sample was lost by volatilization before analysis. Naphthalene has a relatively high vapor pressure compared to the other 17 PAHs measured.

Table 2.2. Relative concentrations in leachate

Substance	Minimum (mg/L)	Maximum (mg/L)	Solubility (mg/L)
Sum of 18 PAHs	0.6	3.2	N/A
Naphthalene	0.03	0.3	34.4
Phenanthrene	0.2	1.1	1.0
Benzo[a]pyrene	0.00066	0.026	Highly insoluble
Phenols	0.7	6.0	Highly soluble

Source: Whiticar et al., 1994.

The TOC released from untreated (control) timbers was as high or higher than the TOC released from treated timbers. TOC ranged from 11 to 261 mg/L from untreated timbers and from 11 to 194 mg/L from creosote-treated timbers. Much of the TOC released from the untreated timbers was thought to be resin acids.

2.1.3 General review papers of PAH leaching

Two papers review creosote leaching research. Cooper (1991) summarized losses of creosote from treated wood in laboratory and environmental exposures, and Sinnott (2000) described the timing of loss of creosote components based on reviews of a number of studies. Other references, including some of the specific studies cited above, draw general conclusions concerning factors that affect PAH leaching from treated wood. In this section, we list and briefly summarize the factors generally recognized as being the most important in determining PAH leach rates from treated wood.

(i) Wood species, density, and surface area

Leaching varies markedly between woods of different species, probably for many complex reasons that are not well understood (Cooper, 1991; Rao and Kuppusamy, 1992). In southern pine and Douglas fir, leaching decreases as wood density increases (Leach, 1960; Miller, 1972; both as cited in Cooper, 1991). There have been no systematic analyses of leaching rates by species or wood density.

Leaching also predictably increases as the surface area to volume ratio of the wood increases (Leach, 1960; Colley and Burch, 1961; Stasse and Rogers, 1965; Gjovik, 1977; Miller, 1977; all as cited in Cooper, 1991), so the shape and configuration of structures built from treated wood can be a factor in predicting or limiting overall leaching rates.

(ii) Preservative formulation and loading rate

Different preservatives are known to leach at different rates (Cooper, 1991), but the magnitude of the variability is not well quantified. In the United States, currently only the P1/P13 formulation of creosote is approved by AWPA for use in aquatic systems, and BMPs specify a preferred loading rate.[1] In recent studies, use of this single formulation and loading rate has allowed for investigation of leaching related to non-formulation variables. In the past, however, a number of creosote formulations and application methods and rates were used, complicating the comparison of leaching studies from different eras. Therefore, few quantifiable conclusions can be drawn about how different preservative formulations and application methods have influenced leaching rates over time.

In addition, as the preservative loading rate increases, the leaching rate may increase (Cooper, 1991). The effect of loading rate is small and inconsistent relative to environmental factors such as temperature and water flow rate.

(iii) Individual PAH compounds

The water solubilities of creosote components influence their relative leaching rates in predictable ways (Ingram et al., 1982; Cooper, 1991; Whiticar et al., 1994). Lower boiling point, low molecular weight compounds, which are compounds with an atomic mass less than 200 atomic mass units (amu), and 1 to 3 benzene rings, comprise approximately 61% of the PAH compounds in creosote. These compounds dissolve more readily in the water column than the heavier PAHs. The higher molecular weight PAHs, which are compounds with an atomic mass

1. Dual treatment of pilings with chromated copper arsenate and P1/P13 creosote is also approved by AWPA, but the leaching characteristics of dual treated wood versus wood treated with only P1/P13 creosote have not been studied.

greater than 200 amu, and 3 or more rings, are lost from wood more slowly. These heavier PAHs are more likely to accumulate in sediments than remain dissolved in the water column. Brooks (1994) presents a compilation of percent composition for 13 PAHs in creosote-treated wood.

(iv) Environmental factors

The factors that affect leaching rates most significantly are temperature, water chemistry, water flow, and disturbance or abrasion. Figure 2.1 illustrates higher leaching rates in freshwater than saltwater, and the effect of temperature on leaching rate (Ingram et al., 1982). Figure 2.2 shows that the combined effects of temperature and flow are greater than the effects of either alone (Xiao et al., 2002). Becker et al. (2001) found that leaching of PAHs and N-containing heterocyclic compounds was greatest in deionized water, less in a slightly acidic, buffered solution, and least in a solution containing humic substances.

Disturbance and abrasion, which expose more surface area and sections of wood farther from the surface, can maintain higher leaching rates over time. For example, Bestari et al. (1998a) estimated that most of the leaching they observed in their outdoor freshwater mesocosm derived from the outer 1 mm of the piling surface. Removal of the outer 1 mm of the piling surface would expose less weathered wood, and could increase leaching long after the initial immersion. Indeed, most of the investigators in the studies reviewed in this section applied epoxy to cut ends of samples to minimize the effect of disturbance and abrasion as confounding factors.

(v) Time since treatment

In general, in the absence of disturbance and abrasion, leaching decreases with time since treatment whether the wood is kept in storage or placed in the water. Numerous investigators report substantial losses by volatilization of certain creosote components during dry storage (Stasse and Rogers, 1965; Stasse, 1966; Arsenault, 1973; Ingram et al., 1984; all as cited in Cooper, 1991). Whiticar et al. (1994) document both volatilization losses and rainwater leaching losses of creosote components during outdoor storage. Similarly, numerous investigators have documented a decline in leaching rates over time following installation in water (Leach, 1960; Colley and Burch, 1961; Stasse and Rogers, 1965; Gjovik, 1977; Miller, 1977; all as cited in Cooper, 1991). Furthermore, the WWPI BMPs and other post-treatment processing can reduce the rate of leaching (WWPI and Canadian Institute of Treated Wood, 1996; WWPI, 2002a).

2.2 Models of PAH Leaching Rates

Three investigators have used mechanistic understanding of leaching rates and relevant factors to develop models of PAH leaching rates. Dr. K. Brooks, Dr. T. Poston, and Dr. Y. Xiao each lead the development of models to describe the leaching of PAHs from treated wood. In the sections

below, we describe and evaluate these models. Because of the influence of multiple factors on PAH leaching rates and the relative paucity of empirical data on PAH leaching that can be used directly in an environmental risk assessment, the available PAH leaching rate models should be viewed as incompletely calibrated to realistic environmental settings, but still useful approximations of known first-order mechanistic processes that affect leaching and transport of PAHs, including in environmental settings.

2.2.1 Model descriptions

Brooks CREOSS model

Dr. K. Brooks developed a model to predict water column and sediment concentrations of PAHs near creosote-treated wood installations. He has written two descriptive papers (Brooks, 1994, 1997) and produced several versions of a spreadsheet model, the most recent of which is called "CREOSS" (copy in Appendix to this report; Brooks, 2004a). Versions of the model have been reviewed by industry and government representatives. These reviews have included comparisons between model predictions and environmental PAH concentrations. The model has not been published in the peer-reviewed literature.

Below, we describe the equations CREOSS uses to calculate a water concentration of PAHs and a leaching rate of PAHs. The model is not set up to calculate a leaching rate, but by rearranging some of the equations, a leaching rate can be derived.

Migration factor

The first step in deriving a leaching rate is the calculation of a unitless 'migration factor' (Equation 1). Calculation of the migration factor requires the user to input water temperature and salinity. The migration factor increases with water temperature and decreases with salinity. Default input values are 30 ppt salinity and 15ºC.

$$\text{Migration factor} = \left[24.4 + \left(0.78 \times \text{WaterTemp}\right) - \left(0.58 \times \text{Salinity}\right)\right] \qquad \text{Eq. 1}$$

Water concentration

The migration factor is used to calculate a water concentration (Equation 2). The equation also requires inputs of piling radius, an "age factor," a "retention factor," and a water flow rate term ("model velocity").

$$\text{Water concentration} = \frac{\left[\left(1{,}000{,}000 \times \text{MigrationFactor}\right) \times \left(3.14 \times 2 \times \text{PilingRadius}\right) \times \text{AgeFactor} \times \text{RetentionFactor} \times \text{WaterPartitioningCoefficient}\right]}{\left(2 \times 86{,}400 \times \text{PilingRadius} \times \text{ModelVelocity}\right)} \qquad \text{Eq. 2}$$

The term "water partitioning coefficient" describes the partitioning of PAHs between the dissolved and particulate compartments in the water column, and for the purpose of this equation, a default of 1.0 (indicating that all of the PAHs are assumed to be in the dissolved state) is used.

The piling age factor is calculated as an exponential decay function (Equation 3),

$$\text{Piling age factor} = \exp\left(\frac{-\text{piling age in years}}{10}\right) \qquad \text{Eq. 3}$$

The default value for piling age in the CREOSS model, 0 years, yields the highest possible piling age factor. Over the short term (less than one year), the decline in value of the piling age factor is nearly linear. After 10 years, the piling age factor decays to 0.37, or a leaching rate that is 37% of the rate at installation (Figure 2.3). After 20 years, it decays to 0.14.

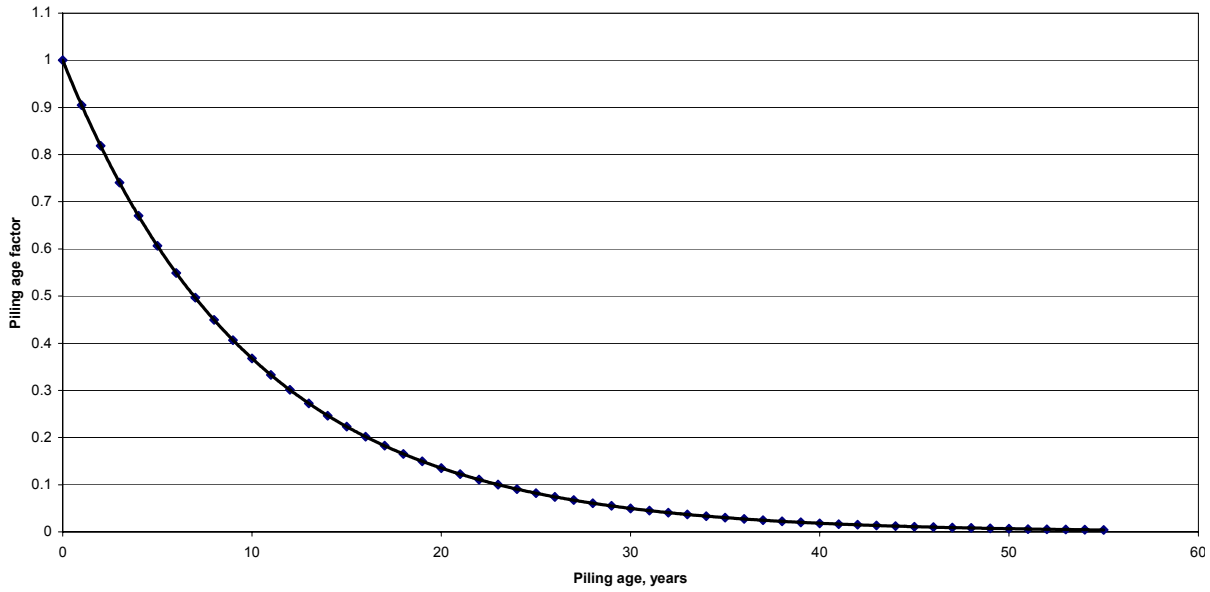

Figure 2.3. Piling age factor value over time, CREOSS model.

Source: Brooks, 2004a.

The retention factor is calculated as (Equation 4):

$$\text{Retention factor} = \exp\left\{0.5\left[\left(\frac{\text{piling retention in pcf}}{22.4}\right)-1\right]\right\} \quad \text{Eq. 4}$$

Although the retention factor is calculated as an exponential function, the formula yields a linear output over the range of realistic input values for piling retention, and the potential range of values is small. Using the default value for piling retention in CREOSS, 27 pcf, the retention factor is 1.108.

Leaching rate

The predicted leaching rates (µg/cm^2 per day) in fresh and seawater, calculated using the default values, are (Equation 5):

$$\text{Leaching rate} = \left[24.4+(0.78\times\text{WaterTemp})-(0.58\times\text{Salinity})\right]\times\exp\left(\frac{-\text{Pilingage}}{10}\right)\times\exp\left\{0.5\left[\left(\frac{\text{Piling retention in pcf}}{22.4}\right)-1\right]\right\} \quad \text{Eq. 5}$$

40.0 µg/cm^2 per day in freshwater, and 20.7 µg/cm^2 per day in seawater (at 30 ppt salinity).

Poston et al. (1996) model

The Poston et al. (1996) water concentration model depends on assumptions about the leaching rate of PAHs from wood. The model uses a freshwater leaching rate of 40 µg/cm^2 per day of total creosote, from Brooks (1994), that is assumed to be constant over the first 4 days following piling installation. The model assumes that the components of creosote migrate from the treated wood in proportion to their concentration in the wood. The proportions are taken from a Brooks (1994) compilation of percent composition for 13 PAHs in creosote-treated wood. This assumption is not supported by laboratory leaching experiments.

Xiao et al. (2002) model

This report presents insufficient details to permit an analysis of the leaching model presented.

2.2.2 Applicability and limitations of leaching rate models

Brooks (2004a) CREOSS model

The CREOSS model is complex, and documentation is essential to understand the model design. Currently, model documentation (Brooks, 1997) is older than the most recent model version, and certain contradictions are apparent. For example, the model documentation (Brooks, 1997) states that different leaching rates are used for LPAH and HPAH. The spreadsheet, however, uses the same migration rates for all components of creosote. The model documentation itself states that this assumption is not supported by laboratory studies. In addition, the documentation of the model is not sufficient to address issues such as unit consistency and the treatment of time, and it is difficult to determine which portions of the current model are mechanistic, which are empirical, what data the empirical portions are based on, and how calibration was accomplished.

In CREOSS, leaching rate is unaffected by water velocity. Water velocity only modifies a final water concentration. This conflicts with empirical data from Xiao et al. (2002), who found large increases in leaching rates as water velocity increased (Figure 2.2).

Empirical results from Ingram et al. (1982) and modeled results from CREOSS under the conditions tested in Ingram et al. (1982) are shown in Figure 2.4. Over the range of water temperatures tested by Ingram et al., leaching rates increased with temperature. The lowest temperature in the Ingram et al. test was 20°C, and if the slope of the temperature-leaching relationship above 20°C holds below 20°C, then CREOSS might over-predict leaching rates in both fresh and seawater at temperatures below 20°C.

Figure 2.4 also includes three data points from Miller (1977) and one from Graham (1991; as cited in Brooks, 1997). The Miller data points are for three pilings that were immersed in seawater for two years at a mean temperature of 10.4°C. The loss rate from one of the pilings, 89.9 $\mu g/cm^2$, is nine times greater than the maximum loss rate measured by Ingram et al. (1982) at the lowest temperature they tested in seawater (20°C). The loss rate from the other two similarly exposed pilings was zero (the two data points overlie one another in Figure 2.4). Brooks (1997) averaged the three Miller (1977) loss rates. Graham's single reported average leaching rate, 27.93 $\mu g/cm^2$ per day at 19ºC in seawater (Figure 2.4), is nearly triple that reported by Ingram et al. (1982). Discrepancies between migration rates reported by Ingram et al. (1982), Miller (1977), and Graham (1991) may be at least partially explained by the fact that Ingram et al. used WWPI BMPs, which specify treatment practices intended to minimize post-treatment loss of preservative. Regardless, it is unclear whether data from these studies were used to develop or calibrate the CREOSS model, or if the studies were cited for comparison.

