Ambient Water Quality Criteria for Polycyclic Aromatic Hydrocarbons (PAHs)

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1.0 INTRODUCTION

Polycyclic aromatic hydrocarbons (PAHs) are organic compounds which are non-essential for the growth of plants, animals or humans; yet, they are ubiquitous in the environment. When present in sufficient quantity in the environment, certain PAHs are toxic and carcinogenic to plants, animals and humans. This document discusses the characteristics of PAHs and their effects on various water uses, which include drinking water, aquatic life, wildlife, livestock watering, irrigation, recreation and aesthetics, and industrial water supplies.

A significant portion of this document discusses the effects of PAHs upon aquatic life, due to its sensitivity to PAHs. For the most part, the discussion is based on recent reviews, but current research articles documenting the toxic effects of PAHs were also consulted. The data obtained from the literature were used in formulating appropriate water quality criteria for PAHs in ambient waters in British Columbia.

Where possible, criteria for each water use are recommended to protect the user from the deleterious effects of PAHs. Water quality standards, objectives and criteria and accompanying rationales from other jurisdictions were reviewed and their suitability for British Columbia water was considered.

KEY to ABBREVIATIONS USED

Polycyclic Aromatic Hydrocarbons (PAHs)

Abbreviation Full Word/Phrase

ACR Acridine
ANA acenaphthene
ANTH anthracene
9-MAN 9-methylanthracene
BAN benzanthracene
B[a]ANTH benz[a]anthracene or benzo[a]anthracene
B[b]CH benzo[b]chrysene
B[b]FLAN benzo[b]fluoranthene
B[j]FLAN benzo[j]fluoranthene
B[k]FLAN benzo[k]fluoranthene
B[a]P benzo[a]pyrene
B[ghi]PERY benzo[g,h,i]perylene

BP benzopyrene
CH chrysene
D[a,h]AN dibenzo[a,h]anthracene
DMB[a]AN dimethylbenz[a]anthracene
FLAN fluoranthene
FL fluorene
I[123-cd]PY indeno[1,2,3-cd]pyrene
NA naphthalene
1-MNA 1-methylnaphthalene
2-MNA 2-methylnaphthalene
3-MNA 3-methylnaphthalene
mNA methylnaphthalenes
d-MNA dimethylnaphthalenes
t-MNA trimethylnaphthalenes
PERY perylene
PH phenanthrene
1-MPH 1-methylphenanthrene
PY pyrene

Other Abbreviations Used

Abbreviation Full word/phrase

AET apparent effects threshold
BAET benthic apparent effects threshold
b.w. or bw body weight
dw dry weight
HPAH high molecular weight PAHs
LOEL lowest observed effect level
LPAH low molecular weight PAHs
MFO mixed-function oxidase (or oxygenase) enzyme system
NOEL No observed effect level
PAHs polycyclic aromatic hydrocarbons
TPAH total PAHs
ww wet weight, fresh weight
MW Molecular weight

2.0 PAHs and THEIR CHARACTERISTICS

The discussion in this chapter is mainly based on Neff (1979) and Handbook of Chemistry and Physics (Weast, 1968). Other sources of information, if any, are also referenced at appropriate places.

2.1 Characteristics

Polycyclic aromatic hydrocarbons (also known as polynuclear aromatic hydrocarbons) are composed of two or more aromatic (benzene) rings which are fused together when a pair of carbon atoms is shared between them (Figure 1). The resulting structure is a molecule where all carbon and hydrogen atoms lie in one plane. Naphthalene ($C_{10}H_8$; MW = 128.16 g), formed from two benzene rings fused together, has the lowest molecular weight of all PAHs. The environmentally significant PAHs are those molecules which contain two (e.g., naphthalene) to seven benzene rings (e.g., coronene with a chemical formula $C_{24}H_{12}$; MW = 300.36 g). In this range, there is a large number of PAHs which differ in number of aromatic rings, position at which aromatic rings are fused to one another, and number, chemistry, and position of substituents on the basic ring system.

Physical and chemical characteristics of PAHs vary with molecular weight (Table 1). For instance, PAH resistance to oxidation, reduction, and vapourization increases with increasing molecular weight, whereas the aqueous solubility of these compounds decreases. As a result, PAHs differ in their behaviour, distribution in the environment, and their effects on biological systems. PAHs can be divided into two groups based on their physical, chemical, and biological characteristics. The lower molecular weight PAHs (e.g., 2 to 3 ring group of PAHs such as naphthalenes, fluorenes, phenanthrenes, and anthracenes) have significant acute toxicity to aquatic organisms, whereas the high molecular weight PAHs, 4 to 7 ring (from chrysenes to coronenes) do not. However, several members of the high molecular weight PAHs have been known to be carcinogenic (Table 1).

Figure 1

FIGURE 1

Structure and numbering of selected PAHs

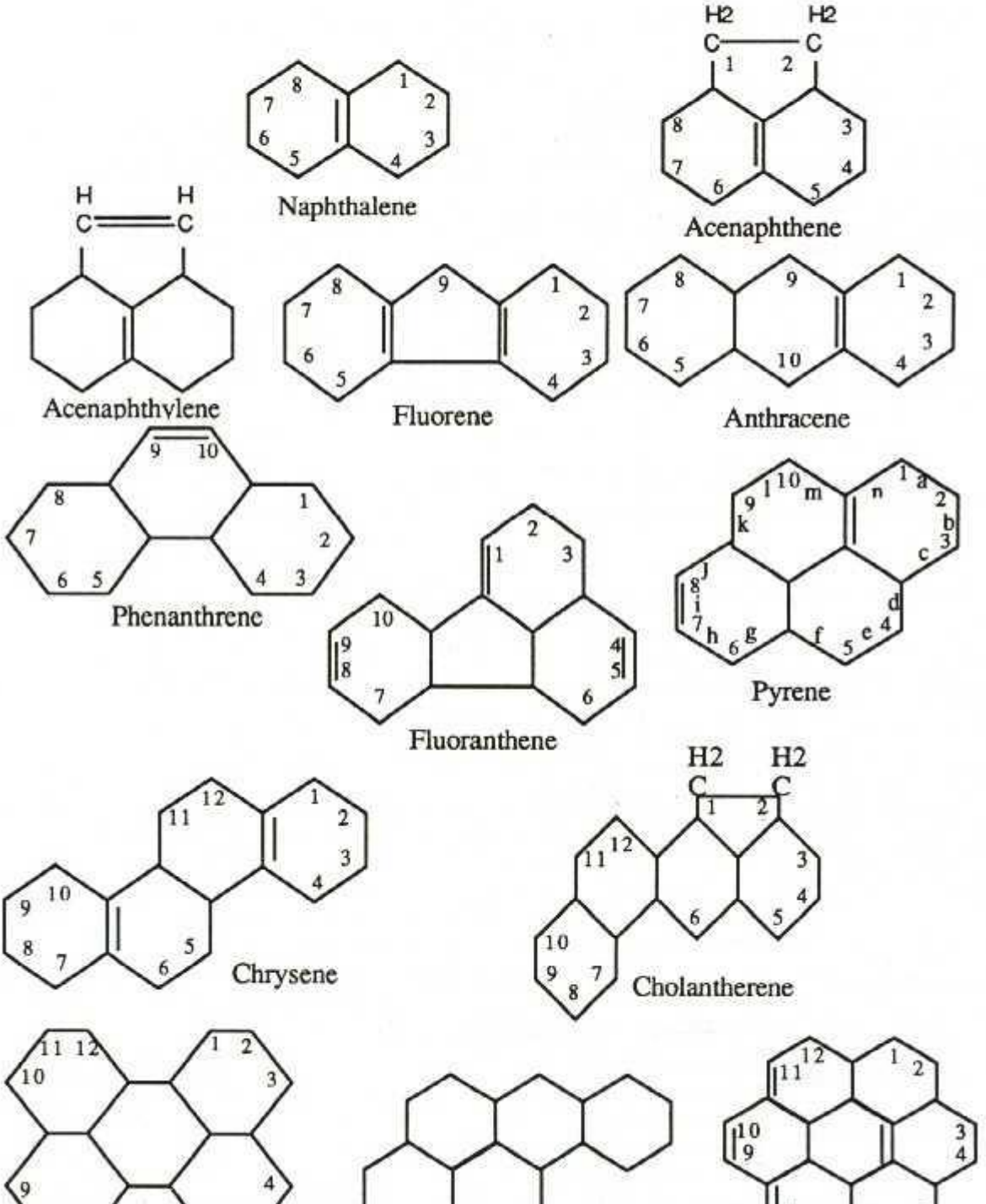


TABLE 1

Physical-chemical characteristics of some PAHs *
(From Neff, 1979; CCREM, 1987; NRCC, 1983; USPHS, 1990)

PAH	***Mol.Wt.* (g)**	***Solubility at 25 °C* (µg/L)**	***Vap. Pressure at 25 °C* (mm Hg)**	***Log Kow (Log Koc)***	***Carcino-genicity***	***Benzene (and total) rings***
Naphthalene	128.2	12500 to 34000	1.8×10^{-2}	3.37	NC	2
Acenaphthylene	152.2	3420	10^{-3} - 10^{-4}	4.07 (3.40)	NC	2
Acenaphthene	154.2			3.98 (3.66)	NC	2
Fluorene	166.2	800		4.18 (3.86)	NC	2 (3)
Anthracene	178.2	59	2.4×10^{-4}	4.5 (4.15)	NC	3
Phenanthrene	178.2	435	6.8×10^{-4}	4.46 (4.15)	NC	3
Acridine	179.2			(4.48)	NC	3
2-Methylanthracene	192.3	21.3		4.77	NC	3
9-Methylphenanthrene	192.3	261		4.77	NC	3
1-Methylphenanthrene	192.3	269		4.77	NC	3
Fluoranthene	202.3	260		4.90 (4.58)	NC	3 (4)
9,10-Dimethylanthracene	206.3	56		5.13	NC	3
Benzo[a]fluorene	216.3	45		5.34	NC	3 (4)
Benzo[b]fluorene	216.3	29.6		5.34	NC	3 (4)
Pyrene	202.1	133	6.9×10^{-7}	4.88 (4.58)	NC	4
Benz[a]anthracene	228.3	11.0	1.1×10^{-7}	5.63 (5.30)	C	4

Naphthacene	228.3	1.0		5.65	NC	4
Chrysene	228.3	1.9		5.63 (5.30)	WC	4
Triphenylene	228.3	43		5.63		4
Benzo[b]fluoranthene	252.3	2.4		6.04 (5.74)	C	4 (5)
Benzo[j]fluoranthene	252.3	2.4		6.21	C	4 (5)
Cholanthrene	254.3	2.0		6.28	C	4 (5)
7,12-Dimethylbenz[a]anthracene	256.3	1.5		6.36	SC	4
Dibenzo[a,h]fluorene	266.3	0.8		6.57	WC	4 (5)
Dibenzo[a,g]fluorene	266.3	0.8		6.57	C	4 (5)
Dibenzo[a,c]fluorene	266.3	0.8		6.57	WC	4 (5)
3-Methylcholanthrene	267.3	0.7		6.64	SC	4 (5)
Benzo[ghi]fluoranthene	214.2	0.5		6.78	NC	4 (5)
Benzo[a]pyrene	252.3	3.8	5.5×10^{-9}	6.06 (5.74)	SC	5
Benzo[e]pyrene	252.3	2.4	5.5×10^{-9}	6.21	NC	5
Perylene	252.3	2.4		6.21	NC	5
Indeno(1,2,3-cd)pyrene	276.3	-		6.58 (6.20)	C	5(6)
Dibenz[a,h]anthracene	278.3	0.4		6.86 (6.52)	C	5
Benzo[ghi]perylene	276.4	0.3	1.0×10^{-10}	6.78 (6.20)	NC	6
Coronene	300.3	0.14	1.5×10^{-11}	7.36	NC	7

* NC= non-carcinogenic; WC=weakly carcinogenic; C=carcinogenic; SC=strongly carcinogenic; Kow=Octanol/water partition coefficient; Koc= partitioning coefficient for organic carbon

2.2 Nomenclature

Several systems of nomenclature have been used to describe PAH ring structures. The most important rules of the system adopted by the International Union of Pure and Applied Chemistry (IUPAC) are outlined below and briefly illustrated in Figures 1 and 2.

1. The structure diagram of PAH is written such that the greatest possible number of rings is in a horizontal row.
2. Horizontal and vertical axes are drawn through the centre of a horizontal row, while orienting the molecule in such a way that maximal number of rings (those which are not lined up horizontally) are placed in the upper right quadrant and the minimal number of rings in the lower left quadrant.
3. Carbon atoms are numbered in a clockwise direction starting with the carbon atom that is not a part of another ring and is in the most counterclockwise position of the uppermost ring or, if there is a choice, of the uppermost ring farthest to the right. Carbon atoms common to two or more rings are not numbered.
4. Ring faces, which are not common to two rings, are lettered in alphabetical order with the side between carbon atoms 1 and 2 designated "a". Alphabetical order is continued clockwise around the molecule.
5. Compounds (or isomers) formed by the addition of a component are named with numbers and letters enclosed in brackets. These are placed immediately after the name of the added component to describe where a substituent group is attached or where a ring is fused to the face of the molecule. Appropriate letters are used where a ring is fused to more than one face of the molecule.
6. The structural formulas used show aromatic rings as plain hexagons and a methylene group as CH2.

A few PAHs (e.g., phenanthrene and anthracene) depart from these rules of nomenclature as seen in Figure 2. Further details on rules of PAH (and other organic molecules) nomenclature, and exceptions to them, can be found in the Handbook of Physics and Chemistry (Weast, 1968).

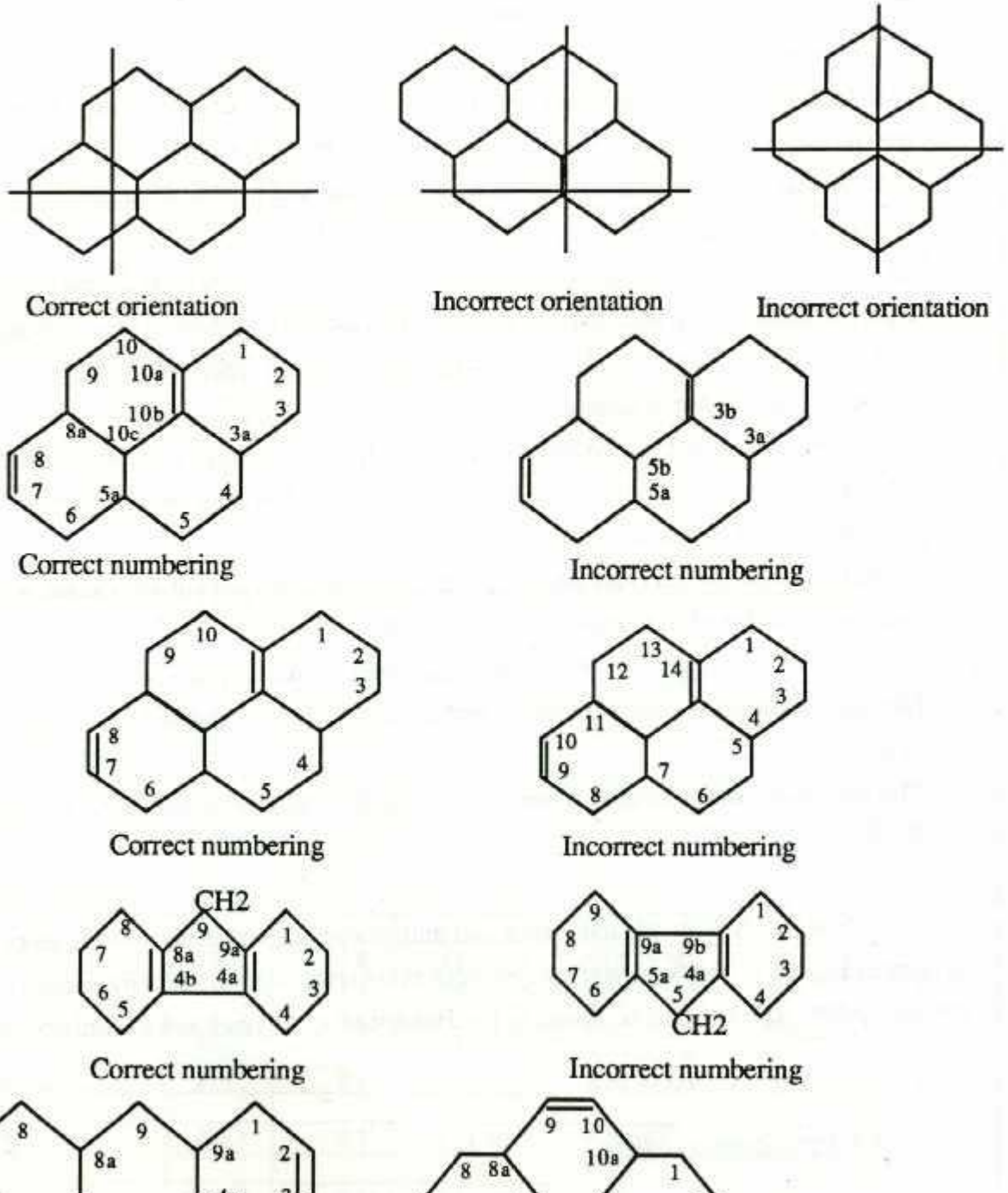
2.3 Production and uses

Among a large number of compounds in the category of polycyclic aromatic

Figure 2

FIGURE 2

Selected rules to numbering PAHs and organic molecules



hydrocarbons, only a few are manufactured in North America (Table 2). These PAHs are mostly used as intermediaries in pharmaceutical, photographic, and chemical industries. Naphthalenes are also used in the production of fungicides, insecticides, moth repellent, and surfactants. PAH-specific uses are also shown in Table 2.

TABLE 2

Production, uses, and producers of PAHs

PAH	***Commercial name(s)***	***Uses***	***Producers****
Acenaphthene	1,2-Dihydronaphthalene, 1,8-Dihydronaphthalene, 1,8-Ethylenenaphthalene, Periethylene naphthalene,	Chemical intermediary in pharmaceutical & photographic industries; to a limited extent in the production of soaps, pigments and dyes, insecticides, fungicides, plastics, and processing of certain foods	4 and 16
Acridine	2,3,5,6-Dibenzoquinoline, 9-Azanthracene, Benzo(b)quinoline	Laboratory chemical (as a dye) & to a limited extent in pharmaceuticals	4, 16, and 18
Anthracene	Paranaphthalene, Tetra olive N2G, Green oil	As a dye or chemical intermediary for dyes, diluent for wood preservatives	4 and 16
Fluorene	*o*-Biphenylenemethane, Diphenylenemethane	Unknown	4 and 11
Naphthalene		In the production of phthalic anhydride, carbaryl insecticide, beta-naphthol, tanning agents, moth repellent, and surfactants	1-3, 5-10, 17, 19, & 20
Pyrene	Benzo[d,e,f]phenanthrene, Beta-pyrene		4 and 12
Quinoline	1-Azanaphthalene, 1-Benzazine, Chinoline, Benzopyridine	In the preparation of hydroxyquinoline sulfate, niacin, some dyes; as a solvent for resins & terpines; decarboxylation agent	1, 3, 13, 14, and 15

* 1. A & C. American Chem. Ltd., Montreal, Que
2. A & K PetroChem Ind. Ltd., Concord, Ont
3. ACP Chemicals Inc., Saint-Leonard, Que
4. Aldrich Chem. Co., Inc., Milwaukee, WI
5. Allied Chem. Canada, Mississauga, Ont
6. Allied Signal Inc., Morristown, NJ
7. Anachemia Canada Ltd., Montreal, Que
8. Aristech Chem. Co. Inc., Pittsburgh, PA
9. Ashland Chem. Co. Inc., Columbus, OH
10. Carbochem Inc., Mississauga, Ont
11. Chemical Dynamics Co., Inc., Milwaukee, WI
12. Chemsyn Science Laboratories, Lenexa, KS
13. Crowley Chem. Co., New York, NY
14. General Intermediates of Canada, Edmonton, Alta
15 Howard Hall Int'l, Cos Cob, CT
16. Jonas Chem. Corp., Brooklyn, NY
17. Mary & Baker CDA Inc., Mississauga, Ont
18. Polyscience, Inc., Warrington, PA
19. Recochem Inc., Montreal, Que
20. Texaco Chemical Co., Houston, TX

3.0 FORMS AND TRANSFORMATIONS

This chapter is based primarily on information presented in Moore and Ramamoorthy (1984) and Neff (1979). Where appropriate, other sources of information were also referenced.

3.1 Physico-chemical properties

Polycyclic aromatic hydrocarbons are non-polar, hydrophobic compounds, which do not ionize. As a result, they are only slightly soluble in water (Table 1). In general:

(1) PAH solubility in water decreases as the molecular weight increases.
(2) Alkyl (i.e., CH2- group) substitution of the aromatic ring results in an overall decrease in the PAH solubility, although there are some exceptions to this rule. For example, Benz[a]anthracene is less soluble than either methyl- or ethylbenz[a]

anthracene.
(3) Molecules with a linear arrangement tend to be less soluble than angular or perifused[1] molecules. For instance, anthracene is less soluble than phenanthrene, and naphthacene is less soluble than chrysene or benz[a]anthracene.

The solubility of PAHs in water is enhanced three- to four-fold by a rise in temperature from 5 to 30 °C. Dissolved and colloidal organic fractions also enhance the solubility of PAHs which are incorporated into micelles (a micelle is composed of an aggregate of surface-active molecules, or surfactants, each possessing a hydrophobic hydrocarbon chain and an ionizable hydrophilic group) (Neff, 1979).

Vapor pressure characteristics determine the persistence of PAHs in the aquatic environment. Two- to 3-ring PAHs are very volatile, while PAHs with 4 or more rings show insignificant volatilizational loss under all environmental conditions (Moore and Ramamoorthy, 1984).

Due to their hydrophobic nature, PAHs entering the aquatic environment exhibit a high affinity for suspended particulates in the water column. As PAHs tend to sorb to these particles, they are eventually settled out of the water column onto the bottom sediments. Thus, the PAH concentrations in water are usually quite low relative to the concentrations in the bottom sediments (Moore and Ramamoorthy, 1984). The sorptive characteristics of PAHs have been exploited in waste treatment processes such as coagulation, flocculation, sedimentation, and filtration with sand or activated carbon.

3.2 Photo-chemical transformations

PAHs are degraded through the process of photooxidation. The photo-induced oxidation of PAHs in the aqueous phase is brought about by singlet oxygen, ozone, HO- radical, and other oxidants. Photooxidation by singlet oxygen appears to be the most dominant process for the breakdown of PAHs and other organics in water (Zafiriou, 1977). Under ozone and light, the half-lives of several PAHs vary between a few minutes to a few hours. The most common products of photolysis are endoperoxides that undergo secondary reactions to yield a variety of products including diones (Figure 3).

PAHs differ in their sensitivity to photooxidation. Nagata and Kondo (1977) studied photodegradation of several PAH compounds in mixed acetone-water or carbon tetrachloride(CCl_4)-water solvents. Anthracene, phenanthrene, and benz[a]anthracene were the most sensitive PAHs, whereas chrysene, fluorene, pyrene, and benzo[a]pyrene were relatively resistant to photodegradation. Since the solvents used (e.g., acetone versus CCl_4) may exert strong influence on photosensitivity of PAHs,

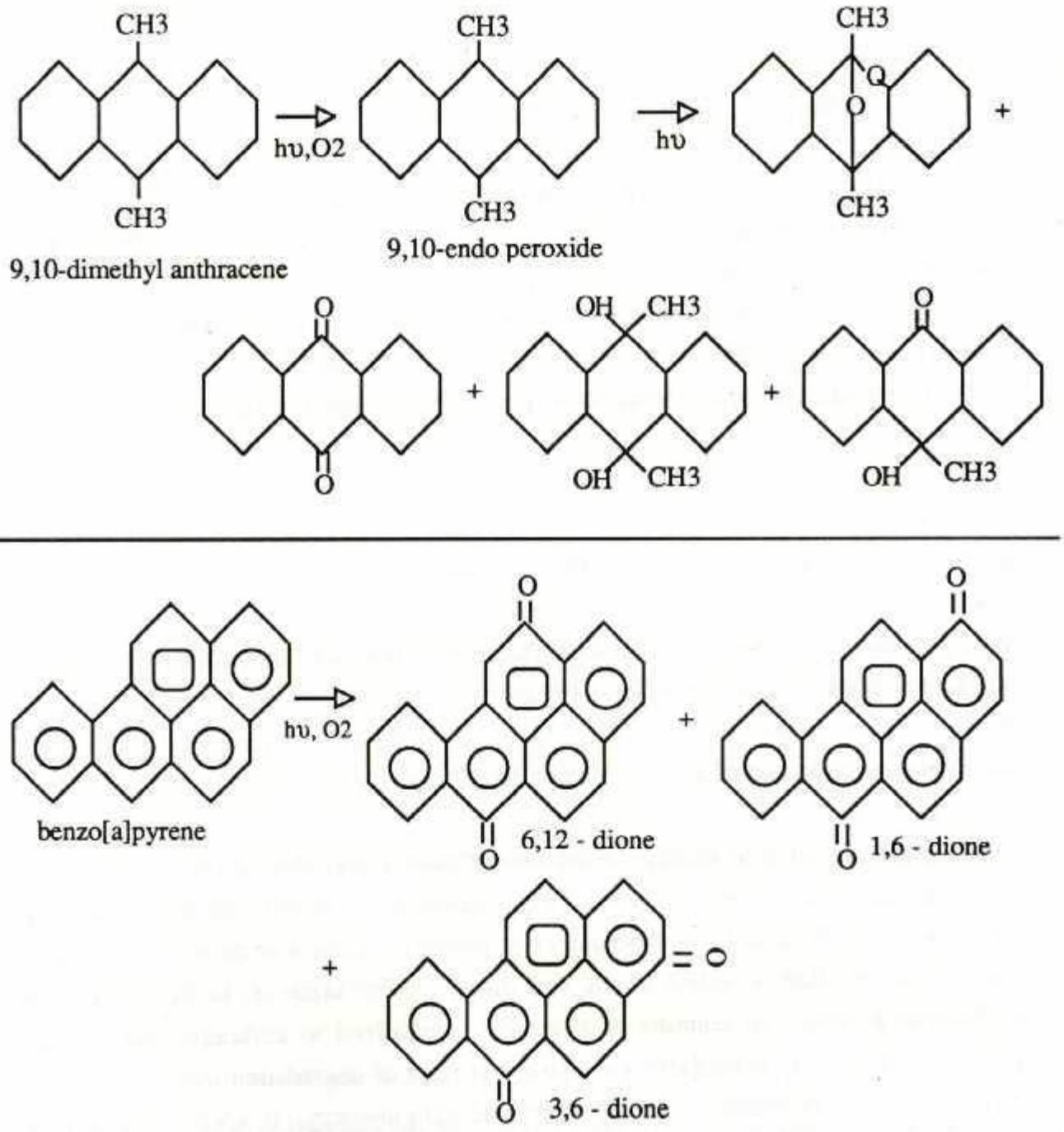
caution must be exercised in predicting the fate of these compounds in natural waters based on laboratory observations.

PAHs attached to particulate matter are more susceptible to photolysis than PAHs in solution. Also, the oxidative pathway for the sorbed PAHs is different from those in solution, and is not intermediated by endoperoxides in yielding quinones or diones (a general name for quinones under IUPAC rules) as the end product. For instance, in the presence of light, anthracene adsorbed to alumina or silica gel is oxidized to anthraquinone. Studies with particulate-associated benzo[a]pyrene show that rates of degradation increase with increasing dissolved oxygen concentration, temperature and light intensity. In a water column, the rate of photodegradation will decrease with depth as a result of (a) decrease in light intensity through absorption and scattering by water and suspended solids, and (b) decrease in temperature and

Figure 3

FIGURE 3

Photolysis products of 9,10-dimethyl anthracene and benzo[a]pyrene
(From Moore and Ramamoorthy, 1984)



dissolved oxygen. Photooxidation of PAHs is negligible in bottom sediments (Neff, 1979; Moore and Ramamoorthy, 1984).

