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Canadian Soil Quality Guidelines

CARCINOGENIC AND OTHER POLYCYCLIC AROMATIC HYDROCARBONS (PAHs)

(Environmental and Human Health Effects)

Scientific Criteria Document (revised)

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NOTE TO READERS

This scientific criteria document provides the background information and rationale for the development of Canadian Soil Quality Guidelines for the protection of environmental and human health for potentially carcinogenic and other PAHs. They were developed under contract by UMA Engineering Ltd., with further revisions by Health Canada and Environment Canada. For additional technical information regarding these guidelines, please contact:

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CCME (Canadian Council of Ministers of the Environment), 2010. Canadian Soil Quality Guidelines for Carcinogenic and Other Polycyclic Aromatic Hydrocarbons (Environmental and Human Health Effects). Scientific Criteria Document (revised). 216 pp.

GLOSSARY OF TERMS

ADI	Allowable Daily Intake.
ATSDR	Agency for Toxic Substances and Disease Registry.
Alkyl-substituted PAHs (and alkyl-PAH)	Those polycyclic aromatic hydrocarbons having at least one alkyl sidechain (methyl, ethyl or other alkyl group) attached to the aromatic ring structure.
Alternant PAHs	Those PAHs with a core ring structure composed entirely of benzenoid rings.
Apoptosis	Programmed cell senescence (aging) and death.
B[a]P	Benzo[a]pyrene.
B[a]P PEF	Benzo[a]pyrene potency equivalence factor.
B[a]P TPE	Benzo[a]pyrene total potency equivalents.
CAEAL	Canadian Association of Environmental Analytical Laboratories.
CCME	Canadian Council of Ministers of the Environment.
CEPA	Canadian Environmental Protection Act.
CEQGs	Canadian Environmental Quality Guidelines.
CYP	Cytochrome P450 oxidase(s).
CWS	Canada-Wide Standards.
DTED	Daily threshold effects dose.
FID	Fluorescence induced detection.
Genotoxic	Tendency to cause mutations.
HPAH	Higher Molecular Weight PAHs.
HPLC	High Pressure Liquid Chromatography.
IARC	International Agency for Research on Cancer.
ILCR	Incremental Lifetime Cancer Risk.
IPCS	International Program on Chemical Safety.
IRIS	Integrated Risk Information System.
Initiation	Those stages of carcinogenesis between internalization of a carcinogen and interaction with the cell's DNA leading to a potentially non-lethal mutation.
LOAEL	Lowest observed adverse effects level.
LPAH	Lower molecular weight PAHs.
K_{OW}	Octanol-water partition co-efficient.
K_{OC}	Organic carbon-water partition co-efficient.
Kinetically favoured PAHs	PAHs preferentially produced during combustion.
Lentic	Freshwater basin (e.g. lake) ecosystem.
Lotic	Running water ecosystem.
$mg \cdot kg^{-1}$	milligrams/kilogram soil (parts per million).
MPC	Maximum Permissible Concentration (Netherlands).
MRL	Minimum Risk Level (ATSDR toxicological threshold value).
NATO I-TEF	North Atlantic Treaty Organization International Toxicity Equivalence Factors.
NCI	National Cancer Institute.
NCSRP	National Contaminated Sites Remediation Program.
NEC	Nutrient and Energy Cycling check value.
NIOSH	National Institute of Occupational Health and Safety (USA).

NOAEL	No observed adverse effects level.
Non-alternant PAHs	PAHs containing four, five, and six-member, non-aromatic ring structures.
PAHs	Polycyclic aromatic hydrocarbons.
Petrogenic	Petroleum-derived.
Pyrogenic	Combustion-derived.
PBPK	Physiologically based pharmacokinetic (a type of contaminant fate model applicable to the uptake, transformation and elimination of contaminants in animal models).
PCBs	Polychlorinated biphenyls.
PHCs	Petroleum hydrocarbons.
PM	Particulate matter. Airborne particles with a diameter of < 10 μm (PM_{10}) are generally considered to be readily respirable by humans.
PSL1	Priority Substances List 1 – Canadian Environmental Protection Act (CEPA).
$PSQG_{DH\text{-}PICA}$	Preliminary soil quality guideline for human health protection based on pica soil ingestion.
Progression	A progressive loss of feedback control that is important for normal cell division, and a progression towards malignancy and spread.
Promotion	Secondary cell transformations that allow the cell and its future progeny to escape normal physiological controls.
RfC	Reference Concentration.
RfD	Reference Dose.
RsD	Risk specific Dose.
SBT_{FL}	Soil-Based Thresholds for Freshwater Life protection, assuming a non-polar narcosis (critical body residue: CBR) mode of toxicity: Back-calculated from a CBR-type Water-Based Threshold (SBT_{CBR}).
SQG_{DH}	Human health-based Soil Quality Guideline for protection of direct contact to soil (i.e., ingestion, inhalation, dermal contact).
SQG_{E}	Soil Quality Guideline for environmental protection.
SQG_{FI}	Soil Quality Guideline for human health protection based on food ingestion (produce, meat, and milk).
SQG_{FL}	Soil Quality Guideline for protection of freshwater life.
SQG_{HH}	Soil Quality Guideline for human health protection.
SQG_{I}	Soil Quality Guideline for protection of livestock and wildlife based on soil and food ingestion. (The terms SQG_{1C} and SQG_{2C} are also used for primary consumers and secondary consumers, respectively.)
SQG_{IAQ}	Soil Quality Guideline for the protection of indoor air quality.
SQG_{IR}	Soil Quality Guideline for the protection of irrigation water.
SQG_{LW}	Soil Quality Guideline for protection of livestock based on water consumption.
SQG_{NEC}	Soil Quality Guideline check value for the protection of nutrient and energy cycling.
$SQG_{OM\text{-}E}$	Soil Quality Guideline check value for off-site migration of soils in consideration of environmental health risks.
$SQG_{OM\text{-}HH}$	Soil Quality Guideline check value for off-site migration of soils in consideration of human health risks.

SQG_{PW}	Soil Quality Guideline for the protection of potable water.
SQG_{SC}	Soil Quality Guideline for soil contact by soil-dependent organisms (e.g., plants and invertebrates).
SQGTG	Soil Quality Guidelines Task Group.
TDI	Tolerable Daily Intake.
TEQ	Toxicity Equivalence Quotient.
TPHCWG	Total Petroleum Hydrocarbon Criterion Working Group (USA).
TRV	Toxicity reference value.
Thermodynamically favoured PAHs	More energetically stable PAHs, especially produced during the long-term diagenesis of organic matter.
USEPA	United States Environmental Protection Agency.
UST	Underground storage tank.
Unsubstituted PAHs	Polycyclic aromatic hydrocarbons made up entirely from carbon and hydrogen atoms, and having no alkyl groups around the aromatic ring structure.
WBT_{CBR}	Water-Based Thresholds based on a critical body residues approach and assumed water-organism equilibrium partitioning of lipophilic contaminants.
WHO	World Health Organization.

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ABSTRACT

This scientific criteria document provides the background information and rationale for the derivation of human health and environmental soil quality guidelines for potentially carcinogenic and other polycyclic aromatic hydrocarbons (PAHs). Canadian soil quality guidelines were developed in the late 1990s for naphthalene and benzo[a]pyrene; however, partially harmonized assessment and risk management approaches were not previously available for any of the other PAHs typically found as complex mixtures at many contaminated sites.

This document contains a review of recent scientific information on the chemical and physical properties of potentially carcinogenic and other commonly analyzed unsubstituted PAHs, a brief review of sources and emissions in Canada, the expected environmental fate, and the toxicological significance of these PAHs to soil microbial processes, plants, animals and humans. This information is further used to derive soil quality guidelines for PAHs to protect human health and ecological receptors for four major land use scenarios: agricultural, residential/parkland, commercial and industrial. These guidelines were developed according to procedures described in *A Protocol for the Derivation of Environmental and Human Health Soil Quality Guidelines* (CCME, 2006).

According to the 2006 CCME soils protocol, both environmental and human health soil quality guidelines are developed and the lowest values of the two generated, for each of the four land use types, is recommended by CCME as the Canadian Soil Quality Guidelines.

Preliminary human health soil quality guidelines (SQG_{HH}) that have been derived for the carcinogenic PAHs include compound-specific limits for each of the four land uses, based on cancer-type endpoints. The guidelines are also based on assumptions regarding the relative cancer potencies of the individual PAHs. A critical review of the PAH relative potency schemes shows that there have been cases where the experimentally determined cancer potency of a mixture was under-estimated based on assumed PAH cancer potencies relative to benzo[a]pyrene. The accuracy of predictions based on relative potencies is influenced by the type of PAH-containing mixture to which a mammal is exposed as well as the route of exposure (oral, dermal, inhalation, other). Cancer risks from PAH contamination at coal tar and creosote release sites, for example, may be under-estimated using the best available relative cancer potency schemes.

Human health soil quality guidelines (SQG_{HH}) for non-carcinogenic PAHs have not been presented in this document, as well as for additional carcinogenic PAHs not presented herein. It is recommended that Health Canada be contacted directly for guidance on these substances (http://www.hc-sc.gc.ca/ewh-semt/contamsite/index_e.html).

The preliminary environmental soil quality guidelines (SQG_{E}) for PAHs include consideration of assumed threshold-acting (non-carcinogenic) effects on soil ecological functioning, and specifically on soil invertebrate and plant responses. For soil invertebrates, the major mode of toxic action might be non-polar narcosis, although other modes of toxicological action (photoinduced toxicity, for example) might be important in some cases, and may be inherent in some of the underlying toxicological data used to develop the preliminary direct soil contact

guidelines. The existing scientific knowledge on PAH environmental risks based on soil nutrient and energy cycling, soil invertebrates, and plants is very limited in spite of some recent advances. The ability to manage risks to ecological receptors of PAH contamination in soils will probably continue to evolve at a rapid pace in the earliest part of the 21st century.

There is still considerable uncertainty regarding the role of soil and site properties in modifying PAH bioavailability and toxicological responses based on direct soil contact pathways.

The minimum data requirements were met for calculating direct contact environmental soil quality guideline (SQG_{SC}) values for three PAHs: anthracene (using the LOEC approach), and fluoranthrene and benzo[a]pyrene (based on the preferred weight-of-evidence approach) (CCME, 2006). However, there is still considerable uncertainty around these estimates, and insufficient data to calculate soil quality guidelines for this exposure scenario for the larger suite of unsubstituted and alkyl-substituted PAHs.

Given that PAHs might influence soils and plants through baseline toxicity (e.g., through internalization into lipid pools and induction of a wide range of non-specific effects such as reduced active transport of ions across membranes), the effects are likely to overlap and be additive with other non-polar organics in the contaminant mixture, including other aromatic and aliphatic petroleum hydrocarbon constituents. It is anticipated, therefore, that some protection is afforded to soil ecological functioning based on direct soil contact through application of the Canada-Wide Standards for Petroleum Hydrocarbons.

PAH environmental soil quality guidelines were also calculated in consideration of freshwater life protection (SQG_{FL}) for sites where there is potential for groundwater mediated transfer of PAHs to an adjacent surface water body capable of supporting aquatic life, using existing (CCME, 1999a) water quality guidelines for aquatic life protection for naphthalene, acenaphthene, fluorene, phenanthrene, anthracene, fluoranthene, pyrene, benz[a]anthracene, and benzo[a]pyrene. Additional soil-based threshold values for the protection of freshwater life were investigated for the remaining unsubstituted PAHs using a critical body residues/non-polar narcosis approach to develop acceptable threshold values in the aquatic receiving environment. The values for the protection of aquatic life were derived in part using a groundwater model adapted from Domenico and Robbins (Domenico, 1987), as described in CCME (2006).

For agricultural and residential/parkland land uses, preliminary SQG_I were developed in consideration of soil and food ingestion by cows as a representative livestock species, and mule deer, meadow voles and American robins as representative wildlife species.

For protection of human health, for all land uses, direct contact guidelines (SQG_{DH}) based on combined ingestion, inhalation and dermal exposures have been calculated as 0.6 $mg \cdot kg^{-1}$ benzo[a]pyrene "Total Potency Equivalents" (B[a]P TPEs) and 5.3 $mg \cdot kg^{-1}$ B[a]P TPEs, based on incremental lifetime cancer risks (ILCRs) of 10^{-6} and 10^{-5}, respectively. B[a]P TPEs are calculated using PAH cancer Potency Equivalence Factors (PEFs) adapted from the World Health Organization (WHO/IPCS 1998). B[a]P TPEs include the concentration of B[a]P itself.

For sites contaminated with coal tar residues or creosote mixtures, the available studies indicate that B[a]P relative potency schemes, coupled with use of the B[a]P cancer slope factor, may not adequately predict cancer risks of the mixture. Therefore, a three-fold safety factor should be employed when calculating B[a]P TPEs for sites affected by creosote or coal tar before comparison with the SQG_{DH}. In other words, for these complex mixtures, total B[a]P equivalent concentrations (derived by applying the TPE values) should be multiplied by three (3) prior to risk characterization by comparison to the PAH guideline values.

Also for the protection of human health, soil quality guidelines for the protection of potable water (SQG_{PW}) component values were derived for individual carcinogenic PAHs. It should be noted that the individual SQG_{PW} component values are not stand alone soil quality guidelines. Rather, each has been incorporated into the "Index of Additive Cancer Risks" (IACR) equation, to account for the combined effects of individual PAHs in the mixture. The potable water component values (which are the same for all land uses) are as follows:

benz[a]anthracene:	0.33 $mg \cdot kg^{-1}$;
benzo[b+j+k]fluoranthene:	0.16 $mg \cdot kg^{-1}$;
benzo[g,h,i]perylene:	6.8 $mg \cdot kg^{-1}$;
benzo[a]pyrene:	0.37 $mg \cdot kg^{-1}$
chrysene:	2.1 $mg \cdot kg^{-1}$;
dibenz[a,h]anthracene:	0.23 $mg \cdot kg^{-1}$;
indeno[1,2,3-c,d]pyrene:	2.7 $mg \cdot kg^{-1}$.

For protection of the non-human environment, soil quality guidelines for agricultural and residential/parkland sites are recommended as follows:

anthracene:	2.5 $mg \cdot kg^{-1}$;
benzo[a]pyrene:	20 $mg \cdot kg^{-1}$
fluoranthene:	50 $mg \cdot kg^{-1}$.

For protection of the non-human environment, soil quality guidelines for commercial and industrial sites are recommended as follows:

anthracene:	32 mg·kg-1;
benzo[a]pyrene:	72 mg·kg-1;
fluoranthene:	180 mg·kg-1;

Although guideline values for certain pathways were determined for additional PAHs, an overall SQG_E could not be recommended for many of these due to the lack of data for derivation of soil contact guidelines.

Typically, Canadian Soil Quality Guidelines for the protection of environmental and human health are recommended based on the lowest guidelines among all of the various pathway-specific and receptor-specific environmental and human health SQG values. However, in the case of PAHs, because the guidelines for some pathways are based on concentrations of

individual PAHs, while guidelines for other pathways are based on additive toxicity of a mixture of PAHs using B[a]P TPEs or IACR, it is not possible to directly compare all guidelines to select the lowest value. Therefore, in applying the guidelines, it is recommended that users compare PAH concentrations measured in soil samples to the guidelines for all applicable exposure pathways.

RÉSUMÉ

Le présent document scientifique fournit des renseignements de base et expose les raisons qui justifient l'élaboration de recommandations pour la qualité des sols en vue de la protection de l'environnement et de la santé humaine contre les hydrocarbures aromatiques polycycliques (HAP) potentiellement cancérogènes et non cancérogènes. Des recommandations ont été élaborées pour le naphtalène et le benzo[*a*]pyrène à la fin des années 1990; toutefois, on ne disposait auparavant d'aucune méthode partiellement harmonisée d'évaluation et de gestion des risques pour aucun des autres HAP généralement présents sous forme de mélange complexe dans de nombreux lieux contaminés.

Le document comprend un examen des renseignements scientifiques récents sur les propriétés physiques et chimiques d'HAP potentiellement cancérogènes et d'autres HAP non substitués couramment analysés, un bref exposé des sources et des émissions de ces HAP au Canada, de leur devenir dans l'environnement et de leur toxicité pour les processus microbiens dans le sol ainsi que pour les plantes, les animaux et les humains. Ces renseignements sont également utiles pour élaborer des recommandations pour la qualité des sols applicables aux HAP en vue de protéger la santé humaine et les récepteurs écologiques sur des terrains à quatre grandes vocations : agricole, résidentielle/parc, commerciale et industrielle. Ces recommandations ont été élaborées conformément aux procédures décrites dans le *Protocole d'élaboration de recommandations pour la qualité des sols en fonction de l'environnement et de la santé humaine* (CCME, 2006).

Conformément au protocole de 2006 du CCME, des recommandations distinctes sont élaborées aux fins de la protection de l'environnement et de la santé humaine, et les valeurs les plus faibles, entre les deux ensembles de valeurs obtenus pour chacun des quatre types de terrain, deviennent la recommandation canadienne pour la qualité des sols.

Les recommandations préliminaires pour la qualité des sols en vue de la protection de la santé humaine (RQS_{SH}) qui ont été élaborées pour les HAP cancérogènes comprennent des limites propres aux composés pour chacun des quatre types de terrains, limites fondées sur les effets cancérogènes. Les recommandations s'appuient également sur des hypothèses concernant le potentiel cancérogène relatif de chacun des HAP. Un examen critique des profils de potentiel cancérogène relatif des divers HAP montre que le potentiel cancérogène d'un mélange, tel que déterminé en laboratoire, a dans certains cas été sous-estimé lorsque le calcul avait été fait d'après le potentiel cancérogène présumé des HAP par rapport à celui du benzo[*a*]pyrène. L'exactitude des prédictions basées sur le potentiel cancérogène relatif dépend du type de mélange contenant des HAP auquel un mammifère est exposé de même que de la voie d'exposition (orale, cutanée, inhalation, autre). Les risques de cancer associés à la présence

d'HAP sur des sites contaminés par du goudron de houille et de la créosote, par exemple, peuvent être sous-estimés si on utilise les meilleurs profils de potentiel cancérogène relatif.

Le présent document ne contient pas de recommandations pour la qualité des sols en vue de la protection de la santé humaine (RQS_{SH}) applicables aux HAP non cancérogènes, ainsi que pour d'autres HAP cancérogènes non considérés ici. Veuillez communiquer directement avec Santé Canada pour obtenir des directives sur ces substances (http://www.hc-sc.gc.ca/ewh-semt/contamsite/index_f.html).

Les recommandations préliminaires pour la qualité des sols en vue de la protection de l'environnement (RQS_{E}) prennent en considération les effets-seuils (non cancérogènes) présumés sur les fonctions écologiques des sols et, plus précisément, sur les réponses des plantes et des invertébrés du sol. La narcose non polaire pourrait constituer le principal mode d'action toxique chez les invertébrés du sol, mais d'autres modes (toxicité photoinduite, par exemple) pourraient s'avérer importants dans certains cas et être inhérents à certaines données toxicologiques utilisées pour élaborer les recommandations préliminaires visant le contact direct avec le sol. Les connaissances scientifiques actuelles sur les risques environnementaux des HAP, basées sur le cycle des nutriments et de l'énergie, les invertébrés du sol et les végétaux, sont très limitées malgré des avancées récentes. La capacité de gérer les risques que pose la contamination des sols aux HAP pour les récepteurs écologiques continuera probablement à augmenter rapidement en de début de 21^{e} siècle.

Il y a encore beaucoup d'incertitude quant à l'incidence des propriétés du sol et des sites sur la biodisponibilité des HAP et les réponses toxicologiques associées aux mécanismes de contact direct avec le sol.

On possède suffisamment de données pour élaborer des recommandations pour la qualité des sols en vue de protéger l'environnement contre l'exposition à trois HAP par contact direct avec le sol (RQS_{CS}) : l'anthracène (méthode de la CMEO) et le fluoranthène et le benzo[*a*]pyrène (méthode du poids de la preuve) (CCME, 2006). Toutefois, il existe beaucoup d'incertitude au sujet de ces estimations, et il manque des données pour élaborer des recommandations quant à l'exposition, par cette voie, au grand groupe des HAP non substitués et des HAP alkyl-substitués.

Vu la toxicité possible des HAP pour les sols et les végétaux (internalisation dans les réservoirs lipidiques et induction de multiples effets non spécifiques, tels que la réduction du transport actif des ions à travers les membranes), les effets sont susceptibles de se superposer en partie et de s'ajouter à ceux des autres composés organiques non polaires dans le mélange de contaminants, y compris d'autres constituants des hydrocarbures pétroliers aromatiques et aliphatiques. On présume donc que l'application du Standard pancanadien relatif aux hydrocarbures pétroliers procure une certaine protection aux fonctions écologiques des sols contre les effets découlant du contact direct avec le sol.

Des recommandations pour la qualité des sols applicables aux HAP ont également été élaborées pour protéger la vie aquatique (RQS_{VA}) sur les sites où des HAP peuvent être transférés par les eaux souterraines dans un plan d'eau de surface adjacent capable d'entretenir des organismes aquatiques. Elles sont fondées sur les recommandations pour la qualité des eaux en vue de la

protection de la vie aquatique (CCME, 1991a) qui s'appliquent au naphtalène, à l'acénaphtène, au fluorène, au phénanthrène, à l'anthracène, au fluoranthène, au pyrène, au benzo[*a*]anthracène et au benzo[*a*]pyrène. Des concentrations-seuils dans le sol ont été examinées pour la protection de la vie aquatique (dulcicole) contre les autres HAP non substitués; la démarche, visant à établir des concentrations-seuils acceptables dans les eaux réceptrices, était fondée sur les teneurs critiques dans les organismes ou la narcose non polaire. Les concentrations définies pour la protection de la vie aquatique ont été calculées en partie à l'aide d'un modèle relatif aux eaux souterraines adapté de Domenico et Robbins (Domenico, 1987), comme il est décrit dans le protocole de 2006 du CCME.

En ce qui concerne les terrains à vocation agricole et résidentielle/parc, des RQS_I préliminaires ont été élaborées d'après l'ingestion de sol et de nourriture par la vache (animal d'élevage représentatif) et par le cerf-mulet, le campagnol des prés et le Merle d'Amérique (espèces sauvages représentatives).

Pour assurer la protection de la santé humaine, pour tous les types de terrains, on a élaboré des recommandations relatives au contact direct (RQS_{CD}) par ingestion, inhalation et contact cutané combinés, soit une équivalence de toxicité totale relative au benzo[*a*]pyrène, ou ETT relative au B[*a*]P, de 0,6 $mg \cdot kg^{-1}$ et 5,3 $mg \cdot kg^{-1}$, d'après un risque accru de cancer pour toute une vie (RACV) de 10^{-6} et 10^{-5}, respectivement. Les ETT relative au B[*a*]P sont calculées à l'aide des facteurs d'équivalence de toxicité (FET) de l'Organisation mondiale de la Santé (WHO/IPCS 1998). Les ETT relative au B[*a*]P comprennent les concentrations du B[*a*]P lui-même.

Dans les sites contaminés par des résidus de goudron de houille ou des mélanges de créosote, les études indiquent que les profils de potentiel cancérogène par rapport au B[*a*]P, combinés au coefficient de cancérogénicité du B[*a*]P, ne permettent pas toujours de prédire adéquatement les risques de cancer associés au mélange. Il faut donc appliquer un facteur de sécurité de 3 pour calculer l'ETT relative au B[*a*]P dans des sites contaminés par la créosote ou le goudron de houille avant de procéder aux comparaisons avec la RQS_{CD}. En d'autres mots, pour ces mélanges complexes, il convient de multiplier par trois (3) les concentrations équivalentes totales de B[*a*]P (calculées en appliquant les valeurs de l'ETT) avant de caractériser les risques par comparaison aux recommandations visant les HAP.

Afin de protéger la santé humaine, des valeurs pour des composantes constituant les recommandations pour la qualité des sols en vue de la protection de l'eau potable (RQS_{EP}) ont été établies pour chacun des HAP cancérogènes. Il convient de noter que les valeurs des composantes le RQS_{EP} ne sont pas des recommandations pour la qualité du sol à part entière. Chacune de ces valeurs a plutôt été intégrée dans l'équation de l'indice de risque cumulatif de cancer (IRCC) afin de tenir compte des effets combinés des HAP individuels dans le mélange. Les valeurs des composantes dans l'eau potable s'établissent comme suit (tous terrains confondus) :

benzo[*a*]anthracène	0,33 $mg \cdot kg^{-1}$
benzo[*b*+*j*+*k*]fluoranthène	0,16 $mg \cdot kg^{-1}$
benzo[*g*,*h*,*i*]pérylène	6,8 $mg \cdot kg^{-1}$
benzo[*a*]pyrène	0,37 $mg \cdot kg^{-1}$

chrysène	2,1 $mg \cdot kg^{-1}$
dibenzo[*a*,*h*]anthracène	0,23 $mg \cdot kg^{-1}$
indéno[1,2,3-*c*,*d*]pyrène	2,7 $mg \cdot kg^{-1}$

Aux fins de la protection de l'environnement non humain, les recommandations pour la qualité des sols visant les terrains agricoles et résidentiels/parcs sont les suivantes :

anthracène	2,5 $mg \cdot kg^{-1}$
benzo[*a*]pyrène	20 $mg \cdot kg^{-1}$
fluoranthène	50 $mg \cdot kg^{-1}$

Aux fins de la protection de l'environnement non humain, les recommandations pour la qualité des sols visant les terrains commerciaux et industriels sont les suivantes :

anthracène	32 $mg \cdot kg^{-1}$
benzo[*a*]pyrène	72 $mg \cdot kg^{-1}$
fluoranthène	180 $mg \cdot kg^{-1}$

Bien que des recommandations relatives à certaines voies d'exposition aient été élaborées pour d'autres HAP, aucune RQS_E globale ne peut être faite pour bon nombre d'entre eux en raison du manque de données sur le contact avec le sol.

Règle générale, les Recommandations canadiennes pour la qualité des sols en vue de la protection de l'environnement et de la santé humaine sont fondées sur les plus faibles valeurs de toutes les RQS propres à chacune des voies d'exposition et à chacun des récepteurs. Toutefois, dans le cas des HAP, étant donné que les recommandations pour certaines voies d'exposition sont fondées sur les concentrations d'HAP individuels tandis que celles visant d'autres voies d'exposition sont fondées sur la toxicité cumulative d'un mélange d'HAP calculée d'après les ETT relatives au B[*a*]P ou l'IRCC, on ne peut comparer directement toutes les recommandations en vue de choisir la valeur la moins élevée. Donc, en appliquant les recommandations, il convient de comparer les concentrations d'HAP mesurées dans les échantillons de sol à celles recommandées pour toutes les voies d'exposition considérées.

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In 2010, this document was revised to improve the understanding of how to implement the PAH soil quality guidelines (e.g. Figures 8-1 and 8-2 were added and Table 8-1 was redesigned). In addition, errors in Tables 7-9 and 7-10 were corrected.

1. INTRODUCTION

Canadian Environmental Quality Guidelines (CEQGs) are intended to protect, sustain and enhance the quality of the Canadian environment, its value for social and economic well-being, and its intrinsic value. The guidelines are generic numerical concentrations or narrative statements that specify levels of toxic substances or other potential stressors in the environment, established using the best available scientific information, and below which risks to humans or wildlife are expected to be negligible. Further, CEQGs are intended to ensure no contaminant-related encumbrances to the specified uses of water, sediment or soil. These values are nationally endorsed through the Canadian Council of Ministers of the Environment (CCME) and are recommended for managing release sites and surrounding environs containing toxic substances and other parameters (for example, nutrients, pH) of concern in the ambient environment.

Through the National Contaminated Sites Remediation Program (NCSRP), the CCME Subcommittee on Environmental Quality Criteria established the framework for the Canadian Soil Quality Guidelines for Contaminated Sites in 1991. An interim set of soil quality criteria was adopted from values that were used in different provinces within Canada (CCME, 1991) in response to the urgent need to begin remediation of high priority "orphan" sites with historical contamination. Although the NCSRP program officially ended in March of 1995, the development of soil quality guidelines has been guided subsequently under the direction of the CCME Soil Quality Guidelines Task Group (SQGTG) based on a continuing need for scientifically defensible national soil quality guidelines for managing contaminant and stressor risks in and associated with Canadian soils, with a particular focus on the investigation and remediation of contaminated sites.

Canadian Soil Quality Guidelines (CSQGs) are developed according to a nationally approved protocol developed under the auspices of CCME (CCME, 2006). According to this protocol, both environmental and human health soil quality guidelines are developed for each of four land use scenarios: agricultural, residential/parkland, commercial, and industrial. The lowest value generated by the two approaches (human health based and ecologically based derivation) for each of the four land uses is recommended by CCME for incorporation within the Canadian Soil Quality Guidelines. CSQGs for a number of substances were developed using this protocol and summarized in a comprehensive report entitled *Canadian Environmental Quality Guidelines* (CCME, 1999). The interim soil quality criteria (CCME, 1991) should be used only when soil quality guidelines have not since been developed for a particular substance using environmental and human health risk-based protocols (CCME, 2006).

The "Protocol for the Derivation of Environmental and Human Health Soil Quality Guidelines" was updated in 2006, and forms part of the foundation for this document (CCME, 2006). Note that the CCME protocols for the derivation of water quality guidelines were also being updated in 2006.

1.1 Objectives of this Document

The contamination of soil by mixtures containing polycyclic aromatic hydrocarbons (PAHs) is widespread in Canada due to the near ubiquitous nature of its major sources: namely, the release of various petroleum hydrocarbon or coal-derived products as well as the production of PAHs through a variety of combustion processes/types such as vehicle exhaust or a wide variety of industrial processes. CCME risk-based soil quality guidelines have been adopted within the last five years for two individual PAHs: (i) naphthalene, a commonly occurring, lower molecular weight PAH that has not been confirmed by the scientific and health community to be a known or probable human carcinogen[1]; and (ii) benzo[a]pyrene, a higher molecular weight PAH that is commonly found in soils contaminated with coal tar or combustion-derived PAH mixtures, and which is recognized to be an exceptionally potent carcinogen among the more commonly studied suite of PAHs.

For the 16 to 18 PAHs most commonly analyzed as part of environmental investigations and research, there were no risk-based CCME soil quality guidelines prior to this assessment for any but the two mentioned above. Humans and other living organisms, however, are generally exposed to complex mixtures of PAHs at PAH-contaminated sites.

This scientific criteria document is intended to address current management gaps for PAH-contaminated soils. The document contains a review of information on the chemical and physical properties of primarily "unsubstituted" PAHs (see Chapter 2 for an explanation), a brief review of sources and emissions in Canada, the expected environmental fate based on key environmental process, and toxicological significance based both on cancer and a myriad of other toxicity endpoints for humans, microbes, plants, and animals other than humans. This information is used to derive preliminary soil quality guidelines for higher molecular weight (4 to 7 ring) unsubstituted PAHs that are potentially carcinogenic to humans and wildlife, and for unsubstituted PAHs that may be toxic based on a range of mechanisms.

In the case of soil microbial communities, soil invertebrates, plants and animals, soil quality guidelines (SQGs) for environmental health are derived for PAHs based on the toxicological endpoints from the literature that were intended to be both the most sensitive and most ecologically relevant for Canadian environmental protection goals. In practice, the shortage of available scientific information directly imposed limitations on this intent.

The development of these guidelines parallels recent efforts by other countries to re-evaluate especially the human health risks of PAHs. The derivation of Canadian SQGs benefits from the broader international efforts. In particular, interested readers may wish

[1] Naphthalene has previously been nominated by the US National Institute of Environmental Health Services (NIEHS) for review and possible listing in the National Toxicology Program (NTP) report on carcinogens. Based on NTP bioassays, there is evidence of carcinogenicity in male and female rats, and equivocal evidence for carcinogenicity of naphthalene in female mice.

to refer to recent substantive compilations of PAH sources, fate, and effects by the World Health Organization (WHO/IPCS, 1998), the Agency for Toxic Substances and Disease Registry of the U.S. Department of Health and Human Services (ATSDR, 1995c), and the European Union Risk Assessment Report on Naphthalene (European Commission 2001). All of these documents are available on-line.

Chapter 2 discusses what PAHs are, as well as their role as carcinogens. Overall, this evaluation and derivation was focused on higher molecular weight, four to seven ring PAHs – especially benz[a]anthracene, benzo[b]fluoranthene, benzo[j]fluoranthene, benzo[k]fluoranthene, benzo[a]pyrene, chrysene, dibenz[a,h]anthracene, and indeno[1,2,3-c,d]pyrene. For ecological receptors, the focus is not on carcinogenesis, but rather on other ecologically relevant toxicological endpoints. This is because contaminant risks to animals other than rare and endangered species are typically managed through policy decision based on potential impairment to whole sub-populations, populations or communities. The role of individual cancers in reduced fecundity or survivorship of wildlife or domestic animals is not clear. For animal ecological receptors, cancer-type endpoints are of very doubtful ecological relevance, but may nonetheless be of strong interest to some members of the Canadian public in the context of ecological indicators.

The Canadian Soil Quality Guidelines presented in this document are intended as generic guidance, and the procedures used for their derivation are specifically intended to ensure an adequate minimum level of human health and ecological protection at the vast majority of sites where they might be used. They are risk-based soil contaminant thresholds that, if not completely accurate, may err on the side of being overly protective relative to actual risks at a specific site – a thoroughly considered trade-off for the fact that such "bright-line" management tools are generic estimates that are easily applied with a lesser need for either expertise in their use and interpretation or detailed information on the characteristics of a particular site *vis-à-vis* ecologically relevant processes, contaminant fate in soil, subsurface soils, water and biota, and human activities relevant to contaminant exposure. Site-specific conditions should nonetheless be considered in the application of the generic soil quality guidelines. The reader is referred to CCME (1999a) for guidance on appropriate and inappropriate uses of the guidelines, as well as their place as Tier I contaminated soil management tools within a larger, 3-tiered framework (with Tier II comprising site-specific adjustments to the generic guidelines, and Tier III comprising detailed, site-specific risk assessment).

The Canadian Soil Quality Guidelines are derived to approximate maximum soil concentrations leading to no- or low-effects level (or threshold level) responses, or no unacceptable incremental cancer risks[2], based only on the toxicological data, the uncertainty about its relevance for actual risks, and associated environmental fate information available for the contaminant(s) of potential concern. They do not

[2] Generally, cancer risks that result in an excess population risk of less than 1 in 1,000,000 are considered *de minimus*, and *insignificant*. Occupational risks are generally tolerated at higher levels (ca. 1/100,000 to 1/10,000). Specific jurisdictions, however, may have developed more prescriptive policies regarding acceptable cancer risks.

incorporate information related to socioeconomic, technological feasibility, or political issues. Such non-scientific factors are for consideration by site managers and their agents at the site-specific level, as their influence is expected to vary across different provincial and territorial jurisdictions. The reader is directed to the applicable laws, regulations and guidance in the jurisdiction they are working within for the applicable implementation procedures.

1.2 Overarching Issues for Carcinogenic and Other PAHs

PAHS were among the first set of substances to be assessed as part of the "Priority Substance List 1" (PSL1) under the Canadian Environmental Protection Act (CEPA). According to Environment Canada (1994) –

> "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."

PAHs collectively comprise a suite of hundreds of individual compounds (see Chapter 2). Very little toxicological information, however, exists for the majority of these. The CEPA PSL1 review considered environmental risks of only nine individual PAHs based on the toxicological data available at the time, including acenaphthene, anthracene, benz[a]anthracene, benzo[a]pyrene, fluoranthene, fluorene, naphthalene, phenanthrene, and pyrene.

1.2.1 Human Health

Several of the PAHs have long been recognized as having the potential to cause cancer in a wide variety of vertebrate species, including humans, as well as some invertebrates. Indeed, the much increased risks of lung cancer in cigarette smokers is attributed by the majority of toxicologists and epidemiologists to the inhalation of combustion-derived PAHs (Band *et al.*, 2002), at least in part. In addition, PAHs may play a role in the health effects of respirable particulate matter, often measured as PM_{10} or $PM_{2.5}$ concentrations in air (Clemons *et al.*, 1998).

Human exposure to PAHs has been associated with an increased risk of developing cancer in a variety of tissues, including the lung, bladder, stomach, and skin (including the scrotum), depending on the mode of exposure and the form of PAH (IARC, 2006). Excess incidences of lung cancer have been associated with PAH exposure in a variety of occupational settings, including coal gassification, coke production, paving and roofing, and various occupations involving exposure to creosote or soot. There is evidence for an increased risk for skin and scrotal cancers from occupational exposure to creosote and

coal tar, while excess incidences of stomach and colorectal cancer have been observed for coal gas production workers.

Clearly, cancer as a whole is based on a large variety of causal agents, and exposure to environmental carcinogens such as PAHs is deemed to be a minor contributor relative to smoking, diet, other aspects of lifestyle, and genetic pre-disposition. Regardless of the contribution of carcinogenic PAH exposure from contaminated sites, the Canadian cancer statistics demonstrate that the Canadian public is likely to maintain a broad interest in cancer, especially for potential non-voluntary exposures to carcinogens as might occur at or near contaminated sites.

1.2.2 Environmental Health

The current management void for soil contamination by PAHs other than naphthalene and benzo[a]pyrene potentially applies to non-human receptors in the environment as well. For non-vertebrate animals, plants and microbes, as well as for shorter lived vertebrates, toxicological endpoints other than carcinogenesis are likely to be more important for individual health and population fitness. Photoinduced PAH toxicity, for example, merits examination as a potentially important ecologically relevant endpoint for plants and some species of soil-associated animals. Scientific data on photoinduced toxicity has been used to develop Canadian water quality guidelines for freshwater life protection for some of the PAHs (see Section 5.6). Some PAHs have been labeled as endocrine disrupting substances by toxicologists (Clemons *et al.*, 1998; Safe *et al.*, 1997; Santodonato, 1997).

This is not meant to suggest that an increased PAH-induced cancer incidence in terrestrial and aquatic wildlife populations is not important as a risk management concern in the context of environmentally sustainable human development. There is a large body of scientific literature on the appearance of cancer in a number of anatomic sites, including liver, in bottom-dwelling fish (Kleinjans and van Schooten, 2002); for example, the case of English sole in the urbanized Duamish Estuary of Puget Sound. In the terrestrial environment, however, there are few definitive studies of genotoxicity or carcinogenicity in invertebrate or vertebrate animals that can be confidently ascribed to exposure to environmental carcinogens (Kleinjans and van Schooten, 2002).

Van Schooten *et al., (*1995) evaluated PAH-DNA adducts in the earthworm species (*Lumbricus terrestris*) and demonstrated that such adducts were formed when earthworms were kept on industrially contaminated soils for several weeks. Adduct formation increased with exposure time. They also noted that earthworms tend to form different adduct patterns in comparison with laboratory-reared rodents or fish. A similar time-dependent increase in adduct formation in earthworms kept on PAH contaminated soils, was observed by Walsh *et al., (*1995), as reported in Kleinjans and van Schooten (2002).

No scientific studies were found for terrestrial animals where increased cancer incidence, as opposed to markers of genotoxicity, was ascribed to soil contamination. Further, for

non-endangered species, the contaminant risks to wildlife and other ecological receptors attributable to individual terrestrial sites have generally been managed toward the protection of populations or sub-populations, unlike the practice for human beings for which protection of individuals is important in and of itself. Increased cancer incidence in animals other than humans theoretically has the potential to reduce population fitness; however, cancer often has a latent period such that most individuals affected are older, and have may have already passed the age at which reproductive output is maximized. For this and other reasons, the relationship between cancer incidence, reproductive impairment, and population fitness is unclear.

1.2.3 PAH Mixtures

The major sources of PAHs to soils at any given location invariably contribute a mixture of PAHs, not just single compounds. Various PAH source types can be distinguished based on the characteristic compositions of PAH mixtures, but the contaminated soil matrix is nonetheless challenging from an environmental risk assessment perspective, since in a PAH contaminated soil there is likely to be a diverse compositional range of non-carcinogenic, and carcinogenic PAHs of varying potency.

Few CCME soil quality guidelines or environmental quality benchmarks from other international jurisdictions address the human health and/or environmental risks of mixtures, with a few key exceptions. In fact, environmental quality guideline development within the international scientific and regulatory community has been overwhelmingly focused on the environmental and human health risks associated with single compounds. Generic soil screening levels or soil quality guidelines for the broader suite of PAHs have not been developed in the United States, European Community (including Netherlands), Australia and New Zealand, or elsewhere up to 2005, owing in large part to the many challenges associated with such an undertaking. Current pharmacologically based pharmacokinetic (PBPK) models, used for estimating an internally relevant dose for the purpose of risk assessment, are essentially restricted to single compounds, with a few exceptions.

Humans and ecological receptors are rarely exposed to individual toxicants at contaminated sites. In addition, the behaviour and toxicity of chemicals in mixtures may substantially differ from observations of individual chemicals. For example, the tendency of both non-carcinogenic and potentially carcinogenic PAHs to induce (or sometimes suppress) the cytochrome P450 oxidase (CYP) 1A1 enzyme system may directly influence the subsequent metabolic modification and activation of potentially carcinogenic PAHs.

1.3 Viable Approaches for Assessing the Environmental Risks of Complex Contaminant Mixtures

Pohl *et al., (*1997) summarized the various approaches that were used “on a case-by-case basis” to assess the human health risks of chemical mixtures by the Agency for Toxic Substances and Disease Registry (ATSDR) in the United States. Table 1-1 provides a

summary of the diverse approaches that have been employed by ATSDR for deriving human-health protective "minimum risk levels" (MRLs).

Table 1-1: Examples of ATSDR Approaches for the Establishment of Human Health Minimum Risk Levels (MRLs) for Contaminant Mixtures *(from Pohl et al., 1997).*

MRL Based on One Chemical in Mixture and Others Covered Based on Assumptions About Equivalency	MRL Based on Toxicity Data for Whole Mixtures	MRL Based on Toxicity of Several Components within Mixture	No MRL Derived in Light of Perceived Variability of Mixture Composition
polychlorinated dibenzo-*p*-dioxins	polychlorinated biphenyls	PAHs (non-carcinogenic endpoints)	Automotive gasoline
polychlorinated dibenzofurans	polybrominated biphenyls		Stoddard fuel
	jet fuels		hydraulic fluids
	Otto fuel II		mineral-based crankcase oils
	fuel oils		

MRLs are established by the ATSDR for non-carcinogenic risks as the highest no-observed adverse effects level (NOAEL) or the lowest observed adverse effects level (LOAEL) for the most sensitive test organism and relevant toxicological endpoint.

Since 1997, toxicity assessments for petroleum hydrocarbon products in the United States have evolved to compensate for variable mixture compositions across products released and across sites, using estimates of toxicity for 17 different mixture sub-fractions - referred to as the Total Petroleum Hydrocarbon Criterion Working Group (TPHCWG) sub-fractions (TPHCWG, 1997a,b).

In Canada, some examples of contaminant mixtures that have been addressed for the derivation of environmental quality guidelines include the following:

1. **Management based on toxicity of whole mixtures**:

   polychlorinated biphenyls (PCBs) comprising up to 209 individual congeners – managed based on information about the toxicity of PCB Aroclor technical fluids.

2. **Management based on the toxicity of mixtures that are narrower sub-fractions of larger mixtures**:

petroleum hydrocarbon (PHC) constituents in the case of soil invertebrate and plant protection [i.e., based on separate assessment of CWS F1 (nC6-nC10), F2 (nC10-nC16), F3 (nC16-C34), F4 (>C34) fractions].

3. **Management based on toxicological equivalence to a single surrogate**:

   (a) Polychlorinated dibenzo-*p*-dioxins and polychlorinated dibenzofurans, comprising up to 210 individual congeners – managed based on estimated potency of interaction [NATO I-TEFs (International Toxicity Equivalence Factors) and more recently revised World Health Organization TEFs] with cytosolic Ah-receptor relative to 2,3,7,8-TCDD and assumed additivity of effects.

   (b) nonylphenol and its ethoxylates (CCME, 2002) – Canadian soil quality guidelines based on a toxicity equivalence (TEQ) scheme from Servos *et al*., (2000) assuming an additive, narcosis-type mode of action.

   (b) Canada-Wide Standards for Petroleum Hydrocarbons for human health protection [In part: the reference doses used for many of the TPHCWG sub-fractions, used in turn to develop Canadian soil quality standards for human health protection were often based on single surrogate toxicity data (see TPHCWG, 1997b, and CCME, 2008)].

In general, there are only a few options for addressing the environmental risks of chemical mixtures. The range of options, along with some of the pros and cons of each, is summarized in Table 1-2.

The USEPA (1999, 2000a) developed a guidance document on the assessment of human health risks of complex mixtures. Three major approaches were discussed therein: (i) use of toxicity data for the mixture of interest (e.g., use of creosote toxicity data applied to evaluate risks at a creosote-contaminated site; (ii) use of toxicity data for a similar mixture; and (iii) evaluation of the mixture through a detailed examination of its individual components. Overall, the USEPA (1999, 2000a) approach is summarized graphically in Figure 1-2.

For PAHs, there is limited information on the carcinogenicity or non-carcinogenic toxicity of PAH-containing mixtures such as coal tar, creosote, and mineral oils. Mixtures that are considered by the International Agency for Research on Cancer (IARC) to be probable human carcinogens (IARC Group 1) include coal tars, coal tar pitches, unrefined and mildly treated mineral oils, shale oils, soots, tobacco smoke, as well as byproducts of aluminum production, coal gasification, and coke production.

A range of options is explored herein for deriving soil quality guidelines for the potentially carcinogenic and other PAHs. These included an evaluation based on toxicity of individual PAHs, critical evaluation of currently available schemes for assessing

relative potencies of PAHs, and an examination of toxicity data for whole PAH-containing mixtures such as mineral oils and coal tar.

Table 1-2: Analysis of Risk Assessment Approaches for Chemical Mixtures within the Context of Environmental Quality Guidelines Development

Approach	Advantages	Disadvantages
1. Select most toxic chemical from mixture and assume an equivalent toxicity on a concentration or molar basis of all mixture constituents.	Likely to over-estimate rather than under-estimate risks and is therefore appropriate for generic application.	Requires at least some knowledge about relative toxicity of compounds, when choosing most toxic component.
2. Treat mixture as a single entity and develop risk-based guidance by assuming the mixture acts as a single toxicant (especially using laboratory toxicity and/or epidemiological studies based on whole mixture exposures).	Toxicological threshold information is based on actual data, which accounts for mixture effects.	If composition of mixture varies (e.g., based on differences in source types, persistence, or differential partitioning) toxicity data from one mixture may not adequately represent others.
3. Divide mixture into fractions with similar environmental fate and toxicological properties and define a generic toxicity reference value for each of these.	Accounts to some extent for compositional variation in the overall variation by taking into account relative composition as sub-fractions.	Sub-fraction data is generally not available; level of effort required to generate sub-fraction toxicity data is high owing to need to artificially create sub-fractions for toxicity testing.
4. Ignore potential cumulative toxicity of many or most individual constituents in mixture, and evaluate/manage risks based only on individual substances.	Avoids practical challenges of assessing mixtures.	May fail to account for the major component of toxicological risk.
5. Assign relative toxicities to different components of mixture based on either their experimentally observed relative potency/toxicity or predictions based on molecular structure -activity relationships.	Enables the establishment of relative toxicity or potency in the absence of detailed laboratory toxicity data and the predictive models can be used to manage related compounds for which no data exist.	Structure-activity models tend to predict relative activity based only on one step of a multi-step pathway, from exposure to mixtures in the external medium to induction of the ecologically relevant toxic action (e.g., relative tendency of different constituents to bioaccumulate may be different than relative tendencies to undergo metabolic modification, or undergo interaction with one or more receptors).

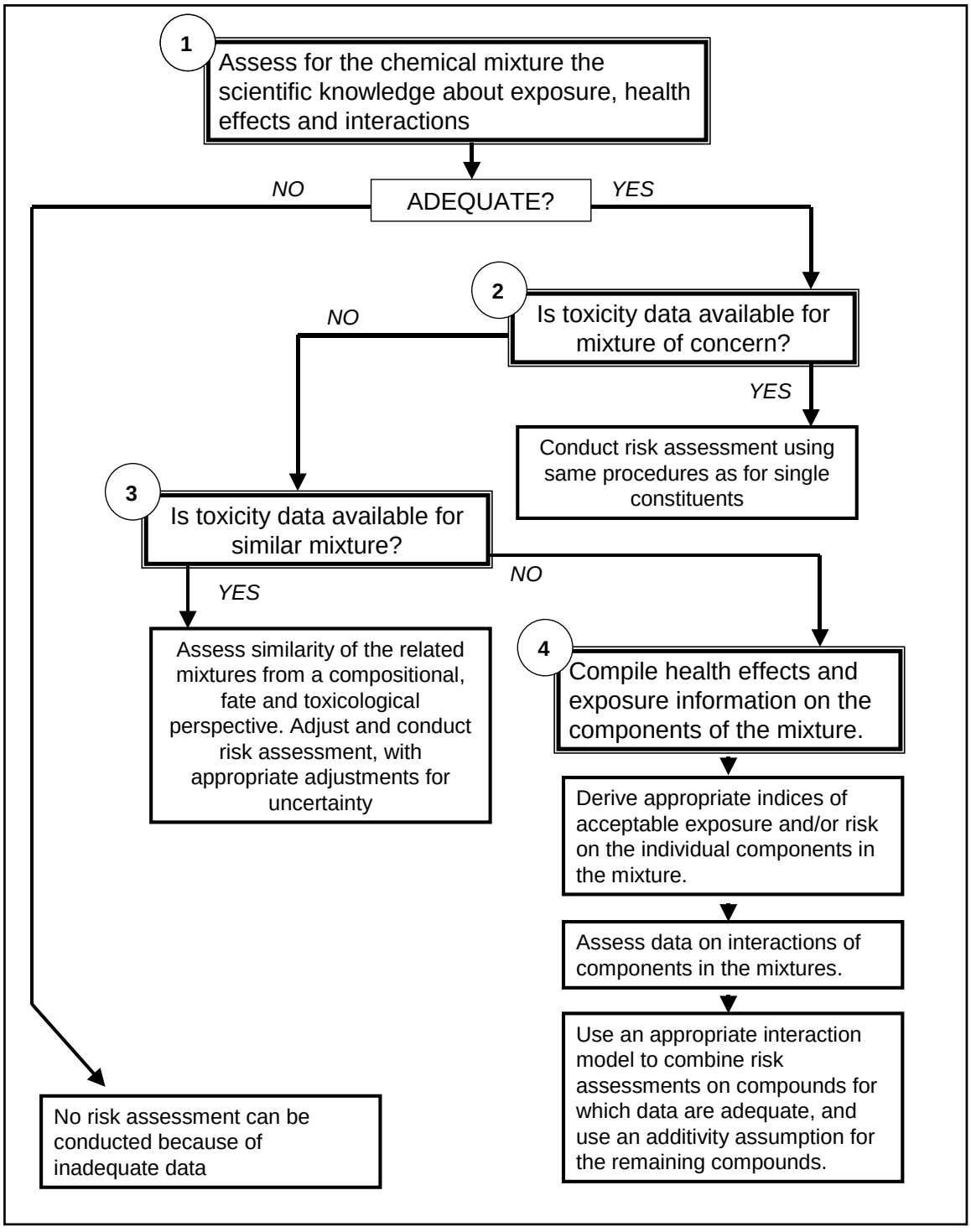


Figure 1-1: Scheme for assessing the environmental risks of complex contaminant mixtures.

2. BACKGROUND INFORMATION

2.1 What are Carcinogenic and Other PAHs?

PAHs are a group of complex hydrocarbons comprised of two or more fused benzenoid rings. Figure 2-1 shows the 16 most commonly evaluated unsubstituted PAHs.

PAHs can be produced during combustion, released from fossil fuels and other petroleum products, or formed from natural plant and bacterial products. PAHs are common constituents of fuels and lubricating oils. Obvious possible sources of PAH inputs to a site include fuel spills, and leaks from aboveground and underground storage tanks (USTs). The combustion of fuels and coal has caused widespread environmental PAH contamination on a global scale in association with atmospheric transport pathways (Laflamme and Hites, 1978). The combustion of organic material, especially of coal and wood, can also produce PAHs. Many PAHs also have natural sources; for example, through combustion of organics during forest fires or as primary or secondary products of natural plant and microbial metabolism. Sources of PAHs are described in more detail in Section 2.5.