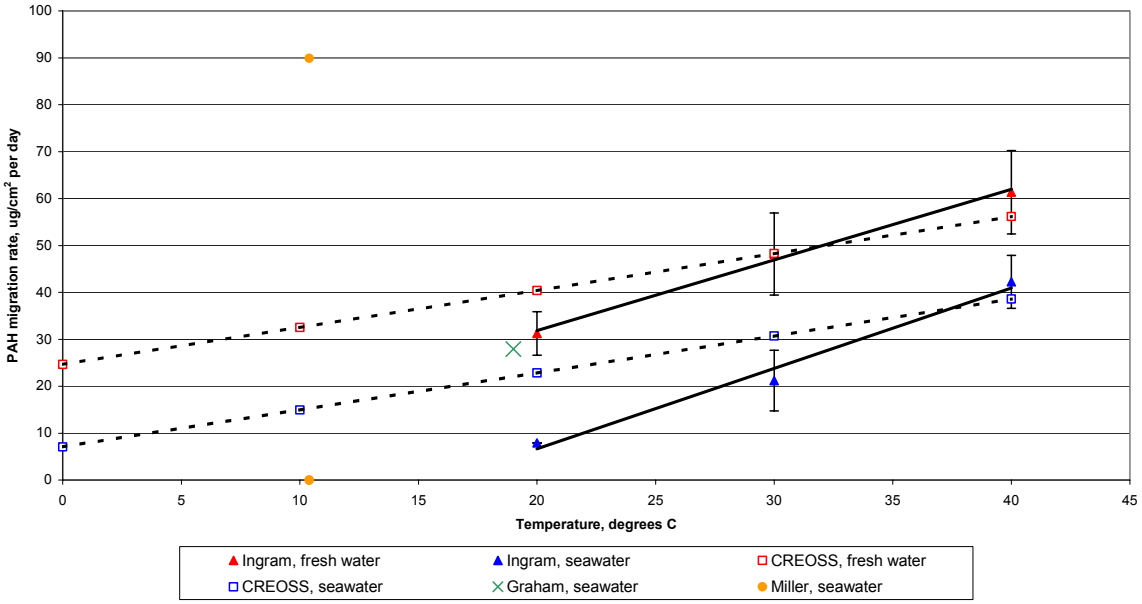

Figure 2.4. Temperature and salinity effects on leaching.

Sources: Ingram et al., 1982; CREOSS model output, Brooks, 2004a.

In CREOSS, all components of creosote leach equally as migration rates change due to changes in temperature, salinity, or other factors. Therefore, if the model over-predicts the overall migration rate, as suggested by comparison to empirical data in Figure 2.4 for temperatures below 20°C, the migration rate and amount of the less water soluble fractions leached is likely to be more over-predicted than the migration rate and amount leached of the remaining, more water-soluble fractions of creosote.

Poston et al. (1996)

The Poston et al. (1996) model uses Dr. Brooks' leaching assumptions. The applicability and limitations of those assumptions, as described above, also apply to the Poston et al. model.

2.2.3 Conclusions

Although the existing models do not account for all of the relevant data and do not contain complete documentation in some cases, they appear to adequately represent many first-order mechanisms of PAHs transport from creosote-treated wood into aquatic environments. The

models account for differential transport related to salinity, temperature, water flow rate, time since application, and chemical constituents. In Section 2.4, we evaluate how well environmental concentrations are predicted by the models.

2.3 Predicting Environmental Concentrations of PAH Resulting from the Use of Treated Wood

PAH leaching rates can be used to predict concentrations in the environment. Predicted environmental concentrations can then be compared to toxic effects thresholds (Chapters 3 and 4). PAHs in the aquatic environment are present in both dissolved form and adsorbed to particulate materials. The fate and transport of PAHs in the environment depends on concentrations in both the water column and sediments. Lower boiling point, low molecular weight compounds dissolve more readily in the water column than the heavier PAHs. The higher molecular weight PAH compounds in creosote tend to accumulate in sediment rather than remain dissolved in the water column.

Transport models estimate concentrations of PAH compounds in the surrounding water column and sediments. Below, we describe a transport model that predicts water column and sediment concentrations in tidal and non-tidal flows, and a transport model that estimates mean water column concentrations based on the ratio of the mass of contaminant leached to the volume of receiving water. The underlying objective of the models, to predict the PAH concentrations that occur under realistic environmental conditions, is the same, although their approaches differ.

2.3.1 Description of the available models

Brooks (1997)

Model description

Brooks (1997) developed a spreadsheet transport model that predicts water column and sediment concentrations of creosote-borne PAHs that leach from treated wood. The model estimates average concentrations in the water column in a cylindrical volume of water surrounding a piling, and PAH deposition in sediments with distance from the piling.

Fifteen input parameters (Table 2.3) that can be measured or estimated by the user are required to run the model. A set of recommended default input parameters for freshwater, marine, and estuarine environments is provided for cases where field data are not available and cannot be estimated (Brooks, 1997).

Table 2.3. Required input parameters

Parameter	Units
Piling retention of creosote	pounds per cubic foot (pcf)
Average piling radius	cm
Piling age	years
Average annual water temperature	°C
Salinity	parts per thousand (ppt)
Sediment particle settling velocity	0.05 cm/s for silt; 0.0005 cm/s for clay
Sediment density	g/cm^3
Steady state current speed	cm/sec, measured at slack tide
Average maximum tidal speed	cm/sec
Redox potential discontinuity	cm
Sediment total organic carbon	%
Sediment total PAH standard	parts per million (ppm) TOC
Maximum allowable sediment PAH	ppm TOC
PAH water partition coefficient	unitless
PAH sediment partition coefficient	unitless

The model calculates a series of intermediate outputs including migration, age factor, retention factor, degradation coefficient, model velocity, and geometry factor as follows:

$$\text{Migration rate } (\mu g/cm^2 \text{ per d}) = 24.4 + 0.78\ T - 0.58\ S \qquad \text{Eq. 6}$$

$$\text{Age factor} = \exp\left(\frac{-A}{10}\right) \qquad \text{Eq. 7}$$

$$\text{Retention factor} = \exp\left[\frac{\left(\frac{Ret}{22.4} - 1\right)}{2}\right] \qquad \text{Eq. 8}$$

$$\text{Degradation coefficient} = \frac{\exp\left[\left(\frac{4 - RPD}{3}\right)^3\right]}{0.047\ T} \qquad \text{Eq. 9}$$

$$\text{Model velocity (cm/s)} = V_{ss} + 0.64\ V_{max} \qquad \text{Eq. 10}$$

$$\text{Geometry factor} = 1 + \frac{V_{model}}{10} \qquad \text{Eq. 11}$$

where:

T	=	temperature (°C)
S	=	salinity (ppt)
A	=	age of piling (years)
Ret	=	initial creosote retention of piling (pounds per cubic foot)
RPD	=	redox potential discontinuity (cm)
V_{ss}	=	steady state velocity (cm/s)
V_{max}	=	maximum tidal velocity (cm/s)
V_{model}	=	model velocity.

These intermediate outputs are then used to calculate concentrations of leached PAH in the water column and sediments. Equation 12 is used in the model to estimate water column concentrations of PAHs leached from creosote-treated pilings:

$$PAH_{water}\ (pptr) = \frac{83333.3 \times WPC \times M_r \times A_f \times Ret_f \times R_p \times G_f}{\left[\left(1800 \times V_{model} + R_p\right)^2 - R_p{}^2\right]} \qquad \text{Eq. 12}$$

where:

WPC	=	water partition coefficient (defines the proportion of PAH assumed to be dissolved in the water column)
M_r	=	migration rate
A_f	=	age factor
Ret_f	=	retention factor
R_p	=	piling radius
G_f	=	geometry factor.

The tidal current equation (an input to model velocity) is based on an equation for harmonic motion, assuming a frequency of 12 hours. Transport appears to be based on advection only (molecular and turbulent diffusion are not considered), but it is not clear how advective transport is modeled.

The predicted water column concentration appears to be an average concentration, for some unspecified length of time, calculated over a volume that is dependent on the model velocity (V_{model}). However, we were unable to confirm this from the available documentation, and replication of the model was beyond the scope of this report. Therefore, we recommend further documentation and peer review of the model before reaching any definitive conclusions from these modeling results.

In addition, dimensional analysis of the equation shows inconsistent units.

The equation used in the model to estimate sediment PAH concentrations is:

PAH sediment accumulation ($\mu g/cm^2$ per day) =
Deposition H Degradation H SPC H G_f Eq. 13

where:

$$\text{Deposition } (\mu g/cm^2 \text{ per day}) = \frac{M \times A_f \times Ret_f \times R_p}{\left[\left(\frac{V_{model}}{V_{vert}}\right) \times \left(r + R_p\right)\right]}$$

SPC = sediment partition coefficient (defines the proportion of PAH assumed to be adsorbed to sediment)
V_{vert} = particle settling velocity (cm/s)
r = distance from the piling perimeter where the concentration is predicted (cm).

The equation for sediment deposition appears to be mechanistically based. Average deposition is calculated by dividing the loss per unit area of the piling that partitions to the sediment by the incremental area over which that sediment is deposited. Sediment concentrations (in units of µg/g dry weight or ppm) are obtained by dividing the sediment accumulation by the sediment density. The recommended input for the particle settling velocity value (necessary to calculate the incremental area over which deposition occurs) is also mechanistically based; it is derived using Stokes' law for the settling velocities of small particles. The sediment deposition equation does not consider post-deposition disturbance such as bioturbation, or sediment mixing from current, propeller wash, or other disturbances.

Model assumptions include:

- Marine-grade creosote contains 80.14% total PAH (TPAH; Environment Canada, 1993), of which 19.57% is HPAH and 60.57% is LPAH.

- HPAH and LPAH migrate from the piling in proportion to their content in whole creosote.
- Once released into the environment, all HPAHs are adsorbed to the silt-clay fraction and settle to the bottom sediment.
- Once released into the environment, 4.83% of the LPAHs are adsorbed to the silt-clay fraction and settle to the bottom sediment and 95.17% of the LPAHs are dissolved in the water column where they degrade with determinable half-lives. Brooks (1997) indicates that these values were determined by assuming minimal mineralization of HPAH in the water column and combining the relative proportions of LPAH and HPAH in whole creosote with the relative proportions reported for contaminated sediments. Explanation for the numbers used is not provided.
- No volatilization of LPAH occurs.
- Ambient water pH does not affect the migration of PAH from creosote-treated wood.
- Tidal flows are harmonic with a frequency of 12 hours.
- The receiving water volume is large in comparison with the total amount of PAH lost from the structure.

Model predictions

As an example, for a 13 in diameter piling submerged in 20 feet of seawater that leaches 1.24 grams of PAH per day, the model predicts a water column concentration of 0.003 ppb total PAH. The volume of water containing this concentration and the length of time that this concentration persists are not specified, but it appears that the concentration is an average that might not be representative of instantaneous concentrations at a particular location. The predicted sediment PAH concentration is 5.89 ppm within 25 cm of the piling and 2.00 ppm 1 m from the piling. These results are based on inputs of a maximum tidal current of 2.5 cm/s and an RPD of 3 cm.

Estimated sediment concentrations appear to be influenced by current velocity and oxygen availability in the sediments. As expected, environments with poor circulation (low velocity) and low sediment oxygen availability (low RPD) are predicted to pose the greatest risk for elevated sediment concentrations of PAHs.

Volatilization and turbulence (which would increase dilution), and abrasion of pilings (which would increase PAH loading in sediment as abraded wood particles become water logged and sink) are not considered in the model. In addition, since the model assumes that the receiving

water volume is large in comparison with the total amount of PAH lost from the structure, it is less applicable to small systems and systems lacking circulation.

Poston et al. (1996)

Model description

Poston et al. (1996) developed a "box" plume model to estimate PAH concentrations around creosote-treated wood pilings. The model estimates spatially averaged water column concentrations of PAH compounds for one-day units in a hypothetical rectangular plume. The model does not predict sediment PAH concentrations.

The source of PAHs in the model is a vertical "footprint" (in square meters) perpendicular to the current representing an assumed number of pilings (50, 100, or 350) compressed into a plane of a given area. Several footprint areas can be modeled to simulate different configurations of the pilings (200, 400, 800, and 1,524 m^2). Smaller footprint areas model higher density configurations. The volume of the box plume is determined by the surface area of the vertical plane of the source and the distance that water flows in one day (determined by the current velocity).

Initial runs were conducted at a maximum velocity of 40.6 cm/s. Four additional current velocities were also simulated: 0.3, 0.5, 1.0, and 10 cm/s. A conceptual model of the plume dimensions is shown in Figure 2.5.

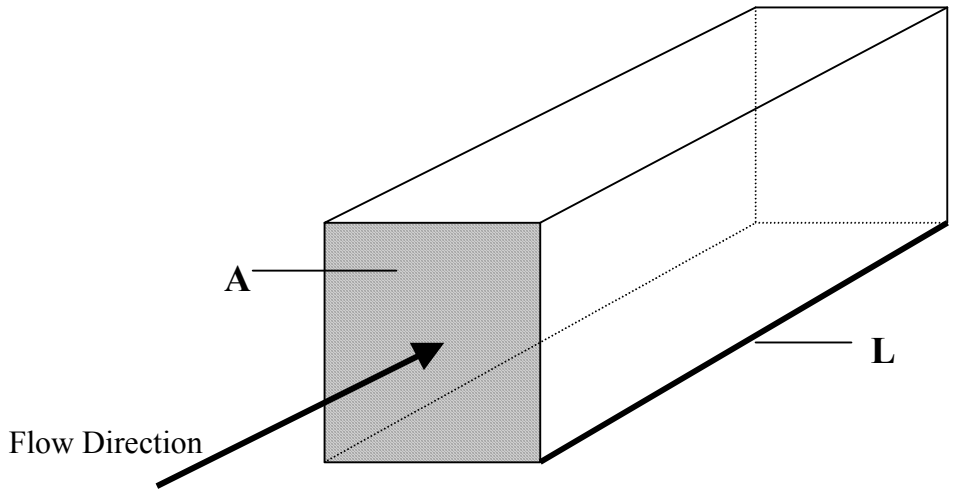

Figure 2.5. Conceptual schematic of the Poston et al. (1996) box plume. The vertical plane A represents the pilings (different piling configurations result in different values of A) and the length L is the distance that water flows away from the pilings in one day.

The volume of the plume is calculated as follows:

$$V = A H L \qquad \text{Eq. 14}$$

where:

A = vertical footprint (representative of the pilings)
L = current velocity H 86,400 seconds/day H 1 day.

The concentration in the plume is calculated by dividing the amount of contaminant leached from all pilings in a 24-hour period by the plume volume. This yields an average concentration throughout the "box" plume. The amount of PAH leached per day is calculated as follows:

$$\text{Mass of leachate (ug)} = \text{number pilings H surface area of piling H leaching rate per day} \qquad \text{Eq. 15}$$

The key assumptions incorporated into the model are as follows:

- There is no lateral mixing.
- Exposure concentrations modeled for the first day do not change for at least four days.
- Leaching is constant for a period of four days at a rate of 40 $\mu g/cm^2$/day for total PAHs (Brooks, 1994). Leaching rates for individual compounds were obtained by multiplying the percent composition in creosote of the compound (as reported in Brooks, 1994) by the total PAH leaching rate.
- Each piling has a diameter of 30 cm and 10 m of submerged length with a surface area of 94,284 cm^2.
- All pilings are assumed to be installed in one day.

Model predictions

Model results are expressed in terms of exceedence of a toxic threshold. For each of the PAH compounds evaluated in the model, the model compares the predicted plume concentration to a published toxic threshold for that compound to get a toxic unit (TU). Toxic units greater than 0.1 are summed to calculate a total TU. The toxic thresholds used in the model for each PAH are

LC_{50}s, or concentrations at which 50% of test organisms are killed (Table 2.4).[2] An overall TU >1.0 indicates an overall toxic threshold for all of the PAHs combined.

Table 2.4. LC_{50}s used to calculate toxic unit values for each PAH in the Poston model

PAH compound	LC_{50} (µg/L)
Naphthalene	3,852
Acenaphthylene	474
Acenaphthene	480
Fluorene	337
2,6-dimethylnaphthalene	172
Anthracene	140
Phenanthrene	140
Fluoranthene	18
Chrysene	4.02
Benz(a)anthracene	2.7
Benzo(a)pyrene	1.09
Dibenzo(a,h)anthracene	0.57
Benzo(k)fluoranthene	0.23
Benzo(g,h,i)perylene	0.1
Indeno(1,2,3-c,d)pyrene	0.03
Source: NMFS, 1996.	

Modeled PAH concentrations increase with the number and density of pilings, and decrease with flow rate. For example:

- For the 350-piling scenario, concentrations of all PAHs modeled exceeded the 0.1 TU threshold at flows of ≤ 0.8 cm/s, regardless of footprint size. At a flow rate of 7.0 cm/s, PAH concentrations exceeded the threshold for footprints of 400 m^2 and smaller.

- For the 100-piling scenario, predicted concentrations of PAHs exceeded the 0.1 TU at all modeled flow rates for footprint areas of 800 m^2 and less.

2. Note that compounds listed in this table do not match exactly the major creosote constituents listed in Table 1.3, which introduces some additional uncertainty regarding the applicability of the model to creosote-treated woods.

- For the 50-piling scenario, flows of 1 cm/s or less and footprint areas of 400 m^2 and less resulted in predicted concentrations greater than the threshold.

This model is a simplified transport model that depicts average concentrations in a hypothetical box plume. Sedimentation, volatilization, lateral mixing, and turbulent mixing (which would decrease concentrations in the water column) are not considered in the model. Poston et al. (1996) acknowledge that the model probably over-predicts concentrations for water column PAHs and that model results are "approximate estimates at best."