Chlorination and ozonation, treatments used for destruction of pathogens in drinking water and sometimes oxidation of organics in industrial wastewater effluents, have been thought to remove PAHs from water. Harrison *et al.* (1976a, b) studied the influence of chlorination on eight PAHs (i.e., fluoranthene, pyrene, benz[a]anthracene+chrysene, benzo[b+j+k]fluoranthene, benzo[a+e]pyrene, perylene, indeno[1,2,3-cd]pyrene, and benzo[ghi]perylene). The rate of oxidation was dependent on PAH type (e.g., pyrene oxidized most rapidly and fluoranthene most slowly), temperature (the oxidation rate increased slightly from 5 to 20 °C), and pH (PAH concentration in water decreased with decreasing pH). These investigators also evaluated the efficiency of water treatment processes, including chlorination, for removal of these PAHs from drinking water (the concentrations among 8 PAHs ranged from 0.069 to 0.15 µg/L, and the total PAH concentration was 0.628 µg/L). It was found that (a) filtration removed 50% of the total PAH attached to fine particulates in water, while (b) chlorination, at 17 °C and pH = 7.5, removed about 60% of the PAHs remaining in the filtered water. The total PAH concentration in water was 0.13 µg/L after treatment. Ozonation was less efficient than chlorination for the removal of PAHs from water.

Chlorine and ozone react with PAHs to produce quinones and polychlorinated aromatics, some of which may be highly toxic to aquatic organisms (Green and Neff, 1977).

3.3 Biological transformations

3.3.1 Bacteria and fungi

PAHs are subject to biodegradation by microorganisms present in soil, sewage, and water. Microbial metabolism of PAHs may result in either complete or incomplete hydrocarbon degradation, depending upon several environmental (e.g., pH, temperature, dissolved oxygen and redox state) and molecular factors (e.g., PAH type including the number and position of fusion of aromatic rings in the molecule). Lower molecular weight PAHs tend to oxidize completely to form CO_2 and H_2O while the heavier PAHs will degrade partially to yield various oxygenated metabolites (e.g., various phenolic and acid metabolites, *cis*-dihydrodiol, etc.).

Lee and Takahashi (1977) studied the degradation of fluorene (initial concentration = 30 µg/L), naphthalene (initial concentration = 50 µg/L), methylnaphthalene (initial concentration = 50 µg/L), and benzo[a]pyrene (initial concentration = 16 µg/L) by

marine bacteria isolated from various depths in a controlled ecosystem enclosure in Saanich Inlet, British Columbia. Water in the enclosure was contaminated with No. 2 fuel oil to a concentration of 10-20 µg/L total non-volatile petroleum hydrocarbons. Prior to the addition of oil, naphthalene (10 µg/L/d) and methylnaphthalene (10 µg/L/d) degraded slowly, while fluorene and B[a]P were not metabolized at all in 48 h. Three days after the oil dosing, the metabolism (or degradation) of naphthalene (26 µg/L/d) and methylnaphthalene (250 µg/L/d) increased greatly, but B[a]P degraded at a barely detectable rate (1.0 µg/L/d) while fluorene was still unmetabolized.

Microbial degradation of PAHs is one of the main processes responsible for removing these substances from bottom sediments and the water column. Delaune *et al*. (1981) noted in their studies that the rate of bacterial metabolism of PAHs in estuarine sediment was significantly lower in acidic, anoxic conditions. Poor water quality or heavy pollution of a water body may increase the residence time of PAHs.

This does not imply that PAHs may reside in the bottom sediments indefinitely. Anaerobic and facultative bacteria present on the sediments are also capable of metabolizing these substances although at a much slower rate than their aerobic counterparts (Delaune *et al*., 1981). The residence time of a PAH in sediment may thus be longer in anaerobic conditions, but biotransformation will still be occurring. Should the compounds be located deep within the sediment layer, however, degradation may or may not occur depending upon the sediment structure and bioavailability of the PAH.

The degradation of PAHs by fungi is unlike bacterial degradation, but resembles that in mammals as a result of fungi possessing a cytochrome P-450 (a heme protein) enzyme system. For instance, the fungus *Cunninghamella elegans* degrades naphthalene by the arene oxide-*trans*-naphthalene dihydrodiol pathway characteristic of mammals (Ferris *et al*., 1973).

The degradation of PAHs in water, sediment, and soils is shown in Table 3.

TABLE 3
Biodegradation of PAHs in water, sediment, and soils
(From Lee & Ryan, 1983; Heitcamp & Cerniglia, 1987; Sims, 1986; Niimi & Palasso,1986)

Biological Component	***PAH***	***Biodegradation rate***	***Comment***
Water (uncontaminated & from heavily oiled river)+	NA	125 to 320-d half-life (14-d)	10C (22C)

	2-MNA	390 to 530-d half-life (16-d)	7C (22C)
	PH	180-d half-life (36-d)	8C (27C)
Sediment (uncontaminated & from heavily oiled river)+	ANTH	95 to 141-d half-life (57-d)	18C
	B[a]ANTH	1 100-d half-life (16-d)	15C
	FL	37-d half-life	10C
	CH	510-d half-life (79-d)	10C
Water & sediment (from pristine contaminated, with petrogenic, ecosystems)+	NA	4.4-wk half-life (2.4-wk)	22C
	2-MNA	20-wk half-life (14-wk)	22C
	PH	18-wk half-life (4-wk)	22C
	PY	not detected (34-wk half-life)	22C
	B[a]P	not detected (>200-wk half-life)	22C
Soil (with &without amendment)*	FL	64-d half-life (39-d)	
	PH	69-d half-life (23-d)	
	ANTH	28-d half-life (17-d)	
	FLAN	104-d half-life (29-d)	
	PY	73-d half-life (27-d)	
	B[a]ANTH	123-d half-life (52-d)	
	CH	70-d half-life (42-d)	
	B[b]FLAN	85-d half-life (65-d)	
	B[k]FLAN	143-d half-life (74-d)	
	B[a]P	91-d half-life (69-d)	
	B[ghi]PERY	74-d half-life (42-d)	
	D[ah]AN	179-d half-life (70-d)	
	I[123-cd]PY	57-d half-life (42-d)	

Rainbow trout (*O. mykiss*) (mean fish wt. 715-875 g)	FL	7-d half-life	amount fed=3.95 mg
	PH	9-d half-life	amount fed=3.51 mg
	ANTH	7-d half-life	amount fed=3.55 mg
	FLAN	6-d half-life	amount fed=3.28 mg

+ half-lives for contaminated systems are in parenthesis; *half-lives with soil amendments (i.e., manure and lime) are in parenthesis

3.3.2 Animals

In animals, the mixed-function oxygenase(or oxidase) (MFOs) enzyme systems are responsible for the biotransformation of PAHs and other exogenous (e.g., xenobiotics or foreign compounds such as PCBs, pesticides, etc.) as well as endogenous organic substances (e.g., steroids and hormones). The MFO systems are usually associated with the endoplasmic reticulum of microsomal tissues located in the livers of vertebrates and the hepatopancreas of invertebrates; they have also been found in other organs of both groups. Not all invertebrates and vertebrates possess the MFO systems though this may be due to the lack of appropriate technology to detect these enzymes rather than the lack of the system.

The function of the MFOs is primarily to detoxify xenobiotics by converting these lipophilic materials into a more water soluble form, thus expediting their excretion from the organism. Detoxification of PAHs is not a simple process. Before formation of non-toxic and harmless end products by various enzymatic and nonenzymatic reactions, PAHs are converted to arene oxide intermediates followed by formation of derivatives of *trans*-dihydrodiols, phenols, and quinones. These intermediate products are known to be toxic, carcinogenic, and/or mutagenic.

Biological half-lives for some PAHs in rainbow trout are shown in Table 3.

Aquatic organisms may serve to remove a significant fraction of these compounds from the body of water. Pelagic organisms may take up PAHs directly from the water column

or benthic organisms may absorb these substances from contact with both the bottom sediments and the overlying water. Considering the tendency of light molecular weight PAHs to volatilize from the water and of heavier PAHs to settle out with the sediments, it seems logical to assume that pelagic animals are exposed to lower overall PAH concentrations. However, uptake of these compounds tends to occur much more rapidly in the solubilized form. Therefore, in a high concentration, short exposure situation, pelagic organisms may actually be more at risk than their benthic counterparts. The toxicity, carcinogenicity, and mutagenicity of PAHs vary with the molecular weight of the compound, the degree of alkylation, and with the mode of accumulation (water, food or sediment) by the organism (Neff, 1979; Moore and Ramamoorthy, 1984). Thus, the effects of these compounds upon an aquatic organism are not only highly dependent on the source of PAHs, but also upon the feeding behavior and habitat of the particular species.

3.3.3 Terrestrial plants

Terrestrial plants can take up PAHs through their roots and/or leaves and translocate them to various plant parts (Edwards, 1983). However, relatively little is known about the fate of PAHs within the plants. Dorr (1970) found a decline in B[a]P concentrations in rye plants after 30 days of growth, following a period (20 days) of increasing concentrations due to uptake from nutrient solution and soil containing the PAH. The decline in B[a]P concentration was attributed to degradation or chemical changes in B[a]P within the plants. Using 14C-B[a]P, Durmishidze *et al*. (1974) demonstrated chemical transformations of B[a]P (mostly to organic acids) within a number of plant species. Durmishidze (1977) reported similar results with both B[a]P and B[a]ANTH. The amount of B[a]P catabolized over a 14-d period varied from 2 to 18% of the B[a]P assimilated and depended upon plant species. The catabolism of anthracene by soybeans was demonstrated by Edwards *et al*. (1982). More recently, Negishi *et al*. (1987) demonstrated that a soybean leaves can oxidize B[a]P to its alcohols that are qualitatively similar to those produced by mammalian microsomes and eukaryotic microorganisms.

1 Polycyclic compounds in which two rings have two, and only two, atoms in common are said to be "ortho-fused". Such compounds have n common faces and 2n common atoms. Polycyclic compounds in which one ring contains two, and only two, atoms in common with each of two or more rings of a contiguous series of rings are said to be 'ortho- and peri-fused'. Such compounds have n common faces and fewer than 2n common atoms.

4.0 OCCURRENCE in the ENVIRONMENT

4.1 Natural sources

In nature, PAHs may be formed three ways: (a) high temperature pyrolysis of organic materials, (b) low to moderate temperature diagenesis of sedimentary organic material to form fossil fuel, and (c) direct biosynthesis by microbes and plants (Neff, 1979).

4.1.1 Fires

Forest fires, prairie fires, and agricultural burning contribute the largest volumes of PAHs from a natural source to the atmosphere. The actual amount of PAHs and particulates emitted from these sources varies with the type of organic material burned, type of fire (heading fire vs. backing fire), nature of the blaze (wild vs. prescribed; flaming vs. smouldering), and intensity of the fire. PAHs from fires tend to sorb to suspended particulates and eventually enter the terrestrial and aquatic environments as atmospheric fallout (Eisler, 1987).

In the atmosphere, PAHs may undergo photolytic and chemical (ozone) transformations. However, most of the material does not degrade quickly in the atmosphere and thus may reside in the environment for extended periods of time. During this atmospheric entrainment, winds may distribute these particle-sorbed PAHs in a global manner such that they appear even in remote areas of the Arctic or Antarctica. There has been some contention, however, that the world-wide distribution of PAH may actually be due to natural PAH sources in proximity to these remote areas (Clarke and Law, 1981; Platt and Mackie, 1981).

4.1.2 Fossil fuels

PAHs occur naturally in bituminous fossil fuels, such as coal and crude oil deposits, as a result of diagenesis (i.e., the low temperature, 100-150 °C, combustion of organic material over a significant span of time). This process favors the formation of alkylated PAHs; the unsubstituted (or the parent) compounds being relatively low in abundance in these sources (NRCC,1983).

It has been suggested that 70-75% of the carbon in coal is in aromatic form; the 6-membered ring aromatics are dominant with a small 5-membered ring fraction present as well (Neff, 1979). PAHs such as benz[a]anthracene, benzo[a]pyrene, benzo[e]pyrene, dibenzo[c,d,m]pyrene, perylene, and phenanthrene have been

identified in coal samples (Woo *et al*., 1978). Atwater and Mavinic (1985) analyzed wastewater and sludge samples from 11 coal operations across Canada In wastewater, naphthalene and phenanthrene were detected at levels >10 µg/L, whereas anthracene, benzo[k]fluoranthene, and dibenzo[a,h]anthracene levels were <10 µg/L. Naphthalene, phenanthrene, anthracene, fluorene, and pyrene were usually found in sludges at µg/g levels.

The PAH make-up of crude oil and refined petroleum products is highly complex and variable and no two sources have the same composition (Table 4).

Under natural conditions, fossil fuels contribute a relatively small volume of PAHs to the environment. Because most oil deposits are trapped deep beneath layers of rock, there is little chance to emit PAHs to the surface environment. There are some petroleum bodies (e.g., tar sands) which, being near the surface, are capable of contributing PAHs to both atmospheric and aquatic surroundings. These deposits are small in number and are likely to contribute little to the overall volume of PAH in the environment.

4.1.3 Other sources

Volcanic activity and biosynthesis by bacteria and plants are other natural sources of PAHs. Relative to fires, these sources contribute small amounts to the environment. There is still some uncertainty as to whether or not biosynthesis of PAH in vegetation, fungi and bacteria is actually occurring or whether PAH levels in these organisms have been acquired from other sources (Neff, 1979). More sophisticated experimental techniques and equipment are required to resolve these questions.

4.2 Anthropogenic sources

Incomplete combustion of organic matter at high temperature is one of the major anthropogenic source of environmental PAHs. The production of PAHs during pyrolysis (i.e., partial breakdown of complex organic molecules during combustion to lower molecular weight

TABLE 4

PAH concentrations in a crude oil and two distillate fuel oils
(From Neff, 1979)

Compound	***Kuwait Crude***	***No. 2 fuel oil*** **(µg/g)**	***Bunker C residual oil***

	(µg/g)		(µg/g)
Naphthalene	400	4000	1000
1-Methylnaphthalene	500	8200	2800
2-Methylnaphthalene	700	18900	4700
Dimethylnaphthalenes	2000	31100	12300
Trimethylnaphthalenes	1900	18400	8800
Fluorenes	<100	3600	2400
Phenanthrene	26	429	482
1-Methylphenanthrene	-	173	43
2-Methylphenanthrene	89	7677	828
Fluoranthene	2.9	37	240
Pyrene	4.5	41	23
Benz[a]anthracene	2.3	1.2	90
Chrysene	6.9	2.2	196
Triphenylene	2.8	1.4	31
Benzo[ghi]fluoranthene	<1		
Benzo[b]fluoranthene	<1		
Benzo[j]fluoranthene	<1		
Benzo[k]fluoranthene	<1		
Benzo[a]pyrene	2.8	0.6	44
Benzo[e]pyrene	0.5	0.1	10
Perylene	<0.1	-	22
Benzo[ghi]perylene	<1		

free radicals) and pyrosynthesis (i.e., combination of free radicals containing one or more carbons) is a function of the temperature. In studying the effects of temperature (550 to 1 000 °C) on pyrosynthesis of PAH from styrene, Commins (1969) found that the yields of all PAHs (ranging in molecular weight from naphthalene to coronene) peaked at 780 °C, decreasing at higher and lower temperatures.

The environmental sources of PAHs of pyrolitic origin are many (Neff, 1979):

(a) Charcoal-broiled steaks, and commercially available smoked food products have been identified to contain PAHs.

(b) Conditions are ideal for PAH pyrosynthesis within a cigarette flame.
(c) Burning of fossil fuels is an important source of PAHs in the environment. Significant quantities of benzo[a]pyrene and other PAHs have been identified in vehicular exhaust.
(d) Many heat and electrical generating facilities burn fossil fuels and produce, as byproducts, liquid, solid, and gaseous wastes that may be rich in PAHs.
(e) Catalytic breakdown of crude petroleum to produce hydrocarbon fuels and other refined products results in the production of PAHs. Many of the PAHs thus produced become concentrated in the high boiling residual oil (e.g., Bunker C and No. 2 fuel oils - Table 4) and asphalt. Significant quantities of PAHs may also be released in flue gas.
(f) The production of coke involves subjecting hard coal to high temperatures (1400 °C) in a reducing atmosphere, conditions ideal for pyrosynthesis of PAHs. Lao *et al.* (1975) identified 75 PAHs in air-filter samples of gaseous coke oven emissions.
(g) Coal tars, produced by the high temperature treatment of coal, are also known to contain a host of PAHs. These PAHs are derived either from PAHs indigenous to the coal or from pyrolysis of coal hydrocarbons.
(h) Incineration is a valuable means of waste disposal and waste reduction. PAHs in the stack gases, solid residues, and wastewaters from municipal incinerators have been identified (Davies *et al.,* 1976). It has also been found that PAHs released each day in solid residues were 10 times more than in the stack gases and 100 times more than in the wastewater (Davies *et al.,* 1976).

There are many other anthropogenic sources of pyrolytic PAHs. In fact, any industrial or domestic process in which organic carbon is subjected to high temperature will result in the production of some PAHs. Treated wood has also been recognized as a source of PAHs in water and sediments.

In general, anthropogenic sources can be divided into two categories: sources that discharge directly into a body of water, and sources that discharge into the atmosphere.

The sources of PAHs which may discharge directly into aquatic environment include: accidental spillage and/or leakage of PAH-containing fluids (e.g.,waste oils, gasoline, etc.), industrial and domestic wastewaters, urban runoff, discharges originating from landfills, and use of creosoted pilings for docks and other shoreline structures.

Atmospheric PAH emissions fall into two groups: (i) those which originate from stationary sources, and (ii) those which originate from non-stationary sources. Stationary sources include coal and gas-fired boilers; coal gasification and liquifaction plants; carbon black, coal tar pitch and asphalt production; coke-ovens; catalytic cracking towers; petroleum refineries and related activities, electrical generating plants; industrial incinerators; municipal incinerators, agricultural and refuse burning, and any

other industry that entails the use of wood, petroleum or coal to generate heat and power. These sources contribute PAHs to the environment either through the formation of these compounds during industrial processing or through pyrolysis of the above mentioned fuels for energy generation. These PAHs, if not degraded in the atmosphere, are sorbed onto particulates in the air and are then deposited onto bodies of water, as well as the surrounding terrestrial environment.

Non-stationary sources of PAHs usually refer to automobiles or other vehicles which use petroleum products as a fuel. Temperatures within an internal combustion engine are often sufficient enough to convert a fraction of the fuel or oil into PAHs via pyrolysis. These compounds are then emitted to the atmosphere through exhaust fumes whereupon they sorb onto particulates. Most PAHs are then photolytically degraded or are deposited onto street surfaces. Precipitation then washes these PAHs into stormwater drainage systems eventually flushing them into the aquatic environment.

4.3 Aquatic environmental loading

According to Eisler (1987) approximately 228 000 metric tons of PAHs are discharged to the aquatic environment per annum as a result of human activity (Table 5). Petroleum spillage and/or leakage of a major and/or a minor nature is the largest contributor to this loading and amounts to 170 000 tons (roughly 75%) of this total. The other major contributor is the atmospheric fallout from the sources listed in section 4.2, which adds an accumulated total of 50 000 tons to aquatic systems. The remaining mass of PAH is contributed through industrial wastewater effluents, sewage effluents and from runoff. The PAH mixtures disposed of in this manner are highly variable and complex due to the large number of sources contributing to this discharge.

TABLE 5

Major Sources of PAHs in the Atmospheric and Aquatic Environments
(From Eisler, 1987)

Sources	***Annual Input of TPAHs*** **(metric tons)**
ATMOSPHERE	
Forest and prairie fires	19 513
Agricultural burning	13 009
Refuse burning	4 769

Enclosed incineration	3 902
Heating and power	2 168
Total	43 361
AQUATIC ENVIRONMENTS	
Petroleum spillage	170 000
Atmospheric deposition	50 000
Wastewaters	4 400
Surface land runoff	2 940
Biosynthesis	2 700
Total	230 040

4.4 Levels in sediment, water and biota

4.4.1 Water

PAH concentrations in fresh waters vary widely, depending upon such factors as proximity of the waterbody to the source, source type, and season (Moore and Ramamoorthy, 1984).

From a review of data collected in Europe, Neff (1982a, b) noted that drinking water from various sources (e.g., ground water, reservoirs, rainwater, etc.) typically contains 0.2 to 80 ng/L B[a]P and 4 to 4 000 ng/L total PAH. The average concentration of total PAH in drinking water from U.S.A. and Europe, respectively, was quoted to be 15 and 50 ng/L by Lee and Grant (1981). According to Lee and Grant, the concentrations ranging from 50 to 250 ng PAH/L represent the low level contamination of fresh surface water by PAHs, whereas the concentrations ranging from 200 to 1 000 ng PAH/L represent the medium level contamination.

The Great Lakes Science Advisory Board (GLSAB) (1983) has reported concentrations of several PAHs in open waters of the Great Lakes water system (Table 6). In general, the Great Lakes are relatively uncontaminated by PAHs. Although the data for each of the lakes in this system were not available, it is likely that there would be a considerable discrepancy between them as substantial portions of certain lakes (e.g., Lake Ontario, Lake Erie) are more impacted by human activity than others.

TABLE 6 ***Concentration of PAHs in Surface Waters of the Great Lakes Water System*** (From Great Lakes Science Advisory Board, 1979)	
PAH	***Mean Value[a]***
	ng/L
Anthracene	6
Phenanthrene	24
Fluoranthene	15
Benzo[a]pyrene	12
Chrysene	14
Pyrene	14

[a] n=6

PAH concentrations in ambient estuarine and oceanic waters are not well addressed in the literature. The available data are based on estimates of total aromatics by ultraviolet, infrared, and fluorescence techniques, which may be subject to considerable interference from non-PAH materials. The PAH concentrations in marine waters from national and international sources are shown in Table 7. It can be seen that, in each water body, the PAH concentration was a function of the sampling depth with the maximum value recorded near the surface. Marty *et al.* (1978) also indicated that PAHs (e.g., phenanthrene, alkylphenanthrene, perylene, fluoranthene, and pyrene) in seawater tend to concentrate in the surface microlayer. Several organisms (plankton, fish eggs) are located in this microlayer and may potentially be impacted to a greater extent than those organisms located in sub-surface waters.

TABLE 7 ***Distribution of Polycyclic Aromatic Hydrocarbons in Marine and Estuarine Waters*** (From Neff, 1979)		
Area and Year	***Depth***	***Aromatic hydrocarbons (mean and standard deviation, based on UV absorbance)***
	metres	**ng/L**

Baltic Sea (1973)	1 10-50 1 m above sediment	277 ± 121 52 ± 9 47 ± 13
Nova Scotia to Gulf Stream (1973)	1 10 25	30.8 ± 10 16.8 ± 4 8.6 ± 9
Sargasso Sea off Bermuda (1974-76)	1 30 300 1 200 2 000	31 ± 10 1 ± 0 1 ± 1 4 0
Mediterranean Sea (1975)	1	148 ± 36
Atlantic Ocean (1976)	surface	400

Reports on specific PAH concentrations in marine waters are few. Niaussat and Auger (1970) found 1 600 ng/L B[a]P and 3 050 ng/L perylene in the Clipperton Lagoon in the Pacific Ocean. Levels of B[a]P ranging from non-detectable to 400 ng/L were found in the Polynesian atolls of Moruroa and Hao (Niaussat *et al.*, 1975). Gschwend *et al*. (1982) found that naphthalene concentrations in Vineyard Sound, USA, ranged from < 1.0 to 35 ng/L over sixteen months.

In water 15 m away from an oil separator platform and brine outfall in Trinity Bay, Texas, USA, Armstrong *et al*. (1977) detected single ring aromatic hydrocarbons (e.g., benzene, toluene, xylene, etc.) as well as naphthalene (0.40 µg/L), 1-methylnaphthalene (0.20 µg/L), 2-methylnaphthalene (0.60 µg/L) and dimethylnaphthalenes (0.70 µg/L). No other LPAH or HPAH were detected in the water although the effluent discharged contained significant quantities of fluorene, phenanthrene and their alkyl derivatives.

Data on PAHs in British Columbia waters are limited. Wan (1991) measured concentrations of 16 PAHs (see Table 12 for the list of PAHs) in the ballasts from five railway rights-of-way and the adjacent ditches (6 locations) flowing to salmon streams in the Lower Mainland of British Columbia. Unlike the ballasts and ditch sediments, PAHs were not consistently found in the ditch water. The average[2] concentrations ranging from 0.4 µg/L for acenaphthylene and benzo[a]pyrene to 208 µg/L for fluoranthene were found in the ditch water. Highest concentrations in the water were detected where power and telecommunication line poles were erected in the railway ditches. Among lower molecular weight PAHs, high mean concentrations were found for acenaphthene (8.3 µg/L), anthracene (9.7 µg/L), naphthalene (82.7 µg/L), and

phenanthrene (112.9 µg/L). Among high molecular weight PAHs, mean concentrations for benz[a]anthracene (32.3 µg/L), benz[b]fluoranthene (25.5 µg/L), benz[k] fluoranthene (14.0 µg/L), chrysene (76.0 µg/L), fluoranthene (207.7 µg/L), and pyrene (125.8 µg/L) were the highest.

The more recent samples collected by the British Columbia Ministry of Environment, Lands and Parks from Duteau Creek and Christina Lake in the Okanagan area, and Spectacle, Old Wolf, Quamichan, Lizard, and Maxwell Lakes on Vancouver Island indicated that the concentrations of the 16 PAHs (see Table 12 for the list of PAHs) were mostly less than the detection limit (0.01 µg/L); only one sample (Quamichan Lake) recorded a significant number (0.03 µg/L) for naphthalene (Nagpal, 1992). Note that no anthropogenic sources were detected in the vicinity of these creeks and lakes.

4.4.2 Sediment

PAH concentrations reported in this section are expressed on a dry weight (dw) basis in surface sediments at the bottom of water columns, unless indicated otherwise.

PAHs are slightly soluble in water. Binding to particulate matter (especially organic), they tend to accumulate in the bottom sediments. Levels of PAH in sediments vary, depending on the proximity of the sites to areas of human activity. Sediment concentration and distribution of PAHs may also fluctuate due to biodegradation of these chemicals, a process which is reliant upon abiotic and biotic factors which are dependent on site characteristics.

In the surface sediment samples collected from the Great Lakes system, 27 PAHs were identified. Among those commonly found were perylene, pyrene, benzopyrenes, benzoperylenes, fluoranthenes, benzofluoranthenes, and chrysene. The total PAH concentrations in sediments from Lakes Ontario, Erie, and Huron, respectively, were 14 µg/g, 54 µg/g, and 1.2 µg/g (GLSAB, 1983).

PAHs in sediments are elevated near industrial and urban centres. In British Columbia, this trend was evident in the Greater Vancouver area. Dunn and Stich (1975) demonstrated the impact of municipal effluent on sediment PAH concentrations in samples collected near the Iona Island sewage treatment outfall when it discharged onto Sturgeon Bank in shallow water. B[a]P levels of 121 µg/g were detected at a distance of about 0.7 km from the sewage outfall. As this distance increased, however, the concentrations of B[a]P dropped rapidly, registering a value of <1.0 µg B[a]P/g past 5 km.

Recently, Fanning *et al*. (1989) sampled sediments near the Iona sewage treatment plant outfall which now discharges at depth beyond Sturgeon Bank. They found that the total PAH concentration did not exceed 0.10 µg/g. Levels ranging from 0.166 to 0.177 µg total PAH/g were measured in sediments from the same area by Harding *et al*. (1988). Sediments sampled from Sturgeon and Roberts Banks were below 0.10 µg total PAH/g (Harding *et al*., 1988).