The unsubstituted PAHs shown in Figure 2-1 are a very small subset of a much larger suite of unsubstituted and substituted PAHs. “Substituted” PAHs exhibit other substituents at one or more positions around the ring structure; these substituents include alkyl-groups, chlorines and/or bromines, hydroxy-, dihydroxy-, or dihydrodiol- groups, arene oxides, methylsulfones, or other groups.

There is a large body of scientific literature on unsubstituted PAHs, including studies of physicochemical properties, environmental fate, ecotoxicology and particulars of environmental degradation [Varanasi (1989) and Wilson and Jones (1993) provide good reviews]. The environmental and toxicological significance of substituted PAHs, however, is largely unknown; yet alkyl-substituted PAHs, in particular, comprise a much greater portion of most petroleum deposits and products than unsubstituted PAHs.

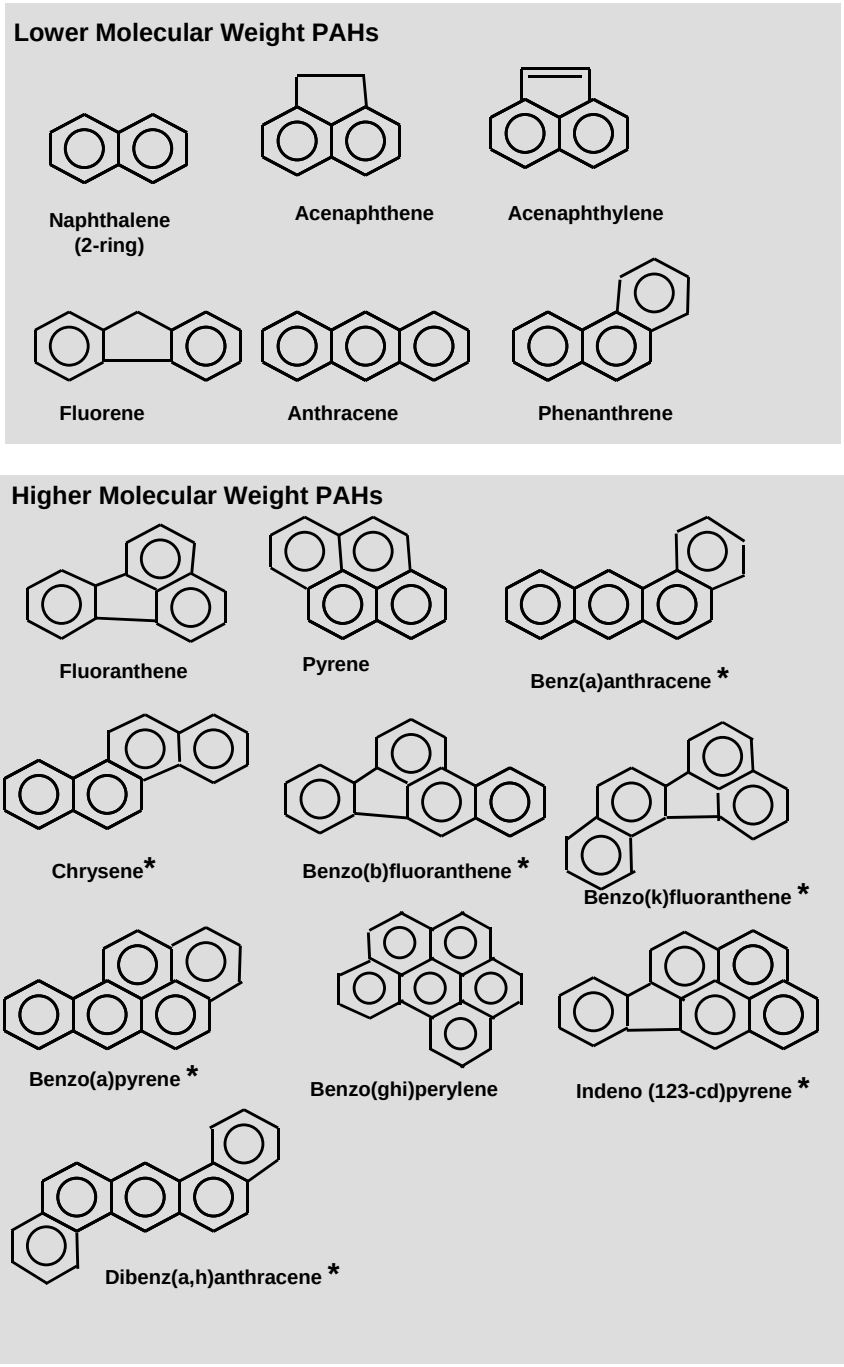


Figure 2-1: Structure of sixteen commonly evaluated unsubstituted PAHs.
Those indicated with an asterisk (*) were of particular interest based on possible carcinogenicity.

PAHs are often divided into two classes: low molecular weight PAHs (LPAHs) and high molecular weight PAHs (HPAHs) (Figure 2-1). LPAHs (e.g., naphthalene, acenaphthene, acenaphthylene, fluorene, anthracene, phenanthrene) tend to have a core structure of two to three benzenoid rings (six-sided aromatic rings of carbon). HPAHs tend to have molecular structures of four or more benzenoid rings, and include fluoranthene, pyrene, benzo[a]pyrene, and benzofluoranthenes. The discrimination between different PAHs based on molecular weight is a useful one, since the hydrophobicity, tendency for bioaccumulation, resistance to biodegradation, and overall environmental persistence generally increase with increasing molecular weight. LPAHs such as naphthalene tend to be more acutely toxic to aquatic organisms than HPAH since they are more water-soluble.

PAHs may be further categorized in alternant and non-alternant PAHs. Alternant PAHs are those with a core ring structure composed entirely of benzenoid rings (In Figure 2-1: naphthalene, phenanthrene, anthracene, pyrene, benz[a]anthracene, chrysene, benzo[a]pyrene, benzo[g,h,i]perylene, dibenz[a,h]anthracene). Non-alternant forms also include four, five, and six-member, non-aromatic ring structures; for example, acenaphthene, and benzo[k]fluoranthene.

The toxicity and environmental cycling of PAHs may be further modified by the presence of various molecular side groups around the central ring structure. This derivation focuses primarily on the higher-molecular weight, unsubstituted PAHs shown in Figure 2-1. A brief discussion of some of the substituted forms is also provided.

Alkylated PAHs, having attached carbon-hydrogen chains (especially methyl groups, but also isopropyl or other alkyl groups), have been frequently identified in environmental samples. In addition, the environmental chemistry of halogenated PAHs, especially those containing chlorine or bromine atoms, is of strong current interest to several environmental chemists. These aromatic chloro- and bromohydrocarbons may be produced by the combustion of PCB oils, municipal waste incineration, fuel consumption, wood fires, or other methods. Processes such as pulp krafting or bleaching, waste water disinfection and wood preservative/pesticide manufacture could also produce halogenated PAHs (e.g., polychloroanthracenes, polychloropyrenes). Basic research on alkylated and halogenated PAHs in the environment has been very limited up to the last five to ten years; therefore, knowledge of environmental cycling or toxicity is very limited.

Alkyl-PAHs can be analyzed as individual isomers (e.g., as 2,6-dimethylnaphthalene) as well as the sums of homologue groups (C0-, C1-, C2-, C3-, C4-, C5-,...) derived from total ion chromatograms using gas chromatographic/mass spectrometric analysis. For example, the two alkyl-PAHs shown in Figure 2-2 would have the same molecular weight, and would jointly be reported as C3-naphthalenes; i.e., naphthalene compounds with three carbons attached as part of side groups. Homologue group totals are derived assuming that all compounds with identical mass are structural isomers, and the chromatographic peaks do not represent other, unrelated compounds.

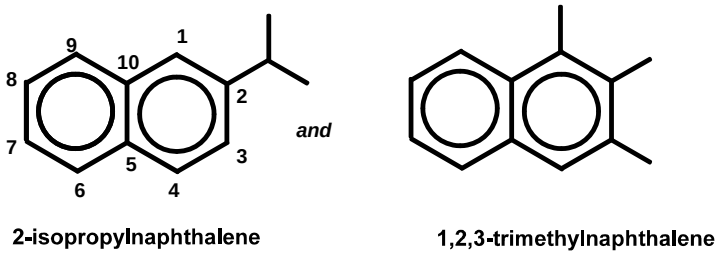


Figure 2-2: Example of Alkyl-PAHs

Possible concern over alkylated PAHs derives from both the routine occurrence of relatively high concentrations in hydrocarbon-contaminated environmental samples and the potential for adverse effects on living organisms. Lee *et al., (*1981) reviewed studies of the toxicity of alkylated PAHs relative to their unsubstituted (parent) analogues: Some alkylated PAHs are less toxic than the unsubstituted compound, whereas other alkylated forms are considerably more toxic. Chrysene, for example, is considered to be only slightly carcinogenic (cancer-causing) to rodents, while 5-methylchrysene is a far more potent carcinogen, and is commonly used as a model carcinogen in scientific studies. The location of alkyl groups around the ring structure may strongly influence carcinogenicity and other toxic effects, and understandings from quantitative structure activity relationships in the last ten years have improved our predictive ability in this area.

Anthracene is generally considered to be non-carcinogenic, whereas 9,10-dimethylanthracene may be strongly carcinogenic (Searle, 1984). Similarly, while unsubstituted benz[a]anthracene and 1- or 2-methylbenz[a]anthracene are only weakly carcinogenic, 7- and 12-methylbenz[a]anthracene are more potent carcinogens. The C2-benz[a]anthracene 1,12-dimethylbenz[a]anthracene does not induce carcinogenic lesions in laboratory models, whereas 7,12-dimethylbenz[a]anthracene is strongly carcinogenic (Searle, 1984) and has also been used extensively as a model carcinogen for cancer research. The effect of degree and position of methylation on carcinogenicity of benz[a]anthracene is apparent in Table 2-1.

The relative carcinogenicity of different positional isomers (PAHs differing only in the position of the alkyl group) is related in part to the fact that PAHs must undergo metabolic activation in the receptor organism to act as a carcinogen (described in greater detail in the next section). It is the production of electrophilic, metabolic intermediates of PAHs that facilitates covalent bonding to the host cell DNA, leading possibly to neoplastic cell transformation. The position and number of alkyl groups will directly influence the stereochemistry (three dimensional molecular shape) of the 'activated' metabolic intermediates, as well as the electron charge delocalization around the ring structure. Whereas 7,12-dimethylbenz[a]anthracene is strongly carcinogenic, 5,7,12-trimethyl-benz[a]anthracene is not (Pullman and Pullman, 1955).

Table 2-1: Carcinogenic Activity of Unsubstituted and Alkyl-substituted Benzanthracenes

(after Searle, 1984) ('-' inactive, '+' weak, '++' moderate, '+++' strong)

Compound	Experimentally Determined Activity
benz[a]anthracene	+
1-methylbenz[a]anthracene	±
2-methylbenz[a]anthracene	±
3- methylbenz[a]anthracene	±
4-methylbenz[a]anthracene	+
5-methylbenz[a]anthracene	+
6-methylbenz[a]anthracene	++
7-methylbenz[a]anthracene	+++
8-methylbenz[a]anthracene	++
9-methylbenz[a]anthracene	+
10-methylbenz[a]anthracene	+
11-methylbenz[a]anthracene	+
12-methylbenz[a]anthracene	++
1,12-dimethylbenz[a]anthracene	-
7,12-dimethylbenz[a]anthracene	++++

2.2 What is Cancer?

Much of the great interest in PAHs is based on the role of these substances in causing cancerous tumours in humans and laboratory rodent models. Among the earliest insights into environmental carcinogenesis was the observation in 1775 by British surgeon Percival Pott that London chimney sweeps tended to have a very high incidence of scrotal cancer, and that this originated from occupational exposure to PAH-containing soot (Boström *et al.,* 2002). Volkman, in 1875, later observed elevated skin cancers in workers in the coal tar industry, and there has since been a large body of evidence confirming the carcinogenic potential of soot, coal tar, and coal tar pitch (Boström *et al.*, 2002).

Cancer is exceedingly complex as a human health/toxicological endpoint, being a multi-step, multi-mechanism process. Overall, carcinogenesis involves genotoxic events (i.e., the formation of genetic mutations) and agents that are known mutagens are sometimes, but not always, demonstrated carcinogens as well. Carcinogenesis also necessarily involves altered gene expression at the transcriptional, translational and/or post-translational level (epigenetic events), altered cell survival, and perturbations to normally programmed cell senescence and death (apoptosis).

The process of carcinogenesis can be divided into three major phases (Figure 2-3) following uptake from the environment of chemical carcinogens such as benzo[a]pyrene: (i) **initiation**; (ii) **promotion**, and (iii) **progression**.

Uptake of PAHs across the gastrointestinal tract following the incidental ingestion of soil or dust or via dermal contact has been less well studied than the initiation stage of carcinogenesis. Initiation includes those steps between internalization of the PAH dose and interaction with the cell's DNA leading to a potentially non-lethal mutation. The internalized PAHs are not themselves potent mutagens, and parent PAHs have little tendency to bind to DNA. Initiation commences for carcinogenic PAHs through activation by the cytochrome P450 oxidase (CYP) suite and phase II biotransformation enzymes. These catalyze the metabolic conversion of PAHs and other heterocyclic or polyaromatic hydrophobic substances to much more hydrophilic forms in preparation for excretion from the body.

2.2.1 Initiation

Conversion of PAHs to more reactive intermediates, especially by CYP1A-type isoforms of the cytochrome P450 oxidase enzyme complex, is the first step in mutagenesis/ genotoxicity. CYP1A enzyme activity itself may be inducible at the transcriptional and translational level by not just those PAHs demonstrated to be mammalian carcinogens, but also by PAHs and other polyaromatic compounds that have little tendency to cause cancerous lesions in laboratory rodents. The dependence of carcinogenesis on multiple stages and mechanisms makes the evaluation of dose-response relationships very challenging.

Inhibition of toxicity may occur, for example, when the metabolic modification of a PAH to its more toxic intermediate is prevented or minimized by the preferential metabolism of another co-occurring compound. Conversely, an enhancement of toxicity can occur when prior exposure to a CYP-active xenobiotic results in enhanced enzyme activity, and an increased rate of conversion of potentially carcinogenic PAHs to their more toxic metabolic intermediates. Overall, the carcinogenic or otherwise toxic potential of a specific PAH will depend on which enzymes have been induced, the relative affinity for the suite of enzymes for the internalized chemical mixture, and the relative toxicity of the metabolized compounds (WHO/IPCS, 1998).

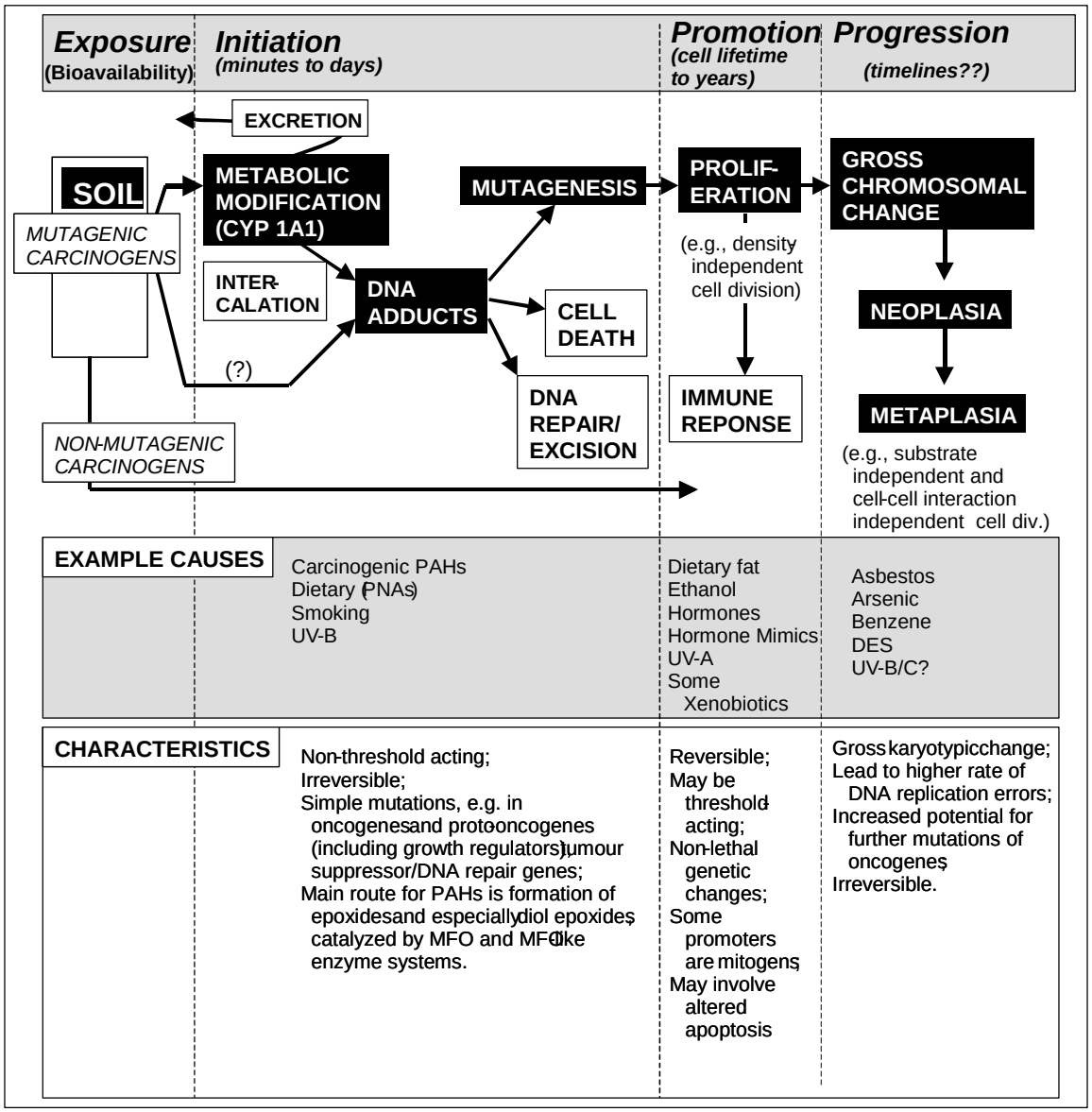


Figure 2-3: Theoretical model for cancer induction by PAHs

The cancer initiation stage is perhaps the best understood mechanistically of all the stages of carcinogenesis as a result of PAH exposures. For potentially carcinogenic PAHs to interact with DNA and subsequently increase the probability of mutagenesis, there is generally a prior requirement for metabolic modification. Initiation of carcinogenesis by benzo[a]pyrene, for example, is hypothesized to begin with the CYP-dependent conversion to start with oxidation at the 7-, and 8- positions to form a 7,8-epoxide, followed by subsequent hydrolysis to 7,8-dihydrodiol (steps 1 and 2 in Figure 2-3). A second epoxidation step yields the ultimate benzo[a]pyrene derived carcinogen: 7,8-dihydrodiol-9,10-epoxide.

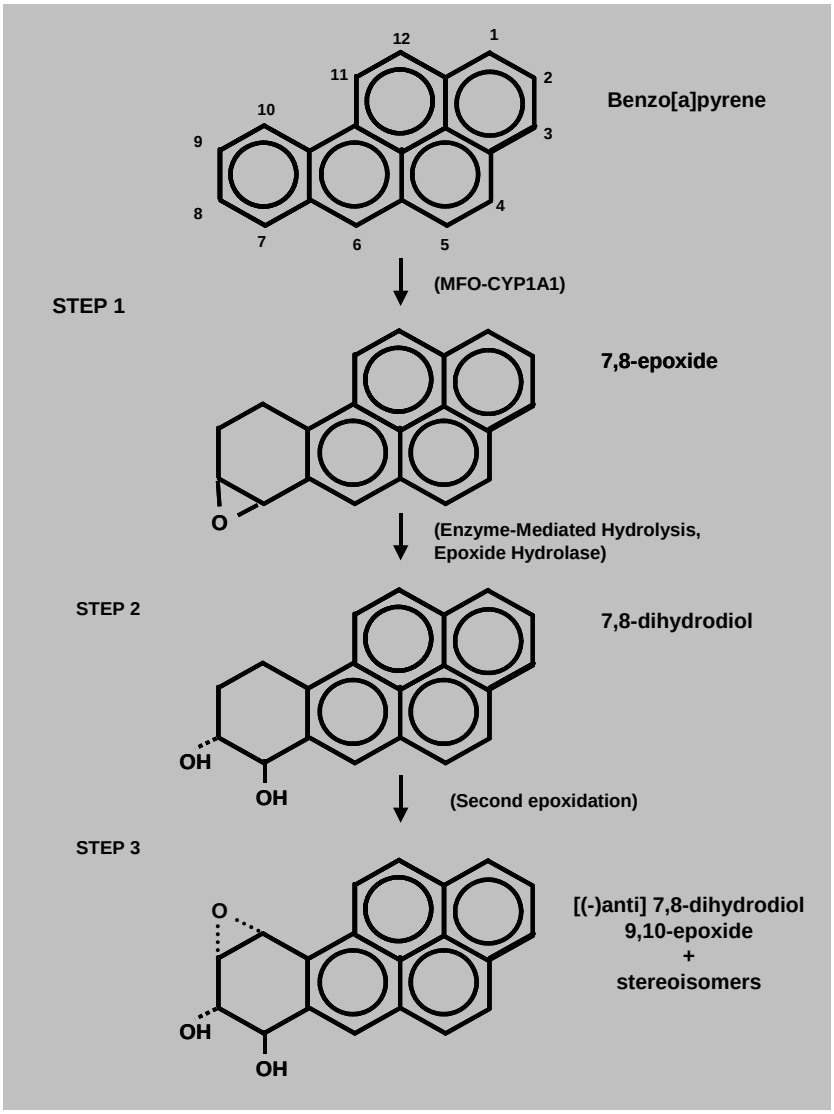


Figure 2-4: Metabolic activation route for benzo[a]pyrene.

In addition to CYP1A1, other P450 cytrochrome oxidases such as CYP1A2, CYP1B1 and CYP3A4 may participate in metabolic modification of PAHs. All of these enzyme systems, in turn, tend to be activated following the interaction of partially planar aromatic organic substances with the Ah-receptor (Aryl hydrocarbon receptor), found in the cytosol of the vast majority of cell types in tissues of virtually all vertebrates examined so far. The Ah-receptor is also the primary site of action of other polyaromatic substances such as 2,3,7,8-tetrachlorodibenzo-*p*-dioxin.

If the 7,8-dihydrodiol structure shown in Figure 2-4 was examined in a three-dimensional view, it would become more apparent that there are several possible stereoisomers of it, depending on whether the hydroxyl group on the 8- position or the 9,10-epoxide ring are positioned away from or toward the viewer (the rest of the aromatic ring structure is a flat, planar structure). The diol epoxides with the highest cancer potency in mammalian

cell cultures and laboratory rodent models are believed to be the *anti*-diastereomers, and especially isomers with an *R*-absolute configuration at the benzylic arene carbon (Boström *et al.,* 2002). Cancer potency differences, therefore, are not associated just with gross variations in the structure of individual PAHs, but also with small stereochemical differences in the corresponding metabolites.

The position of the dihydrodiol complex and the epoxide group, as well as the stereochemistry of the groups for any of the PAHs determines the shape of the electron density cloud locally and across the entire molecule. This in turn directly influences the affinity of the metabolically-modified PAH to bind to (or intercalate with) a variety of regions on the DNA double-helix of higher animals, or the RNA of some prokaryote or eukaryote organelle systems. In other words, some PAHs tend to form stronger PAH-DNA "adducts" following their metabolic conversion than others.

The complexity and diversity of molecules and the associated vast array of emergent properties that arise tends to obscure the simple fact that all molecules can be described in terms of their atomic nuclei and electronic density cloud (Mezey *et al*., 1996). A half century ago, Pullman (1945, 1947) introduced the terms "K- and L-region" to describe the reactivity of PAH based on Hückel molecular orbital calculations (Figure 2-5). Discrete energy values for the electron cloud delocalization at the K- and L- regions of a PAH molecule were thought to be correlated with its carcinogenic potency. The term "bay region" was later introduced (Figure 2-5) to better account for structural correlates of carcinogenicity, since categorizations based on the presence of a K- and L-region were later found to be incompatible with emerging experimental results.

It is widely believed that PAHs with a bay region are carcinogenic, and this is based on two conditions. First, the epoxide group of an ultimate metabolite must be directly adjacent to the bay region, as in the case of the 7,8-dihydrodiol-9,10-epoxide of benzo[a]pyrene. Second, the hydroxy groups of the diol epoxide must be added in the "pre-bay region". The presence of the epoxide group in the bay region facilitates ring opening of the saturated benzene ring to which it is attached, since the delocalization energy forming the carbonium ion is higher based on this configuration.

The carbonium ion thus formed is capable of serving as an alkylating agent, and thus reacting with DNA, to form a PAH-DNA adduct, potentially leading to a mutation.

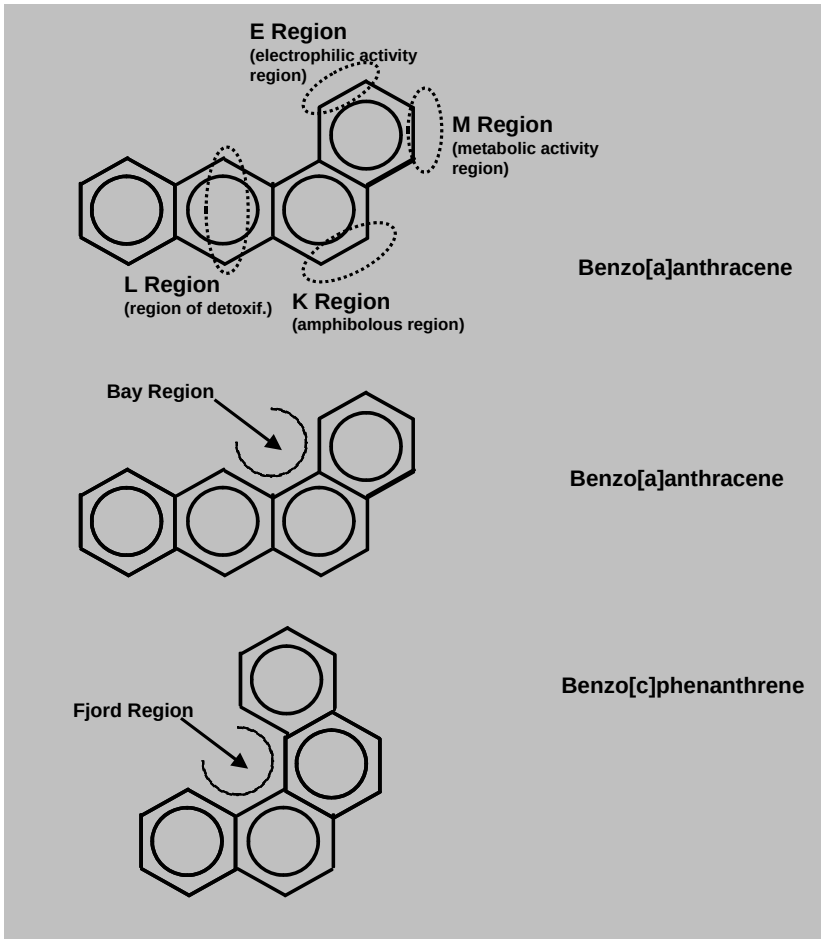


Figure 2-5: PAH structures that have been associated with carcinogenicity

Tumour initiation involves mutation, although there are a large number of biochemical mechanisms that are capable of repairing this initial mutation and thereby arresting carcinogenesis at the initiation stage. As part of the overall initiation event, PAH-DNA adducts can be removed and the DNA repaired based on DNA repair mechanisms. Mutations occur during the imperfect replication of the DNA template during the cell cycle, the probability of which theoretically increases with the number of adducts present in an organism. Many types of modifications to DNA are lethal transformations to the cell, in which case it will no longer divide and proliferate.

As shown in Figure 2-3, alterations in DNA that are neither repaired nor lead to immediate cell death have the potential to result in individual cells with an abnormal ability to divide and proliferate.

Genotoxicity, as the major part of initiation can be further potentiated by co-carcinogens; for example, by other non-carcinogenic PAHs that increase the rate of bioactivation and/or decrease the rate of detoxification and elimination from the body of reactive intermediate metabolites.

2.2.2 Promotion and Progression

Once genetic material is compromised through alkylation with reactive intermediates of PAHs (i.e., through DNA adduct formation), such changes may result in mutation unless either the DNA is repaired by ubiquitous DNA repair mechanisms, or the cell itself ceases to become viable and is removed by apoptosis (programmed cell death).

Promotion involves secondary cell transformations that allow the cell and its future progeny to escape normal physiological controls.

The early stages of promotion are also relatively well understood. Mutations that occur in genes that serve to control the expression of other genes are generally assumed to be responsible for the promotion of carcinogenesis. For example, the "p53 tumor suppressor gene" is believed by many to be turned off by an initiated cell thereby allowing an initiated cell to be promoted and progress into a tumor. Such genes are referred to by cancer researchers as "proto-oncogenes" or "oncogenes", although they are exceedingly important for normal physiological cell function and cell-cell interactions beyond their identified role in cancer promotion. Alterations that lead to changes in the expression of or response to "epidermal growth factor", for example, can lead to cancer promotion in combination with other cell transformations.

In multi-cellular organisms, cells tend to undergo mitotic division only after there is adequate replication of critically important cellular components, and after the DNA itself has undergone replication. Mutations in genes that adversely influence the cell cycle and timing of cell division tend to further influence the departure of the cell line from the normative state, since genetic stability – the ability of the cell to replicate and divide the DNA without error – is compromised. Transformed cell lines are often characterized by highly abnormal karyotypes and nuclear size, with varying degrees of polyploidy or aneuploidy, missing chromosomes, chromosomal re-arrangements, increased incidence of 'satellite DNA' and with a host of less visually obvious genetic aberrations.

At least two other categories of impaired cell division and growth regulation are implicated in the etiology of various cancer types. Normal cells interact with each other, and their division is limited by cell-cell interactions to prevent over-crowding. Promotion, therefore, may be associated with further genetic change resulting in impaired cell-cell feedback of mitotic control.

Another condition for the normal mitotic division of many cells is the association between the cell, along with its exocellular matrix, and the underlying substrate – typically an architectural backbone or substratum, in some cases called a basement membrane, that ensures that cell division and specialization occurs within a complex three-dimensional structure that leads to emergent properties in whole tissues. Another category of transformation involves the alterations of specific oncogenes resulting in loss of feedback control of cell-division based on an association with its substrate.

Transformed cells, that achieve an ability to undergo mitosis within a fluid environment, and removed from the stratum that is normally present, are further capable of "metastasis" – re-locating to and proliferating at secondary sites throughout the body, away from the original site of initiation.

Overall, promotion and progression involve a progressive loss of feedback control that is important for normal cell division, and a progression towards malignancy and spread. Cancer cells progressively lose molecular feedback mechanisms that otherwise serve to prevent cell division at inappropriate times or with inappropriate frequency, over-crowding, and proliferation through other parts of the body. The diversity of possibilities for cancer outcomes increases from initiation through promotion and progression, with the role of apoptosis and immunocompetency being difficult to predict, given the current state of knowledge. The potential for divergence of cancer outcomes between exposure routes, between tissue types, between individuals, and between different species increases from initiation through promotion and progression.

Chemical promoters, unlike chemical mutagens, do not require the interaction with DNA to exert their influence. The resulting changes - sometimes referred to as "epigenetic events" - are considered to be reversible. Tumour progression, following initiation, is thought to be irreversible in the majority of cases; however, chemical promoters can accelerate tumour promotion. The subsequent removal from exposure to promoters can result in a subsequent deceleration in tumour promotion.

2.2.3 Formally Defined Carcinogenic PAHs

The International Agency for Research on Cancer (IARC) provides detailed expert review of the available scientific information on the carcinogenicity of individual substances or mixtures. Table 2-2 (after Collins *et al*., 1998) summarizes the IARC classification for a wide variety of PAHs.

Note that more recently, IARC (2002) identified naphthalene as a Group 2B substance (possibly carcinogenic to humans).

The World Health Organization, through its International Program on Chemical Safety, is engaged in extensive technical evaluations of the toxicological implications to humans of a variety of substances. In 1998, the WHO released "Environment Health Criteria 202: Selected Non-Heterocyclic Polycyclic Aromatic Hydrocarbons" (http://www.inchem.org/documents/ehc/ehc/ ehc202.htm). Those PAH considered to be carcinogenic by the expert group that produced the technical evaluation, per the corrigendum to this report are shown in Table 2-3.

Table 2-2: IARC classifications of the carcinogenicity of several individual PAHs

IARC Group 1: Carcinogenic to humans	**IARC Group 2a:** Probably carcinogenic to humans	**IARC Group 2b:** Possibly carcinogenic to humans	**IARC Group 3:** Not classifiable as to carcinogenic potential due to limited or inadequate data.
• Coal tars • Coal tar pitches • Coke production • Mineral oils • Shale oils • Soots • Tobacco smoke	• Benzo[a]pyrene • Benz[a]anthracene • Dibenz[a,h]anthracene • Creosote	• Benzo[b]fluoranthene • Benzo[j]fluoranthene • Benzo[k]fluoranthene • Dibenz[a,e]pyrene • Dibenz[a,h]pyrene • Dibenz[a,I]pyrene • Dibenz[a,l]pyrene • Dibenz[a,h]acridine • Dibenz[a,j]acridine • Indeno[1,2,3-c,d]pyrene • 5-Methylchrysene • 7H-dibenzo[c,g]carbazole • Carbon black extracts • Naphthalene	• Anthanthrene • Anthracene • Benz[a]acridine • Benz[c]acridine • Benzo[g,h,i]fluoranthene • Benzo[g,h,i]perylene • Benzo[c]phenanthrene • Benzo[e]pyrene • Carbazole • Chrysene • Dibenz[a,c]anthracene • Dibenz[a,j]anthracene • Dibenz[a,e]fluoranthene • Fluoranthene • Fluorene • 1-Methylchrysene • 2-Methylchrysene • 3-Methylchrysene • 4-Methylchrysene • 6-Methylchrysene • 2-Methylfluoranthene • 1-Methylphenanthrene • Perylene • Phenathrene • Pyrene • Triphenylene

Table 2-3: Summary of results of tests for genotoxicity and carcinogenicity for the 33 polycyclic aromatic hydrocarbons

(adapted from WHO/IPCS, 1998).

Compound	Genotoxicity	Carcinogenicity
Acenaphthene	(?)	?
Acenaphthylene	(?)	No studies
Anthanthrene	(+)	+
Anthracene	-	-
Benz[a]anthracene	+	+
Benzo[b]fluoranthene	+	+
Benzo[j]fluoranthene	+	+
Benzo[g,h,i]fluoranthene	(+)	(-)
Benzo[k]fluoranthene	+	+
Benzo[a]fluorine	(?)	(?)
Benzo[b]fluorine	(?)	(?)
Benzo[g,h,i]perylene	+	-
Benzo[c]phenanthrene	(+)	(+)
Benzo[a]pyrene	+	+
Benzo[e]pyrene	+	?
Chrysene	+	+
Coronene	(+)	(?)
Cyclopenta[c,d]pyrene	+	+
Dibenz[a,h]anthracene	+	+
Dibenzo[a,e]pyrene	+	+
Dibenzo[a,h]pyrene	(+)	+
Dibenzo[a,i]pyrene	+	+
Dibenzo[a,l]pyrene	(+)	+
Fluoranthene	+	(+)
Fluorene	-	-
Indeno[1,2,3-c,d]pyrene	+	+
5-Methylchrysene	+	+
1-Methylphenanthrene	+	(-)
Naphthalene	-	(?)
Perylene	+	(-)
Phenanthrene	(?)	(?)
Pyrene	(?)	(?)
Triphenylene	+	(-)

+, positive; -, negative; ?, questionable; (), result derived from small database

This list of potentially carcinogenic PAHs is slightly larger than is recognized by some jurisdictions such as Health Canada, the International Agency for Research on Cancer (IARC) or the USEPA. For the purpose of this exercise, CCME has provisionally defined the carcinogenic PAHs of interest to include benzo[a]pyrene, benz[a]anthracene,

benzo[b]fluoranthene, benzo[k]fluoranthene, dibenz[a,h]anthracene, and indeno[1,2,3-c,d]pyrene as well as the possible or known carcinogens chrysene and benzo[j]fluoranthene.

It should be appreciated, however, that the formal classification of individual PAHs as confirmed, probable or possible carcinogens is highly dependent on the degree to which they have been assessed using a variety of experimental and epidemiological approaches - especially for the large number of alkylated PAHs. There is little doubt that at least some of the presently unclassifiable PAHs will be confirmed as potentially carcinogenic in the future.

2.3 Physical and Chemical Properties

Table 2-4 provides a concise summary of the structural, physical and chemical properties of the most commonly evaluated unsubstituted PAHs. The octanol-water partition co-efficient (K_{OW}), organic carbon-water partition co-efficient (K_{OC}), Henry's Law constant, vapour pressure, and aqueous solubility are chemical specific properties that are of direct relevance in predicting the environmental fate of a substance, including its multi-media partitioning behaviour, bioavailability, and resistance to biodegradation. Chapter 4 examines in more detail the interrelationship between PAH physical-chemical properties and expected environmental fate and toxicity.

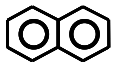
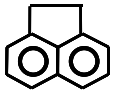
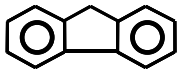
Although in theory the physical-chemical properties of a substance are an inherent characteristic of the molecular structure, the chemical and partitioning co-efficients are often derived through experimental studies or through mathematical prediction. Their actual value, therefore, may vary according to the method used to establish them, and between different researchers. In addition, the specific values of properties such as aqueous solubility vary as a function of the temperature at which the solubility is being measured. The values provided in Table 2-4, therefore, should be regarded as approximate estimates based on various reviews of the currently available scientific literature. The values were not independently assessed as part of this derivation, but rather extracted from the on-line CHEMFate database created on behalf of the USEPA.

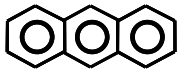
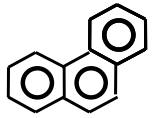
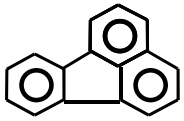
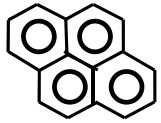
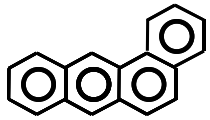
In general, PAHs become increasingly less soluble in water with an increasing number of benzenoid or other rings, and increasing molecular weight. Naphthalene, a two-ring PAH, is the most soluble, with an estimated aqueous solubility of around 32 mg/L at 25^{o} C. Indeno[1,2,3-c,d]pyrene, a six-ring PAH has a much more limited aqueous solubility at room temperature of approximately 2.2×10^{-5} $mg \cdot L^{-1}$, or 22 $ng \cdot L^{-1}$.

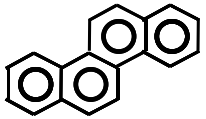
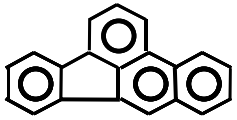
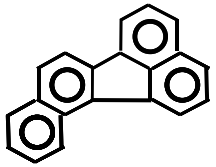
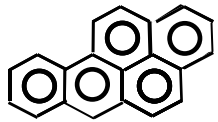
Lower molecular weight PAHs also tend to be more volatile than higher molecular weight PAHs (compare the vapour pressures at room temperature), and more readily partition into air from pure water (indicated by Henry's Law constant).

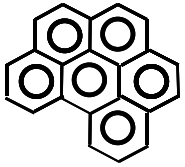
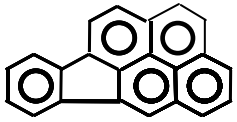
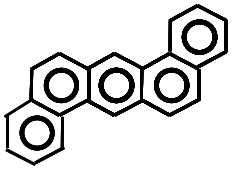
Table 2-4: Physical-chemical properties of PAHs of interest

(from "CHEMFate" database, unless indicated otherwise).

PAH (synonyms)	**Structure**	**CAS Registry No.**	**Mol. Wt.**	**Aqueous Solubility ($mg \cdot L^{-1}$) at room temp**	**Vapour Pressure (mm Hg), room temp**	**Henry's Law Constant ($atm \cdot m^3 \cdot mol^{-1}$)**	**Log K_{OW}**	**Log K_{OC}**
Naphthalene	 $C_{10}H_8$	91-20-3	128	31.7	8.5×10^{-2}	4.83×10^{-4}	3.36[A]	2.7 to 3.0
Acenaphthene	 $C_{12}H_{10}$	83-32-9	154	3.9	2.5×10^{-3}	1.55×10^{-4}	3.92[A]	3.3 to 3.6
Acenaphthylene	 $C_{12}H_8$	208-96-8	152	16.1	9.12×10^{-4}	1.13×10^{-5}	3.9 to 4.1	3.75
Fluorene	 $C_{13}H_{10}$	86-73-7	166	1.9	6.33×10^{-3} to 8.42×10^{-3}	6.34×10^{-5} to 1.00×10^{-4}	4.18	3.45 to 3.95

PAH (synonyms)	**Structure**	**CAS Registry No.**	**Mol. Wt.**	**Aqueous Solubility ($mg \cdot L^{-1}$) at room temp**	**Vapour Pressure (mm Hg), room temp**	**Henry's Law Constant ($atm \cdot m^3 \cdot mol^{-1}$)**	**Log K_{OW}**	**Log K_{OC}**
Anthracene (anthracin; green oil; paranaphthalene)	 $C_{14}H_{10}$	120-12-7	178	0.043 to 0.075	$2{,}67 \times 10^{-6}$	1.93×10^{-5} to 6.5×10^{-5}	4.45, 4.55[A]	4.2 to 4.4
Phenanthrene (Phenanthrin)	 $C_{14}H_{10}$	85-01-8	178	1.15	1.12×10^{-4}	2.33×10^{-5}	4.46, 4.55[A]	3.4 to 4.3
Fluoranthene (Benzo[j,k]fluorene; idryl; 1,2-benzacenaphthene; 1,2-(1,8naphthalenediyl)benzene)	 $C_{16}H_{10}$	206-44-0	202	2.6×10^{-1}	1.23×10^{-8}	1.3×10^{-5} to 1.6×10^{-5}	4.95	4.62
Pyrene (Benzo[d,e,f]phenanthrene)	 $C_{16}H_{10}$	129-00-0	202	1.35	2.45×10^{-6} to 4.59×10^{-6}	1.1×10^{-5}	4.88 to 5.18	4.6 to 5.1
Benz[a]anthracene (Benz[a]anthracene; Benzo[b]phenanthrene; tetraphene; 1,2-benzanthracene; 1,2-benanthrene; 2,3-benzophenanthrene;naphanthracene)	 $C_{18}H_{12}$	56-55-3	228	9.4×10^{-3}	3.05×10^{-8} to 1.05×10^{-7}	3.35×10^{-6}	5.7[A]	5.3

PAH (synonyms)	**Structure**	**CAS Registry No.**	**Mol. Wt.**	**Aqueous Solubility ($mg \cdot L^{-1}$) at room temp**	**Vapour Pressure (mm Hg), room temp**	**Henry's Law Constant ($atm \cdot m^3 \cdot mol^{-1}$)**	**Log K_{OW}**	**Log K_{OC}**
Chrysene (Benzo[a]phenanthrene; 1,2-benzophenanthrene)	 $C_{18}H_{12}$	218-01-9	228	2.0×10^{-3} to 6.3×10^{-3}	6.23×10^{-9}	9.46×10^{-5}	5.7^{A}	5.1
Benzo[b]fluoranthene (Benz[e]acephenanthrylene; benzo[e]fluoranthene; 2,3-fluoranthrene; 3,4-benzofluoranthene; 3,4-benz[e]acephenanthrylene)	 $C_{24}H_{14}$	205-99-2	252	1.5×10^{-3}	5.0×10^{-7}	1.11×10^{-4}	6.2^{A}	5.2
Benzo[j]fluoranthene	$C_{24}H_{14}$	205-82-3	252	6.8×10^{-3}	1.5×10^{-8}	1.0×10^{-6}	6.12	4.7 to 4.8
Benzo[k]fluoranthene (Dibenz[b,j,k]fluorene; 11,12-benzofluoranthene; 2,3,1',8'-binaphthylene; 8,9-benzofluoranthene)	 $C_{24}H_{14}$	207-08-9	252	8.0×10^{-4}	2.0×10^{-9}	8.29×10^{-7}	6.2^{A}	4.3 (?)
Benzo[a]pyrene	 $C_{20}H_{12}$	50-32-8	252	1.6×10^{-3}	5.49×10^{-9}	1.13×10^{-6}	5.97 6.11^{A} 6.58^{B}	6.0 to 6.7

PAH (synonyms)	**Structure**	**CAS Registry No.**	**Mol. Wt.**	**Aqueous Solubility ($mg \cdot L^{-1}$) at room temp**	**Vapour Pressure (mm Hg), room temp**	**Henry's Law Constant ($atm \cdot m^3 \cdot mol^{-1}$)**	**Log K_{OW}**	**Log K_{OC}**
Benzo[g,h,i]perylene (1,12-benzoperylene)	 $C_{22}H_{12}$	191-24-2	276	2.6×10^{-5}	1.0×10^{-10}	1.41×10^{-07}	6.7[A]	5.61
Indeno[1,2,3-c,d]pyrene	 $C_{22}H_{12}$	193-39-5	276	2.2×10^{-5}	1.0×10^{-10}	1.6×10^{-6}	6.6	6.2
Dibenz[a,h] anthracene	 $C_{22}H_{14}$	53-70-3	278	$2,49 \times 10^{-3}$	1.0×10^{-10}	1.47×10^{-8}	6.50, 6.69[A]	5.8 to 6.5

A: USEPA, 1996; B: CHEMFATE DATABASE –(http://esc.syrres.com/efdb/Chemfate.htm)

Major aspects of PAH fate and toxicity in contaminated soils and sediments are related to the degree of lipophilicity (fat solubility), described by the K_{OW}, and the tendency to partition into organic carbon coating the surface of particulates or found in fine colloids, described by K_{OC}. Higher molecular weight PAHs exhibit much greater tendency than lower molecular weight PAHs to remain sorbed to soils or sediments rather than partition into water or air. They also tend to more readily bioaccumulate in organisms from water; for example, through transfer from surface water to aquatic organisms, or through uptake by bacteria, plant rootlets or soil mesofauna from water in soil pore space.

In a two-phase system with water and octanol – a surrogate of biological lipids – naphthalene with a log10 K_{OW} of ~3.36 at equilibrium would tend to partition into the octanol at a ratio of 2,300 parts for every part partitioned into water. Indeno[1,2,3-c,d]pyrene, with a log10 K_{OW} of ~6.6, would tend to partition into octanol at a ratio of approximately 4 million parts for every part partitioned into water.

This analysis is somewhat over-simplistic relative to PAH partitioning at the majority of contaminated sites, since there may be far more than two compartments present (e.g., soil, groundwater, dissolved organic carbon); there may be departures from equilibrium (e.g., if microbial decomposition is actively removing or converting substances at a rate that exceeds desorption rates), or other processes may be important (e.g., co-solvent effects). The physical-chemical properties, nonetheless, offer general guidance on the tendency of a substance to partition into soil, water, air, and biota.

2.4 Relevant Analytical Methods

Detailed reviews of sampling and analytical methods for PAHs in air, water, soil, food and other matrices are provided in WHO/IPCS (1998), ATSDR (1995c), Environment Canada (1994), Environment Canada (1999) and elsewhere. In general, PAH analyses tend to be relatively complicated. On the one hand, the variability across the lower- to higher-molecular weight PAHs in volatility tends to result in loss to air of naphthalene and other lower MW PAHs; and on the other hand, lipophilicity tends to result in incomplete recovery of higher molecular weight PAHs from organic-rich solid matrices such as soil, sediment, or tissues. Specialized extraction and analytical methods have been proposed for different types of sample matrices, each of which may pose different challenges.

For soil samples, the overall quantification of PAHs includes sample collection and handling, sub-sampling in the laboratory, extraction, and quantification. The vast majority of PAH analysis is currently carried out using either gas chromatography (using FID or mass spectrometry detection) or high-pressure liquid chromatography (HPLC).

CCME (1999b) adopted soil quality guidelines for benzo[a]pyrene in 1999. Appropriate analytical methods for the larger suite of unsubstituted PAHs are the same as for benzo[a]pyrene.

Analytical methods of benzo[a]pyrene in soil, as recommended by CCME in 1993 (CCME, 1999b) include U.S. EPA Method 8270. The most recent version of this method is entitled "U.S. EPA Method 8270D (Revision 4) Semivolatile Organic Compounds by Gas Chromatography/Mass Spectrometry (GC/MS)". The method is intended for all types of solid waste matrices, soils, groundwater and surface water. The detection limit for soil and sediment samples tends to be around 0.7 $mg \cdot kg^{-1}$ wet wt.

The availability of this relatively complex and sophisticated U.S. EPA method notwithstanding, relevant jurisdictions in Canada may choose to encourage or allow the use of a variety of performance-based alternatives, and interested parties should consult with the appropriate parties regarding acceptable methods. Commercial analytical laboratories in Canada are routinely certified for PAH analysis by the Canadian Association of Environmental Analytical Laboratories (CAEAL) as partial assurance of the accuracy and reproducibility of PAH analytical data.

2.5 Sources to the Canadian Environment

There is a complex mixture of unsubstituted and substituted PAHs in most environmental samples, the composition of which may depend on the source(s) of input, and possible environmental transformations (Figure 2-6)**.** The expected impact on the ecosystem may also be inferred from this composition. The signature, or compositional pattern of PAHs, for example, can be used to distinguish between combustion-based (pyrogenic) versus petrogenic inputs, or more recent, plant-derived PAHs. The PAH composition in contaminated environmental samples is also useful in delineating between different anthropogenic sources, especially in urbanized, industrialized areas.

Alkyl-PAHs and unsubstituted PAHs can be categorized as –

(i) kinetically-favoured: those preferentially produced through combustion processes (i.e., pyrogenic PAHs);

(ii) thermodynamically-favoured: PAHs which are energetically stable over long periods of time and tending to dominate in more mature environmental/geological compartments such as crude oil or coal deposits (i.e., petrogenic PAHs); and

(iii) PAH derived directly from plant products, and synthesized, at least in part, through biologically-mediated processes.

These categorizations serve simply as a rule of thumb about what the predominant PAHs are likely to be. Most environmental PAH mixtures include trace quantities of the vast majority of the sixteen unsubstituted PAHs described earlier on in this chapter.

Kinetically-favoured, combustion-derived PAHs are found in the environment as a result of natural sources such as production during forest fires, or in association with volcanic eruptions. Many human activities involving combustions and emissions, however, contribute to a greater overall concentration in the global environment of benzo[a]pyrene

and other suspected carcinogenic PAHs relative to natural sources. Automobile and truck transport exhausts and coal-fired power generation are two major sources of combustion-derived PAHs to the environment.

