Canadian Environmental Protection Act

PRIORITY SUBSTANCES LIST ASSESSMENT REPORT

POLYCYCLIC AROMATIC HYDROCARBONS

Government of Canada
Environment Canada
Health Canada

Also available in French under the title:
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Synopsis

Polycyclic aromatic hydrocarbons (PAHs) are emitted into the Canadian environment from both natural and anthropogenic sources. Forest fires, which release approximately 2000 tonnes of PAHs per year, are the single most important natural source of PAHs in Canada. However, since releases from that source are generally widely separated in time and space across the country, they do not result in continuous exposure in any specific area. Anthropogenic sources are numerous and result in emissions of PAHs into all environmental compartments. The greatest anthropogenic sources of PAHs released to the atmosphere are aluminum smelters [925 tonnes/year (t/yr)], with most of these emissions being released from smelters that use the Horizontal Stud Söderberg process. Major sources of PAHs to the aquatic and soil environments include creosote-treated products (up to 2000 t/yr), spills of petroleum products (76 t/yr), metallurgical and coking plants (4 t/yr), and deposition of atmospheric PAHs (amount unknown).

With the exception of some of the lighter compounds, which volatilize from water or soil, PAHs are relatively non-volatile and of low solubility in water. In air, soil, and water, PAHs are mostly adsorbed to particulate matter, on which they are transported and can be degraded slowly. Since degradation is very slow in sediments, this medium is the major environmental sink for PAHs.

Polycyclic aromatic hydrocarbons have been measured in water from railway and utility ditches at concentrations higher than those recognized to cause harmful effects to biota under laboratory conditions. Concentrations of PAHs in surface waters elsewhere in the country are orders of magnitude lower, and are below concentrations that cause harmful effects to aquatic biota.

At several sites in Canada, including the tidal flats of Muggagh Creek in the vicinity of the tar ponds in Sydney Harbour, Nova Scotia, and near a former oil gasification plant at Kettle Creek, Port Stanley, Ontario, populations of aquatic organisms have been adversely affected by contamination by PAHs. Moreover, PAHs in sediments from Hamilton Harbour, Ontario, have been associated with mortality of sensitive aquatic invertebrates under laboratory conditions.

Under laboratory conditions, neoplastic effects such as liver tumours in aquatic organisms have been associated with exposure to PAHs. Field evidence also supports this association. In Vancouver harbour, neoplastic liver lesions were observed in up to 75% of the English sole caught in areas where sediments are highly contaminated by PAHs.

Polycyclic aromatic hydrocarbons generally do not absorb light of wavelengths critical to global warming. Unlike substances associated with depletion of stratospheric ozone, they are non-halogenated compounds of low to moderate persistence in the atmosphere. Given these properties and the low steady-state concentrations of PAHs in the atmosphere, they are not considered to contribute significantly to stratospheric ozone depletion, global warming, or ground-level ozone formation.

Owing principally to the limitations of the available data base, it was necessary to focus the human health assessment primarily on a small number of PAHs and consider exposure from air only.

Based primarily on the results of carcinogenicity bioassays in which PAHs have been administered to experimental animals by inhalation (benzo[*a*]pyrene only) and dermal application, and on supporting data, the five PAHs considered in the human health assessment (benzo[*a*]pyrene, benzo[*b*]fluoranthene, benzo[*j*]fluoranthene, benzo[*k*]fluoranthene, and indeno[*1,2,3-cd*]pyrene) have been classified as "Probably Carcinogenic to Humans" (i.e., substances for which there is believed to be some chance of adverse effects at any level of exposure). For such substances, estimated exposure is compared to quantitative estimates of cancer potency to characterize risk and provide guidance for further action, i.e., analysis of options to reduce exposure. Based on consideration solely of the potential effects of PAHs on human health by two limited approaches, the priority for analysis of options to reduce exposure would be moderate to high.

Based on these considerations, it has been concluded that polycyclic aromatic hydrocarbons are entering the environment in a quantity or concentration or under conditions that may have harmful effects on the environment. Polycyclic aromatic hydrocarbons are not considered to constitute a danger to the environment on which human life depends. The PAHs benzo[*a*]pyrene, benzo[*b*]fluoranthene, benzo[*j*]fluoranthene, benzo[*k*]fluoranthene, and indeno[*1,2,3-cd*]pyrene may constitute a danger in Canada to human life or health.

1.0 Introduction

The *Canadian Environmental Protection Act* (CEPA) requires the Minister of the Environment and the Minister of Health to prepare and publish a Priority Substances List that identifies substances, including chemicals, groups of chemicals, effluents, and wastes, that may be harmful to the environment or constitute a danger to human health. The Act also requires both Ministers to assess these substances and determine whether they are "toxic" as defined under Section 11 of the Act which states:

> "...a substance is toxic if it is entering or may enter the environment in a quantity or concentration or under conditions
>
> (a) having or that may have an immediate or long-term harmful effect on the environment;
>
> (b) constituting or that may constitute a danger to the environment on which human life depends; or
>
> (c) constituting or that may constitute a danger in Canada to human life or health."

Substances that are assessed as "toxic" as defined under Section 11 may be placed on Schedule I of CEPA. Consideration can then be given to developing regulations, guidelines, or codes of practice to control any aspect of these substances' life cycle, from the research and development stage through manufacture, use, storage, transport, and ultimate disposal.

The assessment of whether polycyclic aromatic hydrocarbons (PAHs) are "toxic", as defined under CEPA, was based on the determination of whether they **enter** or are likely to enter the Canadian environment in concentrations or quantities or under conditions that could lead to **exposure** of humans or other biota at levels that could cause adverse **effects**.

The term polycyclic aromatic hydrocarbons refers to the compounds made up of carbon and hydrogen atoms grouped into rings containing five or six carbon atoms. They are called "PAH derivatives" when an alkyl or other radical is introduced to the ring, and "heterocyclic aromatic compounds" (HACs) when any one carbon atom in a ring is replaced by a nitrogen, oxygen, or sulphur atom. Heterocyclic aromatic compounds and PAH derivatives were not considered in this report since little is known about their presence in the Canadian environment.

Polycyclic aromatic hydrocarbons constitute a class of chemical products that include about 100 individual compounds. Based on available data (NRCC, 1983; Bjørseth, 1983; Bjørseth and Ramdhal, 1985; LEI, 1990; EAG, 1990), a preliminary list was developed that included individual compounds representative of the diversity of chemical, environmental, and toxicological properties of PAHs. From this list, 13 compounds were selected for assessment. Sufficient data were available to assess

presence in the Canadian environment and the effects on biota for 9 compounds: acenaphthene, anthracene, benz[*a*]anthracene, benzo[*a*]pyrene (B[*a*]P), fluoranthene, fluorene, naphthalene, phenanthrene, and pyrene.

Owing principally to limitations of available data, it was necessary to focus the human health assessment primarily on a small number of compounds for which the available data base would be adequate for evaluation of effects. In addition, carcinogenicity was considered to be the critical effect, since the doses of PAHs that induce non-neoplastic effects in experimental animals are considerably greater than those associated with increases in tumour incidence. The PAHs selected for human health assessment were benzo[*a*]pyrene (B[*a*]P), benzo[*b*]fluoranthene (B[*b*]F), benzo[*j*]fluoranthene ([B[*j*]F), benzo[*k*]fluoranthene (B[*k*]F), and indeno[*1,2,3-cd*]pyrene (IND).

These compounds were selected for the following reasons. (a) They have been classified in Groups 1 or 2 ("Carcinogenic to Humans" or "Probably Carcinogenic to Humans") of the classification scheme for this endpoint developed for the assessment of "toxic" under Paragraph 11(c) of CEPA (EHD, 1994) on the basis of data reviewed by the International Agency for Research on Cancer (IARC) (IARC, 1983). (b) Available data were considered sufficient to develop a preliminary estimate of potency for the most sensitive endpoint (i.e., carcinogenicity) in a target organ (i.e., the lung) relevant to one of the principal media of exposure in the general environment for which sufficient information was available to estimate exposure (i.e., air). Available data were inadequate to develop estimates of exposure for, or carcinogenic potency of, a broad range of PAHs in the gastrointestinal tract following ingestion.

This focused approach to assessment of potential effects on human health was necessary principally because of the limited available data. Because of its limited scope, it should be considered as an interim approach only. For example, the five PAHs addressed here represent less than 25% (0.1 to 23.5%) of the 13 PAHs determined to be present at various locations in a recent survey of ambient air in Canada (Dann, 1992a). In addition, owing to the limitations of the data base, exposure from sources other than air has not been considered. Upon acquisition of additional data as suggested in Section 4.0, "Recommendations for Research", of this report, it may be possible to develop a more comprehensive approach to assessment.

Polycyclic aromatic hydrocarbons are a major component of creosote and, to a lesser extent, of waste crankcase oils. Creosote-impregnated waste materials and waste crankcase oils have been assessed separately as part of the CEPA Priority Substances List assessment program. These substances are therefore not considered directly in this assessment.

Creosote as a pesticide was not assessed in this report. The pesticide creosote is subject to the provisions of the *Pest Control Products Act*, and its regulatory status as a heavy-duty wood preservative is currently being re-evaluated (Agriculture Canada, 1992). As part of the pesticide re-evaluation process, Environment Canada and Health

Canada will assess the potential effects on the environment and on human health resulting from these pesticidal uses of creosote.

Information relevant to the entry and environmental exposure and effects of PAHs was identified from reviews (Slooff *et al.*, 1989; EAG, 1990; NRCC, 1983; NRC, 1983; Bjørseth, 1983; Bjørseth and Ramdhal, 1985; U.S. EPA, 1987; Ortech International, 1990; CCME, 1989; 1991), searches of data bases [Analytical Abstracts, EMBASE, Enviroline, POLLUTION ABSTRACT, International Register of Potentially Toxic Chemicals (IRPTC)], and from research reports appearing in scientific journals, and university and government publications available at the end of 1992. Works published since 1992 were considered when appropriate. In addition, to generate data essential for this assessment, studies were done by Environment Canada to characterize atmospheric concentrations of PAHs. Although much of the research on PAHs has been conducted outside of Canada, Canadian data on sources, use patterns, fate, and effects of PAHs on the environment were emphasized where available.

Data relevant to the assessment of whether PAHs are "toxic" to the environment obtained after the completion of the environmental sections of this report (i.e., May 1993) were not considered for inclusion. Similarly, data relevant to the assessment of whether PAHs are "toxic" to human health obtained after the completion of these sections of the report (i.e., July 1993) have not been incorporated.

Although review articles were consulted where considered appropriate, original studies that form the basis for the determination of "toxic" under CEPA (entry, and environmental exposure and effects) were critically evaluated by the following staff of Environment Canada. Original studies relevant to the estimation of carcinogenic potency for PAHs considered in this assessment have been critically evaluated by the following staff of Health Canada.

Environment Canada	Health Canada
R. Chénier	P.K.L. Chan
C. Fortin	M.E. Meek
A. Germain	
C. Gonthier	
B. Nadon	
F. Perron	
S. Ringuette	
R. van Coillie	

Quantitative estimates of carcinogenic potency were provided by S. Bartlett and M. Walker of Health Canada.

In this report, a synopsis that will appear in the *Canada Gazette* is presented. An extended summary of the technical information that is critical to the assessment is presented in Section 2.0. This information is presented in greater detail in supporting

documentation that is available upon request. The assessment of whether PAHs are "toxic" as defined in CEPA is presented in Section 3.0.

As part of the review and approvals process established by Environment Canada for its contributions to Priority Substances List (PSL) assessments, sections of the supporting documentation dealing with presence in the Canadian environment were reviewed externally by J.F. Payne (Fisheries and Oceans Canada); sections dealing with sources and releases were reviewed by M. Lalonde (Alcan Ltd), G. Éthier and N. Sherwin (Canadian Petroleum Products Institute), and by representatives of provincial ministries of the environment, including D. Bezak (Manitoba), C. Bisco (Prince Edward Island), P. Blagden (Newfoundland), G. Chow (Saskatchewan), G. Houle (Quebec), L. Johnston (Northwest Territories), J. Knight (New Brunswick), C.S. Liu (Alberta), C. Mackinnon (Nova Scotia), T. Wakelin (British Columbia), and D. Yap (Ontario). Environmental components of the Assessment Report were reviewed by P. Hodson (National Water Research Institute), L.S. McCarty (L.S. McCarty Scientific Research and Consulting), and A.J. Nantel (*Centre de toxicologie du Québec*). The draft health-related sections of the supporting documentation and Assessment Report were approved by the Standards and Guidelines Rulings Committee of the Bureau of Chemical Hazards of Health Canada. The final Assessment Report was reviewed and approved by the Environment Canada/Health Canada CEPA Management Committee.

Copies of this Assessment Report and the unpublished supporting documentation are available upon request from:

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2.0 Summary of Information Critical to the Assessment of "Toxic"

2.1 Identity, Properties, Production, and Uses

Polycyclic aromatic hydrocarbons (PAHs) are organic substances made up of carbon and hydrogen atoms grouped into at least two condensed aromatic ring structures. These are divided into two categories: low molecular weight compounds composed of fewer than four rings and high molecular weight compounds of four or more rings. "PAH derivatives" include PAHs having an alkyl or other radical attached to a ring; "heterocyclic aromatic compounds" (HACs) include PAHs having any one carbon atom in a ring replaced by a nitrogen, oxygen, or sulphur atom. This report addresses only unsubstituted PAHs, containing only carbon and hydrogen atoms (Figure 1).

Pure PAHs are usually coloured, crystalline solids at ambient temperature. The physical properties of PAHs vary with their molecular weight and structure (Table 1). Except for naphthalene, they have very low to low water solubilities, and low to moderately high vapour pressures. Their octanol-water partition coefficients (K_{OW}) are relatively high, indicating a relatively high potential for adsorption to suspended particulates in the air and in water, and for bioconcentration in organisms (NRCC, 1983; Slooff *et al.*, 1989). Generally, PAHs only weakly absorb light of infrared wavelengths between 7 and 14 μm (Sadtler Research, 1982), the wavelengths usually absorbed by chemicals involved in global warming (Ramanathan, 1985).

Polycyclic aromatic hydrocarbons are present in the environment as complex mixtures that are difficult to characterize and measure. They are generally analyzed using gas chromatography coupled with mass spectrometry (GC-MS), or by using high pressure liquid chromatography (HPLC) with ultraviolet (UV) and fluorescence detectors (U.S. EPA, 1982; Bjørseth and Ramdhal, 1985; Slooff *et al.*, 1989; Tardif and Chiu, 1992).

Polycyclic aromatic hydrocarbons are largely produced through the combustion or pyrolysis of organic matter either naturally or through human activity; very small amounts may be produced by diagenesis or biosynthesis (NRCC, 1983). Factors such as the type and quantity of fuel, the temperature and duration of combustion, and the availability of oxygen determine the nature and extent of PAH formation (NRCC, 1983).

Some PAHs are used commercially, but few data are available on the volumes used. Based on limited voluntary reporting of commercial activities for the Domestic Substances List, the following PAHs were reported as being produced or manufactured in Canada in 1986: naphthalene [more than 1000 tonnes (t) produced, 10 to 100 t imported]; anthracene (0.1 to 1 t produced, 1 to 10 t imported); fluorene (0.1 to 1 t produced, <1 t imported); and acenaphthene, benzo[*a*]pyrene, chrysene, and pyrene (<0.1 t imported for each) (Environment Canada, 1992). According to SRI (1985), roughly 70% of the naphthalene produced in the United States and Japan is used in the production of phthalic acid, and this is the expected major use of naphthalene in Canada.

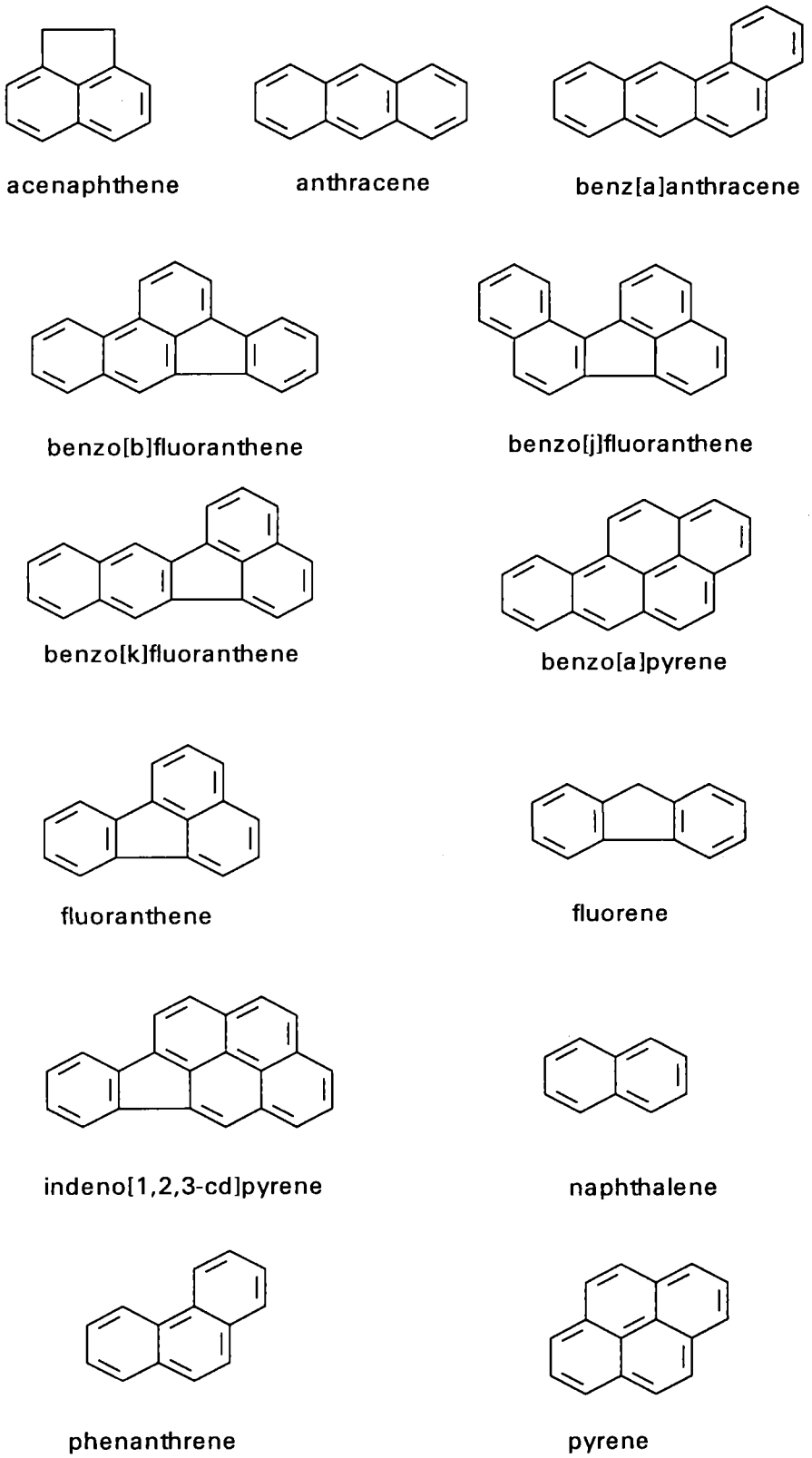

Figure 1 **Chemical Structures of Polycyclic Aromatic Hydrocarbons**

Table 1 Physical Properties of Polycyclic Aromatic Hydrocarbons

Compound (C.A.S. N°)	Molecular weight	log K_{ow}	Water solubility at 25°C (mg/L)	Melting point (°C)	Vapour pressure at 25°C (mPa)
naphthalene (91-20-3) [1]	128.16	3.5	31.7	80.5	11 960
acenaphthene (83-32-9) [1]	154.21	4.33	3.42	95	594
fluorene (86-73-7) [1]	166	4.18	1.98	116.5	94.7
phenanthrene (85-01-8) [1]	178.24	4.5	1.29	101	90.7
anthracene (120-12-7) [1]	178.24	4.5	0.045	216	25
pyrene (129-00-0) [1]	202.26	4.9	0.135	156	91.3×10^{-6}
fluoranthene (206-44-0) [1]	202.26	5.1	0.26	111	1328
benz[*a*]anthracene (56-66-3) [1]	228	5.6	0.0057	162	14.7×10^{-3}
benzo[*a*]pyrene (50-32-8) [1,2]	252.32	6.0	0.0038	179	0.37×10^{-6}
benzo[*b*]fluoranthene (205-99-2) [2]	252.32	6.06	0.014	168	0.13×10^{-5} to 0.133 at 20°C
benzo[*j*]fluoranthene (205-82-3) [2]	252.32			166	
benzo[*k*]fluoranthene (207-08-9) [2]	252.32	6.06	0.0043	217	2.8×10^{-9}
indeno[*1,2,3-cd*]pyrene (193-39-5) [2]	276	6.4	0.00053	164	1.3×10^{-5}

[1] compounds addressed in the assessment of effects on the environment
[2] compounds addressed in the assessment of effects on human health

References: ATSDR, 1990b; Merck Index, 1989; Slooff *et al.*, 1989; CRC, 1988; NRCC, 1983; SRI, 1980; Santodonato *et al.*, 1979, in Smith, 1984; Mackay and Shiu, 1977.

Creosote is a complex and variable mixture of compounds distilled from coal tar. Seventeen PAHs, including the 13 compounds addressed in this report, account for about 63% of the creosote mixture (Environment Canada, 1988). Although Canadian production of creosote has been as high as about 45 $\times 10^6$ L/yr at the end of the 1940s, production has dropped to 12 to 14 $\times 10^6$ L (20 000 t) in 1990 (Envirochem, 1991). Creosote is used mainly as a wood preservative for treating railway ties and marine and freshwater pilings. Transmission and telephone poles have not been treated with creosote in Canada since the mid-1970s.