Goyette and Boyd (1989a) noted that the sediment PAH concentrations for Vancouver Harbour (Table 8) were considerably higher than those reported for the Fraser River estuary (results from Fanning *et al.* reported above). The major PAH compounds found in sediments were phenanthrene in the low molecular weight PAH range (i.e., LPAH) and fluoranthene, pyrene, chrysene, benzo[k]fluoranthene, and benzo[b]fluoranthene in the high molecular weight PAH range (i.e., HPAH). Carcinogenic PAHs including benzo[a]pyrene and indeno[1,2,3-cd]pyrene were also present. B[a]P concentration ranged from 0.73 to 1.6 µg/g in the Inner Harbour sediments and 1.9 to 3.0 µg/g in the Port Moody Arm sediments. During a two-year sampling period, the heavily industrialized Port Moody Arm and moderately industrialized Inner Harbour test sites yielded significantly elevated PAH levels compared to the lesser impacted Outer Harbour site. These investigators also concluded that PAH data for sediments in Vancouver Harbour were insufficient to estimate a baseline level.

Sediment samples taken from Estevan Sound, British Columbia, were found to contain 0.0034-0.010 µg/g, 0.0034-0.016 µg/g, and 0.027-0.068 µg/g of LPAH (phenanthrene+ anthracene), HPAH (chrysene+triphenylene+benz[a]anthracene+benzofluoranthenes) and TPAH, respectively (Cretney *et al*., 1983). This site is located approximately 120 km or greater (linear distance) from the aluminum smelter in the Kitimat Arm and is not subject to any other forms of human impact; consequetly, it may be considered sufficiently uncontaminated to reflect background PAH levels.

TABLE 8

Ranges of mean PAH concentrations (µg/g) in the Vancouver Harbour sediments

(From Goyette and Boyd, 1989a)

Site	***Lower (2-3 ring) PAH*** #	***Higher PAH*** #	***Total PAH***
Outer Harbour 1985/86 1987	0.32 0.31 - 0.79	1.13 1.45 - 3.04	1.45 1.76 - 3.83

Inner Harbour 1985/86 1987	0.32 - 7.51 0.59 - 3.39	0.71 - 8.82 1.82 - 14.03	1.03 - 14.11 2.41 - 17.42
Port Moody Arm 1985/86 1987	0.71 - 1.84 1.06 - 5.64	2.23 - 3.68 2.97 - 31.97	2.94 - 4.93 4.04 - 36.73

Low molecular weight PAH include naphthalene, acenaphthylene, acenapthene, anthracene, phenanthrene, and fluorene; High molecular weight PAH include fluoranthene, pyrene, chrysene, benzo[a]anthracene, benzo[b]fluoranthene, benzo[k]fluoranthene, benzo[a]pyrene, benzo[ghi]perylene, dibenzo[ah]anthracene, and indeno[1,2,3-cd]pyrene.

Cretney *et al*. (1983) also sampled and dated sediment cores collected off Emsley Point at the southern extension of north Kitimat Arm, possibly impacted by the aluminum smelter (established in early 1950s). The results showed a dramatic decrease in the total PAH concentration from 2.0 µg/g at 0-3 cm depth to 0.026 µg/g at 75-78 cm depth. It was also evident that a rapid accumulation began sometime between 1944-1959. Prior to this period, the TPAH concentration of 0.031 ± 0.006 µg/g sediment (or 1.8 ± 0.3 µg/g Carbon) was fairly constant for over a century.

Similar trends (i.e, decrease in PAH with increasing depth) were observed by Heit *et al*. (1981) in sediment core samples collected from Sagamore and Woods lakes in the Adirondack region of New York. The sudden increase in the PAH concentrations of surface sediments was credited to the increase in atmospheric particulates originating from various combustion sources, such as industry, vehicles and heating processes (Heit *et al*. , 1981; NRC, 1983). The average background PAH concentrations in the sediments are shown in Table 9.

TABLE 9

Average (± standard deviation) background concentrations (µg/g) of polycyclic aromatic hydrocarbons in 2 New York lakes sediments
(From Heit *et al.,* 1981)

Polycyclic Aromatic Hydrocarbons	***Sagamore Lake (depth > 9-11 cm)***	***Woods Lake (depth > 6-9 cm)***
Phenanthrene	0.020 ± 0.006	0.040 ± 0.010

Anthracene	< 0.002	0.003 ± 0.001
Fluoranthene	0.010 ± 0.006	0.015 ± 0.005
Pyrene	0.008 ± 0.004	0.050 ± 0.030
Benzo[a]fluorene	< 0.004	0.005 ± 0.002
Benz[a]anthracene	< 0.001	0.002 ± 0.002
Chrysene/triphenylene	< 0.004	0.006 ± 0.005
Benzo[b]fluoranthene	0.004 ± 0.002	0.040 ± 0.020
Benzo[k]fluoranthene	0.002 ± 0.001	0.007 ± 0.002
Benzo[e]pyrene	< 0.002	0.007 ± 0.003
Benzo[a]pyrene	< 0.002	0.004 ± 0.003
Perylene	2.600 ± 1.400	0.500 ± 0.300
Indeno[cd]pyrene	0.003 ± 0.001	0.006 ± 0.006
Dibenzo[a,c/a,h]anthracene	< 0.004	< 0.003
Benzo[ghi]perylene	0.029 ±0.016	0.083 ± 0.047
Coronene	< 0.003	< 0.003
Dibenzo[a,e]pyrene	< 0.003	< 0.003

Swain and Walton (1990a, b) measured PAH concentration in sediments collected from several sites in the Fraser River (freshwater sediments) and Boundary Bay (marine sediments), in British Columbia. In most samples PAH levels were below the detection limits (i.e., 0.005 µg/g for acenaphthene, acenaphthylene, anthracene, fluorene, naphthalene, and phenanthrene; 0.02 µg/g for benzo[a] pyrene, benzo[b]fluoranthene, benzo[g,h,i]perylene, benzo[k]fluoranthene dibenzo[a,h] anthracene, and indeno[1,2,3-c,d]pyrene; and 0.01 µg/g for benz[a]anthracene, chrysene, fluoranthene, and pyrene). The maximum PAH concentrations for the freshwater and marine sediments are shown in Table 10.

TABLE 10

Maximum concentrations (µg/g) of polycyclic aromatic hydrocarbons in the Fraser River and Boundary Bay sediments, in British Columbia
(From Swain and Walton, 1990a,b)

Polycyclic Aromatic Hydrocarbons	***Fraser River (freshwater)***	***Boundary Bay (marine)***
Acenaphthene	< 0.005	0.081

Acenaphthylene	0.053	0.062
Anthracene	0.070	0.097
Benz[a]anthracene	0.012	0.072
Benzo[a]pyrene	0.100	<0.02
Benzo[b]fluoranthene	0.069	0.190
Benzo[ghi]perylene	0.091	0.650
Benzo[k]fluoranthene	0.150	0.130
Chrysene	<0.010	0.074
Dibenzo[a,h]anthracene	0.370	1.240
Fluoranthene	0.085	0.910
Fluorene	0.005	0.079
Indeno[1,2,3-c,d]pyrene	0.32	0.400
Naphthalene	0.13	0.021
Phenanthrene	0.400	0.092
Pyrene	0.040	0.300

Wan (1991) measured concentrations of 16 PAHs (see Table 12 for the list of PAHs) in the ballasts from five railway rights-of-way and the adjacent ditches (6 locations) flowing to salmon streams in the Lower Mainland of British Columbia. All 16 PAHs were found in the sediments of all ditches sampled adjacent to the rights-of-way. The mean concentrations for LPAHs varied between 0.25 µg/g for acenaphthylene and 36.7 µg/g for phenanthrene, and for HPAHs between 0.29 µg/g for dibenz[a,h]anthracene and 91.3 µg/g for fluoranthene. The author noted that the PAH mean concentrations in the ditches were several times higher than those in the Inner Harbour sediments of Burrard Inlet (Goyette and Boyd, 1989 a); also, the 16 PAHs were not detected[3] in the ditch sediments of pristine parkland and agricultural pump stations.

4.4.3 Biota

PAH concentrations in biota depend upon their proximity to the source of pollution, species ability to biotransform, and bioavailability of soil- or sediment-sorbed aromatic hydrocarbons.

The level of anthracene in seaweed collected from Osaka Harbour, Japan, averaged 4 ng/g dw. The average HPAH concentrations in seaweed for this site ranged from 2 ng/g dw for dibenzo[a,h]anthracene to 72 ng/g dw for benzo[a]pyrene (Obana and

Kashimoto, 1981). Harrison *et al*. (1975) reported 60 ng/g ww TPAH for marine algae from Greenland while Lee and Grant (1981) reported up to 60 ng B[a]P/g dw for marine algae. Seuss (1976) observed that 10-50 ng/g dw of B[a]P was taken up by the freshwater alga, *Chlorella vulgaris.* Lee and Grant (1981) stated that the worldwide B[a]P concentration for marine plankton ranged up to 400 ng/g dw.

To study baseline levels of B[a]P, Dunn and Young (1976) collected mussels (*Mytilus californianus,* and *Mytilus edulis)* from 19 mainland and 6 island stations situated throughout the Southern California Bight. The coastal area in this region is inhabited by about 5% of the U.S.A. population. At both mainland and island stations, levels of contamination in mussels taken from locations at least 1 km from piers and wharfs were generally at or near the detection limit of 0.1 ng/g ww. The samples which recorded elevated levels of B[a]P were those in which the mussels were growing directly on creosoted pilings (e.g., up to 8.2 ng/g ww), or were growing near large harbours or marinas (e.g., up to 2.3 ng/g ww). The data collected in Oregon from a relatively pristine Alsea bay site showed non-detectable levels of B[a]P (< 0.4 ng/g dw or < 0.10 ng/d ww) in the tissues of gaper clams (*Tresus capax*), blue mussels (*Mytilus edulis*) and softshell clams (*Mya arenaria*) (Mix *et al*., 1977).

In British Columbia, PAHs in shellfish were first reported by Dunn and Stich (1975). Levels up to 0.2 ng B[a]P/g ww were measured in mussels (*M. californianus*) from the open west coast of Vancouver Island, 5 km from human activity; 42.8 ng/g ww B[a]P were found in mussels from a poorly flushed inlet (False Creek) with heavy boat and industrial use. At four out of five sites in the Vancouver Harbour, B[a]P uptake by mussels fluctuated seasonally. These seasonal fluctuations were attributed to variations in pollution pattern rather than physical differences such as temperature, or physiological differences related to the breeding cycle of the organisms.

Duncan (1984) monitored PAHs in commercial shellfish from seven British Columbia locations, with pacific oysters (*Crassostrea gigas*) collected at five of the stations (e.g., Henry Bay, Denman Island, Comox Harbour, Cortes Island, and Barkley Sound), butter clams (*Saxidomas giganteus*) from the sixth (i.e., Seal Islets), and geoducks (*Panopea generosa*) from the seventh location (i.e., Courtenay area). The low to moderate levels of PAH (and metals, which are not shown here) led the investigator to conclude that the major shellfish harvesting sectors of B.C. were located in areas of good water quality (Table 11).

TABLE 11

PAH concentrations in commercial shellfish from British Columbia
(From Duncan, 1984)

Compound	***C. gigas***	***S. giganteus***	***P. generosa***
	ng/g wet weight	***ng/g wet weight***	***ng/g wet weight***
Fluoranthene	23.5-96.5	6.7	44.0
Benz[a]anthracene	1.8-5.0	1.7	3.9
Benzo[b]fluoranthene	2.0-5.1	0.6	3.2
Benzo[k]fluoranthene	0.6-1.9	0.1	0.9
Benzo[a]pyrene	0.3-0.8	0.4	0.9
Indeno[1,2,3-cd]pyrene	0.2-0.5	0.3	0.6
Benzo[b]chrysene	0.1-0.4	0.5	0.4

Crustaceans possess an MFO system which is capable of converting most PAHs into water- soluble metabolites. Most of these compounds and their resulting products are distributed in the hepatopancreas (the main site of MFO), although residual levels are often detectable in other organs and tissues (Dunn and Stich, 1975). For example, American lobsters (*Homerus americanus*) held in creosoted tidal ponds in Nova Scotia were found to have 35 times as much PAHs in the hepatopancreas than in the tail muscle (Uthe *et al*., 1984). This study found that hepatopancreatic levels of these substances tended to be greater than tail muscle concentrations regardless of whether the lobster was held in a contaminated pond or was freshly obtained from a relatively pristine site. In a similar study of PAH uptake in *H. americanus* from a minor diesel oil spill in Arnold's Cove, Newfoundland, Williams *et al*., 1985 noted that PAHs preferentially concentrate in the hepatopancreas of the animals.

Goyette and Boyd (1989a) analyzed Dungeness crab (*Cancer magister*) tissues (muscle and hepatopancreas) for PAHs. The results of Goyette and Boyd are reproduced in Table 12. The animals were caught from False Creek, upper Indian Arm, Coal Harbour, and Port Moody Arm (Ioco) off Vancouver Harbour. PAH concentrations in both muscle and hepatopancreas tissues were non-detectable in samples from upper Indian Arm (detection limit = 0.02 µg/g dw except for indeno(1,2,3-cd)pyrene, dibenzo(a,h)anthracene, and benzo(g,h,i)perylene which was 0.06 µg/g dw). Crab samples from False Creek had the highest concentrations in both muscle (0.025 - 0.169 µg total PAH/g dw) and hepatopancreas (0.148 - 1.24 µg total PAH/g dw)

tissues. Similar to the Dunn and Stich (1975) observations above, hepatopancreas tissue was the primary site for PAH accumulation.

MFOs in fish have been extensively studied and are believed to be very similar to those of mammals. These enzymes are located in microsomal tissues present in the livers of these animals and serve as a detoxifier of toxic substances. Enzymatic activity of MFOs in fish is more effective in metabolizing PAHs than it is in lower animals (e.g., invertebrates). As a result, these vertebrates tend to bioaccumulate few PAHs (Lawrence and Weber, 1984). West *et al*., (1984) observed that higher molecular weight PAHs, which include the largest class of chemical carcinogens, do not accumulate in fish. Muscles of six species of fish in Lake Ontario were found to contain 3-8 µg/kg ww of TPAH (Eisler, 1987), while trout tissues from Lake Maskinonge, Ontario, did not exceed individual PAH concentrations of 1.5 µg/kg ww (Pancirov and Brown, 1977). Carp (*Cyprinus carpio* , a herbivore) from Hamilton Harbour and Detroit River contained 0.003-0.243 µg/kg ww of PAH (Perylene, Benzo[k]fluoranthene, Benzo[a]pyrene, and Coronene), while Northern Pike (*Esox lucius* , a carnivore) from the same

TABLE 12

PAH concentrations* in Dungeness crab tissue sampled from Vancouver Harbour in 1986 and 1988
(From Goyette and Boyd, 1989a)

Location	***Coal Harbour***		***Ioco***		***False Creek***	
	Hepato+	***Muscle***	***Hepato***	***Muscle***	***Hepato***	***Muscle***
	µg/g dry weight		**µg/g dry weight**		**µg/g dry weight**	
Lower Molecular Weight Polycyclic Aromatic Hydrocarbons (LPAH)						
Naphthalene	<0.02-0.021	<0.02	<0.02-0.080	<0.02	0.021-0.076	0.010-0.069
Acenaphthylene	<0.02	<0.02	<0.02-0.010	0.006-0.010	0.010-0.024	<0.02-0.010
Acenaphthene	0.020-0.460	<0.02-0.028	<0.02-0.180	0.005-0.010	0.039-0.270	0.005-0.010
Fluorene	<0.02-0.067	<0.02-0.010	<0.02-0.170	<0.02	0.010-0.060	0.005-0.010

Phenanthrene	<0.02-0.015	<0.02	<0.02-0.270	<0.02	0.010-0.150	0.005-0.015
Anthracene	<0.02	<0.02	<0.02-0.076	<0.02	0.005-0.061	<0.02-0.005
Total LPAH	<0.02-0.563	<0.02-0.038	<0.02-0.786	0.011-0.020	0.095-0.119	0.025-0.119
High Molecular Weight Polycyclic Aromatic Hydrocarbons (HPAH)						
Fluoranthene	<0.02	<0.02	<0.02-0.110	<0.02	0.010-0.190	<0.02-0.010
Pyrene	<0.02	<0.02	<0.02-0.10	<0.02	0.010-0.140	<0.02-0.010
Benz[a]anthracene	<0.02	<0.02	<0.02	<0.02	0.010-0.059	<0.02-0.010
Chrysene	<0.02	<0.02	<0.02	<0.02	0.023-0.140	<0.02-0.010
Benzo[b+k] fluoranthene	<0.02	<0.02	<0.02	<0.02	<0.02-0.052	<0.02-0.005
Benzo[a]pyrene	<0.02	<0.02	<0.02	<0.02	<0.02-0.021	<0.02-0.005
Indeno(1,2,3-c,d) pyrene	<0.06	<0.06	<0.06	<0.06	<0.06	<0.06
Dibenzo[a,h] anthracene	<0.06	<0.06	<0.06	<0.06	<0.06	<0.06
Benzo[g,h,i] perylene	<0.06	<0.06	<0.06	<0.06	<0.06	<0.06
Total HPAH	<0.02 or <0.06	<0.02 or <0.06	<0.02-0.210	<0.02 or <0.06	0.053-0.602	<0.06-0.050
Total PAH	<0.02-0.563	<0.02-0.038	<0.02-0.996	0.011-0.020	0.148-1.240	0.025-0.169

*Levels below the detection limit are semi-quantitative estimates of PAHs.
+Hepatopancreas

sources contained 0.016-0.074 µg/kg of the same (GLSAB, 1983). English Sole (*Parophrys vetulus*) sampled from two urban bays and two non-urban bays in Puget

Sound, Washington were all found to contain <0.05 µg/kg dw of total aromatic hydrocarbons in muscle tissues.

Goyette and Boyd (1989b) examined PAH levels in the liver and muscle tissues of English Sole from Vancouver Harbour. Concentrations ranging from 0.001-0.037 µg/g dw of LPAH and trace-0.074 µg/g of HPAH were detected in the fish livers from the outer Harbour. The inner Harbour fish liver samples contained 0.013 µg/g fluoranthene, 0.001 µg/g anthracene and 0.014 µg/g phenanthrene. In Port Moody Arm, only phenanthrene was detected at 0.019 µg/g dw. By comparison, the muscle samples contained non-detectable levels of both LPAH and HPAH except for phenanthrene and fluoranthene which were present in trace amounts and 0.013 µg/g dw, respectively.

In several fish species collected from the North and Main Arms of the Fraser River, British Columbia, Swain and Walton (1989) found PAHs in both muscle and liver tissues. The PAH concentrations were much greater in the liver than in the muscle samples (Table 13).

TABLE 13

PAH concentrations (µg/g wet weight) in muscle and liver (in parenthesis) tissues of the Fraser River (British Columbia) fish samples

(From Swain and Walton, 1989)

PAH	Largescale sucker	Northern squawfish	Peamouth chub	Redside shiner	Staghorn sculpin	Starry flounder	Threespine sticklebac k
Acenaphthene	<0.004	<0.004 (0.035)	<0.004 (0.027)	<0.004	<0.004	<0.004	0.008
Acenaphthylene	<0.004	<0.004 (0.022)	<0.004 (0.046)	<0.004	<0.004 (0.032)	<0.004 (0.081)	<0.004
Anthracene	<0.004	<0.004 (0.020)	<0.004	<0.004	<0.004	<0.004	<0.004
Benz[a] anthracene	<0.01	<0.01 (0.035)	<0.01 (0.093)	<0.01	<0.01	<0.01	<0.01
Benzo[a]pyrene	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02
Benzo[b] fluoranthene	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02

Benzo[g,h,i] perylene	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02
Benzo[k] fluoranthene	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02
Chrysene	<0.01	<0.01	<0.01 (0.077)	<0.01	<0.01	<0.01	<0.01
Dibenzo[a,h] anthracene	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02
Fluoranthene	<0.01	<0.01	<0.01	<0.01	<0.01	0.01	0.011
Fluorene	<0.004 (0.020)	<0.004 (0.046)	<0.004 (0.024)	<0.004	<0.004 (0.067)	<0.004 (0.048)	<0.004
Indeno[1,2,3-c,d]pyrene	<0.02	<0.02 (0.12)	<0.02	<0.02	<0.02	<0.02	<0.02
Naphthalene	<0.004 (0.074)	<0.004 (0.23)	<0.004 (0.16)	<0.004	<0.004 (0.095)	<0.004 (0.12)	<0.004
Phenanthrene	<0.004 (0.025)	<0.004 (0.034)	0.02 (0.060)	<0.004	<0.004 (0.038)	0.02 (0.071)	0.026
Pyrene	<0.01	<0.01 (0.090)	<0.01 (0.12)	<0.01	<0.01	<0.01	<0.01

2 (Based on 6 measurements at 6 locations, including those containing non-detectable levels; the non-detectable levels were considered to have a zero value. The dection limits for LPAHs and most of HPAHs were 0.1 µg/L and 0.5 µg/L, respectively; the detection limit for benz[g,h,i]perylene, dibenz[a,h]anthracene, and indeno[1,2,3-cd]pyrene was 0.8 µg/L.

3 the detection limits for LPAHs, and chrysene = 0.01 µg/g; benz[a]anthracene, fluoranthene, and pyrene = 0.05 µg/g; benz[b]fluoranthene, benz[k]fluoranthene = 0.08 µg/g; benz[a]pyrene, benz[g,h,i]perylenedibenz[a,h]anthracene, and indeno[1,2,3-cd]pyrene = 0.10 µg/g.

5.0 DRINKING WATER SOURCES

5.1 Effects

The following discussion is based on a recent review commissioned by the US Public Health Service (USPHS, 1990) on the toxicology of PAHs. Most of the toxicological information was derived from experimental animals exposed to PAHs under controlled conditions.

Mackenzie and Angevine (1981) administered B[a]P by gavage (i.e., introduction of the contaminant into the stomach by tube) to pregnant CD-1 mice during gestation at the rate of 10, 40, and 160 mg/kg body weight (bw)/d, and found that the viability of the litters at birth was significantly reduced in the highest dosed group. In all treatment groups, the mean pup weight was significantly reduced by 42 days of age. To study postnatal development and reproductive functions, these investigators bred F1 progeny (which were exposed prenatally to B[a]P) with untreated animals. It was found that the F1 progeny from the 10 mg/kg bw/d group experienced decreased fertility with associated alterations in gonadal morphology and germ-cell development. The F1 progeny from higher dose groups exhibited total sterility.

The development of forestomach tumors (papillomas and carcinomas) was studied by Neal and Rigdon (1967) in mice exposed to dietary B[a]P. In mice fed 33 mg B[a]P/kg bw/d for periods of 1 to 7 days the incidence of forestomach tumor increased following 2 or more days of exposure (total dose= 2 mg/animal), while mice fed 13.3 mg B[a]P/kg bw/d for 110 days (total dose= 4.48 mg/animal) did not develop tumors. It was suggested that there are no cumulative carcinogenic effects of B[a]P or its metabolites in mice.

Chronic oral administration of a total dose of 4.5 g anthracene/rat in the diet of BD1 or B111 rats for 78 weeks did not produce tumors (Druckrey and Schmahl, 1955). The carcinogenic potential of PAHs is shown in Table 1.

Studies related to effects on humans from exposure to PAHs, singly or collectively, are rare. Sax (1979) reported a lethal concentration of 100 µg/g naphthalene for human children (an accidental ingestion). Epidemiological studies have shown increased mortality due to lung cancer in humans exposed to coke-oven emissions, roofing-tar emissions, and cigarette smoke. Each of these mixtures contains B[a]P, chrysene, benz[a]anthracene, benzo[b]fluoranthene, and dibenzo[a,h]anthracene, as well as other potentially carcinogenic PAHs and other carcinogenic and potentially carcinogenic

chemicals, tumor promoters, initiators, and cocarcinogens such as nitrosamines, coal tar pitch, and creosote (USPHS, 1990). Because of the complex nature of the mixtures, it is difficult to evaluate the contribution of any single PAH to the total carcinogenicity of these mixtures.

Humans can be exposed to PAHs via air, water, and food. In the U.S., Santodonato (1981) estimated general population exposure to total PAH, benzo[a]pyrene, and carcinogenic PAHs (i.e., total of benzo[a]pyrene+ benzo[j]fluoranthene+ indeno[1,2,3-cd]pyrene). The results (Table 14) suggested that drinking water was a minor contributor of PAH body burden in humans.

Lioy *et al*. (1988) conducted a multimedia study of human exposure to B[a]P in a rural town in New Jersey. The major industry in the town was a grey-iron pipe manufacturing plant which contributed to high PAH levels in the atmosphere. The mean outdoor air concentration of B[a]P was 0.0009 µg/m^3 (B[a]P concentration in homes varied from 0.0001 to 0.0081 µg/m^3), whereas the maximum concentration of B[a]P in samples of food was 0.001 µg/g wet weight. Ingestion of B[a]P was estimated to range between 0.01 and 4.0 µg/person/wk. B[a]P concentration in drinking water was less than the detection limit of 0.0001 µg/L. In comparing the inhalation and ingestion pathways in each home, these investigators found that potential intake could be similar in each medium.

5.2 Criteria from other jurisdictions

Drinking water criteria for PAHs from various jurisdictions are listed in Table 15.

Based on the premise that drinking water should be comparable in quality with unpolluted ground water, the World Health Organization (WHO) in 1970 and 1971 recommended a limit of 0.2 µg/L for the sum of six PAHs in drinking water (i.e., fluoranthene, benzo[a]pyrene, benzo[g,h,i]perylene, benzo[b]fluoranthene, benzo[k]fluoranthene, and indeno[1,2,3-cd]pyrene) (WHO, 1984). Concentrations of these indicator PAHs ranged from 0.01-0.05 µg/L in ground water and 0.05-0.25 µg/L in relatively unpolluted rivers, with higher levels in polluted rivers and effluents of the world. Subsequent studies, however, revealed that the concentrations of these PAHs in drinking water were considerably lower than the recommended WHO standards (WHO, 1984).

TABLE 14

Human Exposure to PAHs

Source	***Benzo[a]pyrene***	***Carcinogenic PAHs***	***Total PAHs***
	µg/d (%)	**µg/d (%)**	**µg/d (%)**
Air	0.0095-0.0435(0.6-2.7%)	0.038(90%)	0.207(11-52%)
Water	0.0011(<0.1%)	0.0042(10%)	0.027(1.5-7%)
Food	0.16-1.6(94-97%)	-	0.16-1.6(40-87%)
Total	**0.17 - 1.6**	**0.042**	**0.4 - 1.8**

TABLE 15

PAH Criteria for Drinking Water from various Jurisdictions

CRITERIA STATEMENT	***CRITERIA VALUE* (µg/L)**	***JURISDIC-TION***	***DATE***	***REFERENCES***
Drinking water standard: (B[a]P+FLAN +B[b]FLAN+B[k]FLAN+B[g,h,i] PERY+I[1,2,3-cd]P)	0.2	WHO	1970-1971	WHO (1984)
Drinking water standard: B[a]P	0.01	WHO	1984	WHO (1984)
Ambient criteria to protect human health from ingesting contaminated water and organisms: Total PAH ·cancer risk level = 10-5 ·cancer risk level = 10-6 ·cancer risk level = 10-7	0.02800 0.00280 0.00028	USEPA	1980	USEPA (1980)

Guidance value for drinking water supplies: ·Naphthalene ·Acenaphthene, Fluorene, Anthracene, Phenanthrene, Fluoranthene and Pyrene ·B[a]ANTH, Chrysene, B[b]FLAN, B[k]FLAN, and B[a]P .	10 50 0.002	New York	1985	New York State, 1985
Maximum acceptable concentration in drinking water for B[a]P	0.01	Canada	1989	HWC (1989)
Drinking water quality standards: -B[a]P -all other PAHs	0.03 0.029	Kansas	1988	FSTRAC, 1988
Drinking water quality standards	25	Maine	1988	FSTRAC, 1988
Drinking water quality standards	0.028	Minnesota	1988	FSTRAC, 1988
Drinking water quality standards: -B[a]P -all other PAHs	10 30	New Mexico	1988	FSTRAC, 1988
Drinking water quality standards (all PAHs)	1.0	New Jersey	1989	NJDEP (1989)

The World Health Organization (1984) also recommended a guideline of 0.01 µg/L for B[a]P alone, based on (a) available toxicity data for B[a]P (Neal and Rigdon, 1967) and its association with other PAHs of known carcinogenicity, (b) a linearized multistage model for lifetime cancer exposure risk, considering 1 in 100 000 as an acceptable risk.