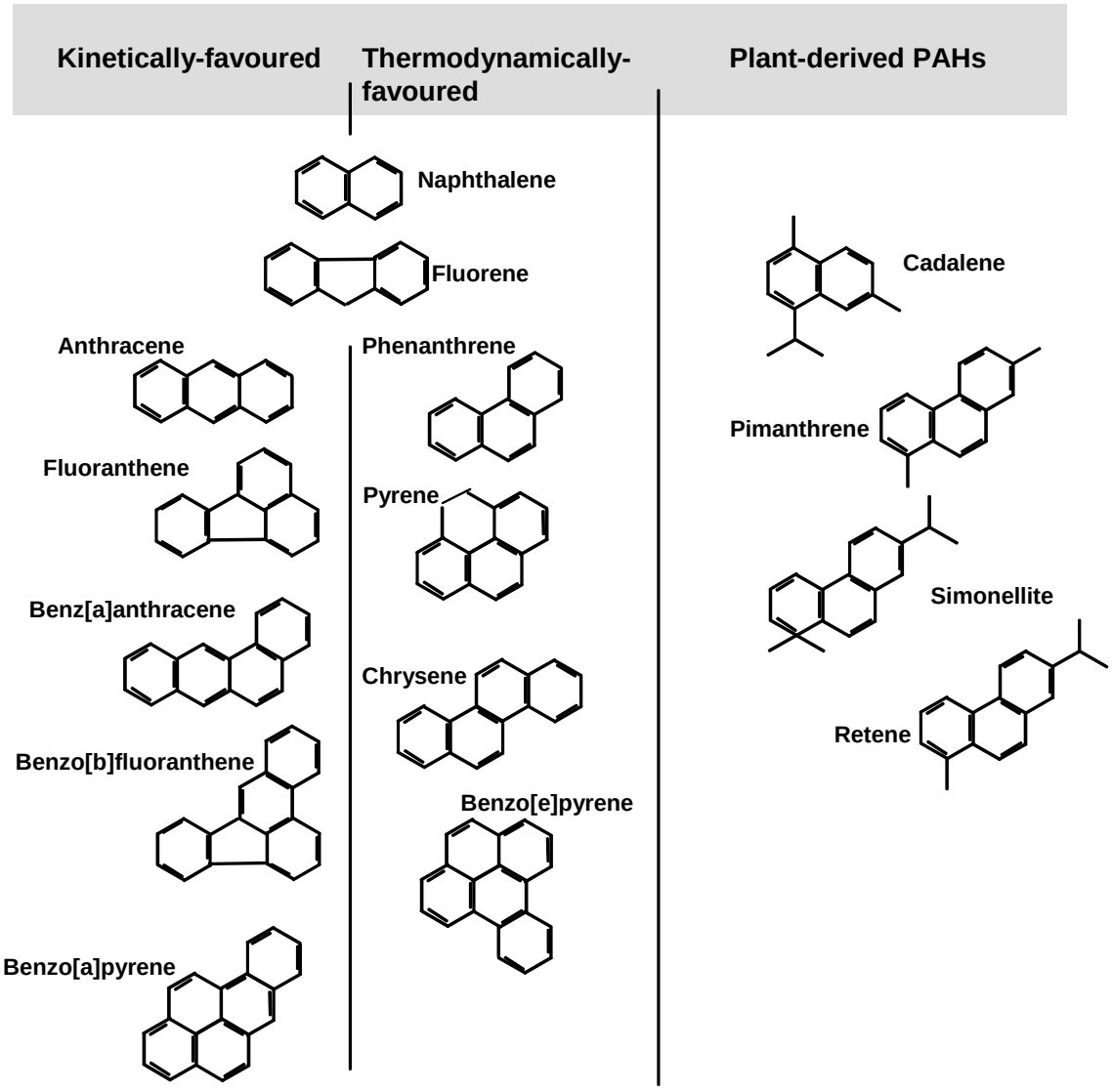


Figure 2-6: **Structure of Several Petrogenic, Pyrogenic, and Plant-Derived PAHs.**

Crude oil and refined product spills are the major overall source of thermodynamically-favoured (petrogenic) PAHs to soils and sediments, especially in urbanized and industrialized areas of Canada. Naphthalene, phenanthrene, and pyrene are particularly common PAHs in soil contaminant mixtures arising from petroleum-hydrocarbon-based releases.

Some PAH contaminant sources exhibit co-dominance of both pyrogenic and petrogenic types owing to the complex processes involved in the PAH source type. Crankcase and

other lubricating oils used in internal combustion engines, for example, contain a variety of petrogenic PAHs. The PAH composition changes, however, as the oil is used, since various combustion-derived PAHs accumulate with increased use. Coal tar residues from former manufactured gas plant (MGP) sites tend to have a highly complex and highly variable PAH composition, since the coal tar was produced by the heating of PAH-containing coal or coke to produce coal gas as a heating and light source prior to the wide-spread availability of natural gas in the mid- to late-twentieth century. Different processes at different MGP sites undoubtedly resulted in differences in the PAHs produced as a result. In addition, heating oil or diesel was added to the coal at some of the MGP sites but not others to increase the BTU content of the resulting coal gas. Therefore any MGP site may exhibit soils contaminated with PAHs derived from the coal and/or coke stock-piled and used at the site; coal tar produced in large quantities as a byproduct; heating oil and diesel used to augment coal gas production but released through chronic spillage prior to use; and complex combinations of the three.

A small number of both unsubstituted and alkylated PAHs are known natural products of plant and microbial compounds. The C4-phenanthrene *retene* (1-methyl-7-isopropal phenanthrene) may be derived from di- and tri-terpenoid precursors produced by plants (Bouloubassi and Saliot, 1993). Similarly, *pimanthrene* (1,7-dimethyl phenanthrene), *cadalene* (4-isopropyl-1,6-dimethylnaphthalene) and *simonellite* (1,1-dimethyl-1,2,3,4-tetrahydro-7-isopropylphenanthrene) may be produced from naturally produced plant terpenoids (Yunker and Macdonald, 1995).

There is also limited evidence that perylene may be formed through natural, biologically-mediated, petrogenic, or combustion-type processes.

Some individual alkyl-PAH isomers have been identified as markers for specific anthropogenic sources: acenaphthene, acenaphythlene, 4,5-methylene phenanthrene and others.

2.5.1 PAH Source Signatures

Gabos *et al., (*2001) examined PAH signatures, along with dioxins/furans and PCBs, derived from forest fires in northern Alberta, in river sediment samples. The parent (non-alkylated) PAHs were more abundant at the un-burned reference site than the burned sites, and the river sediment from the burned areas exhibited a predominance of alkyl-PAH over parent PAHs. The major conclusion derived from the study was that forest fires appear to contribute predominantly naturally derived PAHs including retene (Figure 2-6), rather than pyrogenic PAHs such as unsubstituted fluoranthenes/pyrenes, which are major components of anthropogenic combustion sources and long-range transport PAHs.

Figures 2-7 to 2-9 provide histograms of the relative distributions of alkylated and unsubstituted PAHs in various source materials and in some environmental samples affected by primarily anthropogenic inputs. The presence of alkylated PAH, especially those with two to four rings (e.g., alkyl-naphthalenes, alkyl-phenanthrenes) is highly indicative of a petroleum-based source (Law and Biscaya, 1994). In contrast, production

or release of PAHs through combustion processes usually favours the release of unsubstituted or lesser-alkylated forms of PAH over more highly alkylated forms, and the proportion of alkylated to unsubstituted PAHs varies as a function of combustion temperature (Aboul-Kassim and Simoneit, 1995). Coal and wood smoke, for example, contain a phenanthrene mixture with unsubstituted phenanthrene constituting the highest concentration, followed by an exponential decline in C1-homologues (phenanthrenes with one methyl group attached), then C2-homologues (two attached methyl group or two alkyl carbons), C3- and finally C4-homologues (Aboul-Kassim and Simoneit, 1995). Vehicular emissions peak at the C1-phenanthrene.

Diesel, in particular, contains high concentrations of 1- and 2-methylnaphthalene relative to unsubstituted naphthalene (Lee *et al.*, 1992: Table 2-5).

Table 2-5: Concentration of Some Naphthalenes in Diesel, and Chemical Properties

Compound	Aqueous Solubility ($25^{o}C$)	Log K_{ow}	Log K_{dw} (diesel-water partition coefficient - avg.)	conc. range ($mg \cdot L^{-1}$) in neat diesel
1-methyl-naphthalene	27	3.87	4.30	2,000-4,000
2-methyl-naphthalene	26	4.00	4.42	3,500-9,000
naphthalene	32	3.35	3.68	350-1,500

Coal tar tends to exhibit a strong dominance of unsubstituted PAH over alkylated forms, for both the naphthalene and anthracene + phenanthrene series, with a progressive decline in concentration associated with an increase in the extent of alkylation. Crude oil, on the other hand contains markedly more of the C2-homologue than either more- or less-highly alkylated forms.

Yunker and Macdonald (1995) examined alkyl-PAH homologue distributions in suspended and deposited sediments from the Mackenzie River shelf, Beaufort Sea (Figure 2-8). The relative contributions in the samples of C0 to C5 homologues for different PAH were as follows: the phenanthrene+anthracene series (3-ringed PAHs), and fluoranthene+pyrene series (4-ringed PAHs) exhibited a maximum average concentration for the C1-homologues; the naphthalenes exhibiting the highest concentrations were the C1- to C3- homologues. The concentrations and pattern of PAHs and other hydrocarbons in the Mackenzie River samples was attributed to petrogenic sources, primarily natural releases from hydrocarbon deposits farther up the river (e.g., the Norman Wells oil field).

Table 2-6 summarizes some of the studies wherein PAHs have been used as organic geochemistry markers to elucidate PAH sources and environmental fate.

Overall, sites with possible anthropogenic PAH contamination of surface and subsurface soils, groundwater, surface water, and contiguous lands and water include those where -

- petroleum hydrocarbon extraction, refinement, use and/or disposal has taken place;
- there has been potential for the enhanced deposition and/or post-depositional geochemical focusing of combustion-derived PAHs (e.g., soils and sediments receiving large amounts of stormwater runoff);
- coal tar and creosote were produced or used in materials treatment or preservation; and
- various limited other uses and releases occurred.

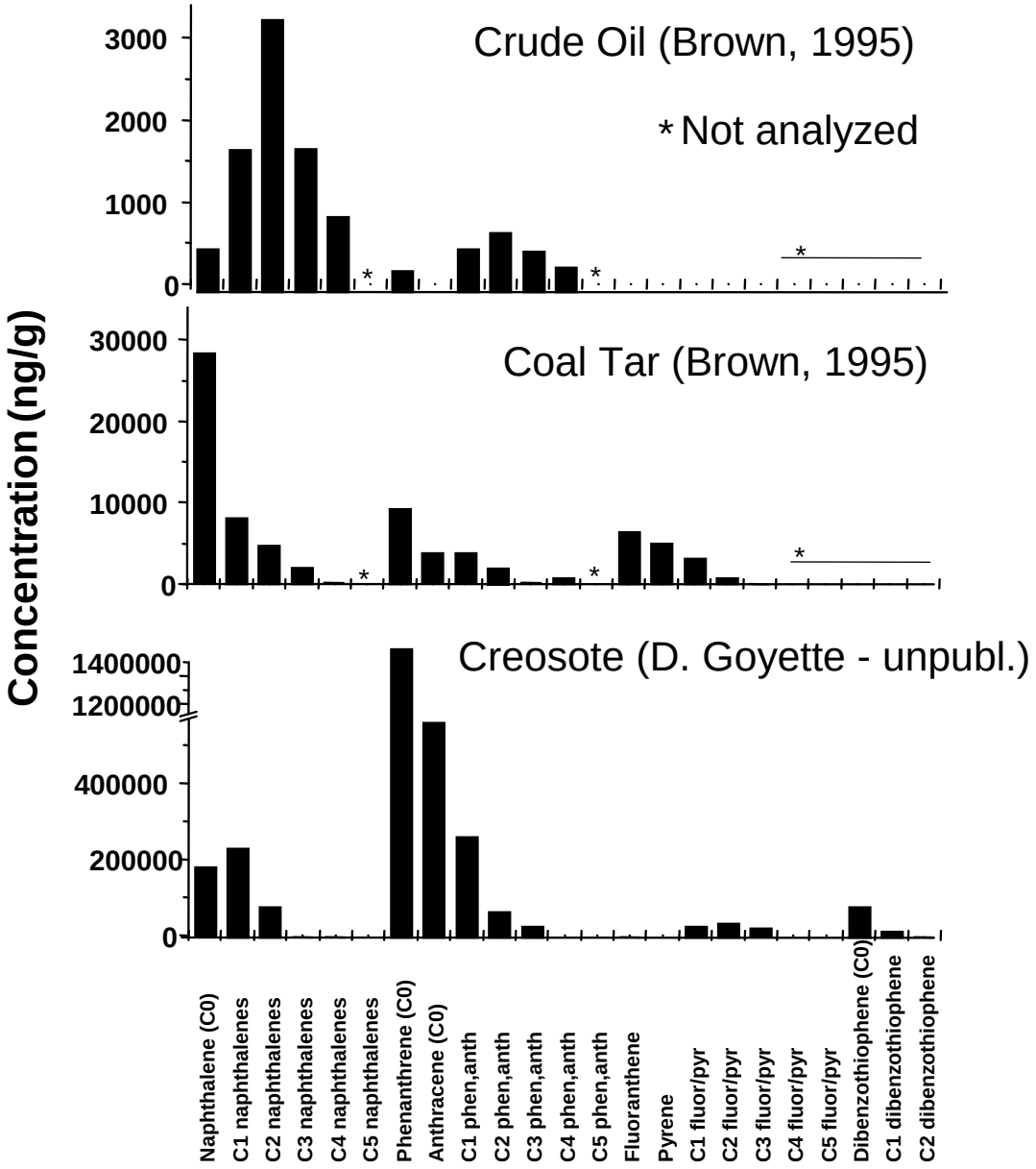


Figure 2-7: Representative Alkyl-PAH Source Signatures from Petroleum Sources.

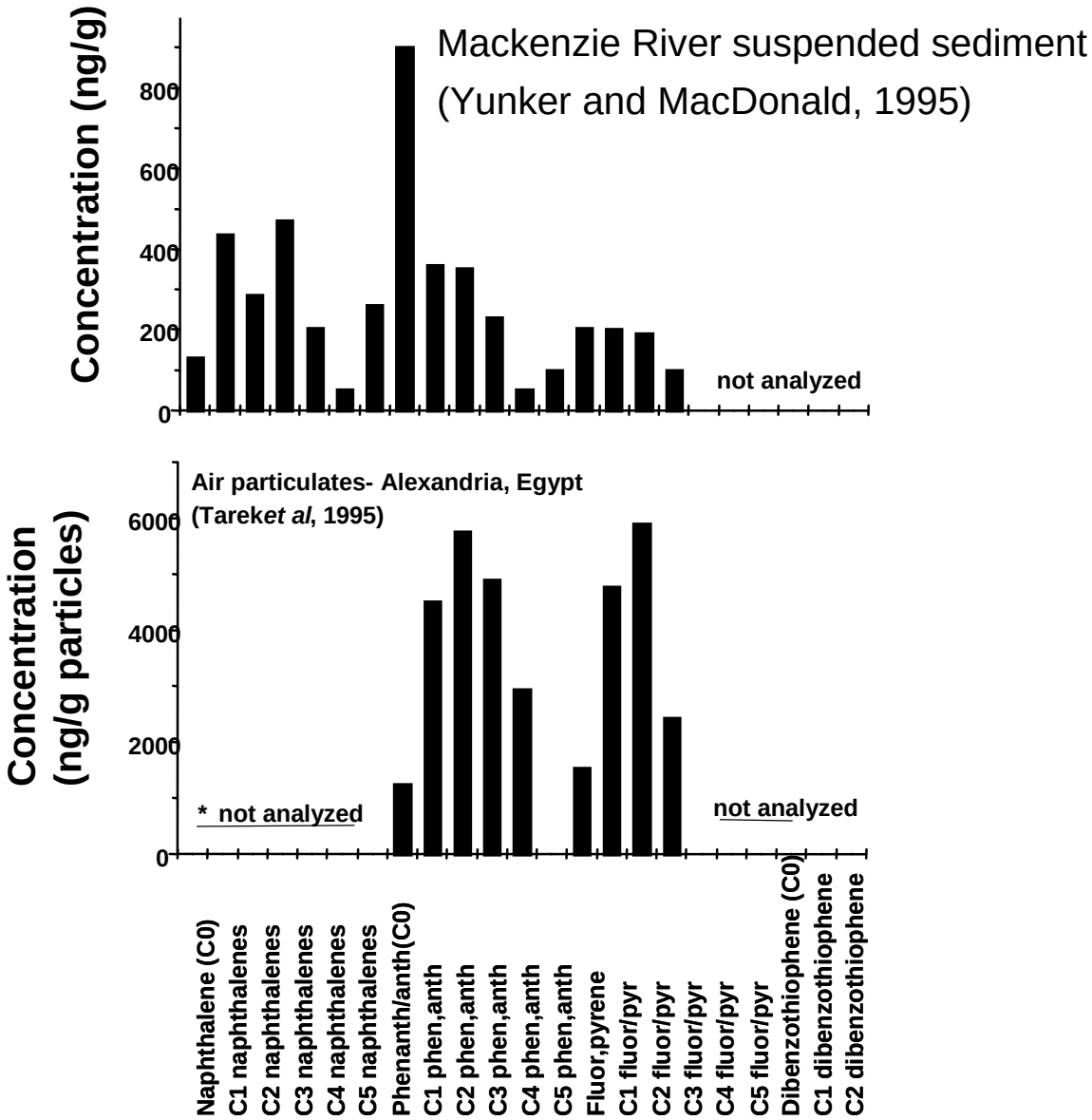


Figure 2-8: Some Representative Alkyl-PAH Source Signatures

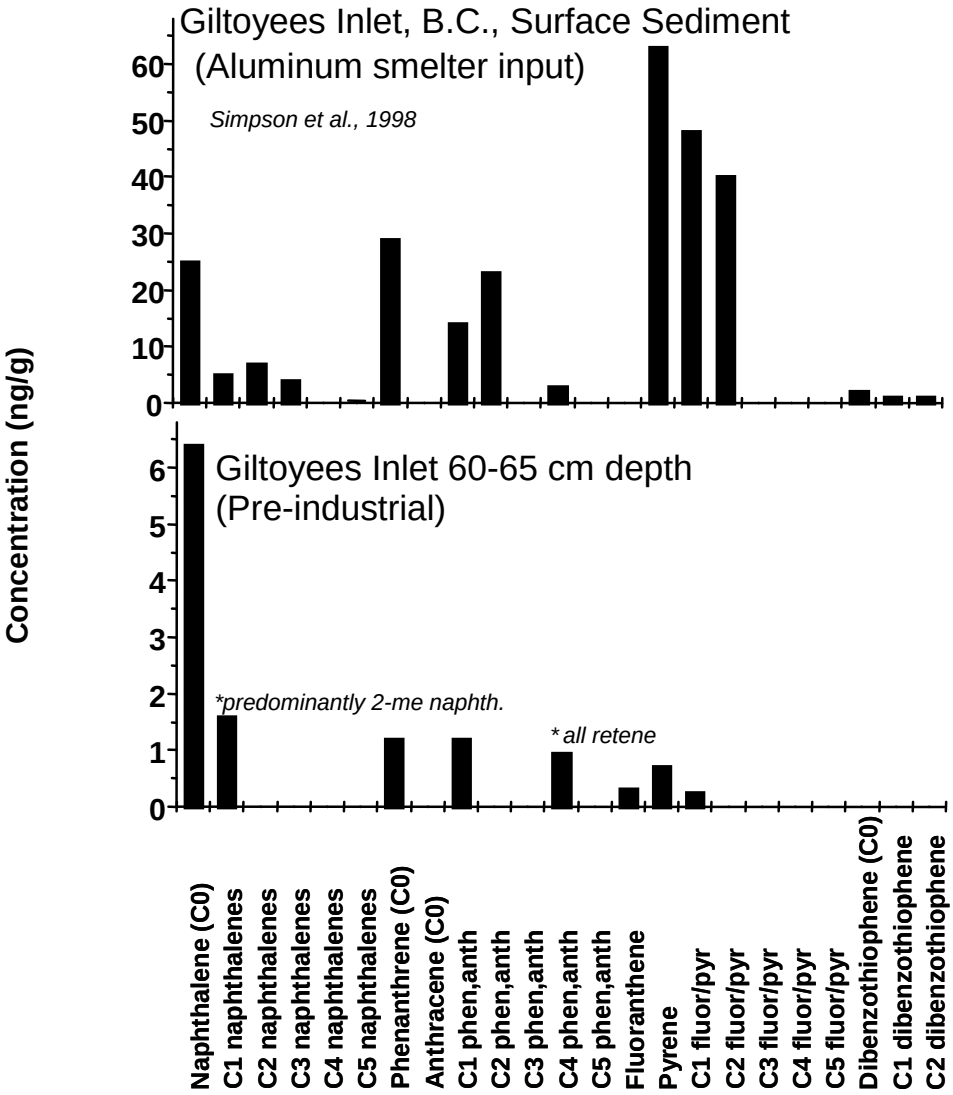


Figure 2-9: Additional Examples of Published Information on Environmental PAH Signatures

Table 2-6: Examples of Organic Geochemistry Studies which Include Alkyl-PAH Data

Title	Reference
Dissolved, particulate and sedimentary naturally derived polycyclic aromatic hydrocarbons in a coastal environment: geochemical significance.	Bouloubassi, I. and A. Saliot, 1993. Mar. Chem. 42: 127-143
Fluxes and transport of anthropogenic and natural polycyclic aromatic hydrocarbons in the western Mediterranean Sea.	Lipiatou, E. and A. Saliot, 1991. Mar. Chem., 32: 51-71.
Organic geochemistry of sediments from the continental margin off southern New England, U.S.A. - Part II. Lipids.	Venkatesan, M.I., E. Ruth, S. Steinberg and I.R. Kaplan, 1987. Mar. Chem., 21: 267-299.
Geochemistry and fluxes of hydrocarbons to the Beaufort Sea shelf: a multivariate comparison of fluvial inputs and coastal erosion of peat using PCA.	Yunker, M.B., R.W. Macdonald, B.R. Fowler, W.J.Cretney, S.R. Dallimore and F.A. McLaughlin, 1991. Geochim. Cosmochim. Acta, 55: 255-273.
Alkane, terpene and polycyclic aromatic hydrocarbon geochemistry of the Mackenzie River and Mackenzie shelf: Riverine contributions to the Beaufort Sea coastal sediment.	Yunker, M.B., R.W. MacDonald, W.J. Cretney, B.R. Fowler and F. A. McLaughlin, 1993. Geochim. Cosmochim. Acta, 57: 3041-3061.
Maturity determination of organic matter in coals using the methylphenanthrene index.	Kvalheim, O.M., A.A. Christy, N. Telnaes and A. Bjorseth, 1987. Geochim. Cosmochim. Acta, 51: 1883-1888.
Thermodynamic calculations on alkylated phenanthrenes: geochemical applications to maturity and origin of hydrocarbons.	Budzinski, H., Ph. Garrigues, M. Radke, J. Connan and J.-L. Oudin, 1993. Org. Geochem., 20: 917-926.
Distribution of naphthalenes in crude oils from the Java Sea: source and maturation effects.	Radke, M., J. Rullkotter and S.P. Vriend, 1994. Geochem. Cosmochim. Acta, 58: 3675-3689.

2.6 Levels in the Canadian Environment

Naturally occurring sources of PAHs to the environment include forest fires, volcanic eruptions, diagenesis, and biosynthesis. Human activities, however, are considered to be a major source of PAH contamination (Neff, 1979; NRC, 1983; NRCC, 1983; Bjørseth and Ramdhal, 1985; Slooff *et al*., 1989).

The distribution and magnitude of certain atmospheric emissions of PAHs are related to human population density, which in turn influences the prevalence of residential heating, transportation, and waste incineration. Other emission types, however, depend on the availability of power (aluminum smelters) or on the presence of natural resources (open air fires and agricultural burning, sawmill residue incinerators).

The sources of PAHs that enter water and soil are varied and include –

- dispersion from creosoted materials (Wan, 1993);
- accidental oil spills;
- precipitation and atmospheric deposition;
- industrial processes (creosote, coal tar, asphalt, land-farming) (AMAI, 1986a; AMAI, 1986b; 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 $t \cdot yr^{-1}$ (LGL, 1993).

Spills of petroleum hydrocarbons result in 76 $t \cdot yr^{-1}$ of PAHs being released into the Canadian environment. About 88% of the total number of spills occurs on land and 12% on water (LGL, 1993).

2.6.1 Soil

Concentrations of up to 100 µg·kg^{-1} (or 0.1 mg·kg^{-1}, ppm) of individual PAHs have been detected in uncontaminated soil, although higher concentrations of pyrene, phenanthrene, chrysene, and benzo[a]pyrene have been found. These are, nonetheless, atypically high concentrations.

The concentrations in soil near industrial sources of PAH may be elevated by three orders of magnitude or more relative to background soil concentrations. Among the highest concentrations of PAHs found in soils were those near former coking plants (CCME, 1989). The Resources Development Research Centre (RDRC) identified 144 potential sites of former coking plants in Canada, 80 of which were confirmed sites (RDRC, 1987). Although the majority of these sites (70) were 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 benzo[a]pyrene, the only compound investigated in 1985, varied from the detection limit (unspecified) to 1,300 mg·kg^{-1} (d.w.). The site has now been restored and monitoring studies entailing more than 800 soil samples have shown that the benzo[a]pyrene concentration is less than 10 mg·kg^{-1} (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 mg·kg^{-1} before site restoration, with a median concentration of 18 mg·kg^{-1} (Tecsult, 1989).

Kieley *et al., (*1986) reported median and maximum concentrations of 965 and 16,000 mg·kg^{-1} (d.w.), respectively, 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 benzo[a]pyrene concentration in soil was reported to be 2.4 mg·kg^{-1} (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. Fluoranthene and pyrene exhibited median concentrations of 0.75 mg·kg^{-1} and 0.50 mg·kg^{-1}, respectively. Benzo[a]pyrene was not detected, however. In the vicinity of the plant, total concentrations of PAHs in soil were reported to be 9,810 mg·kg^{-1} (ETL, 1984).

According to the World Health Organization (WHO/IPCS, 1998):

> "Near industrial sources, individual PAH levels of up to 1 g/kg (1000 µg/g) soil have been found. The concentrations in soil from other sources, such as automobile exhaust, are in the range 2-5 mg/kg (2-5 µg/g). In unpolluted areas, the PAH levels were 5-100 µg/kg (0.005-0.1 µg/g) soil."

ATSDR (1995c) provided an estimate of the range of background PAH concentrations found in soils removed from direct PAH-containing source releases, which is provided

herein as Table 2-7. In Canada, the Ontario Ministry of the Environment (OMOE, 1999) provided estimates of background soil PAH concentrations, as provided in Table 2-8.

Table 2-7: Estimates of Background Soil Concentrations of PAHs

ATSDR (1995c)

PAH	Concentrations ($mg \cdot kg^{-1}$)	
	Rural	**Agricultural**
Acenaphthene	0.0017	0.006
Acenaphthylene		0.005
Fluorene		0.01
Anthracene		0.011 to .013
Phenanthrene	0.03	0.05 to 0.14
Fluoranthene	0.0003 to 0.04	0.12 to 0.21
Pyrene	0.001 to 0.02	0.10 to 0.15
Benz[a]anthracene	0.005 to 0.02	0.056 to 0.11
Chrysene	0.04	0.08 to 0.12
Benzo[b]fluoranthene	0.02 to 0.02	0.06 to 0.22
Benzo[k]fluoranthene	0.01 to 0.11	0.06 to 0.25
Benzo[a]pyrene	0.002 to 1.3	0.005 to 0.9
Benzo[e]pyrene		0.05 to 0.13
Benzo[g,h,i]perylene	0.01 to 0.07	0.07
Indeno[1,2,3-c,d]pyrene	0.010 to 0.015	0.06 to 0.1

Table 2-8: Ontario Typical Range (OTR) Estimates-Background Soil PAH Concentrations (top 5 cm)

PAH	OTR_{98} ($mg \cdot kg^{-1}$) (Lower Conf. Limit – Upper Confidence Limit)	
	Old Urban Parkland	**Rural Parkland**
Naphthalene	0.075 (0.069-0.097)	0.006 (*provisional)*
1-methyl naphthalene		0.006 (*provisional)*
2-methyl naphthalene		0.006 (*provisional)*
Acenaphthene	0.032 (0.021-0.180)	0.006 (*provisional)*
Acenaphthylene	0.047 (0.035-0.061)	0.030 (0.022-0.050)
Fluorene	0.039 (0.030-0.18)	0.0094 (*provisional*)
Phenanthrene	0.31 (0.24-0.36)	0.092 (0.043-1.2)
Anthracene	0.058 (0.055-0.085)	0.006 (*provisional)*
Pyrene	0.49 (0.41-0.66)	0.11 (0.054-1.4)
Fluoranthene	0.56 (0.46-0.60)	0.14 (0.080-2.0)
Benz[a]anthracene	0.36 (0.26-0.39)	0.049 (0.030-0.39)
Chrysene	0.35 (0.29-0.46)	0.099 (0.055-0.48)
Benzo[b]fluoranthene	0.30 (0.25-0.42)	0.15 (0.11–0.31)
Benzo[k]fluoranthene	0.26 (0.20-0.34)	0.006 (*provisional)*

Benzo[a]pyrene	0.30 (0.22-0.37)	0.039 (0.029-0.10)
Dibenz[a,h]anthracene	0.077 (0.065-0.15)	0.052 (0.007-0.11)
Indeno[1,2,3-c,d]pyrene	0.23 (0.17-0.33)	0.054 (0.033-0.11)
Benzo[g,h,i]perylene	0.28 (0.16-0.68)	0.081 (0.049-0.14)

* OTR_{98} is the 97.5% upper estimate of background soil concentrations based on non-parametric techniques.

In general, rural parkland soils tended to exhibit a PAH concentration range that was about 4-fold to 10-fold lower than parkland soils in older urban environments in Ontario.

Fraser and Small (2001) compared PAHs in surface soil samples collected at distances of 5, 10 or 20 km from the Sydney Tar Ponds with soils in communities adjacent to the Tar Ponds. The PAH concentrations in background soils are provided in Table 2-9. The summary data is influenced primarily by the analytical detection limits used (0.05 $mg \cdot kg^{-1}$) and the fact that the majority of the 90 samples collected did not contain PAHs at concentrations above the detection limits.

Table 2-9: Background PAH Concentrations ($mg \cdot kg^{-1}$) in Soils 5-20 km from the Sydney Tar Ponds (from Fraser and Small, 2001).

PAH	N	Min	Max	Geomean*	St. Dev.
Naphthalene	90	<0.05	0.66	0.053	
1-methyl naphthalene	90	<0.05	0.23	0.053	0.03
2-methyl naphthalene	90	<0.05	0.28	0.053	0.04
Acenaphthene	90	<0.05	0.63	0.051	0.06
Acenaphthylene	90	<0.05	<0.05		
Fluorene	90	<0.05	0.86	0.052	0.08
Phenanthrene	90	<0.05	6.6	0.062	0.70
Anthracene	90	<0.05	1.4	0.053	0.14
Fluoranthene	90	<0.05	4.3	0.066	0.48
Pyrene	90	<0.05	3.5	0.062	0.38
Benz[a]anthracene	90	<0.05	4.5	0.058	0.47
Chrysene	90	<0.05	1.6	0.060	0.19
Benzo[b]fluoranthene	90	<0.05	0.64	0.055	0.07
Benzo[k]fluoranthene	90	<0.05	0.64	0.059	0.07
Benzo[a]pyrene	89	<0.05	0.69	0.055	0.07
Indeno[1,2,3-c,d]pyrene	90	<0.05	0.81	0.054	0.09
Dibenz[a,h]anthracene	90	<0.05	0.22	0.051	0.02
Benzo[g,h,i]perylene	90	<0.05	0.67	0.054	0.08

* values below the detection limit were assumed to be at the detection limit for the purpose of calculating geometric mean concentrations.

According to Environment Canada (1999) –

> "In general, soils containing <0.1 mg/kg B[a]P are considered uncontaminated, soils containing 0.1 – 1.0 mg/kg B[a]P are considered slightly contaminated and soils containing 1 – 10 mg/kg B[a]P are considered to be significantly contaminated.

The Electric Power Research Institute (EPRI) has recently sponsored two major studies on PAHs in background soils; one in Illinois and one in northern and western New York State. A country-wide study is planned by EPRI beginning in 2004. The Illinois Environmental Protection Agency reviewed the EPRI background study and has proposed that the data may be relevant for amending risk-based soil remediation objectives in urban areas[3]. The upper 95th percentile concentrations for urbanized areas in Illinois are tabulated below:

Table 2-10: Background PAH Concentrations (mg·kg^{-1}) in Illinois Urban Soils – Upper 95%ile

PAH	Chicago	Metropolitan Statistical Area (pop. ≥ 50,000)	Areas Outside of Metropolitan Statistical Areas
Benz[a]anthracene	1.1	1.8	0.72
Chrysene	1.1	2.7	1.1
Benzo[b]fluoranthene	1.5	2.0	0.70
Benzo[k]fluoranthene	1.0	1.7	0.63
Benzo[a]pyrene	1.3	2.1	0.98
Indeno[1,2,3-c,d]pyrene	0.86	1.6	0.51
Dibenz[a,h]anthracene	0.20	0.42	0.15

Similarly, the Massachussetts Department of Environmental Protection has published "Anthropogenic Background Levels" for PAHs[4].

Wilcke and Amelung (2000) examined PAH and other persistent organic contaminant concentrations in soil samples (top 10 cm) with moderate clay content collected at a variety of latitudes in the native North American (Great Plains) prairie. Site locations range from central Saskatchewan to southern Texas. The results (Table 2-11) were similar to background PAH concentrations in Europe.

[3] EPRI Project Opportunity (July 2003). Nation-wide Study of Background PAH Levels (www.epri.com)
[4] http://www.state.ma.us/dep/bwsc/files/workgrps/numbers/pahback.htm

Table 2-11: Background PAH Concentrations ($mg \cdot kg^{-1}$) in Topsoils from Native North American Grassland (from Wilcke and Amelung, 2000)

PAH	Concentration Range
Naphthalene	0.0063 – 0.179
Acenaphthene	0.0005 – 0.0041
Acenaphthylene	0.0003 – 0.0038
Fluorene	0.0010 – 0.0088
Phenanthrene	0.031 – 0.087
Anthracene	0.0004 – 0.0021
Fluoranthene	0.0047 – 0.029
Pyrene	0.0033 – 0.011
Benz[a]anthracene	0.0006 – 0.0027
Chrysene	0.0014 – 0.0083
Benzo[b+j+k]fluoranthene	0.0014 – 0.011
Benzo[a]pyrene	0.0002 – 0.0027
Indeno[1,2,3-c,d]pyrene	0.0004 – 0.0040
Dibenz[a,h]anthracene	0.0001 – 0.0008
Benzo[g,h,i]perylene	0.0005 – 0.0034

The overall range of background PAH soil concentrations as discussed above (over three orders of magnitude from urbanized to remote environments) serves as an indication that background concentrations need to be assessed using data relevant for specific sites.

2.6.2 Sediments

In areas exposed to industrial pollutants, high concentrations of PAHs have been reported in sediment, which acts as a short-term to longer-term sink for PAH. 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 $mg \cdot kg^{-1}$ for total PAHs and less than 0.01 $mg \cdot kg^{-1}$ for benzo[a]pyrene; maximum concentrations were 0.86 and 0.05 $mg \cdot kg^{-1}$, respectively. For phenanthrene, the median was 0.07 $mg \cdot kg^{-1}$ and the maximum was 0.10 $mg \cdot kg^{-1}$. Pyrene was not detected in more than half of the samples; the maximum value was 0.05 $mg \cdot kg^{-1}$ (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 $mg \cdot kg^{-1}$; median and maximum benzo[a]pyrene concentrations: 0.03 and 0.04 $mg \cdot kg^{-1}$).

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

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 $mg \cdot kg^{-1}$, respectively, with a total PAH concentration (12 compounds) of 2,830 $mg \cdot kg^{-1}$ 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 $mg \cdot kg^{-1}$ near the effluent outfall of the steel complex; the 13 PAHs considered here accounted for 251 $mg \cdot kg^{-1}$ (Kieley *et al.*, 1988). The concentration of total PAHs was 0.0029 $mg \cdot kg^{-1}$ 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 $mg \cdot kg^{-1}$. Other reported median and maximum values were 0.72 and 60.0 $mg \cdot kg^{-1}$ for fluoranthene; 0.61 and 33.0 $mg \cdot kg^{-1}$ for pyrene; and 0.515 and 28 $mg \cdot kg^{-1}$ for benzo[a]pyrene.

In the sediments of Montreal Harbour, Quebec, the median and maximum concentrations were 5.7 and 66 $mg \cdot kg^{-1}$ for phenanthrene, respectively, and 2.8 and 39.0 $mg \cdot kg^{-1}$ for pyrene. The median concentration of benzo[a]pyrene was 1.2 $mg \cdot kg^{-1}$. The median concentration of total PAHs was 14.2 $mg \cdot kg^{-1}$ (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 $mg \cdot kg^{-1}$, with a range from 1.6 to 1470 $mg \cdot kg^{-1}$. The median concentrations for pyrene and fluoranthene were 17.3 and 20.1 $mg \cdot kg^{-1}$, respectively, and their maxima were 280 $mg \cdot kg^{-1}$ (pyrene) and 189 $mg \cdot kg^{-1}$ (fluoranthene). The median concentration of benzo[a]pyrene was 9.5 $mg \cdot kg^{-1}$ (Murphy *et al.*, 1993).

The median and maximum concentrations of total PAHs in the sediments of Kettle Creek, downstream from an old oil-gasification complex at Port Stanley, Ontario, were 28 $mg \cdot kg^{-1}$ and 499 $mg \cdot kg^{-1}$, respectively. For benzo[a]pyrene, median and maximum concentrations were 1.0 and 40.2 $mg \cdot kg^{-1}$ (Canviro, 1988).

In Vancouver Harbour, British Columbia, the median and maximum concentrations for total PAHs were 5.4 $mg \cdot kg^{-1}$ and 36.8 $mg \cdot kg^{-1}$ (Boyd *et al.*, 1989). Median and maximum concentrations were 0.74 $mg \cdot kg^{-1}$ and 5.55 $mg \cdot kg^{-1}$ for pyrene; 0.64 and 5.63 $mg \cdot kg^{-1}$ for fluoranthene; and 2.42 $mg \cdot kg^{-1}$ and 0.134 $mg \cdot kg^{-1}$ for benzo[a]pyrene. In Kitimat, B.C., the median for total PAHs was 31.2 $mg \cdot kg^{-1}$, ranging from 0.14 to 258 $mg \cdot kg^{-1}$ (Goyette, 1991; Simpson *et al.*, 1998). Median and maximum concentrations were 2.9 and 27.0 $mg \cdot kg^{-1}$ for fluoranthene, 2.2 and 23.1 $mg \cdot kg^{-1}$ for pyrene, and 4.35 and 39 $mg \cdot kg^{-1}$ for benzo[a]pyrene.

2.6.3 Groundwater

Few published studies are available concerning the presence of PAHs in groundwater in Canada; however, PAHs are routinely measured in groundwater samples from petroleum hydrocarbon, creosote and coal tar contaminated sites. Near a former refinery at Pincher Creek, Alberta, the concentrations of pyrene ranged from below the detection limit to 300 $\mu g \cdot L^{-1}$, with a median concentration of about 30 $\mu g \cdot L^{-1}$ (ETL, 1984). Concentrations of fluorene at this site ranged from below the detection limit to 230 $\mu g \cdot L^{-1}$; the median was 40 $\mu g \cdot L^{-1}$ (ETL, 1984). At Newcastle, New Brunswick, naphthalene was detected at a concentration as high as 2.8 $\mu g \cdot L^{-1}$ and benzo[a]pyrene as high as 0.32 $\mu g \cdot L^{-1}$ in groundwater near a wood preserving plant (WMS, 1989).

2.6.4 Surface Water

Concentrations of PAHs in water may include both dissolved PAHs and extractable PAHs adsorbed to suspended particles in the water column. This imposes challenges for sampling and subsequent quantification since PAHs tend to be predominantly sorbed to sediments, and since the amount of sediment sampled is often poorly replicable rendering the PAH results highly variable. There is no easy solution for this issue, since the separation of truly dissolved PAHs from particle-bound PAHs during or after sample collection includes the possibility for further changes in partitioning. The presence of PAHs at concentrations exceeding their theoretical solubility limits (Table 2-4) is an indication that a major portion of the overall PAH mass is associated with particulate and/or dissolved organic matter.

Truly dissolved PAH concentrations in water samples from lentic, lotic, and marine systems tend to be very low owing to the very limited solubility of PAHs and the very high K_{OW}s, especially for those with four to six rings.

Concentrations of PAHs in the Atlantic provinces were low, with a median concentration of B[a]P below the detection limit (0.0008 to 0.001 $\mu g \cdot L^{-1}$) and a maximum concentration of 0.009 $\mu g \cdot L^{-1}$ (Wong and Bailey, 1990). The highest PAH concentration in the St. Lawrence River was for phenanthrene, for which the median concentration was 0.018 $\mu g \cdot L^{-1}$ and the maximum value was 0.12 $\mu g \cdot L^{-1}$ (Envirodat, 1993). Concentrations of PAHs in the Niagara River ranged from 0.0021 to 0.064 $\mu g \cdot L^{-1}$, with a median of 0.013 $\mu g \cdot L^{-1}$ (Kuntz, 1990), while they ranged from below the detection limit (0.0004 $\mu g \cdot L^{-1}$) to 0.006 $\mu g \cdot L^{-1}$ 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 0.011 to 0.55 $\mu g \cdot L^{-1}$) than those recorded in June 1986 (0.054 to 1.82 $\mu g \cdot L^{-1}$) at the same sites, indicating that localized run-off following ice break-up 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 $\mu g \cdot L^{-1}$ for naphthalene, 1642 $\mu g \cdot L^{-1}$ for phenanthrene (Wan, 1991), 2,035 $\mu g \cdot L^{-1}$ for fluoranthene, and 5,356 $\mu g \cdot L^{-1}$ for total PAHs (Wan 1993).

In the Atlantic provinces, concentrations of benzo[a]pyrene in marine waters ranged from below the detection limit (0.0008 to 0.001 $\mu g \cdot L^{-1}$) to 0.016 $\mu g \cdot L^{-1}$, while concentrations of fluoranthene ranged up to 0.11 $\mu g \cdot L^{-1}$. The detection frequency was low for benzo[a]pyrene (2% of samples analyzed), but higher for fluorene (25%) (Wong and Bailey, 1990). The highest concentration of total PAHs measured in harbours in Nova Scotia was 0.88 $\mu g \cdot L^{-1}$ (O'Neill and Kieley, 1992).

The metallurgical sector (metals and coking plants) released about 3.9 $t \cdot yr^{-1}$ 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 $t \cdot yr^{-1}$ of PAHs into the aquatic environment, based on estimated emissions in 1989 (Lane *et al.,* 1990).

2.6.5 Drinking Water

In a survey of seven water treatment plants in the area of Niagara Falls, benzo[a]pyrene was not detected (detection limit 1.0 $\mu g \cdot L^{-1}$) (OMOE, 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 0.001 $\mu g \cdot L^{-1}$), fluoranthene (0.02 and 0.03 $\mu g \cdot L^{-1}$), and pyrene (twice at 0.04 $\mu g \cdot L^{-1}$). The range of compounds examined was not specified (OMOE, 1989). In Quebec, the mean concentrations of PAHs in treated water determined recently ranged from <0.005 to 0.62 $\mu g \cdot L^{-1}$ for fluoranthene, <0.005 to 0.040 $\mu g \cdot L^{-1}$ for benzo[k]fluoranthene, to 0.040 $\mu g \cdot L^{-1}$ for benzo[b]fluoranthene, and were <0.005 $ng \cdot L^{-1}$ for benzo[a]pyrene, indeno[1,2,3-c,d]pyrene and benzo[g,h,i]perylene (Ayotte and Larue, 1990).

In a survey conducted in the Atlantic Region from 1985 to 1988 (Environment Canada, 1989a-d), the concentrations of fluoranthene, benzo[a]pyrene, benzo[b]- and benzo[k]fluoranthene, indeno[1,2,3-c,d]pyrene and benzo[g,h,i]perylene were determined (detection limits of 0.001, 0.001, 0.001, 0.001, 0.005, 0.005 $\mu g \cdot L^{-1}$, 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 $\mu g \cdot L^{-1}$ was reported. At this particular site, B[b]F (0.001 to 0.005 $\mu g \cdot L^{-1}$), B[k]F (0.001 to 0.003 $\mu g \cdot L^{-1}$), and B[a]P (0.001 to 0.003 $\mu g \cdot L^{-1}$) were also detected. In Prince Edward Island, fluoranthene was detected at low levels (range, 0.001 to 0.012 $\mu g \cdot L^{-1}$) 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 $\mu g \cdot L^{-1}$), B[a]P (0.001 to 0.003 $\mu g \cdot L^{-1}$), and B[g,h,i]P (0.005 to 0.021 $\mu g \cdot L^{-1}$) were also detected. In Nova Scotia, only fluoranthene was detected in raw sources, at levels ranging from the detection limit to 0.008 $\mu g \cdot L^{-1}$. In New Brunswick, several samples contained fluoranthene at levels ranging from 0.001 to 0.005 $\mu g \cdot L^{-1}$. Concentrations of B[b]F, B[k]F, and B[a]P ranged up to 0.002, 0.001, and 0.003 $\mu g \cdot L^{-1}$, respectively, in samples collected at a well in Fredericton.

2.6.6 Ambient (Outdoor) Air

Forest fires may represent 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). Other important sources of PAHs to ambient air include emissions from: residential wood heating (474 $t \cdot yr^{-1}$); agricultural burning and open-air fires (358 $t \cdot yr^{-1}$); the incineration of wood residues by saw mills in tepee burners (249 $t \cdot yr^{-1}$); and transportation (201 $t \cdot yr^{-1}$).

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 $ng \cdot m^{-3}$ (mean of 10.0 $ng \cdot m^{-3}$), with a maximum of 40.4 $ng \cdot m^{-3}$ (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 $ng \cdot m^{-3}$ (mean of 1,687 $ng \cdot m^{-3}$) at Jonquière and 435 $ng \cdot m^{-3}$ (mean of 1519 $ng \cdot m^{-3}$) at Shawinigan, with maxima of 10,400 and 16,390 $ng \cdot m^{-3}$, respectively. In Jonquière, the highest concentrations of individual PAHs were recorded for phenanthrene and fluoranthene. The maximum concentrations for B[a]P were 305 $ng \cdot m^{-3}$ in Jonquière and 460 $ng \cdot m^{-3}$ 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 $ng \cdot m^{-3}$ in winter (mean of 507 $ng \cdot m^{-3}$), with a maximum of 1,000 $ng \cdot m^{-3}$. Phenanthrene, fluoranthene, and fluorene were present at the higher PAH 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 $ng \cdot m^{-3}$ (mean of 69 $ng \cdot m^{-3}$) and the maximum measured was 289 $ng \cdot m^{-3}$ (Dann, 1991). In Ontario, Canada, up to 140 $ng \cdot m^{-3}$ benzo[k]fluoranthene, 110 $ng \cdot m^{-3}$ perylene, 110 $ng \cdot m^{-3}$ benzo*[a]*pyrene, 90 $ng \cdot m^{-3}$ benzo[g,h,i]perylene, and 43 $ng \cdot m^{-3}$ fluoranthene were found near a steel mill (Potvin *et al.,* 1980).

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 $t \cdot yr^{-1}$ 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).

2.6.7 Indoor Air and House Dust

PAHs can be found in indoor air as a result of residential heating and tobacco smoke at average concentrations of 1-100 $ng \cdot m^{-3}$, with a maximum of 2,300 $ng \cdot m^{-3}$. Environmental tobacco smoke, unvented radiant and convective kerosene space heaters, and gas cooking and heating appliances may be important sources of PAHs in indoor air (Chuang *et al.*, 1991). Chrysene/triphenylene, pyrene, and fluoranthene were dominant among the PAHs found in fine particle emissions from natural gas home appliances (Rogge *et al.*, 1993).

A large volume of literature exists on the effects of tobacco smoke on human lungs (see IARC, 1986). On the basis of more than 100 prospective and retrospective studies in more than 15 countries, cigarette smoke has been shown to be by far the most important single factor contributing to the development of lung cancer. Cigarette mainstream smoke contains a wide variety of PAHs with reported concentrations of benzo[a]pyrene ranging from approximately 5-80 ng/cigarette; sidestream smoke concentrations are significantly higher with sidestream/mainstream concentration ratios for benzo[a]pyrene ranging from 2.5 to 20 (Hoffmann and Hoffmann 1993; IARC 1983). Other studies have shown levels of 11 ng per cigarette benzo[a]pyrene in mainstream smoke and 103 ng per cigarette in sidestream smoke; the corresponding values were 6.8 and 7.6 ng per cigarette for benzo[e]pyrene, 20 and 497 ng per cigarette for chrysene and triphenylene, and 13 and 204 ng per cigarette for benz[a]anthracene (Grimmer *et al.*, 1987). In sidestream smoke, PAH with four or more rings were responsible for 83% of the total carcinogenic activity (Grimmer *et al.*, 1988).

2.6.8 Commercially Sold Food

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

Plants grown on PAH-contaminated soils, nonetheless, have only a limited ability to take in through the roots and translocate anthropogenic PAHs to the aboveground plant biomass – especially for higher molecular weight PAHs. One mode of plant contamination is via the deposition of PAH-containing fine particulates onto plant surfaces.

Terrestrial animals that are used for food by humans, including domestic stock, may be exposed to PAHs by wet and dry deposition of PAH-containing particulates onto soil and plant surfaces followed by ingestion. Incidental soil ingestion while burrowing, grazing, and preening is another potential source of PAH uptake into terrestrial animals, and might be important when these animals are foraging or otherwise inhabiting areas with PAH contaminated soils arising from direct input sources. Virtually all vertebrate animals, however, have the enzymatic capabilities to metabolically modify PAHs and then eliminate the metabolites (e.g., in urine). The tissue residues of PAHs in animals living on PAH-contaminated soils or in PAH-contaminated water, therefore, have occasionally been found to occur at concentrations that are higher than in equivalent samples from

background or reference locations; however, the tissue residue PAH concentrations tend to be much lower than would be expected based on magnitude of PAH contamination of the soil. Overall, PAHs tend to have a limited ability to bioaccumulate in most terrestrial mammals, and very limited if any ability to biomagnify (i.e., to increase in concentration at successively higher levels in the trophic pyramid). Again, this is attributed to the fact that PAHs can be readily metabolized.

Considerable research has been carried out on the use of PAH metabolites such as hydroxypyrene, measured in tissues, as biomarkers of PAH exposure. Few studies, however, have assessed the risks to consumer organisms including humans of the dietary exposure to PAH metabolites.

In cooked food, the method of cooking is generally the primary determinant of the PAH content (Santodonato *et al.,* 1981). A report prepared under contract for Agriculture Canada presented the results 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 0.0018 $mg \cdot kg^{-1}$ in vegetables/fruits for B[b]F, not detected to 0.0003 $mg \cdot kg^{-1}$ in meat/poultry/fish for B[k]F, and not detected to 0.0011 $mg \cdot kg^{-1}$ 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).

In other studies of meat, poultry, and fish in Canada, benzo[k]fluoranthene was detected at concentrations up to 0.0003 $mg \cdot kg^{-1}$ and benzo[a]pyrene up to 0.0011 $mg \cdot kg^{-1}$ (Environment Canada, 1994). The concentrations of fluoranthene, pyrene, benz[a]anthracene, chrysene, benzo[b]fluoranthene, benzo[k]fluoranthene, benzo[a]pyrene, benzo[e]pyrene, perylene, benzo[g,h,i]perylene, indeno[1,2,3-cd]pyrene, and dibenz[a,h]anthracene were measured in milk, milk powder, and other dairy products in Canada (Lawrence & Weber, 1984), the Netherlands (de Vos *et al.,* 1990), and the United Kingdom (Dennis *et al.,* 1983, 1991). The concentrations ranged from < 0.00001 $mg \cdot kg^{-1}$ for benzo[k]fluoranthene and dibenz[a,h]anthracene to 0.0027 $mg \cdot kg^{-1}$ for pyrene. Canadian infant formula was found to contain 0.008 $mg \cdot kg^{-1}$ fluoranthene, 0.0048 $mg \cdot kg^{-1}$ pyrene, 0.0017 $mg \cdot kg^{-1}$ benz[a]anthracene, 0.0007 $mg \cdot kg^{-1}$ benzo[b]fluoranthene, 0.0012 $mg \cdot kg^{-1}$ benzo[a]pyrene, 0.0006 $mg \cdot kg^{-1}$ perylene, 0.0003 $mg \cdot kg^{-1}$ anthracene, and 0.0012 $mg \cdot kg^{-1}$ indeno[1,2,3-cd]pyrene (Lawrence & Weber, 1984). Fluoranthene, but no other PAH, was identified in unspecified fruits and vegetables in Canada at levels of not detected to 0.0018 $mg \cdot kg^{-1}$ (Environment Canada, 1994).