Coal derivatives and other petroleum products contain varying concentrations of PAHs. The concentration of PAHs (14 compounds, including 6 of the 13 considered in this report) in unleaded gasoline was determined to be 0.0054% (Westerholm *et al.*, 1988). On the basis of reported use of gasoline in Canada in 1989 [35 000 ML (megalitres); (Statistics Canada, 1989)], it is estimated that approximately 1500 t of PAHs are present in the gasoline supply, annually.

2.2 Entry into the Environment

Polycyclic aromatic hydrocarbons may be released into the environment through natural phenomena such as forest fires, volcanic eruptions, diagenesis, and biosynthesis. Altlhough PAHs are naturally present in coal derivatives and petroleum (NRCC, 1983), human activities are considered to be a major source of release of PAHs to the environment (Neff, 1979; NRC, 1983; NRCC, 1983; Bjørseth and Ramdhal, 1985;

Slooff *et al.*, 1989). The distribution and magnitude of certain emissions of PAHs are related to human population density (residential heating, transportation); however, others depend on the availability of power (aluminum smelters) or on the presence of natural resources [open air fires and agricultural burning, sawmill residue incinerators (teepee burners)].

Estimates for atmospheric emissions of PAHs in Canada were based on point and diffuse sources from 45 source-sectors (LGL, 1993). Estimated emissions of PAHs from Canadian sources for 1990 are outlined in Table 2. Forest fires represented the single largest source of PAHs to the environment, releasing about 2010 t of PAHs into the atmosphere, or 47% of the total atmospheric emissions inventoried. The aluminum smelting industry was the second largest source of atmospheric emissions of PAHs, accounting for 21% (925 t). In Canada, there are three processes used in aluminum smelting, all using coal tar pitch in the reduction of alumina: the Horizontal Stud Söderberg (HSS) process, which is the oldest one, the Vertical Stud Söderberg (VSS) process, and the pre-baked anode (PB) process. Through plant modernization programs, the Canadian aluminum smelting industry reduced HSS-related PAH emissions by 53% from 1983 to 1990. Nevertheless, although the HSS process accounted for only 20% of the total Canadian production of aluminum in 1990, it accounted for 75% of the PAHs emitted by the aluminum industry (LGL, 1993).

Other important sources of PAHs to ambient air include emissions from: residential wood heating (474 t/yr); agricultural burning and open air fires (358 t/yr); the incineration of wood residues by saw mills in teepee burners (249 t/yr); and transportation (201 t/yr).

The sources of PAHs that enter water and soil are varied and include: dispersion from creosoted materials (Wan, 1991; 1993); accidental oil spills; precipitation and atmospheric deposition; industrial processes (creosote, coal tar, asphalt, land-farming) (AMAI, 1986a;b; RDRC, 1987; Tecsult, 1989; Vandermeulen, 1989); municipal effluents; and disposal (burial) of wastes containing PAHs (Jackson *et al.*, 1985; van Coillie *et al.*, 1990). Polycyclic aromatic hydrocarbons can also reach groundwater and fresh and marine surface water by leaching through soil and by surface run-off (Wakeham *et al.*, 1980; Slooff *et al.*, 1989; Wan, 1991; LGL, 1993).

An estimate of the quantity of PAHs discharged to water and soil from creosote-treated wood products has been attempted based on the PAH content in creosote, the volume of treated wood in use, the retention rates of the compounds for different species of wood, and an estimated 20% release or loss of compounds during the time the treated wood was in service (40 years for pilings, 50 years for railway ties). According to these calculations, creosote-related PAH releases to soil and water could be up to 2000 t/yr (LGL, 1993).

Spills of petroleum hydrocarbons result in 76 t/yr of PAHs being released into the Canadian environment. About 88% of the total number of spills occur on land and 12% on water (LGL, 1993).

Table 2 Annual Atmospheric Emissions of Polycyclic Aromatic Hydrocarbons in Canada During 1990 (LGL, 1993)

Sources	PAH releases	
	tonnes	%
Anthropogenic Sources		
Industrial Processes		
Aluminum plants	925	21
Metallurgical (including ferro-alloy)	19.5	0.4
Coke production	12.8	0.3
Asphalt production	2.5	0.1
Petroleum refineries	0.1	< 0.1
Combustion Sources		
Residential Heating		
Wood	474	11.0
Others	29	0.7
Open air fires/agricultural burning	358	8.3
Incineration		
Teepee burners	249	5.8
Municipal (with sludges)	1.3	< 0.1
Industrial	1.1	< 0.1
Transportation		
Diesel	155	3.6
Gasoline	45	1
Other	1.2	< 0.1
Thermal Power Plants	11.3	0.3
Industrial Combustion		
Wood	5.7	< 0.1
Other	10.2	0.2
Commercial and Institutional Heating	2.7	0.1
Cigarettes	0.2	< 0.1
Natural Sources		
Forest Fires	2010	47
Total	4314	100

The metallurgical sector (metals and coking plants) released about 3.9 t/yr of PAHs into water in 1990 (LGL, 1993). This total does not include run-off from a tar pond at a steel plant in Sydney, Nova Scotia, that released close to 0.8 t/yr of PAHs into the aquatic environment, based on estimated emissions in 1989 (Lane *et al.*, 1990).

Atmospheric deposition has been estimated to be the main source of PAHs to soils and sediments (Slooff *et al.*, 1989; Christensen and Zhang, 1993). Deposition rates of PAHs to terrestrial and aquatic surfaces in the north-eastern United States have been estimated at 0.8 to 3 ng/cm^2 per year in non-urban areas and up to 35 ng/cm^2 per year in sites closer to urban areas (Gschwend and Hites, 1981; Hites and Gschwend, 1982). The magnitude of the atmospheric deposition of PAHs in Canada resulting from the long-range transport from foreign sources cannot be assessed owing to a lack of information. A total of 484 t/yr of PAHs was estimated to enter the Great Lakes as a result of dry deposition of PAHs from sources in Canada and the United States (Eisenreich *et al.*, 1981, cited in NRCC, 1983).

2.3 Exposure-related Information

2.3.1 *Fate*

With the exception of some of the lighter compounds that volatilize from water or soil, PAHs are relatively non-volatile and of low solubility in water. In the atmosphere, they are mostly found adsorbed to particulate matter that can be removed by wet or dry deposition onto water or soil. Polycyclic aromatic hydrocarbons released to soil will adsorb to particulate matter and will slowly be degraded by microbial activity or transported adsorbed to soil particles by surface run-off. In aquatic systems, PAHs generally adsorb to suspended matter or sediments where they persist. Thus, although most PAHs are emitted to the atmosphere, sediments are the major environmental sink for these compounds.

The atmosphere is the main medium of transport for PAHs (Suess, 1976; Bjørseth *et al.*, 1979). Polycyclic aromatic hydrocarbons in the atmosphere are primarily associated with suspended particulates, although they are also present in the vapour phase (Pupp *et al.*, 1974; Pierce and Katz, 1975; Miguel and Friedlander, 1978; van Vaeck and van Cauwenberghe, 1978; Neff, 1979; NRCC, 1983; Bjørseth and Ramdhal, 1985; Slooff *et al.*, 1989). Atmospheric particulates and adsorbed PAHs can be transported over long distances (Lunde and Bjørseth, 1977; Neff, 1979; NRCC, 1983) before they are removed from the atmosphere either through dry or wet deposition to soil or water, or through chemical transformation (Neff, 1979; NRCC, 1983; Van Noort and Wondergem, 1985; Ligocki, *et al.*, 1985). In addition to direct deposition to soils, PAHs can be deposited onto or absorbed by plants, from which they can be washed by rain, oxidized, or deposited into soil as a result of plant decay (Eisler, 1987).

Ozone-induced oxidation and hydroxylation are the two most important mechanisms by which PAHs are transformed in the atmosphere; both of these reactions are activated by sunlight (Lyman *et al.*, 1982; NRCC, 1983; Slooff *et al.*, 1989). The photo-oxidation half-lives in air for different PAHs vary from 0.4 to 68.1 hours;

photolysis half-lives vary from 0.37 to 25 hours, excluding the long half-life for naphthalene (1704 to 13 200 hours) (U.S. EPA, 1990; Slooff *et al.*, 1989). These chemical transformations are affected by several factors, including the nature of the particles to which the atmospheric PAHs are adsorbed (Korfmacher *et al.*, 1980; NRCC, 1983; Behymer and Hites, 1988) and the quantity of PAHs adsorbed to the particulate matter (Kamens *et al.*, 1988; Slooff *et al.*, 1989). Polycyclic aromatic hydrocarbons are more persistent when they are bound to particulates with a high organic carbon content and when present in large quantities on the particulates. Minor transformation pathways for PAHs include reactions with nitrogen oxides (NO_X) and sulphur dioxide (SO_2). Such reactions, however, occur mostly at the points of emission where temperatures vary between 100 and 200°C (Atkinson *et al.*, 1979).

As in the atmosphere, PAHs in the water column are generally associated with particulates (Harrison *et al.*, 1975; Wakeham *et al.*, 1980; Germain and Langlois, 1988). Volatilization, photolysis, hydrolysis, biodegradation, and adsorption to particulate matter followed by sedimentation are the main processes governing the fate of PAHs in water (NRCC, 1983; Eisler, 1987, Slooff *et al.*, 1989). The rate of volatilization depends on weather conditions, movement of water, and the molecular weight of the compounds (NRCC, 1983; Slooff *et al.*, 1989). Polycyclic aromatic hydrocarbons of low molecular weight may volatilize from water, as indicated by the volatilization half-lives of naphthalene (0.4 to 3.2 hours; Slooff *et al.*, 1989; Southworth, 1979) and anthracene (17 hours; Southworth, 1979). A high molecular weight PAH such as pyrene, however, has a volatilization half-life ranging from 115 hours to 3.2 years (Southworth, 1979; Lyman *et al.*, 1982). Many of the PAHs in oil spilled on water volatilize (NRCC, 1983). Polycyclic aromatic hydrocarbons can be biodegraded in water. Half-lives have been estimated to range from 0.5 to 20 days for naphthalene and from 0.6 to 5.2 years for pyrene under aerobic conditions (U.S. EPA, 1990). Photo-oxidation in water also occurs, with estimated half-lives of 8.6 days to 1.2 years for B[*a*]P (Smith *et al.*, 1978) and 0.1 to 4.4 years for anthracene (Radding *et al.*, 1976). For most PAHs in the water column, sedimentation constitutes the primary removal mechanism (NRCC, 1983).

Sediments are the final environmental sink for PAHs (Payne *et al.*, 1988; Vandermeulen, 1989) where they persist and transform very slowly. Biodegradation half-lives of sediment-bound PAHs range from 0.3 to 129 days for naphthalene and from 0.3 to 58 years for B[*a*]P (Herbes and Schwall, 1978). Polycyclic aromatic hydrocarbons in sediments are relatively stationary. Near Seattle, Washington, 63% of particle-bound PAHs were found at the bottom of Puget Sound less than 100 m away from their point of entry (Murphy *et al.*, 1988). Nonetheless, sediments may be partially resuspended and are then subject to transport processes (Windsor and Hites, 1979; Larsen *et al.*, 1986).

Polycyclic aromatic hydrocarbons are removed from soils principally by volatilization and microbial activity, the extent of which varies, depending on several factors such as temperature, soil type, presence of other contaminants, and previous contamination (Beak, 1981; Bulman *et al.*, 1985; PACE, 1988; ASTDR, 1990b; Cooper, 1991; Wild *et al.*, 1991). Low molecular weight PAHs volatilize more rapidly than high molecular weight PAHs (Slooff *et al.*, 1989; Wild and Jones, 1993). Biodegradation

half-lives in soil have been estimated for various PAHs, including anthracene (170 days [Herbes and Schwall, 1978] to 8 years [Wild *et al.*, 1991)]); phenanthrene (from 2.5 to 210 days [Sims and Overcash, 1983]) to 5.7 years [Wild *et al.*, 1991]); and B[*a*]P (8.2 years [Wild *et al.*, 1991] and 0.3 to 58 years [Herbes and Schwall, 1978]). In farming operations in which refinery wastes and sewage sludge are applied to the soil, low molecular weight PAHs (two and three rings) are expected to volatilize or biodegrade within three to four month; higher molecular weight PAHs (more than three rings) can be substantially biodegraded in a four-month period, but repeated applications of oily sludges containing PAHs may result in accumulation of these compounds in soil (PACE, 1988).

Contamination of groundwater by PAHs can occur as a result of leaching through soils, especially when PAHs are accompanied by mobile organic solvents or when channels are present in the soil (Bedient *et al.*, 1984; Slooff *et al.*, 1989). Naphthalene was the most mobile PAH reported below a creosote-contaminated site in the United States; concentrations of naphthalene at a depth of 3 m were 5% of those at a depth of 0.2 to 0.5 m (Wang *et al.*, 1983). Contamination of groundwater has been observed following application of oily sludges to soil (PACE, 1988).

Polycyclic aromatic hydrocarbons can accumulate in a variety of organisms. Bioconcentration factors (BCFs) ranging from 4 to 7800 have been reported for various PAHs in unicellular algae (Lu *et al.*, 1977; Dobrowsky and Epifanio, 1980; Casserly *et al.*, 1983; Mailhot, 1987). Polycyclic aromatic hydrocarbons can be oxidized by these organisms, although only 10 to 37% of accumulated B[*a*]P was oxidized by the green alga, *Selenastrum capricornutum* (Warshawsky *et al.*, 1983).

In aquatic invertebrates, bioconcentration from water has been measured in the cladoceran *Daphnia pulex* after one hour of exposure to five PAHs; the BCFs were correlated with the K_{OW}s for individual PAHs, and ranged from 131 for naphthalene to 10 109 for benz[*a*]anthracene (Southworth *et al.*, 1978). For several invertebrates, bioconcentration of PAHs from sediments has also been observed, especially when PAH loadings in the sediments were high (Eadie *et al.*, 1983; Pruell *et al.*, 1986). For example, the BCF of anthracene increased from 1800 to 9096 in the marine amphipod *Hyalella azteca* when sediment was added to water containing 8 µg/L of anthracene (Landrum and Scavia, 1983). Biotransfer through food can also occur; 44% of the B[*a*]P content of the diatom *Thalassiosira pseudomona* was transferred to larvae of the clam *Mercenaria mercenaria* in 24 hours (Dobrowsky and Epifanio, 1980). The rate of elimination of PAHs in aquatic invertebrates is much lower than the rate of uptake (NRCC, 1983), resulting in a long half-life for PAHs in invertebrates. For example, in the mussel *Mytilus edulis*, depuration half-lives for B[*a*]P, benz[*a*]anthracene, and fluoranthene were 15, 18, and 30 days, respectively (Pruell *et al.*, 1986).

Based on assays conducted in laboratories with radio-labelled PAHs, bioconcentration factors for fish range from 23 for ^{14}C-naphthalene to 675 for ^{14}C-anthracene (Spacie *et al.*, 1983; Linder and Bergman, 1984; Solbakken *et al.*, 1984); these values may be overestimated since radioactive counting detects metabolites as well

as the original compounds. In addition to direct bioconcentration from water, uptake from food may also occur, as observed in the field in English sole (*Parophrys vetulus*) (Malins *et al.*, 1984). However, the biomagnification of PAHs up aquatic food chains is not expected to occur since the elimination of PAHs and their metabolites is relatively rapid in fish (EAG, 1990). Following exposure for 5 hours to PAHs, the depuration half-lives in the bluegill sunfish (*Lepomis macrochirus*) were 17 hours for ^{14}C-anthracene and 67 hours for ^{14}C-B[*a*]P (Spacie *et al.*, 1983). In alevin of the Atlantic cod (*Gadus morhua*), the half-lives were less than 24 hours for ^{14}C-naphthalene and ^{14}C-B[*a*]P (Solbakken *et al.*, 1984).

Plants take in more PAHs from the atmosphere than from the soil; plants with large leaves, therefore, generally contain more PAHs than those with narrow leaves (Edwards, 1983). The outer surfaces of plants, such as fruit peel, contain more PAHs than the internal structures. After uptake, there is little transfer or translocation of PAHs within the plant (Edwards, 1986).

2.3.2 *Concentrations*

Polycyclic aromatic hydrocarbons are widely distributed in the environment and have been detected in numerous media to which humans and biota are exposed including air, water, food, soil, sediment, and tobacco smoke. Most available data pertain to concentrations near known sources of PAHs, rather than from uncontaminated sites.

Air. Concentrations of PAHs in ambient air have been measured in British Columbia, the Yukon Territory, Manitoba, Ontario, Quebec, and Nova Scotia. Concentrations for the 13 PAHs considered in this report are presented in Table 3. It is not always possible to separate the [*b*] and [*k*] isomers or to isolate the [*j*] isomer of benzofluoranthene with most current analytical methods. Tardif and Chiu (1992) successfully analyzed these compounds in a limited number of samples; the three isomers were present in consistent proportions at the seven sites sampled. The concentrations of the [*b*], [*j*], and [*k*] isomers of benzofluoranthene presented in Table 3 were calculated based on these proportions.

Data on atmospheric concentrations of PAHs are available for one rural site located at Walpole Island, Ontario. The median concentration of total PAHs was 7.2 ng/m^3 (mean of 10.0 ng/m^3), with a maximum of 40.4 ng/m^3 (Dann, 1990). The highest concentrations for individual PAHs were recorded for phenanthrene and fluoranthene, while anthracene, B[*a*]P, and indeno[*1,2,3-cd*]pyrene were detected in fewer than half the samples.

The highest concentrations of PAHs in ambient air in Canada were measured at stations located about 1 km from aluminum smelters using the Horizontal Stud Söderberg process in Jonquière and Shawinigan, Quebec. The median concentrations of total PAHs (the sum of 26 compounds) were 693 ng/m^3 (mean of 1687 ng/m^3) at Jonquière and 435 ng/m^3 (mean of 1519 ng/m^3) at Shawinigan, with maxima of 10 400 and 16 390 ng/m^3, respectively. In Jonquière, the highest concentrations of individual PAHs were recorded for phenanthrene and fluoranthene. The maximum concentrations

Table 3 Concentrations of Polycyclic Aromatic Hydrocarbons in Ambient Air (ng/m^3) in Canada

Sector	Aluminum plant*						Wood heating				Rural	
Location	Kitimat[4]		Jonquière[5]		Shawinigan[6]		Whitehorse[5]		Sept-Îles[6]		Walpole[7]	
Number of samples	24		42		31		5		24		62	
Sampling period	1990		1989 to 1991		1989 to 1991		1982 to 1983		1990 to 1991		1988-1989	
Statistical value	Mean	Median	Mean	Median	Mean	Median	Mean	Median	Mean	Median	Mean	Median
naphthalene	3.10	2.00	26.00**	3.26**	12.45	4.33	na	na	43.74	1.29	na	na
acenaphthene	15.10	5.40	121.72	11.99	55.00	9.85	na	na	4.52	0.90	0.32	0.23
fluorene	15.63	9.85	41.45	8.34	46.24	10.97	26.47	22.15	15.48	6.15	0.66	0.39
phenanthrene	57.81	45.50	371.26	197.78	389.65	93.98	271.43	170.77	50.35	22.11	4.18	2.93
anthracene	4.07	2.35	42.92	9.47	29.90	8.47	22.81	14.81	8.65	4.02	< 0.05	< 0.05
pyrene	14.36	10.00	195.89	90.42	206.78	62.07	49.41	43.78	12.28	5.19	0.73	0.51
fluoranthene	22.14	16.00	272.27	132.13	261.00	87.41	50.89	45.68	15.54	6.69	1.32	0.95
benz[*a*]anthracene	2.52	1.65	57.71	8.69	35.14	4.89	9.81	6.97	3.09	1.14	0.07	0.04
benzo[*a*]pyrene	2.06	1.15	36.37	7.15	28.47	4.21	7.66	5.39	1.93	0.68	0.08	< 0.05
benzo[*b,j,k*]fluoranthene[1]	7.90	6.05	166.60	62.91	149.06	29.24	12.75	9.55	5.65	2.38	0.31	0.16
benzo[*b*]fluoranthene[2]	4.42	3.39	93.3	35.23	83.47	16.37	7.14	5.35	3.16	1.33	0.17	0.09
benzo[*j*]fluoranthene[2]	1.96	1.51	41.65	15.73	37.26	7.31	3.19	2.39	1.41	0.60	0.08	0.04
benzo[*k*]fluoranthene[2]	1.50	1.15	31.65	11.95	28.32	5.56	2.42	1.81	1.07	0.45	0.06	0.03
indeno[*1,2,3-cd*]pyrene	1.88	0.92	30.57	8.38	24.81	5.66	5.86	3.60	1.62	0.49	0.10	< 0.05
Total PAHs [3]	155.50	110.03	1687.04	692.88	1519.13	434.79	506.50	498.11	202.09	60.52	9.97	7.19

Table 3 Concentrations of PAHs in Ambient Air (ng/m^3) in Canada (continued)

Sector	Urban: Road Transportation and combustion											
Location Number of samples Sampling period	Vancouver[5] 11 1985		Winnipeg[9] 10 1989		Windsor[7] 55 1988-1989		Toronto[10] 42 1984-1986		Montreal[10] 23 1989-1990		Sydney[11] 39 1981-1982	
Statistical value	Mean	Median	Mean	Median	Mean	Median	Mean	Median	Mean	Median	Mean	Median
naphthalene	na	na	na	na	na	na	na	na	na	na	na	na
acenaphthene	na	na	0.06	0.00	5.21	2.40	2.24	1.66	1.93	1.30	na	na
fluorene	na	na	0.94	0.96	4.65	2.80	5.51	4.50	3.12	2.30	na	na
phenanthrene	na	na	5.26	4.62	34.79	22.67	15.64	14.47	19.59	18.98	2.20	0.34
anthracene	na	na	0.45	0.28	3.77	0.63	1.60	0.94	1.89	1.28	na	na
pyrene	4.92	5.30	1.44	1.13	6.91	4.56	3.87	3.36	6.65	4.10	3.82	0.49
fluoranthene	6.76	8.50	1.55	1.13	11.08	7.03	4.91	4.82	9.60	5.45	3.52	0.36
benz[*a*]anthracene	0.81	0.70	0.19	0.12	0.74	0.23	0.40	0.17	1.16	0.28	4.40	0.32
benzo[*a*]pyrene	0.35	0.30	0.09	< 0.05	0.82	0.37	0.30	0.16	0.56	0.29	1.74	0.29
benzo[*b,j,k*]fluoranthene[1]	1.32	1.20	0.37	0.16	2.71	1.22	1.26	0.83	5.48	2.11	3.64	0.74
benzo[*b*]fluoranthene[2]	0.74	0.67	0.21	0.09	1.52	0.68	0.71	0.46	2.51	1.18	2.04	0.41
benzo[*b*]fluoranthene[2]	0.33	0.30	0.09	0.04	0.68	0.31	0.31	0.21	1.12	0.53	0.91	0.19
benzo[*k*]fluoranthene[2]	0.25	0.23	0.07	0.03	0.51	0.23	0.24	0.16	1.85	0.40	0.69	0.14
indeno[*1,2,3-cd*]pyrene	1.89	1.14	0.19	0.12	0.87	0.53	0.46	0.29	0.96	0.51	1.95	0.31
Total PAHs [3]	20.41	19.10	73.58	10.47	89.28	62.16	47.58	44.09	69.01	57.50	31.16	3.95

na - not analyzed

[1] Total for [*b*], [*j*], and [*k*] isomers of benzofluoranthene

[2] Values for [*b*], [*j*], and [*k*] isomers of benzofluoranthene calculated using proportions for isomers reported by Tardif and Chiu (1992)

[3] Total of all PAHs analyzed at each location

[4] Johnson, 1991

[5] Ringuette *et al.*, 1993

[6] LEI, 1992a; b

[7] Dann, 1992b; 1991

[8] Faulkner, 1985

[9] Bezak, 1990

[10] Dann, 1989

[11] Atwell *et al.*, 1984

* Kitimat: Vertical Stud Söderberg
Shawinigan and Jonquière: Horizontal Stud Söderberg

for B[*a*]P were 305 ng/m^3 in Jonquière and 460 ng/m^3 in Shawinigan (Ringuette *et al.*, 1993).