The USEPA (1980) criteria, cited as total PAH concentration in untreated ambient waters, are designed to protect human health from consumption of contaminated water and contaminated organisms inhabiting the water. The USEPA criterion at the 10-5 cancer risk level is less stringent than the Health and Welfare Canada and WHO (1984)

criteria, considering that the USEPA criterion, although expressed in terms of total PAH, is actually based on B[a]P.

The New York State guidelines for several lower molecular weight PAHs(e.g., naphthalene, acenaphthene, fluorene, anthracene, phenanthrene, fluoranthene, and pyrene) and high molecular weight PAHs (e.g., Benz[a]anthracene, Chrysene, Benzo[b]fluoranthene, Benzo[k]fluoranthene, and Benzo[a]pyrene) are for class AA waters. The class AA waters are designated for use as drinking water supplies which will meet the drinking water standards with treatment and/or disinfection.

See Guideline Summary: Drinking Water Sources on the ambient water quality guideline website for current PAH guideline recommendations.

6.0 AQUATIC LIFE

6.1 Freshwater

6.1.1 Lethal and acute effects

The acute (96-h LC_{50}) and lethal effects of PAHs in the freshwater environment are shown in Table 16. Figure 4 shows a graphic summary of the data.

The toxicity of PAHs to aquatic organisms is determined by several factors which include: (a) the PAH type (e.g., molecular weight, alkyl substitution, etc.), (b) the species of the organism exposed, and (c) the duration and the type of exposure to a given PAH (Table 16). In general, fish appear to be the most sensitive of the aquatic organisms to PAHs (Figure 4). However, there are exceptions to this general trend. For instance, the 96-h LC_{50} for acenaphthene (ANA) was lower for the alga *Selenastrum capricornutum* (EC_{50} for cell count = 520 µg/L) than for brown trout (*Salmo trutta*) (LC_{50} = 580 µg/L) or fathead minnow (*Pimephales promelas*) (LC_{50} = 610 µg/L). The longer exposure periods reduce the LC_{50}s for both cladoceran (*Daphnia magna*) and bluegill (*L. macrochirus*) exposed to acenaphthene and fluoranthene (FLAN) (LeBlanc, 1980; Buccafusco et al., 1981).

The minimum LC_{50} for relatively more soluble and lower molecular weight PAHs, containing 3 or less aromatic (benzene) rings in their structure (Table 1), was found for

rainbow trout (*Oncorhynchus mykiss*) exposed to phenanthrene (LC_{50} = 30 µg/L, Table 16).

The higher molecular weight PAHs (containing more than 3 aromatic rings in their structure) such as benz[a]anthracene and and benzo[a]pyrene, have also been shown to be acutely toxic to invertebrates at low concentrations (5-10 µg/L - Table 16). In natural aquatic environments, this condition may not be achieved because of the low solubility of HPAHs. Note that the 96-h LC_{50} (5 µg/L) for *Daphnia pulex* exposed to B[a]P was higher than its solubility (3.8 µg/L) in water. The solubility of B[a]ANTH (Table 1) and the 48-h LC_{50} for *Daphnia pulex* exposed to B[a]ANTH were nearly identical.

Alkyl homologues of PAHs are generally more toxic to aquatic life than the parent compound. For instance, the 48-h EC_{50} for *Daphnia pulex* exposed to anthracene (750 µg/L) was much higher than that obtained when the organisms were exposed to methyl anthracene (48-h EC_{50}=96 µg/L) or 9-methoxy anthracene (48-h EC_{50}=400 µg/L) (Table 16).

TABLE 16

Lethal and Acute Toxicity of PAHs to Freshwater Aquatic Life

Organism	***PAH***	***EC_{50}/LC_{50}* (µg/L)**	***Duration* (hours)**	***System*[1]**	***References***
Alga (*S. capricornutum*)	NA	2 960	4	S,U	Millemann *et al*., 1984
Diatom (*N. palea*)	NA	2 820	4	S,U	Millemann *et al*., 1984
Alga (*C. vulgaris*)	NA	33 000	48	S,U	Kauss & Hutchinson, 1975
Cladoceran (*D. magna*)	NA	8 570	48	S,U	USEPA, 1978
D. magna	NA	17 000	24	S,U	LeBlanc, 1980
D. magna	NA	8 600	48	S,U	LeBlanc, 1980
D. magna	NA	6 600-13 200	24	S,U	Crider *et al*., 1982
D. magna	NA	3 400-4 100	48	S,U	Crider *et al*., 1982

D. magna	NA	2 160	48	S,M	Millemann *et al*., 1984
Cladoceran (*D. pulex*)	NA	3 400	48	S,U	Geiger & Buikema Jr., 1981
D. pulex	NA	2 920-3 890	48	S,U	Geiger & Buikema Jr., 1982
D. pulex	NA	1 000	96	S,M	Trucco *et al* ., 1983
Amphipod (*G. minus*)	NA	3 930	48	S,M	Millemann *et al*., 1984
Midge (*C. tentans*)	NA	2 810	48	S,M	Millemann *et al*., 1984
Snail (*P. gyrina*)	NA	5 020	48	S,M	Millemann *et al*., 1984
Dragonfly (*S. cingulata*)	NA	1 000-2 500	96	S,U	Correa and Coler, 1983
Mosquitofish (*G. affinis*)	NA	220 000	24	S,U	Wallen *et al*., 1957
G. affinis	NA	165 000	48	S,U	Wallen *et al*., 1957
G. affinis	NA	150 000	96	S,U	Wallen *et al*., 1957
Fathead minnow (*P. promelas*)	NA	7 900	96	FT,M	DeGraeve *et al*., 1982
P. promelas	NA	1 990	96	FT,M	Millemann *et al*., 1984
Largemouth Bass (*M. salmoides*)	NA	680	168	FT,M	Millemann *et al*., 1984
Rainbow trout (*O. mykiss*)	NA	1 600	96	FT,M	DeGraeve *et al*., 1982
O. mykiss	NA	120	648	FT,M	Millemann *et al*., 1984
O. mykiss	NA	110	648	FT,M	Black *et al* ., 1983

Coho Salmon (*O. kisutch*)	NA	5 600	<6		Holland *et al*., 1960
O. kisutch	NA	2 100	96	FT,M	Moles *et al*., 1981
O. kisutch	NA	3 200	96		Neff, 1985
Cladoceran (*D. pulex*)	1,3 d-MNA	770 (EC_{50})	48	S	OMOE, 1990
D. pulex	2,6 d-MNA	190 (EC_{50})	48	S	OMOE, 1990
Alga (*S. capricornutum*)	ANA	520	96		USEPA, 1978
Cladoceran (*D. magna*)	ANA	41 200	48	S,U	USEPA, 1978
D. magna	ANA	41 000	48	S,U	LeBlanc, 1980
Midge (*Paratanytarsus sp*.)	ANA	60-1 650	48	S,M	Lemke and Anderson, 1984
Snail (*A. hypnorum*)	ANA	245 000	96	FT,M	Holcombe *et al*., 1983
Bluegill (*L. macrochirus*)	ANA	1 700	96	S,U	USEPA, 1978
Bluegill (*L. macrochirus*)	ANA	7 200	24	S,U	Buccafusco *et al*., 1981
L. macrochirus	ANA	1 700	48	S,U	Buccafusco *et al*., 1981
Rainbow trout (*O. mykiss*)	ANA	1 130	48	FT,M	Holcombe *et al*., 1983
O. mykiss	ANA	800	72	FT,M	Holcombe *et al*., 1983
Brown trout (*S. trutta*)	ANA	650	48	FT,M	Holcombe *et al*., 1983
S. trutta	ANA	600	72	FT,M	Holcombe *et al*., 1983
S. trutta	ANA	580	96	FT,M	Holcombe *et al*., 1983

Fathead minnow (*P. promelas*)	ANA	1 600	96	FT,M	Holcombe *et al*., 1983
P. promelas	ANA	610	96	FT,M	Cairns & Nebeker, 1982
Channel catfish (*I. punctatus*)	ANA	1 720	96	FT,M	Holcombe *et al*., 1983
Mayfly (*H. bilineata*)	FL	5 800	120	S,M	Finger *et al*., 1985
Snail (*M. potosensis*)	FL	5 600	96	S,M	Finger *et al*., 1985
Amphipod (*G. seudolimnaeus*)	FL	600	96	S,M	Finger *et al*., 1985
Cladoceran (*D. magna*)	FL	430	48 (EC_{50})	S,M	Finger *et al*., 1985
Cladoceran (*D. pulex*)	FL	210	48 (EC_{50})	S	OMOE, 1990
Bluegill (*L. macrochirus*)	FL	910	96	S	Finger *et al*., 1985
Rainbow trout (*S. gairdneri*)	FL	820	96	S	Finger *et al*., 1985
Cladoceran (*D. pulex*)	ANTH	750 (EC_{50})	48	S	OMOE, 1990
Fathead minnow (*P. promelas*)	ANTH	360	24	S,M	Kagan *et al* ., 1985
Cladoceran (*D. pulex*)	methyl-ANTH	96 (EC_{50})	48	S	OMOE, 1990
Cladoceran (*D. pulex*)	9-methoxy ANTH	400 (EC_{50})	48	S	OMOE, 1990
Alga (*S. capricornutum*)	ACR	900	96		Blaylock *et al* ., 1985
Amphipod (*G. minus*)	ACR	1 870	48	S,M	Milleman *et al* ., 1984
Cladoceran (*D. magna*)	ACR	2 050	48	S,M	Milleman *et al* ., 1984

Copepod (*D. clavipes*)	ACR	1 180	142	S,M	Cooney & Gehrs, 1984
Cladoceran (*D. pulex*)	ACR	2 920	24	S,M	Southworth *et al* ., 1978
Midge (*C. tentans*)	ACR	1 860	48	S,M	Millemann *et al*., 1984
Snail (*P. gyrina*)	ACR	11 000	48	S,M	Millemann *et al*., 1984
Fathead minnow (*P. promelas*)	ACR	2 900	96	FT,M	Blaylock *et al* ., 1985
P. promelas	ACR	2 240	96	S,M	Millemann *et al*., 1984
Largemouth bass (*M. salmoides*)	ACR	1 020	168	FT,M	Black *et al*., 1983
Rainbow trout (*O. mykiss*)	ACR	320	648	FT,M	Black *et al*., 1983
Cladoceran (*D. pulex*)	benz[a]ACR	449	24	S,M	Southworth *et al* ., 1978
Alga (*S. capricornutum*)	PH	940	4	S,U	Millemann *et al*., 1984
Diatom (*N. palea*)	PH	870	4	S,U	Millemann *et al*., 1984
Cladoceran (*D. magna*)	PH	700 (EC_{50})	48	S,M	Millemann *et al*., 1984
Cladoceran (*D. pulex*)	PH	1 140	48	S,U	Geiger & Buikema Jr., 1981
D. pulex	PH	960-1 280	48	S,U	Geiger & Buikema Jr., 1982
D. pulex	PH	350 (EC_{50})	48	S	OMOE, 1990
D. pulex	PH	100	96	S,M	Trucco *et al* ., 1983
Amphipod (*G. minus*)	PH	460	48	S,M	Millemann *et al*., 1984

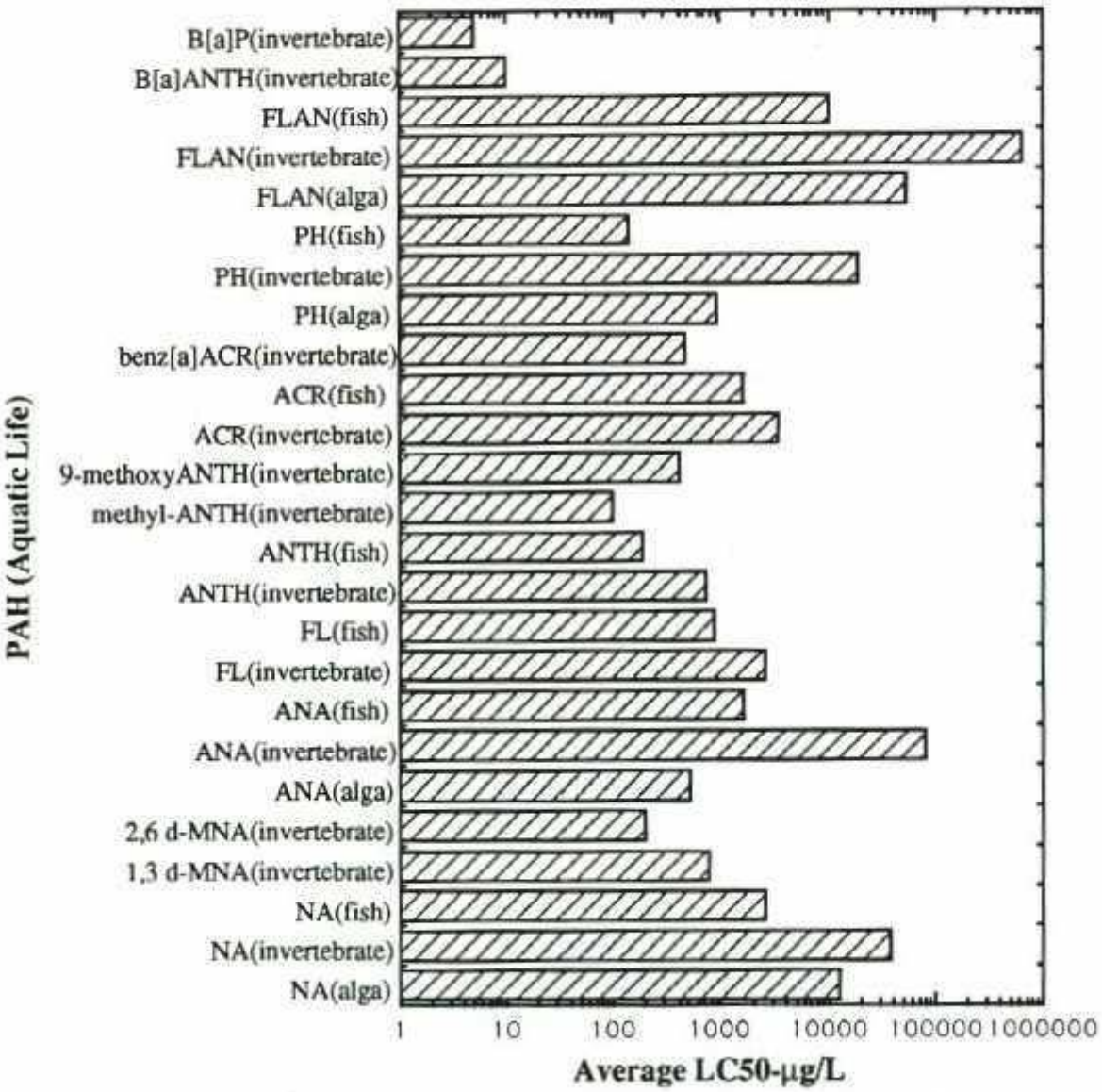
Midge (*C. tentans*)	PH	490	48	S,M	Millemann *et al*., 1984
Mosquitofish (*G. affinis*)	PH	150 000	96		USEPA, 1970
Rainbow trout (*O. mykiss*)	PH	30	648	FT,M	Millemann *et al*., 1984
Largemouth bass (*M. salmoides*)	PH	250	168	FT,M	Millemann *et al*., 1984
Alga (*S. capricornutum*)	FLAN	54 400	96		USEPA, 1978
Cladoceran (*D. magna*)	FLAN	325 000	48	S,U	USEPA, 1978
D. magna	FLAN	1.3 x 106	24	S,U	LeBlanc, 1980
D. magna	FLAN	3.2 x 105	48	S,U	LeBlanc, 1980
Fathead minnow (*P. promelas*)	FLAN	200	24	S,M	Kagan *et al* ., 1985
Bluegill (*L. macrochirus*)	FLAN	3 980	96	S,U	USEPA, 1978
L. macrochirus	FLAN	>32 000	24	S,U	Buccafusco *et al*., 1981
L. macrochirus	FLAN	4 000	48	S,U	Buccafusco *et al*., 1981
Cladoceran (*D. pulex*)	B[a] ANTH	10	48	S,M	Trucco *et al* ., 1983
Cladoceran (*D. pulex*)	B[a]P	5	96	S,M	Trucco *et al* ., 1983

1 S= static; FT= flow through; M= measured; U= unmeasured

Figure 4

FIGURE 4

PAH toxicity (LC50s) to freshwater aquatic life



6.1.2 Sublethal and chronic effects

Sublethal and chronic effects of PAHs on growth and physiological processes of aquatic algae and plants are shown in Table 17. The data suffer from some major drawbacks: (a) short exposure periods, (b) exposure levels higher than PAH aqueous solubilities, and (c) lack of constancy in the PAH concentration during the experiments. Bastian and Toetz (1982) exposed *Anabaena flos-aquae* in open flasks to several PAHs for 14 days. Within 7 days, acenaphthene, fluorene, naphthalene, and pyrene completely disappeared from solution, whereas the benzanthracene, phenanthrene, chrysene and fluoranthene concentrations were reduced to 85%, 77%, 62%, and 49%, respectively, of the initial value at the end of the 14-d period. Additionally, the concentrations of several PAHs used in the experimental solution were greater than their individual aqueous solubilities (Table 1). In the study on nitrogen fixation by *Anabaena flos-aquae,* Bastian and Toetz (1985) used short-term exposure (2 h) to minimize losses of PAHs during the experiment. Several PAHs were observed to reduce the nitrogen fixation by the alga, but the long-term effects of the PAHs are difficult to predict from these short-term studies (Table 17).

The data on long-term or chronic effects of PAHs on freshwater animals are few and suffer from the same drawbacks as noted above for aquatic plants (Table 18). Brown *et al*. (1975) exposed bluegill (*Lepomis macrochirus*) to 1 000 µg/L benz[a]anthracene and found 87% mortality in 6 months. The B[a]ANTH concentration used by these investigators in their study was much higher than the aqueous solubility of the PAH (Table 1). Finger *et al*. (1985) reported 12% mortality in bluefish exposed to 500 µg/L fluorene for 30 days.

Teratogenic effects during organogenesis (7- to 24-d post fertilization) were studied by Hannah *et al*. (1982) and Hose *et al*. (1984) in rainbow trout (*Oncorhynchus mykiss*) exposed to B[a]P-contaminated sand (1-500 µg/g) (Table 18). Gross anomalies (e.g., microphthalmia) were noted in a significant population of fish (6.8%) exposed to the contaminated sand; the average aqueous concentration was 0.2 µg B[a]P/L (Hose *et al*., 1984).

The minimum concentrations of lower molecular weight PAHs: naphthalene, acridine, and phenanthrene, causing gross developmental anomalies in rainbow trout, were found to be much higher (than B[a]P) at 230, 410, and 85 µg/L, respectively (Black *et al*., 1983).

TABLE 17

Sublethal and Chronic Toxicity of PAHs to Freshwater Algae and Plants

Organism	***PAH***	***Conc* (µg/L)**	***Effects***	***References***
Blue-green alga (*A. flos-aquae*)	NA	15 480	30-50% decrease in the N_2 fixation rate in 2 h	Bastian & Toetz, 1985
A. flos-aquae	NA	2 080	16% decrease in the N_2 fixation rate in 2 h	Bastian & Toetz, 1985
A. flos-aquae	NA	14 851	56% increase in biomass in 14 d	Bastian & Toetz, 1982
Chlamydomonas angulosa	NA	8 960	EC_{50} for photosynthesis in 3 h exposure	Hutchinson *et al*., 1980
Alga (*C. vulgaris*)	NA	330-30 000	decrease in growth rate	Kauss & Hutchinson, 1975
Chlamydomonas angulosa	1-MNA	1 700	EC_{50} for photosynthesis in 3 h exposure	Hutchinson *et al*., 1980
Chlamydomonas angulosa	2-MNA	3 550	EC_{50} for photosynthesis in 3 h exposure	Hutchinson *et al*., 1980
Blue-green alga (*A. flos-aquae*)	ANA	2 427	26% increase in biomass in 14 d	Bastian & Toetz, 1982
Blue-green alga (*A. flos-aquae*)	ANA	421-4619	no decrease in the N_2-fixation rate in 2 h	Bastian & Toetz, 1985
Duunliella bioculata	FL	550	72 h-EC_{50} for decreased growth	Heldal *et al*., 1984
Blue-green alga (*A. flos-aquae*)	FL	612	19.5% decrease in the N_2 fixation rate in 2 h	Bastian & Toetz, 1985
A. flos-aquae	FL	1 089	65% inhibition in cell growth in 14 d	Bastian & Toetz, 1982
Chara sp.	FL	20 300	EC_{50} for production in 21 d pre-emergent exposure	Finger *et al*., 1985
Chlamydomonas angulosa	ANTH	42	3 h-EC_{50} for photosynthesis	Hutchinson *et al*., 1980
Chlorella vulgaris	ANTH	42	3 h-EC_{50} for photosynthesis	Hutchinson *et al*., 1980
S. copricornutum	ANTH	17 800	EC_{50} for decreased cell growth in 4 - 7 d	Cody *et al*., 1984

Chlamydomonas angulosa	PH	890	EC_{50} for photosynthesis in 3 h exposure	Hutchinson *et al*., 1980
Blue-green alga (*A. flos-aquae*)	PH	134	15-40% decrease in the N_2 fixation rate in 2 h	Bastian & Toetz, 1985
Blue-green alga (*A. flos-aquae*)	FLAN	434	20-28% decrease in the N_2 fixation rate in 2 h	Bastian & Toetz, 1985
A. flos-aquae	FLAN	38	38% inhibition in growth in 14 d	Bastian & Toetz, 1982
A. flos-aquae	FLAN	417	complete inhibition of cell growth in 14 d	Bastian & Toetz, 1982

TABLE 17 (Continued)

Sublethal and Chronic Toxicity of PAHs to Freshwater Algae and Plants

Organism	***PAH***	***Conc (µg/L)***	***Effects***	***References***
Blue-green alga (*A. flos-aquae*)	BAN	29.9	29% decrease in the rate of N_2 fixation in 2 h	Bastian & Toetz, 1985
A. flos-aquae	BAN	5 and 29	48% reduction in cell growth in 14 days	Bastian & Toetz, 1982
A. flos-aquae	BAN	18	inhibited growth by 16%	Bastian & Toetz, 1982
A. flos-aquae	BAN	29	inhibited growth for 14 days	Bastian & Toetz, 1982
Green alga (*S. capricornutum*)	B[a] ANTH	1 830	30% reduction in algal growth	Schoeny *et al*., 1988
Green alga (*S. capricornutum*)	B[a] ANTH	2.3-22 800	EC_{50} for decreased cell growth in 4 - 7 d exposure	Cody *et al*., 1984
Blue-green alga (*A. flos-aquae*)	PY	159	no decrease in the rate of N_2 fixation in 2 h	Bastian & Toetz, 1985
Chlamydomonas angulosa	PY	202	EC_{50} for photosynthesis in 3 h exposure	Hutchinson *et al*., 1980

Blue-green alga (*A. flos-aquae*)	CH	13.9	17% decrease in the rate of N_2 fixation in 2 h	Bastian & Toetz, 1985
A. flos-aquae	CH	1.9	35% reduction in cell growth in 14 days	Bastian & Toetz, 1982
Green alga (*S. capricornutum*)	B(a)P	1.5	EC_{50} for growth	Schoeny *et al*., 1988
Green alga (*S. capricornutum*)	B(a)P	25	EC_{50} for decreased cell growth in 4 - 7 d exposure	Cody *et al*., 1984

TABLE 18

Sublethal and Chronic Toxicity of PAHs to Freshwater Animals

Organism	***PAH***	***Conc.* (µg/L)**	***Effect***	***References***
Cladoceran (*D. magna*)	NA	>5 000	decreased motility, sluggish behavior; decreased haemoglobin concentration	Crider *et al*., 1982
Cladoceran (*D. pulex*)	NA	330-680	longer lifespan; greater or equal number of live young than controls	Geiger and Buikema Jr., 1982
Prawn (*M. kistnensis*)	NA	595.7	decreased protein levels, increased amino acid concentration, and amino acid enzyme activity	Sarojini *et al* ., 1987
Fathead minnow (*P. promelas*)	NA	>850	reduced egg hatchability; reduced fry length & weight	DeGraeve *et al*., 1982
P. promelas	NA	>4 380	100% mortality	DeGraeve *et al*., 1982
Coho salmon (*O. kisutch*)	NA	400-700	less aggressive feeding behavior; reduced rate of growth	Moles *et al*., 1981
Rainbow trout (*O. mykiss*)	NA	8	97% hatchability at embryo-larval stages	Black *et al*., 1983

O. mykiss	NA	15	91% hatchability at embryo-larval stages	Black *et al*., 1983
O. mykiss	NA	46	85% hatchability at embryo-larval stages	Black *et al*., 1983
O. mykiss	NA	230	35% hatchability at embryo-larval stages; gross anomalies in 7% of exposed fish	Black *et al*., 1983
Largemouth bass (*M. salmoides*)	NA	239	gross anomalies in 6% of exposed fish vs 1% in fish exposed to 28 µg NA/L	Black *et al*., 1983
Midges (*C. riparius*)	FL	600	significant reduction in larval midges	Finger *et al*., 1985
Daphnia magna	FL	125	significant reduction in reproduction in 14 days	Finger *et al*., 1985
Bluegill (*L. macrochirus*)	FL	500	12% mortality in 30 days; 65% reduction in growth	Finger *et al*., 1985
L. macrochirus	FL	250	25% reduction in growth	Finger *et al*., 1985
Cladoceran (*D. magna*)	ACR	400	NOEL: effect: number of broods and number of young/brood	Parkhurst *et al*., 1981
Cladoceran (*D. magna*)	ACR	800	LOEL: effect: number of broods and number of young/brood	Parkhurst *et al*., 1981
D. magna	ACR	400	NOEL: effect: number of young/brood	Blaylock *et al*., 1985
Rainbow trout (*O. mykiss*)	ACR	410	74% hatchability and gross anomalies in 21% of fish at embryo-larval stages	Black *et al*., 1983
O. mykiss	ACR	98	92% hatchability and gross anomalies in 2% of fish at embryo-larval stages	Black *et al*., 1983

TABLE 18 (Continued)

Sublethal and Chronic Toxicity of PAHs to Freshwater Animals

Organism	***PAH***	***Conc.* (µg/L)**	***Effect***	***References***
O. mykiss	ACR	12	99% hatchability in fish at embryo-larval stages	Black *et al*., 1983
Rainbow trout (*O. mykiss*)	PH	4	95% hatchability at embryo-larval stage; gross anomalies in 1% of exposed fish	Black *et al*, 1983
O. mykiss	PH	6	84% hatchability at embryo-larval stage; gross anomalies in 1% of exposed fish	Black *et al*, 1983
O. mykiss	PH	38	44% hatchability at embryo-larval stage; gross anomalies in 6% of exposed fish	Black *et al*, 1983
O. mykiss	PH	85	14% hatchability at embryo-larval stage; gross anomalies in 43% of exposed fish	Black *et al*, 1983
Bluegill (*L. macrochirus*)	B[a] ANTH	1 000	87% mortality in 6 months	Brown *et al*., 1975
Rainbow trout (*O. mykiss*)	B[a]P	0.8 - 3.0 + (Sand= 1-500 µg/g)	gross anomalies in 5.3 to 14.3% of exposed fish vs 2.6% in control fish; anomalies observed: immaturity, kyphosis, cyclopia, microphthalmia, anophthalmia, lack of retinal pigment, reduced yolk sac, albinism	Hannah *et al*., 1982
O. mykiss	B[a]P	0.08 - 30 + (Sand= 1-500 µg/g)	microphthalmia most prevalent anomaly; 6.8% & 17.1% at 0.2 µg/L and 0.3 µg/L; respectively. Other anomalies: skeletal	Hose *et al*., 1984

			deformities (Cranial, vertebral, cartilage)	

6.1.3 Photo-induced effects

Several PAHs, accumulated by aquatic organisms during exposure, have been shown to be severely toxic when the contaminated organisms were exposed to sunlight or ultraviolet radiation (Table 19). For instance, Bowling *et al*. (1983) found that 12.7 µg/L anthracene was fatal to bluegill sunfish (*Lepomis macrochirus*) in 48 hours in an outdoor channel in bright sunlight. No mortality was noted in fish exposed to the PAH in the shaded area of the channel. But, when shading was removed after day 4 (when anthracene concentration in water had dropped to zero and fish were allowed to depurate for 24 hours), all fish previously in the shaded area died within 24 hours. It was concluded that direct sunlight exposure of anthracene-contaminated fish, and not the toxic anthracene photoproducts in the water, was responsible for the mortality of the bluegill.