The amount of PAH formed during roasting, baking, and frying depends markedly on the conditions (Lijinsky & Shubik, 1964). In an investigation of the effect of the method of cooking meat, including broiling (grilling) on electric or gas heat, charcoal broiling, and

broiling over charcoal in a no-drip pan, it was shown that the formation of PAH can be minimized by avoiding contact of the food with flames, cooking meat at lower temperatures for a longer time, and using meat with minimal fat (Lijinsky & Ross, 1967). The most likely source of PAH is melted fat that drips onto the heat and is pyrolysed (Lijinsky & Shubik, 1965). The exact chemical mechanism for the formation of PAH is unknown. In one study, the highest concentration of benzo[a]pyrene (0.130 $mg \cdot kg^{-1}$) in cooked meat was found in fatty beef, and the concentration appeared to be proportional to the fat content (Doremire *et al.,* 1979). Levels of about 0.05 $mg \cdot kg^{-1}$ were detected in a charcoal-grilled T-bone steak (Lijinsky & Ross, 1967), in heavily smoked ham (Toth & Blaas, 1972), and in various other cooked meats (Potthast, 1980). Usually, benzo[a]pyrene levels up to 0.0005 $mg \cdot kg^{-1}$ have been found (Prinsen & Kennedy, 1977).

2.6.9 Human Tissues

Very little is known about the retention and turnover of PAHs in mammalian species. It can be deduced from the limited data available on hydrocarbon body burdens that PAHs themselves do not persist for long periods and must therefore turn over reasonably rapidly. The effects of chemical carcinogens are likely to be related to both the dose and the duration of exposure. The total intake of PAH over a 70-year lifespan is estimated to amount to the equivalent of 300 mg of benzo[a]pyrene (Lutz & Schlatter, 1992).

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.

Average benzo[a]pyrene levels (measured by ultraviolet spectroscopy) in tissues taken from the autopsy of normal people of a wide age range were 0.0032 $mg \cdot kg^{-1}$ dry tissue weight in liver, spleen, kidney, heart, and skeletal muscle and 0.002 $mg \cdot kg^{-1}$ in lung (Gräf, 1970; Gräf *et al.*, 1975). When cancer-free liver and fat from six individuals were assayed for nine hydrocarbons by co-chromatography with authentic standards, pyrene, anthracene, benzo[b]fluoranthene, benzo[g,h,i]perylene, benzo[k]fluoranthene, and benzo[a]pyrene were detected at average levels of 0.00038 $mg \cdot kg^{-1}$ wet weight in liver and 0.0011 $mg \cdot kg^{-1}$ wet weight in fat. Pyrene was the most abundant PAH present (Obana *et al.,* 1981).

3. EXISTING GUIDELINES AND REGULATORY APPROACHES, AND LIMITATIONS

Prior to this assessment, the only Canadian soil quality guidelines for PAHs were for naphthalene and benzo[a]pyrene. In the case of the Canadian soil quality guideline for benzo[a]pyrene, which was adopted in its current form in 1999, the existing toxicity data were deemed to be insufficient to calculate a soil protective value for any of the ecological receptor groups. There are a few more soil screening values for individual PAHs contained in the CCME 1991 soil criteria, but these were not risk-based numbers, and the roots of their derivation are unclear in some cases. Table 3-1 summarizes CCME guidelines for individual PAHs in soil, sediment, and water.

Additional PAH soil clean-up guidance for individual provinces and territories within Canada were not selected for inclusion herein unless they have been developed subsequent to, and were based on new information and analysis relative to, their CCME counterparts. Further, this summary is not intended as an exhaustive review. The information is intended simply to facilitate a brief comparison between the existing and newly developed Canadian PAH guidelines and soil PAH thresholds used in other jurisdictions.

Kalf *et al., (*1997) summarized the derivation of the Netherlands Environmental Quality Objectives for ten PAHs. Maximum permissible concentrations (MPCs) are provided in Table 3-2. The Netherlands has also developed “target intervention values” (not shown in the table). The Netherlands, like Canada, has a longer history than most countries (around a decade) of attempts to derive soil quality guidelines – not just for human health protection but also for ecological protection. Ideally, such soil threshold values are established based on relevant peer-reviewed or other experimental ecotoxicological data. This is rarely the case, however, and the Netherlands has attempted to address the knowledge gap in some instances by adopting quantitative structure activity approaches, or in some instances through the use of aquatic toxicity data to predict soil organismic responses, after making allowances for equilibrium partitioning and differences in the soil-water versus truly aqueous test environment. In the case of PAHs, the Netherlands soil MPCs were developed in light of very limited soil toxicity data in the literature. MPCs could be calculated from the available soil ecotoxicity data for anthracene, benz[a]anthracene, and benzo[a]pyrene. Soil MPCs for the remaining PAHs were derived using an equilibrium partitioning approach.

Table 3-1: Existing CCME environmental quality guidelines for PAHs as of 2003.

Substance	Drinking Water ($\mu g \cdot L^{-1}$)	Freshwater Life ($\mu g \cdot L^{-1}$)	Sediment ($\mu g \cdot kg^{-1}$)				Soil ($mg \cdot kg^{-1}$)			
			Freshwater		Marine		Agri.	Res./Park	Comm.	Indust.
			ISQG	PEL	ISQG	PEL				
Naphthalene		1.1	34.6	391	34.6	391	0.1	0.6	22	22
2-Methylnaphthalene			20.2	201	20.2	201				
Acenaphthylene			5.87	128	5.87	128				
Acenaphthene		5.8	6.71	88.9	6.71	88.9				
Fluorene		3.0	21.2	144	21.2	144				
Anthracene		0.012	46.9	245	46.9	245				
Phenanthrene		0.4	41.9	515	86.7	544				
Pyrene		0.025	53.0	875	153	1398				
Fluoranthene		0.04	111	2355	113	1494				
Benz[a]anthracene		0.018	31.7	385	74.8	693				
Chrysene			57.1	862	108	846				
Benzo[a]pyrene	0.01	0.015	31.9	782	88.8	763	0.1	0.7	0.7	0.7
Dibenz[a,h]anthracene			6.22	135	6.22	135				
Acridine		4.4								
Quinoline		3.4								

Table 3-2: Netherlands "Maximum Permissible Concentrations" (MPCs) for PAHs

(Kalf *et al.*, 1997)

PAH	MPC_{WATER} ($\mu g \cdot L^{-1}$)	MPC_{SOIL} ($mg \cdot kg^{-1}$)	$MPC_{SEDIMENT}$ ($mg \cdot kg^{-1}$)
Naphthalene	1.2	0.14	0.14
Anthracene	0.07	0.12	0.12
Phenanthrene	0.3	0.51	0.51
Fluoranthene	0.3	2.6	2.6
Benz[a]anthracene	0.01	0.25	0.36
Chrysene	0.34[A]	10.7	10.7
Benzo[a]pyrene	0.05	0.26	2.7
Benzo[g,h,i]perylene	0.033[A]	7.5	7.5
Benzo[k]fluoranthene	0.04	2.4	2.4

Notes: A – based on QSAR calculations and an assumed non-polar narcosis mode of toxicological action.

The Danish Environmental Protection Agency summarized the currently available criteria for soil, air and groundwater (Table 3-3) as part of their guidelines for the remediation of contaminated sites (http://www/mst.dk/udgiv/publications/2002/87-7972-280-6/html/kap06_eng.htm). Denmark has also established a PAH groundwater quality criterion of 0.2 $\mu g \cdot L^{-1}$.

Table 3-3: Danish PAH Soil Quality Criteria (2002) – Sensitive Land Use

Substance	Soil Quality Criteria (Human Health)	Soil Quality Criteria (Ecological Risk)	Background Level
PAH, total	1.5 $mg \cdot kg^{-1}$	1.0 $mg \cdot kg^{-1}$	Not provided
Benzo[a]pyrene	0.1 $mg \cdot kg^{-1}$	0.1 $mg \cdot kg^{-1}$	Not provided
Dibenz[a,h]anthracene	0.1 $mg \cdot kg^{-1}$		Not provided

Several jurisdictions outside of Canada have established PAH investigation and remediation guidelines for PAHs in consideration of human health risks, but in the absence of further or simultaneous consideration of various ecological receptors. New Zealand, for example, developed "Guidelines for Assessing Petroleum Hydrocarbon Contaminated Sites in New Zealand" in 2001 (Table 3-4). New Zealand was not comfortable developing similar guidelines for soils in consideration of ecological protection. New Zealand used a benzo[a]pyrene equivalence approach to develop soil acceptance criteria for carcinogenic PAHs, using the relative cancer potency scheme developed and modified by the USEPA (see Chapter 6).

Within the United States, individual regions of the USEPA have developed human health-based PAH soil screening levels to expedite contaminated sites assessment and

remediation, as have several states (e.g., Massachussetts, through the Department of Environmental Protection). USEPA has set a guideline for potentially carcinogenic PAHs in drinking water of 28 $ng \cdot L^{-1}$. The New Jersey Department of Environmental Protection and Energy proposed in 1992 and subsequently modified in 1999 “soil cleanup criteria” in consideration of human health protection. These are listed in Table 3-5.

Table 3-6 provides up-to-date human health risk-based “Preliminary Remediation Goals” developed by the USEPA is support of the investigation and remediation of Superfund sites in the United States.

There are at least two issues of interest that arise from the comparison of soil quality criteria across jurisdictions. First, even when evaluating values that are intended only for human health protection, and in spite of the fact that all numbers reviewed herein are considered to be recently derived risk-based thresholds for PAHs in soils, there are still substantial differences in the derived values, arising in large part from differences in methodologies. For benzo[a]pyrene in residential soils, for example, CCME (1999b) adopted a human health based soil quality guideline of 0.7 $mg \cdot kg^{-1}$. Soil criteria with a similar intent and using exposure estimates for more than one media derived in Denmark, New Zealand, New Jersey and by the USEPA (PRG) were 0.1, 0.27, 0.66, and 0.062 $mg \cdot kg^{-1}$ benzo[a]pyrene, respectively.

Second, the soil protective thresholds for different PAHs are likely to vary a great deal.

Table 3-4: New Zealand Human Health Based PAH Guidelines for Assessing and Managing Petroleum Hydrocarbon Contaminated Sites, 2001

(numbers in brackets likely correspond to concentrations associated with formation of a residual separate phase)

Land Use/ Soil Type/ Depth	Naphthalene ($mg \cdot kg^{-1}$)	Non-carcinogenic (Pyrene) ($mg \cdot kg^{-1}$)	Carcinogenic (B[a]P equiv.) ($mg \cdot kg^{-1}$)
Agricultural			
Sand			
0 to 1 m	7.2	(160)	0.027
1 to 4 m	70	NA	(25)
> 4 m	80	NA	NA
Sandy Silt			
0 to 1 m	7.2	(160)	0.027
1 to 4 m	83	NA	(25)
> 4 m	(130)	NA	NA
Silty Clay			
0 to 1 m	7.2	(160)	0.027
1 to 4 m	(330)	NA	(25)
> 4 m	(1,100)	NA	NA
Clay			
0 to 1 m	7.2	(160)	0.027
1 to 4 m	(360)	NA	(25)
> 4 m	(1,200)	NA	NA
Pumice			
0 to 1 m	7.2	(160)	0.027
1 to 4 m	140	NA	(25)
> 4 m	(220)	NA	NA
Peats & Highly Org.			
0 to 1 m	7.2	(160)	0.027
1 to 4 m	(2,700)	NA	(25)
> 4 m	(3,500)	NA	NA
Residential			
Sand			
0 to 1 m	58	(1,600)	0.27
1 to 4 m	70	NA	(25)
> 4 m	80	NA	NA
Sandy Silt			
0 to 1 m	63	(1,600)	0.27
1 to 4 m	83	NA	(25)
> 4 m	(130)	NA	NA
Silty Clay			
0 to 1 m	69	(1,600)	0.27
1 to 4 m	(330)	NA	(25)
> 4 m	(1,100)	NA	NA
Clay			
0 to 1 m	71	(1,600)	0.27
1 to 4 m	(360)	NA	(25)
> 4 m	(1,200)	NA	NA

Land Use/ Soil Type/ Depth	Naphthalene ($mg \cdot kg^{-1}$)	Non-carcinogenic (Pyrene) ($mg \cdot kg^{-1}$)	Carcinogenic (B[a]P equiv.) ($mg \cdot kg^{-1}$)
Pumice			
0 to 1 m	49	(1,600)	0.27
1 to 4 m	140	NA	(25)
> 4 m	(220)	NA	NA
Peats & Highly Org.			
0 to 1 m	72	(1,600)	0.27
1 to 4 m	(2,700)	NA	(25)
> 4 m	(3,500)	NA	NA
Commercial/Industrial			
Sand			
0 to 1 m	(190)	NA	(11)
1 to 4 m	(230)	NA	(25)
> 4 m	(260)	NA	NA
Sandy Silt			
0 to 1 m	(210)	NA	(11)
1 to 4 m	(270)	NA	(25)
> 4 m	(420)	NA	NA
Silty Clay			
0 to 1 m	(230)	NA	(11)
1 to 4 m	(1,100)	NA	(25)
> 4 m	(3,500)	NA	NA

Table 3-5: New Jersey Human Health Based Soil Cleanup Criteria, 1992 (revised 1999).

PAH	Residential Direct Contact Soil Criteria ($mg \cdot kg^{-1}$)	Non-Residential Direct Contact Soil Criteria ($mg \cdot kg^{-1}$)	Impact to Groundwater Criteria ($mg \cdot kg^{-1}$)
Naphthalene	230	4,200	100
Acenaphthene	3,400	10,000	100
Fluorene	2,300	10,000	100
Anthracene	10,000	10,000	100
Pyrene	1,700	10,000	100
Fluoranthene	2,300	10,000	100
Benz[a]anthracene	0.9	4	500
Chrysene	9	40	500
Benzo[b]fluoranthene	0.9	4	50
Benzo[k]fluoranthene	0.9	4	500
Benzo[a]pyrene	0.66	0.66	100
Dibenz[a,h]anthracene	0.66	0.66	100

Table 3-6: U.S. EPA Human Health Based Preliminary Remediation Goals for Soil ($mg \cdot kg^{-1}$)

	Acenaphthene	Anthracene	Benz[a]-anthracene	Benzo[b]-fluoranthene	Benzo[k]-fluoranthene	Benzo[a]pyrene	Chrysene	Dibenz[ah]-anthracene	Fluoranthene	Fluorene	Indeno[1,2,3-c,d]pyrene	Naphthalene	Pyrene
RESIDENTIAL SOIL													
Cancer: 1×10^{-6}													
soil-inhale			12,000	12,000	12,000	12,000	12,000	12,000			12,000		
soil-dermal			2.1	2.1	21	0.21	213	0.21			2.13		
soil-ingest			0.88	0.88	8.8	0.088	88	0.088			0.88		
Combined			**0.62**	**0.62**	**6.2**	**0.062**	**62**	**0.062**			**0.62**		
Non-cancer: Haz. Quot. = 1													
soil-inhale	17,000	330,000								23,000		58	180,000
soil-dermal									8,600				
soil-ingest	4,700	23,000							3,100	3,100		1,600	2,300
Combined	**3,700**	**22,000**							**2,300**	**2,700**		**56**	**2,300**
INDUSTRIAL SOIL													
Cancer: 1×10^{-6}													
soil-inhale			26,000	26,000	26,000	26,000	26,000	26,000			26,000		
soil-dermal			4.6	4.6	46	0.46	457	0.46			4.6		
soil-ingest			3.9	3.9	39	0.39	392	0.39			3.9		
Combined			**2.1**	**2.1**	**21**	**0.21**	**211**	**0.21**			**2.1**		
Non-cancer: Haz. Quot. = 1													
soil-inhale	56,000	1,100,000								74,000		190	580,000
soil-dermal									48,000				
soil-ingest	61,000	310,000							41,000	41,000		20,000	31,000
Combined	**29,000**	**240,000**							**22,000**	**26,000**		**190**	**29,000**

4. ENVIRONMENTAL FATE PROCESSES

PAHs are ubiquitous in the environment, partly because they are transported over long distances without significant degradation (Lunde, 1976; De Wiest, 1978; Bjorseth & Olufsen, 1983; McVeety & Hites, 1988), e.g., from the United Kingdom and the European continent to Norway and Sweden during winter (Bjorseth *et al.*, 1979). PAHs are sparingly soluble in water and therefore have an affinity for sediment, soil, and biota. When found in air and water, the PAH compounds are generally found adsorbed to particulate matter. Thus, although most PAHs are emitted to the atmosphere, sediments and soils are the major environmental sinks for these compounds. 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 be deposited into soil as a result of plant decay (Eisler, 1987). Removal of PAHs from the environment is normally associated with biodegradation or photodegradation processes. The rates of degradation vary and generally decrease with increasing numbers of aromatic rings.

4.1 Multimedia Partitioning

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 the surface of water bodies, soil, plant surfaces and impervious surfaces.

Polycyclic aromatic hydrocarbons released to soil will adsorb to particulate matter where they will be slowly degraded by microbial activity or transported by surface runoff. In aquatic systems, PAHs generally adsorb to suspended matter or sediments, where they tend to persist.

Contamination of groundwater by PAHs can occur as a result of leaching through soils, especially when mobile organic solvents accompany PAHs 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).

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).

Henry's law constant gives a rough estimate of the equilibrium distribution ratio of concentrations in air and water but cannot predict the rate at which chemicals are transported between water and air. The constants for PAHs are very low, ranging from 4.8×10^{-4} $atm \cdot m^3 \cdot mol^{-1}$ for naphthalene to 4.8×10^{-4} $atm \cdot m^3 \cdot mol^{-1}$ for dibenz[a,h]anthracene. The rates of removal and volatilization of PAHs are strongly dependent on environmental conditions such as the depth and flow rate of water and wind velocity. Although PAHs are released into the environment mainly in air, considerably higher concentrations are found in aqueous samples because of the low vapour pressure and Henry's law constants of PAH. The volatilization half-life for naphthalene from a water body was found experimentally to be 6.3 h, whereas the calculated value was 2.1 h (Klöpffer *et al.*, 1982). Calculations based on a measured air:water partition coefficient for river water 1 m deep with a water velocity of 0.5 m/s and a wind velocity of 1 m/s gave a volatilization half-life of 16 h for naphthalene (Southworth, 1979). The value calculated for evaporative loss of naphthalene from a 1-m water layer at 25°C was of the same order of magnitude (Mackay & Leinonen, 1975). Naphthalene was volatilized from soil at a rate of 30% after 48 h, with negligible loss of PAHs with three or more rings (Park *et al.*, 1990).

4.2 Transformations

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) (USEPA, 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 Rites, 1988) and the quantity of PAHs adsorbed to the particulate matter (Kamens *et al.*, 1988; Slooff *et al.*, 1989). PAHs 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 ($S0_2$).

4.3 Microbially-Mediated and Abiotic Degradation and Persistence Estimates

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 (USEPA, 1990). The few results available from standard tests for biodegradation in water show that PAHs with up to four aromatic rings are

biodegradable under aerobic conditions but that the biodegradation rate of PAHs with more aromatic rings is very low. Biodegradation under anaerobic conditions is slow for all components (Neff, 1979). The reactions normally proceed by the introduction of two hydroxyl groups into the aromatic nucleus, to form dihydrodiol intermediates. Bacterial degradation produces *cis*-dihydrodiols (from a dioxetane intermediate), whereas metabolism in fungal or mammalian systems produces *trans*-dihydrodiol intermediates (from an arene oxide intermediate). The differences in the metabolic pathways are due to the presence of the cytochrome P450 enzyme system in fungi and mammals. Algae have been reported to degrade benzo[a]pyrene to oxides, peroxides, and dihydroxydiols. Owing to the high biotransformation rate, the concentrations of PAHs in organisms and water are usually not in a steady state. Freely dissolved PAH may be rapidly degraded under natural conditions if sufficient biomass is available and the turnover rates are fairly high.

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 months; 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).

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). PAHs are inherently biodegradable, and low molecular mass compounds can be completely degraded by acclimated microorganisms.

Biodegradation is the major mechanism for removal of PAH from soil. PAH with fewer than four aromatic rings may also be removed by volatilization and photolysis. The rate of biodegradation in soil depends on several factors, including the characteristics of the soil and its microbial population and the properties of the PAH present. Temperature, pH, oxygen content, soil type, nutrients, and the presence of other substances that can act as co-metabolites are also important (Sims & Overcash, 1983). Biodegradation is further affected by the bioavailability of the PAH. Sorption of PAH by soil organic matter may limit the biodegradation of compounds that would normally undergo rapid degradation (Manilal & Alexander, 1991); however, no significant difference was found in the biodegradation rate of anthracene in water with 10 and 1000 $mg \cdot L^{-1}$ suspended material (Leslie *et al.,* 1987).

After biodegradation of pyrene by a *Mycobacterium* sp., *cis*- and *trans*-4,5-pyrene dihydrodiol and pyrenol were the initial ring oxidation products. The main metabolite was 4-phenathroic acid. The ring fission products were 4-hydroxyperinaphthenone and cinnamic and phthalic acids (Heitkamp *et al.,* 1988). The pyrene-metabolizing

Mycobacterium sp. can also use phenanthrene and fluoranthene as the sole source of carbon. Phenanthrene was degraded and 1-hydroxy-2-naphthoic acid, *ortho*-phthalate, and protocatechuate were detected as metabolites.1-Hydroxy-2-naphthoic acid did not accumulate, indicating that it was further metabolized (Boldrin *et al.,* 1993).

A strain of *Arthobacter* sp. was isolated that was capable of metabolizing fluorene as a sole energy source: 483 nmol·mL^{-1} were degraded completely within 36 h, and four major metabolites were detected: 9-fluorenol, 9 *H*-fluoren-9-one, 3,4-dihydrocoumarin, and an unidentified polar-substituted aromatic compound. Fluorenol was not degraded further, suggesting that it and fluorenone are products of a separate metabolic pathway from that which produces dihydrocoumarin, the polar compound, and the energy for cell growth. The bacteria could also degrade phenanthrene (Grifoll *et al.,* 1992).

The degradation of PAH was studied in a culture made from activated sludge, polychlorinated biphenyl-degrading bacteria, and chlorophenol-degrading mixed cultures, adapted to naphthalene. The metabolites of naphthalene were 2-hydroxybenzoic acid and 1-naphthalenol, those of phenanthrene were 1-phrenanthrenol and 1-hydroxy-2-naphthalenecarboxylic acid, and that of anthracene was 3-hydroxy-2-naphthalenecarboxylic acid. The authors concluded that the biotransformation pathway proceeds via initial hydroxylation to ring cleavage, to yield the *ortho* or *meta* cleavage intermediates, which are further metabolized via conventional metabolic pathways (Liu *et al.,* 1992).

PAH metabolism by fungi has some similarities to metabolism in other biological groups discussed above. For example, *Cunninghamella elegans* in culture metabolizes benzo[a]pyrene to the *trans*-7,8-diol, the *trans*-9,10-diol,3,6-quinone, 9-hydroxybenzo[a]pyrene, 3-hydroxybenzo[a]pyrene, and 7,8-dihydro-7,8-dihydroxybenzo[a]pyrene (Cerniglia, 1984). In a further experiment, *C. elegans* metabolized about 69% of added fluorene after 24 h. The major ethyl acetate-soluble metabolites were 9-fluorenone (62%), 9-fluorenol, and 2-hydroxy-9-fluorenone (together, 7%). The degradation pathway was similar to that in bacteria, with oxidation at the C9 position of the five-member ring to form an alcohol and the corresponding ketone. 2-Hydroxy-9-fluorenone had not been found as a metabolite previously (Pothuluri *et al.,* 1993).

4.4 Fate in Surficial Soils

PAHs are adsorbed strongly to the organic fraction of soils and sediments. Some PAHs may be degraded biologically in the aerobic soil layer, but this process is slow because sorption to the organic carbon fraction of the soil reduces the bioavailability. For the same reason, leaching of PAHs from the soil surface layer to groundwater is assumed to be negligible, although detectable concentrations have been reported in groundwater.

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; ATSDR, 1990; 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).

In Kidman sandy loam, the biodegradation rates varied between 0.23 h^{-1} (or 5.5 d^{-1}) for naphthalene and 0.0018 d^{-1} for fluoranthene. In a study with sandy loams, forest soil, and roadside soil partially loaded with sewage sludge from a municipal treatment plant, the following half-lives (in days) were found: 14-48 for naphthalene, 44-74 for acenaphthene plus fluorene, 83-193 for phenanthrene, 48-210 for anthracene, 110-184 for fluoranthene, 127-320 for pyrene, 106-313 for benz[a]anthracene plus chrysene, 113-282 for benzo[b]fluoranthene, 143-359 for benzo[k]fluoranthene, 120-258 for benzo[a]pyrene, 365-535 for benzo[g,h,i]perylene, and 603-2030 for coronene (Wild & Jones, 1993).

4.5 Aqueous Fate and Geochemistry

Photo-oxidation in water 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). As previously described, sediments are a primary sink for PAHs (Payne *et al.*, 1988; Vandermeulen, 1989) where they persist and transform very slowly. 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 then subject to transport processes (Windsor and Hites, 1979; Larsen *et al.*, 1986).

5. BEHAVIOUR AND EFFECTS IN BIOTA

5.1 Soil Microbial Processes

There are very limited data available for the toxicity of PAHs on ecologically important microbial functional groups. Based on the literature review conducted, only one study was located on the toxicity of PAHs to microbes. Sverdrup *et al.,* (2002c) documented the microbial toxicity of fluorene, fluoranthene, pyrene, and phenanthrene (Appendix II).

Sverdrup *et al., (*2002c) found that all PAHs tested affected the nitrification process, and at very high concentrations weakly affected the total number of protozoans; none affected bacterial diversity (as measured by denaturant gradient gel electrophoresis). Therefore, nitrification was the most sensitive of the three toxicity indicators evaluated for pyrene, fluoranthene, phenanthrene, and fluorene.

There is an extensive body of literature on the relationship between PAH biodegradation and microbial biochemistry, physiology, community structure, soil properties, bioavailability to microorganisms, and co-metabolism. A review of this information is beyond the scope of this document.

In general, the presence of petroleum hydrocarbons in soils, including PAHs, can both reduce and augment heterotrophic microbial activity or microbial diversity. Many studies have demonstrated that various microbial and fungal groups have an ability to utilize some of the PAHs, including naphthalene, phenanthrene, and pyrene, as sole carbon sources, or to co-metabolize PAHs. The rate of microbial biodegradation of PAHs is influenced by the concentration present and pre-conditioning of the microbial community to encourage the presence of PAH-degrading types. At high PAH concentrations, microbial activity may be suppressed as the toxic effects overcome the value of the contaminant as an energy source to the bacteria.

Biodegradation is likely to be influenced by the bioavailability of the PAHs to the microbial consortia. In turn, bioavailability is related to how tightly sorbed the PAHs are to soil particles, or whether some fraction of the PAHs are biologically inaccessible by virtue of being occluded in particles (as in the case of many combustion-derived vitreous particles) or otherwise internalized in a manner that reduces partitioning into the soil pore water.

5.2 Terrestrial Plants

5.2.1 Bioavailability/Bioaccumulation

Four main pathways exist for chemicals in soils to enter plants. These include -

i) root uptake from soil solutions via conduction channels and possible translocation into the transpiration stream of xylem (i.e., liquid phase transfer);

ii) vapour/aerosols in the surrounding air (gas or vapour phase transfer) into the stomata of stems and/or shoots or absorption by roots from soil air spaces and potential translocation by the phloem);

iii) external contamination of shoots by wind- or airborne soil and dust (particle phase), which is retained in the cuticle or penetrates within; and,

iv) uptake in oil cells or channels of oil containing plants such as carrot, cress, and parsnip.

Organic contaminants can be divided into three major categories in terms of their environmental fate tendencies:

i) substances that are volatile and easily released from soils to the atmosphere;

ii) substances that are rapidly mineralized (metabolized) by microbes and exhibit little persistence; and

iii) substances that are persistent, being strongly adsorbed to the soil (organic) matrix).

Higher molecular weight PAHs such as benzo(a)pyrene fit in the third, while lower molecular weight PAHs such as naphthalene also tend to exhibit a more limited persistence. A combination of these pathways and mechanisms will reflect the total contaminant concentration of a given plant (Bell and Failey, 1991; Simonich and Hites, 1995; Duarte-Davidson and Jones, 1996).

Roots of vascular plants are the main site of nutrient and water uptake from the soil and are therefore a likely point of entry for other chemicals as well. This occurs most actively in the root hair zone located approximately 20 to 40 mm above the root cap where the largest surface area for absorption is found. From this point, absorbed substances may pass through the living (*symplastic* – phloem and cell cytoplasm) and/or non-living (*apoplastic* – cell walls and xylem) tissues via mass flow and diffusion to other areas of the plant. Movement of substances through the symplast occurs at a relatively slow rate of centimetres per hour, moving within cell cytoplasm where they may be placed in close proximity to reactants such as enzymes and other chemicals. Apoplastic movement is much faster (occurring at rates of metres per hour) operating under positive pressure or tension from transpiration streams generated upward by the plant stem and leaves (Bell and Failey, 1991).

Chemicals entering the plant may be restricted to either the symplast, apoplast, or may be mobile in both systems (ambimobile). Initial uptake by the root is a function of its

concentration in the soil solution (Finlayson and MacCarthy, 1973). Although chemical contaminant uptake by roots from the soil is generally a passive process initially, the uptake and transport occurs through a series of successive partitions between different phases as follows:

soil solids ⇒ soil water/vapour ⇒ roots ⇒ transpiration stream

⇒ plant stem ⇒ plant leaves.

Some organic compounds, such as pesticides, have been found to be taken up in a two-phase process (Schwarz and Jones, 1997). These include an initial rapid phase in which the compound diffuses passively into root apparent free space, potentially followed by a second slower and longer accumulation phase in which the compound passively permeates the entire volume of exposed tissue. For some pesticides; such as 2,4-D, atrazine, napropamide, and picloram; this second phase may be active or metabolically dependent using a carrier –mediated mechanism (see Schwarz and Jones, 1997, for additional references).

A number of soil processes can affect the bioavailability of organic contaminants to plants including adsorption/desorption (affecting chemicals available in soil solution for biotic interaction), leaching (potentially to groundwater), abiotic and biotic degradation, and volatilization (i.e., vapour-phase partitioning between the soil solution and soil air spaces) (O'Connor, 1996). Beck *et al., (*1996) noted that photochemical, as well as chemical transformations are also possible.

PAHs may be bound within soils (via lignification), mineralized (ultimately to CO_2 and water) or metabolized outside or within the plant at any point in the above process. The chemical may, as a result, be found as freely (chemically) extractable or non-extractable residues in plants, or extractable conjugates bound to plant materials such as carbohydrates and amino acids (Bell and Failey, 1991; Banks *et al*., 2000; Schnoor, 1997; Duarte-Davidson and Jones, 1996; Harms, 1996). Schnoor (1997) cites examples from the literature such as the chlorinated aliphatic compound, trichloroethylene, which has been found to be mineralized to CO_2 and less toxic aerobic metabolites (e.g., trichloroethanol and chloroacetic acids). The author, however, also states "*…there are no reports in the literature of aromatic compounds (e.g., PAHs) being completely mineralized by plants*" (Schnoor, 1997).

The extent to which an organic contaminant in soil initially enters the roots of a plant from the soil depends on its water solubility, Henry's law constant (linking the partial pressure of the compound and its gas concentration in solution), and the octanol-water partition coefficient (or K_{OW} - the degree to which an organic compound prefers an oil or lipid over water). Other influencing factors include the organic content of the soil medium (which tends to bind or sequester organic contaminants such as hydrocarbons) and associated pH; temperature; moisture; chemical species of the contaminant; and the species of plant, including its available root surface area, stage of plant growth and lipid content (Simonich and Hites, 1995; Duarte-Davidson and Jones, 1996; Al-Assi, 1993).

Uptake of anthracene from soil by wheat and barley was examined by GRI/PERF (2000) at various concentrations, with aging, and with varying soil organic carbon content. As the concentration of anthracene increased from <1 to 250 $mg \cdot kg^{-1}$, uptake also increased, with greater uptake in the wheat than the barley (GRI/PERF, 2000). Aging decreased uptake, such that at 203 days of aging (the longest period tested), uptake declined by 29% and 32% in the wheat and barley, respectively. Organic carbon content was also found to be an important factor affecting availability.

Lipophilic organic compounds such as PAHs, with a low solubility in water, high Henry's law constant and high K_{OW} (> 10^4) are bound strongly to the root surface and/or soils and are not readily translocated within plants (Schnoor, 1997). These generally tend to partition into the epidermis or outer layers of the root tissue (or peel) and remain there bound to lipids in cell walls; transfer into the inner root or xylem is very slow or non-existent. Duarte-Davidson and Jones (1996) add that these compounds may remain bound in the outer root tissue for the lifetime of the plant due to their high persistence. Duarte-Davidson and Jones (1996), in their preliminary screening of organic contaminants in sludge-amended soils for potential transfer to plants and grazers, further noted that few organic compounds have a high potential for root uptake and translocation.

Al-Assi (1993), in his literature review of PAH uptake in crops, indicates that a majority of studies have involved experiments on sewage sludge-amended soils (see Wild and Jones, 1992) or roots grown in PAH-amended nutrient solutions (see Edwards, 1986). Al-Assi (1993) studied the uptake of PAHs by alfalfa and fescue in soil contaminated with pristane, anthracene, and pyrene to a total concentration of 100 $mg \cdot kg^{-1}$ (or ppm) between a four- and 24-week growth period. The investigator found that PAH concentrations in plant shoots were low for both plant species, decreased over time, and appeared to be independent of soil PAH concentrations. Pyrene was not detected in plant roots of either species. It was concluded that PAHs in soil do not accumulate to any significant degree in the crop plants studied. Although the investigator did not specifically address the issue of accumulation in shoots, the results would also suggest that atmospheric deposition on shoot surfaces may have also played a role in the study.

The general consensus in the literature is that the root uptake pathway of organic contaminants such as hydrocarbons and PAH constituents from the soil by plants is extremely limited. Low molecular weight PAHs (i.e., with two, three or four rings) may be taken up by roots and translocated within the plant, but do not appear to accumulate or magnify in concentration relative to concentrations in the soil (EPRI, 1992). High molecular weight PAHs (i.e., five or more rings) may sorb to plant roots, but are not expected to translocate or accumulate within the plant (EPRI, 1992). Simonich and Hites (1995) conclude that:

> "…many controlled exposure experiments and field measurements have shown that the uptake of lipophilic organic (contaminants) through roots is not a significant pathway of accumulation….In general, lipophilic (contaminants) are not translocated within the plant and metabolism is not significant."

The above conclusion was supported for PAHs by the results of a study that was designed to optimize conditions for plant uptake of PAHs and minimize the competing processes of soil sorption and microbial degradation (EPRI, 1993). In this study, a rapidly growing herbaceous plant, white sweetclover (*Melilotus alba*), was exposed to high concentrations (250 to 500 $mg \cdot kg^{-1}$) of low molecular weight radio-labelled PAHs (naphthalene, phenanthrene, pyrene, and fluoranthene) in soils of low organic carbon content (<1%). Even under these conditions, after an exposure of 5 days, only 0.8% of the naphthalene and less than 0.02% of the other three PAHs were found in the above-ground foliage.

For organic contaminants such as PAHs, O'Connor *et al.,* (1991; see also Duarte-Davidson and Jones, 1996; and Beck *et al*., 1996) contend that airborne sources are regarded as the primary source of plant contamination in experiments with sludge amended soils. A few rare exceptions have been noted in the literature with respect to garden produce and dioxin/furan-contaminated soil (Hülster *et al.,* 1994).

Although there is little evidence to suggest that substances such as PAHs, and other organic contaminants in soils accumulate to any significant degree in plants beyond the peripheral root tissues, there is, nevertheless, a growing amount of evidence on the ability of various crop plants and grasses to degrade and dissipate petroleum contaminated soil (Banks *et al*., 2000). This occurs primarily in the rhizosphere of plants where high concentrations of bacterial populations use the organic compounds as carbon substrate for growth, resulting in the breakdown and eventual mineralization of the contaminants. This may also be augmented by the release of root exudates (e.g., sugars, alcohols and acids which stimulate bacterial activity), enzymes and the build-up of organic carbon (via root turnover) in the soil (Schnoor, 1997).

Degradation of PAHs, BTEX (benzene, toluene, ethylbenzene, and xylenes) and phenolics has been observed in a number of perennial grass and crop species such as alfalfa, fescue (*Festuca* sp.) big blue stem (*Andropogon* sp.), Indian grass (*Sorghastrum* p.), switchgrass (*Panicum* sp.), Canadian wild rye (*Elymus* sp.), little bluestem (*Schizachyrium* sp.), and side oats grama (*Bouteloua* sp.) grown in petroleum contaminated soil (Aprill and Sims, 1990; Schwab and Banks, 1994; Banks *et al*., 2000). In most of these cases, plant uptake, adsorption, weathering, and soil leaching could not account for the observed removal of PAHs; leaving microbial biodegradation in the rhizosphere as the most probable explanation for the decrease. Similar properties have also been shown for BTEX in legumes, in addition to alfalfa, such as clover and cowpeas (Schnoor, 1997). Crested wheatgrass (*Agropyron* sp.) has been demonstrated to mineralize PCP and phenanthrene in flow-through plant chamber experiments, converting up to 22% of the original PCP concentration in the soil to $^{14}CO_2$ (Ferro *et al.,* 1994).

5.2.2 Toxicity

Terrestrial plants exhibit a wide range of variability in tolerance to PAH-contaminated soil. Henner *et al., (*1999) identified plants growing on three abandoned manufactured gas plant sites in France (Table 5-1). They also examined inhibition of emergence and

growth reduction using maize (*Zea mays)*, barley *(Hordeum vulgare*), fescue (*Festuca rubra*), colza (*Brassica napus)*, rye grass (*Lolium perenne*), alfalfa (*Medicago sativa*), and red clover *(Trifolium pratense*) in field and laboratory trials on coal tar contaminated soils. Soils with appreciable concentrations of more volatile, water soluble components of less weathered contaminated soils (for example, benzene, toluene, xylenes, styrene, indene, and naphthalene) induced phytotoxic responses. Plant germination and growth was not inhibited, however, in weathered soils, with visually identifiable coal tar, but without detectable hydrocarbon odors. At the sites, the plants – all common pioneering/opportunistic species – had deep and extensive root growth, and in many cases the roots grew through coal tar residues. Furthermore, there was no inhibition of germination or growth of lupin seeds (*Lupinus albus*) planted in uncontaminated agricultural soils spiked with benzo[a]pyrene, benz[a]anthracene, or dibenz[a,h]anthracene concentrations in the range of 31 to 155 mg PAH·kg^{-1} soil.

Lower molecular weight PAHs with a log K_{OW} less than approximately 4.0 to 4.6 tend to be toxic to plants at sufficiently high soil concentrations, while higher molecular weight PAHs have very limited potential for phytotoxicity, likely due to the limited potential for desorption from soils in the root zone, and hence limited bioavailability.

Table 5-1: Plant species identified growing on three manufactured gas plant sites

(from Henner et al., 1999)

Apiaceae	*Daucus carota*	Papaveraceae	*Papaver rhoeas*
Asteraceae	*Artemisia vulgaris*	Poeaceae	*Cynodon dactylon*
	Cirsum arvensis		*Digitaria sanguinalis*
	Erigeron canadensis		*Festuca rubra*
	Matricaria perforata		*Poa annua*
	Sonchus oleracerus	Ranunculaceae	*Clematis vitalba*
	Tanacetum vulgare	Rosaceae	*Ronce*
Boraginaceae	*Echium vulgare*	Solanaceae	*Solanum dulcamara*
Brassicaceae	*Diplotaxis tenuifolia*		*Solanum nigrum*
	Sinapis arvensis	Verbasceae	*Verbascum thapsus*
	Sysimbrium officinale	Betulaceae	*Betula alba*
Caryophyllaceae	*Silene dioica*	Salicaceae	*Populus tremula*
Chenopodiaceae	*Chenopodium hybridum*		
	Chenopodium murale		
Hypericaceae	*Hypericum perforatum*		
Crassulaceae	*Sedum acrum*		
	Sedum album		
	Loganiaceae buddleia		
Onagraceae	*Epilobium angustifolium*		
	Epilobium perforatum		

Laboratory-based plant toxicity data that are used to derive soil quality guidelines using CCME or other protocols often do not capture the range of potential modifying factors for phytotoxicity in a field setting. Joner and Leyval (2001) examined the influence in the laboratory of combined effects of the positive influences of arbuscular mycorrhiza and the potentially negative effects of a mixture of three PAHs [500 $mg \cdot kg^{-1}$ of anthracene (3-ring) and chrysene (4-ring) and 50 $mg \cdot kg^{-1}$ dibenz[a,h]anthracene (5-ring)]. Mycorrhizal inoculations (*Glomus mossaea* P2, BEG 69) of topsoil without PAHs increased the shoot dry weight of clover 100% and 30% after 8 and 16 weeks growth, respectively. The shoot growth of ryegrass was not affected, since this is not an N-fixing plant. Approximately 20% of the clover roots were colonized at both harvest periods by the mycorrhizal organism. In PAH-spiked topsoil, the addition of mycorrhizal organisms enhanced clover shoot yield (dry weight) by approximately 2-fold, but during the 8 day sampling period this was still only about 60% of the yield of clover in mycorrhizal spiked soil without the PAHs. The PAHs inhibited mycorrhizal colonization of clover roots relative to the non-PAH treatment by about 50%. Effects of PAHs on both mycorrhizal colonization of clover roots and on shoot yield were further exacerbated in treatments with both PAH additions and the further addition of a surfactant that was intended to increase PAH bioavailability. This study illustrates a small portion of the complexity of plant responses at PAH-contaminated field sites.

Joner and Leyval (2001) did not find evidence of PAH metabolism in the arbuscular mycorrhizal fungus.

Gaspar *et al.* (2002) demonstrated the ability of the arbuscular mycorrhizal fungus *Glomus geosporum* to grow in a substrate spiked with 100 $mg \cdot kg^{-1}$ phenanthrene. However, percentages of colonization of maize roots were higher in controls than in treated substrates with either phenanthrene or phenanthrene plus yeast. Cabello (1997) found in field experiments that hydrocarbon contamination affects the arbuscular mycorrhizal fungal population associated with plants.

5.3 Terrestrial Invertebrates

5.3.1 Bioavailability/Bioaccumulation

There has been a strong interest in the last half decade in soil sorption properties and 'aging' of contaminants in soil, which has the potential to influence the bioavailability – and hence toxicity - of hydrophobic organic contaminants to soil invertebrates and plants based on direct-contact exposure routes, or based on soil ingestion (Reeves *et al.,* 2001).

A detailed review of this large and rapidly evolving body of knowledge is beyond the scope of this document. Alexander (2000) provides a review. Several researchers have conducted research primarily on earthworms as a model to test variations in bioavailability of hydrophobic organic contaminants from soils based on (i) soil properties such as organic carbon content, (ii) contaminant properties, and (iii) freshly introduced versus older contaminant introductions into the soil. Kreitinger *et al.* (2007) studied uptake and toxicity to earthworms of PAHs in soil. They found that the fraction

of PAHs that was rapidly released through supercritical CO_2 extraction was a much better predictor of toxicity than measured total PAH in the soil. They also found that the bioavailability of PAHs from soils in which most of the organic carbon content was of anthropogenic origin was 5 to 50-fold lower than the bioavailability of PAHs that were freshly added to soil (Kreitinger *et al.*, 2007). Bioconcentration factors for uptake by earthworms of anthracene, fluoranthene, and pyrene from soil were found to range from 0.99 to 2.34, depending on the PAH and whether the PAH had been aged or not (GRI/PERF 2000).

There is little question that there exist geochemical mechanisms that have the potential to limit PAH bioavailability in soils to soil invertebrates. Historically, the scientific ability to quantitatively and accurately predict sorption-associated variations in bioavailability and toxicity has been extremely limited. However, the recent development of methods for measuring concentrations of PAHs in soil porewater, such as solid-phase microextraction (Hawthorne *et al.*, 2005) or supercritical fluid extraction (Hawthorne and Grabanski, 2000; Hawthorne *et al.*, 2001), holds promise for assessing site-specific bioavailability and ecological risk (USEPA, 2006). One area of active research is the development of field measurement/indicator approaches that can be used to predict variations in bioavailability to the larger suite of soil-dwelling organisms that might be exposed at a contaminated site.

5.3.2 Toxicity

There are very few studies on the toxicity of PAHs to terrestrial invertebrates. One of the better studies on field populations of soil invertebrates exposed to PAHs was by Best *et al.* (1978). Field abundances of soil mites (*Acari*), collembolans, other soil mesofauna and larger arthropods were exposed to naphthalene, in a surface-applied granular form, and the abundances of various groups compared with reference plots. Soil mites (including mesostigmatid, orbatid, and prostigmatids), collembolans, the spider *Gammonota inornata*, and arthropods were generally observed to exhibit a 50% reduction in abundance (EC_{50}) after 8 days exposure at a naphthalene application rate of 8 to 21 g naphthalene$\cdot m^{-2}$. This can be roughly equated with a soil concentration by assuming a soil specific gravity of approximately 1.7 and a mixing depth of 1 cm, which would result in EC_{50} estimates in the range of 450 to 1,300 mg$\cdot kg^{-1}$ naphthalene. While this seems like a very high concentration, it is important to understand that short-term acute toxicity in soil invertebrate populations generally occurs at much higher contaminant concentrations than chronic effects on reproduction, or other physiological processes that could directly influence population fitness over periods of one month or more.

Blakely *et al. (*2002) examined field populations of soil invertebrates at a 0.5 ha site contaminated with creosote. The concentration of 2- to 4-ring PAHs was significantly correlated with soil bulk density. Concentrations of 5-ring PAHs were negatively correlated with soil electrical conductivity. The correlation between PAH concentrations and soil properties potentially confounded the interpretation of various significant correlations between soil PAH concentrations and soil community responses. Abundance of nematodes in the Apelenchoididae and Paratylenchidae families was negatively

correlated with benzo[a]pyrene concentration (r = 0.25 to 0.26). The correlational nature of the study precludes an evaluation of threshold-of-effects levels; however, the total number of soil mites was apparently reduced with increasing PAH soil concentrations. No direct association between collembolans and PAH concentrations was noted. The authors noted that different species within one family or order may have contrasting responses to PAH-related stresses. One interesting aspect of this study was the use of several nematode community maturity indices and comparison with soil PAH concentrations.

Sverdrup *et al.* (2001; 2002a,b,d,e) reviewed previous studies of PAH toxicity to soil organisms, and presented detailed chronic toxicity data for three new species (*Folsomi fimetaria, Enchytraeus crypticus,* and *Eisenia veneta*) and for four to eight PAHs in a single, standardized soil type. Survival exhibited a rather steep dose-response curve in all three species tested, while growth and reproduction exhibited a more gradual concentration-response slope. The larger difference in sensitivity between the three species was approximately 23-fold (for acridine). Calculated mortality NOEC values for the three species were in the range of approximately 10 to 100 $mg \cdot kg^{-1}$ pyrene, fluoranthene, phenanthrene or fluorene.

Increasingly, laboratory-based toxicity tests employing soil invertebrates, and/or field-based observations are being used in order to provide a more direct measure of the relationship between PAH contamination and ecological risks. Sayles *et al. (*1999), for example, measured changes over time in land-farmed soil with respect to the toxicity to earthworm survival (14 day earthworm survival: *Eisenia fetida* and *Lumbricus terrestris)*, *Allium* (onion) mitotic anomalies, *Avena sativa* and *Lactuca sativa* germination and root elongation, Ames mutagenicity test, and Microtox response (bioluminescent microbial assay). The authors concluded that a large battery of tests is needed to assess toxicity and soil remediation, given the disparity in observed responses across the tests.

5.4 Livestock

The available literature was surveyed for toxicity data for PAHs or PAH-containing mixtures based directly on studies on cattle or similar domesticated ungulate or other livestock. No studies could be located with appropriate conclusions on dose-response relationships between the individual PAHs and acute or chronic toxicological responses in livestock (Appendix VII).

Coppock and Campbell (1997), recently provided a thorough review of petroleum hydrocarbon risks to livestock. This document should be consulted for more information on the state of the science. There is a large body of published information, especially in veterinary journals, mostly anecdotal and symptom accounts of accidental poisonings of livestock, often through the ingestion of mineral spirit carriers for topical remedies applied to the coat, or through the direct ingestion of petroleum products such as mogas or diesel. Many of these studies provide details of symptoms and acute pathology, which may be diagnostic of petroleum hydrocarbon poisoning.

Less than a half-dozen studies have value in assigning a threshold dose for cattle or other livestock of petroleum hydrocarbon mixtures. Chalmers (1997) reviewed threshold dose estimates for crude oil in cattle, which range from >1.25 to 8 $mL \cdot kg^{-1}$ (b.w.). This information is reproduced herein as Table 5-2. Similar information was used in the development of soil standards for agricultural land uses under the recently adopted Canada-Wide Standards for Petroleum Hydrocarbons.

Unweathered oil (with a specific gravity of 0.843) exhibited a threshold dose of 2.5 $mL \cdot kg^{-1}$ (adapted from Stober, 1962).

Table 5-2: Threshold Doses for Crude Oil in Cattle

(adapted from Chalmers, 1997)

Oil Type	Composition	Threshold Dose
Unweathered Oil	100 mL = 84.3 g Carbon = 84.6% (19% arom.) Hydrogen = 11.92% Nitrogen = 0.71% Sulfur = 2.46%	2.5 to 5 $mL \cdot kg^{-1}$ bw = 2.1 to 4.2 $g \cdot kg^{-1}$ bw
Weathered oil	Water 10% by wt. 100 mL = 91.0 g Carbon = 83.6% (21% arom.) Hydrogen = 11.56% Nitrogen = 0.49% Sulfur = 2.8%	8 $mL \cdot kg^{-1}$ (b.w.) = 7.3 $g \cdot kg^{-1}$ bw
Venezeula crude oil (naphtha-based)	100 mL = 87.5 g Carbon = 85.6% (19% arom.) Hydrogen = 12.95% Nitrogen = 0.46% Sulfur = 1.58%	= 4.0 $mg \cdot kg^{-1}$
Bunker “C” oil	100mL = 97.0g Carbon = 86% (19% arom.) Hydrogen = 11% Nitrogen and Oxygen = 0.46% Sulfur = 2.5%	> 1.25 $mL \cdot kg^{-1}$ = > 1.1 $g \cdot kg^{-1}$ bw

A study by Rowe *et al. (*1973) involved the treatment of 11 cattle (varying in age from 6 months to 3.5 years) with sweet crude, sour crude, or kerosene. Crude oil dosages ranged from 37 $mL \cdot kg^{-1}$ body weight, given as a single dose, to 123 $mL \cdot kg^{-1}$ (b.w.) given as five doses over a five-day period. Kerosene dosages ranged from a single dose of 19.8 $mL \cdot kg^{-1}$ (b.w.) to 61.6 $mL \cdot kg^{-1}$ (b.w.) given as five doses over five days. In addition, 3 separate groups of five calves were administered crude oils and kerosene at a rate of 8 $mL \cdot kg^{-1}d^{-1}$ (b.w.) for up to 14 consecutive days. A dose of 8 $mL \cdot kg^{-1}d^{-1}$ (b.w.) to one calf

produced only mild signs of pneumonia, from which recovery occurred. Higher single doses to calves or adults resulted in a variety of more severe effects, including mortality for some doses and individuals. A threshold dose of 8 $mL \cdot kg^{-1}d^{-1}$ (b.w.) for 14 days is consistent with the LOAEL derived by Coppock and Campbell (1997).

Coppock and Mostrom (1997) summarized the available information on PHC constituent toxicity to cattle or other livestock based on reproductive impairment, a potentially important chronic endpoint.

For individual constituents other than the PAH benzo(a)pyrene, little mammalian reproductive dose-response data exist. For benzo(a)pyrene, the available information is based on mice and rats (see Appendix VII). No directly applicable livestock toxicity data were located for individual PAHs for behavioural, teratogenic, or other endpoints.

ATSDR (1995c), WHO/IPCS (1998) and other recent compendia were consulted for "minimum risk levels" (MRLs) or other toxicity reference values based on chronic to subchronic sublethal mammalian toxicity studies for individual PAHs. Since the underlying primarily mammalian toxicity data (Appendix VII) are the same as is available for estimating risks to wildlife species, guiding studies are discussed in more detail in the next section.

5.5 Other Mammals and Wildlife

It is often challenging to find relevant toxicological data for terrestrial mammalian, avian, amphibian or reptile receptors. For example, two studies were located on the effects of water-borne PAHs on amphibian tadpole development - with and without photoinduced toxicity (Appendix V). No data could be located, however, for the major portion of passerine bird species (Appendix VI) that might be found at an agricultural or residential/parkland site in Canada. Similarly, no information on PAH toxicity to wildlife mammalian species, except deer mice, could be located (Appendix VII).