High concentrations were also measured in urban areas where heating by wood combustion is prevalent. In Whitehorse, Yukon, the median concentration of total PAHs was 498 ng/m^3 in winter (mean of 507 ng/m^3), with a maximum of 1000 ng/m^3. Phenanthrene, fluoranthene, and fluorene were present at the greatest concentrations (Ringuette *et al.*, 1993). In Sept-Îles, Quebec, the concentrations of PAHs in winter were ten times higher than those measured in summer (Germain and Bisson, 1992).

High concentrations of PAHs have been reported in the vicinity of transportation sources. In downtown Montreal, the median concentration of PAHs was 57 ng/m^3 (mean of 69 ng/m^3) and the maximum measured was 289 ng/m^3 (Dann, 1991).

Tobacco Smoke. The levels of PAHs in tobacco smoke have been identified in mainstream and sidestream smoke from cigarettes, cigars, and pipes. The concentrations of PAHs in the mainstream smoke, expressed as µg/100 cigarettes, ranged from 0.4 to 2.2 for B[*b*]F, 0.6 to 2.1 for B[*j*]F, 0.6 to 1.2 for B[*k*]F, 0.5 to 7.8 for B[*a*]P, and 0.4 to 2.0 for IND (IARC, 1983).

Surface Water. Concentrations of PAHs in fresh water at selected sites in Canada are presented in Table 4. Concentrations may include both dissolved PAHs and extractable PAHs adsorbed to suspended particles in the water column. Concentrations of PAHs in the Atlantic provinces were low, with a median concentration of B[*a*]P below the detection limit (0.8 to 1 ng/L) and a maximum concentration of 9 ng/L (Wong and Bailey, 1990). The highest PAH concentration in the St. Lawrence River was for phenanthrene, for which the median concentration was 17.6 ng/L and the maximum value was 119 ng/L (Envirodat, 1993). Concentrations of PAHs in the Niagara River ranged from 2.1 to 63.9 ng/L, with a median of 13.4 ng/L (Kuntz, 1990), while they ranged from below the detection limit (0.4 ng/L) to 6 ng/L in the Detroit River near Windsor, Ontario (Kaiser *et al.*, 1985). In the Mackenzie River (Northwest Territories), water samples collected under the ice cover in March 1988 had lower concentrations of total PAHs (from 10.7 to 546 ng/L) than those recorded in June 1986 (54 to 1824 ng/L) at the same sites, indicating that run-off following ice breakup carried PAHs into the river (Nagy *et al.*, 1987; 1989). The highest concentrations of PAHs in water in Canada were reported for water samples from ditches beside utility and railway lines near Vancouver, B.C. (Wan, 1991; 1993). The highest mean concentrations were measured near utility poles treated with creosote, with values of 488 µg/L for naphthalene, 1642 µg/L for phenanthrene (Wan, 1991), 2035 µg/L for fluoranthene, and 5356 µg/L for total PAHs (Wan 1993).

In the Atlantic provinces, concentrations of B[*a*]P in marine waters ranged from below the detection limit (0.8 to 1 ng/L) to 16 ng/L, while concentrations of fluoranthene ranged up to 113 ng/L. The detection frequency was low for B[*a*]P (2%), but higher for fluorene (25%) (Wong and Bailey, 1990). The highest concentration of total PAHs measured in harbours in Nova Scotia was 880 ng/L (O'Neill and Kieley, 1992).

Table 4 Concentrations of Polycyclic Aromatic Hydrocarbons in Fresh Water (μg/L) in Canada

Location	Railway ditches without poles [1]	Railway ditches with poles [1]	Utility right-of-way ditches [1]	Farm ditches [1]	Niagara River [2]	Quebec [3]	Nova Scotia [3]	New Brunswick [3]	Newfound-land [3]
Number of samples	3	2	4	2	51				
Sampling period	1990 to 1991	1990 to 1991	1990 to 1991	1991	1988 to 1989	1987 to 1993	1987 to 1993	1987 to 1993	1987 to 1993
Statistical value	mean	median	mean	median	median	median	median	median	median
naphthalene	0.19	8.5	0.17	0.35	0.0036	*	*	*	*
acenaphthene	0.57	206	0.63	1.04	*	0.01 (n=44)	*	*	*
fluorene	0.22	116	1.72	0.3	0.0007	0.015 (n=44)	0.007 (n=45)	*	*
phenanthrene	0.44	1027	7.67	0.4	0.0021	0.0176 (n=53)	*	*	*
anthracene	0.13	81	1.65	0.16	0.0002	*	*	*	*
pyrene	0.19	1233	3.41	0.19	0.0003	0.015 (n=57)	0.005 (n=52)	*	*
fluoranthene	0.26	2035	3.08	0.3	0.0008	*	*	*	*
benz[*a*]anthracene	0.12	195	0.69	0.1	0.0003	*	*	*	*
benzo[*a*]pyrene	0.10	43	0.48	0.1	0.0002	0.00046 (n=57)	0.001 (n=168)	0.001 (n=78)	0.001 (n=79)
benzo[*b*+*k*]fluoranthene [4]	0.10	144	1.1	0.19	*	*	*	*	*
indeno[*1,2,3-cd*]pyrene	0.15	17.6	0.4	0.15	*	*	*	*	*
Total PAHs [5]	3.97	5356.3	23.2	5.56	0.0036	*	*	*	*

[1] Wan, 1993
[2] Kuntz, 1990
[3] Envirodat, 1993
[4] Total for [*b*] and [*k*] isomers of benzofluoranthene reported by the authors; data not available for [*j*] isomer
[5] Total of all PAHs analyzed at each location
* less than limit of detection or not reported

Groundwater. Few studies are available concerning the presence of PAHs in groundwater in Canada. Near a former refinery at Pincher Creek, Alberta, the concentrations of pyrene ranged from below the detection limit to 300 µg/L, with a median concentration of about 30 µg/L (ETL, 1984). Concentrations of fluorene at this site ranged from below the detection limit to 230 µg/L; the median was 40 µg/L (ETL, 1984). At Newcastle, New Brunswick, naphthalene was detected at concentrations as high as 2.8 µg/L and B[*a*]P as high as 0.32 µg/L in groundwater near a wood preserving plant (WMS, 1989).

Drinking Water. Few data are available on the levels of PAHs in drinking water, particularly for those compounds being considered principally with respect to human health in this assessment. B[*a*]P was not detected in a survey of seven water treatment plants in the area of Niagara Falls (detection limit 1.0 µg/L) (MOE, 1984). The only PAHs detected in 2006 analyses of treated drinking water conducted in the Ontario Drinking Water Surveillance Program in 1987 were B[*k*]F (twice at 1 ng/L), fluoranthene (20 and 30 ng/L), and pyrene (twice at 40 ng/L). The range of compounds examined was not specified (MOE, 1989). In Quebec, the mean concentrations of PAHs in treated water determined recently ranged from <5 to 623 ng/L for fluoranthene, <5 to 40 ng/L for B[*k*]F, to 40 ng/L for B[*b*]F, and were less than 5 ng/L for B[*a*]P, IND, and benzo[*ghi*]perylene (Ayotte and Larue, 1990).

In a survey conducted in the Atlantic Region from 1985 to 1988 (Environment Canada, 1989a;b;c;d), the concentrations of fluoranthene, B[*a*]P, B[*b*]F, B[*k*]F, IND, and benzo[*ghi*]perylene (B[*ghi*]P) were determined (detection limits of 0.001, 0.001, 0.001, 0.001, 0.005, 0.005 µg/L, respectively). In Newfoundland, only fluoranthene was detected at all sites. Concentrations were near the detection limit, except for the water supply at Baie Verte for which a maximum value of 0.054 µg/L was reported. At this particular site, B[*b*]F (0.001 to 0.005 µg/L), B[*k*]F (0.001 to 0.003 µg/L), and B[*a*]P (0.001 to 0.003 µg/L) were also detected. In Prince Edward Island, fluoranthene was detected at low levels (range, 0.001 to 0.012 µg/L) in every supply. In one sample collected in 1986 from a well at St. Eleanors, P.E.I., B[*b*]F (0.001 to 0.003 µg/L), B[*a*]P (0.001 to 0.003 µg/L), and B[*ghi*]P (0.005 to 0.021 µg/L) were also detected. In Nova Scotia, only fluoranthene was detected in raw sources, at levels ranging from the detection limit to 0.008 µg/L. In New Brunswick, several samples contained fluoranthene at levels ranging from 0.001 to 0.005 µg/L. Concentrations of B[*b*]F, B[*k*]F, and B[*a*]P ranged up to 0.002, 0.001, and 0.003 µg/L, respectively, in samples collected at a well in Fredericton.

Sediments. Data on concentrations in sediments have been identified for all provinces, except Manitoba and Saskatchewan. Representative concentrations are summarized in Table 5. The highest concentrations were reported in sediments collected in or around industrialized harbours close to known sources of PAHs. The concentrations presented in this section are expressed on a dry weight (d.w.) basis, unless otherwise noted.

Table 5 Concentrations of Polycyclic Aromatic Hydrocarbons in Sediments (μg/g dry weight) in Canada

Location	Vancouver Harbour, B.C. [1]	Kitimat Arm, B.C. [2]	Hamilton Harbour, Ont. [3]	Kettle Creek, Ont. [4]	Montreal Harbour, Que. [5]	Muggah Creek, Sydney Harbour [6]	Sydney Harbour, N.S. [7]	Luxton and Mountain Lakes, N.S. [8]	Lake ELA375, Ont. [9]
Number of samples	23	6	25	17	42	1	38	8	9
Sampling period	1987	1990	1988 to 1989	1987	1989	1980	1986	1980s	1990
Statistical value	median	median	median	median	median	single result	median	median	median
naphthalene	0.40	0.14	19.8	*	0.2	*	0.05	0.24	0.014
acenaphthene	0.05	0.06	3.8	*	0.2	*	< 0.05	< 0.01	0.001
fluorene	0.10	0.11	7.0	*	0.4	*	< 0.02	0.04	0.007
phenanthrene	0.51	0.34	25.8	*	2.8	655	0.54	0.07	0.059
anthracene	0.12	0.26	10.9	*	0.6	*	< 0.05	< 0.01	0.004
pyrene	0.74	2.20	22.8	*	2.8	413	0.61	< 0.01	0.045
fluoranthene	0.64	2.90	34.2	*	2.8	607	0.715	< 0.01	0.063
benz[*a*]anthracene	0.24	2.45	8.8	*	1.1	414	0.37	< 0.01	0.017
benzo[*a*]pyrene	0.34	4.35	8.9	1.0	1.2	109	0.515	< 0.01	0.025
benzo[*b*+*k*]fluoranthene[10]	0.41	6.4	15.3	*	2.3	184.6	0.57	< 0.01	0.098
indeno[*1,2,3-cd*]pyrene	0.16	3.3	15.7	*	1.0	71.5	0.29	< 0.01	0.087
Total PAHs [11]	5.36	31.24	282.5	28	14.2	2830	5.20	0.51	0.56

[1] Boyd *et al.*, 1989
[2] Goyette, 1991
[3] Murphy *et al.*, 1993
[4] AEL, 1988
[5] Environnement Illimitée Inc., 1990
[6] Sirota *et al.*, 1983
[7] Kieley *et al.*, 1988
[8] Keizer, 1990
[9] Lockhart, 1990
[10] Total for [*b*] and [*k*] isomers of benzofluoranthene; data not available for [*j*] isomer
[11] Total of all PAHs analyzed at each location

* less than detection limit or not reported

Sydney Harbour, Nova Scotia, is a major site of PAH contamination in Canada. The highest levels of PAHs were measured in the South Arm near the mouth of Muggah Creek, where a steel production complex is located. The lowest concentrations were measured in the Northern Arm (Sirota *et al.*, 1983). At the mouth of Muggah Creek, the concentrations of phenanthrene, pyrene, and B[*a*]P were 655, 413, and 109 µg/g, respectively, with a total PAH concentration (12 compounds) of 2830 µg/g in 1981. A decrease in the steel plant production followed by the closure of the coking facility in 1988 led to a drop in PAH concentrations in sediments. In 1986, close to the same locations, the maximum concentration of total PAHs (18 compounds) was reported to be 310 µg/g near the effluent outfall of the steel complex; the 13 PAHs considered here accounted for 251 µg/g (Kieley *et al.*, 1988). The concentration of total PAHs was 0.0029 µg/g at the least contaminated station of Sydney Harbour, located 6.5 km from Muggah Creek. The median concentration of total PAHs for all the stations was 5.2 µg/g. Other reported median and maximum values were 0.72 and 60.0 µg/g for fluoranthene; 0.61 and 33.0 µg/g for pyrene; and 0.515 and 28 µg/g for B[*a*]P.

In comparison, median concentrations in sediments at sites remote from pollution sources in Luxton and Mountain lakes in Kejimkujik National Park, Nova Scotia, were 0.51 µg/g for total PAHs and less than 0.01 µg/g for B[*a*]P; maximum concentrations were 0.86 and 0.05 µg/g, respectively. For phenanthrene, the median was 0.07 µg/g and the maximum, 0.10 µg/g. Pyrene was not detected in more than half of the samples; the maximum value was 0.05 µg/g (Keizer, 1990). Lockhart (1990) observed similar results in two experimental lakes in Northern Ontario (median and maximum total PAH concentrations: 0.56 and 0.85 µg/g; median and maximum B[*a*]P concentrations: 0.03 and 0.04 µg/g).

In the sediments of Montreal Harbour, Quebec, the median and maximum concentrations were 5.7 and 66.0 µg/g for phenanthrene, and 2.8 and 39.0 µg/g for pyrene. The median concentration of B[*a*]P was 1.2 µg/g, with a maximum of 29.0 µg/g. The median concentration of total PAHs was 14.2 µg/g and the maximum value was 278 µg/g (Environnement Illimitée Inc., 1990).

In Hamilton Harbour, Ontario, where the two largest steel mills in Canada are located, the reported median for total PAHs was 285 µg/g, with a range from 1.6 to 1470 µg/g. The median concentrations for pyrene and fluoranthene were 17.3 and 20.1 µg/g, respectively, and their maxima were 280 µg/g (pyrene) and 189 µg/g (fluoranthene). Median and maximum concentrations of B[*a*]P were 9.5 µg/g and 69.2 µg/g (Murphy *et al.*, 1993).

The median concentration of total PAHs in the sediments of Kettle Creek, downstream from an old oil-gasification complex at Port Stanley, Ontario, was 28 µg/g; the maximum value was 499 µg/g. For B[*a*]P, median and maximum concentrations were 1.0 and 40.2 µg/g, respectively (Canviro, 1988).

In Vancouver Harbour, British Columbia, the median concentration for total PAHs was 5.4 µg/g and the maximum was 36.8 µg/g (Boyd *et al.*, 1989). Median and maximum concentrations were 0.74 µg/g and 5.55 µg/g for pyrene; 0.64 and 5.63 µg/g for fluoranthene; and 2.42 µg/g and 0.134 µg/g for B[*a*]P. In Kitimat, B.C., the median for total PAHs was 31.2 µg/g, ranging from 0.14 to 258 µg/g (Goyette, 1991). Median and maximum concentrations were 2.9 and 27.0 µg/g for fluoranthene, 2.2 and 23.1 µg/g for pyrene, and 4.35 and 39 µg/g for B[*a*]P.

Soils. The highest concentrations of PAHs in soils are found near former coking plants (CCME, 1989). The Resources Development Research Centre identified 144 potential sites of former coking plants in Canada, 80 of which are confirmed sites (RDRC, 1987). Although the majority (70) of these sites are located in Ontario, the RDRC identified sites in all provinces except Prince Edward Island. The site of a former coking facility in Lasalle, Quebec, was investigated after the facility closed in 1976. Concentrations of B[*a*]P, the only compound investigated in 1985, varied from the detection limit (unspecified) to 1300 µg/g (d.w.). The site has now been restored and monitoring studies entailing more than 800 soil samples have shown that the B[*a*]P concentration is less than 10 µg/g (d.w.) (ARGUS Groupe Conseil Inc., 1991). At the former coking facility in Sorel, Quebec, the maximum concentration of total PAHs (16 compounds) was reported to be 11 473 µg/g before site restoration, with a median concentration of 18 µg/g (Tecsult, 1989).

Kieley *et al.* (1986) reported median and maximum concentrations of 965 and 16 000 µg/g (d.w.) for total PAH (12 PAH compounds) in a soil sample collected at the site of a wood preserving plant in Newcastle, New Brunswick. In Ottawa, Ontario, decontamination measures were implemented at a site that was contaminating the Rideau River. The B[*a*]P concentration in soil was reported to be 2.4 µg/g before the restoration (RDRC, 1987). The site of a former refinery in Pincher Creek, Alberta, was investigated after the refinery was dismantled. Before site restoration, high PAH concentrations (primarily alkylated PAHs) were observed in the soil. Maximum concentrations for fluoranthene and pyrene were both 260 µg/g and their median concentrations were 0.75 and 0.50 µg/g, respectively. B[*a*]P was not detected, however. In the vicinity of the plant, total concentrations of PAHs in soil were reported to be 9810 µg/g (ETL, 1984).

Biota. Concentrations of PAHs in biota in Canada are presented in Table 6.

In marine organisms on the Atlantic coast, 12 PAHs were detected in lobster (*Homarus americanus*) from Sydney Harbour, Nova Scotia, in 1981. A mean concentration of B[*a*]P of 0.031 µg/g (d.w.) was recorded in the tails of the lobster and 0.84 µg/g (w.w.) in the hepatopancreas. Total concentrations of PAHs were 2.29 µg/g (d.w.) and 72.7 µg/g (w.w.), respectively (Sirota *et al.*, 1983). Other studies have revealed high PAH concentrations in other organisms in the area (Matheson *et al.*, 1983; Uthe and Musial, 1986; Kieley *et al.*, 1988). For example, mussels (*Mytilus edulis* and *Modiolus modiolus*) collected in Sydney Harbour contained up to 4.2 µg/g (w.w.) of PAHs (O'Neill and Kieley, 1992).

Table 6 Concentrations of Polycyclic Aromatic Hydrocarbons in Biota (μg/g) in Canada

Location	Vancouver Harbour, B.C. [1]		Fraser River, B.C. [2]	St. Lawrence River [3,4]		Sydney Harbour (South Arm) N.S. [5]		Sydney Harbour, N.S. [6]
Species	Dungeness crab *(Cancer magister)* liver (d.w.)*	English sole *(Parophris vetulus)* liver (d.w.)	prickly sculpin *(Cottus asper)* liver (w.w.)*	mussels *(Mytilus edulis)* (w.w.)	northern pike *(Esox lucius)* (w.w.)	lobster *(Homarus americanus)* tail muscle (d.w.)	lobster *(Homarus americanus)* hepatopancreas (w.w.)	mussels *(Modiolus modiolus, Mytilus edulis)* (w.w.)
Number of samples	3	4	4	25	7	10	10	9
Sampling period	1986	1986	1981	1989	1989	1980	1980	1986
Statistical value	median	median	median	median	median	mean	mean	median
naphthalene	**	**	0.040	0.026	0.210	**	**	0.061
acenaphthene	**	**	< 0.160	0.002	**	**	**	< 0.007
fluorene	< 0.06	< 0.06	< 0.10	0.009	0.030	**	**	< 0.006
phenanthrene	0.037	0.016	0.020	0.024	0.040	0.43	3.18	0.064
anthracene	< 0.001	< 0.001	**	0.002	0.001	**	**	< 0.05
pyrene	< 0.010	< 0.010	< 0.040	0.009	0.004	0.20	4.82	0.042
fluoranthene	0.013	0.013	**	0.026	0.020	0.49	11.55	0.025
benz[*a*]anthracene	< 0.001	< 0.001	**	0.007	**	0.478	18.4	0.035
benzo[*a*]pyrene	< 0.005	< 0.005	**	0.002	**	0.03	0.84	0.004
benzo[*b*+*k*]fluoranthene[7]	0.008	< 0.005	**	**	**	0.84	2.49	0.015
indeno[*1,2,3-cd*]pyrene	< 0.060	< 0.060	**	**	**	0.04	0.82	0.027
Total PAHs[8]	**	**	0.078	0.153	0.405	2.29	72.7	0.680

[1] Goyette and Boyd, 1989
[2] Chapman *et al.*, 1981
[3] Langlois, 1989
[4] Lapierre, 1989
[5] Sirota *et al.*, 1983
[6] Kieley *et al.*, 1988
[7] Total for [*b*] and [*k*] isomers of benzofluoranthene; data not available for [*j*] isomer
[8] Total of all PAHs analyzed at each location

* d.w. - dry weight; w.w. - wet weight
** less than limit of detection (variable for each compound or study) or not reported

On the Pacific coast, studies have been conducted in Vancouver Harbour, British Columbia. Total concentrations of PAHs in mussels collected near creosoted barricades and wharfs were between 54 and 215 µg/g (w.w.) (Goyette and Boyd, 1989). In 1986, appreciable concentrations of phenanthrene [0.830 µg/g (d.w.)] and pyrene [0.36 µg/g (d.w.)] were measured in the livers of Dungeness crab (*Cancer magister*). These compounds were also detected in the livers of English sole (*Parophris vetulus*), but in lower concentrations [0.037 µg/g for phenanthrene and 0.021 µg/g for pyrene, (d.w.)] (Goyette and Boyd, 1989).