2.3.2 Comparison to field data

(i) Brooks (1997)

Brooks (1997) compared his predicted sediment concentration results to measured sediment concentrations at two sites in British Columbia. Belcarra Bay has poor circulation, a maximum tidal current of 3.9 cm/s, an average salinity of 15.7 ppt, and an average RPD of 1 cm. The newest piling was 1.5 years old. The model over-predicted sediment concentrations 1 m from the piling but under-predicted concentrations at 3 and 5 m from the piling. Approximate predicted and measured concentrations, estimated from Figure 3 in Brooks (1997), are shown in Table 2.5.

Table 2.5. Approximate predicted and actual concentrations at Belcarra Bay, British Columbia

Distance from piling (m)	Predicted PAH concentration (ppm)	Measured PAH concentration (ppm)
1	9	4
3	5.5	10
5	3	8.5
10	0	Negligible
20	0	Negligible
40	0	Negligible

Source: Figure 3; Brooks, 1997.

Westham Island Bridge had greater circulation, an average maximum tidal current of 18.1 cm/s, an average salinity of less than 2 ppt, and an average RPD of 1.25 cm. The newest piling was 8 years old. The model somewhat over-predicted sediment concentrations at 0.5 and 2.0 m from the piling. Predicted and measured concentrations were similar at 5 m from the piling (Table 2.6).

Table 2.6. Approximate predicted and actual concentrations at Westham Island Bridge, British Columbia

Distance from piling (m)	Predicted PAH concentration (ppm)	Measured PAH concentration (ppm)
0.5	0.56	0.17
2	0.17	0.03
5	0.08	0.07

Source: Figure 4; Brooks, 1997.

(ii) Goyette and Brooks (1998, 2001)

Goyette and Brooks (1998, 2001) compared modeled versus measured concentrations of PAHs in surface water and sediment for sets of 6 treated pilings, both weathered and unweathered, and a set of untreated pilings in a poorly flushed basin of Vancouver Island, British Columbia. They also included a no-piling control. The pilings were all Douglas fir, and the new (unweathered) pilings were treated to a retention of 27 pcf using WWPI BMP standards. The weathered pilings had an unspecified retention and were not treated to BMPs. The pilings, with an average diameter of 30 cm each, were installed in 6-piling dolphins (a dolphin is a boat-mooring structure composed of multiple closely spaced pilings) having a minimum base diameter of 2.5 m. The current direction and speed were assessed over a two-day period, and determinations of "upstream" and "downstream" sampling directions and locations were made on the basis of this assessment.

In this study, the modeled total PAHs included all PAHs leached from creosote, but the measured total PAHs included only 17 measured PAHs (potentially underestimating the total). The site was said to have been selected as a "worst-case" scenario, with low current speed, but one of the site-selection criteria involved the presence of oxic sediments (having an RPD greater than 3 cm below the sediment surface). Sediments above the RPD are oxic, which increases the rate of PAH breakdown, and sediment samples for PAH analysis were routinely collected from the top (0-2 cm) layer during the study.

Goyette and Brooks (1998, 2001) used a 1994 version of Dr. Brooks' model to predict an increase of 336 ng/L total PAH in the water column within 15 cm of individual pilings This predicted concentration was approximately 11 times greater than the maximum of 30.8 ng/L total PAH measured with semi-permeable membrane sampling devices (SPMDs). Water column results were dependent on the ability of the SPMDs to accurately reflect water column concentrations. Although method blanks are documented, no positive controls were reported, and percent recovery was not noted.

PAHs were regularly measured in the upstream and downstream directions from each dolphin, beginning at 0.5 m. Although the distribution of PAHs was observed to be patchy (there was high spatial, and between-replicate, variability, and small oily particles were observed in the sediment samples), the model generally also predicted sediment concentrations that were higher than those observed in the upstream and downstream directions. However, a single sampling event sampled sediments within the dolphins and in a direction described as "offshore" of the dolphins. This sampling event revealed far higher sediment concentrations both inside the dolphins and outside the dolphins in the offshore direction than those observed close to, but outside of, the dolphins in the upstream and downstream directions, and also far higher than model predictions (Table 2.7).

Table 2.7. Observed and predicted sediment PAH concentrations in the Sooke Basin study, day 384/385

Dolphin type	Direction relative to dolphin	Distance from dolphin (m)	Predicted PAH concentration (all), ppm	Observed PAH concentration (17 PAHs), ppm
BMP unweathered	Downstream	0.5	~24	16.1
	Downstream	1.0	~19	5.7
	Inside dolphin	0.0	N/A	30.8
	Offshore	0.5	N/A	68.3
	Offshore	2.0	N/A	2.9
Weathered	Downstream	0.5	~24	10.8
	Downstream	2.0	~6	6.3
	Offshore	0.5	N/A	33.8
	Offshore	2.0	N/A	15.3
	Inside dolphin	0	N/A	47.4

Also, the peak of sediment concentrations occurred somewhat sooner than predicted, giving the appearance of model under-prediction earlier in the experiment followed by over-prediction later in the experiment.

The results of these comparisons of modeled to measured PAH concentration data are not sufficient to make any specific quantitative conclusions about the accuracy of the model in predicting actual sediment concentrations. The variability in the results is understandable because of the many site-specific conditions and simplifying assumptions required to run the model. For instance, Brooks (1997) presents the age of the newest piling, but it is unknown whether just some, or all, of the pilings were installed or replaced at that time. Also, both the number and density of pilings, factors that the Poston et al. (1996) considers to be important, are not included in the Brooks (1997) transport model.

2.3.3 Applicability of the models

The aquatic systems that the Brooks (1997) and Poston et al. (1996) models simulate are highly complex systems that are difficult to describe quantitatively. Numerous simplifying assumptions were necessary to construct the models. For example, tidal currents are very complex and are influenced by many highly variable factors. Turbulence, the main process by which mixing occurs in these systems, is a chaotic three-dimensional process that is notoriously difficult to model. Furthermore, the leaching rates used in the model as inputs are themselves model results with their own set of uncertainties.

Despite these uncertainties and assumptions, the leaching and transport models have value in qualitatively describing many first-order factors related to PAH leaching from treated wood and movement in the environment. For example, Brooks (1997) recognizes the importance of oxygen availability in the system, and sediments with a thin layer of oxygenated sediments (a small RPD) result in higher predicted concentrations of PAH compounds than well oxygenated sediments. Both the Brooks (1997) and Poston et al. (1996) models incorporate flow rate as a critical variable affecting concentrations in the environment, and the Poston et al. (1996) model also incorporates piling density (and thus surface area), which laboratory studies have confirmed to be important.

2.4 Conclusions

The modeling of PAH leaching rates from treated wood and the resulting environmental concentrations are important for evaluating the environmental risk from treated wood structures. Our review and evaluation of the available information and models on PAH leaching and environmental concentrations, supports the following:

The rate of leaching of PAH is greater:

- In freshwater than in seawater
- At high temperatures than at low temperatures
- At high flow rates than at low flow rates
- From less dense wood than from denser wood
- From freshly treated wood than from wood that has either been stored after treatment or been exposed to water
- From end grain than from face grain

- At a higher wood surface area to volume ratio
- From wood that has not been treated to the WWPI BMPs than from wood that has been treated to the BMPs.

Also, leaching is faster for the more water-soluble compounds. Variations in the leaching rates of PAHs from same-species wood samples can be surprisingly large (Miller, 1977, as cited in Brooks, 1997; Rao and Kuppusamy, 1992). In addition to PAHs, compounds such as N-heterocycles can leach from treated wood, and this issue has not been thoroughly investigated in the literature. Most leaching studies to date, with the exception of Becker et al. (2001), have focused on PAH leaching.

The Brooks leaching model, in its most current incarnation (the CREOSS spreadsheet model) incorporates only temperature, salinity, piling age, and creosote retention factors in the calculation of leaching rate. The predicted leaching rates generally behave as expected based on the results of laboratory studies for all components considered, but agreement with laboratory observations, where comparisons can be made, could be improved, particularly at temperatures below 20°C (Figure 2.4). The model ignores the effect of water flow rate on leaching rate, relies on studies of older installations probably missing peak leaching rates, and it assumes an equal migration rate of all components of creosote, which is not supported by laboratory observations. However, the model appears to be adequate for predicting many first-order factors that explain laboratory and field observations.

The transport models for predicting environmental concentrations of PAHs in surface water and sediment are based on assumptions regarding modeled leaching rates and environmental parameters such as water flow, surface area of treated wood, and sediment settling and movement (Poston et al., 1996; Brooks, 1997). The inputs needed to run these models are highly site-specific. The models can provide site-specific predictions where site-specific conditions are known, and they are useful for evaluating the relative importance of different environmental variables on environmental concentrations of PAHs. The models may not fully describe transient concentrations of PAHs, particularly shortly after installation of treated wood in water, or during severe disturbances (especially abrasion).

The transport models indicate that environmental concentrations decrease with increasing flow rates, due to increasing dilution. However, several leaching studies suggest that the rate of leaching also increases with increasing flow rates. This raises the issue of the relative contributions of leaching rate and dilution to water column concentrations of PAHs, since both are affected by changes in flow rate. As flow rate increases, both leaching and dilution increase, but their effects on water column concentration oppose one another. Available data are not sufficient to answer questions regarding the net outcome of this relationship under various realistic scenarios; that is, at any point, as flow rate increases, will the increase in leaching

outweigh the increase in dilution? The results in Xiao et al. (2002) appear to show leaching doubling or more than doubling under some circumstances, when flow rate simply doubles, but this issue has not been thoroughly investigated.

The current models only incorporate flow velocity into the dilution portion of the model, and do not consider the effect that increased velocity may have on leaching rate. It is possible that, because of this omission, the models under-predict actual concentrations. However, these models do not consider lateral mixing or turbulence, both of which increase dilution and mitigate the effect of increased leaching due to increased flow rates.

3. Toxicity of Creosote to Estuarine Organisms

This chapter discusses the environmental toxicity of creosote, including constituents of creosote that are known to be toxic (Section 3.1), the routes by which toxic constituents expose organisms (Section 3.2), the toxicity of various constituents to organisms under environmentally relevant conditions (Section 3.3), and the concentrations at which biological effects begin to occur (Section 3.4).

3.1 Toxic Components of Creosote

As described in Section 1.4, the chemical composition of creosote is very complex. This compositional complexity can obscure the toxicity of the mixture and of particular constituents in environmental settings. PAHs are the dominant class of compounds in creosote, comprising 85-90% of creosote's mass. PAHs are the most comprehensively studied group of chemicals found in creosote, due largely to the potency of some as carcinogens, and to their widespread, and apparently increasing, occurrence in the environment (e.g., Van Metre et al., 2000).

Although a number of PAHs have been well studied regarding their potency as carcinogens, less is known about the non-carcinogenic toxicities of PAHs and other components of creosote to aquatic and marine organisms. This appears to be true particularly for creosote components such as alkylated PAHs and heterocycles. However, while there is little information concerning interactive effects of creosote components, there is a reasonable body of work addressing the effects of creosote per se, both in laboratory exposures and from field studies.

Many studies of PAHs and creosote compounds in aquatic and marine environments distinguish between LPAHs and HPAHs. Generally, LPAHs are PAHs with two or three fused benzene rings; HPAHs are PAHs containing four or more rings. In some cases, such as fluorene and fluoranthene, a 5-carbon aromatic cyclic ring replaces benzene. The focus of the rest of this chapter is the current state of knowledge about routes of exposure and the toxicity of creosote components and the mixture itself, highlighting the sensitive endpoints that drive risk assessments.

3.2 Routes of Exposure

Meador et al. (1995) provided a thorough review of the literature on factors governing the bioaccumulation of PAHs in marine organisms (invertebrates and fish). Their conclusions are supported by subsequent studies, and while they focused on parent PAHs (i.e., non-alkyl-substituted compounds), their analysis probably holds for other major components in creosote such as phenolics, alkylated PAHs, and heterocycles.

Meador et al. (1995) concluded that the major routes of exposure for marine animals were uptake of waterborne chemicals and through the diet. Waterborne chemicals include those in the interstitial water (ISW) of sediments. ISW is probably the compartment governing the bioavailability of many organic chemicals in marine systems. Direct uptake of sediment-bound chemicals (e.g., through the integument of worms and fish) appears to be negligible. This conclusion is supported by studies that demonstrate that the water-soluble fractions of contaminated sediments generally drive the toxicity of chemicals in bulk sediments (e.g., Roberts et al., 1989; Swartz et al., 1989; Padma et al., 1998). Thus, in hazard assessments of PAH-contaminated sediments, K_{oc}, which describes equilibrium partitioning between the organic carbon of sediment and the surrounding ISW, becomes a key driver for exposure assessment. K_{ow}s are often used to estimate K_{oc}s for individual compounds (see Swartz et al., 1995).

The diet can also be an important source of PAHs and related creosote chemicals (Meador et al., 1995), and particularly so for deposit-feeding invertebrates and for fish that feed on invertebrates. Malins et al. (1985) reported elevated concentrations of PAHs and related chemicals in the stomach contents of marine fish inhabiting a creosote-polluted site in Puget Sound, Washington. However, absorption efficiencies of dietary PAHs may be limited; Niimi and Dookhran (1989) reported uptake efficiencies of 2% to 32% for various PAHs.[1]

The relative roles of uptake from the water column (including ISW) and the diet vary greatly, depending on factors such as the organism's life history, physico-chemical characteristics of specific compounds (such as solubility and K_{ow}), and environmental variables (such as sediment organic carbon content). In general, water column uptake is more important for chemicals with higher solubility (or lower K_{ow}), and also is more important for filter-feeding organisms. However, as Meador et al. (1995) stress, the route of uptake is, in the long run, relatively unimportant. Over time, equilibrium among media (sediments, water, and biota) will occur and the same tissue burdens (or other measures of exposure, as described below) will occur regardless of route of exposure.

1. However, many hydrophobic organic compounds have higher uptake efficiencies than this. Niimi and Dookhran (1989) packed one gelatin capsule per day into ground trout diet, which may not be comparable to ingesting invertebrate prey.

The assessment of exposure to, and accumulation of, creosote hydrocarbons is complicated by metabolism. Halogenated hydrophobic chemicals, such as PCBs, chlorinated dioxins and furans, and chlorinated hydrocarbon pesticides, such as DDT, that are also of concern in marine and estuarine systems, are highly resistant to metabolism by most organisms. In contrast, creosote hydrocarbons are not halogenated and consequently are readily prone to metabolism by many organisms. Among estuarine and marine animals, metabolic capacity is generally very high in fish (and other vertebrates), intermediate in crustaceans, and very limited in bivalves (Meador et al., 1995). For this reason, tissue concentrations of creosote hydrocarbons provide a reasonably accurate measure of exposure in bivalves, but an inaccurate measure in vertebrates. Crustaceans are probably intermediate in this respect.

Vertebrate metabolism is discussed in more detail in Section 3.3. Briefly, the metabolism of hydrocarbons gives rise to relatively hydrophilic metabolites, most of which are excreted via the bile in vertebrates, and to reactive metabolites that can bind to cellular macromolecules such as DNA. Thus, measures such as concentrations of bile metabolites and DNA adducts are often used as measures of hydrocarbon (mainly PAH) exposure in vertebrates.

3.3 Toxicities

Information concerning the toxicity of creosote and constituent chemicals is not equally complete for all constituents. Much information is available concerning PAHs, particularly HPAHs, in part because of the potent carcinogens in this group. There is also some information concerning the toxicities of heterocycles and creosote. Phenolics appear to be the least studied of the key components of creosote.

Most toxicity experiments published in the peer-reviewed literature have been conducted by exposing aquatic biota either to creosote-spiked water or to sediments or sediment elutriates containing creosote, which may be either spiked with whole creosote or field-collected product.

These exposures are not directly equivalent to exposures conducted using leachates (either in water or sediment) from treated wood, for several reasons. Although all creosote exposure experiments contain PAHs and other compounds, the individual constituents and their concentrations can be heavily influenced by weathering and by the leaching process itself. Also, many creosote-spiking experiments use solvents such as acetone to increase the aqueous bioavailability of the mixture's more hydrophobic components. And, finally, sediments for use in creosote bioassays are often field-collected from sites where creosote was released from wood treatment facilities. At such sites, other toxicants such as pentachlorophenol and metals are frequently present in potentially toxic amounts, making it difficult to determine what portion of observed toxicity is attributable to the creosote in the sediments.