Relevant data for many substances from laboratory studies on rodents are available, for example through EPA's Integrated Risk Information System (IRIS). Considerable laboratory data exist for rat, mouse or other mammalian surrogates of human health for many contaminants, which are then used by the USEPA, US National Institute of Occupational Safety and Health, World Health Organization, Health Canada, and other risk assessment/management agencies for deriving human health protective benchmarks; i.e., reference doses or concentrations (RfDs and RfCs respectively), allowable or tolerable daily intakes (ADIs and TDIs, respectively), and so on.

Typically, laboratory mammal toxicity studies are chosen for the further development of human health protective benchmarks for contaminant exposure if they provide a relevant and potentially reproducible toxicity estimate (LOAEL and/or NOAEL) for a chronic or sub-chronic exposure. Such benchmarks are converted to generically applicable threshold-of-effect levels by applying various formally defined uncertainty factors to account for (i) intra-specific variations in sensitivity, (ii) inter-specific variations in

sensitivity, (iii) extrapolation of potential for important non-lethal adverse responses from mortality-type endpoints, (iv) extrapolation of potential for chronic effects from acute or sub-chronic exposure regimes, and (v) other aspects pertaining to professional confidence in the study design, including availability of corroborating studies.

Appendices V, VI, and VII of this document provide a collation of available, non-carcinogenic toxicity data for vertebrate test species excluding fish. According to ATSDR (1995c), no data exist on the effects of inhalation exposures to PAHs by vertebrate species for any of the following types of effects: reproduction, neurological effects, developmental effects, and genotoxic effects. The emphasis herein, however, is on oral exposures rather than dermal exposures, as well as on toxicity endpoints other than cancer. Toxicity endpoints deemed to be of particular relevance to the fitness of livestock and wildlife populations include mortality, reproduction, growth, and immunocompetence (disease resistance).

Chronic toxicity mammalian data for any of the PAHs is notably absent (ATSDR, 1995c), while sub-chronic or intermediate data tend to be very limited. In some cases, therefore, it is necessary to use acute toxicity data.

5.5.1 Naphthalene

According to the IRIS database, there are no adequate chronic oral dose-response data for naphthalene in humans or animals. The guiding study (BCL, 1980) used for assessing mammalian toxicity in the absence of chronic exposure reported a decreased body weight in rats exposed by gavage to naphthalene for 90 days (sub-chronic exposure). A NOAEL and LOAEL of 100 and 200 $mg \cdot kg^{-1} \cdot day^{-1}$, respectively, was found for terminal body weight decreases greater than 10% of control values in male rats. Thirty-five Fischer 344 rats (10/sex/dose) received doses of naphthalene in corn oil at 0, 25, 50, 100, 200, or 400 $mg \cdot kg^{-1}$ for 5 days/week for 13 weeks (BCL, 1980). The NOAEL and LOAEL for decreased terminal body weight (> 10% decrease compared with controls) were 200 and 400 $mg \cdot kg^{-1} \cdot day^{-1}$, respectively, in females and 100 and 200 $mg \cdot kg^{-1} \cdot day^{-1}$ in males. Accounting for adjustments of rat body weight and duration of exposure, the adjusted NOAEL and LOAEL for decreased body weight were 71 and 143 $mg \cdot kg^{-1} \cdot day^{-1}$, respectively.

Shepard (1986) examined changes in reproductive success of pregnant rabbits exposed to naphthalene 3 times: on days 20, 22 and 24 of the gestational period. A calculated NOAEL for this study was 16 $mg \cdot kg^{-1}$.

Administration to rabbits of 0.1 to 1 $mg \cdot kg^{-1}$ (b.w.) per day naphthalene by subcutaneous injection for 123 days resulted in severe oedema and a high degree of vacuolar and collicular degeneration in the brain; necrosis of nerve cells also occurred. The author suggested that hypoxaemia resulting from haemolytic anaemia was responsible for this finding (Suja, 1967; cited in WHO/IPCS, 1998).

5.5.2 Acenaphthene

USEPA (1989a, in ATSDR, 1995c) observed an increased relative liver weight in sub-chronically exposed CD-1 mice. The mice were orally exposed to acenaphthene once per day over 13 weeks, resulting in a NOAEL of 175 $mg \cdot kg^{-1} \cdot day^{-1}$ and a calculated LOAEL of 350 $mg \cdot kg^{-1} \cdot day^{-1}$.

5.5.3 Acenaphthylene

Little information was found on toxicological thresholds for acenaphthylene in terrestrial vertebrates. A LOAEL for immune responses in rats, based on a 12-day (acute) exposure period was 180 $mg \cdot kg^{-1}$ (ATSDR, 1995c).

5.5.4 Fluorene

A sub-chronic USEPA (1989b) study as cited in ATSDR (1995c) was located on CD-1 mice orally exposed to fluorene. Mice were exposed once per day for 13 weeks. Groups of 25 male and 25 female CD-1 mice were given 0, 125, 250, or 500 $mg \cdot kg^{-1}$ (b.w.) per day fluorene suspended in corn oil by gavage for 13 weeks. The calculated NOAEL for was 125 $mg \cdot kg^{-1} \cdot day^{-1}$. The LOAEL for increased relative and absolute liver weight and hematological effects was 250 $mg \cdot kg^{-1} \cdot day^{-1}$.

5.5.5 Anthracene

ATSDR (1995c) cited the study by USEPA (1989c) on CD-1 mice sub-chronically exposed to anthracene via the oral route. The mice were exposed for 13 weeks, once per day, and extensive observations were made of cardiovascular, gastrointestinal, hematological, skeletomuscular, liver, renal, endocrine, and dermal characteristics along with body weight. Anthracene was administered to groups of 20 each male and female CD-1 (ICR)BR mice by gavage at a dose of 0, 250, 500, or 1000 $mg \cdot kg^{-1}$ (b.w.) per day for at least 90 days. No treatment-related effects were noted on mortality, clinical signs, body weights, food consumption, ophthalmological findings, the results of haematology and clinical chemistry, organ weights, organ-to-body weight ratios, and gross histological and histopathological findings. A NOAEL for all observed endpoints was 1,000 $mg \cdot kg^{-1} \cdot day^{-1}$ anthracene.

5.5.6 Phenanthrene

No studies were found that examined either sub-chronic or chronic oral exposures of mammalian species to phenanthrene. Two acute mortality data points are available, however. Eisler (1987) summarized a rat acute (single oral exposure) mouse LD_{50} of 700 $mg \cdot kg^{-1} \cdot day^{-1}$ (b.w.). There is very low confidence in this estimate.

5.5.7 Fluoranthene

ATSDR (1995c) cited the study by USEPA (1988) on CD-1 mice sub-chronically exposed to fluoranthene via the oral route. The mice were exposed for 13 weeks, once per day, and extensive observations were made of cardiovascular, gastrointestinal, hematological, skeletomuscular, liver, renal, endocrine, and dermal characteristics along with body weight. A LOAEL for increased relative liver weight in mice was 250 $mg \cdot kg^{-1} \cdot day^{-1}$ fluoranthene, with a NOAEL of 125 $mg \cdot kg^{-1} \cdot day^{-1}$.

Knuckles *et al*. (2003) orally exposed 40 each male and female F-344 rats to fluoranthene by blending into Purina rat diet. The targeted doses were 0 (control), 150, 750 or 1500 mg $fluroanthene \cdot kg^{-1} \cdot day^{-1}$. Urine was collected during days 29, 59 and 89 from 10 rats/sex/group and analyzed for glucose, bilirubin, ketone, specific gravity, blood, pH, protein, nitrite, urobilinogen and leukocytes. Animals were also sacrificed at 30-, 60- and 90-day time points. Haematological parameters were assessed in trunk blood. Subchronic effects on white blood cell and/or red blood cell counts, hematocrit, and haemoglobin were noted at doses greater than 150 $mg \cdot kg^{-1} \cdot day^{-1}$. Overall, a sub-chronic oral NOAEL for the study was determined to be 150 $mg \cdot kg^{-1} \cdot day^{-1}$.

This NOAEL suggests that F-344 rats are less sensitive to fluoranthene administered orally than CD-1 mice. The CD-1 mice were exposed for 13 weeks, similar to the 90 d exposure period used in the Knuckles *et al.* (2003) study; however, liver changes were not assessed in the rats.

5.5.8 Pyrene

For pyrene, a guiding laboratory rodent study, based on USEPA (1989d) is summarized below. Male and female CD-1 mice (n = 20/sex/group) were exposed to pyrene in a corn oil carrier via gavage with 0, 75, 125, or 250 $mg \cdot kg^{-1} \cdot day^{-1}$ for 13 weeks. Observed response variables encompassed body weight changes, food consumption, mortality, clinical pathological evaluations of major organs and tissues, and hematology and serum chemistry. Kidney lesions, described as minimal or mild in all dose groups, were characterized by the presence of multiple foci of renal tubular regeneration, often accompanied by interstitial lymphocytic infiltrates and/or foci of interstitial fibrosis. Relative and absolute kidney weights were reduced in the two higher dosage groups. Based on the results of this study, the low dose (75 $mg \cdot kg^{-1} \cdot day^{-1}$) was considered the NOAEL and 125 $mg \cdot kg^{-1} \cdot day^{-1}$ the LOAEL for nephropathy and decreased kidney weights.

5.5.9 Benz[a]anthracene

Nousiainen *et al.* (1984; in ATSDR, 1995c) exposed Wistar rats by oral gavage to benz[a]anthracene over a four day (acute) period. No sub-chronic or chronic toxicity data

could be located. The dose corresponding to a NOAEL for hepatic, renal and gastrointestinal toxicity was 150 $mg \cdot kg^{-1} \cdot day^{-1}$.

Silkworth *et al*. (1995) exposed mice to a single oral dose of benz[a]anthracene at concentrations of 0.1, 1, 10, or 100 $mg \cdot kg^{-1}$. The LOAEL for immunosuppression in the mice (>50%) was 100 $mg \cdot kg^{-1} \cdot day^{-1}$ with a NOAEL of NOAEL of 10 $mg \cdot kg^{-1}$.

5.5.10 Chrysene

Silkworth *et al.* (1995) observed significantly affected immune response in mice given a single oral dose of 100 $mg \cdot kg^{-1}$ chrysene (acute LOAEL). The corresponding NOAEL concentration was 10 $mg \cdot kg^{-1}$.

5.5.11 Benzofluoranthenes

There is very little toxicological information on benzo[b]-, benzo[j]-, or benzo[k]fluoranthene. Silkworth *et al.* (1995) examined immunocompetence of mice exposed using a single oral dose to several PAHs, at concentrations of 0.1, 1, 10 and 100 $mg \cdot kg^{-1}$ PAH (b.w). For both benzo[b]fluoranthene and benzo[k]fluoranthene, no effect on immunocompetence was observed at 10 $mg \cdot kg^{-1}$ (NOAEL), but greater than 50% suppression of immune response was observed in mice orally exposed to 100 $mg \cdot kg^{-1}$ (b.w.) (LOAEL).

5.5.12 Benzo[a]pyrene

Female CD-1 mice were acutely exposed by gavage to benzo[a]pyrene for 10 days (Mackenzie and Angevine, 1981) and the weight of the pups recorded. Benzo[a]pyrene decreased the percentage of pregnant CD-1 females that reached parturition and produced a high incidence of sterility in the progeny (Mackenzie and Angevine, 1981). The LOAEL for benzo[a]pyrene-induced reproductive toxicity in parental mice was 160 $mg \cdot kg^{-1} \cdot day^{-1}$, and the LOAEL for reduced fertility in the progeny of exposed animals was 10 $mg \cdot kg^{-1} \cdot day^{-1}$.

According to ATSDR (1995c), the route of exposure and the strains of test animals used are major factors in benzo[a]pyrene reproductive toxicity. Benzo[a]pyrene administered in the diet caused no adverse effects on fertility of Swiss mice (Rigdon and Neal, 1965) but reduced the incidence of pregnancy in female rats (Rigdon and Rennels, 1964).

5.5.13 Benzo[g,h,i]perylene

No information was found on mammalian toxicity of benzo[g,h,i]perylene.

5.5.14 Indeno[1,2,3-c,d]pyrene

No information was found on mammalian toxicity of indeno[1,2,3-c,d]pyrene.

5.5.15 Dibenz[a,h]anthracene

Dickerson *et al*. (1994) exposed deer mice (juvenile males) to dibenz[a,h]anthracene via intraperitoneal injection over 11 days. A 50% reduction in immune response (ED50) was observed at a concentration of 0.048 $mg \cdot kg^{-1} \cdot day^{-1}$.

5.5.16 Summary

Several of the above-cited most sensitive endpoints (e.g., for acenaphthene, fluorene, and fluoranthene) are based on changes in the liver, presumed to be indicative of varying degrees of hepatotoxicity. The induction of foci of altered hepatocytes is often seen in rats and mice that also develop liver tumors. These foci have altered enzyme activities and higher rates of cell proliferation than normal hepatocytes.

Table 5-3: Daily Threshold Effect Doses (DTEDs) for PAHs in Vertebrate Species

PAH	Species	Endpoint ($mg \cdot kg^{-1} \cdot day^{-1}$)	Exposure Duration	Original Reference
Naphthalene	Fischer 344 rats	LOAEL (body weight): 143 $mg \cdot kg^{-1} \cdot day^{-1}$	5 days/week x 13 weeks	BCL, 1980.
Final THRESHOLD DOSE = LOAEL/5 = 143 $mg \cdot kg^{-1} \cdot day^{-1}$/(5) = 28.6 $mg \cdot kg^{-1} \cdot day^{-1}$				
Acenaphthene	CD-1 Mice	LOAEL (sub-chronic: hepato.): 350 $mg \cdot kg^{-1} \cdot day^{-1}$	13 wk: oral (gavage)	USEPA, 1989a
Final THRESHOLD DOSE = LOAEL/5 = 350 $mg \cdot kg^{-1} \cdot day^{-1}$/(5) = 70 $mg \cdot kg^{-1} \cdot day^{-1}$				
Acenaphthylene	*Limited data – no THRESHOLD DOSE calculated*			
Fluorene	CD-1 Mice	LOAEL (sub-chronic: hematological): 250 $mg \cdot kg^{-1} \cdot day^{-1}$	13 wk: oral (gavage)	USEPA, 1989b
Final THRESHOLD DOSE = LOAEL/5 = 250 $mg \cdot kg^{-1} \cdot day^{-1}$/(5) = 50 $mg \cdot kg^{-1} \cdot day^{-1}$				
Anthracene	Mice	NOAEL (subchronic): 1000 $mg \cdot kg^{-1} \cdot day^{-1}$	13 wk: oral	USEPA, 1989c

PAH	Species	Endpoint ($mg \cdot kg^{-1} \cdot day^{-1}$)	Exposure Duration	Original Reference
Final THRESHOLD DOSE = NOAEL/5 = 1000 $mg \cdot kg^{-1} \cdot day^{-1}$/(5) = 200 $mg \cdot kg^{-1} \cdot day^{-1}$				
Phenanthrene	Rat	LD50 (acute): 700 $mg \cdot kg^{-1} \cdot day^{-1}$		Eisler, 1987.
Final THRESHOLD DOSE = LD50/5 = 700 $mg \cdot kg^{-1} \cdot day^{-1}$/(5) = 140 $mg \cdot kg^{-1} \cdot day^{-1}$				
Fluoranthene	CD-1 Mice	LOAEL (nephrotox. and liver weight): 250 $mg \cdot kg^{-1} \cdot day^{-1}$	13 weeks: oral (gavage)	USEPA, 1988
Final THRESHOLD DOSE = LOAEL/5 = 250 $mg \cdot kg^{-1} \cdot day^{-1}$/(5) = 50 $mg \cdot kg^{-1} \cdot day^{-1}$				
Pyrene	CD-1 Mice	LOAEL (nephrotox.): 125 $mg \cdot kg^{-1} \cdot day^{-1}$.	13 weeks: oral (gavage)	USEPA, 1989d
Final THRESHOLD DOSE = LOAEL/5 = 125 $mg \cdot kg^{-1} \cdot day^{-1}$/(5) = 25 $mg \cdot kg^{-1} \cdot day^{-1}$				
Benz[a]anthracene	Mice	LOAEL (immuno-sup-pression): 100 $mg \cdot kg^{-1} \cdot day^{-1}$.	oral	Silkworth *et al.*, 1995
Final THRESHOLD DOSE = LOAEL/5 = 100 $mg \cdot kg^{-1} \cdot day^{-1}$/(5) = 20 $mg \cdot kg^{-1} \cdot day^{-1}$				
Chrysene	Mice	LOAEL (immune) 100 $mg \cdot kg^{-1}$ bw	Single oral dose	Silkworth *et al.* 1995
Final THRESHOLD DOSE = LOAEL/5 = 100 $mg \cdot kg^{-1} \cdot day^{-1}$/(5) = 20 $mg \cdot kg^{-1} \cdot day^{-1}$				
Benzo[b]fluoranthene	Mice	LOAEL (immune) 100 $mg \cdot kg^{-1}$ bw	Single oral dose	Silkworth *et al.* 1995
Final THRESHOLD DOSE = LOAEL/5 = 100 $mg \cdot kg^{-1} day^{-1}$/(5) = 20 $mg \cdot kg^{-1} day^{-1}$				
Benzo[k]fluoranthene	Mice	LOAEL (immune) 100 $mg \cdot kg^{-1}$ bw	Single oral dose	Silkworth *et al.* 1995
Final THRESHOLD DOSE = LOAEL/5 = 100 $mg \cdot kg^{-1} \cdot day^{-1}$/(5) = 20 $mg \cdot kg^{-1} \cdot day^{-1}$				
Benzo[a]pyrene	CD-1 mice	LOAEL (Reprod.): 10 $mg \cdot kg^{-1} \cdot day^{-1}$	10 days: oral (gavage)	Mackenzie and Angevine, 1981
Final THRESHOLD DOSE = LOAEL/5 = 10 $mg \cdot kg^{-1} \cdot day^{-1}$/(5) =2 $mg \cdot kg^{-1} \cdot day^{-1}$				
Benzo[g,h,i]perylene	*No data*			
Indeno[1,2,3-c,d]pyrene	*No data*			
Dibenz[a,h]anthracene	*Limited data – no THRESHOLD DOSE calculated*			

Foster Wheeler (1997) proposed wildlife toxicity reference values of relevance at sites contaminated with Total Petroleum Hydrocarbons (TPH), with a single toxicity threshold established to each of PHC-derived aliphatics varying in size from an effective boiling point range of C6 to nC18, and for aromatics from nC9 to nC31. Effects doses were

converted to their NOAEL equivalent and then normalized to the corresponding dose for a mouse or animal with similar body mass. The data from all studies were then ranked.

A 10^{th} percentile NOAEL of the aliphatics toxicity data, for a mouse, was estimated as 1.5 $mg \cdot kg^{-1} \cdot day^{-1}$, and this is deemed to be a reasonable terrestrial vertebrate TRV in the absence of better scientific knowledge. Similarly, the estimated 10^{th} percentile NOAEL for the aromatics toxicity data, for a mouse, was 0.6 $mg \cdot kg^{-1} \cdot day^{-1}$. This draft aromatics TRV is considerably lower than the DTEDs estimated above for the various PAHs.

5.6 Aquatic Organisms

There is an extensive body of scientific literature on the exposure of aquatic organisms to PAHs, and associated effects owing especially to problems associated with oil and fuel spills, urban stormwater runoff, and contaminated sediments in urbanized and industrialized areas. PAH-related cancers have been documented in both bottom-dwelling fish populations and, to a lesser extent, in resident invertebrate populations for species such as soft-shell clams.

The ECOTOX database contains an extensive number of laboratory toxicity data (> 500 individual data points) for PAHs and aquatic organisms. A review of these and other key studies is beyond the scope of this document.

Several studies have been carried out on the measurement of PAH metabolites in freshwater and marine animals as indicators of PAH environmental exposure (e.g., Luthe *et al*., 2002). Several studies have also been carried out on PAH-DNA adduct formation in marine and freshwater spp. (Shaw and Connell, 2001, provide a good review).

Kerr *et al.* (1999), like others, have developed a series of reporter gene bioassays for detecting potentially mutagenic substances, including PAHs, in the aquatic environment.

Di Toro *et al.* (2000), and Di Toro and McGrath (2000), developed models for predicting thresholds of toxicity in water and in sediment for non-polar compounds such as PAHs. The available studies indicate that aquatic organisms can be affected by PAHs based on cancer-related effects, through endocrine disruption, through photo-induced toxicity, and through a myriad of other effects mediated through PAH-molecular receptor interactions. Virtually all aquatic organisms, however, are likely to experience some “baseline” toxicity when exposed to PAHs, through what has been termed ‘Type I narcosis’. When an organism bioaccumulates hydrophobic organic contaminants to a sufficient level, the body burden (largely contained within lipid-rich structures such as cell membranes) can reach a concentration beyond which cell membrane functions become impaired. After a critical body burden is reached, the loss of cell membrane integrity results in narcosis; i.e., through a large number of theoretical mechanisms such as loss of membrane polarization and impaired function of membrane proteins.

Di Toro *et al.* (2000) collated the major portion of PAH LC_{50} data for aquatic organisms for the purpose of refining a “critical body residue” model for predicting threshold

toxicity levels. A total of 33 species including amphibians, fishes, arthropods (insect and crustacean), mollusks, polychaetes, coelenterates, and protozoans were represented in the toxicity data, which included not just experiments on PAHs but also on other chemicals with a type I narcosis mode of action. Based on a critical body residue approach, Di Toro *et al., (*2000) developed "final chronic values" (FCVs) for PAH concentrations in water that were protective of aquatic life, as follows:

- naphthalene: 320 $\mu g \cdot L^{-1}$
- acenaphthylene: 530 $\mu g \cdot L^{-1}$
- acenaphthene: 95 $\mu g \cdot L^{-1}$
- fluorene: 66 $\mu g \cdot L^{-1}$
- anthracene: 36 $\mu g \cdot L^{-1}$
- phenanthrene: 32 $\mu g \cdot L^{-1}$
- pyrene: 17 $\mu g \cdot L^{-1}$
- fluoranthene: 12 $\mu g \cdot L^{-1}$
- benz[a]anthracene: 3.8 $\mu g \cdot L^{-1}$
- chrysene: 3.5 $\mu g \cdot L^{-1}$
- benzo[b]fluoranthene: 1.1 $\mu g \cdot L^{-1}$
- benzo[a]pyrene: 1.6 $\mu g \cdot L^{-1}$
- dibenz[a,h]anthracene: 0.6 $\mu g \cdot L^{-1}$

The Canadian water quality guidelines for the protection of aquatic life have been developed using ecotoxicity data that includes mechanisms other than non-polar narcosis (e.g., photo-induced toxicity) (Table 5-4). The Canadian water quality guidelines are between 16 and 3,000-fold lower than the Final Chronic Values of Di Toro *et al.*, (2000).

Table 5-4: Summary of Existing PAH Canadian Water Quality Guidelines for the Protection of Aquatic Life

Parameter	Guideline ($\mu g \cdot L^{-1}$)	LOEL ($\mu g \cdot L^{-1}$)	Safety Factor Applied	Endpoint	Comment
Naphthalene	1.1*	11	10	Chronic LOEL for survival in rainbow trout	LOEL is geometric mean of 8 and 15
Acenaphthene	5.8	580	100	96-h LC_{50} for brown trout	32-35d LOEL for growth of fathead minnow embryos is 495 $\mu g \cdot L^{-1}$; LOEL not used because of low acute threshold
Fluorene	3.0	125	10	14-d LOEL for reproduction in *D. magna*	LOEL multiplied by an additional factor of 0.24 to correct for a difference in nominal vs. measured concentration
Anthracene	0.012	1.2	100	0.25-h LT_{50} for *D. pulex* (phototoxicity)	
Phenanthrene	0.4	4	10	Chronic LOEL for survival in rainbow trout	
Pyrene	0.025	2.5	100	LC_{50} for	

Parameter	Guideline ($\mu g \cdot L^{-1}$)	LOEL ($\mu g \cdot L^{-1}$)	Safety Factor Applied	Endpoint	Comment
				mosquito larvae (phototoxicity)	
Fluoranthene	0.04	4	100	1-h LC_{50} for *D. magna* (phototoxicity)	
Chrysene	NRG	0.7	N/A	24-h LC_{50} for *D. magna* (photoxicity)	Minimum data requirements not met.
Benz[a]anthracene	*0.018 (interim)*	1.8	100	65-h LT_{50} for fathead minnows (phototoxicity)	Minimum data requirements not met, but interim guideline derived
Benzo[a]pyrene	0.015	1.5	100	4-h LC_{50} for *D. magna* (phototoxicity)	

*** interim value for marine life protection is 1.4 $\mu g \cdot L^{-1}$. Marine life guidelines have not been developed for PAHs other than naphthalene.**

6. HUMAN AND MAMMALIAN UPTAKE, PHARMACOKINETICS, AND EFFECTS

6.1 Human Exposure Estimates

Total daily PAH intake for humans were estimated by de Kok and van Maanen (2000) to vary between 25 to 300 $\mu g \cdot day^{-1}$, excluding those individuals who are also occupationally exposed. Total daily PAH intake for humans for the general American populace was estimated by Santodonato *et al*. (1981) to vary from 0.2 to about 20 $\mu g \cdot day^{-1}$. It is worth noting the span in such estimates and the high degree of uncertainty, as well as questions about the applicability to the Canadian populace. The general population is primarily exposed via consumption of food and as a result of cigarette smoking. Charbroiled grilling and smoked meats may be a substantial source of PAH exposure in some human populations. Depending on an individual's lifestyle, the life-long intake (i.e., 70-78 years) of benzo[a]pyrene for non-occupationally exposed humans may add up to 29 mg, integrating respiratory, gastrointestinal and percutaneous absorption.

6.2 Pharmacokinetics

The following information is derived from detailed reviews contained in WHO/IPCS (1998) and ATSDR (1995c). PAHs may be absorbed through the gastrointestinal (GI) tract, the pulmonary tract, and/or the skin.

Some studies suggest that the gastrointestinal absorption of benzo[a]pyrene following ingestion of soils is low in humans, while other studies indicate that approximately 90 to 99% of B[a]P can be absorbed following oral ingestion (reviewed by Magee *et al.* 1996). In general, oral absorption in animals varies among the PAH compounds depending on the lipophilicity. Oral absorption may increase with the presence of oils in the GI tract, and the more lipophilic PAHs will tend to more strongly partition into lipid; however, resistance to desorption from more refractory non-polar substances in the diet may offset this.

Tang *et al.* (2006) used physiologically based in vitro tests to evaluate the oral bioaccessibility of PAHs in soils collected from a variety of public sites in Beijing. Soil PAH concentrations ranged from 0.112 $mg \cdot kg^{-1}$ to 27.8 $mg \cdot kg^{-1}$. Oral bioaccessibility of total PAHs ranged from 9.2 % to 60.5 % in small intestinal simulation and 3.9 % to 54.9 % in gastric simulation. Van de Wiele *et al.* (2004) also used physiologically based in vitro tests to evaluate the bioaccessibility of PAHs in soil from a recreational area under simulated high soil ingestion rates. The total PAH concentration in soil was 49 $mg \cdot kg^{-1}$. The percentage of total PAHs released from the soil matrix ranged from 0.1 % to 1.4 %. The bioaccessibility of benzo(a)pyrene and phenanthrene in lampblack-contaminated soils from manufactured gas plant sites has been evaluated using in vitro (simulated gastrointestinal system) and in vivo (mouse) test methods (Stroo *et al.*, 2005). The percentage of benzo(a)pyrene solubilized in the simulated gastrointestinal system varied

from 0.5 % to 5.0 %, while that of phenanthrene ranged from 0.8 % to 15.0 %. In vivo test results indicate a range of 0.6 % to 1.1 % for the uptake of phenanthrene in mice six hours after a gavage treatment.

Percutaneous absorption of PAHs appears to be rapid for both humans and animals, but absorption of PAHs following inhalation, oral, or dermal exposure may be affected by vehicle of administration.

There was no information available on the distribution of PAHs in humans. PAHs and their metabolites are distributed to tissues by transport through the blood. Therefore, PAHs reach more-perfused tissues rapidly following exposure and are eliminated more slowly from less perfused tissues (Bartosek *et al.*, 1984). A large fraction of orally absorbed benzo[a]pyrene is believed to be transported by lipoproteins from the gastrointestinal tract to the blood via the thoracic duct lymph flow (Busbee *et al.*, 1990).

PAHs appear to be widely distributed in tissues of animals following oral and inhalation exposure, with the relative concentrations varying according to degree of lipid content. The equilibrium partition based on lipophilicity, however, is offset by non-equilibrium conditions during active uptake as well as through the removal of parent PAHs through metabolic modification, with potentially different rates of cytochrome P450 oxidase enzyme activity in different tissues and organs. Peak tissue concentrations tend to occur earlier with higher exposure levels.

Placental transfer of PAHs appears to be limited, and therefore fetal levels are not as high as maternal levels.

Metabolism of PAHs, as discussed in Chapter 2, occurs in all tissues in the majority of animal species examined and involves several possible pathways. PAH metabolism has been studied extensively both *in vitro* and i*n vivo*. The metabolism products include epoxide intermediates, dihydrodiols, phenols, quinones, and their various combinations. The phenols, quinones, and dihydrodiols can all be conjugated to glucuronides and sulfate esters; the quinones also form glutathione conjugates.

More limited information is available on PAH metabolism in non-mammalian groups with the exception of microbes, based on a strong interest in contaminant biodegradation mechanisms in soil, sediment and water.

In general, feces are the major elimination route of PAHs in animals following inhalation exposure. Excretion of benzo[a]pyrene appears to be high following low-level exposure in rats but low in dogs and monkeys. PAHs are eliminated to a large extent within 2 days following low- and high-level oral exposure in rats. Following dermal exposure, elimination of PAHs occurs rapidly in the urine and feces of guinea pigs and rats.

Using human cadaver skin, it was shown that 23.7±9.7% of the applied benzo[a]pyrene penetrated into the skin (Wester *et al.*, 1990). These results suggest that substantial

metabolism and/or binding of benzo[a]pyrene takes place in viable human skin, therefore limiting the amount of PAH available to penetrate the skin into the systemic circulation.

More recently, Shatkin et al. (2002) developed a fugacity-based model to evaluate the availability of polycyclic aromatic hydrocarbons (PAHs) from soil to human skin. The model (adapted from McKone and Howd, 1992) was based on a rapidly desorbing and slowly desorbing soil compartment. Modelled predictions of dermal availability were validated by comparing with experimentally derived estimates (previously published) using radiolabelled PAHs and 350 μm skin held in vitro on a Franz diffusion cell. In most cases, the modeled percent absorption predicted the empirically derived estimates within one standard deviation of the mean modeled estimate, and the empirically derived dermal absorption estimated agreed with the modeled results very well (within a factor of two or less). Shatkin et al. (2002) provided modeled predictions of dermal uptake fraction assuming worst-case conditions for soil organic carbon content, soil contact time, soil moisture and other input parameters. They predicted that a PAH-contaminated soil with an organic carbon fraction (f_{oc}) of 2.4% would result in less than 35% of the phenanthrene being dermally absorbed. The percent dermal absorption theoretically will be much higher for lower K_{OW} PAHs such as acenaphthene, anthracene, phenanthrene, fluoranthene, naphthalene, pyrene and fluorene, than for higher K_{OW} PAHs such as benzo(a)pyrene and benz(a)anthracene. Sartorelli et al. (1999) provided experimental results that support this view.

The retention versus elimination of PAHs in mammalian species is especially of importance for chronic exposures, yet there is little relevant information available. It appears that PAHs themselves do not persist for long periods and must therefore turn over reasonably rapidly, primarily through metabolism to other compounds. During metabolism, however, some metabolites can become covalently bound to tissue constituents such nucleic acids (DNA and RNA) and proteins. Protein-bound metabolites are likely to persist for periods that do not exceed the normal lifetime of the protein itself. RNA and DNA adducts are expected to have different rates of persistence. Most DNA adduct are removed relatively rapidly by excision and repair, although small fractions can persist for long periods.

The persistence of non-repaired adducts in tissues, such as mouse skin, is of considerable interest (WHO/IPCS, 1998) since one of the basic features of the two-stage mechanism of carcinogenesis (Berenblum & Shubik, 1947) is that application of the tumour promoter can be delayed for many months without markedly reducing the eventual tumour yield.

6.3 Toxicity

6.3.1 CYP-induced carcinogenic activation

PAHs with two or three aromatic rings (for example, naphthalene, acenaphthene, acenaphthylene, fluorene, anthracene, phenanthrene) have been shown consistently to have no or very limited tendency to bind to the Ah-receptor, and to induce CYP enzymes

such as EROD (*ethoxyresorufin-O-deethylase*) (Bosveld *et al.,* 2002). It is suggested that these lighter molecular weight PAHs do not meet the structural requirements for having an affinity to bind to the Ah-receptor. Bosveld and other researchers (reviewed in Bosveld *et al.,* 2002) found that benzo[k]fluoranthene was among the most potent inducers of EROD or reporter gene activity *in vitro.*

Chapter 2 provides a detailed description of mechanisms of PAH human health effects as a result of interaction with the Ah-receptor, and induction of especially CYP1A1.

6.3.2 Acute Toxicity

According to the World Health Organization (WHO/IPCS, 1998) there have been a limited number of studies in which human subjects voluntarily undertook exposures to PAHs. Following dermal application, anthracene, fluoranthene, and phenanthrene induced specific skin reactions, and benzo[a]pyrene induced reversible, regressive skin abnormalities that were classified as neoplastic proliferations.

Recorded incidents of accidental intake in humans, especially in children, provide limited information on acute, systemic toxicity.

Naphthalene appears to exhibit a lethal oral dose of 5,000-15,000 mg for adults (ca. 70 to 210 $mg \cdot kg^{-1}$ for a 70 kg adult) and 2,000 mg taken over two days for a child (ca. 125 $mg \cdot kg^{-1}$, assuming a 16 kg toddler). The typical effect after dermal or oral exposure is acute haemolytic anaemia, which can also affect fetuses transplacentally. By comparison, acute LD_{50} data for laboratory animal exposures for naphthalene, based on oral exposures, are in the range of 350 to 9,400 $mg \cdot kg^{-1}$.

Effects induced in animals following generally acute exposure to high concentrations of benzo[a]pyrene include inflammation of the skin, hyperplasia, hyperkeratosis, pneumonitis, modifications of the lymph nodes, ulceration, reduction in growth and fertility rates and the induction of immunosuppressive effects (CCME, 1999b).

6.3.3 Sub-chronic and Chronic Toxicity (other than Cancer)

While there is no relevant information available for humans, the potential for human health effects can be appreciated to a limited extent by laboratory mammal studies. The reader is directed to WHO/IPCS (1998) as well as ATSDR (1995c) for a more exhaustive review.

Dibenzo[a,h]anthracene injected subcutaneously in male rats 5 days/week x 4 weeks resulted in various haemolymphatic changes, including the appearance of blood cells in lymph spaces, along with the abnormal presence of large pigmented cells. Dogs exposed one time to naphthalene also temporarily became anemic. Sub-chronic exposure of mice to acenaphthene has been shown to result in hepatotoxicity. The growth of rats was

inhibited by feeding a diet enriched with benzo[a]pyrene at 1,100 $mg \cdot kg^{-1}$ for more than 100 days.

Fluorene has been observed to result in hypoactivity in orally exposed mice, along with a decrease in erythrocyte count, decreased haemoglobin concentration, and increase in liver, spleen and kidney weight. Similar signs have been observed in mice exposed to fluoranthene (USEPA, 1988).

Rabbits exposed by subcutaneous injection at 0.1-1 $mg \cdot kg^{-1}$ (b.w.) per day naphthalene for 123 days experienced severe oedema and a high degree of vacuolar and collicular degeneration in the brain; necrosis of nerve cells also occurred. The author suggested that hypoxaemia resulting from haemolytic anaemia was responsible for this finding (WHO/IPCS, 1998).

Overall, sub-chronic exposures to PAHs have been associated with reduced growth rates, hepatotoxicity, cataracts (in rabbits) and, especially, haematological changes. Few, if any, immunological effects have been observed.

Virtually all studies on long-term chronic exposures to PAHs were designed to assess carcinogenic endpoints, and have little value therefore for assessing the potential for long-term chronic effects in human populations other than through increased cancer risk. One study documented the deposition of iron in lymph glands after long-term exposures of mice (weekly subcutaneous injections of 0.25 mg over 40 weeks) to anthracene, benz[a]anthracene, or dibenz[a,h]anthracene. Few other effects were noted.

6.3.4 Genotoxicity and Carcinogenicity

The genotoxicity and carcinogenicity of PAHs have been addressed in Chapter 2 of this report, and are explored in more detail in Section 6.6.

6.3.5 Reproductive Effects, Embryotoxicity, and Teratogenicity

No direct information exists on the reproductive toxicity of PAHs to humans or of teratogenic effects. Laboratory rodent studies, however, have been reported for anthracene, benz[a]anthracene, benzo[a]pyrene, chrysene, dibenz[a,h]-anthracene, and naphthalene.

According to the World Health Organization (WHO/IPCS, 1998) embryotoxicity has been reported in response to exposure to naphthalene, benz[a]anthracene, benzo[a]pyrene, and dibenz[a,h]anthracene.

The lowest dose associated with embryotoxicity for each of these four PAHs is as follows:

Benz[a]anthracene

Female rats injected subcutaneously on days 1 through 15 of gestation at a rate of 5 mg/animal/day experienced intraplacental hemorrhaging, followed by fetal death and resorption occurring up to day 18 (Wolfe and Bryan, 1939; reported in WHO/IPCS, 1998).

Benzo[a]pyrene

Pregnant female mice fed diets containing 120 $mg \cdot kg^{-1}$ B[a]P/day for days 2 through 10 of gestation experienced increased intrauterine toxicity, as well as malformations in the embryos (Legraverend *et al*, 1984; as reported in WHO/IPCS, 1998). Pregnant female rats subjected to subcutaneous injection of benzo[a]pyrene at 5 mg/animal/day on days 1 through 11 or 16 of gestation experienced profuse vaginal hemorrhaging, intraplacental hemorrhaging on day 14, and fetal death up to day 18 (Wolfe and Bryan, 1939; reported in WHO/IPCS, 1998).

Dibenz[a,h]anthracene

Pregnant female rats dosed subcutaneously on days 1 through 8 or 18 of gestation at 5 mg/animal/day exhibited intraplacental hemorrhaging, with fetal death and resorption (Wolfe and Bryan, 1939, in ATSDR, 1995),

Figure 6-1, from WHO/IPCS (1998) summarizes the postulated mechanism of ovotoxicity of PAHs.

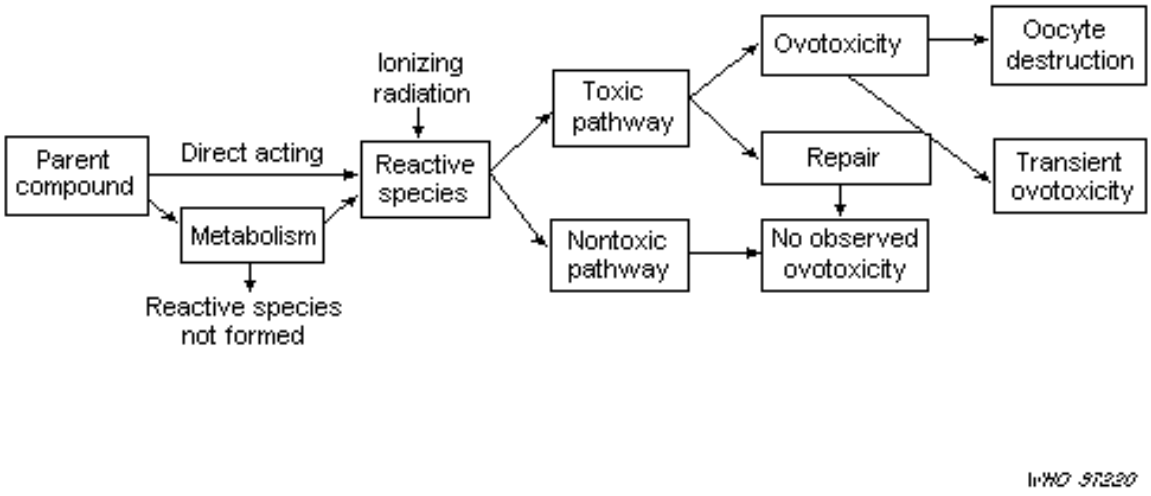


Figure 6-1: Proposed mechanism of ovotoxicity
(from WHO/IPCS, 1998)

Postnatal effects of benzo[a]pyrene on mouse offspring have been investigated in three studies, based on either oral, intraperitoneal or dermal exposure of the mother. Adverse effects included an increased incidence of tumours, immunological suppression, and reduced fertility (WHO/IPCS, 1998).

An oral dose study was conducted by Mackenzie and Angevine (1981). CD-1 mice were exposed via gavage at doses of 0, 10, 40 and 160 $mg \cdot kg^{-1}$ (b.w.) per day during days 6 through 17 of gestation. The lower dose (10 $mg \cdot kg^{-1}$ (b.w.) per day) markedly impaired fertility and reduced testis weight in the progeny of the exposed mice.

6.3.6 Neurotoxicity

No information was found on PAH neurotoxicity.

6.3.7 Effects on Immunocompetence

Immunotoxicity of benzo[a]pyrene has been observed in at least four laboratory rodent studies. Immunosuppressive effects of dibenz[a,h]anthracene in whole organism, laboratory rodent studies have also been observed. Conversely, immunotoxicity has not been observed in experiments using naphthalene, even though naphthalene is known to induce renal and pulmonary toxicity (WHO/IPCS, 1998).

6.3.8 Endocrine Disruption

As mentioned in Chapter 1, some PAHs have been labeled as endocrine disrupting substances by toxicologists (Clemons *et al.,* 1998; Safe *et al.*, 1997; Santodonato, 1997). However, there are few epidemiological or whole animal studies that have examined links between PAH exposure and endocrine-related toxicity endpoints. The major evidence for the role of PAHs and endocrine-disrupting substances is based on their ability to bind to endogenous receptors that mediate endocrine response pathways, mostly based on *in vitro* assays. The primary evidence that PAHs behave as endocrine disruptors is based on their role as agonists to the cytosolic Ah-receptor (AhR). AhR-PAH associations are discussed in Chapter 2 in the context of cytochrome P450 oxidase induction and carcinogenic activation. Since there may be several functional implications of Ah-receptor and PAH interactions, it is probably premature to label PAHs as endocrine-disruptors.

Band *et al., (*2002) conducted an epidemiological study of the link between smoking and breast cancer, based on surveys of 1,431 women under 75 years of age and listed on the British Columbia cancer registry in 1988-89. This cohort was compared with 1,502 age-matched individuals selected from the 1989 voter's list. The results of the Band *et al., (*2002) study indicated that susceptibility to breast cancer among smokers differs between pre- and post-menopausal women, and that susceptibility was higher when the start of smoking occurred within 5 years of the onset of menarche.

Various other investigators have previously postulated that the risk of breast cancer in women who smoke may be higher due to greater exposures to carcinogens. Results from a variety of studies, however, have been equivocal. A positive association has been observed in some but not a clear majority of studies. Band *et al., (*2002) proposed that the potentially competing carcinogenic and anti-estrogenic properties of cigarette smoke could account for the discrepant results across studies.

It is known from animal studies that the susceptibility of breast tissue to carcinogenesis is dependent on the stage of cell differentiation; with susceptibility being greatest around the time that ovarian function begins. Overall, various studies suggest that human breast tissue is more prone to cancer when exposed to carcinogens during periods of rapid cell proliferation (e.g., during puberty) and when differentiation is incomplete. This may have implications for human sensitivity to environmental carcinogens encountered via other exposure routes as well, although few if any studies are available which address this issue.

6.4 Epidemiological Studies of PAH Exposure in Human Populations

Several epidemiological studies have been conducted of occupationally exposed human sub-populations. For example, researchers have examined workers exposed at coke ovens during periods of coal coking and coal gasification, at asphalt works, foundries, and aluminum smelters, and to diesel exhaust.

Workers from aluminum smelters employing Söderberg potrooms, coke-oven workers, and asphalt workers were found to have significantly elevated lung tumour rates. The highest risks so far observed have been for coke-oven workers, with a standardized mortality ratio of 195 (WHO/IPCS, 1998). Aluminum plant workers where the Söderberg electrode process has been used have had significantly elevated urinary bladder cancer, asthma-like symptoms, lung function abnormalities, and chronic bronchitis. Coke-oven workers were found to have decreased serum immunoglobulin levels and decreased immune function.

Occupational exposure to naphthalene for five years was reported to have caused cataracts.

The epidemiological evidence of increased cancer in occupationally exposed workers has in many cases been accompanied by corroborating dose-response data, as well as exposure biomarker data. For example, workers from coke plants, aluminum manufacturing, wood impregnation plants, foundries, and asphalt plants exhibited increased 1-hydroxypyrene excretion or DNA adducts. Coke-oven workers and workers impregnating wood with creosote, have been among the worst-case examples of occupationally exposed individuals based on exposure biomarkers. Creosote applicators may bioaccumulate up 95% of total of PAH through the skin, in contrast to the general

population whom uptake predominantly via food and tobacco smoking (WHO/IPCS, 1998).

According to the World Health Organization (IPCS/WHO), 1998):

> "Estimates of the risk associated with exposure to PAHs and PAH mixtures are based on estimates of exposure and the results of epidemiological studies. Data for coke-oven workers resulted in a relative risk for lung cancer of 15.7. On this basis, the risk of the general population for developing lung cancer over a lifetime has been calculated to be 10^{-4} to 10^{-5} per ng of benzo[a]pyrene per m^3 air.
>
> In other words, about one person in 10,000 or 100,000 would be expected to develop lung cancer in his or her lifetime as a result of exposure to benzo[a]pyrene in air."

6.5 Information on the Carcinogenicity of PAH-Containing Mixtures

The International Agency for Research on Cancer (IARC) has evaluated the carcinogenicity of several PAH-containing complex mixtures. A summary of the IARC evaluations is provided in Table 6-1 (after Boström *et al.,* 2002). One possible means of developing generic soil quality guidelines based on carcinogenicity for such complex mixtures would be to use data for the complex mixture of concern. The carcinogenicity of several complex mixtures has been evaluated in detail by ATSDR, and a summary for each is provided below.

The toxicology and carcinogenicity of some PAH-containing mixtures such as diesel fuel, fuel oils and gasoline has been evaluated by regulatory agencies and institutes, including the US Agency for Toxic Substances and Disease Registry (ATSDR, 1995a, 1995b), the International Agency for Research on Cancer (IARC, 1987, 1989), and the World Health Organization International Programme on Chemical Safety (WHO/IPCS, 1982, 1998).

Unfortunately, no Canadian or other international agency, including ATSDR, has developed cancer slope factors for any of the potentially carcinogenic PAH-containing mixtures based on oral exposures. The California Environmental Protection Agency (OEHHA: Office of Environmental Health Hazard Assessment) has developed an inhalation slope factor for diesel exhaust; i.e., $(1.1\ mg \cdot kg^{-1} \cdot d^{-1})^{-1}$. The USEPA has developed an inhalation unit risk value for coke oven emissions of 6.2×10^{-4} $(g \cdot m^{-3})^{-1}$. Neither of these two inhalation-based values are of relevance for developing generic soil quality guidelines for potentially carcinogenic PAHs. After extensive review, therefore, the use of data for representative mixtures was ruled out for the development of cancer-based soil quality guidelines. These assessments were nevertheless useful for assessing non-cancer type responses to such mixtures.

Other aspects that argue against the use of mixtures data is that the laboratory toxicity data that are available are based on dosages expressed primarily as mass/unit of the whole

product, not the constituent higher molecular weight PAHs. Conversion of such data to PAH-equivalent concentrations, therefore, would in each case require a parallel estimate of percent of the mixture comprised of PAHs.

Table 6-1: Summary of IARC evaluations of PAH-containing complex mixtures and exposure scenarios

Group 1 Mixtures: Carcinogenic to Humans • Coal tars • Coal tar pitches • Mineral oils – untreated • Mineral oils – mildly treated • Shale oils • Soots • Tobacco smoke • Aluminum production • Coal gasification • Coke production	**Group 2b Mixtures: Possibly Carcinogenic to Humans** • Bitumen extracts • Carbon black extracts • Diesel fuels – marine • Fuel oils – heavy • Gasoline • Gasoline exhausts
Group 2a Mixtures: Probably Carcinogenic to Humans • Creosotes • Diesel exhausts • Petroleum refining	**Group 3 Mixtures: Not Classifiable** • Bitumen • Carbon black • Coal dust • Crude oil • Diesel fuels – light • Fuel oils – light • Jet fuel • Mineral oils – highly refined • Petroleum solvents

Furthermore, many of these mixtures, including coal tars, coal tar creosotes, and mineral oils tend to have a highly variable PAH contribution depending on the original source material, heat and conditions of the various processes, and presence of other substances in the mixture during PAH *de novo* production or partitioning from the native source.

6.5.1 Coal Tars and Coal Tar Creosotes

Creosote is a common name for a variety of products. Wood creosote, for example, may be derived from resins extracted from the leaves of the creosote bush (*Larrea* spp.) or beechwood (*Fagus* spp.). Wood creosotes tend to be made up of relatively simple phenolics, including phenol, methyl-phenols and guaiacols.

Of much greater interest in the context of carcinogenic PAHs are “coal tar creosotes” (distillation products of coal tar), and coal tar pitch (a residue produced during the distillation of coal tar). Neither tends to be produced naturally. Especially in the past, coal tar residues could be found within chimneys of houses and factories heated with or having boilers or foundries fueled by coal.

Coal tar creosote is among the most widely used wood preservative and general-purpose sealant in North America. In Canada, an assessment of coal tar creosotes was produced in 1993 under the Canadian Environmental Protection Act Priority Substances List program (Environment Canada, 1993).

Coal tars and coal tar creosotes are complex combinations of polycyclic aromatic hydrocarbons (PAHs), phenols, heterocyclic oxygen, sulfur, and nitrogen compounds, typically with variable PAH concentrations and compositions. The coal tar creosotes consist primarily of PAHs and PAH derivatives; most coal tar creosote mixtures are made up of 75% or more by weight of PAHs.

Acute toxicity of coal tar was assessed by Hackett *et al. (*1984) in CD rats exposed over a five-day period. A gavage dose of 740 $mg \cdot kg^{-1} \cdot day^{-1}$ resulted in the death of 10 of 16 animals. Pfitzer *et al. (*1965) observed an LC_{50} in Wistar rats based on a one-time orally administered dose of 1,700 $mg \cdot kg^{-1}$ (b.w). There have been several reported cases of death in large farm animals associated with the ingestion of coal tar creosote (as reported in ATSDR, 2002, and based on references cited therein); however, the effective dose generally could not be ascertained in those studies.

Among the best available chronic toxicity data for mammals was produced by Culp *et al.* (1996, 1998). These researchers exposed B6C3F1 mice (14 dosage groups times 48 mice/group) to coal tar through a gel diet for two years (Culp *et al.*, 1998). One coal tar sample was derived from a mixture from seven manufactured gas plant (MGP) waste sites and another was from two sites included in the first mixture plus samples from a third site with very high benzo[a]pyrene content. Three groups of mice received feed with the following concentrations of benzo[a]pyrene; 5, 25, and 100 $mg \cdot kg^{-1}$ feed. Estimated oral doses for the second mixture were approximately 12, 33, 117, 333, 739, or 1300 $mg \cdot kg^{-1} \cdot day^{-1}$ of coal tar 1 and 40, 120, or 346 $mg \cdot kg^{-1} \cdot day^{-1}$ (0.03, 0.1, and 0.3%) of coal tar 2. Dietary levels of 333 $mg \cdot kg^{-1} \cdot day^{-1}$ coal tar 1 or 346 $mg \cdot kg^{-1} \cdot day^{-1}$ coal tar 2 produced a significant increase in early mortality compared with controls, while lower levels did not.

There appear to be no direct studies on the association between cancer in humans and the ingestion of coal tar or coal tar pitch (ATSDR, 2002). As part of the Culp *et al.* (1996) study, forestomach tumours were found in all three groups of mice fed a benzo[a]pyrene-amended diet, and the incidence increased with dose. In mice fed coal tar residues from MGP waste sites, however, forestomach tumours were not observed, while tumours of the small intestine were observed only in groups fed 0.6 or 1.0% coal tar.