In freshwater organisms, PAHs have been detected in Quebec in numerous species of fish from Lakes St. Francis, St. Louis, and St. Pierre and from the St. Lawrence Estuary. The median concentrations for total PAHs in the livers of northern pike (*Esox lucius*) and yellow walleye (*Stizostedion vitreum*) were 0.40 and 0.45 µg/g (w.w.), respectively (Lapierre, 1989).

In the Fraser River in British Columbia, naphthalene and phenanthrene were detected in the prickly sculpin (*Cottus asper*), an abundant freshwater fish in the region (Chapman *et al.*, 1981). In 1988, PAHs were measured in the livers of five species of fish (Swain and Walton, 1989). Naphthalene was the most frequently detected compound, with the highest concentrations in the liver of the northern squawfish (*Ptychocheilus oregonensis*).

For birds, the only Canadian data identified are from the late 1970s, for herring gulls (*Larus argentatus*) from Pigeon Island and Kingston, Ontario, where levels in liver were measured for 30 PAHs. For the compounds of concern in this report, the mean levels (in µg/kg lipid) were: 0.05 for naphthalene, 0.038 for acenaphthene, 0.152 for anthracene, 0.082 for fluoranthene, 0.038 for benzo[*a*]pyrene, 0.044 for fluorene, and 0.076 for pyrene (Hallett *et al.*, 1977). These levels were slightly lower than those found for fish from Lake Ontario from the same time period (Hallett *et al.*, 1984).

Food. Concentrations of PAHs in uncooked food depend principally on its source. For example, vegetables, fruits, and fish obtained from polluted areas generally contain higher concentrations of PAHs than those from less polluted regions.

In cooked food, the method of cooking is generally the primary determinant of the PAH content and this content varies considerably depending on cooking habits (Santodonato *et al.*, 1981). In a report prepared under contract for Agriculture Canada, the results were reported of an analysis of 8 PAHs in 208 Canadian food composite samples (supplied by Health Protection Branch, Health Canada, for the Total Diet Program) (Das, 1987). Samples for analysis were prepared for consumption just as in the average household kitchen. Raw meats were cooked (methods unspecified); fresh vegetables were cooked (no salt added) or if not cooked, then properly peeled, trimmed, or otherwise cleaned for serving; processed foods were prepared following directions on the label. The concentrations of three of the five PAHs considered principally in the assessment of potential effects on human health ranged from not detected (detection limit unspecified) in all food types examined to 1.78 µg/kg in vegetables/fruits for B[*b*]F, not

detected to 0.30 µg/kg in meat/poultry/fish for B[*k*]F, and not detected to 1.13 µg/kg in meat/poultry/fish for B[*a*]P. However, the percent recoveries were particularly low for B[*a*]P and B[*k*]F in dairy and cereal products (69.3 and 24.6%, respectively) and in the cooking fats/salad oil/margarine/butter group (34.1 and 43.8%, respectively).

2.4 Effects-related Information

2.4.1 *Toxicokinetics*

Polycyclic aromatic hydrocarbons are metabolized to a wide variety of compounds principally by enzymes of the cytochrome P-450 mixed-function oxidase (MFO) system and epoxide hydrolase (IARC, 1983; Wislocki and Lu, 1988). The most important step of primary metabolism is epoxidation. A small proportion of all PAHs will, after epoxidation and subsequent diol formation, be further epoxidized by the microsomal monooxygenases to diol epoxides. Benzo-ring diol epoxides, in which the epoxide forms part of the bay region* of the hydrocarbon molecule, are likely highly reactive and are implicated as the ultimate biologically reactive intermediates involved in the binding of PAHs to macromolecules and resulting toxicity (Santodonato *et al.*, 1981; Varanasi *et al.*, 1989). They may also be detoxified and excreted as metabolites in bile, feces, and urine by conjugation with glutathione or glucuronic acid, or by further metabolism to tetrahydrotetrols (Gelboin, 1980). The cytochrome P-450 activities of the lung are less than those in the liver and intestinal tract (i.e., metabolism to the active species is slow); moreover, the activity of the conjugating enzyme systems and epoxide hydrase in the lung is very low (i.e., detoxification is slow). These differences may account for possible variations in potency of PAHs following inhalation and ingestion (Santodonato *et al.*, 1981).

The bay region diol epoxide intermediates of PAHs are currently considered to be the ultimate carcinogen for most PAHs, although for some, other reactive intermediates may also be important (ATSDR, 1990a;b; IARC, 1983; Santodonato *et al.*, 1981). Though examined principally in animal models *in vitro* and *in vivo*, results of studies on the metabolism of benzo[*a*]pyrene in primary cultures of human hepatocytes and a human hepatoma cell line indicate that these mechanisms are likely relevant to humans (ATSDR, 1990a).

2.4.2 *Experimental Animals and* In Vitro

Non-neoplastic Effects. Carcinogenicity has been the principal focus of research on the biological effects of PAHs; identified limited information indicates that non-neoplastic effects of these compounds in experimental animals are restricted principally to proliferating tissues such as the bone marrow, lymphoid organs, gonads, and intestinal epithelium (Santodonato *et al.*, 1981; ATSDR, 1990a;b). Observed effects include, for example, prominent hemosiderosis, reduction in spleen size with marked

* Bay regions occur in angularly fused benzo-rings; for example, in benzo(a)pyrene, the sterically hindered region between the 10- and 11-positions constitutes the bay region.

cellular depletion, alteration of the enzyme activity in the intestinal mucosa leading to increased production of reactive intermediates and tissue injury, aplastic anemia, myelosuppression, and decreased fertility. The cytotoxicity is probably a consequence of an interaction with DNA in the S-phase of cell proliferation. The doses required to produce such responses in various species of experimental animals, however, are in some cases an order of magnitude higher than those which induce neoplasms (ATSDR, 1990a;b).

Carcinogenicity. In numerous studies in laboratory animals, various PAHs have induced tumours following principally dermal exposure [see, for example, ATSDR (1990a;b), IARC (1983), and Santodonato *et al.* (1981)]. Data are quite limited, however, on induction of tumours following exposure by routes by which the general population are principally exposed (i.e., inhalation and ingestion).

The carcinogenic effects of exposure to PAHs by inhalation have been examined in only a few limited identified studies, all of which were restricted to B[*a*]P (Heinrich *et al.*, 1986; Laskin *et al.*, 1970; Thyssen *et al.*, 1981); moreover, in two of the investigations, animals were concomitantly exposed to other compounds (Heinrich *et al.*, 1986; Laskin *et al.*, 1970). In the study by Heinrich *et al.* (1986), the incidence of lung tumours was increased in female Wistar rats exposed to combustion gases of a coal furnace for an average of 16 hours/day, 5 days/week over a maximum of 22 months. The incidence of respiratory tract tumours was also increased in rats that inhaled 10 ppm (103 mg/m^3) B[*a*]P and the atmospheric irritant, sulphur dioxide (SO_2) (Laskin *et al.*, 1970).

In a study by Thyssen *et al.* (1981), groups of 24 male Syrian golden hamsters were exposed by inhalation (nose only) to 0, 2.2, 9.5, and 45.6 mg/m^3 B[*a*]P for 4.5 hours/day, 7 days a week for the first 10 weeks, and for 3 hours/day for the rest of the exposure period (up to 96 weeks). Though there was a decrease in body weight gain in exposed animals during the first 10 weeks of the study, from the tenth to the sixtieth week, the body weights of all surviving exposed animals were similar to those of the controls (with the exception of the high exposure group). Mean survival was also decreased in the group exposed to 46.5 mg/m^3. The incidences of unspecified tumours of the respiratory tract (nasal cavity, larynx, and trachea) were 0/27 for controls, 0/27 for the low-dose group, 9/26 (34.6%) for the mid-dose group, and 13/25 (52%) for the high-dose group. Exposure-related neoplasms (unspecified) were present in the pharynx (0, 0, 23, and 56% for control, low-, mid-, and high-dose, respectively), oesophagus (0, 0, 0, and 8% for control, low-, mid-, and high-dose, respectively), and forestomach (0, 0, 4, and 4% for control, low-, mid-, and high-dose, respectively). Lung tumours were not observed.

A relationship between the ingestion of benzo[*a*]pyrene and the development of benign and malignant tumours has been documented in several limited studies in experimental animals (ATSDR, 1990a). In the most extensive, though limited, early study in which mice were fed a diet containing benzo[*a*]pyrene at concentrations of 40 to 45 ppm {equivalent to 5.2 to 5.9 mg/[kg (b.w.)•d]} for 110 days, the incidence of

stomach tumours was increased 10% or less, whereas in mice fed a diet containing 50 to 250 ppm B[*a*]P {equivalent to 6.5 to 32.5 mg/[kg (b.w.)•d]} for 70 to 197 days, it exceeded 70%. In a second experiment in which a diet containing 250 ppm B[*a*]P {equivalent to 32.5 mg/[kg (b.w.)•d]} was fed to mice for different periods of time, incidences of tumours of the forestomach (the only tissue examined) were as follows: 2 to 4 days of feeding, 10%; 5 to 7 days of feeding, 30 to 40%, 30 days of feeding, 100%. However, increases in the incidence of forestomach tumours were not observed following administration of a lower concentration of benzo[*a*]pyrene in the diet {100 ppm or equivalent to 13 mg/[kg (b.w.)•d]} for up to 7 days (Neal and Rigdon, 1967).

The carcinogenicity of a wider range of PAHs has been examined in experimental animals following direct introduction into the lungs (see, for example, ATSDR, 1990b; IARC, 1983; Santodonato *et al.*, 1981). In the most extensive of these investigations in which the broadest range of PAHs was examined, various amounts of anthanthrene (ANT), B[*a*]P, benzo[*e*]pyrene (B[*e*]P), B[*b*]F, B[*j*]F, B[*k*]F, benzo[*ghi*]perylene (B[*ghi*]P), or IND dissolved in residue-free acetone and a mixture of 1:1 beeswax and trioctanoin were implanted into the left lung following administration of anaesthetic and thoracotomy of groups of 38 three-month-old, inbred Osborne-Mendel female rats (Deutsch-Wenzel *et al.*, 1983). Operative and post-operative mortality was less than 5%. After surgery, rats were observed until their natural deaths, which occurred up to 32 months following implantation. At the site of implantation, a granulomatous inflammatory response was observed. In some animals, keratinized epidermoid carcinomas invading the extrapulmonary chest wall were observed; other tumours of this type metastasized predominantly into local and distant lymph nodes, heart, uterus, ovaries, adrenal glands, and kidneys. In a small number of animals, there were pleomorphic sarcomas. On the basis of histological and statistical analysis, there was evidence of an exposure-response relationship for increases in tumour incidence for B[*a*]P, B[*b*]F, IND, B[*k*]F, B[*j*]F, and ANT. The incidence of epidermoid carcinomas and pleomorphic sarcomas of the lung at the highest dose for all compounds administered were: control, 0/0; B[*b*]F at 1.0 mg, 13/35 (37.1%); B[*e*]P at 5.0 mg, 1/35 (2.9%); B[*j*]F at 5.0 mg, 18/35 (51.4%); B[*k*]F at 4.15 mg, 12/27 (44.4%); IND at 4.15 mg, 21/35 (60%); ANT at 0.83 mg, 19/35 (54.3%); B[*ghi*]P at 4.15 mg, 4/34 (11.8%); B[*a*]P at 1.0 mg, 33/35 (94.3%).

Based on data reviewed by IARC, B[*a*]P, B[*b*]F, B[*j*]F, B[*k*]F, and IND have also induced tumours in mice in several studies after dermal application, subcutaneous, and/or intramuscular administration (IARC, 1983).

The available data also indicate that the five PAHs considered principally here have genotoxic potential in both *in vitro* and *in vivo* test systems, and that a mammalian metabolic activation system is necessary for the activity in most cases (see for example IARC, 1983).

2.4.3 *Humans*

Though there have been increases in lung and skin tumour incidence in populations occupationally exposed to complex mixtures containing principally PAHs,

these data have been considered inadequate as a basis for assessment of the weight of evidence of carcinogenicity in humans (IARC, 1983; 1987). Moreover, it is not possible, on the basis of these data, to assess effects of individual PAHs. In addition, the composition of mixtures to which these workers (principally those in coke production, roofing, oil refining, or coal gasification) are exposed may vary considerably from those in the general environment.

2.4.4 *Ecotoxicology*

Laboratory Studies. Most of the ecotoxicological studies on PAHs have been conducted on aquatic rather than terrestrial organisms. Effects on survival, growth, reproduction, and induction of neoplasms have been observed following exposure to PAHs. Ecotoxicological effects on aquatic organisms have been reviewed by several authors (see Germain *et al.*, 1993). The broad range of the reported effects concentrations reflects the variation in experimental parameters and designs (Germain *et al.*, 1993).

The most sensitive non-neoplastic test endpoint for freshwater organisms determined in the laboratory for each of the selected PAHs are presented in Table 7. Only data for environmentally relevant endpoints (lethality, growth, reproduction) and for test species indigenous to North America or closely related species were considered.

Lethal and sublethal effects of PAHs have been investigated in the laboratory for different species of aquatic invertebrates (Germain *et al.*, 1993). For example, 96-hour LC_{50}s (concentrations estimated to be lethal to 50% of tested organisms after 96 hours of exposure) for the water flea *Daphnia pulex* were 5 µg/L for B[*a*]P and 1000 µg/L for naphthalene (Trucco *et al.*, 1983); the 2-day IC_{50} (concentration estimated to be inhibitory to 50% of tested organisms after 2 days) for *Daphnia magna* was 430 µg/L for fluorene; and the 30-day LOEC (lowest observed effect concentration) for emergence of the chironomid *Chironomus riparius* was 600 µg/L for fluorene (Finger *et al.*, 1985). Generally, the short-term lethal and sublethal toxicities for *Daphnia* are greater for PAHs with higher octanol/water partition coefficients (EAG, 1990).

Polycyclic aromatic hydrocarbons in sediments have been associated with mortality of nymphs of the mayfly *Hexagenia limbata* in toxicity tests conducted under laboratory conditions (Krantzberg and Boyd 1992; Murphy *et al.*, 1993). Sediment grab samples from a station in Hamilton Harbour highly contaminated by PAHs induced nearly 100% mortality even after chemically treating the sediment in order to reduce toxicity associated with metals.

In aquatic vertebrates, death can occur following short- or long-term exposure to PAHs. For example, reported toxic thresholds for naphthalene include a 72-hour LC_{50} of 240 µg/L for embryos of largemouth bass (*Micropterus salmoides*), a 23-day LC_{50} of 120 µg/L for embryos of the rainbow trout (*Oncorhynchus mykiss*) (Black *et al.*, 1983), and a 96-hour LC_{50} of 7900 µg/L for fathead minnow (*Pimephales promelas*) (DeGraeve *et al.*, 1982). For phenanthrene, the reported 96-hour LC_{50} was 375 µg/L for juvenile

Table 7 Most Sensitive Toxicity Endpoints Reported for Polycyclic Aromatic Hydrocarbons for Freshwater Organisms

Substance	Organism / Life stage	Endpoint	Concentration (µg/L)	Study type	Temperature (°C) / pH	Dissolved O_2 (mg/L) / Hardness (mg $CaCO_3$/L)	Reference
Naphthalene	Rainbow trout, *Oncorhynchus mykiss*/ Early life stage	27-d LC_{50}	110	Flow-through, measured	20.2 to 23.2 / 7.4 to 8.1	7.1 to 8.4 / 86.8 to 116.3	Black *et al.*, 1983
Acenaphthene	Chironomid, *Paratanytarsus* sp./ Larvae	2-d LC_{50}	60	Static, measured	19.0 to 22.4 / 7.6	n.a. / 46	Lemke and Anderson, 1984
Fluorene	Water flea, *Daphnia magna*/ Adults	14-d LOEC (fecundity 44% lower than controls)	125	Flow-through, measured	25 ±1 / 7.2 to 7.4	n.a. / 270	Finger *et al.*, 1985
Phenanthrene	Rainbow trout, *Oncorhynchus mykiss*/ Embryos	90-d LOEC (survival)	8	Flow-through, measured	10.2 / 6.9	74.8 / 50.4	Call *et al.*, 1986
Anthracene	Fathead minnow, *Pimephales promelas*/ 24-h post-hatching	96-h LC_{50}	6.6	Static, measured	22 ± 1 / 7.5 ± 0.06	6.2 ± 0.4 / 339 ±13	Oris *et al.*, 1990
Pyrene	Water flea, *Daphnia magna*/ First instar larvae	48-h LC_{50}	91	Static, unmeasured	23 / n.a.	5-9 / n.a.	Abernathy *et al.*, 1986
Fluoranthene	Blue-green algae, *Anabaena flos-aquae*/ Exponential growth phase	14-d LOEC (inhibition of growth)	38	Static, measured	25 / n.a.	n.a. / n.a.	Bastian and Toetz, 1982
Benz[*a*]anthracene	Blue-green algae, *Anabaena flos-aquae*/ Exponential growth phase	14-d LOEC (inhibition of growth)	5	Static, measured	25 / n.a.	n.a. / n.a.	Bastian and Toetz, 1982
Benzo[*a*]pyrene	Water flea, *Daphnia pulex*/ 2 mm	96-h LC_{50}	5	Static, measured	15 / 7.5	n.a. / n.a.	Trucco *et al.*, 1983

n.a. - not available

rainbow trout (*Oncorhynchus mykiss*) (Call *et al.*, 1986) and the 27-day LC_{50} was 30 µg/L for their embryos (Millemann *et al.*, 1984). The 96-hour LC_{50} for fluorene was 820 µg/L for juvenile rainbow trout (Finger *et al.*, 1985).

Among sublethal effects, the 30-day LOEC for growth inhibition was 250 µg/L for juvenile bluegill sunfish (*Lepomis macrochirus*) exposed to fluorene (Finger *et al.*, 1985); the LOEC for embryos of the fathead minnow (*Pimephales promelas*) exposed to naphthalene was 850 µg/L (DeGraeve *et al.*, 1982).

Light is important for the activation of the toxic effects of several PAHs, including anthracene, phenanthrene, pyrene, and benzo[*a*]pyrene. Photo-activation is well

documented for various aquatic biota, including vertebrates, invertebrates, and macrophytes (Trucco *et al.*, 1983; Newsted and Giesy, 1987; Oris and Giesy, 1987; Huang *et al.*, 1993).

Exposure of fish to PAHs has been associated with various teratogenic effects. After a seven-day exposure to naphthalene at a concentration of 239 µg/L, teratogenic deformities were seen in 6% of embryos of largemouth bass (*Micropterus salmoides*) (Black *et al.*, 1983). Deformities were also observed in 43% of rainbow trout (*Oncorhynchus mykiss*) exposed for 27 days to 85 µg/L of phenanthrene (Black *et al.*, 1983). Eye anomalies were detected in 7% of rainbow trout alevin exposed for 36 days to 0.2 µg/L of B[*a*]P and in 17% of those exposed to 0.3 µg/L. The lowest concentration at which effects were observed was 0.1 µg/L (36-day LOEC for eye anomalies in 2% of rainbow trout fry) (Hose *et al.*, 1984). In addition, clastogenic effects resulting from DNA damage in fish were reported. For example, a formation of numerous secondary micronuclei is observed in red cells of rainbow trout embryos exposed for 36 days to 0.1 µg/L of B[*a*]P (Hose *et al.*, 1984).

The growth of terrestrial flora may be stimulated or inhibited by PAHs, depending on the concentrations to which the plants are exposed. B[*a*]P, for example, stimulates growth in corn, soya, and wheat at soil concentrations of up to 50 mg/kg (d.w.), and inhibits growth at concentrations above this level (Slooff *et al.*, 1989). Polycyclic aromatic hydrocarbons are of low toxicity to terrestrial plants because plants can degrade PAHs and even synthesize certain PAHs (Slooff *et al.*, 1989).

The toxicity of an artificial mixture of 18 PAHs was tested on embryos of four bird species: chicken (*Gallus domesticus*), turkey (*Meleagris gallopava*), mallard duck (*Anas platyrhynchos*), and common eider (*Somateria mollissima*) (Brunström *et al.*, 1990). The doses injected in the yolk sacs were 2.0 and 0.2 mg/kg of egg. Mortality was significantly increased in all four species at the higher dose, and in mallards at the lower dose. When the 18 compounds were tested individually, only benzo[*k*]fluoranthene, B[*a*]P, and indeno[*1,2,3-cd*]pyrene caused significant increases in mortality in chicken embryos at a dose of 2.0 mg/kg of egg. The B[*k*]F also caused significant increases in mortality of the other three species at 0.2 mg/kg of egg. The various PAHs thus appeared to differ substantially in toxicity to bird embryos.