Plants appear to be less sensitive to creosote chemicals than animals in aquatic and marine systems (WHO, 2004). Our discussion focuses on animals, particularly invertebrates and fish. In studies with invertebrates, endpoints that have been examined most frequently include acute toxicity (mortality), phototoxicity (a distinct form of acute toxicity), and effects on populations and community structure. Studies with fishes have included investigations of acute toxicity and phototoxicity, and more basic investigations of reproduction and growth, effects on the immune system, early life stage development, and chemical carcinogenesis. The effects of creosote and creosote chemicals on key endpoints are discussed below.

3.3.1 Toxicity from acute (short-term) exposure

Under standard laboratory conditions, the acute toxicities of water-borne PAHs and alkylated PAHs to marine organisms vary widely among chemicals and test organisms (see reviews by Neff, 1985; and Eisler, 2000). For marine and freshwater invertebrates and fishes, 24- and 96-hour LC_{50}s generally range from approximately 0.1 to 4 mg/L (ppm); crustaceans tend to be relatively more sensitive and fish less sensitive in these tests. Eisler (2000) noted that these concentrations are generally orders of magnitude greater than those encountered in surface waters, including at polluted sites. Few studies of this nature examining heterocycles have been reported.

Other studies have investigated the acute toxicities of creosote itself, either in sediments or water-extracted fractions. Padma et al. (1998) exposed the mysid shrimp *Mysidopsis bahia* to either a creosote-contaminated sediment (Elizabeth River, Virginia) or to the water-soluble fraction (WSF) of this sediment. Chemical analyses were performed on both sample types. As measured by total identified aromatic compounds, they determined 24-hour LC_{50}s to be approximately 180 µg/L (ppb) and 700 µg/L for the water-extractable fraction and sediment, respectively, indicating an approximate four-fold greater toxicity of the WSF. A major difference between the two samples was higher concentrations of low molecular weight PAHs (< three rings) in the WSF compared with the sediment. N-heterocycles were also reported to be higher in the WSF, but no data were provided.

Swartz et al. (1989) measured the acute toxicity of various dilutions of creosote-contaminated sediments and ISW extracted from the sediments, collected from Eagle Harbor, Washington, to the marine amphipod *Rhepoxynius abronius*. They measured 13 PAHs in these samples; heterocycles apparently were not measured. Based on these studies, the 4-day LC_{50} for total PAHs was 666 mg/kg (wet weight). The ISW LC_{50} was found to be 0.89% of the undiluted ISW. Based on data for undiluted ISW provided in Table 4 of Schwartz et al. (1989), this equates to an ISW LC_{50} of 100 µg/L. Dominant PAHs observed in both sample types included acenaphthene, fluorene, phenanthrene, fluoranthene, and pyrene.

Sved and Roberts (1995) constructed a flow-through dilutor system to continually expose the estuarine teleost, spot (*Leiostomus xanthurus*), to selected dilutions of suspended sediment mixed with commercial marine creosote. They measured over 100 compounds, including heterocycles, but provided data for only six PAHs that comprised 64% of total resolvable PAH: naphthalene (21%), acenaphthene (8%), fluorene (6%), phenanthrene (14%), fluoranthene (9%), and pyrene (6%). Based on these exposures, the 96-hour LC_{50} was determined to be 1,740 µg/L, and the no observable effects level (NOEL) was 250 µg/L. In a previous study that involved exposures of spot in this system for 14 days (Sved et al., 1992), mortality, fin erosion, and epidermal lesions were observed at total PAH concentrations as low as 76 µg/L. Induction of hepatic ethoxyresorufin O-deeethylase (EROD) activities were observed at all concentrations tested, down to 16 µg/L. In this study, concentrations of individual chemicals were not reported.

Sved et al. (1997) also used the flow-through dilutor system to compare the toxicity of commercial creosote that had been fractioned into HPAH and LPAH, with the exception that phenanthene and fluoranthene were important components of both. In 10-day exposures of spot, mortality, fin erosion, epidermal lesions, and EROD inductions were observed in fish exposed to HPAH. Of these responses, only limited epidermal lesions were observed in fish exposed to LPAH. Dominant PAHs in the LPAH fraction were acenapthalene (14%), fluorene (12%), phenanthrene (28%), and fluoranthene (9%). Dominant PAHs in the HPAH fraction were phenanthrene (22%), fluoranthene (26%), and pyrene (15%). The authors concluded that the HPAH fraction better mirrors weathered creosote in the field and produces responses similar to field and laboratory responses to exposures to creosote-contaminated sediments.

The identity of key chemicals responsible for the acute toxicity of creosote (as well as some other endpoints described below) remains unresolved. In his review, Neff (1985) concluded that only PAHs in the molecular weight range of naphthalene (MW = 128) to fluoranthene and pyrene (MW = 202) demonstrated significant acute toxicity to aquatic organisms, and within this range, bioaccumulation increases with increasing molecular weight. A number of subsequent studies, including some described above, support this conclusion. However, Padma et al. (1999) concluded that more water-soluble LPAH, perhaps including heterocycles, dominated toxicity in their study. Kuehl et al. (1990) used a fractionation scheme to determine key chemicals in a creosote mixture that were acutely toxic to the water flea *Ceriodaphnia dubia.* They concluded that PCP and low molecular weight heterocycles in the mixture were probably the chemicals responsible. However, in a study of creosote-contaminated sediments in Finland, Hyotylainen and Oikari (1999) noted that, over time, the sediments became enriched in very high molecular weight PAHs, such as benzo[a]pyrene (MW = 252). They concluded that the high molecular weight PAHs appeared to be primarily responsible for the toxicity of the sediments to *Daphnia magna* and the photoluminescent bacterium *Vibrio fischeri*. In a study attempting to elucidate the fractions of weathered middle-distillate oils (a petroleum product that contains many of the chemical components in creosote) toxic to *Mysidopsis bahia*, Barron et al. (1999) concluded that

aromatic compounds (including "classic" PAHs and substituted PAHs) were not primarily responsible for toxicity. However, the relevance of that study to creosote is unclear.

Clearly, assessing the toxicity of complex mixtures such as creosote is very challenging. However, at least for PAHs common in creosote, considerable effort has focused on predicting the cumulative toxicity of PAHs in sediments, which is the key reservoir for creosote-derived chemicals in aquatic and marine systems. One of the proposed approaches that appears to be very useful, and that includes measurements and predictions of acute toxicity of PAHs, is the EPAH model of Swartz et al. (1995). This model incorporates 10-day acute toxicity tests (for acenaphthene, phenanthrene, and fluoranthene) with several sensitive marine and estuarine amphipods (including *Rhepoxynius abronius*), QSAR predictions of the toxicities of 10 additional PAHs (naphthalene, acenaphthylene, fluorene, anthracene, pyrene, benz[a]anthracene, chrysene, benzo[b]fluoranthene, benzo[k]fluoranthene, and benzo[a]pyrene), field chemistry data (sediment concentrations of these 13 PAHs and organic carbon content), and equilibrium partitioning to predict ISW concentrations of each PAH.

Swartz et al. (1995) predicted the 10-day LC_{50}s for these PAHs to range from 0.17 µg/L for benzo[k]fluoranthene to 3,500 µg/L for naphthalene. Their analyses and predictions support the notion that acute toxicity increases with increasing molecular weight (between 2 to 4 ring structures). This might be important in creosote-contaminated systems where higher molecular weight compounds appear to persist and eventually dominate the sediment profile relative to lower molecular weight compounds.

3.3.2 Phototoxicity

Concentrations of PAHs in surface waters rarely approach the concentrations associated with acute toxicity of these compounds under standard laboratory testing protocols (Eisler, 2000). A potentially important exception to this is the enhancement of the acute toxicity of some PAHs to various aquatic invertebrate and fish species examined under ultraviolet (UV) radiation. UV radiation is largely absent in normal indoor lighting. QSAR models have been developed that are reasonably accurate in predicting the phototoxic potencies of aromatic compounds (Ankley et al., 1997). Among the PAHs found to be highly phototoxic are anthracene, fluoranthene, pyrene, benzo[a]pyrene, benz[a]anthracene, and benzo[k]fluoranthene (Newsted and Giesy, 1987). The degree of enhancement of PAH toxicity is dramatic, with measures of acute toxicity generally increasing by one to two orders of magnitude. In animals, phototoxicity is thought to require bioaccumulation of the phototoxic chemical (Weinstein and Oris, 1999). It has also been shown that UV radiation can transform some PAHs (such as anthracene, phenanthrene, and benzo[a]pyrene) into products such as quinones that are more toxic than parent PAHs (Huang et al., 1993). Very recently, it has been reported that some photo-products of anthracene, such as

2-hydroxyanthroquinone, are estrogenic and occur at much greater concentrations than the parent compound in natural waters (Kurihara et al., 2005).

While many studies of phototoxicity have examined water-column-inhabiting organisms, the phenomenon has also been shown to occur in sediment-inhabiting invertebrates. For example, UV-enhanced phototoxicity has been demonstrated in oligochaetes (*Lumbriculus variegatus*) and amphipods (*Rhepoxynius abronius*) exposed via PAH-amended sediments (Ankley et al., 1994; Swartz et al., 1997), as well as in *Lumbriculus variegatus* and the amphipod *Hyalella azteca* exposed to field collected PAH-contaminated sediments (Ankley et al., 1994). UV exposure was also shown to markedly increase the acute toxicity of creosote-contaminated sediments from the Elizabeth River, Virginia, in larvae of the estuarine killifish, *Fundulus heteroclitus* (Meyer and Di Giulio, 2003).

The ecological relevance of UV-mediated PAH toxicity in the environment remains controversial, with some contending that factors operating in the environment ameliorate PAH phototoxicity (McDonald and Chapman, 2002). For example, humic acids that are often abundant in natural waters but generally minimal in most laboratory studies can reduce PAH bioaccumulation and attenuate UV light penetration, and thereby greatly reduce phototoxicity (Weinstein and Oris, 1999).

3.3.3 Carcinogenesis

From the standpoint of human health, the greatest concern for creosote constituents, particularly PAHs and aromatic amines, is the potency of many as mutagens and carcinogens. There is also a very substantial literature concerning chemical carcinogenesis, including PAHs, in fish. The mechanisms by which PAHs produce cancers are very similar in mammals and fish, and fish models have been used extensively in cancer research. Cancer, and the steps leading to it, comprise a key endpoint relevant to chronic exposures of vertebrates to PAHs, and might be important in assessing environmental risks of creosote in aquatic systems.

Many of the epizootics of cancer (predominantly liver neoplasms) described in fish populations in North America have been in areas contaminated by PAHs and associated aromatics such as N heterocycles (Landahl et al., 1990; Johnson et al., 1993; Baumann and Harshbarger, 1995; Myers et al., 2003). Among these are cases where creosote was strongly indicated as the source of the chemicals underlying the observed liver cancers, including cancers in English sole (*Pleuronectes vetulus*) in Puget Sound, Washington (Malins et al., 1985) and in *Fundulus* spp. in the Elizabeth River, Virginia (Vogelbein et al., 1990). In a more recent (2001) survey of Elizabeth River *Fundulus* spp. from the former Atlantic Wood creosote site (which closed in about 1990), Vogelbein and Unger (2003) observed hepatic neoplasm rates of 8% and altered hepatocellular foci (a precancerous lesion) rates of 65%. These rates were diminished somewhat

from earlier surveys. They provided quantitative data for sediment concentrations of 10 LPAHs and 8 HPAHs. Total mean PAH concentration was approximately 490,000 ng/g (ppb), dry weight, of which approximately 440,000 ng/g were HPAHs, including fluoranthene (approximately 125,000 ng/g), pyrene (approximately 71,000 ng/g) and the carcinogens benzo[a]pyrene (approximately 56,000 ng/g), benz[a]anthracene (approximately 41,000 ng/g), chrysene (approximately 60,000 ng/g), and dibenz[a,h]anthracene (approximately 12,000 ng/g). These data support the relative persistence of creosote-derived HPAHs, including carcinogenic PAHs, over time.

Laboratory studies have confirmed a causal link between PAHs and liver cancer and associated lesions in fish, including dibenzo[a,l]pyrene in rainbow trout (*Onchorhynchus mykiss*) (Williams et al., 2003), and benzo[a]pyrene and 7,12-dimethylbenzanthrace (DMBA) in Japanese medaka (*Oryzias latipes*) and guppy (*Poecilia latipes*) (Hawkins et al., 1990). There appears to be variability in sensitivity to chemical carcinogenesis among fish species. However, most species involved in epizootics are benthic species, such as English sole and winter flounder (*Pleuronectes americanus*) in marine systems, and brown bullhead (*Ameriurus nebulosus*) in freshwater systems. These species are frequently in contact with sediments; their life history behavior is thought to increase their exposure to carcinogens and thereby play a role in their sensitivity.

The mechanisms by which PAHs cause tumors appear to be similar in mammals and fish. They are briefly summarized here due to their relationship to the assessment of exposure and effects of creosote hydrocarbons in estuarine and marine systems. To initiate cancer, PAHs must first be metabolized into reactive products that can bind to or otherwise damage DNA. DNA damage occurs when the base sequence is altered and the alteration is passed along during subsequent cell divisions, i.e., a mutation has occurred (see review by Pitot and Dragan, 2001). For a resulting mutation to initiate cellular events leading to cancer, it must occur at a critical site in a gene that codes for a protein that serves a role in cellular growth, regulation, differentiation, or signaling. For example, benzo[a]pyrene has been shown to produce mutations in the DNA-binding regions of the tumor suppressor gene, p53, which leads to loss of the DNA damage surveillance function of the p53 protein (Denissenko et al., 1996). The processes occurring between initiation (DNA damage) and cancer, including promotion and progression, are complex and beyond the scope of this report.

However, mechanisms underlying initiation merit consideration here. Benzo[a]pyrene is the most well studied PAH in terms of DNA damage and cancer initiation, but mechanisms underlying these phenomena generally apply to other genotoxic PAHs also. Benzo[a]pyrene is oxidized mainly in the liver of vertebrates by cytochrome P450 (CYP); in fish the dominant enzyme catalyzing PAH oxidations is CYP1A (Stegeman and Hahn, 1994). Various oxidations to phenolics and epoxide metabolites can occur on the benzo[a]pyrene molecule, and the majority of these oxidations lead to their excretion via the bile due to enhanced water solubility. However,

specific metabolites can be highly reactive with cellular macromolecules, including DNA. In the case of benzo[a]pyrene, the 7,8-diol, 9,10-epoxide (benzopyrene diol epoxide, BPDE) is the best characterized genotoxic metabolite that covalently binds to DNA bases, such as guanine. Cells are equipped with DNA repair machinery that can excise and replace damaged bases. However, bulky adducts such as PAHs can elicit misrepair, with the wrong base inserted to replace the damaged one. If the cell containing the resulting altered base sequence remains viable and able to divide, mutation has occurred, with the potential for carcinogenesis, as described above.

These processes have been used in the biomonitoring of PAHs in the environment and in risk assessments (Myers et al., 1998). Expression of the CYP1A protein is regulated by the aryl hydrocarbon receptor (AHR). Ligands for the AHR can elicit very marked up-regulations of CYP1A that can be readily measured, for example by the EROD enzyme activity assay mentioned earlier, which is highly specific for CYP1A activity. A number of PAHs, particularly HPAHs, are effective AHR agonists that elicit CYP1A inductions. Thus, EROD activity provides a very sensitive biomarker for vertebrate exposures to PAHs, and it has been effective in field studies. This is important for PAHs because they are so readily metabolized by vertebrates and hence not amenable to standard tissue residue analysis. One downside of CYP1A measures is that there are other potent AHR agonists that also induce the protein, including polyhalogenated aromatics such as dioxins and PCBs. An assay that gets around this issue is the measure of PAH metabolites in the bile, which, though somewhat more difficult and less sensitive than EROD, can be very useful. DNA adducts to DNA can also be measured; this is accomplished in field studies principally by the ^{32}P-post-labeling assay (Myers et al., 1998). This is a much more involved assay than the previous two, but very powerful in that it has a clear relationship to cancer. Finally, hepatic anomalies, including pre-neoplastic lesions (lesions suggestive of carcinogenesis) and frank neoplasms can be quantified by standard histopathologic methods. Myers et al. (1998) provide a detailed example from Puget Sound of the integrated use of these markers for biomonitoring, in which the NOAA NMFS laboratory in Seattle, Washington, has been involved for many years.

Thus, the mechanisms underlying PAH metabolism and genotoxicity provide for an array of useful biomonitoring tools, or biomarkers, including:

- CYP1A
- Bile metabolites
- DNA adducts
- Lesions
- Cancer.

Sensitivity and ease of measurement generally decline from the top to the bottom of this list, but biological importance, and perhaps regulatory clout, increases from top to bottom.