Photo-induced toxicity of PAHs due to ultraviolet (UV) radiation was subsequently studied by other investigators (Oris and Giesy, 1985, 1987; Newsted and Giesy, 1987). For the purpose of photochemical considerations, UV light is divided into three bands of varying wavelengths: UV-A (390-315 nm), UV-B (315-285 nm), UV-C (285 nm and lower). Although much of the incident solar ultraviolet radiation (SUVR) is filtered out by the atmosphere, some SUVR of longer wavelengths (290-400 nm) passes through. Several PAHs have shown absorption maxima in the 290-400 nm wavelength range. Previous to the discovery of the photo-induced toxicity reactions in contaminated aquatic organisms, most of the laboratory studies with PAHs were conducted in conventional cool fluorescent lighting to avoid photo-oxidation of the compounds; PAHs absorb little radiation in the visible band.

Bearing in mind that LC_{50} increases as the period of exposure decreases, a comparison of the data in Tables 16 and 19 suggests that photoactivation of PAHs was responsible for the observed increase in acute toxicity seen in invertebrates and fish. Also, higher molecular weight PAHs (e.g., B[a]P), which previously were not considered to be acutely toxic to fish because of their low aqueous solubility, could cause an acute toxic reaction if photoactivation occurred.

The phototoxicity of a PAH is a function of several factors: (a) PAH concentration in tissue, (b) length of exposure to and absorption of SUVR by the organism, (c) the efficiency of conversion of ground-state molecules to the excited triplet state, and (d) the probability of the excited intermediate reacting with a target molecule (Newsted and Giesy, 1987).

TABLE 19

Photo-Induced Toxicity of PAHs to Freshwater Animals

Organism	***PAH***	***Conc* (µg/L)**	***Effects/Comments***	***References***
Cladoceran (*D. magna*)	ACR	440	50% mortality in 0.9 h in UV light	Newsted and Giesy, 1987
Fathead minnow (*P. promelas*)	ACR	525	50% mortality in 4.3 h; UV intensity: 100 µW/cm^2 (UV-A), 20 µW/cm^2 (UV-B)	Oris and Giesy, 1987
Cladoceran (*D. magna*)	ANTH	15	50% mortality in 4.98 h in UV light	Newsted and Giesy, 1987
D. magna	ANTH	20	LC_{50} in 1 h irradiation with UV light at 1300 µW/cm^2	Kagan *et al*., 1985
Mosquito larvae (*A. aegypti*)	ANTH	26.8	24-h LC_{50} at the intermediate light intensity of intensity (150 µW/cm^2 UV-B	Oris *et al*., 1984
Dipteran (*A. aegypti*)	ANTH	150	LC_{50} in 1 h irradiation with UV light at 1300 µW/cm^2	Kagan *et al*., 1985
Bluegill (*l.. macrochirus*)	ANTH	12.7	0% mortality in shaded (to sunlight) portion of an outdoor channel & up to 100% in the unshaded portion	Bowling *et al*., 1983
Bluegill (*l.. macrochirus*)	ANTH	11.9	96-h LC_{50}: at a solar UV-B intensity (170 µW/cm^2) equivlent to a depth of 0.6 m in a typical eutrophic north-temperate lake	Oris *et al*., 1984
Bluegill (*l.. macrochirus*)	ANTH	5 (continu-ous light) 46 (6 h light: 18 h dark)	96-h LC_{50}: based on exposure to solar UV-A radiation (365±36 nm) with water surface intensity of 100 µW/cm^2 and intermittent light-dark regime	Oris and Giesy, 1986

Bluegill (*I.. macrochirus*)	ANTH	26.8	36.5-h LC_{50}: based on continuous exposure to solar UV-B radiation (310±34 nm) with water surface intensity of 14.8 to 170 µW/cm^2	Oris and Giesy, 1985
Fathead minnow (*P. promelas*)	ANTH	5.4	50% mortality in 15.75 h; UV intensity: 95 µW/cm^2 (UV-A), 20 µW/cm^2 (UV-B)	Oris and Giesy, 1987
Fathead minnow (*P. promelas*)	ANTH	360	24-h LC_{50}; photosensitization period=0.5 h	Kagan *et al*., 1985
Cladoceran (*D. magna*)	FLAN	9	50% mortality in 10.8 h in UV light	Newsted and Giesy, 1987
D. magna	FLAN	4	LC_{50} in 1 h irradiation with UV light at 1300 µW/cm^2	Kagan *et al*., 1985
Dipteran (*A. aegypti*)	FLAN	12	LC_{50} in 1 h irradiation with UV light at 1300 µW/cm^2	Kagan *et al*., 1985
Fathead minnow (*P. promelas*)	FLAN	200	24-h LC_{50}; photosensitization period=0.5 h	Kagan *et al*., 1985
Cladoceran (*D. magna*)	1H-B[a] FL	50	50% mortality in 22.95 h in UV light	Newsted and Giesy, 1987
Cladoceran (*D. magna*)	1H-B[b] FL	2	50% mortality in 22.4 h in UV light	Newsted and Giesy, 1987
Fathead minnow (*P. promelas*)	Benza-throne	50	50% mortality in 0.83 h; UV intensity: 100 µW/cm^2 (UV-A), 20 µW/cm^2 (UV-B)	Oris and Giesy, 1987
Cladoceran (*D. magna*)	PY	6	50% mortality in 3.47 h in UV light	Newsted and Giesy, 1987
D. magna	PY	4	LC_{50} in 1 h irradiation with UV light at 1300 µW/cm^2	Kagan *et al*., 1985
Dipteran (*A. aegypti*)	PY	20	LC_{50} in 1 h irradiation with UV light at 1300 µW/cm^2	Kagan *et al*., 1985

Fathead minnow (*P. promelas*)	PY	26	50% mortality in 3.2 h; UV intensity: 100 µW/cm^2 (UV-A), 20 µW/cm^2 (UV-B)	Oris and Giesy, 1987
Fathead minnow (*P. promelas*)	PY	220	24-h LC_{50}; photosensitization period= 0.5 h	Kagan *et al*., 1985
Cladoceran (*D. magna*)	B[a] ANTH	2	50% mortality in 12.51 h in UV light	Newsted and Giesy, 1987
Cladoceran (*D. magna*)	B[b] ANTH	1	50% mortality in 16.43 h in UV light	Newsted and Giesy, 1987
Cladoceran (*D. magna*)	CH	2	50% mortality in 23.98 h in UV light	Newsted and Giesy, 1987
Cladoceran (*D. magna*)	1H-B[k] FLAN	1	50% mortality in 13 h in UV light	Newsted and Giesy, 1987
Cladoceran (*D. magna*)	PERY	1	50% mortality in 18.33 h in UV light	Newsted and Giesy, 1987
Cladoceran (*D. magna*)	B[a]P	2	50% mortality in 4.44 h in UV light	Newsted and Giesy, 1987
Fathead minnow (*P. promelas*)	B[a]P	5.6	50% mortality in 40.05 h; UV intensity: 100 µW/cm^2 (UV-A), 20 µW/cm^2 (UV-B)	Oris and Giesy, 1987
Cladoceran (*D. magna*)	B[e]P	1	50% mortality in 15.26 h in UV light	Newsted and Giesy, 1987
Cladoceran (*D. magna*)	D[ah] AN	4	50% mortality in 3.08 h in UV light	Newsted and Giesy, 1987
Cladoceran (*D. magna*)	B[ghi] PERY	0.2	50% mortality in 13.82 h in UV light	Newsted and Giesy, 1987

Newsted and Giesy (1987) investigated several descriptors (e.g., lowest energy triplet state, phosphorescence lifetime, octanol/water partition coefficient, etc.) to describe photo-induced toxicity of PAHs to *Daphnia magna.* The phototoxicity of PAHs was highly correlated with the lowest energy triplet state[4] (LETS, kJ/mol). It was also found that compounds with long phosphorescence lifetimes[5] (PLT > 3.5 s) were not toxic. Newsted and Giesy used these relationships to classify PAHs into toxic categories of very toxic (LT_{50}[6] < 900 min), moderately toxic (900 < LT_{50} < 9999 min), and non-toxic. Working with the larvae of fathead minnow (*Pimephales promelas*), Oris and Giesy (1987) used a similar technique to separate toxic (i.e., causing phototoxic reaction within 96 h) and non-toxic (non-phototoxic in 96 h) PAHs. The results were similar in most of the cases (Table 20). Although promising, these modelling techniques need further improvement to become useful as a tool in the environmental regulation of numerous PAHs which have not been tested yet.

While modelling photo-induced acute (96-h LC_{50}) and chronic toxic effects in bluegill sunfish (*Lepomis macrochirus*) exposed to anthracene, Oris and Giesy (1986) found that both acute and chronic toxicities were dependent on the length of exposure to solar ultraviolet radiation. As the daily exposure to SUVR was increased, the threshold concentration for predicted effects decreased. Furthermore, the absolute difference between acute and chronic threshold concentrations decreased as the daily exposure to SUVR increased (e.g., at daily exposure of 5 h, the predicted acute concentration = 55 µg/L and chronic concentration = 17 µg/L; and at daily exposure of 20 h, the acute concentration = 7.8 µg/L and chronic concentration = 2.2 µg/L).

The evidence for the photo-induced toxicity of PAHs is recent and its significance in natural aquatic systems is yet to be understood. Aquatic organisms in deep and turbid waters and shaded areas may not be affected by this phenomenon. However, juveniles of most fish are found in the shallow areas of the littoral zone and are subject to photo-induced toxicity of PAHs. Landrum *et al*. (1986) calculated that SUVR of sufficient intensity, which may cause 50% immobilization in the cladoceran (*Daphnia pulex*) exposed to 1.2 µg/L anthracene over a 14-h

TABLE 20

Phototoxicity classification of selected PAHs based on lethal response of Daphnia magna (Newsted and Giesy, 1987) and Pimephales promelas (Oris and Giesy, 1987)

PAH	***Phototoxic Category***	
	*Newsted and Giesy (1987)**	*Oris and Giesy (1987)***

Carbazole	Non-toxic+	Non-toxic++
Fluorene	Non-toxic+	Non-toxic++
Anthracene	Very toxic+	Toxic+
Phenanthrene	Non-toxic+	Non-toxic+
Acridine	Very toxic+	Toxic+
Phenazine	Very toxic++	Toxic++
Fluoranthene	Very toxic+	Non-toxic++
1H-Benzo[a]fluorene	Moderately toxic+	Non-toxic++
1H-Benzo[b]fluorene	Moderately toxic+	Non-toxic++
Pyrene	Very toxic+	Toxic+
Benz[a]anthracene	Very toxic+	Toxic+
Benz[b]anthracene	Very toxic+	Toxic++
Chrysene	Moderately toxic+	Non-toxic++
Triphenylene	Non-toxic+	Not determined
Benzo[k]fluoranthene	Very toxic+	Non-toxic++
Benz[a]acridine	Very toxic++	Toxic++
Benz[c]acridine	Very toxic++	Toxic++
Benzathrone	Very toxic+	Toxic+
Benzo[a]pyrene	Very toxic+	Toxic+
Benzo[e]pyrene	Very toxic+	Non-toxic+
Perylene	Very toxic+	Non-toxic+
Dibenz[a,h]acridine	Very toxic++	Non-toxic++
Dibenz[a,h]anthracene	Very toxic+	Non-toxic+
Dibenz[a,j]anthracene	Very toxic++	Non-toxic++
Benzo[b]chrysene	Very toxic++	Toxic++
Dibenz[a,c]phenazine	Very toxic++	Toxic++
Benzo[b]triphenylene	Very toxic++	Non-toxic++
Benzo[g,h,i]perylene	Very toxic+	Non-toxic+

Coronene	Non-toxic++	Non-toxic++

+ Results based on bioassays; ++ Predicted results from toxicity modelling techniques;
*Very toxic = LT_{50} < 900 min, and Moderately toxic = 900 < LT_{50} <9999 min;
**Toxic = LT_{50} < 96 h, and Non-toxic = LT_{50} > 96 h

daylight cycle, could penetrate to a depth of 7 metres in a lake. Since photo-induced toxicity of PAHs is a function of several factors which can not be duplicated in a laboratory environment, sophisticated experimentation and evaluation techniques are needed to determine the extent to which phototoxicity may proceed in natural aquatic environments.

6.2 Marine water

6.2.1 Lethal and acute effects

Most of the literature on acute and lethal toxic effects (as EC_{50} for aquatic plants and LC_{50} for aquatic animals) in estuarine and marine environments is related to the lower molecular weight PAHs (LPAH), containing 3 or less benzene rings in their structure (Table 21). These compounds are relatively more soluble in water than the higher molecular weight PAHs (HPAH); at saturation, their concentrations in water (Table 1) can exceed the LC_{50}s shown in Table 21.

The PAH concentrations causing lethal effects in marine organisms vary widely. The lowest 96-h LC_{50} of 40 µg/L was recorded for juvenile mysid shrimp (*Mysidopsis bahia*) exposed to fluoranthene (USEPA, 1978). Since this test with shrimp was conducted in a static (as opposed to a flow-through) environment and the PAH concentration was not measured during the experiment, the results of this test were considered to be of secondary[7] importance.

Several trends were established from Table 21: (a) The toxic (LC_{50}) PAH concentration for an organism decreased with longer exposure periods; for instance, the 96-h LC_{50}s (1 900 µg/L for 1-MNA, and 1 300 µg/L for 2-MNA) for Dungeness crab (*Cancer magister*) exposed to two methylnaphthalenes were about four times lower than their 48-h LC_{50}s (8 200 µg/L for 1-MNA, and 5 000 µg/L for 2-MNA) (Caldwell *et al*., 1977); (b) the degree and position of methylation affected PAH toxicity; e.g., dimethylnaphthalene (96-h LC_{50} = 600 d-MNA/L) was more toxic to Dungeness crab than a methylnaphthalene (96-h LC_{50} = 1 300 2-MNA/L),

TABLE 21

Lethal Toxicity of PAHs to Marine and Estuarine Aquatic Life

Organism	***PAH***	***EC_{50}/LC_{50}* (µg/L)**	***Duration* (hours)**	***System*[1]**	***References***
Copepod (*E. affinis*)	NA	3 800	24	S,M	Ott *et al*., 1978:
Amphipod (*Parhyale*)	NA	>5 000	24	S,U	Lee & Nichol, 1978a
Amphipod (*E. pectenicrus*)	NA	2 680	96		Lee & Nichol, 1978b
Polychaete (*N. arenaceodentata*)	NA	3 800	96	S,U	Rossi and Neff, 1978
Pacific Oyster (*C. gigas*)	NA	199 000	96	S,U	LeGore, 1974
Brown Shrimp (*P. aztecus*)	NA	2 500	24	S,U	Anderson *et al*., 1974
Brown Shrimp (*P. aztecus*)	NA	2 500	96	S,U	Tatem *et al.*, 1978
Grass Shrimp (*P. pugio*)	NA	2 350	96	S,U	Tatem, 1976; Tatem *et al*., 1978
Dungeness Crab (*C. magister*)	NA	>2 000	96	FT,M	Caldwell *et al*., 1977
Crab (*S. serrata*)	NA	17 000	96		Kulkarni and Masurekar, 1984
Sheepshead Minnow (*C. variegatus*)	NA	2 400	24	S,U	Anderson *et al*., 1974
Pink Salmon (*O. gorbuscha*)	NA	920	24		Thomas and Rice, 1978
O. gorbuscha	NA	1 200	96	FT,M	Moles and Rice, 1983
O. gorbuscha	NA	1 200	960	FT,M	Moles and Rice, 1983

Dungeness Crab (*C. magister*)	1-MNA	8 200	48	FT,M	Caldwell *et al*., 1977
C. magister	1-MNA	1 900	96	FT,M	Caldwell *et al*., 1977
Sheepshead minnow (*C. variegatus*)	1-MNA	3 400	24	S,U	Anderson *et al*., 1974
Copepod (*E. affinis*)	2-MNA	1 300-1 500	24	S,M	Lee & Nichol, 1978a, b; Ott *et al*., 1978
Grass Shrimp (*P. pugio*)	2-MNA	1 100	96	S,U	Neff *et al*., 1976a; Tatem *et al*., 1978
Brown Shrimp (*P. aztecus*)	2-MNA	700	24	S,U	Anderson *et al*., 1974
Brown Shrimp (*P. aztecus*)	2-MNA	600	96	S,U	Tatem *et al*., 1978
Dungeness Crab (*C. magister*)	2-MNA	5 000	48	FT,M	Caldwell *et al*., 1977
C. magister	2-MNA	1 300	96	FT, M	Caldwell *et al*., 1977
Sheepshead minnow (*C. variegatus*)	2-MNA	2 000	24	S,U	Anderson *et al*., 1974
Copepod (*E. affinis*)	d-MNA	850	24	S,M	Ott *et al*., 1978
Polychaete (*N. arenaceodentata*)	d-MNA	2 600	96	S,U	Neff *et al*., 1976a; Rossi and Neff, 1978
Grass Shrimp (*P. pugio*)	d-MNA	700	96	S,U	Neff *et al*., 1976a; Tatem *et al*., 1978
Brown Shrimp (*P. aztecus*)	d-MNA	80	24	S,U	Anderson *et al*., 1974
Brown Shrimp (*P. aztecus*)	d-MNA	80	96	S,U	Tatem *et al*., 1978

Dungeness Crab (*C. magister*)	d-MNA	3 100	48	FT,M	Caldwell *et al*., 1977
C. magister	d-MNA	600	96	FT,M	Caldwell *et al*., 1977
Sheepshead Minnow (*C. variegatus*)	d-MNA	5 100	24	S,U	Anderson *et al*., 1974
Polychaete (*N. arenaceodentata*)	t-MNA	2 000	96	S,U	Rossi and Neff, 1978
Copepod (*E. affinis*)	t-MNA	320	24	S,M	Ott *et al*., 1978
Alga (*S. costatum*)	ANA	500	96	S,U	USEPA, 1978
Mysid shrimp (*M. bahia*)	ANA	970	96	S,U	USEPA, 1978
Sheepshead minnow (*C. variegatus*)	ANA	2 230	96	S,U	USEPA, 1978
C. variegatus	ANA	3 700	24	S,U	Heitmuller *et al*., 1981
C. variegatus	ANA	2 300	48	S,U	Heitmuller *et al*., 1981
C. variegatus	ANA	2 200	96	S,U	Heitmuller *et al*., 1981
Amphipod (*G. pseudoliminaeus*)	FL	600	96	S,M	Finger *et al*., 1985
Polychaete (*N. arenaceodentata*)	FL	1 000	96	S,U	Rossi and Neff, 1978
Grass Shrimp (*P. pugio*)	FL	320	96		Wofford and Neff, 1978
Sheepshead minnow (*C. variegatus*)	FL	1 680	96		Wofford and Neff, 1978

Polychaete (*N. arenaceodentata*)	PH	600	96	S,U	Rossi and Neff, 1978
Grass Shrimp (*P. pugio*)	PH	370	24		Young, 1977
Polychaete (*N. arenaceodentata*)	1-MPH	300	96	S,U	Rossi and Neff, 1978
Alga (*S. costatum*)	FLAN	45 000	96	S,U	USEPA, 1978
Polychaete (*N. arenaceodentata*)	FLAN	500	96	S,U	Neff *et al*., 1976a; Rossi and Neff, 1978
Mysid shrimp (*M. bahia*)	FLAN	40	96	S,U	USEPA, 1978
Sheepshead minnow (*C. variegatus*)	FLAN	>560 000	96	S,U	USEPA, 1978
C. variegatus	FLAN	>560 000	960	S,U	Heitmuller *et al*., 1981
Polychaete (*N. arenaceodentata*)	CH	>1 000	96	S,U	Rossi and Neff, 1978
Polychaete (*N. arenaceodentata*)	B[a]P	>1 000	96	S,U	Rossi and Neff, 1978
Polychaete (*N. arenaceodentata*)	D[a,h]AN	>1 000	96	S,U	Rossi and Neff, 1978

1 S= static; U= unmeasured; FT= flow through; M= measured

whereas, between the two methylnaphthalenes, 2-MNA was more toxic than 1-MNA (96-h LC_{50} = 1 900 1-MNA/L) (Caldwell *et al*., 1977). With the addition of each methyl group, Ott *et al*. (1978) found that the 24-h LC_{50} for the copepod *Eurytemora affinis* exposed to naphthalene was reduced by approximately one-half; (c) the acute and lethal reactions of HPAH (e.g., benzo[a]pyrene, chrysene, and dibenz[a,h]anthracene) were limited to concentrations much above their solubilities in seawater.

6.2.2 Sublethal and chronic effects

Chronic toxicities of various PAHs are shown in Table 22. The lack of data and a wide variety of end points chosen by the investigators during experimentation prevent a comparison of chronic toxicity levels among PAHs.

The most sensitive chronic effects of naphthalene were observed by DiMichele and Taylor (1978) while studying histopathological and physiological responses in mummichog (*Fundulus heteroclitus*). These investigators found gill hyperplasia in 80% of the fish after a 15-d exposure to 2 µg/L naphthalene; only 30% of the control fish showed the effect. All of the fish exposed to 20 µg NA/L demonstrated necrosis of tastebuds, a change not observed in the control fish.

Ott *et al*. (1978) found that lethal toxicity (24-h LC_{50}) of naphthalene and its alkylated derivatives was determined by the degree of methylation (see Section 6.2.1). This trend was not obvious from low level chronic studies due to the lack of appropriate data (e.g., organism tested, experimental end points, experimental test conditions, etc. were different in different studies). Ott *et al*. found that chronic exposure of copepod *E. affinis* to various naphthalenes (2-MNA, d-MNA, and t-MNA) at a concentration of about 10 µg/L in sea water for the duration of their adult life (maximum 29 days) resulted in significant reductions in the organisms' length of life, total numbers of nauplii produced, and mean brood size. Exposure to all naphthalenes at 10 µg/L resulted in reduced rates of egg production which were, on average, about 50% of those of control animals.

Miller *et al*. (1982) found that the concentration of 1 µg/L chrysene in water increases incidence of molts in pink shrimp (*P. duorarum*) after 28 days.

TABLE 22

Sublethal and Chronic Toxicity of PAHs to Marine Animals

Organism	***PAH***	***Conc* (µg/L)**	***Effects***	***Reference***
Amphipod (*Parhyale*)	NA	10 000	toxic effects on survivors after 1 wk; complete recovery of survivors after 2wk	Lee and Nichol, 1978b
Crab (*S. serrata*)	NA	2 500	elevated amino acid enzymatic activity in blood serum	Kulkarni and Masurekar, 1984

Crab (*S. serrata*)	NA	5 000	elevated amino acid enzymatic activity in blood serum	Kulkarni and Masurekar, 1984
Fiddler crab (*U. pugilator*)	NA	8 000	inhibition of circadian melanin distribution	Staub and Fingerman, 1984
Mummichog (*F. heteroclitus*)	NA	4 000	at 21 C, survival rate 90% at 2-15 salinity, and 50% at 23-33 salinity	Levitan and Taylor, 1979
F. heteroclitus	NA	6 000	at 16 C, survival rate > 95% at 8 & 15 salinities, ~75% at 2 salinity, and 60% at 23 & 33 salinities	Levitan and Taylor, 1979
F. heteroclitus	NA	2	gill hyperplasia during 15-d exposure	DiMichele and Taylor, 1978
F. heteroclitus	NA	20	tastebud necrosis	DiMichele and Taylor, 1978
S. minnow (*C. variegatus*)	NA	620	embryo-larvae test	DeGraeve *et al*., 1982
Pink salmon (*O. gorbuscha*)	NA	380-560	less aggressive feeding behavior; 20% red-uction in food consumption in 40 d	Moles and Rice, 1983
O. gorbuscha	NA	800	little or no feeding initially; 10% feeding at the end of 40 days	Moles and Rice, 1983
O. gorbuscha	NA	380-800	decreased rate of growth; dulled motor response; increased metabolic rate	Moles and Rice, 1983
Copepod (*E. affinis*)	2-MNA	15.03	decreased lifespan; decreased fecundity and reproductive success of females	Ott *et al*., 1978
Cod (*Gadus morhua*)	2-MNA	300	25% abnormal eggs after 4-d exposure	Stene & Lonning, 1984

Gadus morhua	d-MNA	8.16	decreased lifespan; decreased fecundity and reproductive success of females	Ott *et al*., 1978
Gadus morhua	t-MNA	9.27	decreased lifespan; decreased fecundity and reproductive success of females	Ott *et al*., 1978
S. minnow (*C. variegatus*)	ANA	710	geometric mean of effect and no-effect concentrations	USEPA, 1978
Mud crab (*R. harrisii*)	PH	37.5-75	respiration rate unaffected at 37.5 µg/L, but increased with conc. at 75 µg/L	Laughlin Jr. and Neff, 1980
Mysid shrimp (*M. bahia*)	FLAN	16	life cycle chronic effects (specific effect not reported)	USEPA, 1978:
Pink shrimp (*P. duorarum*)	CH	1	increased incidence of molts after 28 days of exposure	Miller *et al*., 1982
P. duorarum	CH	5	increased incidence of molts after 28 days of exposure	Miller *et al*., 1982
English Sole (*P. vetulus*)	B[a]P	1.8-2.4	0.71% abnormalities in embryo/larvae	Hose *et al*., 1982
Sand sole (*P. melanostichus*)	B[a]P	0.10	av. hatching success=28.1% in treated eggs compared to 57.0% in control	Hose *et al*., 1982

Among PAHs studied, B[a]P was found to be the most toxic (Table 22). Five percent of sand sole (*Psettichthys melanostichus*) eggs exposed to 0.1 µg B[a]P/L in water (as compared to 0% in control fish) showed gross anomalies such as overgrowth of tissue originating from the somatic musculature, and arrested development (Hose *et al*., 1982). Also, the hatching success of eggs exposed to 0.1 µg B[a]P/L (average = 28.1%; range = 7.0% to 67.6%) was significantly lower than that of controls (average = 57%; range = 21.6% to 89.6%).

6.3 Mutagenicity, carcinogenicity, and tumor induction

Several PAHs, especially those containing 4 to 6 aromatic rings in their structure, have been shown to be mutagenic, carcinogenic, and inducers of tumors in mammals exposed to high doses of the contaminants in the laboratory. Studies directly linking

PAHs to these effects in fish are not only few in number but also have used excessively high exposure levels of PAHs.

Hendricks *et al*. (1985) exposed rainbow trout (*Salmo gairdneri* now classified as *Oncorhyncus mykiss*) for 6 to 18 months to a diet containing 1000 µg B[a]P/g dry weight. Hepatic neoplasms were observed in 25% of the trout that were fed the B[a]P diet for 18 months; the fish on the control diet did not show the effect at all. Of the affected (i.e., 25%) population, 21% had livers with at least one hepatocellular carcinoma. In the same experiment, Hendricks and coworkers also found that at the end of 18 months the body weight was lower in the B[a]P-fed fish than the control fish; however, the number of mortalities was higher in the control population (5% as opposed to 3% in treated fish) which suggested that B[a]P was not acutely toxic. Similar results were obtained by Hendricks *et al*. (1985) in 10-month old trout (weighing 45-55 g) intraperitoneally injected with B[a]P (dissolved in 0.4 mL propylene glycol) at the rate of 1 mg B[a]P/month for 12 months.