Genotoxic and neoplastic effects have been reported in both vertebrate and invertebrate organisms following metabolism of certain PAHs. Of the compounds selected for this environmental assessment, such effects were observed under laboratory conditions for B[*a*]P, phenanthrene, and naphthalene (Shugart, 1988; Black *et al.*, 1983; Hose *et al.*, 1984; Hose, 1985).

Exposure of fish to PAHs can lead to clastogenic effects resulting from DNA damage. For example, a formation of numerous secondary micronuclei is observed in red cells of rainbow trout embryos exposed for 36 days to 0.1 µg/L of B[*a*]P (Hose *et al.*, 1984). A 24-week exposure (with two six-hour periods per week) to 150 to 240 µg/L of

B[*a*]P caused hepatic neoplastic tumours in 10% of the guppies tested (Hawkins *et al.*, 1990).

Injecting organic extracts from Hamilton Harbour sediments into the sac fry of rainbow trout (*Oncorhynchus mykiss*) induced malignant hepatic tumours similar to those induced by PAHs such as B[*a*]P and 7,12-dimethylbenzanthracene (Metcalfe *et al.*, 1990). These sediment extracts contained mainly 2 to 16 PAHs (0.01 to 52 µg/g for individual compounds) with traces of polychlorinated biphenyls (0.020 µg/g) and organochlorines such as hexachlorobenzene (0.0006 µg/g). The extracts were mutagenic in *Salmonella typhimurium* and generated aromatic DNA adducts in a mouse cell culture.

Stein *et al.* (1990) have reported that sediment-associated PAHs are biologically available to two flatfish, English sole (*Parophrys vetulus*) and starry flounder (*Platichthys stellatus*). Under laboratory conditions, English sole and starry flounder metabolized B[*a*]P to intermediates that bind to hepatic DNA and develop chemically induced hepato-carcinogenesis (Stein *et al.*, 1990).

English sole caught in an area of Puget Sound with minimal contamination by PAHs were exposed parenterally to organic extracts of urban marine contaminated sediment of Eagle Harbour and to B[*a*]P (Schiewe *et al.*, 1991). Polycyclic aromatic hydrocarbons are the principal constituents (90%) of these sediment extracts. Following 13 exposures (every four weeks) over one year to 30 mg of extract/kg (b.w.), 75 mg of extract/kg (b.w.), or 12 mg of B[*a*]P/kg (b.w.), and after a six-month holding time, fish were examined for hepatic lesions. Preneoplastic foci of cellular alteration were observed in 13.6% of the fish exposed to 30 mg of extract/kg (b.w.), in 13.8% of those exposed to 75 mg of extract/kg (b.w.), and in 13.3% of those exposed to 12 mg B[*a*]P/kg (b.w.); in the controls, no alteration was observed. Controls included sediment extracts from an area of Puget Sound with minimal contamination, carrier extracts, and an untreated group of sole. The lesions induced in the laboratory were not distinguishable from those observed in English sole caught in Eagle Harbour, where the contaminated sediments were sampled.

In aquatic invertebrates, B[*a*]P (0.5 µg/L) was shown to be clastogenic (chromosomal abberations and secondary micronuclei) and teratogenic (deformed gastrula) to eggs of sea urchin (*Paracentrotus purpuratus*) following exposure for 48 hours (Hose, 1985).

Environmental Effects Observed *In Situ*. In Sydney Harbour, surveys of the benthic fauna indicated a lower diversity and abundance in the more contaminated South Arm than in the Northwest Arm (Wendland, 1979; Hildebrand, 1982). The lowest diversity was observed near the mouth of Muggah Creek. The most common taxa reported in South Arm were polychaetes and sea anemones, accounting for 42% of the total biomass. Many species such as hermit crabs, limpets, and amphipods were present in Northwest Arm but not in South Arm. The concentration of total PAHs in sediments varied from 13 to 2800 µg/g (d.w.) in the South Arm and from 2.5 to 8.2 µg/g in the Northwest Arm (Matheson *et al.*, 1983). Although high concentrations of heavy metals

were also reported, no relationship was observed between the concentration of metal and faunal distribution. The colour and the strong hydrogen sulphide (H_2S) odour of the sediments suggested that many metals were in sulphide forms, which are less available to biota (Hildebrand, 1982). The sediment ranged from a brown, sandy mud to a "black sludge" mud with a strong tar odour. Stations with this strong odour had the lowest faunal diversity (Hildebrand, 1982). Diversity and abundance of benthic organisms in Sydney Harbour increased with distance from the mouth of the creek. The tidal flats of Muggah Creek [total PAHs: 2830 µg/g (d.w.)] were completely devoid of life.

Changes in populations of benthic invertebrates have been found to be correlated with concentrations of PAHs in sediments in Kettle Creek in Port Stanley, Ontario (AEL, 1988; Canviro, 1988). This stream drains the site of a former oil gasification complex and its sediments have been classified according to three levels of PAH contamination: <9 µg/g, 12 to 40 µg/g, and >80 µg/g (d.w.). A significantly lower population of oligochaetes (benthic worms), generally considered to be tolerant to the contamination, as well as a general impoverishment of the benthic fauna in the areas with the most contaminated sediments were observed.

In studies at several sites in Canada, including the Great Lakes and Vancouver Harbour, and locations in the U.S. (see Table 8), increases in the incidence of neoplasms in fish in both marine and fresh water have been associated with exposure to PAHs.

Baumann and Whittle (1988) reported that fish in the Great Lakes had higher tumour frequencies in areas with high sediment concentrations of B[*a*]P and fluorene. In Hamilton Harbour, where B[*a*]P and fluorene concentrations were greater than 1 and 10 µg/g (d.w.), respectively, 30% of white sucker (*Catostomus commersoni*) had carcinogenic papillomas. In the eastern side of Lake Ontario, B[*a*]P and fluorene were detected between 0.1 and 1 µg/g and 6% of the white suckers had papillomas. In Lake Superior, where B[*a*]P and fluorene were lower than 0.1 µg/g, papillomas were not observed. Brown bullhead (*Ictalarus nebulosus*) caught in Black River (Ohio) had B[*a*]P levels greater than 5 ng/g (w.w.) in tissues; liver tumours were observed in 25 to 35% of the fish. No tumours were observed for those caught in Lake Buckeye, a reference site.

Baumann and Mac (1988) detected hepatic tumours in 30 to 38% of brown bullhead in the Black River, Ohio, and 13 to 19% of those in the Cuyahoga River, Ohio. High concentrations of PAHs were detected in the sediments of these two rivers, individual compounds ranging from 10 to 1000 µg/g (d.w.) in the Black River and from 0.1 to 1 µg/g (d.w.) in the Cuyahoga River. The sediments contained benz[*a*]anthracene and B[*a*]P. Polycyclic aromatic hydrocarbons were found in the bullheads in the two rivers as well as high concentrations of PAH metabolites in the bile of bullheads in the Black River. Organochlorine compounds and metals were not found in high concentrations, compared to other sites where no hepatic tumours were detected.

In a study of carcinogenesis in fish, Maccubbin *et al.* (1985) reported that the stomach contents of white sucker (*Catostomus commersoni*) inhabiting the bottom of Lake Erie may contain appreciable concentrations of PAHs (0.78 µg/g). The

Table 8 Field Population Studies in Fish[1]

Species	Location	Observed effects and frequency of affected organisms (%)	Ranges of concentrations in sediments (µg/g dry weight)	References
Fresh water				
White sucker, *Catostomus commersoni*	Hamilton Harbour (Ont.) Lake Ontario (eastern side) Lake Superior	Papillomas 30 6 < 1	B[*a*]P > 1; Flu > 10 B[*a*]P: 0.1 to 1; Flu: 0.1 to 1 B[*a*]P < 0.1; Flu < 0.1	Baumann and Whittle, 1988
Brown bullhead, *Ictalurus nebulosus*	Black River (Ohio) Lake Buckeye (Ohio) (reference site)	Liver tumours 25 to 35 0	B[*a*]P > 5 ng/g in tissues; Flu > 1 µg/g in organs	Baumann and Whittle, 1988
Brown bullhead, *Ictalurus nebulosus*	Black River (Ohio) Cuyahoga River (Ohio)	Liver tumours 30 to 38 13 to 19	individual PAHs: 10 to 1000 individual PAHs: 0.1 to 1	Baumann and Mac, 1988
Rainbow trout, *Oncorhynchus mykiss*	Hamilton Harbour (Ont.) Oakville Creek (Ont.)	Hepatic neoplasms 8.9 0	Phe: nd to 40.8; Flu: nd to 52; 16 PAHs detected Phe: nd to 0.04; Flu: nd to 0.03; 4 PAHs detected	Metcalfe *et al.*, 1990
Brown bullhead, *Ictalurus nebulosus*	Buffalo River (N.Y.)	Tumours 27	B[*a*]P: 3; Flu: 13; Pyr: 17	Maccubin *et al.*, 1990
Walleye, *Stizostedion vitreum*	Detroit River (Michigan)	Hepatic neoplasms 20	B[*a*]P: 0.12 to 17.7; Flu: 0.12 to 34.6; Pyr: 0.12 to 38.8	Maccubin *et al.*, 1990
Salt water				
English sole, *Parophrys vetulus*	Puget Sound (Wash.) Mukilteo Point Eagle Harbour President Point (reference site)	Hepatic neoplasms 7.5 27 0	Aromatic hydrocarbons 7.8 to 33 2.8 to 120 (90% are PAHs) 1.1	Malins *et al.*, 1985a; b
English sole, *Parophrys vetulus*	Puget Sound (Wash.) Eagle Harbour Dunawish Waterway Useless Bay	One or more types of lesions 90 67.2 6.2	B[*a*]P: 2.3; Flu: 59 B[*a*]P: 0.073; Flu: 0.44 B[*a*]P: 0.005; Flu: 0.019	Krahn *et al.*, 1986
English sole, *Parophrys vetulus*	Vancouver Harbour (B.C.) Port Moody Arm Burrard Narrow Central Channel	Hepatic tumours 75 30 8	Total PAHs 3.1 to 36.8 nr 2.5	Goyette *et al.*, 1988

[1] B[*a*]P - Benzo[*a*]pyrene; Flu - Fluoranthene; Phe - Phenanthrene; Pyr - Pyrene; nd - not detected; nr - not reported

concentrations measured in the fish were correlated with the concentrations in the sediments where the fish were taken. Moreover, the chromatographic profiles for the PAHs in the sediments and the fish were similar. Therefore, it is likely that a portion of the PAHs in the fish was taken up by ingestion of contaminated sediments during feeding. In another study, two species of fish from the Buffalo (New York) and Detroit (Michigan) rivers along the Great Lakes had a high incidence of liver cancer (27 and 20% respectively; Maccubbin *et al.*, 1990). Sediments in these two rivers are highly contaminated by many chemicals, including B[*a*]P (0.12 to 17.7 µg/g), fluorene (0.12 to 34.6 µg/g), and pyrene (0.12 to 38.8 µg/g). To determine the role of these compounds in the etiology of the tumours, the authors measured DNA adducts in fish livers. Several genotoxic aromatic compounds were identified in the nucleotide adducts. An analysis of fish bile revealed recent exposure to polyaromatic compounds.

In marine waters, cancerous hepatic lesions have been observed in English sole (*Parophrys vetulus*) in Vancouver Harbour (Goyette *et al.*, 1988; Brand and Goyette, 1989). The authors reported a high incidence of tumours (75%) where sediments were highly contaminated by hydrocarbons and PAHs (from 3.1 to 6.6 µg/g of total PAHs), including B[*a*]P and the [*b*] and [*k*] isomers of benzofluoranthene. Moderate frequencies of tumours (8 and 30%) were observed at other sites in the harbour.

Malins *et al.* (1984) studied the frequency of hepatic neoplastic and other lesions in English sole (*Parophrys vetulus*) collected in Puget Sound in Washington. They examined the relationship between the aromatic compounds present in sediments and the diet of the fish (Malins *et al.*, 1984; 1985a;b). They observed a high frequency (27%) of cancerous hepatic lesions in English sole in Eagle Harbour of Puget Sound. The sediments in the harbour contained particularly high concentrations of aromatic hydrocarbons (from 2.8 to 120 µg/g, depending on the site), 90% of which were PAHs. At a control site with much lower contaminant concentrations (aromatic content: 1.1 µg/g), the bottom-dwelling fish showed no such hepatic lesions. Moreover, higher concentrations of aromatic hydrocarbons were observed in the stomach contents of sole from Eagle Harbour, along with higher PAH metabolite concentrations in their bile. McCain *et al.* (1990) also found higher PAH levels in the stomach content and higher B[*a*]P metabolites in the bile of juvenile chinook salmon (*Oncorhynchus tsawytscha*) caught in the Duwamish Waterway (discharging in Puget Sound near Seattle, Washington) than those found at the reference site. The salmon collected in Duwamish Waterway showed a reduced immunological memory; the consequence of this reduction in disease resistance is unknown (Arkoosh *et al.*, 1991).

Krahn *et al.* (1986) statistically compared mean PAH metabolite concentrations in the bile of English sole with the incidence of hepatic tumours in the same fish collected at 11 sites in Puget Sound. They found a significant positive correlation between the incidence of neoplasms and other liver lesions and the concentration of PAH metabolite in the fish. These findings provided additional evidence of the relationship between PAHs and cancerous hepatic tumours in bottom-dwelling fish. Since PAHs are rarely the only contaminants present in sediments, their specific role in altering the health of fish is not fully understood. In a review of studies published on liver carcinogenicity, Myers

(1990) noted that the livers of English sole collected from Puget Sound were affected by a broad range of hepatic lesions. The author advanced the hypothesis that these lesions are related to PAH contaminants, on the basis of the statistical associations drawn between PAH levels in sediments and lesion frequencies, and the significant correlations between lesion frequency and the level of aromatic compound metabolites (including numerous PAH metabolites) present in the bile.

Shugart (1990) analyzed B[*a*]P DNA adducts in the brains of belugas (*Delphinapterus leucas*) in the St. Lawrence and the Mackenzie River estuaries. Concentrations of 69 to 206 ng of B[*a*]P adducts per gram of DNA were observed in belugas from the St. Lawrence, while no adducts where detected in those from the Mackenzie.

In terrestrial mammals, higher concentrations of B[*a*]P adduct (geometric mean of 69.9 pmol of B[*a*]P adduct/g of albumin and 7.6 pmol of B[*a*]P adduct/g of hemoglobin) have been detected in the blood proteins of wild marmots (*Marmota monax*) captured 10 km from an aluminum smelter in the Saguenay region than in marmots from a control area (geometric mean of 7.6 pmol of B[*a*]P adduct/g of albumin and 0.4 pmol of B[*a*]P adduct/g of hemoglobin) located in Saint Roch des Aulnaies, Quebec (Blondin and Viau, 1991; 1992).

3.0 Assessment of "Toxic" Under CEPA

3.1 CEPA 11(a) Environment

Polycyclic aromatic hydrocarbons are emitted into the Canadian environment from both natural and anthropogenic sources. Forest fires are the single most important natural source of PAHs in Canada. Since forest fires are separated both in time and space across the country, and release PAHs to the atmosphere, a dispersive medium, they do not result in continuous or chronic release of, or exposure to, PAHs in any specific area. Anthropogenic sources are numerous and result in emissions of PAHs into all environmental compartments. The largest anthropogenic sources of PAHs to the atmosphere are aluminum smelters using the Horizontal Stud Söderberg smelting process. Other important sources to the atmosphere include residential heating with wood, agricultural burning, and burning of wood wastes. Major sources of PAHs to the aquatic and soil environments include creosote-treated products, spills of petroleum products, metallurgic and coking plants, and deposition of atmospheric PAHs. Most anthropogenic sources are point sources releasing PAHs regularly, whether continuously or intermittently (e.g., smelters, coking and metallurgic plants), or diffuse sources releasing PAHs in a circumscribed place or time (e.g., urban sources such as vehicle emissions or wood burning), and can therefore result in chronic exposure of biota to PAHs.

With the exception of some of the lighter compounds, PAHs are relatively non-volatile and of low solubility in water. In the atmosphere, they are mostly found adsorbed to particulate matter that can be deposited to water and soil. In aquatic systems, PAHs adsorb to suspended matter in the water column and are deposited in sediments where they will persist; heavy PAHs, such as benzo[*a*]pyrene, can persist in sediments for several years. Polycyclic aromatic hydrocarbons are removed from soils mainly by volatilization and by microbial activities. Thus, with most PAHs being emitted into the atmosphere, the atmosphere is the major medium for the transport of PAHs, while sediments are the major environmental sink for these compounds.

Polycyclic aromatic hydrocarbons have been measured in air, soil, groundwater, fresh and marine surface water, sediments, and terrestrial and aquatic biota in Canada. Concentrations in all media are generally higher in areas close to known anthropogenic sources of PAHs.

Considerable data are available on toxicity of PAHs to aquatic organisms. In reviewing these data, emphasis was placed on ecologically relevant test end-points for species occurring in Canada or for related species. Since toxicity of many PAHs is photo-activated, it is likely that those studies done in the dark or under conditions of ambient laboratory lighting underestimate the toxicity of PAHs in surface waters.

Concentrations of PAHs in Canada in ambient waters (i.e., not close to anthropogenic sources) are relatively low. The highest concentration for total PAHs in ambient water was 1.8 µg/L, measured shortly after ice breakup in the Mackenzie River.

Concentrations of PAHs reported in ambient water are a few to several orders of magnitude lower than the lowest effects thresholds reported.

The highest concentrations of PAHs in the water column were reported by Wan (1993) in drainage ditches along railway lines (total PAHs of 5356 µg/L) and utility rights-of-way (total PAHs of 23 µg/L), where PAHs were released from utility poles or railway ties treated with creosote. Based on limited sampling, median concentrations in railway ditches with poles (Table 4) were close to or greater than the effects thresholds recorded for aquatic biota (Table 7) for eight of the PAHs selected for this assessment: acenaphthene, fluorene, phenanthrene, anthracene, pyrene, fluoranthene, benzo[*a*]anthracene, and benzo[*a*]pyrene. Mean concentrations in utility right-of-way ditches (Table 4) were close to or greater than the effects thresholds recorded for aquatic biota (Table 7) for three of the PAHs selected for this assessment: phenanthrene, anthracene, and pyrene. No data were identified that would allow the estimation of effects to biota in receiving waters.

Therefore, based on concentrations of PAHs recorded in surface water in Canada and on laboratory-derived toxic effects thresholds, PAHs in ambient water likely do not directly affect aquatic biota, while PAHs in railway and utility ditches could adversely affect biota.

Numerous sites where sediments are contaminated with PAHs were identified in Canada. Sites discussed in this section were selected because concentrations of PAHs in sediments and the biota at the sites are relatively well characterized.

The discharge of wastes from a coking facility resulted in the formation of the tar ponds on the tidal flats of Muggah Creek in Sydney Harbour, Nova Scotia. Although the coking facility was closed in 1988, it is estimated that close to 1 tonne/year of PAHs are released in the estuary by tidal influence. It is recognized that the tidal flats of Muggah Creek contaminated with PAHs are devoid of living organisms. Generally, both the diversity and abundance of benthic organisms increase with distance from Muggah Creek. Also, it has been demonstrated that benthic communities in the more contaminated South Arm of the estuary were less diversified than those from the Northwest Arm.

Hamilton Harbour is a highly contaminated site in the Great Lakes, with the steel mills located close to the harbour being the main source of PAHs. Although they are not the only contaminants at this site, PAHs have been associated with mortality of nymphs of the mayfly, *Hexagenia limbata*, in toxicity tests conducted under laboratory conditions (Krantzberg and Boyd, 1992; Murphy *et al.*, 1993). Sediments from a location in the harbour highly contaminated by PAHs induced nearly 100% mortality even after the sediments were chemically treated to reduce toxicity associated with metals.

Kettle Creek at Port Stanley, Ontario, is another site heavily contaminated with PAHs because of the presence of a former oil gasification complex. AEL (1988) reported a correlation between the concentrations of PAHs in the sediments (Canviro, 1988) and the abundance of the oligochaetes *Limnodrilus cervix* and *L. hoffmeisteri.*

These worms are generally recognized as tolerant to contaminated environmental conditions and were the most abundant species in the study area.

Therefore, evidence from field studies and from laboratory studies using field samples indicate that biota are adversely affected at various Canadian sites contaminated by PAHs of different industrial origins.

Under laboratory conditions, neoplastic and genotoxic effects have been associated with exposure to PAHs for both terrestrial and aquatic organisms. Field evidence is supportive of such an association. In terrestrial systems, the only field evidence available is limited to that demonstrating exposure and possible preliminary stages of chemically induced carcinogenesis. For example, an increase has been observed in the incidence of B[*a*]P DNA adducts in the blood proteins of marmots captured downwind of an aluminum smelter in the Saguenay region. They have also been observed in the brain of aquatic mammals (i.e., beluga whales) in the St. Lawrence River. The ultimate neoplastic and genotoxic effects to wildlife resulting from chronic exposure to PAHs are not known.

In the case of bottom-dwelling fish, the evidence demonstrating that exposure to B[*a*]P is associated with cancer induction is stronger. Injecting organic extracts from Hamilton Harbour sediments into the sac fry of rainbow trout induced malignant hepatic tumours similar to those induced by PAHs such as B[*a*]P and 7,12-dimethylbenzanthracene (Metcalfe *et al.*, 1990). Under laboratory conditions, English sole and the starry flounder metabolized B[*a*]P to intermediates that bind to hepatic DNA and develop chemically induced hepato-carcinogenesis (Stein *et al.*, 1990). Also under laboratory conditions, Schiewe *et al.* (1991) demonstrated that English sole parenterally exposed to extracts from sediments contaminated with high levels of aromatic hydrocarbons or to benzo[*a*]pyrene developed neoplastic hepatic lesions. In Canada, similar cancerous hepatic lesions have been observed in English sole in Vancouver harbour (Goyette *et al.*, 1988; Brand and Goyette, 1989). These authors reported tumour frequencies of up to 75% in English sole caught in Port Moody where sediments are highly contaminated by PAHs, including carcinogens such as B[*a*]P and benzofluoranthene ([*b*] and [*k*] isomers). Lower tumour frequencies were reported in other areas of the harbour.