As part of the Culp *et al.* (1998) study, mice fed coal tar 1 or 2 exhibited a significant concentration-related increase in incidence of neoplasms of the liver, lung, and forestomach and of hemangiosarcomas, histiocytic sarcomas, and sarcomas. Mice treated with coal tar 1 also showed a significant concentration-related increase in incidence of neoplasms of the small intestine. In contrast mice treated with benzo[a]pyrene had a significant concentration-related increase in incidence of neoplasms of the forestomach, esophagus, tongue, and larynx. The lowest coal tar dose associated with an increased incidence of forestomach tumours was 200 $mg \cdot kg^{-1} \cdot day^{-1}$ (61% of exposure group) (Culp *et al.*, 1996) while the lowest dose associated with increased incidence of various other neoplasms was 333 $mg \cdot kg^{-1} \cdot day^{-1}$ (Culp *et al.*, 1998).

The authors felt that the forestomach tumours in coal-tar exposure oral groups might have been due to the benzo[a]pyrene in the mixtures, but that other tumours were probably associated with the presence of other genotoxic compounds in coal tar. The incidence of small intestine tumours was speculated to be due to chemically induced cell proliferation (i.e., mitogenesis) as opposed to mutagenesis.

This research, coupled with follow-up studies by Gaylor *et al.* (2000) and Goldstein *et al.* (1998) suggest that the estimation of cancer potency in coal tar risk assessments based entirely on benzo[a]pyrene concentration is inappropriate when dealing with coal tar contaminated sites. Schneider *et al*. (2002) as cited by Ramesh *et al*. (2004) concluded that the potency of B[a]P equivalents will underestimate the potency of PAH mixtures.

There is clear-cut epidemiological evidence for an association between occupational exposures to coal tar or creosote and increased incidence of skin cancers in humans (ATSDR, 2002). Effects in occupationally exposed groups have included the development of dermatoses (e.g., squamous papillomas), which progressed to carcinoma, usually squamous-cell carcinoma. Coal tar has also been demonstrated to induce an increased incidence of cancer in mice based on dermal exposure (Niemeier *et al*., 1988; WHO/IPCS, 2004). In the WHO evaluation, carcinogenic studies in mice revealed that creosote mixtures were about 5 times more potent than expected based on B[a]P content (WHO/IPCS, 2004). For these studies, as for the oral studies, it is challenging to express the dose on the basis of the concentrations of individual or total PAHs.

ATSDR/IRIS have labeled coal tars and coal tar creosotes as probable human carcinogens (type B1). Quantitative estimates, however, of the carcinogenic potential of coal tar and coal tar creosote based either on oral, inhalation, or dermal exposures, have yet to be produced by Health Canada, IRIS, ATSDR, or WHO/IPCS.

6.5.2 Used Mineral-Based Crankcase Oil (after ATSDR, 1997)

Mineral crankcase oils are synthesized from highly refined crude oils and contain branched and straight-chain aliphatic and aromatic hydrocarbons, including PAHs. Used mineral crankcase oils may also contain various metals (from engine wear or additives), gasoline, solvents, detergents, and other additives. Additives can comprise up to 20% of the total volume of new lubricating oils. Van Donkelaar (1990) described approximately

one hundred and forty different PAHs in used crankcase-lubricating oils, at higher concentrations than in new or fresh oil. The PAH composition and concentration of mineral-based lubricating oils tends to be highly variable, since thermal decomposition is accompanied by the production of newly formed PAHs, and the particulars of production depend on combustion conditions, presence of precursors, and presence of other additives (Bingham, 1988; Ingram *et al.*,1994).

ATSDR (1997) was consulted especially for toxicity and epidemiological studies based on oral and dermal exposures to crankcase oils containing PAHs. No studies were available on human deaths following oral exposures to used crankcase oil. Osweiler *et al.,* (1973) reported increased mortality among cattle believed to have ingested discarded used mineral-based crankcase oil; however, tissue lead levels were elevated in the cattle, and toxic symptoms observed in the affected animals were attributed to ingestion of lead contained in the used oil.

Few if any studies are available on effects on growth, reproduction, or other endpoints from acute, chronic, or sub-chronic exposures to new or used crankcase oil in animals. In particular, no studies were located regarding cancer in humans or animals after oral exposure to used mineral-based crankcase oil.

Four separate studies have been conducted on the dermal exposure of mice and cancer. Several studies have examined the dermal carcinogenicity of used mineral-based crankcase oil in mice (API 1983; Grimmer *et al.*, 1982a, 1982b, 1983; McKee and Plutnick 1989). These studies have shown that the incidence of dermal papillomas and carcinomas among mice is higher after chronic exposure via the dermis to used mineral-based crankcase oil from gasoline-powered cars. No tumours were observed in mice exposed to unused motor oil (API 1983; McKee and Plutnick 1989), whereas the greatest tumor incidence was observed in mice exposed to oil from cars driven the longest distances prior to removing the oil (McKee and Plutnick 1989). The tumor incidence was correlated with PAH content of the oil.

Fractionation of the oil showed tumor induction only with the fraction containing PAHs with more than three rings (Grimmer *et al.*, 1982a, 1982b, 1983). In contrast to used mineral-based crankcase oil from gasoline-powered automobiles, used mineral-based crankcase oil from diesel-powered automobiles showed no increase in tumor incidence, even when the diesel-powered automobiles were driven extremely long distances prior to removal of the oil (McKee and Plutnick 1989).

Cancer effects (dermal papillomas and carcinomas) were observed by Grimmer *et al.* (1982a,b) when CFLP mice were dermally exposed to used crankcase oil twice per week over a 104-week period at a rate of 60 $mg \cdot kg^{-1}$ (b.w).

Overall, the composition and carcinogenicity of crankcase oils is expected to be highly variable, and depends on the extent of prior use in an internal combustion engine before release to the environment. For this reason, and in light of the very limited toxicological information on used crankcase oil, it was concluded that risk of PAH-containing mixtures

in soils could not be addressed based on whole mixture human health or ecotoxicity data for used crankcase oil.

6.6 Critical Evaluation of the Comparative Carcinogenicity of Individual PAH

With the exception of coal tar, whole mixture toxicity data are not generally available to develop environmental quality guidelines based on primarily oral and dermal exposures to PAH-containing mixtures. Therefore, the potential for use of comparative toxicity/potency estimates for PAH substituents, based on current knowledge of structure-activity relationships was examined. The theoretical understanding of some aspects of carcinogenesis is very good indeed, and there are several excellent papers on PAH structure-cancer activity relationships.

A more detailed review of the contemporary literature on PAH quantitative structure-activity relationships (QSAR) is provided in sub-section 6.6.1, below.

6.6.1 Quantitative-Structure Activity Relationships (QSAR) as Predictors of Comparative and Cumulative Toxicity

In general, PAHs with a bay or fjord region (Chapter 2) are more active as mutagens and carcinogens than those PAHs without a bay region. Furthermore, methylation of the PAH at specific positions around the ring structure may either increase or decrease the carcinogenic potential of PAHs (Boström *et al.*, 2002). The methylation of the 5-position of chrysene (i.e., 5-methylchrysene) greatly potentiates mutagenic and carcinogenic effects relative to chrysene, and 5-methylchrysene is a model carcinogen that is commonly used in cancer studies, with a potency that is generally greater than benzo[a]pyrene.

For a PAH to act as a promoter, there appears to be a requirement for interaction with the Ah-receptor (Poland *et al*, 1982). Specific mechanisms of Ah-receptor mediated tumour promotion have been hypothesized to be based on activation of genes transcribed simultaneously with genes coding for cytochrome P450 enzymes, or as a result of the activation of tyrosine kinase and the subsequent phosphorylation of growth factors and hormones (Enan and Matsumura, 1996; as cited in Boström *et al.*, 2002).

In order for a substance to exhibit binding affinity with the Ah-receptor, it must fit into a rectangle with an approximate size of 6.8 by 13.7 angstroms, and must be sufficiently non-polar. Sjögren *et al.* (1996) suggested that affinity of PAHs for the cytosolic Ah-receptor may be a better prediction of carcinogenicity than mutagenicity is.

In light of the emerging knowledge on the relationship between carcinogenicity and PAH structural requirements, it might be expected that the relative carcinogenic potency of different PAHs can be predicted from their molecular structure. In fact, more than a dozen studies have been completed since 1995 on structure-activity relationships for

PAHs and carcinogenic potential (Isu *et al.*, 1996; Mezey *et al.*, 1996; Klopman *et al*, 1998; Borosky, 1999; Vendrame *et al.*, 1999; Lin *et al.*, 2001; Coluci *et al.*, 2002; Dongbin *et al.*, 2002; Kitti *et al.*, 2003).

Some of the recent QSAR (Quantitative Structure Activity Relationship) studies have focused on aspects of PAH environmental fate and effects other than carcinogenic potential. For example, Cossoni *et al. (*2002) used PAH molecular descriptors to predict the boiling point, melting point, and octanol-water partition co-efficient for 82 PAHs, and compared these with various literature values. Shi *et al.* (2001) used QSAR as a predictor of estrogenic potential of a wide variety of organic contaminants, including PAHs.

Several studies have explored QSAR approaches for predicting ecotoxicity of PAHs and other xenobiotics. Vighi *et al.* (2001), for example, used various molecular descriptors to predict toxicity of PAHs to aquatic life, within orders of magnitude – of relevance especially where the experimental data are limited or absent. For a subset of the chemicals, the predicted no-effect concentrations to algae, fish or *Daphnia magna* were in good agreement with the water quality objective proposed by the Scientific Advisory Committee on Toxicity and Ecotoxicity of Chemicals of the European Commission (CSTE, 1994). The predicted no-effect range for naphthalene was 1 $\mu g \cdot L^{-1}$ and for all of the higher molecular weight PAHs – fluoranthene, benzo[b]fluoranthene, benzo[k]fluoranthene, benzo[a]pyrene, benzo[g,h,i]perylene, and indeno[1,2,3-c,d]pyrene – was 0.0001 $\mu g \cdot L^{-1}$.

Krylov *et al.* (1997) developed a structure-activity model for photoinduced PAH toxicity.

Studies of QSAR and PAH carcinogenicity have predominantly focused on one or a few steps in the overall process of PAH uptake, cancer initiation, promotion and progression. For example, Klopman *et al.* (1998) developed a QSAR model that predicts the degree of epoxidation versus hydroxylation of various PAHs during metabolic modification at various sites around PAH molecules. Their model predicted that formation of hydroxylated byproducts of benzo[a]pyrene, primarily at the 1-, 3- and 9-positions, is less likely than the formation of various epoxides, which are predicted to occur following metabolism at the 1-2, 7-8, and 9-10 positions. Predicted P450 metabolites of benzo[a]pyrene were in agreement with various experimental observations. Borosky (1999) focused specifically on molecular models of charge delocalization in PAHs, which in turn directly affects the potential for ring opening and the tendency to form PAH-DNA adducts. Perlow and Broyde (2002) modeled preferences for normal or abnormal nucleotide incorporation by polymerases when these enzymes encounter bulky benzo[a]pyrene-DNA adducts [(+)-trans-anti-[B[a]P]-ATP].

Researchers have variously used quantum chemical properties and structural features of PAHs to examine aspects of metabolism and adduct formation. The reactivity of an atom or bond in a molecule can be expressed by its quantum chemical properties. One approach, for example, is to establish a quantum index derived from atomic co-efficients of either or both of highest occupied molecular orbitals (HOMO) and lowest unoccupied molecular orbitals (LUMO), calculated from a simple Hückel method. The influence of

the quantum properties may be further mediated by structural features of the molecule that contribute to encumbrances around various possible reaction sites.

Vendrame *et al.* (1999) used similar quantum methods, based on predictions of the "local density of state" (LDOS) to predict the relative carcinogenic activity of 81 methylated and non-methylated PAHs. Further, the authors evaluated several different mathematical classification techniques in conjunction with theoretical molecular descriptors, including principal components analysis (PCA) and neural networks (NN). The results of this QSAR study provide an indication of the predicted relative carcinogenic potency of various PAHs based on structural variations.

Figure 6-2 is adapted from Vendrame *et al.* (1999). A summary of the predicted activity of the 80 PAHs relative to benzo[a]pyrene can be calculated based on the ratio of scores on the first principal component for the unknown PAHs to that of benzo[a]pyrene (Table 6-2). PAHs with negative scores on PC-1 are simply listed in Table 6-2 as "inactive".

Vendrame *et al.* (1999) found that they could classify PAHs as carcinogenically active or inactive, using electronic indices, with an accuracy of approximately 78%. Whether using electronic indices, Principal Components Analysis or Neural Networks Analysis, it is clear that several PAHs were misclassified. In the case of the PCA analysis, 9 of 34 PAH appear to have been incorrectly classified as inactive when experimental evidence suggests otherwise, and 4 of 44 PAHs were incorrectly classified as being carcinogens when other lines of evidence indicate that they are inactive (Table 6-2). While such discrepancies can be attributed in part to a low degree of confidence in independent classifications based on very limited evidence, the prediction that 5-methylchrysene, along with 6,12-dimethylbenz[a]anthracene, is inactive undermines the confidence in the QSAR analyses even for simple binomial classification.

The ratio of scores on principal component 1 between individual PAH and benzo[a]pyrene cannot be taken as a quantitative measure of relative potency with any degree of confidence, since (i) the relative position of PAHs in multivariate space may not accurately represent true carcinogenicity in whole organisms; (ii) the relationship between the principal component scores and cancer potencies, if any, may not be linear; and (iii) the actual tumorogenic potential may be related to more than one mode of action (e.g., based on role of PAHs as both initiators and promoters.)

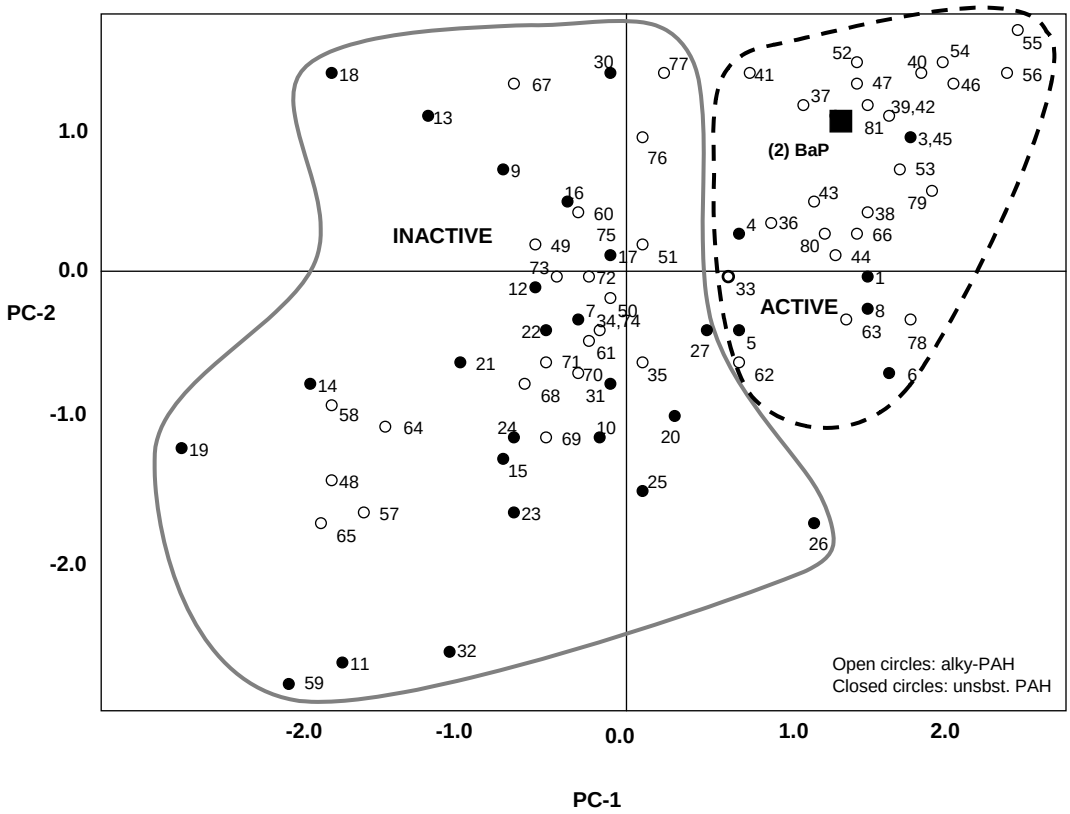


Figure 6-2: Classification of PAHs as carcinogenically active or inactive based on QSAR

(after Vendrame et al., 1999)

(the numbers correspond to PAHs as listed in Table 6-2)

Table 6-2: Predicted Carcinogenic Activity of PAHs Relative to Benzo[a]pyrene From QSAR Studies and Principal Components Analysis

(Adapted from Vendrame et al., 1999). Shaded cells are alkyl-PAH.

PAH	PAH:B[a]P PC-1 score	PAH	PAH:B[a]P PC-1 score
(1) dibenzo[3,4;9,10]pyrene	1.2	(42) 3-methylbenzo[a]pyrene	1.2
(2) benzo[3,4]pyrene (benzo[a]pyrene)	**1**	(43) 1,2-dimethylbenzo[a]pyrene	0.8
(3) dibenzo[3,4;8,9]pyrene	1.3	(44) 2,3-dimethylbenzo[a]pyrene	1.0
(4) dibenzo[3.4;6,7]pyrene	0.5	(45) 3,12-dimethylbenzo[a]pyrene	1.3
(5) dibenzo[1,2'3,4]pyrene	0.5	(46) 1,3-dimethylbenzo[a]pyrene	1.5
(6) naptho[2,3;3,4]pyrene	1.2	(47) 1,4-dimethylbenzo[a]pyrene	1.1
(7) dibenz[1,2;5,6]anthracene	Inactive? [A]	(48) 5-methylbewnzo[c]phenanthrene	Inactive? [A]
(8) tribenzo[3,4;6,7;8,9]pyrene	1.2	(49) 5-methylchrysene	Inactive? [A]
(9) dibenzo[1,2;3,4]phenanthrene	Inactive? [A]	(50) 6,8-dimethylbenz[a]anthracene	Inactive? [A]
(10) tribenzo[3,4;6,7;8,9]pyrene	Inactive? [A]	(51) 7-methylbanz[a]anthracene	Inactive? [A]
(11) dibenzo[1,2;5,6]-phenanthrene	Inactive	(52) 5-methylbenzo[a]pyrene	1.1
(12) benz[1,2]anthracene	Inactive	(53) 7-methylbenzo[a]pyrene	1.3
(13) chrysene	Inactive	(54) 6-methylbenzo[a]pyrene	1.5
(14) benzo[3,4]phenanthrene	Inactive	(55) 1,6-dimethylbenzo[a]pyrene	1.9
(15) dibenz[a1,2;7,8]anthracene	Inactive	(56) 3,6-dimethylbenzo[a]pyrene	1.8
(16) dibenz[a,c]anthracene	Inactive	(57) 4-methylbenzo[c]phenanthrene	Inactive
(17) benzo[1,2]pyrene	**Inactive**	(58) 3-methylbenzo[c]phenanthrene	Inactive
(18) phenanthrene	**Inactive**	(59) 6-methylbenzo[c]phenanthrene	Inactive
(19) triphenylene	**Inactive**	(60) 6-methylbenz[a]anthracene	Inactive
(20) benzo[1,2]naphthacene	**Inactive**	(61) 12-methylbenz[a]anthracene	Inactive
(21) dibenzo[3,4;5,6]phenanthrene	**Inactive**	(62) 6-methylanthanthrene	0.4[B]
(22) picene	**Inactive**	(63) 6,12-dimethylanthanthrene	1.0[B]
(23) tribenz[1,2;3,4;5,6]anthracene	**Inactive**	(64) 1-methylbenzo[c]phenanthrene	Inactive
(24) dibenzo[1,2;5,6]pyrene	**Inactive**	(65) 2-methylbenzo[c]phenanthrene	Inactive
(25) phenanthra[2,3;1,2]-anthracene	**Inactive**	(66) 10-methylbenzo[a]pyrene	1.0[B]
(26) benzo[1,2]pentacene	**Inactive**	(67) 6-methylchrysene	Inactive
(27) anthanthrene	**Inactive**	(68) 3-methylbenz[a]anthracene	Inactive
(28) benzene	**Inactive**	(69) 1-methylbenz[a]anthracene	Inactive
(29) naphthalene	**Inactive**	(70) 11-methylbenz[a]anthracene	Inactive
(30) pyrene	**Inactive**	(71) 9-methylbenz[a]anthracene	Inactive
(31) benzo[g,h,i]perylene	**Inactive**	(72) 2-methylbenz[a]anthracene	Inactive
(32) coronene	**Inactive**	(73) 5-methylbenz[a]anthracene	Inactive
(33) 7,12-dimethylbenz[a]-anthracene	0.5	(74) 8-methylbenz[a]anthracene	Inactive
(34) 6,12-dimethylbenz[a]-anthracene	Inactive? [A]	(75) 2-methylpyrene	Inactive
(35) 6,8,12-trimethylbenz[a]anthracene	Inactive? [A]	(76) 4-methylpyrene	Inactive
(36) 2-methylbenzo[a[pyrene	0.7	(77) 1-methylpyrene	Inactive
(37) 4-methylbenzo[a]pyrene	0.8	(78) 7,10-dimethylbenzo[a]pyrene	1.3[B]
(38) 11-methylvbenzo[a]pyrene	1.2	(79) 6,10-dimethylbenzo[a]pyrene	1.5
(39) 2-methylbenzo[a]pyrene	1.2	(80) 8-methylbenzo[a]pyrene	0.9
(40) 1-methylbenzo[a]pyrene	1.4	(81) 9-methylbenzo[a]pyrene	1.2
(41) 4,5-dimethylbenzo[a]pyrene	0.5		

Notes: A- considered to be carcinogenically active based on the Cavalieri *et al., (*1983) scale.
B- considered to be carcinogenically inactive based on the Cavalieri *et al., (*1983) scale.

Overall, QSAR studies have assisted in advancing the collective mechanistic understanding of cancer, along with its prevention and treatment. The focus on limited

stages within a multistage process of carcinogenesis, however, limits the utility of the emerging understanding on quantitative-structure activities relationships for assessing the relative or absolute cancer-related human-health risks of cancer. In addition, none of the QSAR studies have incorporated a comparison between mathematically/structurally predicted carcinogenicity and actual experimental data on cancer in whole organisms and populations. Based on this, it was concluded that QSAR evidence is very important as corroborating evidence for human health risks from cancer for PAHs where there is very limited direct experimental or epidemiological evidence; however, such studies cannot yet be used to confidently predict relative cancer potency with a high degree of certainty. The currently available knowledge of mechanisms of cancer induction, coupled with QSAR studies, suggests that several of the alklylated and other PAHs not currently identified as probable carcinogens by IARC, Health Canada or others (due to lack of experimental data) exhibit the appropriate structural properties to be carcinogenic.

Unfortunately, each QSAR study dealt with only one stage of carcinogenesis, which is a multi-stage, multi-mechanism process. In particular, most QSAR-type research has focused on the potential for PAHs to form diol epoxides, which may intercalate with DNA and subsequently cause genotoxicity. Some of the available studies establish different relative potencies for the PAHs based on CYP1A1 induction tendency, interaction with the cytosolic Ah-receptor, or – in one case – tendency of the DNA adduct formed by the most favoured diol epoxide to be removed through DNA repair mechanisms. Since guidance from QSAR studies tended to provide an indication of relative potencies for only one step of the multi-stage uptake, initiation, promotion and progression processes, such an approach cannot yet be used to estimate relative potencies based on the overall process.

6.6.2 The Benzo[a]pyrene Potency Equivalence Approach

The major approach advocated by regulatory agencies such as the USEPA (1993, 1999), California EPA (OEHHA, 1992), Netherlands (RIVM, 2000), the UK (UK Environment Agency, 2002), or Provinces of British Columbia and Ontario for assessing the human health risks of PAH-containing mixtures involves the use of “potency equivalence factors” (PEFs), also referred to as “relative potency factors” (RPFs) or “toxicity equivalence factors” (TEFs). These factors are used to relate the carcinogenic potential of other PAHs to that of benzo[a]pyrene (B[a]P).

There are more than a dozen sets of equivalency numbers that have been proposed over the last two decades (Table 6-3). Boström *et al.* (2002) provide an up-to-date review.

Bosveld *et al.*, (2002) compared the potency relative to benzo[a]pyrene of nine PAHs to induce the CYP enzyme ethoxy resorufin-*O*-deethylase (EROD) or other markers of PAH detoxification/activation in cells or in vitro cell lines. They also compared the results of their analysis to similar studies undertaken by Willett *et al.* (1997), Till *et al.* (1999), Bols *et al.* (1999), Fent and Bätscher (2000) and Machala *et al.* (2001). The results are provided in Table 6-3.

The measurement of *in vitro* or *in vivo* biochemical responses to different PAHs provides a far less proximate evaluation of relative cancer risks than the direct observation of cancerous lesion induction in whole organisms. It is nonetheless of interest that the different researchers have provided a similar picture *vis-à-vis* the ranking of relative EROD inducing potency of the different PAHs, and that this ranking departs from relative potency schemes based on laboratory rodent data. In particular, several *in vitro* studies suggest that benzo[b]- and benzo[k]fluoranthene deserve attention based on their potency relative to other PAHs in environmental mixtures.

Bosveld *et al.* (2002) found based on their own and reviewed EROD induction studies that the order of potency increased as follows:

Anthracene ≅ Phenanthrene < Fluoranthene < Naphthalene <

Benzo[g,h,i]perylene < Indeno[I,2,3-c,d]pyrene < Benz[a]anthracene <

Benzo[a]pyrene < Chrysene < Benzo[k]fluoranthene

Conclusions about the relative benzo[a]pyrene-like toxicity of chrysene and benzofluoranthenes, in particular, are expected to have major implications for environmental risk estimates at PAH-contaminated sites.

Table 6-3: Comparison of relative potency estimates for various studies

PAH	Chu and Chen, 1984, as cited in Nisbet and LaGoy, 1992	Krewski *et al*., 1989	Nisbet and Lagoy, 1992	USEPA, 1993	McClure, 1996	OEHHA, 1992; Collins *et al*., 1998
Basis	Inhalation exposures	Inhalation exposures	Inhalation exposures	Inhalation exposures	Primarily dermal, some intraperitoneal or subcutaneous	Mixture of data for inhalation and other exposure routes, esp. mouse skin
acenaphthene			0.001			
acenaphthylene			0.001			
anthracene			0.01			
benz[a]anthracene	0.013	0.145	0.1	0.1	0.1	0.1
benzo[a]pyrene	1	1	1	1	1	1
benzo[b]fluoranthene	0.08	0.14	0.1	0.1	0.1	0.1
benzo[j]fluoranthene		0.061			0.1	0.1
benzo[k]fluoranthene	0.04	0.066	0.1	0.01	0.1	0.1
Benzo(g,h,i)perylene		0.022				
chrysene	0.001	0.0044	0.01	0.001	0.1	0.01
5-methylchrysene						1.0
dibenz[a,h]anthracene	0.69	1.11	5[a]	1.0	1	
dibenz(a,j)acridine						0.1
dibenz(a,h)acridine						0.1
Dibenzo(a,h)pyrene						10
Dibenzo(a,i)pyrene						10
Dibenzo(a,l)pyrene						10
indeno[1,2,3-c,d]pyrene	0.017	0.232	0.1	0.1	0.1	0.1
fluoranthene			0.001			
fluorene			0.001			
2-methylnaphthalene			0.001			
naphthalene			0.001			
phenanthrene			0.001			
pyrene		0.081	0.001			

Notes: a) at low concentrations;

PAH	The Netherlands (RIVM, 1989)	The Netherlands (Baars, 2000)	Health Canada (Meek *et al*., 1994)	British Expert Panel, 1999, (based on Deutsch-Wenzel, 1983)	Ontario (Muller, 1997)[b]	Larsen and Larsen, 1998
Basis			Exposure in rats by lung implantation	Rat lung implant study cited in Meek *et al.*, 1994	Tumour initiation in mouse skin, or rat lung assay data	Various exposure routes
acenaphthene		0.001				
acenaphthylene		0.01				
anthracene	0					0.0005
benz[a]anthracene	0-0.04	0.1		0.1	0.014	0.005
benzo[a]pyrene	1	1	1	1	1	1
benzo[b]fluoranthene		0.1	0.06	0.11	0.11	0.1
benzo[j]fluoranthene		0.1	0.05		0.045	0.05
benzo[k]fluoranthene	0.03-0.09	0.1	0.04	0.03	0.037	0.05
benzo(g,h,i)perylene	0.01-0.03				0.012	0.02
chrysene	0.05-0.89	0.01		0.03	0.026	0.03
5-methylchrysene						
dibenz[a,h]anthracene		1		1.91	0.89	1.1
dibenz(a,j)acridine						
dibenz(a,h)acridine						
Dibenzo(a,h)pyrene						
Dibenzo(a,i)pyrene						
Dibenzo(a,l)pyrene						
indeno[1,2,3-c,d]pyrene	0-0.08	0.1	0.12	0.08	0.067	0.1
fluoranthene	0-0.06	0.01				0.05
fluorene						
2-methylnaphthalene						
naphthalene						
phenanthrene	0.01				0.00064	0.0005
pyrene		0.001			0	0.001

b) study includes relative potency factors for many alkyl-substituted forms.

PAH	Kalberlah *et al.*, 1995 as cited in WHO/IPCS, 1998[c]	Alberta Environment, 2001b	Willett *et al.*, 1997	Clemons *et al.*, 1998	Clemons *et al.*, 1998
Basis		All based on BCMELP (1988) except – (i) benzo(j)fluoranth.; (ii) chrysene (after USEPA, 1993)	EROD Induction potency **relative to B[a]P** in rat hepatoma H4IIE cell culture	**Potency relative to B[a]P (and relative to 2,3,7,8-TCDD) of AhR-mediated expression** in a mouse hep. cells	**Potency relative to B[a]P (and relative to β-estradiol) in MCF-7:ER** mouse cells
acenaphthene					
acenaphthylene					
anthracene				10 (0.0001)	nd
benz[a]anthracene	0.1	0.1	0.08	1 (0.00001)	0.1 (0.0001)
benzo[a]pyrene	1	1	1	1 (0.00001)	1 (0.001)
benzo[b]fluoranthene	0.1	0.1	7.8		
benzo[j]fluoranthene	0.1	0.1			
benzo[k]fluoranthene	0.1	0.1	15	5,000 (0.05)	nd
benzo(g,h,i)perylene	0.01				
chrysene	0.01	0.0044	0.6	1,000 (0.01)	0.5 (0.0005)
5-methylchrysene					
dibenz[a,h]anthracene	1.0	1.1	6.3	5,000 (0.05)	nd
dibenz(a,j)acridine					
dibenz(a,h)acridine					
Dibenzo(a,h)pyrene					
Dibenzo(a,i)pyrene					
Dibenzo(a,l)pyrene					
indeno[1,2,3-c,d]pyrene	0.1	0.2	3.0		
fluoranthene					
fluorene				nd	nd
2-methylnaphthalene					
naphthalene					
phenanthrene					
pyrene				nd	nd

c) The WHO toxicity equivalence factors have been used by the Canadian Food Inspection Agency for establishing interim tolerances for PAHs in imported food substances such as olive pomace oil.

PAH	Klimm *et al.*, 1999	Till *et al.*, 1999	Bols *et al.*, 1999	Fent and Bätscher, 2000	Machala *et al.*, 2001	Bosveld *et al.*, 2002
Basis	EROD induction relative to B[a]P (and relative to 2,3,7,8-TCDD)	EROD induction, rat hepatocytes	EROD induction, Rainbow trout liver cell line	EROD induction, fish cell line	CALUX gene reporter assay	In vitro EROD induction, H4IIE cell line
acenaphthene						
acenaphthylene						
anthracene		0	0	0	0	0
benz[a]anthracene	0.09 (0.000027)	0.10	0.14	0.11	0.08	0.7
benzo[a]pyrene	1 (0.0003)	1	1	1	1	1
benzo[b]fluoranthene	1.3 (0.00038)					
benzo[j]fluoranthene						
benzo[k]fluoranthene	1 (0.00029)	9.6	3.4	6.1	18.2	3.8
benzo(g,h,i)perylene		0.0044	?	0	0	0.002
chrysene		0.26	0.16	0.59	1.1	2.7
5-methylchrysene						
dibenz[a,h]anthracene	0.26 (0.000078)					
dibenz(a,j)acridine						
dibenz(a,h)acridine						
Dibenzo(a,h)pyrene						
Dibenzo(a,i)pyrene		1	1	1	1	1
Dibenzo(a,l)pyrene						
indeno[1,2,3-c,d]pyrene	0.29 (0.000086)	2.7	0.9		3.3	0.3
fluoranthene		0.0004	0	?	0.0003	0.00003
fluorene						
2-methylnaphthalene						
naphthalene		0	0	0	0	0.007
phenanthrene		0	0	?		0
pyrene						

Agencies that have developed B[a]P relative potency schemes tend to have only limited confidence in them. Overall, there are at least four factors that create uncertainty about relative potency schemes: (i) prediction of mixture effects from single substance studies; (ii) prediction of potency for one route of exposure based on experimental data derived using another exposure route; (iii) possible presence of carcinogens or promoters in the mixture that are not measured and/or not addressed in the relative potency scheme, and (iv) uncertainty about the use of laboratory rodent models to predict human cancer potential.

For example, according to USEPA (1999) –

> "The Provisional Guidance for Quantitative Risk Assessment of Polycyclic Aromatic Hydrocarbons (PAH) (EPA/600/R-93/089) described an RPF approach for assessing the carcinogenic risks posed by exposures to non-benzo[a]pyrene (B[a]P) PAH that had been judged by the Agency as B2 substances; i.e., probable human carcinogens. The results of mouse skin carcinogenicity assays for these non-B[a]P B2 PAHs were compared with those of B[a]P to estimate cancer potency. The approach assumed that the B2 PAH had the same cancer slope factor as B[a]P. The ability of these non-B[a]P B2 PAH to elicit rodent skin tumors was quantitatively compared to that of B[a]P; the results of this quantitative comparison were expressed as an "estimated order of potency". Because this approach was limited to the cancer endpoint, based on B[a]P exposure from a single (oral) pathway (for the derivation of the slope factor), and considered only a small subset of the PAH, EPA has described it as an estimated order of potency. This naming reflects the uncertainty EPA felt about the application of this type of approach given the current state of science of PAH."

According to the ATSDR (2002) –

> "The risk assessment of mixtures on human and environmental health is complicated by the paucity of approaches to assess environmental exposures and biological effects of chemical mixtures. Furthermore, the use of single-compound toxicity data to predict the toxicity and health effects of complex mixtures is highly speculative. The difficulty of using single-compound toxicity data to estimate the toxicity of complex mixtures is illustrated by the considerable variation in carcinogenicity of PAHs depending on the components of the mixture (Warshawsky *et al.*, 1993). For instance, when benzo[a]pyrene, a potent animal carcinogen, is added at noncarcinogenic levels to mixtures of carcinogenic or noncarcinogenic PAHs, the skin tumourigenicity of the mixture, as well as the latency of tumor incidence, is changed. Hence, unless the actual complex mixture is evaluated directly for toxicity, it is unlikely that its toxic potency can be interpreted from that of its isolated components"

It should also be noted that the vast majority of relative potency schemes are intended specifically to address tumourogenesis in lungs based on the inhalation of PAHs (e.g., OEHHA, 1992), or have been based on mouse skin/direct application trials. Neither of

these approaches relate well to the uptake of PAHs in incidentally ingested soil, which is expected to be a major exposure pathway at contaminated sites.

Collins *et al.* (1998) described the order of preference for the selection of relative potency values for PAHs, as used to develop the California EPA Office of Environmental Health Hazard Assessment (OEHHA) PEFs, designed for managing air quality issues:

i) derive slope factors based on detailed quantitative risk assessment of individual PAHs (based on availability of adequate dose response data for oral and inhalation exposures in humans);
ii) derive slope factors from "expedited" quantitative risk assessment of humans;
iii) tumour data from exposure path of interest (i.e., inhalation for estimating airborne exposure risks);
iv) tumour data for intratracheal or intrapulmonary administration;
v) tumour data from oral administration;
vi) tumour data for skin-painting studies;
vii) tumour data for sub-cutaneous or intraperitoneal injection;
viii) genotoxicity data; and
ix) structure-activity information (least preferred).

This scheme is based on estimating risks from the inhalation of PAH-containing particulates, as are the OEHHA PEFs provided in Table 6-3. Adapting such a scheme to soil-based exposures suggests that whole animal dose-response data from oral exposure studies would be preferable to data from inhalation, intratracheal, dermal, subcutaneous, intraperitoneal or other types of exposures, provided that applicable human epidemiological data [items (i) and (ii)] are not available.

Few studies that have been used to derive PAH PEFs used oral exposure methods. Based on reviews by other agencies, it is clear that sufficient oral exposure data for the derivation of cancer slope factors exist only for benzo[a]pyrene. No adequate human epidemiological studies are available. The major portion of the remaining available data fits into the less preferred categories, being largely based on intratracheal and skin-painting exposures (Table 6-4). Yet, numerous studies demonstrate that both the types of carcinogenic lesions formed and the dose-response relationship are highly dependent on the route of exposure (Culp *et al.,* 1998).

Some of the relative potency schemes have been critically examined by comparing estimated carcinogenicity based on PAH composition and relative potency with measured potency.

Table 6-4: Summary of data availability (number of data points) for the carcinogenicity of PAHs according to exposure route

*(adapted from WHO/IPCS, 1998)**

PAH	Oral	Dermal	Inhal./ Intratrach.	Subcutan./ Intramusc.	Intraperit./ Intravenous	Other**
LABORATORY RODENTS						
Acenaphthene		1-/1±				
Anthracene	2+	6-/1±	1-	1+/3-	1-/1±	1-
Benz[a]anthracene	2+/1-/1±	7+/7-		4+/4-	3-	2+/1-
Benzo[b]fluoranthene		7+	1+/1-	1+	1+	
Benzo[j]fluoranthene		3+/0-/1±	1+		1+	
Benzo[k]fluoranthene		1+/2-/1±		1+	2-	
Benzo[g,h,i]fluoranthene		2-				
Benzo[a]fluorene		1±				
Benzo[g,h,i]perylene		8-		2-		1-
Benzo[c]phenanthrene		2+/0-/2±		1-/1±		
Benzo[a]pyrene	8+/0-	27+/1-/1±	21+/1-/1±	7+/0-	4+/0-	5+/0-
Benzo[e]pyrene		2+/1-/5±	1-	2+/1-/5±	1±	1-
Chrysene		11+/9-/1±		4+/5-/1±	1+/2-/1±	1-
Coronene		1-/1±				
Dibenz[a,h]anthracene	1+	6+	2+/0-/1±	10+/0-/1±	1+	1-
Dibenzo[a,e]pyrene		3+		2+		1-
Dibenzo[a,h]pyrene		6+		2+	1+	1+
Dibenzo[a,i]pyrene		7+	2+	4+	1+	1+/1-
Dibenzo[a,l]pyrene		7+		1+		2+
Fluoranthene		6-		2-	3+	
Fluorene	2-	3-		1-	1-	
Indeno[1,2,3-c,d]pyrene		2+/1-/2±	1+	1+	1-	
5-methylchrysene		13+		1+/0-/1±	1+/1-	1-
1-methylphenanthrene		1-				
Naphthalene	1-	1-/2±		1-/1±	1-	
Perylene		2-				
Phenanthrene	1+	1+/3-/3±		3-	1-	1-
Pyrene		1+/7-/3±		1-	1-	1-
Triphenylene		2-				
LIVESTOCK						
Benzo[a]pyrene				3-		
DOGS/MONKEYS						
Benzo[a]pyrene			1+	2+/1-		
Dibenz[a,h]anthracene						
Dibenzo[a,I]pyrene						

*Abbreviations and codes: +, positive evidence of carcinogenic effects; -, negative; ±, questionable.

**Other: Includes intramammary injection, bladder implant, and bronchial implant.

Table 6-5: Summary of carcinogenicity data for PAHs based on oral/dietary exposure routes only

(*adapted from WHO/IPCS, 1998)*

COMPOUND/ TEST ORGANISM	SAMPLE SIZE	EXPOSURE REGIME	DURATION	RESULTS	CARCINO-GENICITY.	STATS. MODEL	NOTES*	REFERENCE
ANTHRACENE								
Rat (?)	N=33	6 mg/animal/day, 7x/week	33 months	22/31 alive after 1 year; no tumours after 33 mo	**(–)**		LC	Schmahl & Reuter, cited by Gerarde (1960)
Rat BDI/BDIII	N=28	5-15 mg /animal /day, 6 x/week	78 weeks	2/28 malignant tumours	**(–)**		LD	Schmähl (1955)
BENZ[A]ANTHRACENE								
57/BL Mouse	8-19	0.5 mg/animal, 1x, 8x, 16x	~2 months	0/12 (control); 0/13; 1/19; 1/8 papillomas, no carcinomas	**(?)**	✓	LN	Bock and King (1959)
Newborn B6AF1/J mice	10, 20	1.5 mg/animal, 3x/week.	5 weeks	solvent only: 10% hepatomas, 35% pulmonary adenomas; Treatment: 100% hepatomas, 95% pulmonary adenomas	**(+)**		VAL	Klein (1963)
BENZO[A]PYRENE								
A HE/J Mice	F: n=15	3 mg/animal in sesame oil; 2 x	30 weeks	Increased pulmonary tumours: 16.6 (control: 0.3)	**(+)**	✓	VAL	Wattenberg and Leong, 1970
A/J mice	F: n=15	2 mg/animal, every 2 weeks,	26 weeks	No control: 15/15 with forestomach tumours; 15/15 with pulmonary adenomas	**(+)**	✓	VAL	Sparnins *et al.*, 1986
CFW mice	M/F: 25-73	0.004-1 mg/animal per day; 100-165 days	140-200 d	Dose-dependent gastric tumours (0-90%); control: no tumours	**(+)**		VAL	Neal & Rigdon (1967)
CFW mice	M/F: 9-26	1-20 mg/animal / day, <1-30 d	15-30 d	Dose-dependent gastric tumours (0-100; control: no tumours	**(+)**		VAL	Neal & Rigdon (1967)
White Swiss mice	M/F: 60-175	0.25 and 1 mg/g food, <= 34 weeks	34 weeks	33 and 61 % with Stomach tumours; 53 and 20% with lung tumours.	**(+)**		VAL	Rigdon & Neal (1966)

COMPOUND/ TEST ORGANISM	SAMPLE SIZE	EXPOSURE REGIME	DURATION	RESULTS	CARCINO-GENICITY.	STATS. MODEL	NOTES*	REFERENCE
				Controls: 1% and 21%				
Sprague-Dawley rats	F:9	100 mg·kg^{-1}, 1x	60 days	8/9 with mammary tumours; controls: 8/164 in 310 days	(+)		LN	Huggins and Yang, 1962
LEW/Mai rats	F: 20	625 mg/animal, 1x/week; 8x; 50 mg/animal, 1 ×	90 weeks	67-77% with mammary tumours; Control: 30%	(+)	✓	VAL	McCormick *et al., (*1981)
Syrian golden hamster	M/F: 13	2.5 mg/animal, 4 days/week, ≤ 14 months	≤ 14 months	9/13 with forestomach cancer; 2/13 with papillomas	(+)		VAL	Chu and Malmgren (1965)
DIBENZ[A,H]ANTHRACENE								
Swiss mice	M	1.5 mg/animal in PEG-400, 1x (initiation study)	30 weeks	21 % forestomach papillomas; promoter only: 14%	(?)		LC	Berenblum and Haran (1955)
DBA/2 mice (drinking water)	M/f 21/21; control 25/10	0.8 mg/day/animal in olive oil; 8-9 months	8-9 months	14/14 m and 13/13 f with pulmonary adenomas; 14/14 m and 10/13 f with alveologenic carcinomas; control: 1 mouse with tumour	(+)		VAL	Snell and Stewart (1962)
FLUORENE								
Buffalo rats	f 20	0.05% diet; 4.3 mg/rat per day = 796 mg/rat (total intake) over 6 mo.	10.7 months	2/11 carcinomas (renal pelvis, ureter); control: 4/16 with carcinomas	(?)		LD	Morris *et al.*, 1960
Buffalo rats	f 18	0.05% diet; 4.6 mg/rat per day = 2553 mg/rat (total intake) over 18 months	≤ 20.1	7/18 tumours; control: 4/18 or 15/18 tumours	(?)		VAL	Morris *et al.*, 1960
NAPHTHALENE								
BD I/BD I II	28	10-20 mg/animal,	Life time	No tumours	(–)		LD	Schmähl (1955)

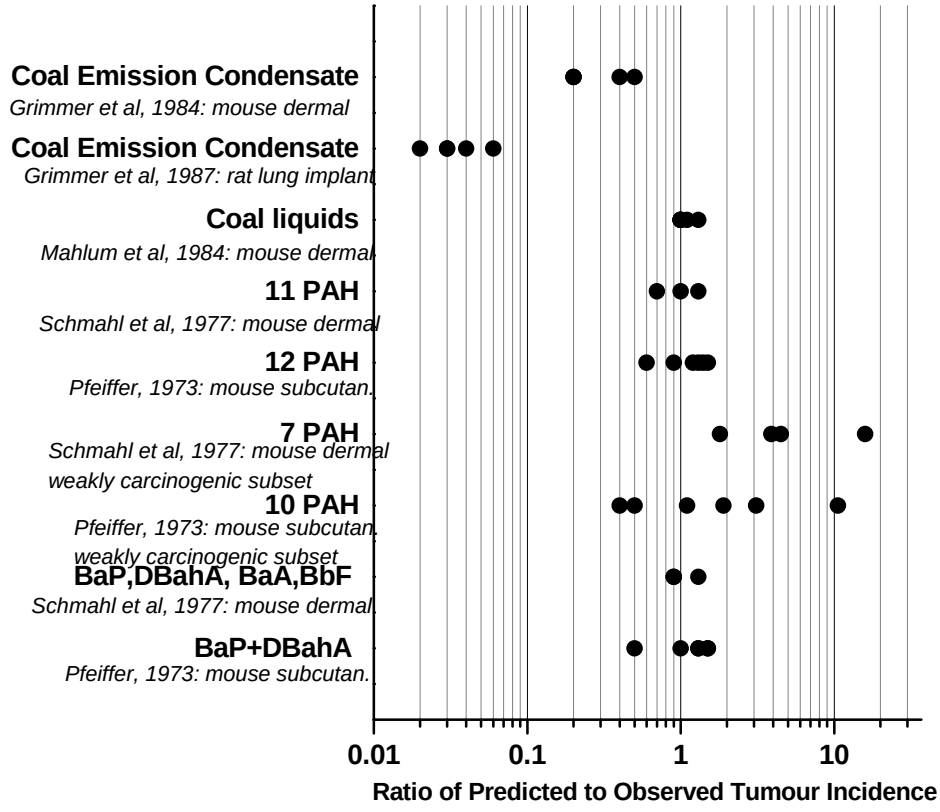
COMPOUND/ TEST ORGANISM	SAMPLE SIZE	EXPOSURE REGIME	DURATION	RESULTS	CARCINO-GENICITY.	STATS. MODEL	NOTES*	REFERENCE
Rats (inbred)		6x/week, 70 weeks						
PERYLENE								
Sprague-Dawley rats	f 10	200 mg/rat, 1x; experiment on mammary tumours	60 days	No tumours at 60 days; Controls: 8/164 after 310 days	**(–)**		LN	Huggins and Yang (1962)

*Study Validity (after WHO/IPCS, 1998): VAL: valid; LD: limited design; LC: limited documentation; LS: limited survival; LN: limited number of animals.

McClure (1996), based on support from the USEPA, independently developed PEFs [referred to in that study as "Estimated Order of Potency" (EOP) factors] for 14 EPA Group B2 PAHs using slope factors calculated primarily from dermal exposure studies, which were then compared with the USEPA/IRIS benzo[a]pyrene slope factor. The cancer potency of dibenz[a,h]anthracene, dibenzo[a,e]fluorene, dibenzo[a,e]pyrene, and dibenzo[a,h]pyrene were estimated to be similar to that of benzo[a]pyrene, and were assigned an EOP of 1.0. The EOP assigned to benz[a]anthracene, the benzofluoranthenes, chrysene, cyclopenta[c,d]pyrene, dibenzo[a,I)pyrene and indeno[1,2,3-c,d]pyrene was 0.1. Dibenzo[a,l]pyrene was assigned an EOP of 100.

Figure 6-3: Ratio of observed tumour incidence to predictions based on PAH "Estimated Order of Potency" factors

(developed by McClure,1996)



The EOPs were subsequently validated using five previously published studies in which the tumour response was quantified in animals exposed to PAH mixtures, for which individual PAHs had also been quantified. The data from the studies were used to construct a polynomial dose-response curve. The observed whole PAH mixture response

was then compared to the predicted response based on the known carcinogenicity of benzo[a]pyrene, and the application of the EOPs for all Group B2 PAHs in the mixture.

The results of the validation are shown in Figure 6-3. The McClure (1996) EOPs differ from the Kalberlah *et al.* (1995) benzo[a]pyrene potency equivalency factors, as cited in WHO/IPCS (1998) only slightly. The EOPs for chrysene and indeno[1,2,3-c,d]pyrene were higher by an order of magnitude than the Kalberlah *et al.* (1995) PEFs. Kalberlah *et al.* (1995), in addition, established a number for benzo[g,h,i]perylene.

McClure (1996) found that there were few published data available to undertake such validation studies. The EOP approach provided a reasonable prediction of tumour incidence for PAH mixtures with more carcinogenic PAHs and, to a lesser extent, for dermally applied coal tar. The EOP approach tended to over-estimate carcinogenicity of mixtures comprised only of weakly carcinogenic PAHs. Finally the EOP approach grossly underestimated observed tumour incidence for lung exposure experiments. This is not too surprising, given that the EOPs were developed based almost entirely on dermal exposure studies. In the context of PAH exposures through either drinking water or incidental soil ingestion, the validation study provides little guidance, unfortunately.

The tendency of benzo[a]pyrene relative potency schemes to under-estimate coal tar carcinogenicity might be due to the presence of unaccounted for carcinogens in the mixture. In particular, Harvey *et al.* (2000) and Koganti *et al*. (2001) found that 7H-benzo[c]fluorene, a major constituent of coal tars, strongly induced DNA-adduct formation.

Schneider *et al.* (2002) compared cancer risks using laboratory rodent data based on the oral or other exposure routes to directly compare the carcinogenic potency of pure benzo[a]pyrene and PAH mixtures for which the composition is known and B[a]P-like carcinogenicity was predicted using relative potency schemes. These researchers were able to re-analyze data from studies wherein the test animals were exposed to both a PAH mixture and to pure B[a]P under the same test conditions. Studies re-evaluated included:

- Culp *et al.* (1998) who exposed B6C3F1 mice orally to two field collected coal tar mixtures;

- Weyland *et al. (*1995) who exposed A/J mice orally to manufactured gas plant residue;

- Robinson *et al. (*1987) who exposed A/J mice via gavage to coal tar paint;

- Mangelsdorf *et al. (*1998) who exposed CD1 mice dermally to two coal tar products;

- Grimmer *et al. (*1982a,b; 1983) who exposed CFLP mice dermally to used engine oil or automotive exhaust condensate;

- Deutsch-Wenzel *et al*. (1984) who exposed CFLP mice to brown coal flue gas condensate;

- Grimmer *et al.* (1985) who exposed CFLP mice dermally to flue gas concentrate;

- Nesnow *et al.* (1983) who exposed SENCAR mice dermally to automobile exhaust extract, roofing tar emission extract, or topside coke oven extract; and

- Grimmer *et al.* (1984; 1987) who exposed OM rats via lung implantation to gasoline engine exhaust condensate, flue gas condensate, or diesel engine condensate.

All of these studies also included the parallel evaluation of carcinogenic endpoints for pure B[a]P exposure, and several of them included the characterization of the exposure mixture in terms of PAH composition. Schneider *et al.* (2002) calculated the ratio in the mixtures of total B[a]P relative potencies (including B[a]P) to B[a]P alone. The results are provided herein as Figure 6-4.

Based on the available data, the use of the USEPA B[a]P relative potency scheme resulted in an accurate prediction of mixture carcinogenic potential in one case, but more often resulted in an inaccurate prediction. In three cases, the use of a B[a]P relative potency scheme over-predicted cancer risks relative to observed results by a factor 1.5 to 3.6. In five cases, however, the use of a B[a]P relative potency scheme resulted in an under-prediction in the range of 3.5 to more than 14-fold. The magnitude of differences noted between predicted and observed risk is actually quite small. Given the uncertainty inherent in the animal studies themselves, a difference of less than one order of magnitude would typically be considered quite minor for the purpose of the acceptability of a model. Such uncertainty, however, needs to be considered for the larger risk-based guidance.