Krahn *et al*. (1986) examined hepatic lesions in English sole (*Parophrys vetulus*) caught from 10 sites in Puget Sound, Washington. A strong positive correlation was found between B[a]P metabolite equivalents (67-2100 µg/kg wet weight) in bile and the prevalence of hepatic lesions (6.2-90%). However, the concentration of metabolites in the bile was highly variable within locations and was not correlated with the PAH (e.g., naphthalene, phenanthrene, and B[a]P) concentration in the sediment. In the same area, Malins *et al*. (1985a, 1985b) found elevated hepatic neoplasms in association with sediments which were heavily contaminated with creosote and also contained PAHs (e.g., acridine and carbazole).

Repeated short-term (five successive 6-h periods separated by weekly intervals) aqueous exposure of viviparous[8] fish (*Poeciliopsis lucida* and *P. monacha*) to 5 000 µg/L 7,12-dimethylbenzo[a]anthracene (DMB[a]AN) induced hepatocellular neoplasms in 25 of 60 fish in 6 to 7 months; tumors were not found in any of the controls (0/59) (Shultz and Shultz, 1982). However, short-term exposures to 250 µg/L 7,12-DMB[a]AN failed to induce neoplasms. Since these results were obtained using concentrations much above the aqueous solubility of the PAH (1.5 µg/L at 25 C - Table 1), they cannot be considered indicative of a natural exposure situation.

In a preliminary work by Hawkins *et al*. (1988), young guppies (*Poecilia latipes*) and medakas (*Oryzias latipes*) were exposed (two 6-h exposures one week apart) to B[a]P and DMB[a]AN concentrations of <5 µg/L (0.45 µm filtrate with no carrier), 30-50 µg/L (carrier-mediated 0.45 µm filtrate), and 150-250 µg/L (carrier-mediated glass fiber filtrate). Preliminary analyses indicated that hepatic neoplasm occurred in both fish exposed to B[a]P and DMB[a]AN. In the guppy, B[a]P-induced hepatic neoplasms (10%

at 24 weeks post initial exposure) were limited to the high exposure group. DMB[a]AN was much more carcinogenic to fish than B[a]P. Medakas, which were affected by all concentrations of DMB[a]AN, also showed numerous non-hepatic lesions at the high DMB[a]AN concentration. The prominence of both hepatic and non-hepatic lesions at high exposure concentrations (which contained particulates) suggested that the insoluble or particulate fraction of PAHs may play an important role in carcinogenesis. Both guppies and medakas are not native to British Columbia; also, PAH concentrations used in these tests are much higher than their aqueous solubilities (Table 1).

Metcalfe and Sonstegard (1984) exposed rainbow trout (*O. mykiss*) embryos at the eyed stage to DMB[a]AN using a micro-injection (0.5 µL) technique. Injections of 0.25 DMB[a]AN/embryo produced grossly visible neoplasms and carcinomas at a frequency ranging from 3.5 to 6.3%. Using a similar technique, Black *et al*. (1985) noted that injections of 10 µg B[a]P/embryo (136.1 µg B[a]P/g) produced a tumor incidence of 8.7% after nine months in rainbow trout, while all control fish appeared healthy.

6.4 Mixed-function oxidases

Mixed-function oxidases (MFOs) are a group of enzymes which are located in hepatic microsomes and play an important role in the biotransformation of PAHs (and other xenobiotics e.g., PCBs, dioxins, and furans) before their excretion from the body. The action of MFOs can either detoxify PAHs or produce more reactive intermediates which are cytotoxic and possibly carcinogenic.

There are several enzyme systems which make up the mixed-function oxidases. They include, for instance, aryl hydrocarbon hydroxylase (AHH), ethoxycoumarin O-deethylation (ECOD), ethoxyresorufin O-deethylation (EROD), etc. Several fish and invertebrate species have been reported to show a mixed-function oxidase activity in response to a PAH exposure.

Gerhart and Carlson (1978) exposed rainbow trout to several PAHs (e.g., 1,2,4-trimethylnaphthalene, phenanthrene, pyrene, fluoranthene, chrysene, and benzo[a]pyrene) in water and through intraperitoneal (ip) injections to screen for MFO activity. Chrysene and B[a]P were found to induce MFO activity in the fish injected with the PAHs. In water, bioaccumulation of B[a]P (average aqueous conc.=0.4 µg/L) resulted in MFO induction in 10 days, whereas bioaccumulation of pyrene (3.9 µg/L) and fluoranthene (3.31 µg/L) did not. Based on these experiments, it was predicted that tissue (excluding liver and gut) concentrations in excess of 0.3 µg/g B[a]P would cause MFO induction in rainbow trout. These investigators did not correlate MFO induction with other effects (e.g., carcinogenic, mutagenic, tumor induction, etc.), but a

comparison of these results with those of Black *et al*. (1985) in section 6.3 suggests that the tissue concentration of 0.3 µg B[a]P/g may induce MFO activity in rainbow trout without producing a tumor.

Walters *et al*. (1979) found naphthalene (200 µg/L), 2-methylnaphthalene (100 µg/L), 2,6-dimethylnaphthalene (100 µg/L), 3-methylcholanthrene (100 µg/L), and benzo[a]pyrene (50 µg/L) increased AHH activity in the marine zooplankton *Calanus helgolandicus* by 191%, 125%, 29%, 146%, and 129%, respectively, over the control population. However, in all cases, the observed mortality in the animals exposed to the PAHs for 7 days was not significantly different from those of the control population.

Recently, Hendricks *et al*. (1985) studied the hepatocarcinogenicity of B[a]P to rainbow trout by dietary exposure (500 and 1 000 µg/g dry weight of diet) and intraperitoneal injection (10-month old fish weighing 45-55 g injected with 1.0 mg B[a]P once a month for 12 months). In 9 weeks, the MFO activity (e.g., EROD) increased significantly in the fish fed both 500 and 1 000 µg/g B[a]P diets over the control population. In the long-term feeding experiments (up to 18 months) with a diet containing 1 000 µg/g B[a]P, these investigators found that the treated population had significantly lower body weight than the control (mortality was similar in both populations). No lesions were found in the fish for the first 6 months, but the incidence of liver neoplasms (basophilic foci+carcinomas) increased to 15% (12%+3%) and 25% (4%+21%) after 12 and 18 months, respectively of feeding B[a]P in the diet. The whole fish B[a]P level at the end of 18 months was estimated to range from 860 to 1 000 µg/g wet weight.

In the intraperitoneal injection experiments, Hendricks *et al*. (1985) found that the incidence of the carcinoma of the liver in rainbow trout increased to 46% (as opposed to 4% in control; based on fish that survived) in 6 months after 12 monthly ip injections of B[a]P. At the end of the experiment, the B[a]P concentration in whole fish, based on the starting weight of the fish at 55 g, was about 218 µg/g. The investigators did not measure the MFO activity in this portion of the study, but mortality of the fish was high (44% in treated fish versus 46% in control), which was attributed to propylene glycol used as the PAH carrier.

6.5 Mixtures of contaminants

In aquatic environments, organisms are exposed to several contaminants at a time. To assess the impact of a PAH on aquatic environments, therefore, interactions between contaminants must be considered. Landrum (1983) found a 50% reduction in the uptake of B[a]P and anthracene by the amphipod *Pontoporeia hoyi* in presence of toluene (expressed as I_{50}9 values). It was noted that I_{50} was a function of several factors, including (a) co-contaminant solubility- I_{50} increases as the co-contaminant

solubility increases, and (b) aqueous solubility of the primary contaminant- the reduction in the uptake of relatively insoluble and hydrophobic B[a]P (I_{50} = 740 µg/L toluene) was more sensitive than anthracene (I_{50} = 2 200 µg/L toluene).

Stein *et al*. (1984, 1987) exposed English sole (*Parophrys vetulus*) to sediments labelled with 3H-benzo[a]pyrene and ^{14}C-Aroclor 1254 (a PCB formulation) either singly or together, and found that the accumulation of B[a]P-derived radioactivity was enhanced in the fish exposed to both contaminants simultaneously relative to exposure to the PAH alone. The formation and accumulation of potentially toxic metabolites of carcinogenic B[a]P in sole liver were also increased by the simultaneous exposure to other contaminants present in a sediment. However, while investigating accumulation of naphthalene, a PCB mixture, and B[a]P by the oyster *Crassostrea virginica*., Fortner and Sick (1985) found several instances where multiple components had antagonistic effects on PAH accumulation.

The literature on interactions between co-contaminants is limited. More work is needed on modelling and quantifying these interactions before they can be applied to real aquatic environmental situations.

6.6 Other modifying factors

Smith *et al*. (as quoted in OMOE, 1990) found that lethal toxicity of waterborne phenanthrene to *Daphnia pulex* increased over a narrow temperature range of 17 °C to 20 °C, but only a marginal effect was noted with 1,3 dimethylnaphthalene. On the other hand, a decrease in temperature increased bioaccumulation of PAHs. This increase in bioaccumulation may be attributed to several factors including decreased rates of depuration and metabolism with decreasing temperature. Higher PAH concentrations were observed in fish and invertebrates at lower temperatures, even though uptake kinetics may be slower (Varanasi *et al., 1981*). In general, the effect of temperature on PAH toxicity and bioaccumulation is minor (Varanasi *et al., 1981*).

Complexation by dissolved organic matter (DOM) reduces the bioavailability of PAHs (Leversee *et al*., 1983; Spacie *et al*., 1983; McCarthy and Jimenez, 1985b; McCarthy *et al*., 1985). Leversee *et al*. (1983) found that the presence of humic acids reduced bioaccumulation of unsubstituted PAHs in proportion to their K_{ow} (B[a]P > anthracene > naphthalene). A reversal in the bioaccumulation trend with humic acid (i.e., higher bioaccumulation at higher humic acid content) was observed for methylcholanthrene, but no explanation was offered.

Humic acids were also shown to increase salting-out of PAHs initiated by increasing salinity. Spacie *et al*. (1983) found that the addition of dissolved humic acids to water

decreased B[a]P accumulation in bluegill sunfish (*Lepomis macrochirus*); however, in the same experiment, the accumulation of anthracene was not affected by the added humic acids.

McCarthy and Jimenez (1985b) and McCarthy *et al*. (1985) investigated the binding and dissociation of several PAHs (B[a]P, benzanthracene, and anthracene) with dissolved humic material (DHM) and PAH uptake and accumulation by bluegill sunfish (*L. macrochirus*) and *Daphnia magna*. A positive logarithmic relationship between Kow and an association constant with dissolved humic material (i.e., Kp) was found. Also, the binding of PAHs with humic acids was completely reversible. The presence of humic acids dramatically reduced the availability of PAHs for uptake by organisms, which lead these investigators to suggest that dissolved organic material has the potential to mitigate effects of PAH in aquatic systems.

Landrum *et al*. (1987) found that the partitioning (expressed as partitioning coefficient Kb) of PAHs binding to dissolved organic carbon was not adequately predicted by Kow; only 46% of the variance in Kb was explained by Kow. Thus, while Kow was an adequate descriptor for PAH binding with dissolved humic material (DHM) of consistent composition, it clearly lacked the capacity for predicting partitioning of PAHs on the basis of dissolved organic carbon (or Kb). Obviously, the composition and complexing properties of the dissolved organic carbon used in the Landrum *et al*. (1987) study were different than those of the standardized DHM used in the McCarthy and coworkers studies. Landrum *et al*. (1987) noted that Kb was more closely related with a sorption coefficient derived through reverse phase separation.

PAHs are also complexed by sediments. However, sediment-bound PAHs may become available to organisms at higher trophic levels through ingestion of benthic organisms living in these sediments. For instance, Landrum and Scavia (1983) found that sediment-associated uptake of anthracene by the amphipod *Hyalella azteca* was slower than aqueous uptake; however, the sediment-associated anthracene accounted for 77% of the steady state body burden in these organisms.

6.7 Bioaccumulation

Aquatic organisms can accumulate PAHs from water, sediment, and food. The literature suggests that PAH uptake by aquatic organisms depends upon several factors: (a) physical and chemical properties of the PAH (e.g., molecular weight, octanol/water partition coefficient, etc.), (b) environmental variables (e.g., suspended matter, dissolved organic matter, bioavailability, temperature, presence of other contaminants, biodegradation, etc.), and (c) biological factors (e.g., PAH metabolism

and depuration rates, feeding characteristics of organisms, fat content of tissue, lifestage, etc.) (McElroy *et al*., 1989).

The octanol/water coefficient (Kow) has been shown to be a good descriptor for accumulation for several PAHs in aquatic organisms (expressed as bioconcentration factor BCF, which is equal to the contaminant concentration in the organism ÷ the contaminant concentration in the aqueous phase) (Southworth *et al*., 1978; Pruell *et al*., 1986). The relationship between Kow and BCF may be modified by the PAH affinity for dissolved organic and solid phase fractions (McCarthy *et al*., 1985). In some cases, the relative availability of PAHs does not appear to be a simple function of their physical and chemical properties. For instance, Varanasi *et al*. (1985) found 4-ring PAHs to be more available than either 3- or 5-ring PAHs from contaminated sediments in Puget Sound, Washington, for accumulation by two species of amphipod (*Rhepoxynius abronius* and *Pandalus platyceros*) and one species of clam (*Macoma nasuta*).

The accumulation of petroleum hydrocarbons by the two populations of the oyster, *Crassostrea virginica*, was positively correlated with their fat content (Stegeman and Teal, 1973). Bioconcentration of PAHs in the fat tissue, however, may be influenced by the lipid composition. Schneider (1982) found that the difference in PCB accumulation in different organs of cod was eliminated when residues were normalized to the neutral lipid or fat (mostly triglycerides) content (rather than extractable lipid content) of the organ. No such data are available for PAHs.

In general, waterborne PAHs are taken up relatively rapidly as compared to sediment-associated PAHs. McElroy (1985) exposed the polychaete, *Nereis virens*, to ^{14}C-benz[a]anthracene introduced directly to the water column or already sorbed to the sediment. The tissue/sediment ratio for *N. virens* and the degree to which accumulated residues were metabolized in a 7-d period, were significantly higher when B[a]ANTH was introduced via the water column. Regarding short-necked clams, *Tapes japonica*, exposed to PAHs added to the water column in a circulating aquarium, or to PAHs in contaminated sediments collected from an urban harbour, Obana *et al*. (1983) observed that the clams reached apparent equilibrium with water concentrations in one day in the first case, while concentrations of most individual PAHs were still increasing in clams exposed to contaminated sediment for 7 days. Investigating the influence of sediment on uptake of PAHs, Landrum and Scavia (1983) estimated that anthracene associated with sediment and pore water contributed 77% of the amphipods' (*Hyalella azteca*) steady-state body burden. Considering reversibility between aqueous phase and sediment-associated PAHs, the differential availability of dissolved versus sorbed PAH should primarily be a kinetic consideration. That is, the particulate PAH reservoir should be the primary source, even though actual uptake may occur via a dissolved pathway.

Aquatic organisms are capable of accumulating PAHs through diet. In cases where uptake from food versus sediment has been compared, the dietary route appears to be more efficient (McElroy, 1985; Corner *et al*., 1976). For instance, in comparing bioaccumulation and metabolism of ^{14}C-anthracene by the omnivorous deposit-feeding polychaete in microcosms where the PAH was introduced either already sorbed to sediments or in a prepared protein-based diet, McElroy (1985) noted that the isotope in the prepared diet was more rapidly metabolized than the isotope that bound to sediments. However, studies comparing direct uptake from solution to that from dietary routes are contradictory. In hard clam larvae (*Mercenaria mercenaria*), Dobroski and Epifanio (1980) found greater efficiency of ^{14}C-benzo[a]pyrene uptake from the water column than from contaminated algae (*Thaslassiosira pseudeonana*), although the contaminated algae contributed significantly to the PAH body burden of the larvae. Lu *et al*. (1977) compared uptake of ^{14}C-benzo[a]pyrene (added to water column) by fish (*Gambusia affinis*), mosquito larvae (*Culex pipiens quinquefasciatus*), and snails (*Physa* sp.) exposed individually and collectively in a model ecosystem. In single species exposures, no bioaccumulation was observed in fish, whereas mosquito larvae and snails attained B[a] P levels (in terms of radioactivity) 40 to 2 000 times that of the water column. When the organisms were exposed together, bioconcentration factors increased dramatically for all groups including fish (up to 1 000 times). On the other hand, in an experimental food chain consisting of diatoms, mussels, and snails exposed to ^{14}C-naphthalene, Clark (1983) found that partitioning from seawater across membranes is a much more important route for PAH accumulation than the trophic transfer. Obviously, as noted from the discussion above, the topic of PAH bioaccumulation needs further exploration.

The bioconcentration factors in aquatic organisms exposed to PAHs in water are shown in Tables 23 and 24.

6.8 Sediment toxicity

The toxicity of PAH adsorbed to sediments is not well studied. It is, however, generally recognized that association with sediments reduces the bioavailability of PAHs.

Swartz *et al*., (1988) calculated 10-d LC_{50}s of 3.68 µg/g (dry weight) phenanthrene and 4.20 µg/g (dry weight) fluoranthene for the marine amphipod *Rhepoxynius abronius* exposed to these PAHs in sediment. However, working with contaminated sediments of Eagle Harbor in Puget Sound, Washington, these investigators found different results (Swartz *et al*., 1989). To assess toxicity, *Rhepoxynius abronius* were exposed to several sediment samples obtained by

TABLE 23

Bioconcentration Factors (BCF) of PAHs in Freshwater Animals

Organism	***PAH***	***Exposure Conc* (µg/L)**	***Duration* (hours)**	***Tissue***	***BCF***	***References***
Cladoceran (*D. pulex*)	NA	1 000	24	whole	118	Southworth *et al*., 1978
Dragonfly nymph (*S. cingulata*)	NA	10	24	whole	1 128	Correa & Coler, 1983
S. cingulata	NA	10	48	whole	1 548	Correa & Coler, 1983
S. cingulata	NA	100	24	whole	154	Correa & Coler, 1983
S. cingulata	NA	100	48	whole	177	Correa & Coler, 1983
Bluegill (*L. macrochirus*)	NA		24	whole	310	McCarthy and Jimenez, 1985a
L. macrochirus	ANA		672		387	USEPA, 1978
Bluegill (*L. macrochirus*)	FL		720		200-1 800	Finger *et al*., 1985
Cladoceran (*D. pulex*)	ANTH	6	24	whole	1 192	Southworth *et al*., 1978
D. magna	ANTH	6	1	whole	200	Herbes, 1976
Cladoceran (*D. pulex*)	ANTH		24		760	Herbes & Risi, 1978
Mayfly (*Hexagonia* spp.)	ANTH		28	whole	3 500	Herbes, 1976
Fathead minnow (*P. promelas*)	ANTH		48-72		485	Southworth, 1979

Rainbow trout (*S. gairdneri*)	ANTH		72		4 400-9 200	Linder *et al*., 1985
Cladoceran (*D. pulex*)	PH	30	24	whole	374	Southworth *et al*., 1978
Cladoceran (*D. pulex*)	9-MAN	6	24	whole	3 896	Southworth *et al*., 1978
Rainbow trout (*O. mykiss*)	FLAN	3.31	72	muscle + kidney	96	Gerhart & Carlson, 1978
O. mykiss	FLAN	3.31	168	muscle + kidney	82	Gerhart & Carlson, 1978
O. mykiss	FLAN	3.31	240	muscle + kidney	123	Gerhart & Carlson, 1978
O. mykiss	FLAN	3.31	504	muscle + kidney	378	Gerhart & Carlson, 1978
Cladoceran (*D. pulex*)	PY	50	24	whole	3 283	Southworth *et al*., 1978
Rainbow trout (*O. mykiss*)	PY	3.89	72	muscle + kidney	24	Gerhart & Carlson, 1978
O. mykiss	PY	3.89	168	muscle + kidney	21	Gerhart & Carlson, 1978
O. mykiss	PY	3.89	240	muscle + kidney	39	Gerhart & Carlson, 1978

O. mykiss	PY	3.89	504	muscle + kidney	72	Gerhart & Carlson, 1978
Cladoceran (*D. pulex*)	B[a] ANTH	6	24	whole	4 646	Southworth *et al*., 1978
Midge (*C. riparius*)	B[a]P		8		166	Leversee *et al*., 1981
Cladoceran (*D. magna*)	B[a]P		6		2 837	Leversee *et al*., 1981
Cladoceran (*D. magna*)	B[a]P	20	72	whole	134 248	Lu *et al*., 1977
Snail (*Physa* spp.)	B[a]P	2.5	72	whole	82 231	Lu *et al*., 1977
Mosquito (*C. pipiens quinquefasciatus*)	B[a]P	2.5	72	whole	11 536	Lu *et al*., 1977
Mosquitofish (*G. affinis*)	B[a]P	2.5	72	whole	930	Lu *et al*., 1977
Bluegill (*L. macrochirus*)	B[a]P		4		12	Leversee *et al*., 1981
Bluegill (*L. macrochirus*)	B[a]P		48		2 657	McCarthy and Jimenez, 1985a
L. macrochirus	B[a]P		48		225	McCarthy and Jimenez, 1985a
Rainbow trout (*O. mykiss*)	B[a]P	0.40	240	muscle + kidney	920	Gerhart & Carlson, 1978
O. mykiss	B[a]P	0.40	240	liver	182	Gerhart & Carlson, 1978
Northern pike (*E. lucius*)	B[a]P		3.36	bile+gall bladder	3 974	Balk *et al*., 1984

E. lucius	B[a]P		19.2	bile+gall bladder	36 656	Balk *et al*., 1984
E. lucius	B[a]P		204	bile+gall bladder	82 916	Balk *et al*., 1984
E. lucius	B[a]P		552	bile+gall bladder	53 074	Balk *et al*., 1984
E. lucius	B[a]P		3.36	liver	259	Balk *et al*., 1984
E. lucius	B[a]P		19.2	liver	578	Balk *et al*., 1984
E. lucius	B[a]P		204	liver	1 276	Balk *et al*., 1984
E. lucius	B[a]P		552	liver	619	Balk *et al*., 1984
E. lucius	B[a]P		3.36	gills	283	Balk *et al*., 1984
E. lucius	B[a]P		19.2	gills	382	Balk *et al*., 1984
E. lucius	B[a]P		204.	gills	373	Balk *et al*., 1984
E. lucius	B[a]P		552	gills	213	Balk *et al*., 1984
E. lucius	B[a]P		3.36	kidney	192	Balk *et al*., 1984
E. lucius	B[a]P		19.2	kidney	872	Balk *et al*., 1984
E. lucius	B[a]P		204	kidney	1 603	Balk *et al*., 1984
E. lucius	B[a]P		552	kidney	2 015	Balk *et al*., 1984
E. lucius	B[a]P		3.36-552	other tissues	<55	Balk *et al*., 1984
Cladoceran (*D. pulex*)	PERY	0.3	24	whole	5 410	Southworth *et al*., 1978

TABLE 24

Bioconcentration Factors of PAHs (BCF) in Marine and Estuarine Animals

Organism	***PAH***	***Exposure Conc.* (µg/L)**	***Dura-tion* (hours)**	***Tissue***	***BCF***	***References***
Polychaete (*N. arenaceodentata*)	NA	150	3-24	whole	40	Rossi, 1977
Clam (*R. cuneata*)	NA	71	24	whole	6.1	Neff *et al* ., 1976a
Clam (*R. cuneata*)	NA	840	24	whole	2.3	Neff *et al* ., 1976b
Blue mussel (*M. edulis*)	NA	32	4	whole	44	Lee *et al* ., 1972
Copepod (*C. helgolandicus*)	NA	1	24	whole	60	Harris *et al* ., 1977a
Copepod (*C. helgolandicus*)	NA	0.20	24	whole	50	Harris *et al* ., 1977b
Copepod (*E. affinis*)	NA	1	216	whole	5 000	Harris *et al* ., 1977b
Brown shrimp (*P. aztecus*)	NA	2.3	72	whole	195	Cox *et al* ., 1975
Fiddler crab (*U. minax*)	NA	23	72	whole	325	Cox *et al* ., 1975
Wharf crab (*S. cinereum*)	NA	2.3	72	whole	404	Cox *et al* ., 1975
Atlantic salmon (*S. salar*)	NA		168	egg	44-83	Kuhnhold and Busch, 1978
Speckled sanddab (*C. stigmaeus*)	NA	21.3	1	muscle	76.9	Lee *et al* ., 1972
C. stigmaeus	NA	21.3	1	liver	133	Lee *et al* ., 1972
C. stigmaeus	NA	21.3	1	gut	22.5	Lee *et al* ., 1972
C. stigmaeus	NA	21.3	1	gills	160.3	Lee *et al* ., 1972

C. stigmaeus	NA	21.3	1	heart	36.6	Lee *et al* ., 1972
Mudsucker (*G. mirabilis*)	NA	32	2	muscle	11.8	Lee *et al* ., 1972
G. mirabilis	NA	32	2	liver	252	Lee *et al* ., 1972
G. mirabilis	NA	32	2	gut	34.8	Lee *et al* ., 1972
G. mirabilis	NA	32	2	gills	37.2	Lee *et al* ., 1972
G. mirabilis	NA	32	2	heart	41.6	Lee *et al* ., 1972
Mudsucker (*G. mirabilis*)	NA	29 000	1	muscle	62.4	Lee *et al* ., 1972
G. mirabilis	NA	29 000	1	liver	15.1	Lee *et al* ., 1972
G. mirabilis	NA	29 000	1	gut	38.4	Lee *et al* ., 1972
G. mirabilis	NA	29 000	1	gills	61.8	Lee *et al* ., 1972
G. mirabilis	NA	29 000	1	heart	17.2	Lee *et al* ., 1972
Starry flounder (*P. stellatus*)	NA	3	336	muscle	220	Roubal *et al* ., 1978
Coho salmon (*O. kisutch*)	NA	3	840	muscle	16	Roubal *et al* ., 1978
Clam (*R. cuneata*)	1-MNA	340	24	whole	8.5	Neff *et al* ., 1976b
Starry flounder (*P. stellatus*)	1-MNA	3	336	muscle	320	Roubal *et al* ., 1978
Coho salmon (*O. kisutch*)	1-MNA	3	840	muscle	38	Roubal *et al* ., 1978
Clam (*R. cuneata*)	2-MNA	480	24	whole	8.1	Neff *et al* ., 1976b

Starry flounder (*P. stellatus*)	2-MNA	3	336	muscle	400	Roubal *et al* ., 1978
Coho salmon (*O. kisutch*)	2-MNA	3	840	muscle	26	Roubal *et al* ., 1978
Brown shrimp (*P. aztecus*)	mNA	15.4	72	whole	234	Cox *et al* ., 1975
Fiddler crab (*U. minax*)	mNA	15.4	72	whole	294	Cox *et al* ., 1975
Wharf crab (*S. cinereum*)	mNA	15.4	72	whole	393	Cox *et al* ., 1975
Clam (*R. cuneata*)	d-MNA	240	24	whole	17.1	Neff *et al* ., 1976b
Brown shrimp (*P. aztecus*)	d-MNA	15.2	72	whole	967	Cox *et al* ., 1975
Fiddler crab (*U. minax*)	d-MNA	15.2	72	whole	1 105	Cox *et al* ., 1975
Wharf crab (*S. cinereum*)	d-MNA	15.2	72	whole	1 625	Cox *et al* ., 1975
Clam (*R. cuneata*)	t-MNA	30	24	whole	26.7	Neff *et al* ., 1976b
American oyster (*C. virginica*)	ANTH	16.7	48	whole	1 160	Lee *et al* ., 1978
Clam (*R. cuneata*)	PH	89	24	whole	32	Neff *et al* ., 1976a
Clam (*M. inquinata*)	PH	3.7	168	whole	10.3	Roesijadi *et al* ., 1978
American oyster (*C. virginica*)	FLAN	3.3	48	whole	2 860	Lee *et al* ., 1978
Grass shrimp (*P. pugio*)	BAN	2.8	3	digestive tract	376	Fox and Rao, 1982
Grass shrimp (*P. pugio*)	BAN	2.8	3	hepato-pancreas	231	Fox and Rao, 1982