Therefore, laboratory and field evidence indicates that PAHs induce neoplastic and genotoxic effects in aquatic biota. Data for mammals indicate that these animals may be susceptible to such effects, but no studies were identified documenting such effects in wild mammals.

Therefore, based on the neoplastic, genotoxic, and population-level effects observed in aquatic biota at sites contaminated with PAHs across Canada, PAHs are considered to be entering the environment in a quantity or concentration or under conditions that are having a harmful effect on the environment.

3.2 CEPA 11(b) Environment on Which Human Life Depends

Polycyclic aromatic hydrocarbons generally only weakly absorb light of wavelengths critical to global warming. Unlike substances associated with depletion of stratospheric ozone (Firor, 1989), PAHs are non-halogenated compounds of low to moderate persistence in the atmosphere. Given these properties and the low steady-state concentrations of PAHs in the atmosphere, they are not considered to contribute significantly to stratospheric ozone depletion, global warming, or ground-level ozone formation.

Therefore, based on available data, PAHs are not considered to be entering the environment in a quantity or concentration or under conditions that constitute a danger to the environment on which human life depends.

3.3 CEPA 11(c) Human Life or Health

Based on the limited available data on concentrations of PAHs in drinking water in Canada, it is likely that it contributes negligibly to total intake. Although data are available on the concentrations in food of several of the PAHs considered principally in this assessment, analytical recoveries were poor. Moreover, levels in food vary considerably depending on cooking habits and food sources. Available data were also inadequate to develop estimates of carcinogenic potency of a broad range of PAHs in the gastrointestinal tract following ingestion. The current assessment focuses, therefore, on exposure in ambient air.

Owing to the possible confounding by concomitant exposure to other substances that may have contributed to observed effects, available epidemiological data are considered inadequate to assess the health risks (including carcinogenicity) of PAHs in humans. The five PAHs considered principally in the assessment of potential risks to human health (benzo[*a*]pyrene, benzo[*b*]fluoranthene, benzo[*j*]fluoranthene, benzo[*k*]fluoranthene, and indeno[*1,2,3-cd*]pyrene) are classified in Group II ("Probably Carcinogenic to Humans") of the classification scheme for carcinogenicity developed for the determination of "toxic" under Paragraph 11(c) of CEPA (EHD, 1994). This classification is based primarily on the results of carcinogenicity bioassays in which PAHs have been administered to experimental animals by inhalation (B[*a*]P only) and dermal application, and supporting data from studies involving routes of administration less relevant to environmental exposure (i.e., direct introduction into body cavities or tissues) and information on genotoxicity and mechanism of action. For such substances, where data permit, the estimated total daily intake or concentrations in relevant environmental media are compared to quantitative estimates of carcinogenic potency to characterize risk and provide guidance for further action (i.e., analysis of options to reduce exposure) (EHD, 1994). The carcinogenic potency is expressed as the dose or concentration that induces a 5% increase in the incidence of relevant tumours ($TD_{0.05}$).

In order to calculate the exposure/carcinogenic potency indices (EPIs) for PAHs, two approaches have been adopted. One is based on the assumption that the carcinogenic potency of each of the components of a mixture of PAHs is equivalent to that of B[*a*]P on a weight basis; the other is based on calculation of relative carcinogenic potencies for several PAHs for which the data base is considered sufficient. Both of these approaches have considerable limitations (as described in this section); moreover, exposure from media other than air has not been taken into account*. The values are, therefore, considered "interim", pending generation of more suitable data for quantitative estimation of exposure and potency.

For the B[*a*]P equipotency approach, the $TD_{0.05}$ for inhaled B[*a*]P has been estimated based on multi-stage modelling of the respiratory tract tumours in Syrian golden hamsters in the study reported by Thyssen *et al.* (1981). The highest exposure group was eliminated from the analysis due to the low average survival time. The $TD_{0.05}$ for B[*a*]P estimated in this manner is 1.57 mg/m^3. Estimated interim EPIs for populations residing in the vicinity of aluminum smelters (Kitimat, Jonquière, and Shawinigan) developed based on this likely conservative approach and the total concentrations of the 17 to 28 PAHs at these locations (156 to 1690 ng/m^3) determined in the national survey (Ringuette *et al.*, 1993) range from 1.0×10^{-4} to 1.1×10^{-3} (156 to 1690 ng/m^3 +1.57 mg/m^3). Therefore, based on this approach, the priority for analysis of options to reduce exposure solely on considerations of potential health effects would be high.

This approach is necessarily limited to only those PAHs that have been monitored in ambient air and does not account, therefore, for all PAHs to which the general population is exposed. Also, the small increases in the incidence of tumours at sites other than the respiratory tract in the study by Thyssen *et al.* (1981) have not been taken into account. Nevertheless, it is likely that the carcinogenic potency of PAH mixtures is considerably overestimated based on the assumption that the potency of all PAHs is similar to that of B[*a*]P since the weight of evidence of carcinogenicity for each of the monitored PAHs has not been considered. Moreover, on the basis of studies in which the carcinogenicity of various PAHs has been examined principally by application to the skin of mice, the potency of B[*a*]P has consistently been greater than that of most other compounds (see, for example, U.S. EPA, 1982). In addition, there are some data that indicate that some PAH-containing mixtures containing B[*a*]P are less potent than B[*a*]P alone (Slaga *et al.*, 1980). Furthermore, based on determination of the incidence of tumour formation after exposure to a combination of two or more PAHs, effects were not additive (see, for example, Grimmer, 1983).

With respect to the alternative approach, however, data available to serve as a basis for estimating the relative potencies of various PAHs in the target organ (i.e., the lung)

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* On the basis of the limited available data on concentrations of PAHs in food (Das, 1987), the estimated intake (based on B[*a*]P equivalents) of three of the selected PAHs (i.e., B[*a*]P, B[b]F, and B[k]F) for an adult (EHD, 1994) is approximately two-fold greater than that inhaled; however, it is likely that there are considerable variations in toxicokinetics and potency by ingestion versus inhalation.

relevant to one of the principal media of exposure in the general environment for which data were sufficient to estimate exposure (i.e., air), are very limited. Indeed, only two adequate studies have been identified relevant to estimation of potency in the lung for a sufficiently broad range of PAHs, those of Thyssen *et al.* (1981), in which there was clear evidence of an exposure-response relationship between inhaled B[*a*]P and respiratory tract tumours in Syrian golden hamsters, and Deutsch-Wenzel *et al.* (1983), in which there were exposure-response relationships for epidermoid carcinomas and pleomorphic sarcomas in Osborne-Mendel female rats administered B[*a*]P, B[*b*]F, IND, B[*k*]F, B[*j*]F, and ANT by pulmonary implantation.

For the relative potency approach, carcinogenic potencies were estimated for the selected PAHs on the basis of multi-stage modelling of tumour incidence (epidermoid carcinomas) in Osborne-Mendel rats exposed in the study by Deutsch-Wenzel *et al.* (1983) to each of the PAHs compared to that in the "solvent-vehicle"-exposed controls. Values were based on the dose that induced a 5% increase in the incidence of relevant tumours calculated on the basis of each fit. The potencies of B[*b*]F, B[*j*]F, B[*k*]F, and IND relative to that of B[*a*]P were computed by dividing the dose calculated to be associated with a 5% increase in tumours for B[*a*]P by those for each compound. The relative carcinogenic potency factors estimated on this basis were 0.06 for B[*b*]F, 0.05 for B[*j*]F, 0.04 for B[*k*]F, and 0.12 for IND (and 1 for B[*a*]P).

B[*a*]P equivalents for the five PAHs addressed in this assessment were calculated by multiplying the concentrations in ambient air by the relative carcinogenic potency factors derived above (Table 9). The values for total B[*a*]P equivalents/m^3 range from 2.72 to 48.98 ng/m^3 for cities near aluminum smelters using Horizontal or Vertical Stud Söderberg processes, 2.42 to 9.12 ng/m^3 for cities where wood stoves are commonly used, 0.13 to 2.25 ng/m^3 for urban cities, and 0.11 ng/m^3 for a rural area.

The EPIs have been calculated on the basis of the $TD_{0.05}$ for inhaled B[*a*]P estimated based on multistage modelling of the respiratory tract tumours in Syrian golden hamsters in the study reported by Thyssen *et al.* (1981), as described above for the B[*a*]P equipotency approach, and the B[*a*]P equivalents in ambient air at different types of sites in Canada for the five specified PAHs. For example, "interim" EPIs for the general population in Canada living near aluminum smelters that use the Horizontal or Vertical Söderberg process (i.e., the population that has the highest exposure to the selected PAHs) range from 1.7×10^{-6} to 3.1×10^{-5} (2.72 to 48.98 ng of B[*a*]P equivalent/m^3 +1.57 mg of B[*a*]P/m^3). On the basis solely of considerations of potential health effects, based on this approach, therefore, the priority for further action (i.e., analysis of options to reduce exposure) would be considered to be moderate.

This approach to estimating relative potency is considered to offer several advantages over those reported previously by, for example, Thorslund and Charnley (1988) and Slooff *et al.* (1989). Estimation of potency is restricted to the target organ for the route of exposure for which sufficient data are available to estimate exposure of the general population (*i.e.*, inhalation in air). In addition, potency estimates for the other PAHs considered here can be compared directly to that for B[*a*]P in the same

Table 9 Levels of PAHs in B[*a*]P Equivalents/m^3 in Canadian Ambient Air

PAH *	Relative carcinogenic potency factors	ng B[*a*]P equivalent/m^3 **			
		A***	B***	C***	D***
B[*a*]P	1.0	2.06 to 36.37	1.93 to 7.66	0.09 to 1.74	0.08
B[*b*]F	0.06	0.27 to 5.59	0.19 to 0.43	0.01 to 0.15	0.01
B[*j*]F	0.05	0.10 to 2.08	0.07 to 0.23	0.01 to 0.06	0.01
B[*k*]F	0.04	0.06 to 1.27	0.04 to 0.10	<0.01 to 0.07	<0.01
IND	0.12	0.23 to 3.67	0.19 to 0.70	0.02 to 0.23	0.01
Total		2.72 to 48.98	2.42 to 9.12	0.13 to 2.25	0.11

* B[*a*]P = benzo[*a*]pyrene; B[*b*]F = benzo[*b*]fluoranthene; B[*j*]F = benzo[*j*]fluoranthene; B[*k*]F = benzo[*k*]fluoranthene; IND = indeno[*1,2,3-cd*]pyrene

** ng B[*a*]P equivalent/m^3 = mean concentrations in ambient air (ng/m^3) x relative potency factor

*** A = cities near aluminum smelters (Jonquière, Kitimat, Shawinigan)
B = wood stove burning (Whitehorse, Sept-Îles)
C = cities (Montreal, Sydney, Toronto, Vancouver, Windsor, Winnipeg)
D = rural area (Walpole Island)

experimental model. Moreover, the weight of evidence for carcinogenicity and study quality has been taken into account, factors that have often not been adequately considered in the previous estimations of potency for PAHs. However, since the type of respiratory tumours induced in the study by Thyssen *et al.* (1981) were not specified, it is unknown whether they were similar to those induced following implantation. In addition, only tumours of the respiratory tract have been taken into account in estimating carcinogenic potency. It should also be recognized that these "interim" EPIs based on relative carcinogenic potencies include only a very small proportion of PAHs in the general environment, for which the available data on concentrations and relative carcinogenic potency were considered adequate [i.e., 0.1% to 23.5% of a total of 13 PAHs determined at various locations in a recent survey of ambient air in Canada (Dann, 1992a)].

Based on the two approaches presented here, both of which are limited by the inadequacies of the data base, the resulting "interim" EPIs and, hence, priority for further action (i.e., analysis of options to reduce exposure) would be moderate to high, on the basis solely of consideration of the potential effects on health of selected polycyclic aromatic hydrocarbons.

On the basis of available data, the PAHs benzo[*a*]pyrene, benzo[*b*]fluoranthene, benzo[*j*]fluoranthene, benzo[*k*]fluoranthene, and indeno[*1,2,3-cd*]pyrene are classified as

"Probably Carcinogenic to Humans" (*i.e.*, substances for which there is considered to be some probability of harm for the critical effect at any level of exposure) and considered to be "toxic" as defined under Paragraph 11(*c*) of the *Canadian Environmental Protection Act*. This approach is consistent with the objective that exposure to substances for which the critical effect is considered not to have a threshold should be reduced wherever possible and obviates the need to establish an arbitrary *de minimis* level of risk for determination of "toxic" under the Act.

Therefore, based on available data, the polycyclic aromatic hydrocarbons benzo[*a*]pyrene, benzo[*b*]fluoranthene, benzo[*j*]fluoranthene, benzo[*k*]fluoranthene, and indeno[*1,2,3-cd*]pyrene are entering the environment in a quantity or concentration or under conditions that may constitute a danger to human life or health.

3.4 Conclusion

Therefore, based on available data, polycyclic aromatic hydrocarbons are entering the environment in a quantity or concentration or under conditions that are having or may have a harmful effect on the environment. They are not considered to be entering the environment in a quantity or concentration or under conditions that constitute or that may constitute a danger to the environment on which human life depends. The polycyclic aromatic hydrocarbons benzo[*a*]pyrene, benzo[*b*]fluoranthene, benzo[*j*]fluoranthene, benzo[*k*]fluoranthene, and indeno[*1,2,3-cd*]pyrene are considered to be entering the environment in a quantity or concentration or under conditions that may constitute a danger to human life or health.

4.0 Recommendations for Research

To permit a more comprehensive assessment, it is recommended that the following research be considered.

- Development of experimental and theoretical models for estimation of the carcinogenic potency of a broad range of PAHs. In particular, bioassays in which animals are exposed by routes by which the general population is principally exposed (i.e., inhalation and ingestion) to a range of PAHs selected to characterize a range of carcinogenic potencies are desirable (high priority).

- Development of analytical methods and additional characterization of concentrations of a broader range of PAHs in ambient air, drinking water, food, and soil (high priority).

- The highest concentrations of PAHs in water were reported from ditches along railway and utility lines. Given the prevalence of railway and utility lines throughout the country, it is recommended that data on effects on biota in the ditches and in receiving waters should be determined (medium priority).

- The neoplastic and related effects of chronic exposure to PAHs should be determined for terrestrial organisms, notably mammals, in areas adjacent to major atmospheric sources of PAHs (low priority).

- To better characterize the effects of PAHs in sediments on fish, fish should be exposed to individual sediment-bound PAHs using environmentally relevant exposure protocols (low priority).

5.0 References

Abernathy, S., A.M. Bobra, W.Y. Shiu, P.G. Wells, and D. Mackay, "Acute Lethal Toxicity of Hydrocarbons and Chlorinated Hydrocarbons to Two Planktonic Crustaceans: The Key Role of Organism-water Partitioning", *Aquat. Toxicol.*, *8*:163-174 (1986).

AEL (Aquatic Ecostudies Limited), "Effect of Polynuclear Aromatic Hydrocarbons on the Benthic Invertebrate Fauna of Kettle Creek at Port Stanley, Ontario", prepared for the Ontario Ministry of the Environment, 21 p. (1988).

Agriculture Canada, "Announcement A 92-02: Re-evaluation of Heavy Duty Wood Preservatives", Agriculture Canada, Food Production and Inspection Branch, Pesticides Directorate, July 2, 1992.

AMAI (André Marsan et Associés Inc.), "LaSalle Coke : étude de caractérisation", prepared for Environment Canada, Environmental Protection, Montreal, Que. (1986a).

AMAI (André Marsan et Associés Inc.), "Monsanto : étude de caractérisation", prepared for Environment Canada, Environmental Protection, Montreal, Que. (1986b).

ARGUS Groupe Conseil Inc., "Les hydrocarbures aromatiques polycycliques dans les sols de sites industriels canadiens", prepared for Environment Canada, Conservation and Protection, Quebec Region, Montreal, Que., 32 p. (1991).

Arkoosh, M.R., E. Casillas, E. Clemons, B. McCain, and U. Varanasi, "Suppression of Immunological Memory in Juvenile Chinook Salmon (*Onchorynchus tsawytscha*) from an Urban Estuary", *Fish Shellfish Immunol.*, *1*:261-277 (1991).

Atkinson, R., K.R. Darnall, A.C. Lloyd, A.M. Winer, and J.N Pitts, Jr., in: *Advances in Photochemistry*, Vol. II, J.N. Pitts. Jr., TG.S. Hammond, and K. Gollnick (eds.), Wiley Publ., New York, NY, p. 375 (1979).

Atwell, L., P. Hennigar, J. Kozak, M. Morin, and C. Oldreive, "Ambient Air-Polynuclear Aromatic Hydrocarbons Study, Sydney, Nova Scotia", Environment Canada, Environmental Protection Service, Atlantic Region and Nova Scotia Department of Environment, Halifax, N.S., 79 p. (1984).

ATSDR (Agency for Toxic Substances and Disease Registry), "Toxicological Profile for Benzo(*a*)pyrene", ATSDR/TP-88-05 (1990a).

ATSDR (Agency for Toxic Substances and Disease Registry), "Toxicological Profile for Polycyclic Aromatic Hydrocarbons", ATSDR/TP-90-20 (1990b).

Ayotte, P. and M. Larue, "Micropolluants organiques : campagnes d'échantillonnage printemps/été 1987 et hiver 1988", Quebec Government, Ministry of the Environment, Urban Ecosystems Branch, 178 p. (1990).

Bastian, M.V. and D.W. Toetz, "Effect of Eight Polynuclear Hydrocarbons on Growth of *Anabaena flos-aquae*", *Environ. Contam. Toxicol.*, *29*:531-538 (1982).

Bastian, M.V. and D.W. Toetz, "Effect of Polynuclear Hydrocarbons on Algal Nitrogen Fixation", *Bull. Environ. Contam. Toxicol.*, *35*:258-265 (1985).

Baumann, P.C. and M. Mac, "Polycyclic Aromatic Hydrocarbons and Tumours in Brown Bullheads from the Black and Cuyahoga Rivers - Cause and Effects", *Can. Tech. Rep. Fish Aquat. Sci.*, 26 p. (1988).

Baumann, P.C. and D.M. Whittle, "The Status of Selected Organics in the Laurentian Great Lakes: An Overview of DDT, PCBs, Dioxins, Furans and Aromatic Hydrocarbons", *Aquat. Toxicol.*, *11*:241-257 (1988).

Beak, "Landspreading of Sludges at Canadian Petroleum Facilities", prepared for the Petroleum Association for Conservation of the Canadian Environment, no. 81-5A, Ottawa, Ont., 97 p. + appendices (1981).

Bedient, P.B., A.C. Rodgers, T.C. Bouvette, M.B. Tomson, and T.H. Wang, "Ground-water Quality at a Creosote Waste Site", *Ground Water*, *22*:318-329 (1984).

Behymer, T.D. and R.A. Hites, "Photolysis of Polycyclic Aromatic Hydrocarbons Adsorbed on Fly Ash", *Environ. Sci. Technol.*, *22*:1311-1319 (1988).

Bezak, D., Manitoba Department of the Environment (unpublished data) (1990).

Bjørseth, A. and T. Ramdhal, *Handbook of Polycyclic Aromatic Hydrocarbons*, Vol. 2, Marcel Dekker (Publ.), New York, NY, 416 p. (1985).

Bjørseth, A, *Handbook of Polycyclic Aromatic Hydrocarbons*, Marcel Dekker (Publ.), New York, NY, 727 p. (1983).

Bjørseth, A., G. Lunde and A. Lindskog, "Long-range Transport of Polycyclic Aromatic Hydrocarbons", *Atmos. Environ.*, *13*:45-53 (1979).

Black, J.A., W.J. Birge, A.G. Westerman, and P.C. Francis, "Comparative Aquatic Toxicology of Aromatic Hydrocarbons", *Fund. Appl. Toxicol.*, *3*:353-358 (1983).

Blondin, O. and C. Viau, "PAH-blood Protein Adducts in Wild Woodchucks: a Useful Bioindicator of Environmental Contamination", 30th Annual Meeting, Society of Toxicology, February 25-March 1, 1991, Dallas, TX (1991).

Blondin, O. and C. Viau, "Benzo(*a*)pyrene Protein Adducts in Wild Woodchucks Used as Biological Sentinels of Environmental Polycyclic Aromatic Hydrocarbons Contamination", *Arch. Environ. Contam. Toxicol., 23*: 310-315 (1992).

Boyd, J., B. Raymond, and D. Goyette, "Vancouver Harbour Benthic Environmental Quality Studies. Trace Metals and Organic Compounds in Sediments - October 1987", Environment Canada, Conservation and Protection, Pacific and Yukon Region, Vancouver, B.C., 62 p. (1989).

Brand, D. and D. Goyette, "Further Studies on the Prevalence of Idiopathic Liver Lesions in English Sole, *Parophrys vetulus*, from Vancouver Harbor, British Columbia, 1987", Environment Canada, Conservation and Protection, Pacific and Yukon Region, Vancouver, B.C., 30 p. (1989).

Brunström, B., D. Broman, and C. Näf, "Embryotoxicity of Polycyclic Aromatic Hydrocarbons (PAHs) in Three Domestic Avian Species, and of PAHs and Coplanar Polychlorinated Biphenyls (PCBs) in the Common Eider", *Environ. Pollut., 67*:133-143 (1990).

Bulman, T.L., S. Lesage, P.J.A. Fowlie, and M.D. Webber, "The Persistence of Polynuclear Aromatic Hydrocarbons in Soil", prepared for PACE (Petroleum Association for Conservation of the Canadian Environment), Ottawa, Ont., 50 p. + appendices (1985).

Call, D.J., L.T. Brooke, S.L. Harting, S.H. Poirier, and D.J. McCauley, "Toxicity of Phenanthrene to Several Freshwater Species", Center for Lake Superior Environmental Studies, University of Wisconsin, Superior, WI (1986).