Schneider *et al.* (2002) concluded that the contribution of benzo[a]pyrene to the carcinogenicity of PAH-containing mixtures depends very much on the exposure route and type of cancer examined, but the B[a]P relative contribution was less variable across industrial or other source types. The authors state –

> "The use of B[a]P toxicity equivalence factors is not suitable because the carcinogenic risk of PAH mixtures is highly dependent on the exposure pathway, therefore it is proposed here to assess the carcinogenic risk of PAH mixtures using dose-response data for the exposure route in question to calculate risk estimates…. ..using only data from the recently carried out study of Culp *et al.,* a risk estimate (slope factor) for all exposure-related tumours was obtained."

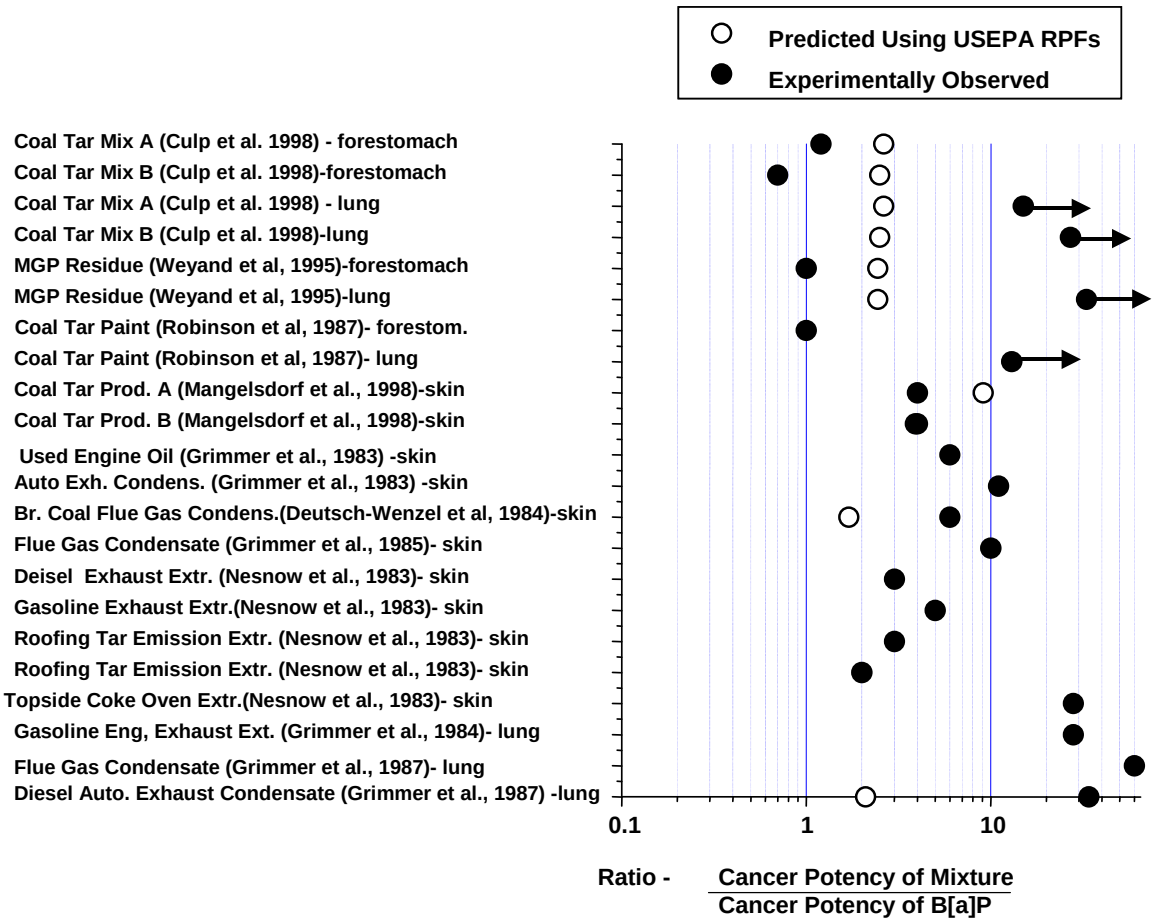


Figure 6-4: Predicted (using the USEPA relative potency scheme) and observed cancer potency ratios between whole mixtures and benzo[a]pyrene
(Adapted from Schneider et al., 2002)

Schneider *et al.*, (2002) proposed the use of a slope factor of 11.5 $(mg \cdot kg^{-1} \cdot day^{-1})^{-1}$ for risks based on oral exposures and applied in consideration of only the mixture's B[a]P content, based on the Gaylor *et al.*, (1998) studies, but further suggested that this slope factor is not relevant for other PAH mixture types such as diesel engine emissions. This still leaves a gap for PAH soil contamination derived from other than coal-tar sources, for which there is inadequate experimental cancer data based on oral exposure routes. Further, humans are more typically exposed at PAH contaminated sites through both oral and dermal exposure, and potentially via inhalation of PAH-containing particulates as well. The Schneider *et al.*, approach would require the availability of a separately derived B[a]P cancer slope factor for each of these exposure routes, something that is not currently possible in light of the available experimental evidence.

Wynder and Hoffman (1959) exposed female Swiss Millerton mice three times weekly for life to individual PAHs (0.1 or 1%). After 6 months, the order of potency to induce papillomas and carcinomas was –

benzo[a]pyrene = dibenz[a,h]anthracene > dibenzo[a,i]pyrene >
chrysene > benzo[e]pyrene > fluoranthene, anthracene, pyrene.

Habs *et al.,* (1980) conducted a similar lifetime skin-paint PAH exposure experiment in female NMRI mice. The observed order of potency was –

benzo[a]pyrene >> benzo[b]fluoranthene > benzo[j]fluoranthene
> benzo[k]fluoranthene, coronene, indeno[1,2,3- c,d]pyrene.

The last group had no carcinogenic effect in this study, while benzo[b]fluoranthene was mildly carcinogenic.

Tumourigenic activity of nonalternant PAHs benzo[b]fluoranthene, benzo[j]fluoranthene, and benzo[k]fluoranthene and indeno[1,2,3-c,d]pyrene and benzo[a]pyrene were evaluated by injecting a total of 0.5, 1.1, 2.1, 2.1, or 0.5 µmol of each compound, respectively, in dimethyl sulfoxide in aliquots of 5, 10, or 20 µl on days 1, 8, and 15 after birth to CD-1 mice (LaVoie *et al*., 1987). The benzofluoranthenes are often major constituents of coal tars and coal tar creosotes. The observed order of potency was –

benzo[a]pyrene > benzo[b]fluoranthene = benzo[j]fluoranthene >
benzo[k-fluoranthene, indeno[1,2,3-c,d]pyrene.

Neither benzo[k]fluoranthene nor indeno[1,2,3-c,d]pyrene were tumourogenic.

There remains the issue that carcinogenic potential of the carcinogenic PAHs may not be strictly additive (Heinrich *et al*., 1994; WHO, 2003), and that there is also a good possibility of further interactions between carcinogenic PAHs and poorly or non-carcinogenic PAHs such as phenanthrene (for example, through competition for binding sites on CYP enzymes). Various studies of exposure to mixture have shown that individual PAHs may interact metabolically in a myriad of ways resulting in synergistic, additive or antagonistic effects, and consequently nothing can be concluded at present on the resulting tumourigenic actions of individual PAHs in complex mixtures (Montizaan *et al.,* 1989; WHO, 2003).

Sjögren *et al.* (1996) examined the ability of various bioassays such as bacterial mutagenicity or Ah-receptor affinity to predict the carcinogenic potency in rodents of 29 different PAHs. Such bioassays include bacterial mutagenicity tests such as the Ames test, enhancement and inhibition tests of microbial mutagenicity, Ah-receptor (AhR) affinity, and induction of various CYP enzymes (e.g., ethoxyresorufin-*O*-deethylase) that bioactivate many PAHs to their proximal carcinogenic form. Overall, the strongest correlate with rodent carcinogenic potency was AhR affinity. The authors concluded that some of these responses were inversely correlated; for example, enzyme induction and mutagenicity were negatively correlated. The authors proposed that bacterial mutagenicity reflects cancer initiation potency, whereas AhR affinity better reflects the promotive effects of some PAHs. Cancer initiation and promotion may be provoked by different PAHs; i.e., reactive metabolites and parent PAHs, respectively. It was

speculated that – unlike the situation for the majority of laboratory rodent studies – initiation may be most important at doses reflecting typical human exposures.

A review of more recent literature revealed similar conclusions regarding the use of a TEF approach with respect to PAH mixtures, especially at coal tar sites (Kroese *et al*., 2001; Koganti *et al*., 2001; and Fitzgerald *et al*., 2004). Many of the studies recommend that B[a]P and its associated TEF approach be reconsidered, however, the current literature regarding PAH mixtures is limited. Kroese *et al*. (2001) estimated carcinogenic risks associated with PAH mixtures to be about 10 to 25 times higher than that of B[a]P alone.

Because there is no other viable approach at this time, the derivation of the preliminary soil quality guidelines for the protection of human health was based on an adaptation of the relative potency scheme recommended by the World Health Organization (WHO/IPCS 1998; from Kalberlah *et al.* 1995), with minor modifications. This is a scheme based on the order of magnitude cancer potency. Any finer-scale assertions about relative potency for more generic application are hard to justify given the current state of knowledge and confounding influences such as route of exposure, or non-additive effects in complex PAH mixtures. It is not currently possible to develop different relative potency schemes across different exposure routes (oral, dermal, inhalation) or mixture types, owing to a lack of data.

The B[a]P PEFs used in the derivation of the human health protective guidelines, therefore, were:

Table 6-6: Benzo[a]pyrene Potency Equivalence Factors

Benz[a]anthracene	0.1
Benzo[a]pyrene	1
Benzo[b+j+k]fluoranthene	0.1
(note: since these three isomers closely co-elute using most contemporary analytical methods, the PEF applies specifically to the total of the three co-eluting PAHs)	
Benzo[g,h,i]perylene	0.01
Chrysene	0.01
Dibenz[a,h]anthracene	1
Indeno[1,2,3-c,d]pyrene	0.1

Regardless of the particulars of the benzo[a]pyrene relative potency scheme used, the best available information suggests that the predicted cancer potency of PAH-containing mixtures may deviate from the actual cancer potency in exposed mammalian species by one order of magnitude or more, and that the deviation may often be in the direction of under-predicting rather than over-predicting cancer risks.

Such uncertainty needs to be considered when undertaking risk assessments of PAH-containing mixtures based on potential for an increased incidence of cancer in humans.

7. DERIVATION OF HUMAN HEALTH AND ENVIRONMENTAL SOIL QUALITY GUIDELINES

7.1 Human Health Guidelines Derivation

Human health soil quality guidelines provide concentrations of contaminants in soil at or below which no appreciable risks to human health are expected. On all land uses, exposure through direct contact (i.e., ingestion, dermal contact, and inhalation) is considered, as well as exposure through the use of ground water as a source of drinking water. For volatile contaminants, exposure through vapours in indoor air is also considered. As most PAHs are not particularly volatile, guidelines for this pathway have not been derived here.

For agricultural land uses, guidelines are also calculated that take into consideration the intake of contaminants from produce, meat, and milk produced and consumed on-site. Food-based exposure scenarios may also be applicable to some residential/parkland sites. The plant, milk and livestock consumption pathway was not included in the derivation of the PAH soil quality guidelines. There is a lack of bioaccumulation in other mammalian species (even though PAHs might be expected to bioaccumulate based on their log K_{ow}) (Eisler, 1987). This is mainly due to the fact that PAHs are metabolized and therefore tend to not bioaccumulate. There is evidence that PAHs in soils may be transferred to various tissues of plants grown in those soils. However, PAH concentrations in the plant tissues are typically substantially lower than those in the soils in which they are grown (Edwards, 1983; Fismes *et al*., 2002; Kulhanek *et al*., 2005; Tao *et al*., 2004; Wild *et al*., 1992). The extent of uptake and the subsequent distribution within the plant tissues is highly variable for different PAH species and different plant species. There is currently insufficient information to evaluate this pathway quantitatively for the development of a PAH soil quality guideline. At sites where appreciable amounts of garden produce are consumed, a lower soil quality guideline value may need to be considered.

The steps used to derive human-health protective soil quality guidelines are generally similar to those used for undertaking a site-specific risk assessment, but in the absence of site-specific information given the generic applicability.

For human health protection, a human health-based soil quality guideline for direct contact (SQG_{DH}) was developed as documented below based on exposures via ingested or inhaled soil particles and dermal exposures. Dermal exposures and primary exposures via the gastrointestinal tract are expected to result in different kinds of cancers (i.e., melanomas and papillomatous growths versus foregut and internal organ tumours for dermal or oral exposures, respectively). However, cancer slope factors based directly on dermal exposures are currently not available. Therefore, the Health Canada oral slope factor of 2.3 $(mg \cdot kg^{-1} \cdot day^{-1})^{-1}$ was used to derive a soil quality guideline based on combined oral, dermal and inhalation exposure. Health Canada is currently in the process of developing a dermal slope factor for benzo(a)pyrene. A separate SQG_{DH} for dermal exposure may be published following the completion of the Health Canada dermal slope factor.

7.1.1 Benzo[a]pyrene SQG_{DH}

Canadian soil quality guidelines for benzo[a]pyrene for the protection of human health were established in 1996 (Health Canada, 1996) based on the 1996 CCME protocol, then edited and reprinted in 1999 (CCME, 1999b). The following derivation provides an update to the derivation of the CCME human health soil quality guidelines for benzo[a]pyrene. Human health guidelines were developed using default exposure factors from CCME (2006). These exposure factors are based on recent Health Canada guidance (HC, 2004a).

Benzo[a]pyrene has been classified as a "substance probably carcinogenic to humans" and therefore is considered as a non-threshold toxicant (a substance for which there is considered to be some probability of harm for the critical effect at any level of exposure), like other carcinogens. The appropriate derivation for a soil quality guideline, therefore, employs a critical RsD (risk-specific dose), based on lifetime incremental risks from soil ingestion (CCME, 2006). For all land uses, the adult was chosen as the receptor when considering lifetime cancer risk (CCME, 2006).

The CCME Soil Quality Guidelines Task Group recommends the development of a soil guideline for a non-threshold toxicant based on an incremental risk from soil exposure of 10^{-6} or 10^{-5} (CCME, 2006); i.e., an incremental risk of 1 in 1 million or 1 in 100,000, respectively. Health Canada considers an incremental risk of less than one in 10^5 to 10^6 to be "essentially negligible" for the purpose of deriving Maximum Acceptable Concentrations (MACs) for carcinogenic chemicals in drinking water (HWC 1989). Some provinces in Canada have adopted through policy an acceptable incremental lifetime cancer risk (ILCR) of 10^{-5}, and others have chosen 10^{-6}. Therefore, soil quality guidelines that are based on a cancer endpoint in humans are presented in this document at ILCRs of both 10^{-6} and 10^{-5}. Detailed derivations for each set of guideline are provided in Appendix I.

The Health Canada risk-specific dose (RsD) for benzo[a]pyrene is derived directly from a cancer slope factor estimated from Neal and Rigdon (1967). This study involved less than lifetime exposures (up to 197 days) to B[a]P administered in the diet to male and female inbred CFW-Swiss mice (n=23 to 73 for selected dose groups) aged 17 to 160 days old at the beginning of the study. The control group involved 289 individuals (n=171 males and 118 females). The doses of B[a]P were: 0, 1, 10, 20, 30, 40, 45, 50, 100, and 250 $mg \cdot kg^{-1}$ (ppm), mixed into a diet of Purina laboratory pellets. The doses were estimated from concentrations of B[a]P measured in food assuming a daily food consumption rate of 4 g. Mice were fed B[a]P for 100 days at the six lowest dose levels and for 107 to 197, 98 to 122, and 70 to 165 days for the three highest dose levels (50, 100, and 250 ppm), respectively. Mice were sacrificed between 88 and 219 days.

The study observed a duration- and dose-related increase in incidence of papillomas and squamous cell carcinomas in the forestomach of exposed mice. No forestomach tumours were observed in the 289 control mice. The majority of the tumours were papillomas,

and no metastases were observed. The exposure and incidence data for gastric tumours are tabulated in Table 7-1.

Table 7-1: Gastric Tumour Data from Neal and Rigdon (1967)

Oral Dose (mg B[a]P·g^{-1} food)	**Calculated Daily Dose** ($mg \cdot kg^{-1} \cdot day^{-1}$)(b.w.)	**Gastric Tumour Incidence**
0	0	0/289
0.001	0.078	0/25
0.01	0.781	0/24
0.02	1.56	1/23
0.03	2.34	0/37
0.04	3.13	1/40
0.045	3.52	4/40
0.05	3.91	24/34
0.1	7.82	19/23
0.25	19.5	66/73

A robust linear extrapolation was used by Health Canada with a surface area correction to determine the slope factor based on an increased incidence of forestomach tumours. At an incremental cancer risk level of 1 in 100,000 (i.e. 10^{-5}), an RsD of 0.00435 µg·kg $bw^{-1} \cdot d^{-1}$ was calculated and at an incremental cancer risk level of 1 in 1 million (i.e. 10^{-6}), an RsD of 0.000435 µg·kg $bw^{-1} \cdot d^{-1}$ was calculated. The study of Neal and Rigdon (1967) was also used by the USEPA (IRIS) to develop an oral slope factor of 7.3 $(mg \cdot kg^{-1} \cdot d^{-1})^{-1}$ based on the geometric mean of four slope factors obtained using different modeling procedures. At a similar incremental cancer risk level, an RsD of 0.0014 $\mu g \cdot kg^{-1} \cdot d^{-1}$ can be calculated, which is approximately four-fold lower than the Health Canada RsD. The OEHHA (1993) derived a cancer slope factor of 11.5 $(mg \cdot kg^{-1} \cdot day^{-1})^{-1}$.

Culp *et al.* (1998) studied lifetime exposures (2 years) to B[a]P administered in the diet to female B6C3F1 mice (n=48 per group) at doses of 0, 5, 25, and 100 ppm B[a]P in the feed. An increased incidence of forestomach tumours was observed and significant mortality was observed at the highest dose within one year of exposure. Gaylor *et al.* (2000) used these data with a linear multistage model to derive an oral slope factor of 1.2 $(mg \cdot kg^{-1} \cdot d^{-1})^{-1}$. This value is approximately six fold less than the current USEPA/IRIS value. Based on an incremental cancer risk level of 1 in 100,000, an RsD of 0.0083 $\mu g \cdot kg^{-1} \cdot d^{-1}$ can be calculated, which is approximately two-fold greater than the Health Canada RsD.

The Ontario Ministry of the Environment (OMOE, 1997) adopted a benzo[a]pyrene oral cancer potency (slope) factor of 0.18 $(mg \cdot kg^{-1} \cdot day^{-1})^{-1}$, also based on a linearized multistage model, but without the further use of an upper 95% confidence interval estimate of the low-dose slope. RIVM adopted a similar benzo[a]pyrene cancer slope factor of 0.2 $(mg \cdot kg^{-1} \cdot day^{-1})^{-1}$.

As discussed in Section 6.6, Schneider *et al.* (2002) proposed the use of a slope factor of 11.5 $(mg \cdot kg^{-1} \cdot day^{-1})^{-1}$ for risks based on oral exposures to coal tar and applied in consideration of only the mixture's B[a]P content, based on the Gaylor *et al.* (1998) studies on coal tar, but further suggested that this slope factor is not relevant for other PAH mixture types such as diesel engine emissions.

Thus, using two separate toxicological studies and variations in modeling approach, calculated RsD values for B[a]P range from 0.0014 to 0.0083 $\mu g \cdot kg^{-1} \cdot d^{-1}$, which is within an order of magnitude. While the more recent study by Culp *et al.* (1998) involved lifetime exposures compared to the study of Neal and Rigdon (1967), the latter study involved a greater number of dose levels (n=9 versus n=3 exposed groups) within the lower range. Furthermore, in the study of Culp *et al.* (1998), one of the three doses (highest) was associated with significant mortality. As a result, currently Health Canada recommends use of the oral RsD of 0.00435 $\mu g \cdot kg\ bw^{-1} \cdot d^{-1}$ for an ILCR of 10^{-5}, and 0.000435 $\mu g \cdot kg\ bw^{-1} \cdot d^{-1}$ for an ILCR of 10^{-6}, based on Neal and Rigdon (1967), for estimating risks to human health. Therefore, the Health Canada oral slope factor for benzo[a]pyrene $[(2.3\ (mg \cdot kg^{-1} \cdot day^{-1})^{-1}]$ (HC, 2004b), was chosen over the alternatives.

The preliminary soil quality guidelines for human health protection are derived assuming the life-long exposure of an adult, since the intent is to minimize cancer risks from benzo[a]pyrene in soil. An adult is generally considered to be the most sensitive receptor to carcinogens, since the probability of cancer increases with duration of exposure.

CCME (2006) allows for the derivation of a soil quality guideline for each of the following three exposure pathways (i) soil ingestion; (ii) particulate inhalation; and (iii) dermal absorption. In the case of B[a]P and other potentially carcinogenic PAHs, a critical review of the literature suggested a potential for systemic modes of toxic action based on either the gastrointestinal or pulmonary uptake route. In addition the major portion of bulk soils have a particle size greater than 2.5 µm which makes them incompatible with exposure regimes used in studies that are the basis for development of inhalation RfCs. It is not known whether dissolution in the upper respiratory tract of PAHs from greater than 2.5 µm can contribute to systemic dose since there are no studies addressing this question. It is expected that greater exposure occurs via the oral route than by inhalation and a significant portion of respired particulate matter is cleared to the GI tract. When these exposures are combined, the oral potency factor is greater, therefore combining these exposure pathways is considered conservative. For dermal exposures, the toxicological literature suggests different types of tumours, at different locations – e.g., as a result of skin painting studies. However, in the absence of a defensible dermal cancer slope factor for benzo(a)pyrene a single $PSQG_{DH}$ is calculated for the three exposure routes (dermal, oral, particulate inhalation) combined.

The preliminary soil quality guidelines for benzo[a]pyrene are:

$0.6\ mg \cdot kg^{-1}$ for an ILCR of 10^{-6}, or

$5.3\ mg \cdot kg^{-1}$ for an ILCR of 10^{-5}.

These guidelines are based on the assumption of a worst-case scenario in which the relative absorption factor for inhalation and ingestion is 100%. This factor assumes that

the bioavailability of PAHs in soil will be the same as the bioavailability of PAHs in the food administered to the test animals in the critical study used to derive the risk specific dose. The bioavailability of PAHs in soil via the oral and inhalation routes will vary significantly depending on factors such as soil conditions, the form of PAHs present in the soil and gastrointestinal conditions. Furthermore, there is insufficient information to relate the bioavailability of PAHs in soils to that in the food administered in the critical study. Therefore, a relative absorption factor of 100% was selected for exposure via inhalation and ingestion.

The magnitude of dermal absorption is expected to be lower than for oral absorption, and a dermal absorption factor was estimated from the available literature. Dermal bioavailability of PAHs in soil is influenced by soil characteristics, such as grain size and organic carbon content (Abdel-Rahman *et al*., 2002). The form of PAH contamination may also influence bioavailability. In particular, experimentally derived dermal absorption factors for benzo(a)pyrene from naturally weathered lampblack samples from a manufactured gas plant are one to two orders of magnitude lower than those for benzo(a)pyrene spiked soil (Stroo *et al*., 2005).

The total percutaneous absorption of benzo(a)pyrene in soil observed in *in vivo* tests with female rhesus monkey was 14.8 % after 24 hours and 15.8 % after 48 hours of exposure (Wester *et al*., 1990). A percent absorption of 56.4 after 24 hours and 49.7 after 48 hours for benzo(a)pyrene in soil was measured by Moody *et al*. (in press) in *in vitro* studies using human skin. Shatkin *et al*. (2002) developed a fugacity-based model to predict dermal uptake of benzo[a]pyrene from soil, and compared the model predictions with nine experimental data points, which agreed within a factor of two. An upper bound estimate based on this study for dermal absorption over a 24 h period is 34%, which is the modeled mean plus one standard deviation. A dermal absorption factor of 34%, based on the study by Shatkin *et al.* (2002), was selected for the derivation of the soil quality guideline.

Full details of the derivation of the guidelines are provided in Appendix I.

These preliminary soil quality guidelines are interpreted to be applicable not just to residential land use sites, but also agricultural, commercial and industrial areas. Especially in the case of commercial and industrial area, humans are expected to be exposed less than 24 h/day over an entire year, and for less than the entire lifespan of an individual. Nonetheless, the available experimental data on cancer induction in mammalian species by B[a]P is for equivalent exposures that are much less than the entire life expectancy of the test animals, and there remains considerable uncertainty about whether incremental cancer risks decrease in direct proportion to any amortization of human exposures based on periods of time approaching years to decades.

The major sources of uncertainty in these estimates are deemed to be as follows:

- cancer slope factor estimate: range of various proposed values for B[a]P of 0.2 to 11 $(mg \cdot kg^{-1} \cdot day^{-1})^{-1}$ relative to Health Canada value of 2.3 $(mg \cdot kg^{-1} \cdot day^{-1})^{-1}$:

- no consideration of benzo[a]pyrene exposures via diet.

This second point is deemed to be of only minor importance. CCME (1999a) provided an estimate of the average daily dietary intake (EDI: estimated daily intake) by Canadians of benzo[a]pyrene, which is 2.0×10^{-3} to 5.9×10^{-3} $\mu g \cdot kg\ bw^{-1} \cdot day^{-1}$, excluding additional PAH exposures through cigarette smoking. While this is in fact higher than the risk-specific dose used by Health Canada to develop a human health-based soil quality guideline (0.4×10^{-3} $\mu g \cdot kg\ bw^{-1} \cdot day^{-1}$, for an ILCR of 10^{-6}), it is irrelevant. For carcinogens, it is assumed that some level of risk exists at any level of exposure other than zero. Therefore, management of cancer-related human health risks is a policy decision based on incremental risks, including those beyond background exposures (CCME, 2006).

7.1.2 Carcinogenic PAH SQG_{DH} Based on B[a]P Relative Potencies

The basis of and limitations associated with the assessment of human cancer risks based on PAH cancer potency equivalence factors (PEFs) relative to benzo[a]pyrene, along with a B[a]P cancer slope factor, is discussed in detail in Section 6.6. Limitations of the approach notwithstanding, it remains the best of several carefully evaluated approaches to manage the cancer risks of PAH-containing mixtures.

The CCME policy-derived human health protection goals remain the same for benzo[a]pyrene and for PAH mixtures with B[a]P-like cancer risks: Concentrations of carcinogenic PAHs in soils should be commensurate with an incremental lifetime cancer risk (ILCR) of 10^{-5} to 10^{-6}.

If there is adequate confidence that the application of B[a]P Potency Equivalence Factors (B[a]P PEFs) predicts the cancer risks of PAH mixtures, then a SQG_{DH} to account for the broader range of carcinogenic PAHs would be the same as if benzo[a]pyrene were the only carcinogenic PAH present; i.e., –0.6 $mg \cdot kg^{-1}$ B[a]P (Total Potency Equivalents) (for a 10^{-6} ILCR) or 5.3 $mg \cdot kg^{-1}$ B[a]P TPE (10^{-5} ILCR). See Figure 8-2 for an example of how to calculate a B[a]P Total Potency Equivalents (TPE) from a mixture of carcinogenic PAHs.

There are two implicit issues here. The first is the assumption of additivity of measured and recognized carcinogenic constituents in the mixture. The second is associated with other compounds in complex mixtures that have not been adequately characterized, either analytically or from a carcinogenic/toxicological perspective.

The World Health Organization B[a]P Potency Equivalence Factor scheme was adopted after critical review (Section 6.6), as in Table 6-6. This scheme reflects an explicit

understanding that the relative cancer potencies can at best be estimated within an order of magnitude, and any greater precision is not warranted relative to the state of knowledge. Further, the scheme is similar to that used in other jurisdictions; e.g., as proposed by the USEPA.

7.1.2.1 Creosote and Coal Tar

It was noted in Section 6.6 that a few studies have compared, predicted and measured potencies of PAH-containing mixtures, and that use of B[a]P relative potency schemes is problematic – since predicted potency may underestimate true cancer potency by up to approximately 15-fold. However, based on studies reviewed in Section 6.6, it is expected that in most cases the degree of under-prediction would be three-fold or less.

Aspects of scientific uncertainty that might lead to an under-prediction or over-prediction of cancer risks based on human exposures to soils contaminated with a mixture of PAHs are outlined in the following table.

Table 7-2: Factors contributing to over/under prediction of toxic potency

Factors contributing to an over-prediction of risks (over-conservatism)	Factors contributing to an under-prediction of risks (lack of conservatism)
• B[a]P cancer slope factor is conservative relative to some jurisdictions and recommendations	• B[a]P cancer slope factor is less conservative than USEPA or California EPA slope factor.
• Overall potency of PAH-containing mixtures might be less than additive (or different PAH might contribute to different cancer types at a different anatomic site)	• Overall potency of PAH-containing mixtures might be greater than predicted assuming additivity.
• Bioavailability of higher MW PAHs from soil likely to be <100%, while derivation assumes 100% relative bioavailability from soil via ingestion and particulate inhalation pathways	• Multimedia exposures not combined for B[a]P SQG_{DH} derivation.
	• PAHs in mixture not accounted for (especially 4-6 ring alkyl-PAHs) that are not currently but may in the future be proven to be carcinogenic

Above all, it is clear that prediction of cancer potency in laboratory rodent models using B[a]P relative potency schemes is likely an under-prediction of measured potency for rodents exposed to coal-tar, but not based on the other PAH-containing mixtures examined.

In light of this, the SQG_{DH} used in concert with the B[a]P Potency Equivalence Factors (Table 6-6) may not be applicable to soils contaminated with coal tar or creosote.

To account for this, a three-fold safety factor should be employed, therefore, when calculating B[a]P TPEs for sites affected by creosote or coal tar before comparison with the SQG_{DH}. In other words, the soil-borne concentrations of creosote or coal tar should be multiplied by a factor of 3 when the risks posed by on-site contamination with these mixtures is being evaluated against the SQG_{DH}. See Figure 8-2 for an example of how to calculate a B[a]P Total Potency Equivalents (TPE) from carcinogenic PAHs found in creosote, or coal tar mixtures. In cases where site information is insufficient to determine whether PAH contamination has resulted from a coal tar or creosote source, the uncertainty factor should be applied.

7.1.3 Evaluation of Soil Thresholds for Benzo[a]pyrene Based on Infant Pica Soil Ingestion Exposure

Concern is often expressed about not just the health effects of long-term human exposures, but also of potential risks associated with acute exposures to high concentrations of a contaminant in soil. Young children who ingest large amounts of soil represent a worst-case scenario for acute exposures. The deliberate ingestion of soil by some young children is called *pica* soil ingestion. No provisions exist within the CCME protocols for the evaluation of soil thresholds assuming pica-type exposures; however, a check of this exposure scenario was calculated herein for those with an interest in the issue.

Assumptions and data:

- Assumes a short-term exposure and acute-type effects only. The calculation does not consider non-threshold type effects such as cancer.

- The sensitive receptor for threshold-type effects is an infant (between the ages 6 months to 6 years).

- Per USEPA guidance, it was assumed that a child involved in pica soil ingestion generally consumes a maximum of 5,000 $mg \cdot day^{-1}$ (5 $g \cdot d^{-1}$ or 0.005 $kg \cdot d^{-1}$).

- ATSDR (1995c) did not develop any acute oral Minimum Risk Levels (MRLs) for PAHs due to the absence of guiding studies. There are, however, four ATSDR (1997) MRLs developed for intermediate exposures (15 to 364 days of exposure):

    - Acenaphthene: 0.6 $mg \cdot kg^{-1} \cdot day^{-1}$ (300-fold UF).
    - Fluorene: 0.4 $mg \cdot kg^{-1} \cdot day^{-1}$ (300-fold UF).
    - Anthracene: 10 $mg \cdot kg^{-1} \cdot day^{-1}$ (100-fold UF).
    - Fluoranthene: 0.4 $mg \cdot kg^{-1} \cdot day^{-1}$ (300-fold UF).

- In addition to this, a screening-level TDI can be developed for benzo[a]pyrene, as follows:

    Nousiainen *et al.* (1984) conducted an acute oral exposure of rats and mice. B[a]P was orally administered at 150 $mg \cdot kg^{-1} \cdot day^{-1}$, once per day, for each of four days. A NOAEL for this study is 150 $mg \cdot kg^{-1} \cdot day^{-1}$. No acute studies were identified with relevant acute toxicological responses at lower doses. Applying a 10-fold uncertainty factor to this NOAEL to account for interspecies differences, as well as another 10-fold uncertainty factor to account for differences in sensitivity yields a B[a]P screening level TDI of 1.5 $mg \cdot kg^{-1} \cdot day^{-1}$.

The acceptable threshold, $PSQG_{DH}$, for PICA exposure based on acute, threshold-type responses can be calculated using the following equation:

$$PSQG_{DH\text{-}PICA} = \frac{(TDI - EDI) \times SAF \times BW}{[(AF_G \times SIR) + (AF_S \times SR) + (AF_L \times IR_S)] \times ET} + BSC \qquad (1)$$

where -

$PSQG_{DH\text{-}PICA}$	=	preliminary direct human health-based soil quality guideline for pica soil ingestion ($mg \cdot kg^{-1}$)		(calculated)
TDI	=	Tolerable daily intake ($mg \cdot kg^{-1}$ per day)	=	1.5 mg B[a]P·day^{-1} (see above)
SAF	=	Soil allocation factor		Disregard for PICA calculation
BW	=	Body weight	=	16.5 kg for a toddler (HC, 2004a)
BSC	=	background soil concentration ($mg \cdot kg^{-1}$)		Disregard for PICA calculation
AF_G	=	relative absorption factor for gut (unitless)	=	1.0, or 100% (assumed)
AF_L	=	relative absorption factor for lung (unitless)	=	1.0, or 100% (assumed)
AF_S	=	relative absorption factor for skin (unitless)	=	0.34 (Shatkin *et al*., 2002) (to convert dermal dose to oral equivalent)
SIR	=	soil ingestion rate ($kg \cdot d^{-1}$)	=	5×10^{-3} kg·day^{-1} (USEPA)
IR_S	=	soil inhalation rate ($kg \cdot d^{-1}$)	=	9.3 $m^3 \cdot d^{-1}$ x 0.76 $\mu g \cdot m^{-3}$ (=7.1 $\mu g \cdot d^{-1}$ or 7.1×10^{-9} $kg \cdot d^{-1}$) (HC, 2004a) *omitted as insignificant relative to pica ingestion*
SR	=	soil dermal contact rate ($kg \cdot d^{-1}$)	=	6.9×10^{-5} kg·day^{-1} [CCME (2006): (hand surface area of 0.043 m^2 x soil adherence factor of 0.001 $kg \cdot m^{-2} \cdot day^{-1}$)+(other surface area of 0.25 m^2 x soil adherence factor of 0.0001 $kg \cdot m^{-2} \cdot day^{-1}$) *omitted as insignificant relative to pica ingestion*
ET	=	exposure term (unitless)	=	1.0; i.e., acute exposure duration is commensurate with expected timescale of TDI

Therefore –

$$PSQG_{DH\text{-}PICA} = \frac{1.5 \text{ mg B[a]P} \cdot kg^{-1} \cdot day^{-1} \times 16.5 \text{ kg bw}}{}$$

$$5 \times 10^{-3} \text{ kg soil} \cdot \text{day}^{-1}$$

$$= 4{,}950 \text{ mg B[a]P} \cdot \text{kg}^{-1} \text{ soil or } 4{,}950 \text{ µg} \cdot \text{g}^{-1} \text{ soil}$$

Based on similar calculations for the other four PAHs for which ATSDR intermediate exposure MRLs are available, the following pica-based soil thresholds would be calculated.

Acenaphthene	1,980 mg·kg^{-1} soil.
Fluorene	1,320 mg·kg^{-1} soil.
Anthracene	33,000 mg·kg^{-1} soil.
Fluoranthene	1,320 mg·kg^{-1} soil.

These numbers seem very high. However, it bears remembering that the B[a]P risk-specific dose (RsD), for an ILCR of 10^{-6}, used in the calculation based on non-threshold responses was 4.4 x 10^{-4} µg·kg bw^{-1}·day^{-1} (equivalent to 7.3 x 10^{-3} µg·day^{-1} for a 16.5 kg toddler), while the acute oral screening level TDI used above was 1.5 mg·kg bw^{-1}·day^{-1}, or 1.5 x 10^{3} µg·kg bw^{-1}·day^{-1}, a difference of almost six orders of magnitude.

7.1.4 Use of Ground Water as a Drinking Water Source – Benzo[a]pyrene

Potable water may be drawn from surface water or groundwater. Soils are hydrologically linked to both surface and groundwater systems, and a major concern with soil contamination is that it can lead to groundwater contamination. Thus, CCME soil quality guidelines include provisions for preventing human health risks associated with the transfer of contaminants from contaminated soils to groundwater systems.

In the generic case, it is conservatively (prudently) assumed that an aquifer directly adjacent to or underlying a site of interest may be used as a source of drinking water for humans presently or potentially in the future. A one-dimensional, four-compartment groundwater model is described in CCME (2006), and further described under section 7.3 for freshwater life protective pathways. A key difference in site assumptions used to estimate acceptable soil concentrations for groundwater mediated transfers to exposure media between surface waters that support aquatic life and potable water supplies is the distance between the point of interest for exposure/use and the lateral edge of the contaminated soil mass. In the case of freshwater life protection, it is assumed in the generic case that contaminants in groundwater travel ≥ 10 m prior to entering a surface water body that may contain aquatic life. In the case of potable water supplies, it is conservatively assumed that there is no lateral separation between the contaminated soil and point of extraction (groundwater well).

The model used to calculate soil quality guidelines for the protection of potable water (SQG_{PW}) considers four processes, as does for the calculation of soil quality guidelines for the protection of freshwater life (SQG_{FL}):

1. partitioning from soil to leachate;
2. transport of leachate from base of contamination to water table;
3. mixing of leachate and groundwater; and,
4. groundwater transport downgradient to the receptor.

Each of these processes results in the sequential dilution of the contaminant from the source to the receptor. In the case of generic guidelines for SQG_{PW}, it is assumed that the base of the contaminated zone is at the water table, and hence there is no dilution in process #2. In addition, no offset is assumed between source and receptor, so there is no dilution in process #4. The following equations are used to calculate SQG_{PW}; guideline calculations for both coarse and fine soil are provided below.

$$SQG_{PW} = C_L \left\{ K_d + \left(\frac{\theta_w + H'\theta_a}{\rho_b} \right) \right\} \qquad (2)$$

where:

SQG_{PW}	=	soil quality guideline for the protection of potable groundwater ($mg \cdot kg^{-1}$);
C_L	=	allowable leachate concentration at source ($mg \cdot L^{-1}$); for default guidelines, this is assumed to be equal to C_z (see calculation below)
K_d	=	distribution coefficient ($cm^3 \cdot g^{-1}$) (see calculation below)
θ_w	=	water filled porosity (unitless); 0.119 for coarse soils, 0.168 for fine soils (CCME 2006)
H’	=	dimensionless Henry’s Law constant (see calculation below)
θ_a	=	air-filled porosity (unitless); 0.241 for coarse soils, 0.302 for fine soils (CCME 2006)
ρ_b	=	soil bulk density in contaminant partitioning zone ($g \cdot cm^{-3}$); 1.7 for coarse soils, 1.4 for fine soils (CCME 2006)

$$C_z = C_{gw} \left\{ 1 + \left(\frac{Z_d K_H i}{IX} \right) \right\} \qquad (3)$$

where:

C_z	=	allowable chemical concentration in leachate at the water table ($mg \cdot L^{-1}$)
C_{gw}	=	allowable chemical concentration in groundwater at the source ($mg \cdot L^{-1}$); for default guidelines, this is assumed to be the same as the allowable chemical concentration in water at the receptor (i.e., drinking water guideline)

Z_d	=	average thickness of mixing zone (m) (see calculation below)
K_H	=	hydraulic conductivity in the saturated zone ($m \cdot y^{-1}$); 320 $m \cdot y^{-1}$ for coarse soils, 32 $m \cdot y^{-1}$ for fine soils (CCME 2006)
i	=	hydraulic gradient (unitless); 0.028 (CCME 2006)
I	=	infiltration rate ($m \cdot y^{-1}$); 0.28 $m \cdot y^{-1}$ for coarse soils, 0.20 $m \cdot y^{-1}$ for fine soils (CCME 2006)
X	=	length of source parallel to groundwater flow (m); 10 m (CCME 2006)

$$K_d = K_{oc} \times f_{oc} \qquad (4)$$

where:

K_d	=	distribution coefficient ($cm^3 \cdot g^{-1}$)
K_{oc}	=	organic carbon partitioning coefficient ($L \cdot kg^{-1}$); 2,200,000 $L \cdot kg^{-1}$ for benzo[a]pyrene (see discussion below)
f_{oc}	=	organic carbon fraction of soil ($g \cdot g^{-1}$); default value of 0.005 assumed (CCME 2006)

$$H' = H \times 42.32 \qquad (5)$$

where:

H’	=	dimensionless Henry’s Law constant
H	=	Henry’s Law constant ($atm\text{-}m^3 \cdot mol^{-1}$); 1.13 x 10^{-6} for benzo[a]pyrene (from Table 2-4)

$$Z_d = r + s \qquad (6)$$

where:

Z_d	=	average thickness of mixing zone (m)
r	=	mixing depth available due to dispersion and diffusion (m); 0.1 (CCME 2006)
s	=	mixing depth available due to infiltration rate and groundwater flow rate (m); (see calculation below)

$$s = d_a \left\{ 1 - e^{-\frac{2.178\,XI}{K_H i d_a}} \right\} \qquad (7)$$

where:

s	=	mixing depth available due to infiltration rate and groundwater flow rate (m)
d_a	=	depth of unconfined aquifer (m); 5 m (CCME 2006)
X	=	length of source parallel to groundwater flow (m); 10 m (CCME 2006)

I	=	infiltration rate ($m \cdot y^{-1}$); 0.28 $m \cdot y^{-1}$ for coarse soils, 0.20 $m \cdot y^{-1}$ for fine soils (CCME 2006)
K_H	=	hydraulic conductivity in the saturated zone ($m \cdot y^{-1}$); 320 $m \cdot y^{-1}$ for coarse soils, 32 $m \cdot y^{-1}$ for fine soils (CCME 2006)
i	=	hydraulic gradient (unitless); 0.028 (CCME 2006)

The log K_{oc} for benzo[a]pyrene has been reported to range from 6.0 to 6.7 (see Table 2-4). Therefore, taking the geometric mean of these values, 6.34, and taking the anti-log, a K_{oc} value of approximately 2,200,000 was estimated.

The Maximum Acceptable Concentration (MAC) in drinking water for benzo[a]pyrene, as established by Health Canada, is 0.00001 $mg \cdot L^{-1}$ (HWC 1988).

Substituting these values into the above equations, SQG_{PW} for B[a]P are estimated to be 0.37 $mg \cdot kg^{-1}$ for coarse-textured soils and 0.27 $mg \cdot kg^{-1}$ for fine-textured soils.

The first dilution factor (DF1) describing a three phase partitioning between the contaminant on the soil, in soil interstitial water, and in soil gas, is the only major mechanism for achieving reductions in mass between the contaminated soil and the point of potable water extraction, based on the generic case protocol provided in CCME (2006). This is likely highly conservative relative to the vast majority of actual PAH contaminated sites in Canada. In addition, it should be noted that the calculation of DF1 is highly sensitive to the K_{OC} value used. Literature-derived empirical or modeled K_{OC} estimates can often vary by an order of magnitude for more highly non-polar substances such as the higher molecular weight PAHs that are of interest here. Such an order of magnitude uncertainty directly translates into an order of magnitude value in DF1, as well as the resulting SQG_{PW}. The K_{OC} for a substance can also vary by as much as three orders of magnitude when determined in the field, depending on various site-specific conditions, such as the forms of organic carbon present in the soil which can vary in their sorption characteristics (Hawthorne *et al.*, 2006). Soil-specific K_{OC} (and K_d) values are often higher than default values reported in the literature (Dondelle and Loehr, 2002; Hawthorne *et al.*, 2006). Therefore, users may wish to consider calculating Tier 2 SQG_{PW} values by using K_{OC} values derived on a site-specific basis as parameter values for the above equations.

For the protection of groundwater as a source of raw water for drinking, the level in coarse soils should generally not exceed 0.37 $mg \cdot kg^{-1}$ (or $\mu g \cdot g^{-1}$) B[a]P or its TEQ in dry soil.

7.1.5 Use of Groundwater for Drinking Water – Other PAHs

Canadian Guidelines for Drinking Water Quality exist only for benzo[a]pyrene; i.e., 0.01 $\mu g \cdot L^{-1}$. The Canadian Guidelines for Drinking Water Qulaity for benzo[a]pyrene was used in concert with the WHO/IPCS (1998; from Kalberlah *et al.* (1995)) relative potency factors (adopted herein as B[a]P PEFs) to derive "Source Guidance Values for Groundwater" (SGVG) for each of the carcinogenic PAHs of interest.

Table 7-3: Calculations for PAH Soil Quality Component Values Protective of Drinking Water Quality Based on Cancer Risks

PAH	log K_{OC}	Henry's Law Constant ($atm \cdot m^3 \cdot mol^{-1}$)	Canadian GDWQ ($\mu g \cdot L^{-1}$)	B[a]P PEF	SGVG ($\mu g \cdot L^{-1}$)[a]	SQG_{PW} component value ($mg \cdot kg^{-1}$)
benz[a]anthracene	5.3	3.35×10^{-6}		0.1	0.1	**0.33**
chrysene	5.1	9.46×10^{-5}		0.01	1.0	**2.1**
benzo[b+j]fluoranthene	4.97*	1.05×10^{-5} *		0.1	0.1	**0.16**
benzo[k]fluoranthene	4.3	8.29×10^{-7}		0.1	0.1	**0.034**
benzo[a]pyrene	6.34*	1.13×10^{-6}	**0.01**	1	--	**0.37**
benzo[g,h,i]perylene	5.61	1.41×10^{-7}		0.01	1.0	**6.8**
indeno[1,2,3-c,d]pyrene	6.2	1.60×10^{-6}		0.1	0.1	**2.7**
dibenz[a,h]anthracene	6.14*	1.47×10^{-8}		1	0.01	**0.23**

*Geometric mean calculated from the range of values given in Table 2-4.

[a] Source guidance values for groundwater were calculated by dividing the Canadian guideline for drinking water quality for benzo[a]pyrene by the relevant B[a]P PEF. This method for developing the SGVG is a deviation from the normal procedure (see section 5.3.2 of CCME 2006).

Table 7-3 summarizes the calculated soil quality guidelines for other potentially carcinogenic PAHs, using equations (2) to (7), above. The SQG_{PW} component value presented in Table 7-3 and calculated in equation (2) above is identical to the $SQG_{PW.}$ The addition of "component values" to SQG_{PW}s has been done to emphasize that they are to be used differently than traditional SQG_{PW}s, i.e. they are not to be used as stand alone soil quality guidelines (see IACR below).

In summary, the groundwater protection soil threshold values based on drinking water-related cancer risks from each PAH would be as follows, based on the use of B[a]P PEFs:

Benz[a]anthracene	0.33 $mg \cdot kg^{-1}$
Chrysene	2.1 $mg \cdot kg^{-1}$
Benzo[b+j+k]fluoranthene	0.16 $mg \cdot kg^{-1}$

Benzo[g,h,i]perylene	6.8 $mg \cdot kg^{-1}$
Indeno[1,2,3-c,d]pyrene	2.7 $mg \cdot kg^{-1}$
Dibenz[a,h]anthracene	0.23 $mg \cdot kg^{-1}$

Note that benzo[b]fluoranthene and benzo[j]fluoranthene tend to strongly co-elute under most gas chromatographic conditions. Furthermore, resolution between benzo[b]fluoranthene, benzo[j]fluoranthene, and benzo[k]fluoranthene is difficult to achieve when all three isomers are present in the soil matrix. Therefore, these three isomers have been considered together in deriving SQG_{HH} values.

The calculation of a corresponding soil quality guideline for each potentially carcinogenic PAH represents a different approach for PAH-containing mixtures than was taken for the exposure scenario based on direct soil ingestion (Section 7.1.2). This is because it is difficult to apply groundwater fate models to chemical mixtures that exhibit a potential range of K_{OC} and K_d values. Subsurface chemical fate is expected to be particularly sensitive to variations across PAH compounds in the organic carbon – water partitioning tendency (K_{OC} values), which vary by approximately four orders of magnitude (range of $\log_{10} K_{OC}$ values: 2.7 to 6.7; Table 2-4).

The relative composition of different PAHs would be expected to change between the contaminated soil source and the drinking water source based on particulars of groundwater-mediated transport.

A major implication of this approach, however, is that the additive carcinogenicity of the PAH mixture is not accounted for. The lifetime excess cancer risk has been determined on the basis of the incremental risk from exposure to each compound individually, which may fall below the risk level associated with the benzo[a]pyrene Canadian Guideline for Drinking Water Quality, but the cumulative risk from exposure to several PAHs in water, simultaneously, would almost certainly exceed the acceptable risk threshold. Conceptually, a test for the additive cancer risks of a mixture of PAHs can be carried out by dividing the known concentration of individual PAHs in a soil, or the projected concentration following remedial activity, by the individual groundwater protective soil values (i.e. SQG_{PW} component values) shown above; i.e., –

Index of Additive Cancer Risk (Drinking Water Check) = (8)

$$
\begin{aligned}
&[\text{Benz[a]anthracene}] / 0.33\ mg \cdot kg^{-1} \\
+\ &[\text{Chrysene}] / 2.1\ mg \cdot kg^{-1} \\
+\ &[\text{Benzo[b+j+k]fluoranthene}] / 0.16\ mg \cdot kg^{-1} \\
+\ &[\text{Benzo[a]pyrene}] / 0.37\ mg \cdot kg^{-1} \\
+\ &[\text{Benzo[g,h,i]perylene}] / 6.8\ mg \cdot kg^{-1} \\
+\ &[\text{Indeno[1,2,3-c,d]pyrene}] / 2.7\ mg \cdot kg^{-1} \\
+\ &[\text{Dibenz[a,h]anthracene}] / 0.23\ mg \cdot kg^{-1}
\end{aligned}
$$

The resulting Index of Additive Cancer Risk (IACR) value is equivalent to a hazard index. An IACR > 1.0 indicates possible incremental lifetime cancer risk (ILCR) based

on drinking water exposures that is greater than the accepted level of risk associated with the benzo[a]pyrene drinking water guideline.

IACR ≤ 1.0	⇒	ILCR ≤ acceptable threshold
IACR > 1.0	⇒	ILCR > acceptable threshold

The actual acceptable concentrations of individual potentially carcinogenic PAHs at a site will depend on and can be predicted from the particular composition of the contaminant mixture. In practice, the acceptable threshold for each PAH can be estimated with prior knowledge of the PAH composition at the site in question. For example, it is possible, using actual site data, to express the percent contribution of each of the carcinogenic PAHs to the total higher molecular weight PAH concentration. This in turn can be used for the entire mixture to evaluate the concentration of total higher molecular weight PAHs for the site at which there is predicted to be an exceedance of the acceptable threshold for incremental lifetime cancer risk. By setting the IACR at 1.0, and the individual PAHs as a proportion of the total carcinogenic PAH concentrations, it is possible to calculate the concentration for each of the PAHs beyond which an acceptable ILCR would be exceeded.

7.2 Environmental Guidelines Derivation

7.2.1 Direct Soil Contact SQG_{SC} for the Protection of Soil Invertebrates and the Plant Community

7.2.1.1 Agricultural and Residential/Parkland Land Use

Three options are available for deriving guidelines for agricultural and residential/parkland land use in consideration of direct soil contact and the maintenance of soil ecological functioning. Depending on the availability and quality of data on toxicity of substances to soil invertebrates and plants, a guideline can be calculated using:

1) Weight-of-Evidence (WOE) approach to calculate a "Threshold Effects Concentration" (TEC), provided minimum data requirements are met; depending on the data available, this approach may be used with all data standardized at an EC_{25} level, or it may use a combination of effects and no-effects data; or

2) Estimation of a TEC by extrapolating from the lowest observable adverse effect concentration (LOAEC) in soil, provided an appropriate LOAEC endpoint is available; or

3) Estimation of a TEC from a median effective concentration (EC_{50}) or median lethal concentration (LC_{50}).