Grass shrimp (*P. pugio*)	BAN	2.8	3	muscle	85	Fox and Rao, 1982
Grass shrimp (*P. pugio*)	BAN	2.8	3	cephalo-thorax	25	Fox and Rao, 1982
American oyster (*C. virginica*)	B[a] ANTH	8.3	48	whole	3 700	Lee *et al* ., 1978
Clam (*R. cuneata*)	CH	66	24	whole	8.2	Neff *et al* ., 1976a
Clam (*M. inquinata*)	CH		168	whole	694	Roesijadi *et al* ., 1978
Pink shrimp (*P. duorarum*)	CH	1	672	cephalo-thorax	248	Miller *et al* ., 1982
Pink shrimp (*P. duorarum*)	CH	5	672	cephalo-thorax	362	Miller *et al* ., 1982
Pink shrimp (*P. duorarum*)	CH	1	672	abdomen	199	Miller *et al* ., 1982
Pink shrimp (*P. duorarum*)	CH	5	672	abdomen	84	Miller *et al* ., 1982
American oyster (*C. virginica*)	B[a]P	8.3	48	whole	2 560	Lee *et al* ., 1978
American oyster (*C. virginica*)	B[a]P		336	whole	242	USEPA, 1980
Clam (*R. cuneata*)	B[a]P	52	24	whole	8.7	Neff *et al* ., 1976a
Clam (*R. cuneata*)	B[a]P	30.5	24	whole	236	Cox *et al* ., 1975
Clam (*M. inquinata*)	B[a]P	0.043	168	whole	861	Roesijadi *et al* ., 1978
Grass Shrimp (*P. pugio*)	B[a]P	2.5	3	digestive tract	154	Fox and Rao, 1982
Grass Shrimp (*P. pugio*)	B[a]P	2.5	3	hepato-pancreas	49	Fox and Rao, 1982
Grass Shrimp (*P. pugio*)	B[a]P	2.5	3	muscle	3.5	Fox and Rao, 1982

Grass Shrimp (*P. pugio*)	B[a]P	2.5	3	cephalo-thorax	10	Fox and Rao, 1982
Atlantic salmon (*S. salar*)	B[a]P		168	egg	71	Kuhnhold and Busch, 1978
Sand sole (*P. melanostictus*)	B[a]P	0.10	144	egg	21 000	Hose *et al* ., 1982

mixing the contaminated sediment of Eagle Harbor (Total PAH = 6 461 µg/g dry weight) with the uncontaminated sediment of Yaquina Bay (Total PAH = 0.10 µg/g dry weight). From these tests, the 4-d LC_{50} value for the Eagle Harbor sediment mixed into Yaquina Bay sediment was calculated to be 666 µg/g wet weight (i.e., concentration of Eagle Harbor sediment in a mixture of sediments from Eagle Harbor and Yaquina Bay). The concentrations of the PAHs (in µg/g dry weight) in the 4-d LC_{50}-mixture were as follows:

Naphthalene = 0.03 Acenaphthylene = 0.02 Acenaphthene= 0.15
Fluorene = 0.21 Phenanthrene = 0.95 Anthracene = 0.07
Fluoranthene= 0.60 Pyrene = 0.35 B[a]ANTH = 0.08
Chrysene = 0.08 B[b]FLAN = 0.03 B[k]FLAN = 0.01
B[a]P = 0.01 Total PAH = 2.59

Since the concentrations in the 4-d LC_{50} Eagle Harbor sediment of both phenanthrene (0.95 µg/g dry weight) and fluoranthene (0.60 µg/g dry weight) were much less than the 10-d LC_{50}s for the individual PAHs (see above; Swartz *et al*., 1988), it was concluded that either the toxic action of other single chemicals alone or joint action between chemicals may have been responsible for the toxicity of diluted Eagle Harbor sediment. Swartz *et al*. (1989) also cautioned against extrapolating LC_{50} data for PAHs in Eagle Harbour sediment to toxicities with bulk PAH concentrations in sediment from other sources. This is because sediment samples have been collected in Eagle Harbor that did not cause acute toxicity, but had fluoranthene and phenanthrene concentrations much higher than the Yaquina Bay-Eagle Harbour sediment mixture which caused 50% mortality in the organisms.

In the above experiment with sediment mixtures, Swartz *et al*., (1989) also compared the interstitial water concentration of phenanthrene in sediments from nine Eagle Harbor stations that caused no amphipod mortality. The highest concentration in Eagle Harbor dilution experiments that caused no mortality was 6.0 µg /L phenanthrene, which is the same (i.e., 5.9 µg/L phenanthrene) as quoted in Tetra Tech., Inc. (1986) as the safe level for amphipods. It was concluded that interstitial water concentration of phenanthrene and other chemicals may provide a better indication of sediment toxicity

than bulk concentration in sediment. Several investigators appear to support this conclusion (Hargis *et al*., 1984; Adams *et al*., 1985).

6.9 Criteria from other jurisdictions

There is a growing concern about PAHs due to the toxic and carcinogenic properties of certain of these substances. Concern is also increasing because of the lack of definite environmental information on these compounds despite the advance of detection technology (e.g., GC/MS, HPLC). Thus, while there is a need for established PAH criteria, few agencies have been able to venture forth with any definite quantitative values (Table 25)

From studies conducted in the Great Lakes basin, GLSAB (1983) recommended that levels of B[a]P not exceed 1.0 µg/g in sediment (dw) and organisms (ww) serving as food items for the protection of aquatic life. This report also suggested that levels of B[a]P be less than 0.01 µg/L in the water column.

The USEPA (1980) did not establish PAH criteria for the protection of aquatic life. The criteria recommended by the agency were related to the protection of human health from ingesting contaminated water and contaminated organisms living in it.

Provisional sediment quality objectives for PAHs in Burrard Inlet, British Columbia, were recently published by Nijman and Swain (1990). These objectives were derived from apparent effect threshold (AET; i.e., sediment concentration of a selected chemical above which statistically significant biological effects always occur) values developed for Puget Sound, Washington, generally using an application factor of 0.1. They also took into account values from relatively uncontaminated areas. For most PAHs (except fluorene, dibenzo[a,h]anthracene, and pyrene), the objectives proposed by Nijman and Swain are lower than the ER-L values (i.e., sediment concentration at the low end or the 10th percentile concentration of the range in which effects had been observed) determined by the National Oceanic and Atmospheric Administration (NOAA) (Long and Morgan, 1990).

The interim criteria for PAHs proposed by the Washington Department of Ecology (WDOE - Table 25) are expressed on the basis of organic carbon; hence, as such, they can not be compared with criteria proposed by other jurisdictions. To carry out a comparison with other jurisdictions, the WDOE interim criteria were expressed on a dry weight basis, assuming an organic carbon content of 1.0% for the sediment (conversion factor used is: µg/g oc= 0.01 µg/g dw sediment). The results (not shown) indicated that the WDOE interim criteria (expressed on sediment basis) were up to 9 times higher for some PAHs than the ER-L values determined by NOAA.

TABLE 25

PAH Criteria for Aquatic Life from other Jurisdictions

CRITERIA STATEMENT	***CRITERIA VALUE****	***JURISDICTION***	***DATE***	***REFERENCES***
Water Ambient water quality criteria for the protection of human health from ingesting contaminated water and organisms: Total PAH · cancer risk level = 10-5 · cancer risk level = 10-6 · cancer risk level = 10-7 For the protection of aquatic life the level of B[a]P in water should be less than 0.01 µg/L	0.02800 µg/L 0.00280 µg/L 0.00028 µg/L 0.01 µg/L	USEPA Great Lakes Science Advisory Board	1980 1988	USEPA (1980) GLSAB, 1988
Fish For the the protection of aquatic life, the levels of B[a]P in organisms serving as a food source for fish should not exceed 1.0 µg/g wet weight	1.0 µg/g ww	Great Lakes Science Advisory Board	1988	GLSAB, 1988
Sediment For the the protection of aquatic life, the levels of B[a]P in the sediment should not exceed 1.0 µg/g dry weight	1.0 µg/g dw	Great Lakes Science Advisory Board	1988	GLSAB, 1988
Lowest Effect Level: Tentative guideline for Total PAH (i.e., ANA+acenephthylene+ANTH+ B[k]FLAN+benzo[b]fluorene+ B[a]ANTH+B[a]P+B[ghi]PERY+	2.0 µg/g dw	Ontario	1991	Persaud *et al*., 1991

CH+D[ah]AN+FLAN+FL+ I[123-cd]PY+NA+PH+PY)				
Interim sediment quality criteria: ·Naphthalene ·Acenaphthylene ·Acenaphthene ·Fluorene	99 µg/g oc 66 µg/g oc 16 µg/g oc 23 µg/g oc	Washington	1991	WDOE, 1991
Interim sediment quality criteria: ·Phenanthrene ·Anthracene ·2-Methylnaphthalene LPAH#1 ·Fluoranthene ·Pyrene ·Benz[a]anthracene ·Chrysene ·Benzofluoranthenes (Total)#2 ·Benzo[a]pyrene ·Indeno[1,2,3-c,d]pyrene ·Dibenzo[a,h]anthracene ·Benzo[g,h,i]perylene HPAH#3	100 µg/g oc 230 µg/g oc 38 µg/g oc 370 µg/g oc 160 µg/g oc 1000 µg/g oc 110 µg/g oc 110 µg/g oc 230 µg/g oc 99 µg/g oc 34 µg/g oc 12 µg/g oc 31 µg/g oc 960 µg/g oc	Washington	1991	WDOE, 1991
Conc. at low end of the range in which effects had been observed (i.e., ER-L): ·Acenaphthene ·Anthracene ·Benz[a]anthracene ·Benzo[a]pyrene ·Chrysene ·D[ah]ANTH	0.15 µg/g dw 0.09 µg/g dw 0.23 µg/g dw 0.40 µg/g dw 0.40 µg/g	National Oceanic & Atmospheric Administration (NOAA)	1990	Long and Morgan, 1990

·Fluoranthene ·Fluorene ·2-methylnaphthalene ·Naphthalene ·Phenanthrene ·Pyrene ·Total PAH	dw 0.06 µg/g dw 0.60 µg/g dw 0.04 µg/g dw 0.07 µg/g dw 0.34 µg/g dw 0.23 µg/g dw 0.35 µg/g dw 4.00 µg/g dw			
Maximum objective levels for Burrard Inlet sediments : ·Naphthalene ·Acenaphthylene ·Acenaphthene ·Fluorene ·Phenanthrene ·Anthracene Total LPAH ·Fluoranthene ·Pyrene ·Benz[a]anthracene	0.20 µg/g dw 0.06 µg/g dw 0.05 µg/g dw 0.05 µg/g dw 0.15 µg/g dw 0.10 µg/g dw 0.50 µg/g dw 0.17 µg/g dw 0.26 µg/g dw 0.13 µg/g dw	British Columbia	1990	Nijman and Swain, 1990
Maximum objective levels for Burrard Inlet sediments : ·Chrysene	0.14 µg/g dw 0.32 µg/g	British Columbia	1990	Nijman and Swain, 1990

·Benzofluoranthene ·Benzo[a]pyrene ·Indeno[1,2,3-c,d]pyrene ·Dibenzo[a,h]anthracene ·Benzo[g,h,i]perylene Total HPAH	dw 0.16 µg/g dw 0.06 µg/g dw 0.06 µg/g dw 0.07 µg/g dw 1.20 µg/g dw			

* Sediment criteria are stated on dry weight (dw) or organic carbon (oc) basis;
#1 LPAH= naphthalene+acenaphthylene+acenaphthene+fluorene+phenanthrene+anthracene;
#2 Total benzoflouranthenes= B[b]FLAN+B[j]FLAN+B[k]FLAN;
#3 HPAH= FLAN+ PY+ B[a]ANTH+ CH+ Total benzofluoranthenes+ B[a]P+ I[123-cd]PY+

D[ah]ANTH+ B[ghi]PERY

CCREM (1987) concluded that there were insufficient data to set guidelines in water; sediment and biota were not considered.

6.10 Recommended Criteria

6.10.1 Water

To protect freshwater aquatic life from long-term and phototoxic effects, and marine aquatic life from long-term effects, it is recommended that the concentration of specified PAHs in water should not exceed those shown in Table 26.

6.10.3 Sediment

For the protection of aquatic life, the concentrations of specified PAHs in sediments containing 1% organic carbon should not exceed those shown below in Table 27. For sediment containing organic carbon content other than 1%, appropriate criteria can be obtained by multiplying values shown in Table 27 by the percent organic carbon content of the sediment (e.g. if the sediment had 5% organic carbon you would multiply the sediment guideline value in Table 27 by 5) .

Table 26			
Recommended Interim Freshwater and Marine Water Quality Criteria*			
PAH	***Freshwater (long-term) (µg/L)***	***Freshwater (Phototoxic effects) (µg/L)***	***Marine water (long-term) (µg/L)***
Naphthalene	1	NR+	1
Methylated naphthalenes	NR	NR	1
Acenaphthene	6	NR	6
Fluorene	12	NR	12
Anthracene	4	0.1	NR
Phenanthrene	0.3	NR	NR
Acridine	3	0.05	NR
Fluoranthene	4	0.2	NR
Pyrene	NR	0.02	NR
Chrysene	NR	NR	0.1
Benz[a]anthracene	0.1	0.1	NR
Benzo[a]pyrene	0.01	NR	0.01

+ NR = not recommended due to insufficient data;
* average concentrations

Table 27					
Recommended Interim Sediment Quality Criteria					
PAH	***Kow (From Table 1)***	***Water Quality Criteria (µg/L) (From Table 26)***		***Sediment Quality Criteria (µg PAH/g dry weight)****	
		Freshwater	***Marine***	***Freshwater***	***Marine***
Naphthalene	2 344	1	1	0.01	0.01
Acenaphthene#	9 550	6	6	0.15	0.15
Fluorene#	15 136	12	12	0.2	0.2
Anthracene	31 623	4	NR+	0.6	NR

Phenanthrene	28 840	0.3	NR	0.04	NR
Acridine	60 399	3	NR	1	NR
Fluoranthene	79 433	4	NR	2	NR
Chrysene	426 580	NR	0.1	NR	0.2
Benz[a]anthracene	426 580	0.1	NR	0.2	NR
Benzo[a]pyrene	1 096 478	0.01	0.01	0.06	0.06

*sediment containing 1% organic carbon; average concentrations
+NR= not recommended due to insufficient data;

#sediment criteria for these PAHs were adjusted to make them compatible with WDOE (see section 6.10.3).

6.11 Rationale

6.11.1 Fresh and Marine waters

PAHs are highly hydrophobic and lipophilic compounds which have the potential to bioaccumulate in aquatic organisms (Dobrowsky and Epifanio, 1980). They are also slightly soluble in water. As a result only a single average criterion was recommended for each PAH.

Because of insufficient data only interim criteria were recommended for PAHs in this document. Chrysene and benzo[a]pyrene in marine water did not meet the minimum data requirements recommended by either the draft B.C. Environment or the CCREM protocols for setting interim criteria. Despite this, interim marine water criteria are proposed for these two PAHs since the available chronic data were considered to be of good quality (i.e., primary data). In other cases where minimum data requirements are not met either for marine or fresh water, a comparison between the available data (e.g., between freshwater and marine data, relative potency factor with regard to phototoxicity) was used to derive the interim criteria for the PAHs (e.g., marine water criterion for acenaphthene and fluorene). For the definition of criteria and interim criteria, refer to the British Columbia (Singleton *et al.,* 1992) and CCREM (1987) protocols for the derivation of water quality criteria/guidelines.

The criteria recommended in this document for the protection of aquatic life from long-term effects of PAHs, are obtained by multiplying the lowest observed effect levels from acute or chronic tests with appropriate application or safety factors. Several safety

factors were used in the derivation of PAH criteria. The choice of an application factor was based on the British Columbia Environment (Singleton *et al.,* 1992) and CCREM (1987) protocols. For instance, the protocols recommend that (a) criteria for persistent contaminants, which show a potential for bioaccumulation in aquatic organisms (e.g., PAHs), may be derived by multiplying the lowest observed LC_{50} or EC_{50} with a safety factor of 0.01 (CCREM, 1987, Singleton *et al*., 1992); (b) where the lowest observed effect level is available from a chronic toxicity test, a safety factor between 0.1 and 0.5 is acceptable (Singleton *et al*., 1992).

To derive PAH criteria, a safety factor of 0.1 was used with most chronic data. For naphthalene in marine water, a safety factor of 0.5 was used along with the lowest observed chronic level; an explanation for this is given in section 6.11.1(a) below. Due to (a) the general paucity of data on PAHs, and (b) the fairly long lifespan (4 to 8 years) for rainbow trout, the 648-h LC_{50} (for rainbow trout) used in the derivation of criteria for certain PAHs (e.g., phenanthrene in freshwater) was considered to be an acute value. The starting values (e.g., LC_{50}, LOEL, etc.), and the safety factors used in the derivation of PAH criteria are shown in Table 28. For most PAHs, the application factors used in the derivation of PAH criteria are consistent with the recommendations of the B.C. and CCREM protocols (Table 28). The case of anthracene in freshwater was an exception; the departure from the usage of the recommended safety factor is explained below.

TABLE 28

Lowest observed effect levels and application factors used in the derivation of water quality criteria to protect aquatic life from long-term effects of PAHs

Polycyclic Aromatic	***Freshwater***			***Marine water***		
Hydrocarbons	***LOEL (µg/L)***	***Type of effect (organism)***	***Safety Factor***	***LOEL (µg/L)***	***Type of effect (organism)***	***Safety Factor***
Naphthalene*	11	Chronic effects (rainbow trout)	0.1	2	Chronic (mummichog)	0.5
Methylated naphthalenes				10.4	Chronic (copepod)	0.1

Acenaphthene	580	96-h LC_{50} (fathead minnow)	0.01			
Fluorene	125	Chronic effects (bluegill)	0.1			
Anthracene	42	3-h EC_{50} (algae)	0.1			
Phenanthrene	30	648-h LC_{50} (rainbow trout)	0.01			
Acridine+	34	Chronic effects (rainbow trout)	0.1			
Fluoranthene	38	Chronic (algae)	0.1			
Chrysene				1	Chronic (pink shrimp)	0.1
Benz[a]anthracene	10	48-h LC_{50} (*Daphnia*)	0.01			
Benzo[a]pyrene				0.1	Chronic (sand sole)	0.1

* geometric mean of LOELs of 8 µg/L and 15 µg/L (Table 18);
+ geometric mean of LOELs of 12µg/L and 98 µg/L (Table 18).

For some PAHs, water quality criteria were also recommended to protect freshwater aquatic life from both phototoxic and long-term (but non-phototoxic) effects of the PAHs. The phototoxic criteria were derived either by multiplying the lowest observed effect level by an appropriate safety factor or by multiplying or dividing a known criterion by the toxic potency factor[10] of a given PAH. The application factors used in the derivation of the phototoxic criteria were selected in the same manner as in the long-term effect criteria.

(a) Naphthalene

The criterion recommended to protect freshwater aquatic life from long-term effects (1.0 µg/L) is based on chronic toxicity of naphthalene to rainbow trout (*O. mykiss*) exposed to 11 µg NA/L (geometric mean of the two LOELs; Black *et al*., 1983 in Table 18). The chronic tests by Black *et al*. (1983) were conducted using a flow-through system in which the concentration of the PAH was measured during the experiment, and, therefore, represented primary data (see footnote 5, page 61). The chronic value of 11 µg/L was the lowest concentration at which an effect of naphthalene was recorded in freshwater organisms (Tables 16, 17 and 18). Based on the recommendations in the CCREM and B.C. Environment protocols for the derivation of water quality criteria, an application factor of 0.1 was applied to derive the recommended interim criterion.

The criterion (i.e., 1.0 µg NA/L) recommended for the protection of marine aquatic life from long-term effects is based on non-lethal chronic effects (gill hyperplasia) of naphthalene to mummichog (*F. heteroclitus*) exposed to 2 µg NA/L for 15 days (DiMichele and Taylor, 1978 - Table 22) and an application factor of 0.5. The chronic value of 2.0 µg NA/L was the lowest concentration at which an effect of naphthalene was recorded in a marine environment.

The safety factor of 0.5 used in developing the marine water criterion was consistent with the B.C. Environment protocol (see section 6.10.1), but it was less conservative than the safety factor of 0.1 used for the freshwater criterion. Since chronic toxicity of naphthalene in fresh and marine water environments was similar, the choice of 0.5 as a safety factor was preferred as it yielded a marine water criterion which was compatible with the freshwater criterion.

(b) Acenaphthene

The criterion of 6 µg/L recommended for the protection of freshwater aquatic life from long-term effects of acenaphthene is based on the minimum lethal concentration (i.e., 96-h LC_{50} of 580 µg/L for fathead minnow, *P. promelas*) observed to have a significant effect on aquatic life (Holcombe *et al*., 1983 - Table 16) and a safety factor of 0.01. The 96-h LC_{50} used in the derivation of the criterion was considered to be a primary data point since it was obtained using a flow-through system in which the PAH concentration was measured during the experiment. Hence, it was chosen as a starting point for the derivation of acenaphthene criterion. Since there are no chronic data on fish and invertebrates, the recommended guideline for acenaphthene is an interim criterion.

The interim water quality criterion for acenaphthene to protect marine aquatic life is the same (6 µg ANA/L) as the freshwater criterion, since the available data suggest that the acenaphthene toxicity (minimum LC_{50}s) is similar in freshwater and marine environments (Tables 15 and 20).

(c) Fluorene

The criterion (12 µg FL/L) for the protection of freshwater aquatic life from the toxic effects of fluorene was based on the lowest observed chronic level of 125 µg/L for *Daphnia magna* (Finger *et al*., 1985 -Table 18). A safety factor of 0.1, which is consistent with the B.C. Environment and CCREM protocols, was applied to derive the criterion. The freshwater criterion for fluorene was classified as an interim criterion due to the lack of chronic data on fish and invertebrates.

The interim water quality criterion for fluorene to protect marine aquatic life is the same (12 µg FL/L) as the freshwater criterion, since the available data suggest that the fluorene toxicity (minimum LC_{50}s) is similar in freshwater and marine environments (Tables 16 and 21).

(d) Anthracene

The water quality criterion for anthracene to protect freshwater aquatic life from photo-induced toxicity of anthracene (0.1 µg/L ANTH) accumulated in fish tissue was based on the Bowling *et al*. (1983) (i.e., up to 100% mortality in fish exposed to 12.7 µg/L anthracene in the unshaded portion of the stream; Table 19) and Oris *et al*. (1984) (96-h LC_{50} of 11.9 µg/L) data. Bowling's results were obtained in a field experiment; hence they were preferred as a starting point for the derivation of the phototoxicity criterion for anthracene. An application factor of 0.01 was used to arrive at the recommended criterion. Since the long-term impact of solar ultraviolet radiation on aquatic life exposed to the PAH is unknown, the recommended criterion is an interim criterion. Aquatic life exposed to anthracene at the criterion level in deeper, turbid, and shaded waters will not be affected by the phototoxic effects of anthracene.

The water quality criterion for the protection of freshwater aquatic life from long-term chronic effects (excluding photo-induced toxicity) of anthracene (4 µg/L ANTH) was based on the EC_{50} of 42 µg/L observed for *Chlamydomonas angulosa* and *Chlorella vulgaris* exposed to anthracene (Hutchinson *et al*., 1980 - Table 17) and an application factor of 0.1. The application factor of 0.1 used in the derivation of the criterion is less stringent than recommended by the CCREM and the B.C. Environment protocols, when LC_{50} or EC_{50} is the starting point. This was justified, however, since the end point of the tests with the algae (i.e., 50% reduction in photosynthesis) was non-lethal; as a result, the EC_{50}s were considered to represent sublethal and chronic effects. Under this assumption, the use of 0.1 as the safety factor was consistent with the B.C. Environment protocol.

A criterion for the protection of marine aquatic life from toxic effects of anthracene was not recommended due to the lack of data.

(e) Phenanthrene

The criterion (0.3 µg/L) recommended to protect freshwater aquatic life from the long-term effects of phenanthrene is based on the minimum concentration (i.e., 648-h LC_{50} of 30 µg/L for rainbow trout, *O. mykiss*) observed to cause a significant effect on aquatic life (Millemann *et al*., 1984 - Table 16), and a safety factor of 0.01. The 648-h LC_{50} used in the derivation of the the criterion was considered a primary data point since it was obtained using a flow-through system in which concentration of the PAH was measured during the experiment. Because of the lack of chronic data on fish and invertebrates, the recommended criterion for phenanthrene should be considered as an interim criterion.

The recommended criterion of 0.3 µg/L for the protection of aquatic life from chronic effects of phenanthrene was further justified by the Black *et al*. (1983) data. These investigators found an LOEL of 4 µg/L for rainbow trout (at the embryo-larval stages) exposed to phenanthrene (Table 18). The lowest observed chronic level of 4 µg/L in conjunction with a safety factor of 0.1 (as recommended by the CCREM and B.C. Environment protocols) yields a criterion which is about the same as that derived using the 648-h LC_{50} as the starting point.

Due to the lack of data, a criterion for the protection of marine aquatic life from the harmful effects of phenanthrene was not recommended.

(f) Acridine

Acridine has been shown to be phototoxic to aquatic organisms (Tables 19 and 20). Therefore, the water quality criterion for acridine was designed to protect freshwater aquatic life from both chronic (3 µg/L) and photo-induced (0.05 µg/L) toxicity.

The water quality criterion (3 µg/L) for the protection of aquatic life from long-term effects (excluding photo-induced toxicity) was based on the lowest observed chronic value of 34 µg/L (= geometric mean of 12 µg/L and 98 µg/L) observed for *O. mykiss* exposed to acridine (Black *et al*., 1983 - Table 18). An application factor of 0.1 was used which was consistent with the CCREM and B.C. Environment protocol recommendations.

The interim criterion for protection against phototoxic effects of acridine was based on (a) the criterion for anthracene (0.1 µg ANTH/L), and (b) the potency of acridine to

induce phototoxic effects. The relative (to anthracene) potency factor for acridine causing phototoxicity in the larvae of the fathead minnow, *Pimephales promelas*, was given to be 2.247 by Oris and Giesy (1987) (i.e., acridine is 2.247 times more phototoxic than anthracene). The phototoxic criterion for acridine was calculated as: 0.1 µg/L ÷ 2.247 = ~0.05 µg/L.

Due to the lack of data, a criterion for the protection of marine aquatic life from the toxic effects of acridine was not recommended.

(g) Fluoranthene

Fluoranthene has been shown to be phototoxic to aquatic organisms (Tables 19 and 20). Thus, the water quality criterion for fluoranthene was designed to protect freshwater aquatic life from both photo-induced toxicity as well as chronic effects.

The water quality criterion (4 µg/L) for the protection of aquatic life from long-term effects (excluding photo-induced toxicity) of fluoranthene was based on chronic effects observed for blue-green algae *A. flos-aquae* exposed to 38 µg FLAN/L (Bastian and Toetz, 1982 - Table 17). A safety factor of 0.1, consistent with the B.C. Environment protocol, was used to derive the interim criterion.

The interim criterion (0.2 µg/L) to protect against phototoxic effects of fluoranthene was based on the benz[a]anthracene (0.1 µg B[a]ANTH/L) criterion and the potency (relative to B[a]ANTH) of fluoranthene to induce phototoxic effects. The relative potency factor calculations, using adjusted mean lethal times, suggested that fluoranthene was 1.94 times less phototoxic to *Daphnia magna* than Benz[a]anthracene (Newsted and Giesy, 1987). The recommended criterion was calculated as: 0.1 µg/L x 1.96 = ~0.2 µg/L fluoranthene.

Due to the lack of data, a criterion for the protection of marine aquatic life from toxic effects of fluoranthene was not recommended.