3.3.4 Development

The effects of PAHs and related hydrocarbons on early life stage development have emerged as important issues relatively recently. In contrast to cancer, where concerns originated in the context of human health and subsequently spread to concerns for aquatic and marine systems, developmental effects have been primarily the purview of environmental studies. Field and laboratory investigations aimed at elucidating the impacts of the *Exxon Valdez* oil spill in Prince William Sound identified significant effects of petroleum hydrocarbons on development in endemic species such as the Pacific herring (*Clupea pallasi*) (Hose et al., 1996; Marty et al., 1997; Middaugh et al., 1998; Carls et al., 1999). Effects observed in exposed embryos included decreased hatching success, DNA damage, reduced heart rates, and gross morphological abnormalities such as scoliosis, pericardial edema, and cranio-facial abnormalities.

The morphological abnormalities observed are very similar to those described for fish embryos exposed to polyhalogenated hydrocarbons, particularly 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD; Walker et al., 1991). This effect, ultimately associated with drastic declines in Great Lakes lake trout populations (Cook et al., 2003), is referred to as blue sac disease, due to the appearance imparted by pericardial edema. Recent studies using zebrafish (*Danio rerio*) as a model have investigated the pathologies and mechanisms underlying TCDD-mediated blue sac disease. TCDD is among the most potent ligands for the AHR and is a very potent inducer of CYP1A. Using gene silencing techniques with antisense morpholinos to block translation of specific genes, it has been shown that activation of the AHR is required for the effects on cardiovascular development that underlie blue sac disease, but the role of CYP1A is unresolved (Prasch et al., 2003; Carney et al., 2004). Given that some PAHs are also effective AHR ligands and CYP1A inducers, petroleum and creosote might have similar developmental effects.

Incardona et al. (2004) investigated the effects of selected PAHs, including an S-substituted heterocycle, on cardiovascular development in the zebrafish. They investigated 2- to 4-ring PAHs, abundant in crude oil, singly and in mixtures. PAHs studied were naphthalene, fluorene, anthracene, phenanthrene, dibenzothiophene, chrysene, and pyrene. Among those, the chemicals exhibiting the most severe effects on cardiovascular development were phenanthrene, dibenzothiophene, and pyrene. Phenanthrene and dibenzothiophene produced particular effects on cardiac conduction resulting in reductions in circulation, which appeared primary to subsequent effects on cardiovascular development. The effects of pyrene were distinct and included anemia, peripheral vascular defects, and neuronal cell death; these effects resemble effects associated with TCDD. Coincidentally, 4-ringed pyrene is an AHR agonist (though far weaker than TCDD), while 3-ringed phenanthrene and dibenzothiophene are not. Incardona et al. (2004) suggested that pyrene may be acting through mechanisms similar to those by which TCDD acts, while the 3-ringed PAHs are directly perturbing atrioventricular conduction. They provide a convincing argument that narcosis is not a likely mechanism for the cardiovascular effects produced by these compounds. Relatedly, Billiard et al. (1999) reported blue sac disease

in rainbow trout and zebrafish embryos exposed to the alkylated 3-ringed PAH, retene (7-isopropyl-1-methylphenanthrene).

In a study directly addressing the effects of creosote on development, Vines et al. (2000) placed Pacific herring embryos in seawater containing creosote-treated wood, with seawater alone and seawater containing untreated wood as controls. Embryos collected from creosote-treated pilings in San Francisco Bay were also examined. This species spawns on a variety of substrates including pilings. In the laboratory studies, all embryos adhering directly to treated wood, and approximately 40-50% of those not adhering, failed to develop beyond the first few days of incubation. Surviving embryos displayed a 93% reduction in heart rate, and moderate to marked arrhythmia. Approximately 15-20% of the embryos exposed to creosote hatched, but all of these exhibited deformities including scoliosis, pericardial edema, and ascites. These effects were not observed in untreated wood or seawater controls. Also, similar effects were observed in embryos collected from the Bay, which displayed a 72% decrease in hatching success and similar deformities in surviving larvae.

Vines et al. (2000) measured total hydrocarbons diffusing from the treated wood by UV fluorescence, and individual components were measured in C-18 extractions of seawater by GC-MS. They concluded that 92.5% of this extract was composed of four 3-ringed PAHs – anthracene, phenanthrene, fluorene, and diphenlethyne – and lesser amounts of furans and non-aromatics. These results are somewhat surprising because the pilings used as the source for the treated wood had been placed in a marina about 40 years before the studies. Based on their results, the authors calculated the LC_{50} for hatching success to be 0.05 mg/L (total hydrocarbons in seawater). A sublethal exposure of 0.003 mg/L significantly reduced hatching success and increased abnormalities in surviving larvae (gross morphology and reduced heart rates); these effects were largely independent of three test salinities (8, 16, and 28 ppt – low, optimal, and high).

Wassenberg and Di Giulio (2004b) exposed *Fundulus* spp. embryos to dilutions of water extracts of sediments from the creosote-impacted portion of the Elizabeth River and observed significantly elevated EROD activities at all dilutions tested (ratios of extract to seawater ranged from 1:5000 to 1:4). They observed deformities, including pericardial edema, deformed hearts, and shortened tails, at the 1:4 dilution. Addition of the PAH-type CYP1A inhibitor α-naphthoflavone, which had no effects on development by itself, effectively inhibited EROD activities at all dilutions studied and greatly enhanced the teratogenic potency of the extract, with significant effects seen down to the 1:1000 dilution. Subsequent studies examining the interactive effects of a model PAH-type AHR agonist (β-naphthoflavone) and CYP inhibitor (α-naphthoflavone) demonstrated a potent synergy between these chemicals. Such synergy has also been demonstrated for creosote-associated PAHs (Wassenberg and Di Giulio, 2004a; Wassenberg et al., in press). For example, fluoranthene, carbazole, and dibenzothiophene were shown to be effective CYP1A inhibitors that markedly enhanced the effects of the AHR agonist

benzo[a]pyrene on cardiovascular development in *Fundulus* spp. Collectively, these studies bring into question current assumptions of additivity for PAH mixtures (Barron et al., 2004) and hence may have relevance for ecological risk assessments of these mixtures, including creosote.

The laboratory studies described above tested aqueous exposures; this is probably an important route of environmental exposure. For example, Petersen and Kristensen (1998) measured bioconcentration factors (BCFs) in the range of 3.25 (naphthalene) to 4.32 (benzo[a]pyrene) for several PAHs in the eggs of several freshwater and marine fishes following aqueous exposures. However, these compounds can be transferred from the mother to the egg, as well. For example, adult female *Fundulus* spp. exposed to benzo[a]pyrene transferred parent compound and metabolites to developing eggs, with the compounds transported in associated with vitellogen that entered the eggs during oogenesis (Monteverdi and Di Giulio, 2000a, 2000b).

3.3.5 Immunotoxicity

PAHs have been shown to impact function of the immune system in mammals (White et al., 1994). Studies with fish, some involving creosote exposures, show similar impacts. Payne and Fancey (1989) exposed winter flounder to sediments contaminated with a petroleum source of PAHs. They observed reduced numbers of melanomacrophage centers (primitive analogs of mammalian lymph nodes and important for the cellular immune system of fish) at exposure levels down to approximately 25 mg/kg, total sediment PAH. Faisal et al. (1991) found that anterior kidney and splenic leukocytes from *Fundulus* spp. captured from a creosote-contaminated site in the Elizabeth River had less cytotoxic activity against a tumor cell line than leukocytes from *Fundulus* spp. from a reference site. Karrow et al. (1999) studied the effects of liquid creosotes added to microcosms on immune responses of rainbow trout exposed for up to 28 days to a range of creosote concentrations (5-100 µl/L). Major effects were concentration-dependent reductions in leukocyte oxidative burst response and in the number of surface immunoglobin positive (sIg+) peripheral blood leukocytes. Major chemicals identified in the microcosms were fluoranthene, pyrene, fluorene, and anthracene, with fluoranthene and pyrene exhibiting the strongest associations with immune system effects. They calculated the lowest observable effects concentration (LOEC) for these effects to be 17 µl/L, corresponding to a total PAH concentration of 611.63 ng/L.

3.3.6 Community effects

Several microcosm and field studies have investigated the effects of creosote on phytoplankton and invertebrate communities. Sibley et al. (2001a, 2001b) examined the effects of marine-grade creosote on freshwater microcosms applied at concentrations ranging from 0.06 to 109 mg/L with a single application. These studies involved about 200 species of phytoplankton and

86 species of zooplankton. Creosote caused a concentration-dependent reduction in zooplankton abundance and number of taxa that were maximal at 5-7 days; most taxa recovered within the 83 days of observation following the application. Community composition varied with time and creosote composition, and interspecific competition appears to have played a factor in the population decline and reduced recovery of rotifera relative to cladocera and copepoda; however, rotifera were generally more tolerant to increased creosote exposure. Based on their results and measured concentrations of total PAH, the authors calculated the EC_{50} for total zooplankton abundance (day 7) to be 2.9 µg/L, and the no observed effects concentration (NOEC) for community effects ($NEC_{community}$) to be 3.7 µg/L. In contrast, creosote had no direct adverse effects on phytoplankton communities. Instead, increases in total abundance and number of taxa were observed, apparently in response to reduced grazing pressure by zooplankton. A similar study was subsequently performed, in which Douglas fir pilings impregnated with the same creosote used in the above studies served as the contaminant source (Sibley et al., 2004). The effects on zooplankton and phytoplankton communities in this study mirrored those observed with direct creosote applications; the NOEC for zooplankton community effects was calculated to be 11.1 µg/L total PAH.

In a controlled field study, Goyette and Brooks (1998, 2001) examined the effects of creosote-treated pilings placed in an uncontaminated marine system (Sooke Basin, Vancouver Island, British Columbia). They investigated the effects of these pilings on endpoints including 10-day amphipod (*Eohaustorius washingtonianus*) toxicity; bacterial toxicity (Microtox^{R}); echinoid fertilization; *in situ* assays in deployed blue mussels (*Mytilus edulis*) for growth, spawning, larval development, and PAH accumulation; and benthic community analysis. Some of these endpoints were tracked for up to 1,540 days post deployment of the pilings. In the first of these reports covering baseline studies and the first 535 days of the piling deployment, Goyette and Brooks (1998) reported significant sediment PAH accumulation up to 7.5 m downstream of pilings (18 µg/g and 7.5 µg/g total PAH at 0.5 m and 7.5 m, respectively), significant toxicity in laboratory tests with sediments collected within 0.65 m of pilings, and significant effects on PAH accumulation and reduced growth rates in mussels deployed within 15 cm of the pilings. Based on Washington State guidelines, they concluded that the greatest risks were posed by phenanthrene, followed by fluorene, acenaphthene, fluoranthene, and chrysene. In the later report covering sampling dates 1,360 and 1,540 days post-deployment, Goyette and Brooks (2001) reported sharply reduced PAH concentrations in sediments and mussels, and toxicity of sediments to amphipods was attributed to elevated sulfide due to anoxic conditions created by the pilings and associated biological communities.

3.3.7 Other effects

Other deleterious effects in estuarine and marine organisms have been noted for creosote and creosote-related compounds, including effects on growth and specific organ systems (Eisler,

2000; WHO, 2004). For example, Borthwick and Patrick (1982) found a 96-hour EC50 for reduced shell deposition in eastern oysters (*Crassostrea virginica*) of 710 µg/L. Also, Rice et al. (2000) observed severe impacts on growth in English sole fed polychaete worms that had ingested PAH-contaminated sediment (other contaminants in the sediment were not reported). Most of the sediment PAHs came from creosote released from a wood treatment plant. PAH concentrations in the worms were 11.3 ppm dry weight. Field-contaminated sediment fed to the worms was first diluted with clean sediment, producing a final concentration (3.3 ppm dry weight) that was 0.1% of the original sediment concentrations. Rice et al. (2000) conducted two similar experiments with this sediment. Although both experiments resulted in severe growth impairment, only the first was significantly different from control, possibly due to low statistical power in the second experiment.

Although a full review of all of these other toxicological endpoints is beyond this scope of this report, the response measures described in the preceding sections of this chapter appear to be appropriate and reasonably protective of aquatic receptors in evaluating wood-treating projects.

3.4 Conclusions: Biological Effects Concentrations

Based on the forgoing review of toxicities, acute toxicity to sediment-inhabiting invertebrates, and chronic effects on reproduction, development, the immune system, and liver (i.e., effects generally leading to liver cancer) in fish merit consideration as adverse effects thresholds (as concentrations in sediment or water). Section 3.4.1 discusses biological effect concentrations in surface water; Section 3.4.2 in sediments.

3.4.1 Biological effects concentrations – surface water

Of the quantitative data available, some are given in terms of sediment concentrations, and some are provided as water column or ISW concentrations. When chemical-specific concentrations are provided in one media, equilibrium partitioning based on K_{ow}s and/or K_{oc}s can be used to make predictions in other media. Unless otherwise noted, concentrations are presented below in terms of total chemical concentrations; these are generally the sums of compounds identified and measured in a given sample, usually TPAH. For water concentrations, some representative effects concentrations are shown in Table 3.1.

The biological effects thresholds for total PAHs in water (Table 3.1) fall within a relatively small range, considering the differences in organisms and endpoints. The lowest value is for immune system effects in rainbow trout (Karrow et al., 1999), but the relationship of this effect to fish health is unclear since the study did not address disease susceptibility. A slight increase in the ratios of liver weights to body weights were observed at 1.0 µg/L. Complete mortality was

Table 3.1. Effects thresholds for PAHs in surface water (concentrations in µg/L)

Organism	Exposure source	Toxicity endpoint	Concentration	Citation
Mysidopsis bahia	Elizabeth River, Virginia, sediment extracts	24-hr LC_{50}	180	Padma et al., 1999
Rhepoxynius abronius	Eagle Harbor, Washington, sediment extracts	96-hr LC_{50}	100	Swartz et al., 1989
Pacific herring	PAHs leaching from ~ 40 year old pilings	LC_{50} for hatching success	50	Vines et al., 2000
Zooplankton	PAHs leaching from pilings placed in microcosms	NEC for communities	11.1	Sibley et al., 2004
Zooplankton	Commercial creosote added to microcosms	NEC for communities	3.7	Sibley et al., 2001b
Pacific herring	PAHs leaching from ~ 40 year old pilings	Significant reduction in hatching success and increased abnormalities in surviving larvae	3	Vines et al., 2000
Zooplankton	Commercial creosote added to microcosms	EC_{50} for abundance	2.9	Sibley et al., 2001b
Trout	Commercial creosote added to microcosms	LOEC for immune effects	0.6	Karrow et al., 1999

observed within 3 days of the initiation of the experiment at the highest concentration of creosote tested, which appears to have been approximately 6 µg/L at the outset of the study. Control mortality was 23% in the course of the 28-day experiment, and no LC_{50} calculations were presented.

3.4.2 Biological effects concentrations – sediment

The biological effects of PAHs in sediment have been widely studied. A complete review of sediment PAH studies and a compilation of calculated threshold concentrations is outside the scope of this report. However, several studies summarize sediment quality guidelines (SQGs) for PAHs. Swartz (1999) includes many thresholds from many studies, and he compares those to his proposed SQG based on the sum of the PAH concentrations (∑PAH). The Oak Ridge National Laboratory compiled sediment toxicity benchmark data in 1997 (Jones et al., 1997). The U.S. EPA has provided guidance for determining sediment toxicity thresholds, including guidance for the use of equilibrium partitioning (e.g., Di Toro and McGrath, 2000) to determine

sediment quality guidelines (Hansen et al., 2003). Thresholds calculated according to this guidance do not carry regulatory authority at this time. Other U.S. EPA publications, such as Ingersoll et al. (2000) and Hellyer and Balog (1999), summarize sediment toxicity thresholds from many other studies, in an attempt to find some consensus.

Similarly, NOAA has compiled sediment toxicity thresholds for PAHs for consideration (not formally adopted), including an analysis of over 1,000 toxicity data points from the early 1990s (Long et al., 1998), and has published Screening Quick Reference Tables (SQuiRTs) that provide a quick reference to four freshwater sediment and five saltwater sediment threshold concentrations for many PAHs, as well as total PAHs (NOAA, 1999a). MacDonald et al. (2000) provide a thorough compendium of sediment quality benchmarks; Appendix III of their report contains well over 100 pages of sediment quality criteria and guidelines from the United States and Canada. Cormack (2001) provides a detailed review of sediment toxicity thresholds and their relation to sediment criteria in U.S. state and federal, and Canadian national and provincial, policies.