Canviro, "PAH Sediment Contamination in Kettle Creek, Port Stanley: Site Investigations and Remedial Recommendations", prepared for the Ontario Ministry of the Environment, London, Ont., by Canviro Consultants, Waterloo, Ont. (1988).

Casserly, D.M., E.M. Davis, T.D. Downs, and R.K. Guthrie, "Sorption of Organics by *Selenastrum capricornutum", Water Res., 17*:1591-1594 (1983).

CCME (Canadian Council of Ministers of the Environment), "Interim Canadian Environmental Quality Criteria for Contaminated Sites", CCME Subcommittee on Environmental Quality Criteria for Contaminated Sites, Report CCME-EPC-CS34, Winnipeg, Man., 20 p. (1991).

CCME (Canadian Council of Ministers of the Environment), "Interim Guidelines for PAH Contamination at Abandoned Coal Tar Sites" Environment Canada, Ottawa, Ont., CCME-TS/WM-TRE004 (1989).

Chapman P.M., D.M. Munday, and G.A. Vigers, "Determination of Contaminant Levels in Fish Species from the Fraser River: Pollutant Ecology", Vancouver, B.C., Report no. 647, 25 p. (1981).

Christensen, E.R. and X. Zhang, "Sources of Polycyclic Aromatic Hydrocarbons to Lake Michigan Determined from Sedimentary Records", *Environ. Sci. Technol.*, *27*:139-146 (1993).

Cooper, P.A., "Leaching of Wood Preservatives from Treated Wood in Service", prepared for Public Works Canada, Ottawa, Ont., 79 p. (1991).

CRC, *Handbook of Chemistry and Physics*, R.C. Weast (ed.), CRC Press, Boca Raton, FL, 68th ed. (1987-1988).

Dann, T., "Polycyclic Aromatic Hydrocarbons in the Ambient Air of Toronto, Ontario and Montreal, Quebec", Pollution Measurement Division, Environment Canada, Ottawa, Ont., 20 p. (1989).

Dann, T., "Ambient Air PAH Concentrations Mixture Analysis", Pollution Measurement Division, Environment Canada, Ottawa, Ont. (unpublished, 1990).

Dann, T., personal communication to Environment Canada, Quebec Region, River Road Environmental Technology Centre, Ottawa, Ont. (1991).

Dann, T., personal communication, River Road Environmental Technology Centre, Ottawa, Ont. (1992a).

Dann, T., "Detroit Incinerator Monitoring Program. Data Report #2", River Road Environmental Technology Center, Environment Canada, Conservation and Protection, Ottawa, Ont., 15 p. (unpublished report) (1992b).

Das, B.S., "Analysis of Polycyclic Aromatic Hydrocarbons (PAH) in Canadian Foods", Contract report for Food Research Centre/Agriculture Canada Research Branch, Ottawa, Ont. (unpublished, 1987).

DeGraeve, G.M., R.G. Elder, D.C. Woods, and H.L. Bergmann, "Effects of Naphthalene and Benzene on Fathead Minnows and Rainbow Trout", *Arch. Environ. Contam. Toxicol.*, *11*:487-490 (1982).

Deutsch-Wenzel, R.P., H. Brune, G. Grimmer, G. Dettbarn, and J. Misfeld, "Experimental Studies in Rat Lungs on the Carcinogenicity and Dose-response Relationships of Eight Frequently Occurring Environmental Polycyclic Aromatic Hydrocarbons", *J. Natl. Cancer Inst.*, *71*:539-544 (1983).

Dobrowsky, C.J. and C.E. Epifanio, "Accumulation of Benzo(*a*)pyrene in a Larval Bivalve Via Trophic Transfer", *Can. J. Fish. Aquat. Sci.*, *37*:2318-2322 (1980).

Eadie, B.J., W.R. Faust, P.F. Landrum, N.R. Morehead, W.S. Gardner, and T. Nalepa, "Bioconcentration of PAH by Some Benthic Organisms of the Great Lakes", in: *Polynuclear Aromatic Hydrocarbons: Formation, Metabolism and Effects*, M. Cooke and A.J. Dennis (eds.), Batelle Press, Columbus, OH, pp. 437-450 (1983).

EAG (Environmental Application Group Ltd.), "The Environmental Toxicology of Polycyclic Aromatic Hydrocarbons", prepared for the Ontario Ministry of the Environment, Toronto, Ont., 296 p. (1990).

Edwards, N.T., "Polycyclic Aromatic Hydrocarbons (PAHs) in the Terrestrial Environment - A Review", *J. Environ. Qual., 12*:427-441 (1983).

Edwards, N.T., "Uptake, Translocation and Metabolism of Anthracene in Bush Bean *Phaseolus vulgaris*", *Environ. Toxicol. Chem. 5*:659-665 (1986).

EHD (Environmental Health Directorate), "Approach to the Assessment of 'Toxic' Under Paragraph 11(*c*) of CEPA", Bureau of Chemical Hazards, Health Canada, Ottawa, Ont. (in press, 1994).

Eisenreich, S.J., B.B. Looney, and J.B. Thornton, "Airborne Organic Contaminants in the Great Lakes Ecosystem (1981)". (As cited in NRCC, 1983.).

Eisler, R., "Polycyclic Aromatic Hydrocarbon Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review", United States Fish and Wildlife Service, Patuxent Wildlife Research Center, Laurel, MD (1987).

Envirochem, "An Inventory of Sources, Uses and Waste Disposal Practices of Creosote in Canada", prepared for Environment Canada, Environmental Protection Service, Western and Northern Region, Edmonton, Alta., 74 p. (1991).

Envirodat, Environmental Data Base, Environment Canada, Eco-health Branch, Ottawa, Ont. (1993).

Environment Canada, "Creosote Wood Preservation Facilities", Conservation and Protection, EPS 2/WP/1, Ottawa, Ont. (1988).

Environment Canada, "Atlantic Region Federal-Provincial Toxic Chemical Survey of Municipal Drinking Water Sources, Data Summary Report, Province of Nova Scotia, 1985-1988", Environmental Protection Service, Water Quality Branch, Atlantic Region, IWD-AR-WQB-89-154 (1989a).

Environment Canada, "Atlantic Region Federal-Provincial Toxic Chemical Survey of Municipal Drinking Water Sources, Data Summary Report, Province of New Brunswick, 1985-1988", Environmental Protection Service, Water Quality Branch, Atlantic Region, IWD-AR-WQB-89-155 (1989b).

Environment Canada, "Atlantic Region Federal-Provincial Toxic Chemical Survey of Municipal Drinking Water Sources, Data Summary Report, Province of Prince Edward Island, 1985-1988", Environmental Protection Service, Water Quality Branch, Atlantic Region, IWD-AR-WQB-89-156 (1989c).

Environment Canada, "Atlantic Region Federal-Provincial Toxic Chemical Survey of Municipal Drinking Water Sources, Data Summary Report, Province of Newfoundland, 1985-1988", Environmental Protection Service, Water Quality Branch, Atlantic Region, IWD-AR-WQB-89-157 (1989d).

Environment Canada, personal communication, D. Dubé, Commercial Chemicals Branch, Conservation and Protection, Hull, Quebec (1992).

Environnement Illimitée Inc., "Caractérisation de la qualité des sédiments du port de Montréal", report prepared for Environment Canada, Conservation and Protection, Quebec Region, Montreal, Que. (1990).

ETL (Enviro-Test Laboratory), "GC/MS Analysis - Gulf/Pincher Creek - Samples Within Process Area", prepared for Gulf Canada Inc. and Monenco Consultants Ltd., 48 p. (1984).

Faulkner, D.A., "Atmospheric Concentration of Polycyclic Aromatic Hydrocarbons (PAH) in Greater Vancouver", Scientific Services Division, Atmospheric Environment Service, Report PAES-85-1, Vancouver, B.C. (1985).

Finger, S.E., E.F. Little, M.G. Henry, J.F. Fairchild, and T.P. Boyle, "Comparison of Laboratory and Field Assessment of Fluorene. Part I: Effects of Fluorene on the Survival, Growth, Reproduction and Behavior of Aquatic Organisms in Laboratory Tests", in: *Validation and Predictability of Laboratory Methods for Assessing the Fate and Effects of Contaminants in Aquatic Ecosystems*, T.P. Boyle (ed.), ASTM, Philadelphia, PA, ASTM STP 865, pp. 120-133 (1985).

Firor, J., "*The Challenge of Global Warming*", D.E. Abrahamson (ed.), Island Press, Washington, D.C. (1989).

Gelboin, H.V., "Benzo(*a*)pyrene Metabolism, Activation, and Carcinogenesis: Role and Regulation of Mixed-function Oxidases and Related Enzymes", *Physiol. Rev.*, *60*:1107-1167 (1980).

Germain, A. and M. Bisson, "Mesure d'hydrocarbures aromatiques polycycliques dans l'air ambiant au Québec (Canada)", in: *Proceedings, 5th Conference on Toxic Substances, April 1-2, 1992. Montreal, Que.*, Environment Canada, pp. 47-57 (1992).

Germain, A. and C. Langlois, "Contamination des eaux et des sédiments en suspension du fleuve Saint-Laurent par les pesticides organochlorés, les biphényles polychlorés et d'autres contaminants organiques prioritaires", *Water Pollut. Res. J. Can.*, *23*:602-614 (1988).

Germain, A., F. Perron, and R. Van Coillie, "PAHs in the Environment: Fate, Ecotoxicity and Regulations", Environment Canada, Conservation and Protection, Quebec Region, Montreal, Que. (1993).

Goyette, D., "Sediments PAH - Vancouver Harbour, Kitimat Arm and Two B.C. Coastal Reference Areas", Environment Canada, Marine and Environmental Quality Branch, Pacific Region, Vancouver, B.C. (Unpublished, 1991).

Goyette, D. and J. Boyd, "Distribution and Environmental Impact of Selected Contaminants in Vancouver Harbour, British Columbia", Environment Canada, Pacific and Yukon Region, Vancouver, B.C., Regional Program Report 89-02, 99 p. (1989).

Goyette, D., D. Brand, and M. Thomas, "Prevalence of Idiopathic Liver Lesions in English Sole and Epidermal Abnormalities in Flatfish from Vancouver Harbor, British Columbia, 1986", Environment Canada, Conservation and Protection, Pacific and Yukon Region, Vancouver, B.C., 48 p. (1988).

Grimmer, G., "*Environmental Carcinogens: Polycyclic Aromatic Hydrocarbons. Chemistry, Occurence, Biochemistry, Carcinogenicity*", CRC Press Inc., Boca Raton, FL (1983).

Gschwend, P.M. and R.A. Hites, "Fluxes of Polycyclic Aromatic Hydrocarbons to Marine and Lacustrine Sediments in the Northeastern United States", *Geochim. Cosmochim. Acta.*, *45*:2359-2367 (1981).

Hallet, D. J., R.J. Norstrom, F.I. Onuska, and M.E. Comba, in: *Proceedings of the 2nd International Symposium on Glass Capillary Chromatography*, R.E. Keiser (ed.), May 1-5, Allgau/Bad Durkheim, Germany (1977).

Hallet, D.J., R.W. Brecher, J.O. Nriagu, and M.S. Simmons, "Cycling of Polynuclear Aromatic Hydrocarbons in the Great Lakes Ecosystem", *Adv. Environ. Sci. Technol.*, *14*:213-238 (1984).

Harrison, R.M., R. Perry, and R.A. Wellings, "Polycyclic Aromatic Hydrocarbons in Raw, Potable and Waste Waters", *Water Res.*, *9*:331-346 (1975).

Hawkins, W.E., W.W. Walker, R.M. Overstreet, J.C. Lyttle, and T.F. Lyttle, "Carcinogenic Effects of Some Polycyclic Aromatic Hydrocarbons on the Japanese Medaka and Guppy in Waterborne Exposures", *Sci. Total Environ.*, *94*:155-167 (1990).

Heinrich, U., F. Pott, U. Mohr, R. Fuhst, and J. Konig, "Lung Tumours in Rats and Mice After Inhalation of PAH-rich Emissions", *Exp. Pathol.*, *29*:29-34 (1986).

Herbes, S.E. and L.R. Schwall, "Microbial Transformation of Polycyclic Aromatic Hydrocarbons in Pristine and Petroleum-contaminated Sediments", *Appl. Environ. Microbiol.*, *35*:306-316 (1978).

Hildebrand, L.P., "Environmental Quality in Sydney and Northeast Industrial Cape Breton, Nova Scotia", Environment Canada, Environmental Protection Service, Contaminants Assessments Branch, Atlantic Region, Dartmouth, N.S., 89 p. (1982).

Hites, R.A. and P.M. Gschwend, "The Ultimate Fates of Polycyclic Aromatic Hydrocarbons in Marine and Lacustrine Sediments", in: *Polynuclear Aromatic Hydrocarbons: Physical and Biological Chemistry*, Sixth International Symposium, M. Cooke, A.J. Dennis, and G.L. Fisher (eds.), Battelle Press, Columbus, OH, pp. 357-366 (1982).

Hose, J.E., "Potential Use of Sea Urchin Embryos for Identifying Toxic Chemicals: Description of a Bioassay Incorporating Cytologic, Cytogenic and Embryologic Endpoints", *J. Appl. Toxicol.*, *5*:245-254 (1985).

Hose, J.E., J.B. Hannah, H.W. Puffer, and M.L. Landolt, "Histologic and Skeletal Abnormalities in Benzo(*a*)pyrene Treated Rainbow Trout Alevins", *Arch. Environ. Contam. Toxicol.*, *13*:675-684 (1984).

Huang, X.D., D.G. Dixon, and B.M. Greenberg, "Impacts of UV Radiation and Photomodification on the Toxicity of PAHs to Higher Plant *Lemna gibba* (Duckweed)", *Environ. Toxicol. Chem.*, *12*:1067-1077 (1993).

IARC (International Agency for Research on Cancer), "Polynuclear Aromatic Compounds, Part 1, Chemical, Environmental and Experimental Data", *IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans*, Vol. 32, Lyon, France, pp. 33-91 (1983).

IARC (International Agency for Research on Cancer), "Overall Evaluations of Carcinogenicity: An Updating of IARC Monographs Volumes 1 to 42", *IARC Monographs on the Evaluation of Carcinogenic Risk to Humans*, Suppl. 7, Lyon, France (1987).

Jackson, R.E., R.L. Patterson, B.W. Graham, J.M. Bahr, D. Bélanger, J. Lockwood, and M. Priddle, "Contaminant Hydrogeology of Toxic Organic Contaminants at a Disposal Site, Gloucester, Ontario. 1. Chemical Concepts and Site Assessment", Environment Canada, National Hydrology Research Institute Report No. 23, Inland Waters Directorate Scientific Series No. 141, Ottawa, Ont. (1985).

Johnson, D., "PAH Levels in Kitimat, B.C.", Ministry of the Environment of British Columbia (unpublished, 1991).

Kaiser, K.L.E., M.E. Comba, H. Hunter, R.J. Maguire, R.J. Tkacz, and R.F. Platford, "Trace Organic Contaminants in the Detroit River", *J. Great Lakes Res.*, *11*:386-399 (1985).

Kamens, R.M., Z. Guo, J.N. Fulcher, and D.A. Bell, "Influence of Humidity, Sunlight and Temperature on the Daytime Decay of Polyaromatic Hydrocarbons on Atmospheric Soot Particles", *Environ. Sci. Technol.*, *22*:103-108 (1988).

Keizer, P.D., Fisheries and Oceans, Biological Sciences Branch, Bedford Institute for Oceanography, Dartmouth, N.S. (unpublished, 1990).

Kieley, K.M., R.A.F. Matheson, and P.A. Hennigar, "Polynuclear Aromatic Hydrocarbons in the Vicinity of Two Atlantic Region Wood Preserving Operations", Environment Canada, Environmental Protection Service, Atlantic Region, Dartmouth, N.S., EPS-5-AR-86-3, 28 p. (1986).

Kieley, K.M., P.A. Hennigar, R.A.F. Matheson, and W.R. Ernst, "Polynuclear Aromatic Hydrocarbons and Heterocyclic Aromatic Compounds in Sydney Harbour- A 1986 Survey, Nova Scotia", Environment Canada, Environmental Protection Service, Atlantic Region, Dartmouth, N.S., EPS-5-AR-88-7, 41 p. (1988).

Korfmacher, W.A, E.L. Wehry, G. Mamantov, and D.F.S Natush, "Resistance to Photochemical Transformations of Polycyclic Aromatic Hydrocarbons Adsorbed on Coal Fly Ash", *Science*, *207*:1094-1099 (1980).

Krahn, M. M., L.D. Rhodes, M.S. Myers, L.K. Mooree, W.D. MacLeod, and D.C. Malins, "Associations Between Metabolites of Aromatic Compounds in Bile and the Occurrence of Hepatic Lesions in English Sole (*Parophrys vetulus*) from Puget Sound, Washington", *Arch. Environ. Contam. Toxicol.*, *15*:61-67 (1986).

Krantzberg, G and D. Boyd, "The Biological Significance of Contaminants in Sediment from Hamilton Harbour, Lake Ontario", *Environ. Toxicol. Chem.*, *11*:1527-1540 (1992).

Kuntz, K.W., Environment Canada, Water Quality Branch, Ontario Region, Burlington, Ont. (unpublished, 1990).

Landrum, P.F. and D. Scavia, "Influence of Sediment on Anthracene Uptake, Depuration and Biotransformation by the Amphipod *Hyalella azteca", Can. J. Fish. Aquat. Sci., 40*:298-305 (1983).

Lane, P. and Associates, Loucks Oceanology Ltd, and Manna Marine Enterprises Ltd., "Sydney Tar Ponds Clean-Up Marine Monitoring Implementation", Project E-222, prepared for Acres International Ltd. (1990).

Langlois, C., Environment Canada, Conservation and Protection, Quebec Region, Montreal, Que. (unpublished, 1989).

Lapierre, L., Environment Canada, Conservation and Protection, Quebec Region, Montreal, Que. (unpublished, 1989).

Larsen, P.F., D.F. Gadbois, and A.C. Johnson, "Polycyclic Aromatic Hydrocarbons in the Gulf of Maine Sediments: Distribution and Mode of Transport", *Mar. Environ. Res.*, *4*:231-244 (1986).

Laskin, S., M. Kuschner, and R.T. Drew, "Studies in Pulmonary Carcinogenesis", in: *Inhalation Carcinogenesis*, M.G. Hanna, Jr., P. Nettesheim, and J.R. Gilbert, (eds.), AEC Symposium Series No. 18, Oak Ridge, TN, Oak Ridge Division of Technical Information, U.S. Atomic Energy Commission, pp. 321-351 (1970).

LEI (Lavalin Environnement Inc.), "Étude sur les hydrocarbures aromatiques polycycliques. Technologies de réduction des émissions et besoins de développement", prepared for Environment Canada, Conservation and Protection, Quebec Region, Montreal, Que. (1990).

LEI (Lavalin Environnement Inc.), "PAH Levels in the Ambient Air in Quebec. Fall 1989 - Winter 1990", Report prepared for the Toxic Substances and Air Pollution Control Division, Environment Canada, Conservation and Protection, Environmental Protection, Quebec Region, Montreal, Que., 103 p. (unpublished, 1992a).

LEI (Lavalin Environnement Inc.), "PAH Levels in the Ambient Air in Quebec. Fall, 1990 - Winter, 1991", Report prepared for the Toxic Substances and Air Pollution Control Division, Environnement Canada, Conservation and Protection, Quebec Region, Montreal, Que., 111 p. (unpublished, 1992b).

Lemke, A.E. and R.L. Anderson, "Insect Interlaboratory Toxicity Test Comparison Study for the Chironomid (*Paratanytarsus* sp.) Procedure", U.S. Environmental Protection Agency, Duluth, MN, EPA-600/3-84-054, 15 p. (1984).

LGL (Lalonde, Girouard, Letendre et Associés), "PAH Emissions into the Environment in Canada - 1990", prepared for Environment Canada, Conservation and Protection, Quebec Region, Montreal, Que. (1993).

Ligocki, M.P, C. Leuenberger, and J.F. Pankow, "Trace Organic Compounds in Rain-II. Gas Scavenging of Neutral Organic Compounds", *Atmos. Environ.*, *19*:1609-1617 (1985).

Linder, G. and H.L. Bergman, "Periodic Depuration of Anthracene Metabolites by Rainbow Trout", *Trans. M. Fish. Soc.*, *113*:513-520 (1984).

Lockhart, W.L., personal communication, Department of Fisheries and Oceans, Freshwater Institute, Winnipeg, Man. (1990).

Lu, P.Y., R.L.. Metcalfe, N. Plummer, and D. Mandel, "The Environmental Fate of Three Carcinogens, Benzo(*a*)pyrene, Benzidine and Vinyl Chloride Evaluated in Laboratory Model Ecosystems", *Arch. Environ. Contam. Toxicol.*, *6*:129-142 (1977).

Lunde, G. and A. Bjørseth, "Polycyclic Aromatic Hydrocarbons in Long-range Transported Aerosols", *Nature*, *268*:518-519 (1977).

Lyman, W.J., W.F. Reehl, and D.H. Rosenblatt, *Handbook of Chemical Property Estimation Methods*, McGraw Hill, New York, NY (1982).

Maccubbin, A.E., P. Black, L. Trzeciak, and J.J. Black, "Evidence for Polynuclear Aromatic Hydrocarbons in the Diet of Bottom-feeding Fish", *Bull. Environ. Contam. Toxicol.*, *34*:876-882 (1985).

Maccubbin, A.E., J.J. Black, and B.P. Dunn, "^{32}P-Post-labeling Detection of DNA Adducts in Fish from Chemically Contaminated Waterways", *Sci. Total Environ.*, *94*:89-104 (1990).

Mackay, D. and W.Y. Shiu, "Aqueous Solubility of Polynuclear Aromatic Hydrocarbons", *J. Chem. Eng. Data*, *22*:399-402 (1977).