(h) Pyrene

Pyrene has been shown to be phototoxic to aquatic organisms (Tables 19 and 20). Therefore, the water quality criterion for pyrene was designed to protect freshwater aquatic life from the photo-induced toxicity. The interim criterion for pyrene was based on the anthracene (0.1 µg ANTH/L) criterion and the potency of pyrene to induce phototoxic effects. The relative potency factor (relative to anthracene) for pyrene causing phototoxicity in the larvae of the fathead minnow, *Pimephales promelas*, was calculated to be 4.656 by Oris and Giesy (1987) (i.e., pyrene is 4.656 times more toxic

than anthracene). The recommended criterion was calculated as: 0.1 µg/L ÷ 4.656 = ~ 0.02 µg/L pyrene.

Due to the lack of data, a criterion for the protection of marine aquatic life from the harmful effects of pyrene was not recommended.

(i) Benz[a]anthracene

Benz[a]anthracene has been shown to be phototoxic to aquatic organisms (Tables 19 and 20). Therefore, the water quality criterion for benz[a]anthracene was designed to protect freshwater aquatic life from both chronic and photo-induced toxicities.

The water quality criterion (0.1 µg/L benz[a]anthracene) for the protection of aquatic life from long-term effects (excluding photo-induced toxicity) was based on a 48-h LC_{50} of 10 µg/L observed for *Daphnia pulex* exposed to benz[a]anthracene (Trucco *et al*., 1983 - Table 16) and an application factor of 0.01. The application factor was consistent with the B.C. Environment and CCREM protocols. The criterion of 0.1 µg/L will also protect freshwater aquatic life from phototoxic effects of B[a]ANTH (see below).

The interim criterion to provide protection against photo-induced toxicity was based on the anthracene (0.1 µg ANTH/L) criterion and the potency of benz[a]anthracene to induce phototoxic effects. The relative potency factor (relative to anthracene) for benz[a]anthracene causing phototoxicity in the larvae of the fathead minnow, *Pimephales promelas*, was given to be 0.763 by Oris and Giesy (1987) (i.e., benz[a]anthracene is 0.763 times is less toxic than anthracene). The phototoxic criterion was calculated as: 0.1 x 0.763 = ~0.1.

Due to the lack of data, a criterion for the protection of marine aquatic life from the harmful effects of benz[a]anthracene was not recommended.

(j) Chrysene

Due to the lack of data, a water quality criterion for the protection of freshwater aquatic life exposed to chrysene was not recommended.

The interim criterion to protect marine aquatic life exposed to chrysene (0.1 µg/L) was based on the chronic toxicity of the PAH to pink shrimp, *P. duorarum* (Miller *et al*. 1982 - Table 22). The minimum concentration causing an effect (i.e., increased incidence of molts after 28 days of exposure) was 1.0 µg CH/L. An application factor of 0.1, consistent with the B.C. Environment protocol, was used to arrive at the interim criterion.

Chrysene did not meet the minimum data requirement recommended by the B.C. Environment and CCREM protocols for setting interim water quality criterion. A water (marine) quality criterion was, however, recommended for chrysene, since the available data were considered to be of good quality. The acceptance of the chrysene criterion from limited data was also justified on the premise that the sediment quality criterion, which was based on its water quality criterion (Tables 27 and 28), protected benthic organisms from harmful effects of chrysene; note that the recommended sediment criterion for chrysene is lower than the apparent effects threshold concentration for oysters in Puget Sound (i.e., WDOE criteria- Figure 5).

(k) Benzo[a]pyrene

The interim criterion for the protection of marine aquatic life exposed to benzo[a]pyrene was based on the lowest observed chronic level of the PAH to sand sole (*P. melanostichus*) (Hose *et al*., 1982, Table 22). The investigators observed that the average hatching success in sand sole exposed to 0.10 µg/L B[a]P was reduced by about 29% compared to the control. An application factor of 0.1, which is consistent with the B.C. Environment protocol, was used to derive the criterion.

The interim criterion (0.01 µg/L benzo[a]pyrene) for the protection of freshwater aquatic life from long-term effects is the same as that recommended for marine aquatic life (see above). The toxicity of B[a]P to aquatic life appears to be of similar magnitude for both freshwater and marine environments (e.g., the LOEL for fresh and marine waters, respectively, are 0.2 µg/L (Hose *et al*., 1984-Table 18) and 0.1 µg/L (Hose *et al*., 1982-Table 22)). The recommended criterion of 0.01 µg/L is 20 times lower than the lowest observed effect level (chronic) for rainbow trout exposed to 0.2 µg/L pyrene (Hose *et al*., 1984, Table 18).

Benzo[a]pyrene is also phototoxic (Tables 19 and 20). Freshwater aquatic life appears to be protected against the phototoxic reaction of B[a]P at the recommended criterion of 0.01 µg/L, which is two orders of magnitude lower than the concentration which caused 50% mortality in *Daphnia magna* exposed to the PAH in solar UV light (Newsted and Giesy, 1987 - Table 18).

(l) Methylated naphthalenes

The interim (1.0 µg/L) marine water criteria for each of mNA, 1-MNA, 2-MNA, 3-MNA, d-MNA, and t-MNA, are based on the fact that methylated naphthalenes (mono-, di-, or tri-) display similar toxicity to copepods at low levels (Ott *et al*, 1978 - Table 22). The mean value of methylated naphthalenes causing chronic effects (decreased fecundity and reproductive success in females) in copepods was calculated to be 10.4 µg/L (i.e.,

geometric mean of 15.01 µg 2-MNA/L, 8.16 µg d-MNA/L, and 9.27 µg t-MNA/L). A safety factor of 0.1, which is consistent with the B.C. Environment protocol, was applied to obtain the interim criterion.

Due to the lack of data, criteria for the protection of freshwater aquatic life exposed to methylated naphthalenes were not recommended.

6.11.2 Fish and shellfish

The criterion for the carcinogenic B[a]P in the edible tissue of fish and shellfish is based on: (a) its potential to cause cancer in animals, (b) human exposure to the PAH from water and food sources, (c) the risk assessment procedure of the USEPA (PSEP, 1986). The following steps were used in the calculation of the recommended criterion:

1. The general form of the linearized multistage model used by the USEPA for risk assessment is:

$$R(d) = q\ d \quad (1)$$

where R(d) is the excess (over background) lifetime risk of cancer at dose d (mg/kg d), and q is the carcinogenic potency factor. The carcinogenic potency factor for B[a]P has been determined to be 11.5 by the USEPA from the dose-frequency-of-tumor relationship. From the above relationship, at the cancer risk level of 7 : 1000 000[11], the acceptable daily dose for a 70-kg person was calculated to be (7 x 1 000 µg/mg x 70 kg ÷ 1000 000 x 11.5) = 0.043 µg/d.

2. Assuming daily consumption of 1.5 L of drinking water by an individual, the maximum body-B[a]P burden from drinking water at the recommended level of 0.01 µg/L (section 5.3) will be equal to 0.015 µg/d.
3. Assuming fish and shellfish to be the main food source of B[a]P, the acceptable intake from fish and shellfish was calculated to be:

$$0.043\ \mu g/g \text{ (step 1)} - 0.015\ \mu g/g \text{ (step 2)} = 0.028\ \mu g/d \quad (2)$$

Since human consumption of fish/shellfish varies, the recommended criteria, as shown in section 6.10.2, were expressed in terms of safe quantity of fish/shellfish (containing B[a]P) which may be consumed on a regular basis.

6.11.3 Sediment

The recommended sediment criteria are based on equilibrium partitioning of PAHs between interstitial water and sediment. This approach is based on the premise that sediment-associated contaminants are inactive and the toxic fraction of the contaminant is the one associated with interstitial water. This approach was preferred for two reasons: (i) the data on sediment toxicity of PAHs to aquatic organisms are lacking in the literature, (ii) the waterborne PAHs are accumulated more rapidly by aquatic organisms than PAHs associated with sediments (Roesijadi *et al*., 1978). Also, several studies in the literature suggest that the accumulation of PAHs from sediments, when it occurs at all, may be attributed in large part to uptake of PAH desorbed from sediment particles into the interstitial water (Neff, 1979; Landrum and Scavia, 1983). It was, therefore, assumed that the adverse effect of contaminated sediment is mostly due to the PAH concentration in the interstitial water in equilibrium with the sediment.

Assuming that partitioning of a contaminant between sediment and interstitial water is at equilibrium, the USEPA (1989) suggested the following relationship between sediment quality criteria (SQC expressed as µg PAH/kg organic carbon) and water quality criteria (WQC expressed as µg PAH/L):

$$SQC = Koc\ WQC \quad (3)$$

where Koc is the partitioning coefficient for particle organic carbon. Since Koc is about one-half the octanol-water coefficient Kow (from Table 1 where data are available), the above relationship was written as follows:

$$SQC = 0.5\ Kow\ WQC \quad (4)$$

or

$$SQC' = (0.5\ Kow\ WQC) \div 100\ 000 \quad (5)$$

where SQC' is the sediment criterion expressed in µg/g dry weight sediment containing 1% organic carbon. Using equation (5) in conjunction with Kow values from Table 1 and the water quality criteria for long-term effects (excluding criteria for phototoxic effects) recommended in Table 26, the sediment quality criteria for PAHs (i.e., the concentration which should not be exceeded for the protection of aquatic life) in both freshwater and marine environments were derived as shown in Table 27.

The sediment criteria derived using equation (5) were compared with those of the Washington Department of Ecology (the recommended numbers were multiplied by 0.01 to express them on the basis of dry weight of sediment containing 1% organic carbon) and the ER-L values of the National Oceanic and Atmospheric Administration

(NOAA), Seattle (Long and Morgan, 1990 - Table 25). Figure 5 suggests that the criteria derived from equation (5) were either the same or lower, except for acenaphthene and fluorene, than the WDOE criteria. The WDOE criteria are essentially the apparent threshold values (AET) obtained for the Puget Sound area. An AET concentration is the sediment concentration of a given contaminant above which statistically significant biological effects always occur; also, by definition, some effects are expected to occur at concentrations less than AET. Obviously, the criteria based on equation 5 will provide more protection from adverse effects of all PAHs considered in this document, except acenaphthene and fluorene. To protect aquatic biota from the adverse effects of acenaphthene and fluorene, the sediment criteria (based on equation 5) for these PAHs were adjusted downward by factors of 2 (for acenaphthene) and 4 (for fluorene), to make them compatible with the WDOE criteria.

The ER-L values of the NOAA were lower than our criteria for acenaphthene, fluorene, anthracene, and fluoranthene. The comparison with the NOAA values may not be a fair one since the NOAA values were developed from field data. Under these conditions where several contaminants are simultaneously present in the environment which may individually be more toxic or act synergistically with PAHs.

6.12 Application of criteria

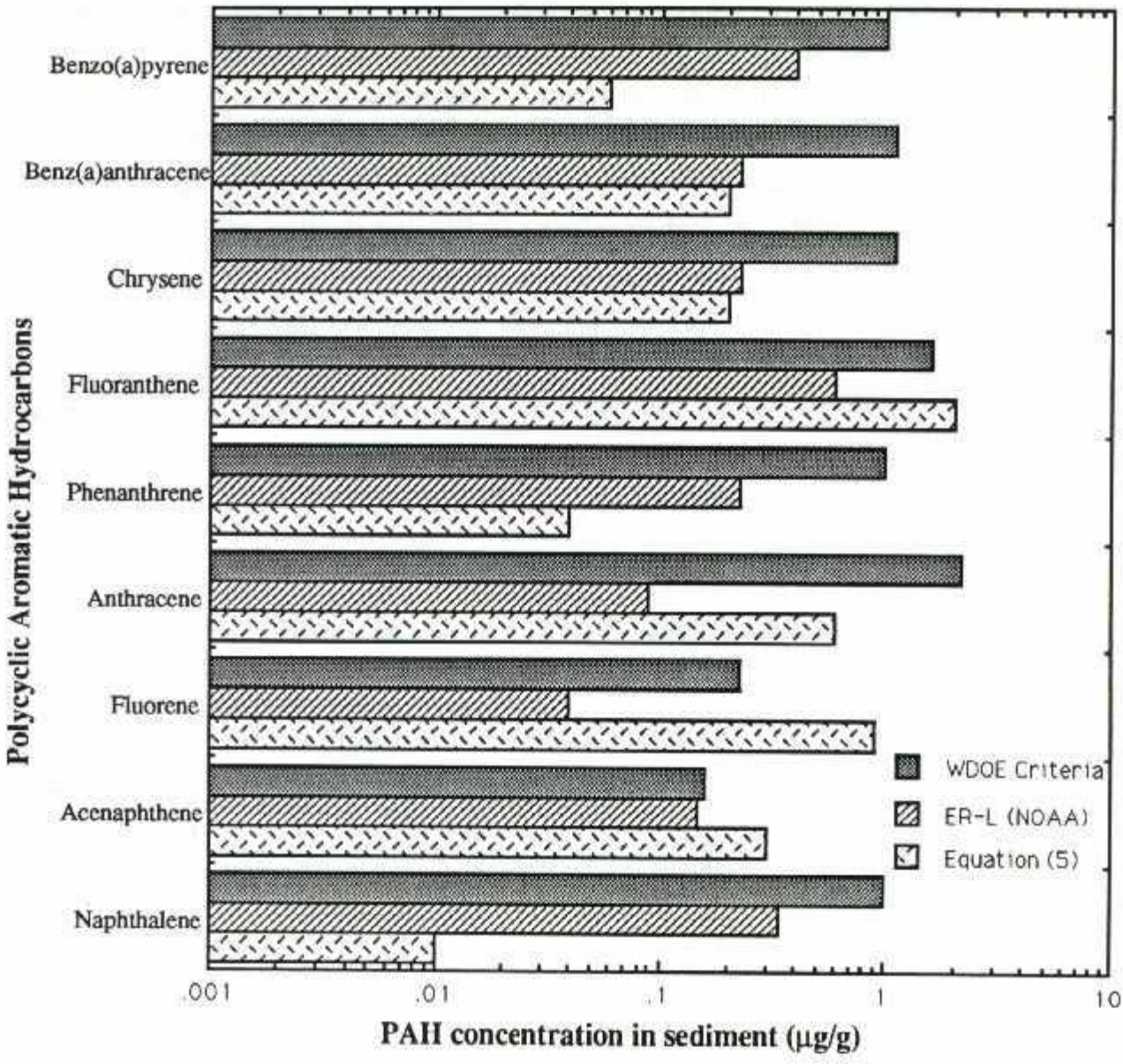
6.12.1 Phototoxic versus long-term criteria

The ecological significance of photo-induced toxicity of PAHs on aquatic environments has not been fully explored. It is, however, evident from data presented in the literature that phototoxicity is relatively (relative to long-term effects in the absence of solar UV radiation) more severe and hazardous to aquatic organisms in clear shallow waters. Juveniles of most fish are found in shallow areas of the littoral zone or on the surface as pelagic larvae and would be extremely vulnerable (Bowling *et al*., 1983). Therefore, it is recommended that where both chronic and phototoxic criteria are provided, the criteria provided for the protection of freshwater aquatic life from phototoxic effects should be given precedence over the criteria to protect against long-term effects of PAHs on aquatic life. If PAH levels exceed the phototoxic criteria, but the aquatic life do not show adverse effects from PAHs introduced into the waterbody by anthropogenic activities, the long-term criteria should be applied to manage and control further deterioration of water quality.

Figure 5

FIGURE 5

Comparison of sediment criteria



6.12.2 PAH levels in smoked fish/shellfish

TABLE 29

B[a]P Concentration in Smoked Fish and Shellfish
(Source: Health and Welfare Canada)

Organism	***B[a]P Conc.*** *(µg/g ww)*	***Organism***	***B[a]P Conc.*** *(µg/g ww)*	***Organism***	***B[a]P Conc.*** *(µg/g ww)*
Haddock1	0.05	Saithe2	<0.10	Oyster2	3.9
Cod1	0.05	Mussle2	1.0	Oyster2	0.4
Herring1	0.05	Mussle2	3.9	Oyster	2.8
Arctic char1	0.05	Mussle2	0.8	Oyster	2.3
Digby chix1	0.05	Mussel (average)	1.5	Oyster	13.3
Sardine2	0.5	Oyster2	7.7	Oyster (average)	2.9
Kippers2	<0.10	Oyster2	1.6		

1 fresh smoked fish; 2 canned smoked fish/shellfish

6.12.3 Analytical limitations

Zenon Environmental Laboratories is currently contracted to provide analytical services to the British Columbia Government. The laboratory has the capability to provide analysis for all PAHs, except acridine, for which water quality and sediment quality criteria are recommended in this document (Table 30). The minimum detectable concentrations for all PAHs are at (e.g., benzo[a]pyrene in water) or below the recommended water and sediment quality criteria.

It is recommended that the capability to measure acridine at a minimum level of 0.3 µg/L in water and 0.1 µg/g dry weight in sediment, and benzo[a]pyrene at 0.001 µg/L in water be developed so that these contaminants can be monitored with confidence at the recommended criteria levels.

Currently, Zenon Environmental Laboratories uses an analytical technique (GC/MS) which has a detection limit of 1.0 ng/g for benzo[a]pyrene in tissue samples. This

minimum detectable concentration is one-half the recommended criterion of 2 ng B[a]P/g wet weight in the the edible tissue of fish and shellfish for moderate consumers (140 g/wk) (section 6.10.2).

Table 30

Recommended Water Quality and Sediment Quality Criteria, and Minimum Detectable PAH concentrations

PAH	***Water (Fresh or Marine)***		***Sediment (Fresh or Marine)***	
	Criteria (µg/L)	***MDC* (µg/L)***	***Criteria (µg/g)***	***MDC* (µg/g)***
Naphthalene	1	0.01	0.01	0.001
Acenaphthene	6	0.01	0.15	0.001
Fluorene	12	0.01	0.2	0.001
Anthracene	4	0.01	0.6	0.001
Phenanthrene	0.3	0.01	0.04	0.001
Acridine	3	not given	1	not given
Fluoranthene	4	0.01	2	0.001
Chrysene	0.1	0.01	0.2	0.001
Benz[a]anthracene	0.1	0.01	0.2	0.001
Benzo[a]pyrene	0.01	0.01	0.06	0.001

* Minimum detectable concentration by Zenon Laboratory

4 The lowest energy triplet state (or LETS) is a condition which is assumed by a PAH molecule excited by the absorption of the appropriate quanta of light, and in which the outer paired electrons of the excited molecule have identical rather than opposing spins.

5 Phosphorescence lifetime (or PLT) is a direct measurement of the radiation dissipation of a molecule from the excited triplet state to the singlet ground state.

6 LT_{50} is defined as exposure time required to yield 50% mortality in organisms exposed to a given contaminant concentration.

[7] A primary study (or the study of primary importance) is one in which tests are carried out in a flow-through environment and the contaminant concentration is measured during the test period. In a study classified as secondary, tests are conducted in a static environment and/or concentration is not measured during the experiment. The criteria apply to both freshwater and marine data sets.

[8] Producing living young instead of eggs from within the body in the manner of nearly all mammals, many reptiles, and a few fishes.

[9] I_{50} is defined as the concentration of the co-contaminant resulting in a 50% reduction in the uptake rate constant.

[10] The toxic potency factor of a PAH is an index of its relative efficacy (which is a unique descriptor of the phototoxic activity of a compound) compared to the one of known phototoxicity.

[11] The risk level of 7: 1000 000 has been used by the Environmental Protection Division, Ministry of Environment, Lands, and Parks, in assessing risk to humans from exposure to toxic substances.

7.0 WILDLIFE

7.1 Effects

The data on PAH toxicity to wildlife (except in laboratory animals such as rats, mice, guinea pigs, etc.) are few. Some pertinent data from the literature are reviewed below.

Patton and Dieter (1980) exposed mallards (*Anas platyrhynchos*) to a diet containing 4 000 µg PAHs/g (mostly as naphthalenes, naphthenes, and phenanthrene) for 7 months. No visible signs of toxicity were evident during the exposure. Although food consumption was not measured, it was believed the toxicant effect was mediated through a decrease in the voluntary intake by the birds because of reduced food palatability.

Hoffman and Gay (1981) studied the embryotoxicity of various PAHs (in a synthetic petroleum mixture) applied to the surface of mallard eggs. 7,12-dimethylbenz[a]anthracene was the most toxic PAH tested. Approximately 0.002

µg/egg (~0.036 µg/kg fresh weight, assuming an average mass of 55 g/egg) of 7,12-dimethylbenz[a]anthracene caused 26% mortality in 18 days. Also, a reduction in the embryonic growth and an increase in the frequency of other anomalies were noted among survivors. Similar results were found with 0.015 µg chrysene/egg. Benzo[a]pyrene (0.002 µg/egg) did not affect mallard survival, but caused a reduction in the embryonic growth and an increased incidence of abnormal survivors.

7.2 Criteria from other jurisdictions

PAH criteria for wildlife were not found in the literature.

7.3 Recommended criteria

PAH criteria for wildlife are not recommended due to the lack of data.

8.0 LIVESTOCK WATER SUPPLY

8.1 Effects

The data on PAH toxicity to livestock animals are few in the literature. Some pertinent data are reviewed below.

Based on an old (1940) study, OMOE (1990) noted that an accidental ingested dose of light sweet crude oil equivalent to 40 mL/kg body weight was lethal to calves in 13 days. Ingestion of kerosene was also lethal to calves over 5 days, but at a lower dose (8 to 12 mL/kg body weight/d). Kerosene contains much less PAH than crude or refined oils; however, no information on the composition of the ingested dose was given by the investigators.

Ellenton (1982) exposed chick embryos to various fractions of Prudhoe Bay crude and fuel oil No. 2. The greatest teratogenicity was associated with the fraction containing 2-3 ringed PAHs (e.g., alkylated naphthalenes, anthracenes, phenanthrenes, fluorenes, and biphenyls) applied at the level of 2.5 mg-equivalent; 71% of the embryos treated with this fraction were abnormal. The fraction containing 4-5 ringed PAHs included alkylated chrysenes, B[a]ANTH, triphenylene, phenylanthracene, B[a]P, perylenes, and benzo[a]perylene. No significant increase in teratogenic effects was observed with this fraction even at the maximum application of 5 mg-equivalent.

No literature on the effects of individual PAHs on livestock through ingestion from diet or drinking water was found.

8.2 Criteria from other jurisdictions

PAH criteria for livestock watering were not found in the literature.

8.3 Recommended criteria

PAH criteria for livestock water supplies are not recommended due to the lack of sufficient information.

9.0 IRRIGATION

9.1 Effects

The toxicity of PAHs to natural and cultivated plants is not well addressed in the literature. Much of the available data were gathered on excised sections of plants and germinated seeds and, therefore, were of limited use.

Deubert *et al* (1979) soaked corn and wheat seeds in a 0.5 - 20 µg/L B[a]P solution. The exposure to B[a]P at the low concentration (0.5 µg/L) appeared to stimulate corn root growth; shoot growth was unaffected for both corn and wheat. In another experiment, acenaphthene (15 400 µg/L) was observed to slow down the elongation rate in the young root tips from maize, while anthracene (17 800 µg/L) did not have an effect (OMOE, 1990).

There is abundant literature on accumulation of PAHs in higher plants grown on PAH-enriched soils. However, it suffers from a lack of detail on PAH accumulated simultaneously from other sources (e.g., air, volatilized fraction in soil, etc.); i.e., the uptake of soil PAH via plant roots could not be addressed adequately from the data.

Edward (1983) reported that PAH uptake rates by plants are dependent on: (a) PAH concentration in the environment, (b) plant species, (c) the nature of plant growth substrate (e.g., soil, water, etc.), (d) PAH solubility, (e) PAH phase (vapour or particulate), and (f) PAH molecular weight.

Edward *et al* (1982) noted that 14C-anthracene uptake by soybeans from nutrient solution was directly proportional to the anthracene concentration in the solution. However, anthracene in soil was quite unavailable for absorption by soybean roots, in comparison to its availability in hydroponic solution (Edward *et al*, 1982). The tendency of a soil to bind PAHs is related to its organic matter content and cation exchange capacity. Field experiments with several agricultural crops, where the soil was treated with fresh compost containing a number of PAHs, suggested little or no uptake of PAHs by plant roots (Ellwardt, 1977).

Concentrations of PAHs in vegetation are generally much less than concentrations in the soil. Wang and Meresz (1981) analyzed onions, beets, tomatoes, and soil for 17 PAHs, and found that the vegetation/soil concentration ratios ranged from 0.0001 to 0.085 for B[a]P and 0.001 to 0.183 for total PAHs. They also found that most of the PAH contamination was in the peels.

Negishi *et al* (1987) suggested that soybeans metabolized B[a]P using a mixed function oxidase similar to mammalian and eukaryotic[12] systems. Their findings, however, were in contrast to Trenck and Sandermann (1980) who, using higher plant cell cultures, concluded that plants did not metabolize B[a]P similarly to mammalian systems.

In controlled environmental conditions, Grimmer and Duvel (1970) noted that vegetable crops were incapable of *de novo* (afresh) synthesis of PAH; however, opposite conclusions were drawn for algae by several other investigators (Knutzen and Sortland, 1982).

Wagner and Wagner-Hering (1971) reported phytotoxic effects for polycyclic aromatic hydrocarbons.. These investigators found that wheat (whole plant) and barley (straw) yields were reduced by about 10% when exposed to 3,4-benzfluoranthene in soils at the rate of 6.3 µg PAH/g dw(soil) and 9.4 µg PAH/g dw(soil), respectively. No other study related to phytotoxic effects of PAHs to terrestrial plants was found in the literature. Phytotoxic effects are apparently not severe in higher, terrestrial plants up to B[a]P soil concentrations of 18 µg PAH/g dw (Sims and Overcash, 1983).

9.2 Criteria from other jurisdictions

PAH criteria for irrigation waters were not found in the literature.

9.3 Recommended criteria

PAH criteria for irrigation waters are not recommended due to the lack of sufficient information.

12 Eukaryote or eucaryote: an organism composed of one or more cells with visibly evident nuclei.

10.0 RESEARCH AND DEVELOPMENT NEEDS

Several research and development needs were identified during the preparation of this document:

1. Several high molecular weight PAHs have been shown to be mutagenic, carcinogenic, and inducers of tumors in laboratory animals exposed to high doses. Whether such conditions will also occur at low exposure levels (i.e., equivalent to those one may be exposed to in the environment), can not be determined from the current literature. More work is needed on low level effects of PAHs on animals, including humans.

2. Several PAHs have been shown to be phototoxic when aquatic organisms contaminated with the organic compounds are exposed to solar ultraviolet radiation. The photo-induced toxicity of the PAHs occurs at much lower concentrations than the laboratory chronic tests (generally performed in the absence of ultraviolet radiation) might suggest. Research is needed to identify the existence, and to assess the impact, of photo-induced toxicity in natural aquatic environments. Confirmation is also required regarding photo-induced toxic reactions of those PAHs which have been designated to be phototoxic based on empirical modelling techniques, but have not been tested in the laboratory or in the field.

3. The data on long-term effects (both phototoxic and non-phototoxic) on freshwater and marine organisms exposed to waters and sediments contaminated with PAHs are lacking in the literature. As a result, water quality or sediment quality criteria for several PAHs could not be recommended in this document, while for other PAHs the recommended criteria are interim. The lack of data on effects of PAHs on the other water uses (e.g., irrigation, livestock watering, wildlife, and contact recreation) was also recognized.

4. Field measurements have linked chronic effects (e.g., carcinogenic, mutagenic, and tumor induction, etc.) suffered by aquatic organisms to PAHs found in the environment (e.g., sediments). Despite such associations, definitive information does not exist about which environmental chemicals or group of chemicals may be responsible for the

observed effects. Clearly, PAHs and other environmental chemicals may not act individually, but through synergistic-antagonistic interactions. Interactions among PAHs and other equally toxic contaminants such as PCBs need to be addressed to develop definitive (not interim) criteria for PAHs.
5. Data on ambient PAH concentrations in British Columbia waters are non-existent. Good water quality data are required to assess the state of the environment in British Columbia with respects to PAHs.

6. Minimum detectable concentrations need to be improved for some PAHs as indicated in section 6.11.3.

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