Table 3.2 lists an important subset of SQGs for PAHs that are well known and often cited, and/or promulgated and enforceable criteria. Each study in Table 3.2 contains threshold endpoints with acronyms for that threshold. An explanation of those thresholds follows.

- Effects Range-Low (ER-L) and Effects Range-Median (ER-M) are the lower 10th percentile and the 50th percentile, respectively, of a database of effects thresholds originally compiled by Long and Morgan (1991). NOAA guidance states that these are not derived as toxicity thresholds; rather, they were intended to be estimates of concentrations below which toxicity is least likely. They are meant to be used for ranking and prioritizing sites with contaminated sediments (NOAA, 1999b).

- Toxicity Effect Level (TEL) and Probable Effect Level (PEL) were derived for the promulgation of sediment quality criteria for the State of Florida (MacDonald, 1994). They divided their biological effects database into a database of effects concentrations and a database of no effects concentrations. They then calculated the TEL as the geometric mean of the 15th percentile of effects concentrations and the 50th percentile of no effects concentrations, and calculated the PEL as the geometric mean of the 50th percentile of the effects concentrations and the 85th percentile of the no effects concentrations (MacDonald, 1994). The Canadian Council of Ministers of the Environment (2003) copied MacDonald's Florida criteria for the Canadian Environmental Quality Guidelines, though they renamed the TEL the Interim Sediment Quality Guideline (ISQG).

Table 3.2. Sediment quality guidelines or criteria for marine/estuarine sediment. See text for explanation of acronyms and the thresholds they represent. MW = molecular weight (g/mol). Concentrations in ppb dry weight (see below).

		NOAA		FL and Env. Canada		WA	BC		Swartz, 1999	
Parameter	**MW**	**ER-L**	**ER-M**	**TEL**	**PEL**	**AET[a]**	**$SedQC_{scs}$**	**$SedQC_{tcs}$**	**∑PAH TEL[a]**	**∑PAH LC_{50}[a]**
Naphthalene	128.2	160	2,100	34.6	391	990	240	470	130	710
2-methylnaphthalene	142.2	70	670	20.2	201	380				
Acenaphthylene	152.2	44	640	5.87	128	660	80	150	30	150
Acenaphthene	154.2	16	500	6.71	88.9	160	55	110	40	230
Fluorene	166.2	19	540	21.2	144	230	89	170	170	900
Anthracene	178.2	85	1,100	46.9	245	2,200	150	290	210	1,140
Phenanthrene	178.2	240	1,500	86.7	544	1,000	340	650	290	1,550
Fluoranthene	202.3	600	5,100	113	1,494	1,600	930	1,800	690	3,710
Pyrene	202.3	665	2,600	153	1,398	10,000	870	1,700	900	4,810
Benzo(a)anthracene	228.3	261	1,600	74.8	693	1,100	430	830	210	1,110
Chrysene	228.3	384	2,800	108	846	1,100	520	1,000	310	1,690
Benzo(a)pyrene	252.3	430	1,600	88.8	763	990	470	920	330	1,790
Dibenz(a,h)anthracene	278.4	63	260	6.22	135	120	84	160		
Sum LPAH		552	3,160	312	1,442	3,700			870	4,680
Sum HPAH		1,700	9,600	655	6,676	9,600			3,060	16,460
Sum TPAH		4,022	44,792	1,684	16,770		10,000	20,000	3,930	21,140

a. Threshold concentrations originally in ppm organic carbon (OC). We assumed 1% OC to convert to ppb dry weight.

Sources: MacDonald, 1994; Swartz, 1999; NOAA, 1999b.
State of Washington: WAC 173-204-320.
Province of British Columbia: B.C. Reg 375/96 Schedule 9.
Canadian Council of Ministers of the Environment, 2003.

- Apparent Effects Thresholds (AETs) are thresholds above which statistically significant biological effects always occur (Swartz, 1999). These threshold concentrations are considerably higher than other thresholds, because they indicate concentrations where deleterious biological effects *will* occur, rather than concentrations where effects might occur, or concentrations below which effects are not likely to occur. The sediment criteria for Washington State are AETs (Gries and Waldow, 1996).

- British Columbia promulgated two separate criteria for PAHs in marine sediment, with a criterion for sediment in a "typical" environment ($SedQC_{tcs}$) and a more conservative criterion for sediment in a "sensitive" environment ($SedQC_{scs}$). These criteria are listed online in Schedule 9 of the Environmental Management Act Contaminant Sites Regulation (B.C. Reg 375/96) (http://www.qp.gov.bc.ca/statreg/reg/E/EnvMgmt/EnvMgmt375_96/375_96.htm; British Columbia Ministry of Labour and Citizen's Services, 2005). We have no guidance for the derivation of these criteria. The $SedQC_{tcs}$ concentrations are similar to the TEL from Florida, and the $SedQC_{scs}$ fall between the TEL and PEL (Table 3.2).

- While at the U.S. EPA, Swartz (1999) proposed sediment criteria based on the ∑PAH model of toxicity to marine and estuarine amphipods. Using data from other studies and translating other thresholds into his ∑PAH metric, Swartz derived a low effects threshold similar to the TEL that he called the "∑PAH toxicity threshold" (which we called the ∑PAH TEL in Table 3.2). He also derived a "∑PAH mixture LC_{50}" at which the concentrations of individual compounds, LPAH, HPAH, or TPAH are sufficient to cause 50% mortality in amphipods (Swartz, 1999).

Table 3.2 shows the wide disparity in sediment quality guidelines for PAHs. As discussed earlier, scientists at the NOAA NMFS Laboratory in Seattle, Washington, have examined the effects of pollutants, particularly PAHs, on benthic fish in Puget Sound for many years, allowing them to derive guidelines using their extensive data set. Here, we summarize some of their work. As described in Section 3.3.3, the liver is an important target for PAHs in some benthic fishes. The worst-case manifestation is cancer, but various biochemical and physiological effects precede the development of cancer, and many of these have been the focus of biomonitoring. Drawing on NOAA's data sets, Horness et al. (1998) developed "hockey stick" regressions to determine sediment thresholds for effects in benthic fish. The analysis focused on liver lesions in English sole in relation to TPAHs in sediments for approximately 30 sites in the Puget Sound comprising a wide gradient of PAH concentrations. Liver lesions evaluated were neoplasms, specific degenerative/necrotic lesions, such as megalocytic hepatosis and nuclear polymorphism, proliferative lesions, and foci of cellular alteration (FCA). Threshold concentrations are the sediment TPAH concentrations (based on dry weight of whole sediments) above which lesions are predicted to occur above background incidences. Threshold values ranged from 54 ng/g (ppb) for FCA to 2,800 ng/g for neoplasms. Values for other lesions ranged between 230 and 940 ppb.

The very low FCA value, however, was deemed insignificant because the confidence interval did not lie entirely within the data range, and it was suggested that FCAs may be a non-threshold response. Among the other thresholds, the most sensitive was 230 ppb, the threshold value for proliferative lesions.

Johnson et al. (2002) built on the hockey stick approach developed by Horness et al. (1998) to incorporate additional endpoints (DNA damage and endpoints associated with reproduction) and to explicitly calculate a sediment quality threshold for PAHs. Again, data for English sole provided the basis for their analysis, and they incorporated the results for liver lesions from the Horness et al. (1998) paper. Threshold values for three reproductive endpoints that were relatively sensitive (inhibition of spawning, infertile eggs, and abnormal larvae) were all calculated to be 630 ppb. The threshold value for DNA damage, measured as PAH-DNA adducts, was 288 ppb. Based on their overall analysis, Johnson et al. (2002) concluded that at sediment concentrations greater than 1,000 ppb, there is a substantial increase in the risk of liver disease and reproductive impairment, and suggested that 1,000 ppb be used as a sediment quality guideline for TPAH in estuarine systems. This SQG is less than the TPAH criteria shown in Table 3.2, though it is close to the 1,684 ppb TPAH criterion for the Florida TEL and Environment Canada's ISQG. Based on the foregoing, a sediment effects concentration of 1 ppm total PAH appears to be a reasonable screening value for use in the evaluation of potential creosote applications. Chapter 4 discusses how this threshold and other toxicity information presented in this chapter can be used in tandem with leaching and mobility information from Chapter 2 to assess the risk to the environment of various creosote treatments and applications.

4. Risk Evaluation

Having established in previous chapters that significant amounts of PAHs (and other contaminants) can leach from creosote-treated wood under environmentally relevant conditions, resulting in toxicity to organisms exposed to nearby surface waters and sediments, this chapter discusses further the risk to aquatic biota, including NOAA trust resources, from the use of creosote-treated wood. Two alternative lines of evidence are available to evaluate potential impacts. Section 4.1 briefly presents the results of previous ecological risk assessments of treated wood products, and Section 4.2 uses the results of the leaching and environmental distribution models described in Chapter 2 in a separate risk evaluation. Section 4.3 discusses the results of empirical laboratory and field studies (including many of the studies discussed in Chapters 2 and 3) designed to evaluate potential biological and/or ecological effects. Section 4.4 then discusses factors that should be considered for site-specific risk assessments.

4.1 Previous Risk Assessments

Sinnott (2000) developed a simulation model to evaluate ecological risks from creosote-treated wood. Using leaching rates from Ingram et al. (1982) and Hochman (1967), as cited by Kelso and Behr (1977), and degradation rates from the U.S. EPA (1979), the author estimated average daily concentrations of naphthalene, phenanthrene, and anthracene for the first month following immersion, and for the subsequent 11 months of immersion, in a 6-foot deep pond. Sinnott concluded that much of the leaching occurs shortly after immersion of treated wood in water, that the PAHs dissipate rapidly or are not present in high enough concentrations to cause harm, and that treated wood is not generally a toxicological problem in aquatic environments. The predicted sum of these compounds in the water column was below New York State water quality standards.

Brooks (1995) conducted an assessment of risks to T&E species in the Columbia River Basin from PAHs released from creosote-treated wood using a version of his leaching and environmental distribution model described in Chapter 2. The author compared predicted sediment PAH concentrations, given two pilings spaced 1 m apart, against then-current Washington State sediment quality criteria (total PAH of 1,330 in ppm TOC, or 25.3 ppm sediment dry weight, at 1.9% TOC), and made the following recommendations:

1. Creosote-treated wood products can be used without further risk assessment when:

    a. The redox potential discontinuity (RPD; a depth measure of the transition between oxic and anoxic sediment) is greater than or equal to 0.5 cm and current speeds are greater than 10.0 cm/sec.
    b. The RPD is greater than or equal to 1.0 cm and the sum of the RPD in cm and the current speed exceeds 7.0 cm/sec.

2. An individual project risk assessment should be required when:

    a. The RPD is less than 0.5 cm deep or when current speeds are less than or equal to 2.0 cm/sec.
    b. A project uses more than four pilings installed in a line parallel to the currents at inter-piling distances less than 1 m.
    c. The sum of the RPD and the current speed is less than or equal to 5.0 cm/sec.
    d. A new project is located within 10 m of an existing creosote-treated wood project.

3. Creosote projects should not be constructed in areas where current speeds are less than or equal to 1.0 cm/sec without further assessment.

4.2 Risk Assessments Using PAH Leaching Models

Chapter 2 described several PAH leaching and environmental distribution models that have been developed to predict the environmental concentrations of PAHs that result from the use of treated wood. Since the models contain many variables, specific scenarios must be assumed to generate model predictions. Table 4.1 lists model predictions for the specific scenarios described in Chapter 2 where the model authors were comparing the results of their models to measured PAH concentrations in specific field settings. Table 4.1 also includes the results for a specific model run for a hypothetical single-piling scenario.

As shown in Table 4.1, in most of the scenarios modeled, the predicted sediment PAH concentrations are well above the 1.0 mg/kg total PAH threshold discussed in Chapter 3. The single exception is the model predictions of Brooks (1997) for the pilings of the Westham Island Bridge in British Columbia that are at least 8 years old and are in an area of high tidal velocity. For the other scenarios, concentrations greater than 1.0 mg/kg are predicted for areas within several meters of the pilings. In the model for Belcarra Bay, British Columbia, Brooks (1997) predicts that the sediment concentration of 9 mg/kg at 1 m within the piling decreases to approximately 0 within 10 m of the piling.

Table 4.1. Environmental PAH concentrations predicted by PAH leaching and distribution models

Source	Scenario description	Environmental medium	Distance from piling/structure (m)	Predicted PAH concentrations mg/kg (dw)
Brooks, 1997	Belcarra Bay, BC; > 1.5 year-old pilings	Sediment	1 3 5 10 20 40	9 5.5 3 0 0 0
Brooks, 1997	Westham Island Bridge, BC; > 8 year old pilings; low salinity water; high tidal current	Sediment	0.5 2 5	0.6 0.2 0.1
Chapter 2 (using model of Brooks, 1997)	Single, 13 inch piling in seawater, 2.5 cm/s tidal current	Sediment	0.25 1	5.9 2.0
Goyette and Brooks, 1998, 2001	6-piling dolphins in Sooke Basin, BC; unweathered	Sediment	0.5 2.0	24 19
	6-piling dolphins in Sooke Basin, BC; weathered	Sediment	0.5 2.0	24 6

In addition to the results shown in Table 4.1, the model of Poston et al. (1996) also predicts that concentrations of PAHs in the water column may be toxic around newly installed pilings of relatively high density (as described in Chapter 2). However, the Poston et al. (1996) model is a simplistic model that most likely overestimates PAH concentrations in the water column. On the other hand, they use toxic thresholds for many individual PAH compounds that are most likely too high, based on the review provided in Chapter 3.

In conclusion, these results indicate that the available models on PAH leaching and environmental distribution predict that PAHs that leach from treated wood are present at concentrations that are predicted to be toxic to aquatic biota under realistic environmental scenarios. The models predict that these affects will be relatively localized around pilings (within approximately 5 m, depending on the specific conditions).

The predictive models applied here and described in Chapter 2 appear to capture the available laboratory data on PAH leaching reasonably well under certain conditions. However, for a variety of reasons discussed in Chapter 2, there is uncertainty in applying the results of laboratory study-based leaching models to field conditions. Depending on the specific field application, the laboratory-based leaching models appear to be more likely to under-predict than

over-predict leaching under field conditions, at least for the initial leaching period that occurs within the first hours and days after construction. Furthermore, there is much uncertainty in modeling actual environmental concentrations from the leaching study models, as also described in Chapter 2. Therefore, the results of the predictive risk assessment models should be interpreted carefully, as they may have substantial (and unquantified) uncertainty. Finally, by their nature, models use simplifying assumptions that may miss uncommon but important conditions that result in temporary or localized concentration spikes that could affect marine organisms.

4.3 Laboratory and Field Studies

Several laboratory and observational field studies have been performed to evaluate the potential impacts of creosote-treated wood products on aquatic biota. These studies allow for a direct assessment of the potential adverse effects and ecological risk associated with use of creosote-treated wood in aquatic habitats.

This section evaluates the potential impacts of creosote leaching first by looking at three highly relevant areas of research: (1) large-scale studies showing creosote leaching from sites where dozens or hundreds of pilings are clustered together, specifically the Charlestown Navy Pier in Boston, Massachusetts, and the Naval Station San Diego (NAVSTA) in San Diego Bay, California; (2) creosote leaching studies performed under the auspices of Dr. Kenneth Brooks, including a study of creosote leaching effects in the Fraser River Estuary in British Columbia and several subsequent studies; and (3) laboratory studies of creosote leaching in aquatic microcosms by University of Guelph researchers. We then provide reviews of several other field and laboratory studies that provide ancillary evidence of creosote leaching in the environment.

4.3.1 Large-scale studies: Charlestown Navy Pier and Naval Station San Diego

In 1987, the National Park Service (NPS) replaced about 90 creosote piles at Pier #2 of the Charlestown Navy Yard in Boston (Graham and Johnsen, 2002). The NPS rejected many of the piles because of insufficient retention. The re-treated piles were then over-impregnated to an average retention that was 25% higher than BMPs specified. The result was a noticeable slick of creosote from the new pilings after installation (Graham and Johnsen, 2002).

Costa and Wade (1989) collected samples and provided analyses of the Pier #2 creosote leachate, including chromatography analyses and sea urchin toxicity tests. The chromatography analyses showed that the surface sheen emanating from the pier was unquestionably creosote, as the peaks in the slick matched the peaks in creosote for all major PAHs except the lightest, most volatile ones. Dissolved PAH concentrations were high in the surface sheen and relatively high in water

directly above the sediment, but dissolved PAHs were undetectable in samples from within the water column below the surface slick. Target PAH concentrations in the water near the sediment were 0.87 to 1.7 µg/L, about 8-14 times higher than the concentrations at a control site near a different pier. The PAH concentration of the surface slick was up to 5,350 µg/L – the authors stated that "it can be presumed that the surface slick is toxic to organisms residing in the surface layer" (Costa and Wade, 1989).