Mailhot, H., "Prediction of Algal Bioaccumulation and Uptake Rate of Nine Organic Compounds by Ten Physicochemical Properties", *Environ. Sci. Technol.*, 21:1009-1013 (1987).

Malins, D.C., B.B McCain, D.W. Brown, S.L. Chan, M.A. Myers, J.T. Landahl, P.C. Prohaska, A.J. Friedman, L.D. Rhodes, D.G. Burrows, W.D. Gronlund, and H.U. Hodgins, "Chemical Pollutants in Sediments and Diseases of Bottom-dwelling Fish in Puget Sound, Washington", *Environ. Sci. Technol.*, *18*:705-713 (1984).

Malins, D.C., M.M. Krahn, D.W. Brown, L.D. Rhodes, M.S. Myers, B. McCain, and S.L. Chan, "Toxic Chemicals in Marine Sediment and Biota from Mukilteo, Washington: Relationships with Hepatic Neoplasms and Other Hepatic Lesions in English Sole (*Parophrys vetulus*)", *J. Nat. Cancer Inst.*, *74*:487-494 (1985a).

Malins, D.C., M.M. Krahn, M.S. Myers, L.D. Rhodes, D.W. Brown, C.A. Krone, B.B. McCain, and S.L. Chan, "Toxic Chemicals in Sediment and Biota from a Creosote-polluted Harbor: Relationships with Hepatic Neoplasms and Other Hepatic Lesions in English Sole (*Parophrys vetulus)", Carcinogenesis*, *10*:1463-1469 (1985b).

Matheson, R.A.F., G.L. Trider, W.R. Ernst, K.G. Hamilton, and P.A. Hennigar, "Investigation of Polynuclear Aromatic Hydrocarbon Contamination of Sydney Harbour, Nova Scotia", Environment Canada, Atlantic Region, EPS-5-AR-83-6, 86 p. (1983).

McCain, B.B., D.C. Malins, M.M. Krahn, D.W. Brown, W.D. Gronlund, L.K. Moore, and S-L. Chan, "Uptake of Aromatic and Chlorinated Hydrocarbons by Juvenile Chinook Salmon (*Onchorynchus tsawytscha*) in an Urban Estuary", *Arch. Environ. Contam. Toxicol.*, *19*:10-16 (1990).

Merck Index, *An Encyclopedia of Chemicals, Drugs and Biologicals*, 11th Edition, S. Budavari, M.J. O'Neil, A. Smith, and P.E. Heckelman (eds.), Merck and Co., Rahway, NJ (1989).

Metcalfe, C.D., G.C. Balch, V.W. Cairns, J.D. Fitzsimons, and B.P. Dunn, "Carcinogenic and Genotoxic Activity of Extracts from Contaminated Sediments in Western Lake Ontario", *Sci. Total Environ.*, *94*:125-141 (1990).

Miguel, A.H. and S.K. Friedlander, "Distribution of Benzo(*a*)pyrene and Coronene with Respect to Particle Size in Pasadena Aerosols in the Submicron Range", *Atmos. Environ.*, *12*:2407-2413 (1978).

Millemann, R.E., W.J. Binje, J.A. Black, R.M. Curham, K.L. Daniels, P.J. Franco, J.M. Giddings, J.F. McCarthy, and A.J. Stewart, "Comparative Acute Toxicity to Aquatic Organisms of Components of Coal-derived Synthetic Fuels", *Trans. Am. Fish. Soc.*, *113*:74-85 (1984).

MOE (Ontario Ministry of the Environment), "Drinking Water Survey of Selected Municipalities in the Niagara Area and Lake Ontario", Toronto, Ont. (1984).

MOE (Ontario Ministry of the Environment), "Drinking Water Surveillance Program Overview Annual Report 1987", Toronto, Ont. (1989).

Murphy, P.P., T.S. Bates, H.C. Curl, R.A. Feely, and R.S. Burger, "The Transport and Fate of Particulate Hydrocarbons in an Urban Fjord-like Estuary", *Estuar. Coast. Shelf Sci.*, *27*:461-482 (1988).

Murphy, T.P., H. Brouwer, M.E. Fox, E.Nagy, L. McArdle, and A. Moller, "Assessment of the Coal Tar Contamination near Randle Reef, Hamilton Harbour", Lakes Research Branch, National Water Research Institute, Burlington, Ont. (1993).

Myers, M.S., "Overview of Studies on Liver Carcinogenesis in English Sole from Puget Sound: Evidence for a Xenobiotic Chemical Etiology I: Pathology and Epizootiology", *Sci. Total Environ.*, *94*:33-50 (1990).

Nagy, E., J.H. Carey, and E.D. Ongley, "Hydrocarbons in the Mackenzie River", Environment Canada, National Water Research Institute, Burlington, Ont., 7 p. (1987).

Nagy, E., J.H. Carey, and E.D. Ongley, "Under-ice Hydrocarbons in the Mackenzie River", Environment Canada, National Water Research Institute, Burlington, Ont. (1989).

Neal, J. and R. H. Rigdon, "Gastric Tumours in Mice Fed Benzo(*a*)pyrene: A Quantitative Study", *Texas Reports on Biology and Medicine, 25*:553-557 (1967).

Neff, J.M., "Polycyclic Aromatic Hydrocarbons in the Aquatic Environment. Sources, Fates and Biological Effects", Applied Science Publishers Ltd., Essex, England, 262 p. (1979).

Newsted, J.L. and J.P. Giesy, "Predictive Models for Photo-induced Acute Toxicity of Polycyclic Aromatic Hydrocarbons to *Daphnia magna*", *Environ. Toxicol. Chem., 6*:445-461 (1987).

NRC (National Research Council), "Polycyclic Aromatic Hydrocarbons: Evaluation and Effects", Committee on Pyrene and Selected Analogues, Board on Toxicology and Environmental Health Hazards, Commission on Life Sciences, National Academy Press, Washington, DC (1983).

NRCC (National Research Council of Canada), "Polycyclic Aromatic Hydrocarbons in the Aquatic Environment: Formation, Sources, Fate and Effects on Aquatic Biota", NRC Associate Committee on Scientific Criteria for Environmental Quality, Publication No. NRCC 18981, Ottawa, Ont., 209 p. (1983).

O'Neill, H.J. and K.M. Kieley, "Polynuclear Aromatic Hydrocarbons: Atlantic Region Data Summary and Review", Environment Canada, Conservation and Protection, Moncton, N.B., 64 p. (1992).

Oris, J.T. and J.P. Giesy, "The Photo-induced Toxicity of Polycyclic Aromatic Hydrocarbons to Larvae of the Fathead Minnow *Pimephales promelas*", *Chemosphere, 16*:1395-1404 (1987).

Oris, J.T., A. Tilghman Hall, and J. D. Tylka, "Humic Acids Reduce the Photo-induced Toxicity of Anthracene to Fish and Daphnia", *Environ. Toxicol. Chem., 9*:575-583 (1990).

Ortech International, "MOE Toxic Chemical Emission Inventory for Ontario Eastern North America", prepared for the Ontario Ministry of the Environment, draft report no. P89-5-5429/06 (1990).

PACE (Petroleum Association for the Conservation of the Canadian Environment), "Fate of Polynuclear Aromatic Hydrocarbons in Refinery Waste Applied to Soil", prepared for PACE by Environment Canada, Conservation and Protection, Wastewater Technology Centre, Burlington, Ont., 156 p. (1988).

Payne, J.F., J. Kiceniuk, L.L. Fancey, U. Williams, G.L. Fletcher, A. Rahimtula, and B. Fowler, "What Is a Safe Level of Polycyclic Aromatic Hydrocarbons for Fish: Subchronic Toxicity Study on Winter Flounder (*Pseudopleuronectes americanus)*", *Can. J. Fish. Aquat. Sci., 45*:1983-1993 (1988).

Pierce, R.C. and M. Katz, "Dependency of Polynuclear Aromatic Hydrocarbon Content on Size Distribution of Atmospheric Aerosols", *Environ. Sci. Technol., 9*:347-353 (1975).

Pruell, R.J., J.L. Lake, W.R. Davies, and J.G. Quinn, "Uptake and Depuration of Organic Contaminants by Blue Mussels *Mytilus edulis* Exposed to Environmentally Contaminated Sediments", *Mar. Biol.*, *91*:497-508 (1986).

Pupp, C., R. Lao, J. Murray, and R. Pottie, "Equilibrium Vapor Concentrations of Some Polycyclic Aromatic Hydrocarbons, As_4O_6 and SeO_2, and the Collection Efficiencies of these Air Pollutants", *Atmos. Environ.*, *8*:915-925 (1974).

Radding, S.B., T. Mill, C.W. Gould, D.H. Liu, H.L. Johnson, D.C. Bomberger, and C.V. Fojs, "The Environmental Fate of Selected Polynuclear Aromatic Hydrocarbons", U.S. Environmental Protection Agency, Office of Toxic Substances, Washington, DC, EPA 560/5-75-009, 122 p. (1976).

Ramanathan, V., R.J. Cicerone, H.B. Singh, and J.T. Kiehl, "Trace Gases and their Potential Role in Climate Change", *J. Geophys. Res.*, 90:(D3), 5547-5566 (1985).

RDRC (Resources Development Research Centre), "National Overview of Abandoned Coal Gasification Works in Canada", Carleton University Resources Development Research Centre Report, for Environment Canada, Conservation and Protection, Ottawa, Ont., 51 p. (1987).

Ringuette, S., A. Germain, C. Gonthier, and F. Perron, "Presence of PAHs in the Canadian Environment - An Overview", Conservation and Protection, Quebec Region, Montreal, Que., 36 p. + appendices (1993).

Sadtler Research Laboratories, "Infrared Spectra of Priority Pollutants and Toxic Chemicals", Sadtler Research Laboratories, Philadelphia, PA (1982).

Santodonato, J., P. Howard, D. Basu, S. Lande, and J.K. Selkirk, "Health Assessment Document for Polycyclic Organic Matters", Washington, DC, U.S. EPA, Report EPA-600/9-79-008 (1979). (As cited in Smith, 1984).

Santodonato, J., P. Howard, and D. Basu, "Health and Ecological Assessment of Polynuclear Aromatic Hydrocarbons", *J. Environ. Pathol. Toxicol.*, *5*:1-368 (1981).

Schiewe, M.H., D.W. Weber, M.S. Myers, F.J. Jacques, W.L. Reichert, C.A. Krone, D.C. Malins, B.B. McCain, S.L. Chan, and U. Varanasi, "Induction of Foci of Cellullar Alteration and Other Hepatic Lesions in English Sole (*Parophrys vetulus*) Exposed to an Extract of an Urban Marine Sediment", *Can. J. Fish. Aquat. Sci.*, *48*:1750-1759 (1991).

Shugart, L.R., "Quantitation of Chemically Induced Damage to DNA of Aquatic Organisms by Alcaline Unwinding Assay", *Aquat. Toxicol.*, *13*:43-52 (1988).

Shugart, L. R., "Detection and Quantitation of Benzo(*a*)pyrene-DNA Adducts in Brain and Liver Tissues of Beluga Whales (*Delphinapterus leucas*) from the St. Lawrence and Mackenzie Estuaries", in: *For the Future of the Beluga: Proceedings of the*

International Forum for the Future of the Beluga, J. Prescott and M. Gauquelin (eds.), Presses de l'Université du Québec, Quebec, pp. 219-223 (1990).

Sims, R.C. and M.R. Overcash, "Fate of Polycyclic Aromatic Compounds in Soil-plant Systems", *Residue Rev.*, 88:1-68 (1983).

Sirota, G.R., J.F. Uthe, A. Streedharan, R. Matheson, G.J. Musial, and K. Hamilton, "Polynuclear Aromatic Hydrocarbons in Lobster (*Homarus americanus*) and in the Vicinity of a Coking Facility", in: *Polynuclear Aromatic Hydrocarbons Formation, Metabolism and Measurement*, M. Cooke and A.J. Dennis (eds.), Batelle Press, OH, pp. 1123-1136 (1983).

Slaga, T.J., G.L. Gleason, G. Mills, L. Ewald, P.P Fu, H.M. Lee, and R.G. Harvey, "Comparison of the Skin Tumour-initiating Activities of Dihydrodiols and Diol-epoxides of Various Polycyclic Aromatic Hydrocarbons", *Cancer Res.*, *40*:1981-1984 (1980).

Slooff, W., J.A. Janus, A.J.C.M. Matthijsen, G.K. Montizaan, and J.P.M. Ros (eds.), "Integrated Criteria Document PAHs", National Institute of Public Health and Environmental Protection (RIVM), Bilthoven, Netherlands, Report No. 758474011, 199 p. (1989).

Smith, I.M., "PAH from Coal Utilisation - Emissions and Effects", IEA Coal Research, London, England, ICTIS/TR29 (1984).

Smith, J.H., W.R. Mabey, N. Bohonos, B.R. Holt, S.S. Lee, T.W. Chov, D.C. Bamberger, and T. Mill, "Environmental Pathways of Selected Chemicals in Freshwater Systems, Part II: Laboratory Studies", U.S. Environmental Protection Agency, Washington, DC, EPA 600/7-78-074, 405 p. (1978).

Solbakken, J.E., S. Tilseth, and K.H. Palmork, "Uptake and Elimination of Aromatic Hydrocarbons and Chlorinated Biphenyl in Eggs and Larvae of Cod *Gadus morhua*", *Mar. Ecol. Prog. Ser.*, *16*:297-301 (1984).

Southworth, G.R., "The Role of Volatilization in Removing Polycylic Aromatic Hydrocarbons from Aquatic Environments", *Bull. Environ. Contam. Toxicol.*, *21*:507-514 (1979).

Southworth, G.R., J.J. Beauchamp, and P.K. Schmieden, "Bioaccumulation Potential of Polycyclic Aromatic Hydrocarbons in *Daphnia pulex*", *Water Res.*, *12*:973-977 (1978).

Spacie, A., P.F. Landrum, and G.J. Leversee, "Uptake, Depuration and Biotransformation of Anthracene and Benzo(*a*)pyrene in the Gill Fish", *Ecotox. Environ. Safety*, *7*:330-341 (1983).

SRI (Stanford Research Institute), "Directory of Chemical Producers", Menlo Park, CA (1980).

SRI (Standford Research Institute), "International Chemical Economics Handbook" (1985). (As cited in Slooff *et al.* 1989.)

Statistics Canada, Catalogue 45-004 Monthly, Volume 44, Number 11, November (1989).

Stein, J.E., W.L. Reichert, M. Nishimoto, and U. Varanasi, "Overview of Studies on Liver Carcinogenesis in English Sole from Puget Sound; Evidence for a Xenobiotic Chemical Etiology II: Biochemical Studies", *Sci. Total Environ.*, *94*:51-69 (1990).

Suess, M.J., "The Environmental Load of Polycyclic Aromatic Hydrocarbons" (1976). (As cited in Bjørseth *et al.*, 1979.)

Swain, L.G. and D.G. Walton, "Fraser River Monitoring - Report on the 1988 Fish Monitoring Program", Ministry of the Environment, British Colombia, 147 p. (1989).

Tardif, M. and C. Chiu, "Method Development on the Analysis of PAH Isomers", Chemistry Division, Technology Development Branch, Environmental Technology Centre, Environment Canada, Reference no. CD920303 (unpublished, 1992).

Tecsult, "Garde côtière canadienne - base de Sorel : étude de caractérisation complémentaire", prepared for Transport Canada (1989).

Thorslund, T. and G. Charnley, "Comparative Potency Approach for Estimating the Cancer Risk Associated with Exposure to Mixtures of Polycyclic Aromatic Hydrocarbons", ICF-Clement Associates, Washington, DC, Interim Final Report prepared for U.S. EPA Office of Health and Environmental Assessment, Contract No. 68-02-4403 (unpublished, 1988).

Thyssen, J., J. Althoff, G. Kimmerle, and U. Mohr, "Inhalation Studies with Benzo[*a*]pyrene in Syrian Golden Hamsters", *J. Natl. Cancer Inst.*, *66*:575-577 (1981).

Trucco, R.G., F.R. Englehardt, and B. Tracey, "Toxicity, Accumulation and Clearance of Aromatic Hydrocarbons in *Daphnia pulex", Environ. Poll., ser. A, 31*:191-202 (1983).

U.S. EPA (United States Environmental Protection Agency), "An Exposure and Risk Assessment for Benzo(*a*)pyrene and Other Polycyclic Aromatic Hydrocarbons", Office of Water Regulations and Standards, Washington, DC, EPA/440/4-85-020 (1982).

U.S. EPA (United States Environmental Protection Agency), 1987: Locating and Estimating Air Emissions from Sources of Polycyclic Organic Matter (POM)". EPA-450/4-84-007. (September, 1987).

U.S. EPA (United States Environmental Protection Agency), "Chemical Fate Rate Constants for SARA Section 113 Chemicals and Superfund Health Evaluation Manual Chemicals", Office of Toxic Substances, Washington, DC, 6-02-4254 (1990).

Uthe J.F. and C.J. Musial, "Polycyclic Aromatic Hydrocarbon Contamination of American Lobster, *Homarus americanus*, in the Proximity of a Coal-cooking Plant", *Bull. Environ. Contam. Toxicol., 37*:730-738 (1986).

van Coillie, R., N. Bermingham, C. Blaise, R. Vezeau, and J. Lakshuminaraganan, "Integrated Ecotoxicological Evaluation of Effluents from Dumpsites", in: *Advances of Environmental Sciences and Technology*, J.M. Wiley, New York, NY, Vol. 22, pp. 161-191 (1990).

van Noort, P.C.M and E. Wondergem, "Scavenging of Airborne Polycyclic Aromatic Hydrocarbons by Rain", *Environ. Sci. Technol., 19*:1044-1048 (1985).

van Vaeck, L. and K. van Cauwenbeerghe, "Cascade Impactor of the Size Distribution of the Major Classes of Organic Pollutants in Atmospheric Particulate Matter", *Atmos. Environ., 12*:2229-2239 (1978).

Vandermeulen, J.H., "PAH and Heavy Metal Pollution of the Sydney Estuary: Summary and Review of Studies to 1987", *Canadian Technical Report of Hydrography and Ocean Sciences*, No. 108 (1989).

Varanasi, U., J.E. Stein, and M. Nishimoto, "Biotransformation and Disposition of PAH in Fish", in: *Metabolism of Polycyclic Aromatic Hydrocarbons in the Aquatic Environment*, U. Varanasi (ed.), CRC Press, Boca Raton, FL, Chapter 4, pp. 93-151 (1989).

Wakeham, S.G., C. Schaffer, and W. Giger, "Polycyclic Aromatic Hydrocarbons in Recent Lake Sediments. I. Compounds Having Anthropogenic Origins", *Geochim. Cosmochim. Acta., 44*:403-413 (1980).

Wan, M.T., "Railway Right-of-way Contaminants in the Lower Mainland of British Columbia: Polycyclic Aromatic Hydrocarbons", *J. Environ. Qual., 20*:228-234 (1991).

Wan, M.T., "Utility Right-of-way Contaminants: Polycyclic Aromatic Hydrocarbons", Environment Canada, Conservation and Protection, Pacific and Yukon Region, North Vancouver, B.C., (unpublished, 1993).

Wang, T.H., C. Curran, P. Bedient, and M. Tomson, "Ground Water Contamination at Conroe Creosote Waste Disposal Site", in *Proceedings, Second International Conference on Ground Water Quality Research*, Tulsa, OK (1983). (As cited in Bedient *et al.*, 1984.)

Warshawsky, D., T. Cody, M. Radike, B.A. Smiddy, and B. Nagel, "Toxicity and Metabolism of Benzo(*a*)pyrene in the Green Algae *Selenastrum capricornutum*", in: *Polynuclear Aromatic Hydrocarbons: Formation, Metabolism and Effects*, M. Cooke and A.J. Dennis (eds.), Batelle Press, Columbus, OH, pp. 1235-1245 (1983).

Wendland, T., "1978-79 Sydney Harbour Survey; Benthic Fauna Distribution and Related Studies", Environmental Technology Project, College of Cape Breton, Sydney, N.S. (1979). (As cited in Hildebrand, 1982.)

Westerholm, R.N., T.E. Alsberg, A.B. Frommelin, M.E. Strandell, V. Ranney, L. Winquist, V. Grigoriadis, and K.E. Egebäck, "Effect of Fuel Polycyclic Aromatic Hydrocarbon Content on the Emissions of Polycyclic Aromatic Hydrocarbons and Other Mutagenic Substances from a Gasoline-fueled Automobile", *Environ. Sci. Technol.*, *22*:925-930 (1988).

Wild, S.R. and K.C. Jones, "Biological and Abiotic Losses of Polynuclear Aromatic Hydrocarbons (PAHs) from Soils Freshly Amended with Sewage Sludge", *Environ. Toxicol. Chem.*, 12:5-12 (1993).

Wild, S.R., M.L. Berrow, and K.C. Jones, "The Persistence of Polynuclear Aromatic Hydrocarbons (PAH) in Sewage Sludge Amended Agricultural Soils", *Environ. Pollut.*, *72*:141-157 (1991).

Windsor, J.G. and R.A. Hites, "Polycyclic Aromatic Hydrocarbons in the Gulf of Maine Sediments and Nova Scotia Soils", *Geochem. Cosmochim. Acta.*, *43*:27-33 (1979).

Wislocki, P.G. and A.Y.H. Lu, "Carcinogenicity and Mutagenicity of Proximate and Ultimate Carcinogens of Polycyclic Aromatic Hydrocarbons", in: *Polycyclic Aromatic Hydrocarbon Carcinogenesis: Structure-Activity Relationships*, S.K. Yang and B.D. Silverman (eds.), Vol. I, CRC Press, Boca Raton, FL, pp. 1-30 (1988).

WMS Associates Ltd., Intera Technologies, and National Water Research Institute, "Investigation of Suspected PAH Contamination of Groundwater, Newcastle, New Brunswick", Fredericton, N.B., WMS 2988, 29 p. (1989).

Wong, L. and H. Bailey, Environment Canada, Conservation and Protection, Moncton, N.B. (unpublished, 1990).