The concentrations of the target creosote PAHs in sediment near Pier #2 pilings were 250 times greater than the concentrations at a control site, with total target PAH concentrations as high as 6,390 µg/g dry weight. The samples were described as having a strong creosote odor (Costa and Wade, 1989). Surface sediment PAH concentrations decreased rapidly with distance from the pilings, reaching background concentrations between 6 and 21 feet from the creosote pilings.

Costa and Wade (1989) collected the leachate in the surface slick and attempted to fertilize sea urchin eggs in the presence of the leachate. Over 50% of the eggs failed to fertilize in all tests in which the test water contained at least 1% leachate. The authors calculated the LOEC at 0.38% leachate, or 20 µg/L PAH, and the NOEC at 0.19% leachate. Their results show that at 0.09% leachate (4.8 µg/L PAH), the most dilute test they performed, 21% of the eggs failed to fertilize, compared to 0.3-0.7% failure using control water. The difference was not statistically significant.

In summary, the approximately 90 new pilings at the Charlestown Navy Pier exuded a surface slick with PAH concentrations of 5,350 µg/L, several orders of magnitude greater than concentrations predicted to impact sea urchin reproductive success. Samples of the slick caused significant toxicity to sea urchins in a reproductive endpoint test at mixtures of 0.38% slick. PAH concentrations in water near the sediment interface were up to 1.7 µg/L, and were not detected in the water column between the sediment and water surface. The sediment near the pilings contained PAH concentrations 250 times greater than the concentrations at a nearby control site (Costa and Wade, 1989).

At NAVSTA in San Diego Harbor, the U.S. Navy made important operational changes in the 1990s in an effort to reduce PAH contamination in San Diego Bay. Specifically, the Navy stopped discharging bilge water directly to the bay, and they made a concerted effort to replace all creosote pilings at the base. In 1997, Katz (1998) examined the changes in dissolved PAH concentrations in the bay after approximately 50% of the creosote pilings had been replaced with plastic, concrete, or untreated pilings.

Katz (1998) compared historical surface water PAH concentrations from 1990 to 1994 with concentrations measured in the summer and fall of 1997. In all studies from both time periods, PAH concentrations were higher near the Navy piers than in other areas within San Diego Bay. In the early 1990s, the two sample sites adjacent to the piers contained average total PAH concentrations of 1.2 and 1.7 µg/L, including a sample at one site that exceeded 8 µg/L in a

surface slick. The average total PAH concentration from 36 samples collected near NAVSTA piers from 1990 to 1994 was 1.1 µg/L, compared to an average of 0.16 µg/L for the 65 samples collected away from the piers. In 1997, after 50% of the pilings were replaced with non-creosote alternatives, the total PAH concentrations in the surface water near the pilings were an order of magnitude lower than the average concentrations between 1990 and 1994, with concentrations between 0.1 and 0.2 µg/L (Katz, 1998). Total average PAH concentrations decreased to 0.12 µg/L at NAVSTA and 0.06 µg/L at non-NAVSTA sites in 1997. Chromatograms of the water samples showed PAHs that matched the pattern for creosote from samples at the Navy pier (Katz, 1998). Unfortunately, this study did not include analyses of sediments or biota in the bay. However, the study does suggest that creosote pilings at NAVSTA were at least partly responsible for elevated PAH concentrations in the surface water of San Diego Bay, and that the program to replace those pilings led to measurable decreases in dissolved PAH concentrations in the Bay.

4.3.2 Fraser River estuary and related studies

In 1994, EVS Consultants (1994) conducted a creosote evaluation project for the Fraser River Estuary Management Program in British Columbia. They examined sediment PAH concentrations and conducted toxicity tests on amphipods and bacteria near creosote piling installations at Belcarra Bay and at Westham Island in the Fraser River Estuary. The pilings at the Belcarra Bay wharf ranged from 2 to 20 years old, and the pilings closest to the Westham Island study site were 8 years old. At Belcarra Bay, sediment PAH concentrations within 10 m of the pilings were significantly higher than reference concentrations, and the survival of amphipods and bacteria exposed to the sediment in laboratory toxicity tests was significantly diminished. Total sediment PAHs were as high as 19.7 µg/g, about 10 times higher than reference concentrations. By contrast, at Westham Island, total sediment PAH did not exceed 0.5 µg/g and was not significantly different than background concentrations. Amphipod survival was similar in sediment collected from the Westham Island site and the control site. EVS Consultants (1994) attributed the different results at Westham Island compared to Belcarra Bay to a higher water flow rate at Westham Island that carried leached creosote away from the site. Furthermore, the newest pilings at the Westham Island site were over 8 years old, compared to the 2 year old pilings at the Belcarra Bay site.

The EVS study in the Fraser River estuary led to a Phase II study (Goyette and Brooks, 1998, 2001) in the Sooke River Basin on Vancouver Island, examining more closely the possible impacts of creosote leaching. Sooke Basin conditions were similar to Belcarra Bay in the Fraser River Estuary but with less background PAH contamination. The Sooke Basin site was away from intense human activity, with weak tidal currents (1.89 cm/s) and no freshwater runoff input. The study consisted of three tests using 6-piling dolphins, one containing a dolphin with newly treated pilings, one with 8 year old weathered pilings, and one with untreated pilings. As at

Belcarra Bay, sediment PAH concentrations near the creosote-impregnated test dolphins in Sooke Basin were elevated compared to background. Table 4.2 shows the sediment PAH concentrations near the weathered piling dolphins. Figure 4.1 shows sediment PAH concentrations near the newly treated dolphins, including the changes in PAH concentration with time (Figure 4.1a) and the changes in PAH concentration with distance from the pilings (Figure 4.1b). It should be noted that surficial samples in this study were collected as the top 2.0 to 2.5 cm, and therefore evaluation of the accumulation of creosote in the surficial sediments at a scale finer than the top 2.0 to 2.5 cm is not possible. Nevertheless, the results of the study document that increased creosote accumulation in sediment was observed downstream of the pilings.

Table 4.2. Summary of sediment PAH concentrations near dolphins containing six weathered creosote pilings in the Sooke Basin, British Columbia. (+) is downstream, (-) is upstream of the pilings. Concentrations immediately before installation are listed as 0 days since installation. PAH concentrations in µg/g.

Distance from pilings (m)	Days since installation	Mean % TOC	Mean LPAH	Mean HPAH	Mean TPAH
0	0	0.9	0.03	0.1	0.13
0.5	0	1	0.04	0.16	0.19
0	384	0.7	5	42.3	47.4
+0.5	14	1.3	34	71	105
+0.5	180	0.9	2.9	11.2	14.1
+0.5	180	1.3	6.2	11.6	17.8
+0.5	384	0.7	1.3	9.5	10.8
+0.5	384	0.6	4.6	29.2	33.8
+2.0	14	1.3	0.8	2.1	2.9
+2.0	180	1.3	0.9	3.9	4.8
+2.0	384	0.6	0.8	5.6	6.3
+2.0	384	0.9	3	12.2	15.3
-2.0	14	1.2	0.6	0.7	1.3
-2.0	180	1.1	0.4	1.1	1.5
-2.0	384	0.7	0.5	3.8	4.3
+5.0	384	0.6	0.3	2	2.3
+10.0	384	1.2	0.1	0.8	0.9

TOC = total organic carbon; LPAH, HPAH, and TPAH = light, heavy, and total polycyclic aromatic hydrocarbons, respectively.

Source: Goyette and Brooks, 1998.

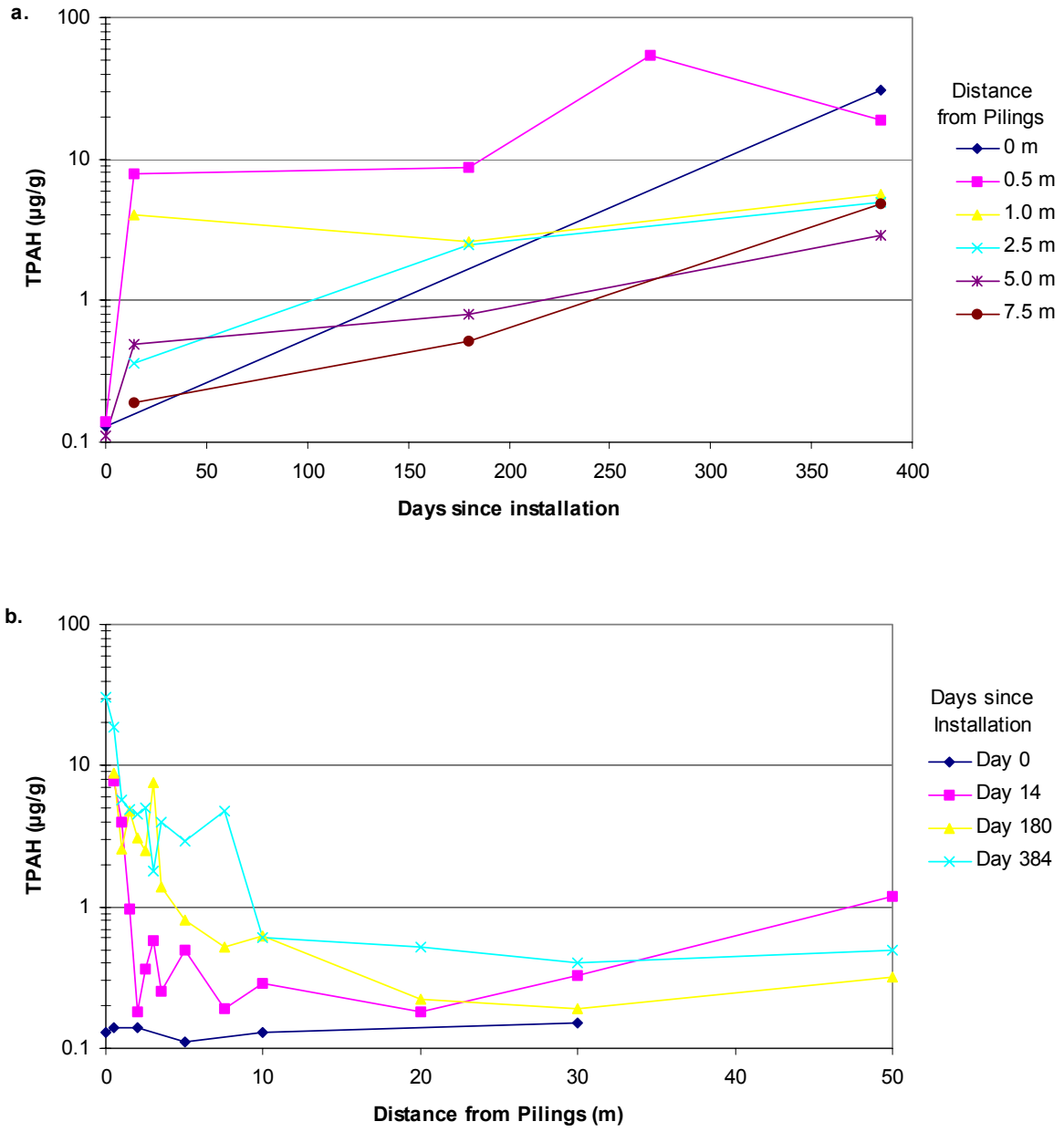

Figure 4.1. Sediment total PAH concentrations downstream (downcurrent) of newly treated pilings in the Sooke Basin study, as they varied with (a) time and (b) distance from the pilings. Note the logarithmic scale for TPAH.

Source: Goyette and Brooks, 1998.

The data in Table 4.2 and Figure 4.1 clearly show increases in sediment PAH concentrations within two weeks after installation, with elevated PAH concentrations remaining over a year after installation and extending up to 50 m from the pilings. The following summarizes the conclusions of Goyette and Brooks (1998) after one year of data from the Sooke Basin study:

- Two weeks after installation of the weathered piling dolphin, the sediment PAH concentration 0.5 m downstream of the pilings (where downstream is the dominant direction of current flow) was over 100 μg/g dry weight, nearly three orders of magnitude greater than the concentration before installation (Table 4.2). The authors suggest that physical abrasion of the treated wood surface during installation may have caused an initial release of creosote.

- Surface sediment PAH concentrations were statistically significantly higher than baseline concentrations to a distance of 7.5 m from the newly treated pilings. Smaller PAH increases occurred at a distance of 50 m (Figure 4.1b).

- The proportion of HPAH to LPAH in the sediment increased notably between Day 14 and Day 384 (Table 4.2), suggesting the preferential loss of the LPAH through solubilization.

- The surface sediment PAH concentrations were highly variable, with poor correlation amongst replicate samples in some cases.

- No significant changes in benthic community structure were observed.

- Toxicity tests on mussels (*Mytilus edulis*) showed slightly elevated PAH body burden and slightly less growth in the presence of sediments contaminated with PAHs leached from the pilings. There were no adverse effects to mussel survival or viability.

In 2001, Goyette and Brooks (2001) published an addendum to the Sooke Basin study, showing results four years after the installation of the dolphins. This study showed a significant decline in PAH concentrations between Year 1 (384 days) and Year 4. An active invertebrate community had become established, with the pilings serving as an artificial reef. Mussels living on pilings contained PAH concentrations less than background concentrations. The limiting factor on biota was reported to be low oxygen and high sulfur due to the accumulation of detritus near the pilings.

Dr. Brooks, who was part of the steering committee for the Fraser River Estuary study and was co-author of the Sooke Basin study, published several more reports examining creosote leaching into the environment. They include a study showing the leaching of PAHs from bridge timbers into an aquatic environment (Brooks, 2000) for the USDA Forest Service (USFS), a study of PAH leaching from creosote timbers in Puget Sound (Brooks, 2003) for the Creosote Council, an

industry group, and a study examining PAH leaching from railroad timbers into wetlands (Brooks, 2004b) for the USFS. His results and conclusions for each of these studies are similar to the results and conclusions from Belcarra Bay (EVS Consultants, 1994), and the Sooke Basin study (Goyette and Brooks, 1998, 2001). Brief summaries of these studies are provided below.

Brooks examined sediment PAH concentrations in Pipe Creek, Indiana, immediately downstream of two bridges built with creosote timbers (Brooks, 2000). At the time of the study, one bridge was 2 years old and one was 17 years old. The results of the study showed the following:

- Sediment PAH concentrations at the older bridge increased from undetectable (< 0.11 µg/g) upstream of the bridge to a maximum of 2.3 µg/g 1.8 m downstream of the bridge. Beyond 1.8 m, PAH concentrations generally decreased with distance downstream of the bridge, though were still detectable (0.5 µg/g) at 10 m, the most downstream location.

- Sediment PAH concentrations at the newer bridge increased from undetectable (< 0.23 µg/g) upstream of the bridge to a maximum of 5.5 µg/g 1.8 m downstream of the bridge. PAHs were still detectable (0.5 µg/g) at 6 m downstream, and were undetectable at 22.8 m downstream.

- The highest PAH concentrations downstream of the newer bridge exceeded toxicity threshold effect levels, whereas none of the concentrations downstream of the older bridge did.

- Despite the toxicity threshold exceedences, the biological data that was collected did not reveal adverse effects on biota from PAHs at either the newer bridge site or the older bridge site (Brooks, 2000).

Brooks (2003) conducted a comprehensive study of sediment PAH concentrations and the effects on biota at several locations in Puget Sound, Washington State, in a manner similar to the Sooke Basin study. This study included wharfs with dozens of creosote-impregnated piers and sites with dolphins similar to Sooke Basin. The results, summarized below, are similar to the results of his previous studies.

- At a long wharf at Fort Worden, total PAH concentrations in sediment were 16 µg/g closest to the pier, 11 µg/g at 2.5 m distance, and 5.4 µg/g at 7.5 m distance. Background concentrations were 0.5 µg/g or less. Within 2 m of the densest cluster of pilings, sediment PAH concentrations were as high as 34 µg/g, compared to 0.07 µg/g at background sites, though some of the PAHs at 2 m were specific HPAHs that are more

characteristic of heavy oil than they are of creosote. Samples from between 0.5 and 2 m from the pilings exceeded the Washington State SQGs for PAHs.

- Near a three-piling dolphin at Fort Ward, the sediment PAH concentration was 11.7 µg/g within 0.5 m of the dolphin and decreased to 0.7 µg/g at 2 m. Background samples contained elevated PAHs at this site.

- Sediment PAH concentrations from around a single piling at Fort Ward and around a three-piling dolphin at Port Townsend's city pier were not significantly higher than at reference sites. However, the reference sites themselves contained PAHs in the sediment (1.9 to 7.6 µg/g).

- At Fort Worden, weak negative correlations were found between the abundance of some invertebrate species and PAH concentrations. Weak positive correlations were also found, particularly between nematodes and PAHs, most likely because nematodes tend to populate the organic-rich sediment found at the base of the pilings. The author states that the effects of biodeposits from the abundant epifaunal community that populates the pilings has a much larger effect on the overall benthic community than do the PAHs that leach from the pilings and accumulate in the sediment.

The most recent study from Dr. Brooks (Brooks, 2004b) examined creosote leaching from railroad ties in wetland areas, with an examination of both PAHs migrating to the railroad bed ballast and PAHs migrating into the wetland. The results of this study showed very little PAH contamination in the wetland. In the second year of the study, PAH concentrations increased by an average of 0.3 µg/g, which was not statistically significant. In 16 wetland sampling events over the course of two years, PAHs were only detected once, and the concentrations were well below toxicity thresholds.

4.3.3 University of Guelph microcosm studies

Researchers at the University of Guelph in Ontario conducted laboratory (microcosm) studies of the distribution of contaminants within the microcosm when exposed to creosote. These include a study where liquid creosote was added directly to the water (Bestari et al., 1998a, 1998b; Sibley et al., 2001b), and another study where recently treated Douglas fir pilings were added to the microcosm (Bestari et al., 1998a; Sibley et al., 2004).

Bestari et al. (1998b) applied 14 different doses of liquid creosote to an aquatic microcosm in a 12,000 L tank, then over the course of several weeks measured the concentration of 15 priority PAHs in water, sediment, and on PVC strips within the sediment. In a concurrent study (Bestari et al., 1998a), they applied 6 different doses of creosote to similar microcosms, but rather than

apply creosote directly, they used recently-impregnated Douglas fir pilings where the creosote leached from the pilings. The results of each study were similar and are summarized below.

- PAH concentrations in water were dose-dependent. In the timber study, the dissolved PAH concentration ranged from 7.3 µg/L with one-half of a piling in the microcosm up to 97.2 µg/L with 6 pilings in the microcosm.

- PAH concentrations in water decreased exponentially with time after initial dosage. The PAH concentration after the highest liquid creosote dosage decreased from 5,800 µg/L on Day 2 to 13.9 µg/L on Day 84. The 6-timber treatment decreased from the maximum of 97.2 µg/L at Day 7 to 6.7 µg/L at Day 84.

- When liquid creosote was applied at concentrations exceeding 590 µg/L, sediment PAH concentrations increased until Day 28, then decreased thereafter in all but the highest dose. The increase in sediment PAHs was dose-dependent. In contrast, in the piling treatment study there was no increase in sediment PAHs at any of the treatment levels at any time during the study or at any distance from the pilings, though none of the water concentrations approached 590 µg/L in that study. The authors suggested that in the piling treatments the HPAHs adsorbed to the PVC liners, and LPAHs were lost to volatilization and possibly to biodegradation in sediment occurring in equilibrium with the PAH removal rate.

Sibley et al. (2001b, 2004) report the effects of these creosote treatments on phytoplankton and zooplankton communities. The responses were similar for both liquid creosote application and creosote leaching from pilings. Zooplankton abundance decreased after the introduction of creosote in a dose-dependent manner, just as the aqueous PAH concentrations increased in a dose-dependent manner. At concentrations greater than 1,100 µg/L, which were found only in the liquid creosote study, zooplankton species composition changed significantly, perhaps due to a drop in rotifer density (Sibley et al., 2001b). For liquid creosote, the estimated NOEC for the zooplankton community was 13.9 µg/L total PAHs after 5 days and 5.6 µg/L total PAHs after 7 days (Sibley et al., 2001b). For leached creosote, the NOEC was 11.1 µg/L total PAHs (Sibley et al., 2004).

By contrast, the phytoplankton abundance and diversity increased in all treatments in both studies, with phytoplankton abundance increasing to up to twice that in the control microcosms (Sibley et al., 2001b, 2004). The authors attribute this to decreased zooplankton grazing pressure and possibly to growth stimulation from compounds in the creosote. Based on these data, the authors conclude that creosote leaching from pilings may cause short-term toxicity to limnetic or benthic communities shortly after deployment, but that long-term effects are unlikely as PAH concentrations decrease exponentially with time (Sibley et al., 2004).

4.3.4 Other studies

Over the past 30 years, many studies have examined the leaching of creosote from impregnated timbers and the resultant environmental concentrations of PAHs near creosote timbers. Short summaries of some of these studies are included below (some are also discussed in Chapter 2, as relevant to leaching).

- Zitko (1975) found elevated PAH concentrations in mussels, clams, periwinkles, and whelks near a wharf in New Brunswick, Canada. Zitko states that creosote-treated wharf timbers are the only source of PAHs to the bay.

- Dunn and Stich (1976) reported benzo(a)pyrene concentrations 3 to 4 times higher in mussels growing on creosote timbers than on nearby rocks and concrete in Vancouver Harbor, British Columbia.

- Ingram et al. (1982) conducted studies of creosote leaching from treated pilings in laboratory tanks, using many different treatments. Dissolved PAH concentrations increased for all 15 PAHs they studied, in both fresh and saltwater. Six compounds (naphthalene, phenanthrene, acenaphthene, dibenzofuran, fluorene, and 2-methylnaphthalene), which comprised 70-80% of their test creosote, were the dominant contaminants in the water. Higher concentrations of leached PAHs were found in freshwater treatments than in saltwater treatments, and higher concentrations emanated from newly treated timbers than from aged timbers. Maximum PAH concentrations occurred within 48 hours of treatment, then concentrations decreased for the remainder of the study.

- Harrington and Crane (1994) found slightly elevated PAH concentrations in clams at and just downstream of a ferry dock in the Sacramento River delta. PAHs were not detectable in surface water or in clams upstream of the dock. PAHs were as high as 0.45 mg/kg in clams on the dock, and 0.20 mg/kg in clams downstream of the dock. The authors concluded that these concentrations were not sufficient to cause an adverse effect to the clams.

- Wendt et al. (1996) found slightly elevated PAH concentrations in sediment and oysters growing near creosote-impregnated dock pilings in South Carolina compared with control sites. Oyster growth was somewhat less near the pilings compared to control sites. None of the differences were statistically significant.

- Graham and Johnsen (2002) describe a surface slick from a dock in Poughkeepsie, New York, that required the dock owners to deploy a boom to contain the spill. Creosote-impregnated pilings at the dock were given as the cause of the slick. No specific data were given.

4.3.5 Conclusions

Overall, the laboratory and field studies described above indicate that treated wood structures can leach PAHs and other toxic compounds into the environment. However, the degree of PAH accumulation to sediment associated with these structures appears to be relatively minor in many settings, particularly in well-circulated waters and over time. PAH accumulation also appears to be relatively limited spatially (within approximately 10 m of the structure) and has not generally been associated with measured, significant, biological effects except in close proximity to the structures. The duration of any biological effects also appears to become attenuated within several months of construction (the time period when leaching rates are likely to be highest).

Nevertheless, there are several factors that suggest that a precautionary principle might be applicable to certain treated wood uses. First, the above studies typically have evaluated responses at the community level (e.g., the benthic invertebrate studies) or to tolerant life stages (e.g., adult oysters and mussels). However, the level of environmental protectiveness applied to T&E species (such as endangered salmonids) should occur at the *individual* rather than the *population* or *community* level. Moreover, field studies have indicated that PAHs can accumulate to potentially deleterious concentrations in poorly circulated water bodies or when the density of treated wood structures is high compared to the overall surface area of the water body. As a result, site-specific evaluations of risk should be conducted for treated wood projects that are proposed for areas containing sensitive life stages, species of special concern, or where water circulation and dilution is potentially low. We discuss considerations associated with such site-specific risk assessments below.

4.4 Factors to be Considered in Aquatic Risk Assessments

The analyses presented in this report demonstrate that PAHs that leach from creosote-treated wood have the potential to accumulate in abiotic media and aquatic biota and to cause toxicity to biota. However, the risk of adverse toxicological effects may be limited in spatial scale and time in many environmental settings and treated wood uses, and vary dramatically depending on case-specific factors such as the nature of the wood and its treatment, environmental conditions, and species of concern. Therefore, in certain settings, site-specific risk assessments should be performed to ensure that projects avoid unnecessary risks to sensitive species or species of

special concern. Conditions that should prompt consideration of a site-specific risk assessment include:

- Low current velocities (e.g., current speeds < 1 cm/sec) and/or relatively little expected mixing coupled with a relatively high density of construction materials

- The presence of sensitive life stages (typically larvae and juveniles) of aquatic organisms, particularly T&E or special status species, in the project location.

When conducting such site-specific risk assessments, Hutton and Samis (2000) identify the following factors that should be considered:

- **Background water quality variables such as salinity**

  The salinity of the receiving environment should be considered because leaching increases with decreasing salinity, as in estuarine environments.

- **Current velocity and direction**

  Although total leaching rates from treated wood can be relatively low, potential environmental effects will be dictated by local water mixing, with poorly mixed waters at greater risk. Information on current velocities – at the specific micro-environment – of the project location (including the influence of the structure itself on ambient current velocities) should be developed and integrated into a site-specific risk evaluation.

- **Proximity to sensitive fish habitat**

  The presence of sensitive life stages, especially T&E species or their essential prey species, should prompt an evaluation of potential risks at that location. Essential fish habitats for Pacific salmon include all streams, lakes, and other water bodies currently or historically accessible to salmon. This includes essentially all estuarine and marine waters of the Pacific Coast. The most sensitive life stages for these species are fry (particularly post swim-up) and juveniles. Because the initial leach rates are higher for treated wood, risk assessments should consider the timing of PAH releases relative to periods when sensitive life stages of fish are present.

- **Timing of proposed construction**

  Because initial leach rates tend to be greater, the timing of proposed construction should be considered with respect to the presence of sensitive life stages of aquatic receptors, water flow rates and temperature, environmental and climatic factors that can influence

mixing and dilution, and the relationship between season, annual hydrograph, and water quality conditions.

- **Size of proposed structure**

  As discussed previously, environmental effects are likely to be greatest when the size of the proposed structure is large relative to the receiving environment. Factors to consider include number and size of pilings, surface area of exposed wood area relative to a mixing zone, density of pilings relative to the mixing zone (to evaluate potential behavioral avoidance responses), and potential effects of structure size on current flows.

- **Application methods**

  Treatment and application methods should be confirmed to meet industry BMPs.

- **Proximity of other treated-wood structures and other sources of contamination that may contribute to cumulative effects**

  In evaluations of site-specific risks, assessments should consider potential effects in light of the cumulative effect of the proposed structure relative to other existing environmental perturbations at the site.

In addition, the Los Angeles District of the U.S. Army Corps of Engineers uses standard permit conditions that apply to creosote-treated pilings placed in navigable waters of the U.S. The standard conditions include the following (personal communication, D.J. Castanon, U.S. Army Corps of Engineers, Los Angeles District, November 30, 2004).

- Creosote-treated pilings shall not be placed in navigable waters or waters of the United States unless all of the following conditions are met:
  - The project involves the repair of existing structures that were originally constructed using wood products.
  - The creosote-treated pilings are wrapped in plastic.
  - The use of plastic-wrapped creosote pilings is restricted to marine waters.
  - Measures are taken to prevent damage to plastic wrapping from boat use. Such measures may include installation of rub strips or bumpers.
  - The plastic wrapping is sealed at all joints to prevent leakage.

- The plastic material is expected to maintain its integrity for at least ten years, and plastic wrappings that develop holes or leaks are repaired or replaced in a timely manner.

These conditions were developed by the Army Corps of Engineers in coordination with the California Department of Fish and Game and NOAA. Furthermore, as presented in Chapter 1, other agencies with jurisdiction over marine waters have begun replacing and restricting the use of creosote-treated wood, including: Washington State Ferries; the Port of Port Angeles, Washington; the Oregon Department of Environmental Quality, State Marine Board; the California Coastal Commission; the Delaware Department of Natural Resource and Environmental Control; the New York State Legislature; and the Rhode Island Coastal Resources Management Program.

Theses various initiatives, restrictions, and standard permit conditions show that regulatory agencies are increasingly recognizing that creosote treatments in marine environments can cause ecological harm under common enough circumstances that new structures should avoid the use of creosote-treated wood, and creosote should be isolated from the environment wherever it is used. Based on the findings of this report, that creosote moves into the environment under a variety of realistic conditions, and environmental levels of contaminants originating from creosote-treated wood are often toxic, precautions to avoid creosote-treated wood where practical, and measures to isolate potential toxic effects appear to be justified. We recommend that similar precautions be implemented by regulating agencies throughout the United States.

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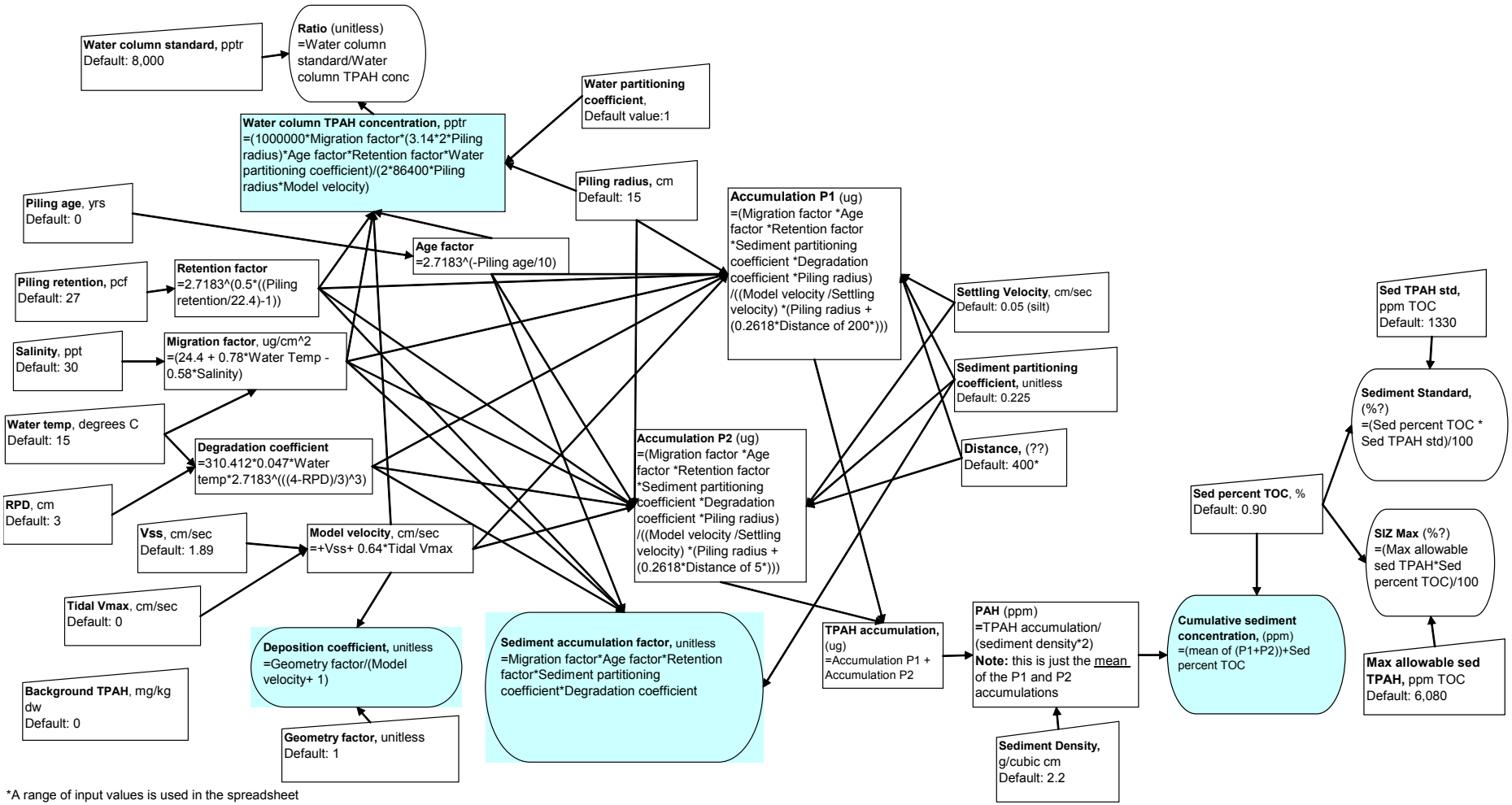
Appendix: CREOSS Model, Dr. K. Brooks



*A range of input values is used in the spreadsheet
Units in parentheses are assumed (not explicit)