For the WOE approach, when sufficient EC_{25} data are not available, as is the case for the PAH datasets, all available "effects" (LOEC, LOAEC, EC_{50}, LC_{50}) and "no-effects" (e.g., NOEC) data are gathered. Other types of effects data, EC_X or LC_X, may be included depending on the value of 'X' and on professional judgment about the value of the data in describing toxicological thresholds for various taxa. At least ten data points from not less than three studies is required for the WOE approach, with data for at least two species of both soil invertebrates and plants.

Table 7-4 shows the available toxicity endpoints data derived from a comprehensive literature review. The detailed data are tabulated in Appendices III and IV. Note that the vast majority of all available data comprised "effects" as opposed to "no-effects" endpoints, and the data were also dominated by median lethal or median effects concentration data. All plant data were deemed to be non-lethal endpoints (growth based on length or biomass; germination/emergence). For the soil invertebrates, the majority of the data come from a series of recent studies by Sverdrup *et al.*, (2001, 2002a, 2002b, 2002d, 2002e), with an emphasis on the species *Folsomia fimetaria, Eisenia veneta*, and *Enchytraeus crypticus*.

There is very limited information on the toxicity of the PAHs of interest to soil invertebrates and plants, and the minimum data requirements for using the WOE approach are met only for fluoranthene and benzo[a]pyrene. There were 92 soil invertebrate endpoints for all PAHs, for which 14 were NOEC data. Only 25 plant toxicity endpoints were found in the literature that were deemed to be useful for assessing PAH phytotoxicity thresholds, and none of these were NOEC data.

Table 7-4: Summary of soil invertebrate and plant PAH toxicity data.

(Potentially carcinogenic, higher MW PAHs that are the focus of this derivation are shown in bold.)

PAH	Number of independent studies	Number of soil invertebrate taxa	Number of plant taxa	Total number of endpoints
Lower MW PAH				
Naphthalene	1	1	0	2
Acenaphthene	1	1	0	2
Acenaphthylene	1	1	0	2
Fluorene	3	3	0	10
Anthracene	4	1	8	16
Phenanthrene	4	4	0	15
Higher MW PAH				
Fluoranthene	5	4	2	16
Pyrene	4	3	0	12
Benz[a]anthracene	2	1	1	4
Chrysene	1	1	0	2
Perylene	2	1	2	4
Benzo[b]fluoranthene	1	1	0	2

Benzo[k]fluoranthene	1	1	0	2
Benzo[a]pyrene	5	3	2	14
Benzo[g,h,i]perylene	0	0	0	0
Indeno[1,2,3-cd]pyrene	1	1	0	2
Dibenzo[a,h]anthracene	2	1	1	4
Other PAH				
9H-Fluorene	3	4	0	8

The available data for fluoranthene and benzo[a]pyrene are illustrated in Figures 7-1 and 7-2, respectively. The species sensitivity distribution for fluoranthene is based on data for four soil invertebrate species (*Eisenia fetida, Eisenia veneta, Enchytraeus crypticus,* and *Folsomia fimetaria*) and two plant species (*Avena sativa* and *Brassica rapa*). The species sensitivity distribution for benzo[a]pyrene is based on data for three soil invertebrates (*Eisenia fetida, Enchytraeus crypticus,* and *Folsomia fimetaria*) and two plant species (*Lupinus albus* and *Cannabis sativa*).

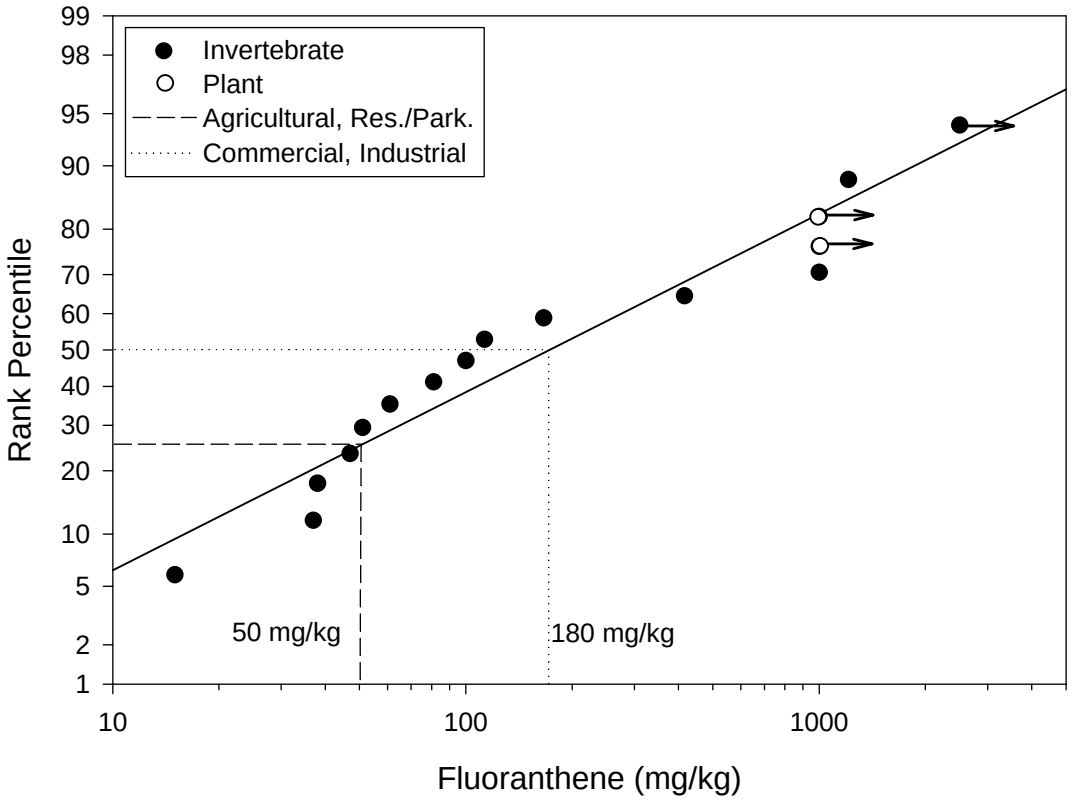


Figure 7-1: Ranked ecotoxicity data on effects of fluoranthene to soil invertebrates and plants.

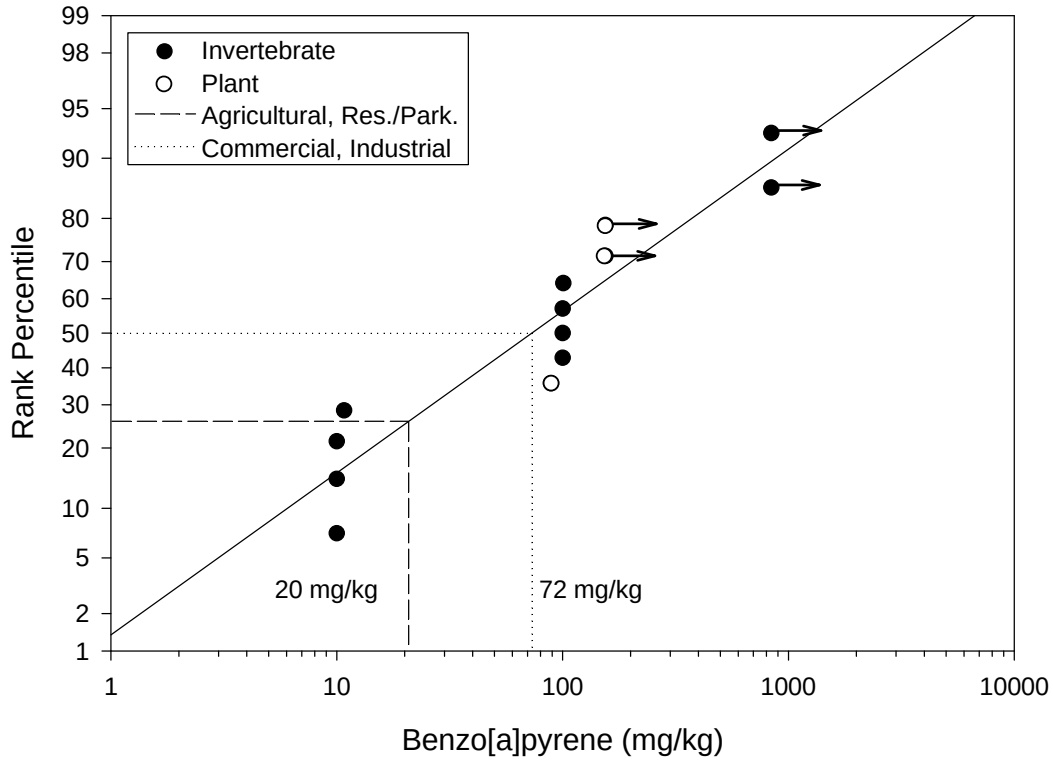


Figure 7-2: Ranked ecotoxicity data on effects of benzo(a)pyrene to soil invertebrates and plants.

As illustrated in Figure 7-1, a soil contact guideline for fluoranthene of 50 $mg \cdot kg^{-1}$ was derived for agricultural and residential/parkland land uses. For benzo[a]pyrene (Figure 7-2), a soil contact guideline of 20 $mg \cdot kg^{-1}$ was derived for agricultural and residential/parkland land uses.

Sverdrup *et al.* (2002a) provide mortality and reproductive toxicity data for 16 unsubstituted PAHs on the springtail *Folsomia fimetaria.* These authors developed the following relationship between reproductive toxicity and the K_{ow} of the PAH, after excluding data for anthracene which appears to be more toxic to soil invertebrates and plants than might be accounted for by narcosis-type effects alone.

$$\text{Log } EC_{10} = -0.97 \log K_{OW} + 4.0 \ (r^2 = 0.80) \qquad (9)$$

Note that the EC_{10} concentration was estimated on the basis of calculated molar pore-water concentration of the PAH (using K_{OW}, the known organic carbon content of the soil and the percent saturation).

This applied only to the PAHs with log $K_{OW} \leq 5.2$. The higher molecular weight PAHs (benz[a]anthracene, chrysene, benzofluoranthenes, perylene, benzo[a]pyrene, indeno[1,2,3-cd]pyrene and dibenz[a,h]anthracene) were all non-toxic to *F. fimetaria* (the LC_{50} values or EC_{10} values for reproduction were estimated at greater than the maximum exposure concentration used; i.e., > 360 to > 1,030 $mg \cdot kg^{-1}$ soil).

Unfortunately the above-described stochastic model accounts for toxicity in only one single test organism and for a single soil type. Further, the relationships described in Sverdrup *et al.,* (2002a) are based on regressing the EC_{10} or LC_{50} values, converted to estimated pore-water molar concentrations using K_{OW} values on K_{OW}, and the regression itself captures some degree of autocorrelation, therefore. There was no relationship between the toxicity (based on mortality or reproductive impairment) of individual PAHs expressed on the basis of initial soil concentration and log K_{OW} or log K_{OC} even though all data were from tests using the same soil type with a standardized organic carbon content and percent saturation.

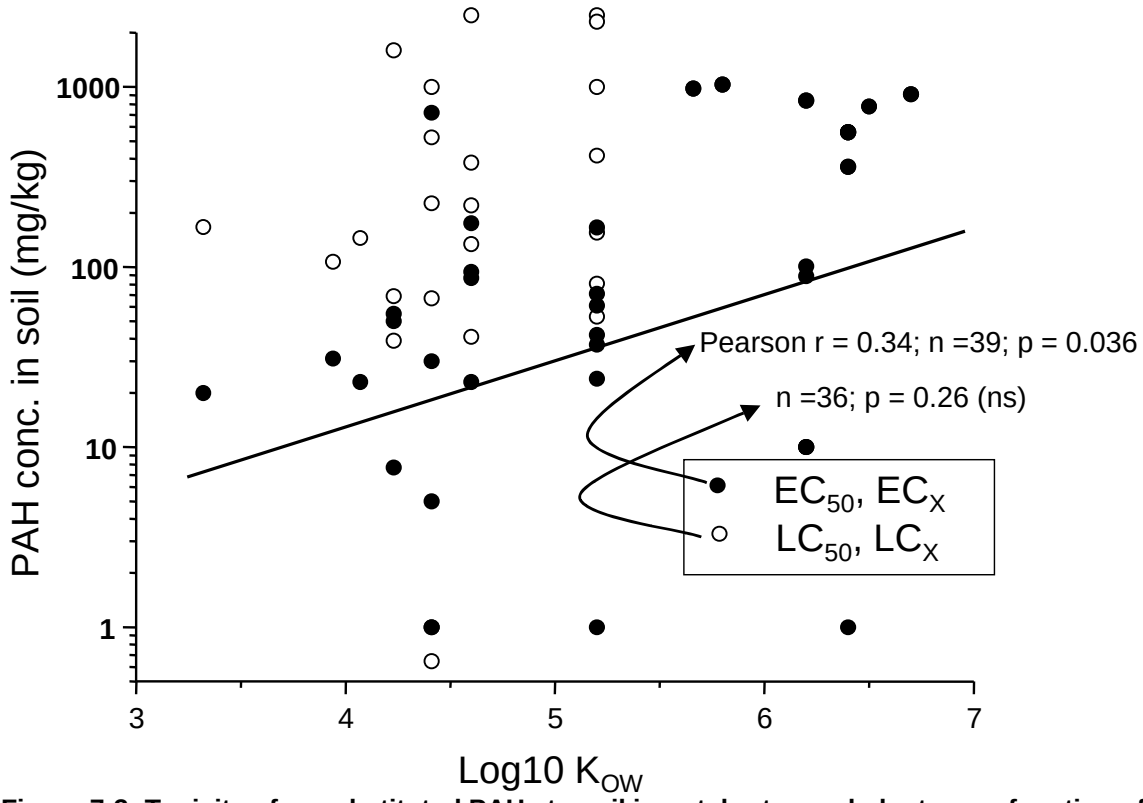


Figure 7-3: Toxicity of unsubstituted PAHs to soil invertebrates and plants as a function of K_{OW}.

All soil invertebrate and plant effects and mortality endpoints were plotted in Figure 7-3 as a function of the log K_{OW} of the individual PAHs. NOEC data were excluded, and where more than one ECx value was available for a specific taxon, only the closest data to a median effects level (EC_{50}) were used. While higher molecular weight PAHs with larger K_{OW} values tended to be slightly less toxic, it is apparent that this interrelationship accounts for only a very small portion of the variation in observed response levels across taxa, across soil types, and between studies. There is little support, therefore, for using a K_{OW}-based QSAR approach for deriving generically applicable risk based standards based on direct soil contact.

It is evident from Figure 7-3 that non-mortality type endpoints were systematically more sensitive than mortality endpoints, as would be expected, and that the range of variability in median effects levels for a specific PAH may vary by up to three orders of magnitude, even for this subset of taxa which is very small relative to the expected number and diversity of species at various field sites in Canada.

Given that a weight-of-evidence approach was not feasible for developing direct soil contact soil quality guidelines for all but fluoranthene and benzo[a]pyrene, the "Lowest Observed Effect Concentration Method" was investigated.

Table 7-5 lists the most sensitive LOEC-type endpoints from Appendices III and IV. There are insufficient data to calculate a soil contact guideline using the CCME (2006) protocols for all unsubstituted PAHs except anthracene, fluoranthene, and benzo[a]pyrene. As noted above, soil contact guidelines have already been derived for fluoranthene and benzo[a]pyrene using the Weight-of-Evidence method. Therefore, the LOEC method was only used to derive a soil contact guideline for anthracene.

Table 7-5: Most sensitive LOEC-type endpoints.

	Organism	Effect	Endpoint	Effective Concentration ($mg \cdot kg^{-1}$)	Exposure Period	Reference
Naphthalene						
	Folsomia fimetaria (Springtail)	Reproduction	EC_{10}	20 (95%CI: 0 - 39)	21 d	Sverdrup *et al*., 2002a
Acenaphthene						
	Folsomia fimetaria (Springtail)	Reproduction	EC_{10}	31 (CI: 0 - 42)	21 d	Sverdrup *et al.*, 2002a
Acenaphthylene						
	Folsomia fimetaria (Springtail)	Reproduction	EC_{10}	23 (CI: 12 - 31)	21 d	Sverdrup *et al.*, 2002a
Fluorene						
	Eisenia veneta (earthworm)	Growth	EC_{10}	31 (CI: 28 - 36)	28 d	Sverdrup *et al.*, 2002e
	Enchytraeus crypticus (potworm)	Reproduction	EC_{10}	25 (CI: 0 - 39)	21 d	Sverdrup *et al.*, 2002d

	Organism	Effect	Endpoint	Effective Concentration ($mg \cdot kg^{-1}$)	Exposure Period	Reference
	Folsomia fimetaria (Springtail)	Reproduction	EC_{10}	7.7 (CI: 5.2 - 10)	21 d	Sverdrup *et al.*, 2001
Anthracene						
	Lolium perenne (Perennial ryegrass)	Growth	LOEC ($EC_{40.8}$)	226	40 d	Leyval and Binet, 1998
	Avena sativa (Common oat)	Growth	EC_{50}	30 (CI: 20 - 45)	32 d	Mitchell *et al.*, 1988
	Folsomia fimetaria (Springtail)	Reproduction	EC_{10}	5 (CI: 3.2 - 12)	21 d	Sverdrup *et al.*, 2002a
Phenanthrene						
	Eisenia veneta (earthworm)	Growth	EC_{10}	25 (CI: 9.0-41)	28 d	Sverdrup *et al.*, 2002e
	Enchytraeus crypticus (potworm)	Reproduction	EC_{10}	40 (CI: 0 - 78)	21 d	Sverdrup *et al.*, 2002d
	Folsomia candida (Springtail)	Mortality	LOEC	380	33-34 d	Crouau *et al.*, 1999
	Folsomia candida (Springtail)	Reproduction	LOEC	220	33-34 d	Crouau *et al.*, 1999
	Folsomia fimetaria (Springtail)	Reproduction	EC_{10}	23 (CI: 9.1 - 38)	21 d	Sverdrup *et al.*, 2001
Fluoranthene						
	Avena sativa (Common oat)	Growth	EC_{50}	>1,000	14 d	Kordel *et al*., 1984
	Brassica rapa (Bird rape)	Growth	EC_{50}	>1,000	14 d	Kordel *et al*. 1984
	Eisenia veneta (earthworm)	Growth	EC_{10}	113 (CI: 101 - 124)	28 d	Sverdrup *et al.*, 2002e
	Enchytraeus crypticus	Reproduction	LOEC	1212	30 d	Achazi *et al.*, 1995
	Enchytraeus crypticus	Reproduction	EC_{10}	15 (CI: 11 - 33)	21 d	Sverdrup *et al.*, 2002d
	Folsomia fimetaria (Springtail)	Reproduction	EC_{10}	37 (CI: 26 - 48)	21 d	Sverdrup *et al.*, 2002a
Pyrene						
	Eisenia veneta (earthworm)	Growth	EC_{10}	38 (CI: 28 - 46)	28 d	Sverdrup *et al.*, 2002e
	Enchytraeus crypticus (potworm)	Reproduction	EC_{10}	11 (CI: 2.3 - 27)	21 d	Sverdrup *et al.*, 2002d
	Folsomia fimetaria (Springtail)	Reproduction	EC_{10}	10 (CI: 7.3 - 13)	21 d	Sverdrup *et al.*, 2001
Benz[a]anthracene						
	Lupinus albus (Lupin)	Growth	LOEC	>155	30 d	Henner *et al.*, 1999

	Organism	Effect	Endpoint	Effective Concentration ($mg \cdot kg^{-1}$)	Exposure Period	Reference
	Folsomia fimetaria (Springtail)	Reproduction	EC_{10}	>980	21 d	Sverdrup *et al.*, 2002a
Chrysene						
	Folsomia fimetaria (Springtail)	Reproduction	EC_{10}	>1030	21 d	Sverdrup *et al.*, 2002a
Benzo[b]fluoranthene						
	Folsomia fimetaria (Springtail)	Reproduction	EC_{10}	>360	21 d	Sverdrup *et al.*, 2002a
Benzo[k]fluoranthene						
	Folsomia fimetaria (Springtail)	Reproduction	EC_{10}	>560	21 d	Sverdrup *et al.*, 2002a
Benzo[a]pyrene						
	Cannabis sativa (Hemp)	Emergence	EC_{50}	89	30 d	Campbell *et al.*, 2002
	Lupinus albus (Lupin)	Growth	LOEC	>155	30 d	Henner *et al.*, 1999.
	Eisenia fetida (earthworm)	Growth	EC_{12}	10	28 d	Achazi *et al.*, 1995
	Eisenia fetida (earthworm)	Reproduction	$EC_{(91)}$	10	28 d	Achazi *et al.*, 1995
	Enchytraeus crypticus (potworm)	Reproduction	$EC_{(18)}$ (LOEC)	40 $\mu mol \cdot kg^{-1}$	30 d	Achazi *et al.*, 1995
	Folsomia fimetaria (Springtail)	Reproduction	EC_{10}	>840	21 d	Sverdrup *et al.*, 2002a
Indeno[1,2,3-c,d]pyrene						
	Folsomia fimetaria (Springtail)	Reproduction	EC_{10}	>910	21 d	Sverdrup *et al.*, 2002a
Dibenzo(a,h)anthracene						
	Lupinus albus (Lupin)	Growth	LOEC	>155	30 d	Henner *et al.*, 1999
	Folsomia fimetaria (Springtail)	Reproduction	EC_{10}	>780	21 d	Sverdrup *et al.*, 2002a

The lowest LOEC for anthracene is from a series of studies by Sverdrup *et al*. (2002a) using the collembolan *Folsomia fimetaria*, among other plant and invertebrate taxa. These studies have been critically reviewed and pass data quality requirements for use in the derivation of CCME soil quality guidelines. Of note is that the toxicity endpoints have been adjusted to account for measured as opposed to nominal (spiked) PAH concentrations in the soil test units.

Sverdrup *et al.* (2002a) provided a collembolan reproduction EC_{10} of 5 $mg \cdot kg^{-1}$ anthracene (95% CI = 3.2 to 12 $mg \cdot kg^{-1}$), based on a 21 d exposure period. Also

emerging from their study was an LC_{50} estimate (same exposure period) of 67 $mg \cdot kg^{-1}$ (95% CI = 44 to 98 $mg \cdot kg^{-1}$). The studies were conducted at 50% soil saturation.

In an earlier study, Sjursen *et al*. (2001) showed that prior exposure to PAHs in soil (at 50% soil saturation) for 21 d resulted in reduced survival relative to controls of *F. fimetaria* subsequently placed without soil in dehydration chambers for the PAHs fluorene, fluoranthene, pyrene and others (anthracene was not investigated). This suggests that the reproduction LOEC for anthracene is higher than if the interactive effects of sub-optimal soil conditions (as might occur seasonally in the field) were taken into account.

The lowest LOEC is divided by an uncertainty factor (UF) to derive a threshold effects concentration (TEC):

TEC = lowest LOEC/UF (10)

On the one hand, we are hampered in our understanding of the larger expected range of species sensitivities by the fact that adequate ecotoxicity data exists for only one soil invertebrate and two plant species. On the other hand, the effects level for the LOEC is 10% as opposed to 25%, and the reproductive EC_{10} is an order of magnitude lower than the LC_{50} for the same (chronic) exposure period.

In light of this, an uncertainty factor of 2 is deemed to be adequate to address inter-taxon variability (especially for soil invertebrates, which are likely to be more sensitive to PAHs in general than plants):

TEC = 5 $mg \cdot kg^{-1}$ / 2 **= 2.5 $mg \cdot kg^{-1}$**

Of the remaining PAHs, there were also insufficient data for any of them to derive soil contact guidelines using the Median Effects Method. Therefore, no soil contact guidelines could be derived for the other PAHs.

In summary, the above described derivations lead to the following SQG_{SC} based on direct soil contact for agricultural and residential/parkland areas:

- Anthracene: 2.5 $mg \cdot kg^{-1}$
- Fluoranthene: 50 $mg \cdot kg^{-1}$
- Benzo[a]pyrene: 20 $mg \cdot kg^{-1}$

7.2.1.2 Commercial and Industrial Land Use

For commercial and industrial land uses, the SQG_{SC} was calculated as the 50^{th} percentile of the plant and invertebrate data, also referred to as the effects concentration low (ECL; CCME, 2006). Using this procedure, the SQG_{SC} value for commercial and industrial

land uses was calculated as 180 $mg \cdot kg^{-1}$ for fluoranthene and 72 $mg \cdot kg^{-1}$ for benzo[a]pyrene (see Figures 7-1 and 7-2).

For anthracene, an ECL was calculated based on the LOEC Method in the CCME (2006) protocol as follows:

$$ECL = (LOEC_1 \times LOEC_2 \times \ldots \times LOEC_n)^{1/n} \quad (11)$$

Where ECL = effects concentration low (mg/kg)
LOEC = lowest observed effects concentration ($mg \cdot kg^{-1}$)
n = number of available LOECs

Thus, for anthracene (Table 7-5) –

$$ECL = (5 \text{ mg} \cdot kg^{-1} \times 30 \text{ mg} \cdot kg^{-1} \times 226 \text{ mg} \cdot kg^{-1})^{1/3} = \mathbf{32\ mg \cdot kg^{-1}}$$

7.2.1.3 Nutrient and Energy Cycling Check

The Nutrient and Energy Cycling check (NEC) applies to the SQG_E for all land uses. Studies on nitrogen fixation and nitrification are preferred, but may be supplemented with studies on nitrogen mineralization and carbon cycling (i.e., decomposition and respiration). The minimum data requirements for the SQG_{SC} derivation methods, as outlined in CCME (2006), also apply to the NEC check process (Weight of Evidence, LOEC, or Median Effects methods). Therefore, the toxicity data for microbial processes from Sverdrup *et al.* (2002c) (Section 5.1) are insufficient to determine the effects of PAHs on nutrient and energy cycling. Further research is necessary to obtain the minimum data necessary to calculate a concentration of PAHs in soil that would be protective of important microbial processes.

The relevant consulted studies are summarized in Appendix II. NOEC and minimum effects concentrations (EC_5, EC_{10}) for individual PAHs were generally in the range of 13 to 3,000 $mg \cdot kg^{-1}$. Effects on soil microorganisms and protozoa, therefore, are likely to occur only at PAH concentrations that are higher than thresholds in soil for the protection of soil invertebrates and plants (Section 7.2.1, above).

7.2.2 Food and Soil Ingestion SQG_I for the Protection of Livestock and Wildlife

To ensure adequate soil quality for the maintenance of grazing and feedlot livestock in agricultural settings, a soil quality guideline protocol is provided in CCME (2006) that accounts for the incidental ingestion of potentially contaminated soil as well as ingestion of contaminated food. CCME (2006) explains the rationale for a focus primarily on herbivorous species. For herbivores, soil and plant ingestion are likely to be the major routes of contaminant exposure.

Recent advances have been made in assessing risks to secondary and tertiary consumers (e.g., first, second and higher order predators) based on food-web mediated transfer of contaminants; however, this is of much lower concern for PAHs than risks to herbivores. Higher molecular weight PAHs exhibit sufficient lipophilicity (e.g., as indicated by K_{OW}) in order to exhibit biomagnification, but have never actually been observed to undergo biomagnification, since the facile metabolic modification of PAHs and subsequent excretion of considerably less lipophilic metabolites tends to limit concentrations in prey items.

For herbivorous livestock and wildlife, the task of quantifying PAH exposure is simplified by the very limited tendency of plants to bioaccumulate PAHs based on uptake from soil and translocation to above-ground plant biomass, as discussed in Section 5.2.1. Direct soil ingestion, therefore, is likely to account for the major portion of PAH uptake in vertebrate herbivores.

Soil quality guidelines for the protection of livestock and grazing wildlife are derived based on a reasonable understanding of threshold concentrations, often based on extrapolation from laboratory toxicity studies.

The available data for toxicity of individual PAHs to vertebrate test species (Appendices V, VI, VII) were collated and an assessment was made of the most sensitive available endpoint, which was then used to derive a "Daily Threshold Effects Dose" (DTED)".

$$\text{DTED} = \text{lowest LOAEL/UF} \qquad (12)$$

Where –

DTED	=	Daily Threshold Effects Dose
LOAEL	=	Lowest Observed Adverse Effects Level
UF	=	Uncertainty Factor (range from 1 to 5)

A summary of the DTEDs calculated for each PAH, and the sensitive endpoints upon which they were based, is provided in Table 5-3.

For calculating a soil quality guideline, it is assumed that 75% of the exposure to the contaminant of concern is attributed to the exposure pathways addressed: i.e., – soil ingestion and ingestion of plants growing on contaminated soils.

Overall, the CCME (2006) soil quality guideline for the protection of herbivorous wildlife and livestock (i.e., primary consumers) can be calculated as -

$$SQG_{1C} = \frac{0.75 \times DTED_{IC} \times BW_{1C}}{(SIR_{1C} \times BF) + (FIR_{1C} \times BCF_1)} \qquad (13)$$

Where –

SQG_{1C} = Soil Quality Guideline for Soil and Food Ingestion

		for Protection of Primary Consumers (i.e., herbivorous wildlife and livestock) (mg·kg^{-1} dw soil)
$DTED_{1C}$	=	Daily Threshold Effects Dose of the primary consumer (mg·kg^{-1} bw_{1C}·d^{-1})
BW_{1C}	=	Body Weight (kg)
SIR_{1C}	=	Soil Ingestion Rate (kg dw soil·d^{-1})
BF	=	Bioavailability factor (unitless): assume to be 1.0 in absence of better knowledge.
FIR_{1C}	=	Food Ingestion Rate of primary consumer (kg dw food·d^{-1})
BCF_1	=	Bioconcentration Factor (unitless: chemical and plant-specific)

Because PAHs exhibit very limited tendency to be accumulated in plant tissues, it is assumed that the exposure via plant ingestion is essentially negligible; i.e., the Bioconcentration Factor is equal to or very closely approaches zero. Therefore, equation 13 simplifies to –

$$SQG_{1C} = \frac{0.75 \times DTED_{1C} \times BW_{1C}}{(SIR_{1C} \times BF)} \qquad (14)$$

Four different receptors were considered in the derivation of soil quality guidelines for soil and food ingestion (SQG_I):

- Dairy cow;
- Mule deer (*Odocoileus hemionus*);
- Meadow vole *(Microtus* spp.) (small herbivore); and
- American robin (*Turdus migratorius*) (foraging bird)

In the case of the American robin, uptake via the ingestion of soil invertebrates (especially earthworms) was considered, and therefore equation 15 for secondary consumers was used.

$$SQG_{2C} = \frac{0.75 \times DTED_{2C} \times BW_{2C}}{[(SIR_{2C} \times BF) + (FIR_{2C} \times BAF_2)]} \qquad (15)$$

Where –

SQG_{2C}	=	Soil Quality Guideline for Soil and Food Ingestion for the Protection of Secondary Consumers (mg·kg^{-1} dw soil)
$DTED_{2C}$	=	Daily Threshold Effects Dose of the secondary consumer (mg·kg^{-1} bw_{2C}·d^{-1})
BW_{2C}	=	Body Weight of the secondary consumer (kg)
SIR_{2C}	=	Soil Ingestion Rate for the secondary consumer (kg dw soil·d^{-1})

BF	=	Bioavailability factor (unitless): assume to be 1.0 in absence of better knowledge.
FIR_{2C}	=	Food Ingestion Rate of secondary consumer (kg dw food·d^{-1})
BAF_2	=	Bioaccumulation Factor from soil to prey (unitless: chemical and prey-specific)

Earthworm whole body concentrations of COPCs were estimated according to the method of Menzie *et al.* (1992) as described in Sample *et al.* (1997):

$$\text{Bioaccumulation Factor } (BAF_2) = \frac{\text{earthworm lipid content } (f = 0.0084)}{0.66 \times \text{organic C content of soil } (F_{oc})} \quad (16)$$

Point estimates for life-history traits of various surrogate wildlife species have been compiled by Sample *et al.* (1996, 1997) and USEPA (2000b) as an aid to conduct screening level ecological risk assessments. These estimates, augmented from additional sources, are provided herein in Table 7-6.

Table 7-6: Reference Values for Selected Ecological Receptors

Species of Interest	Est. Body Wt. (kg)	Food Intake Rate (FIR) (kg dw·kg^{-1} bw·day^{-1})	Soil Intake Rate (SIR) (kg·day^{-1})	Estimated Home Range (ha)	Notes
Cows	410	0.02	0.750	N/A	Alberta Environment, 2001a
Mule deer (*Odocoileus hemionus*)	68 (59 –74)	0.022	0.044		Alberta Environment, 2001a
Meadow Vole (*Microtus pennsylvanicus)*	0.039	0.58	0.00012	0.04 – 0.08	After Sample *et al.*, 1996
American robin (*Turdus migratorius*)	0.077	0.093 kg/day	0.0019	0.12 –0.42	After Sample and Suter, 1994

These exposure factor estimates were combined with the estimated Daily Threshold Effect Doses (DTEDs) developed in Section 5.5 to calculate a series of SQG_I estimates (Table 7-7).

Table 7-7: Estimated SQG_I for individual PAHs using DTED values and Equations 13 and 14.

PAH	DTED ($mg \cdot kg^{-1} \cdot day^{-1}$)	Cow ($mg \cdot kg^{-1}$)	Mule Deer ($mg \cdot kg^{-1}$)	Meadow Vole ($mg \cdot kg^{-1}$)	American Robin ($mg \cdot kg^{-1}$)
Naphthalene	28.6	11,726	33,150	6,971	**8.8**
Acenaphthene	70	28,700	81,136	17,062	**21.5**
Fluorene	50	20,500	57,955	12,187	**15.4**
Anthracene	200	82,000	231,818	48,750	**61.5**
Phenanthrene	140	57,400	162,273	34,125	**43.0**
Fluoranthene	50	20,500	57,955	12,187	**15.4**
Pyrene	25	10,250	28,977	6,094	**7.7**
Benz[a]anthracene	20	8,200	23,182	4,875	**6.2**
Chrysene	20	8,200	23,182	4,875	**6.2**
Benzo[b+j]fluoranthene	20	8,200	23,182	4,875	**6.2**
Benzo[k]fluoranthene	20	8,200	23,182	4,875	**6.2**
Benzo[a]pyrene	2	820	2,318	487	**0.6**
Dibenz[a,h]anthracene	N/A	--	--	--	**--**

(Bolded SQG_I: lowest calculated values for different vertebrate receptors)
NOTE: According to the protocol (CCME, 2006), naphthalene was the only PAH that met the minimum data requirements for calculation of the SQG_I. Therefore, values presented for all other PAHs are considered "provisional", and have not been used in determining the overall SQG_E.

Preliminary soil quality guidelines for PAHs calculated for the protection of the American robin are much lower than for livestock (dairy cow receptor) or for small or large herbivorous mammalian wildlife. This is due to the additional dietary exposure based on PAH uptake into earthworms (Equation 15) followed by ingestion. In these calculations, food (earthworm) intake account for 56% of the internalized dose, assuming a lipid content in the earthworm of 0.84% and a soil organic carbon content of 0.5% (f_{OC} = 0.005), resulting in a worm uptake factor (or BCF) of approximately 2. This modeled BCF is supported by the following BCFs that were measured for earthworms exposed to unaged PAHs in soil: 1.84 for anthracene, 2.34 for fluoranthene, and 2.17 for pyrene (GRI/PERF 2000). Insectivorous and worm-eating small birds and mammals are often estimated to be the worst-case for contaminated soil-associated risks as a result of detailed site-specific risk assessments. This greater apparent potential for exposure is often borne out in tissue residue studies. For example, insectivorous shrews typically exhibit higher concentrations of organic contaminants than co-occurring herbivorous wild mice, voles and other small herbivorous mammals and birds.

The SQG_I values for the American robin were consistently lower than for the other vertebrate receptors, and these are selected as the SQG_I for agricultural and residential/parkland land uses.

7.2.3 Soil Quality Guidelines for the Protection of Livestock Watering

Values for livestock ingesting water (SQG_{LW}) were not calculated. The PAHs that are potentially carcinogenic have sufficiently high K_{oc}s that this route of exposure is unlikely.

7.2.4 Soil Quality Guidelines for the Protection of Aquatic Life

There are pre-existing CCME water quality guidelines for PAHs based on the protection of freshwater life (CCME, 1999a), as follows:

- Naphthalene 1.1 $\mu g \cdot L^{-1}$
- Acenaphthene: 5.8 $\mu g \cdot L^{-1}$
- Fluorene 3.0 $\mu g \cdot L^{-1}$
- Anthracene 0.012 $\mu g \cdot L^{-1}$
- Phenanthrene 0.4 $\mu g \cdot L^{-1}$
- Fluoranthene 0.04 $\mu g \cdot L^{-1}$
- Pyrene 0.025 $\mu g \cdot L^{-1}$
- Benz[a]anthracene 0.018 $\mu g \cdot L^{-1}$
- Benzo[a]pyrene 0.015 $\mu g \cdot L^{-1}$
- *(Quinoline) 3.4 $\mu g \cdot L^{-1}$
- *(Acridine) 4.4 $\mu g \cdot L^{-1}$

(*ignored for the purpose of this derivation exercise.)

CCME (2006) provides a method for calculating acceptable soil concentrations based on modeled concentrations within the aquatic receiving environment through the adoption of a quasi two-dimensional groundwater model based on work by Domenico (1987). The methodology used for protection of aquatic life is similar to that used for the protection of potable groundwater pathway, except that a minimum 10 m lateral offset between source and exposure point is assumed.

The protection of groundwater for freshwater life applies to all land uses. A guideline for each chemical of concern was derived from the water quality guideline for freshwater life for that chemical. Where available, CCME (1999a) water quality guidelines are used.

The protection of aquatic life considers four processes:

1. partitioning from soil to leachate;
2. transport of leachate from base of contamination to water table;
3. mixing of leachate and groundwater; and
4. groundwater transport down-gradient to a discharge point.

Equations used to calculate the soil quality guidelines for the protection of aquatic life are as follows:

$$SQG_{FL} = C_L \left\{ K_d + \left(\frac{\theta_w + H'\theta_a}{\rho_b} \right) \right\} \qquad (16)$$

where:

SQG_{FL} = soil quality guideline for the protection of freshwater life ($mg \cdot kg^{-1}$)
C_L = allowable leachate concentration at source ($mg \cdot L^{-1}$); for default guidelines this is assumed to be equal to C_z (see calculation below)
K_d = distribution coefficient ($cm^3 \cdot g^{-1}$); (see calculation below)
θ_w = water filled porosity (unitless); 0.119 for coarse soils (CCME 2006)
H' = dimensionless Henry's Law constant = H x 42.32
H = Henry's Law constant (see Table 7-9)
θ_a = air-filled porosity (unitless); 0.241 for coarse soils (CCME 2006)
ρ_b = soil bulk density in contaminant partitioning zone ($g \cdot cm^{-3}$); 1.7 for coarse soils (CCME 2006)

$$K_d = K_{oc} f_{oc} \qquad (17)$$

where:

K_{oc} = organic carbon partitioning coefficient ($L \cdot kg^{-1}$) (see Table 7-9)
f_{oc} = organic carbon fraction of soil ($g \cdot g^{-1}$); 0.005 (CCME 2006)

$$C_z = C_{gw} \left\{ 1 + \left(\frac{Z_d K_H i}{IX} \right) \right\} \qquad (18)$$

where:

C_z = allowable chemical concentration in leachate at the water table ($mg \cdot L^{-1}$)
C_{gw} = allowable chemical concentration in groundwater at the source ($mg \cdot L^{-1}$) (see calculation below)
Z_d = average thickness of mixing zone (m) (see calculation below)
K_H = hydraulic conductivity in the saturated zone ($m \cdot y^{-1}$); 320 for coarse soils (CCME 2006)
i = hydraulic gradient (unitless); 0.028 (CCME 2006)
I = infiltration rate ($m \cdot y^{-1}$) = precipitation minus runoff and evapotranspiration; 0.28 for coarse soils (CCME 2006)
X = length of source parallel to groundwater flow (m); 10 (CCME 2006)

$$Z_d = r + s \quad \text{(Note: } Z_d \text{ cannot exceed } d_a\text{)} \qquad (19)$$

where:

r = mixing depth available due to dispersion and diffusion (m)
= 0.01 X

X = length of source parallel to groundwater flow (m); 10 (CCME 2006)
s = mixing depth available due to infiltration rate and groundwater flow rate (m) (see calculation below)

$$s = d_a\left\{1 - e^{-\frac{2.178XI}{K_H i d_a}}\right\} \qquad (20)$$

where:
d_a = depth of unconfined aquifer (m); 5 (CCME 2006)
I = infiltration rate ($m \cdot y^{-1}$) = precipitation minus runoff and evapotranspiration; 0.28 for coarse soils (CCME 2006)
K_H = hydraulic conductivity in the saturated zone ($m \cdot y^{-1}$); 320 for coarse soils (CCME 2006)
i = hydraulic gradient (unitless); 0.028 (CCME 2006)

$$C_w(x,y,z,t) = \left(\frac{C_{gw}}{4}\right)\exp\left\{\left(\frac{x}{2\partial_x}\right)\left[1-\left(1+\frac{4L_s\partial_x}{v}\right)^{1/2}\right]\right\} erfc\left[\frac{x - vt\left(1+\frac{4L_s\partial_x}{v}\right)^{1/2}}{2(\partial_x vt)^{1/2}}\right]$$

$$\left\{erf\left[\frac{(y+Y/2)}{2(\partial_y x)^{1/2}}\right] - erf\left[\frac{y-Y/2}{2(\partial_y x)^{1/2}}\right]\right\} \qquad (21)$$

$$v = \frac{K_H i}{n_e R_f}; \quad R_f = 1 + \frac{\rho_b}{n}K_d \qquad (22; 23)$$

where:
C_w = allowable chemical concentration in water at receptor ($mg \cdot L^{-1}$) (i.e., Canadian water quality guideline for the protection of freshwater aquatic life)
x = distance from source to receptor (m); 10 (CCME 2006)
x,y,z = Cartesian coordinates relating source and receptor (m); y, z assumed to be 0
t = time since contaminant release (years); conservative assumption of 100 used
C_{gw} = allowable chemical concentration in groundwater at source ($mg \cdot L^{-1}$)
∂_x = longitudinal dispersivity tensor = 0.1x = 1
∂_y = lateral dispersivity tensor = $0.1\partial_x$ = 0.1

L_s = decay constant (y^{-1}) in saturated zone (see calculation below)

$$L_s = \frac{0.693}{t_{1/2S}}\left(e^{-0.07d}\right) \qquad (24)$$

where:

d = depth from surface to groundwater surface (m); 3 (CCME 2006)

$t_{1/2S}$ = biodegradation half-life in saturated zone (y); conservative assumption of 999 used

v = velocity of contaminant ($m \cdot y^{-1}$) (see equation above)

K_H = hydraulic conductivity in the saturated zone ($m \cdot y^{-1}$); 320 for coarse soils (CCME 2006)

i = hydraulic gradient (unitless); 0.028 (CCME 2006)

n = total porosity of soil = 1 - ρ_b/2.65 (unitless); 0.36 for coarse soils (CCME 2006)

n_e = effective soil porosity (unitless); generally assumed to be the same as total soil porosity (n)

Y = source width (m) perpendicular to groundwater flow; 10 (CCME 2006)

R_f = retardation factor (unitless) (see equation above)

ρ_b = soil bulk density in saturated zone ($g \cdot cm^{-3}$); 1.7 for coarse soils (CCME 2006)

K_d = distribution coefficient ($cm^3 \cdot g^{-1}$) (see calculation above)

Assumptions implicit in the model include the following:

- the soil is physically and chemically homogeneous;
- moisture content is uniform throughout the unsaturated zone;
- infiltration rate is uniform throughout the unsaturated zone;
- decay of the contaminant source is not considered (*i.e.*, infinite source mass);
- flow in the unsaturated zone is assumed to be one dimensional and downward only (vertical recharge) with dispersion, sorption-desorption, and biological degradation;
- contaminant is not present as a free phase product;
- maximum possible concentration in the leachate is equivalent to the solubility limit of the chemical in water under the defined site conditions;
- groundwater aquifer is unconfined;
- groundwater flow is uniform and steady;
- co-solubility and oxidation/reduction effects are not considered;
- attenuation of the contaminant in the saturated zone is assumed to be one dimensional with respect to sorption-desorption, dispersion, and biological degradation;
- dispersion is assumed to occur in the longitudinal and transverse directions only and diffusion is not considered;

- mixing of the leachate with the groundwater is assumed to occur through mixing of leachate and groundwater mass fluxes; and
- dilution of the plume by groundwater recharge down-gradient of the source is not included.

Table 7-8 provides default assumptions for important generic site properties that are used in the model to derive generic SQG_{FL} for each of the PAHs.

Table 7-8: Generic site and subsurface soil properties.

Descriptor	Symbol	Units	Coarse Soils
Definition – median grain size is		μm	>75
Soil temp.	T	^{o}K	294
Total soil porosity	n	vol/vol	0.36
Soil water content	θ_M	Mass/mass	0.07
Moisture filled porosity	θ_W	vol/vol	0.119
Vapour-filled porosity	θ_A	vol/vol	0.241
Soil bulk density (dry wt basis)	ρ_b	g/cm^3	1.7
Soil permeability to vapour flow	K_V	cm^2	10^{-8}
Saturated hydraulic conductivity	K_H	m/y	320
Recharge (infiltration) rate	I	m/y	0.28
Fraction of organic carbon	f_{OC}	mass/mass	0.005
Hydraulic gradient	i		0.028
Contaminant source width	X	m	10
Contaminant source length	Y	m	10
Distance to receptor (parallel to groundwater flow)	x	m	10
Depth to groundwater	d	m	3
Distance between base of contamination and groundwater	b	m	0
Depth of unconfined aquifer	d_a	m	5
Days with surface temp < 0 ^{o}C.	$D_{1/2US}$	days	365
Time since contaminant release	t	years	100

Generic parameter values for both fine-textured and coarse-textured soils were included in CCME (2006), but the fine-textured case is not considered herein. The SQG_{FL} for individual PAHs is based on the generic coarse-textured soil assumptions.

In addition, some protocols allow for the inclusion of contaminant biodegradation half-life estimates, and degradation in the unsaturated zone and saturated zone can be considered independently. Given the expected high degree of variability in PAH biodegradation rates across soil types and biogeoclimatic zones in Canada, however, it was assumed for the generic case that biodegradation is negligible; i.e., $t_{1/2}$ = 999 years in both the unsaturated and saturated zones.

Some jurisdictions within Canada and elsewhere have adopted policy decisions that allow for a minimum amount of dilution of groundwater that enters a surface water body, and then potentially interacts with aquatic life. Given differences in policies between jurisdictions in Canada, however, no allowance for dilution of groundwater near the groundwater outflow face in the receiving environment has been made herein. The resulting soil quality guidelines, therefore, are lower than is supported in jurisdictions that have adopted a groundwater-surface water 10-fold or other dilution allowance. Some jurisdictions have explicitly rejected such a policy, since benthic invertebrates and other sediment, lakebed, or streambed associated organisms might experience perfusion of undiluted groundwater at the outflow face. If dilution at the surface water body is expected to be significant, site specific assessment should be considered.

Based on the CCME (2006) protocol, generic site assumptions as provided above, pre-existing Canadian Water Quality Guidelines for the protection of freshwater aquatic life ($CWQG_{FAL}$), and estimates of organic carbon-water partitioning for each PAH, SQG_{FL}s were calculated as follows:

Table 7-9: Soil Quality Guidelines for the Protection of Freshwater Life, derived from $CWQG_{FAL}$

	log Koc[a]	**Henry's Law constant ($atm \cdot m^3 \cdot mol^{-1}$)**[b]	**$CWQG_{FAL}$ ($\mu g \cdot L^{-1}$)**	**SQG_{FL} ($mg \cdot kg^{-1}$)**
Naphthalene	2.85	4.83×10^{-4}	1.1	0.013
Acenaphthene	3.45	1.55×10^{-4}	5.8	0.28
Fluorene	3.69	7.96×10^{-5}	3.0	0.25
Anthracene	4.3	3.54×10^{-5}	0.012	no value[c]
Phenanthrene	3.82	2.33×10^{-5}	0.4	0.046
Fluoranthene	4.62	1.44×10^{-5}	0.04	no value[c]
Pyrene	4.84	1.1×10^{-5}	0.025	no value[c]
Benz[a]anthracene	5.3	3.35×10^{-6}	0.018	no value[c]
Benzo[a]pyrene	6.34	1.13×10^{-6}	0.015	8800

a: FromTable 2-4; where a range was given, the geometric mean has been calculated.
b: FromTable 2-4; where a range was given, the geometric mean has been calculated.
c: No SQG_{FL} could be calculated based on the assumed generic site/soil properties and the K_{OC} of the PAH, since the concentration in groundwater at the point of leaching would need to far exceed the solubility limit to account for a concentration that approaches the $CWQG_{FAL}$ at a point 10 m down-gradient.

Where a range of log K_{OC} values was found in the literature, a geometric mean value was used. As noted earlier in section 7.1.5, K_{OC} values for PAHs can vary by up to three orders of magnitude depending on site-specific conditions. Therefore, users may wish to calculate Tier 2 SQG_{FL} values by substituting site-specific K_{OC} values into the above equations.

It is noted in Section 5.6 that $CWQG_{FAL}$ do not exist for all of the PAHs of interest. In the absence of existing values, it is possible to calculate water quality guidelines for the protection of aquatic life using a Critical Body Residues approach (Di Toro *et al.,* 2000). This is the same procedure used to calculate *de facto* water quality benchmarks for the sub-fractions of the Canada-Wide Standards for Petroleum Hydrocarbons (CWS PHC F1 and F2; CCME, 2008). Assuming a narcosis/baseline mode of toxicological action, it was assumed, based on Di Toro *et al.* (2000) that an internalized dose for aquatic life of 3.0 mmol PAH·kg^{-1} lipid was a threshold for chronic, non-lethal toxicity, and that the uptake of PAHs is influenced primarily by $K_{OW.}$ The resulting Critical Body Residue (CBR) water-based thresholds (WBT_{CBR}) were calculated, as follows:

$$WBT_{CBR} = (BRL \times MW \times CF)/K_{OW} \qquad (25)$$

Where:

WBT_{CBR}	=	Water-Based Thresholds based on a critical body residues approach and assumed water-organism equilibrium partitioning of lipophilic contaminants (μg·L^{-1})
MW	=	Molecular weight (mg·$mmol^{-1}$) (substance specific)
CF	=	Unit conversion factor: 1,000 μg = 1 mg
BRL	=	Body Residue (Lipid-basis): It is assumed that a BRL of 3.0 mmol·kg-$lipid^{-1}$ is a threshold for chronic, narcosis-type effects in aquatic organisms (CCME, 2008; Di Toro *et al.,* 2000)
K_{OW}	=	Octanol-water partition co-efficient (substance specific)

Using this approach, calculated WBT_{CBR} and corresponding draft Soil-Based Thresholds for freshwater life protection (SBT_{FL}) values are presented in Table 7-10.

The CBR-based water quality guidelines are uniformly higher than existing Canadian Water Quality Guidelines for the protection of freshwater aquatic life ($CWQG_{FAL}$), where they exist. This is not too surprising, since the CBR-based values only account for baseline, narcosis-type effects, while the actual aquatic toxicity data are more likely a reflection of additional modes of toxicity, including photoinduced toxicity and molecular receptor specific effects. Even when using a non-polar narcosis-type approach, it is noted that virtually all of the higher molecular weight PAHs are sufficiently non-polar to preclude entry into the environment at toxicologically relevant concentrations without exceeding solubility limits at the soil leaching zone, based on model predictions for transport in the dissolved phase.

Where soil quality guidelines for freshwater life (SQG_{FL}) have been calculated based on existing $CWQG_{FAL}$, these will be used preferentially over soil-based thresholds for freshwater life (SBT_{FL}). Bolded values in Table 7-10 indicate the individual PAHs for which there are no existing water quality guidelines in Canada. Therefore, only one

additional soil-based threshold was calculated using the critical body threshold approach; this was a value of 320 $mg \cdot kg^{-1}$ for acenaphthylene.

Table 7-10: Soil Quality Guidelines for the Protection of Aquatic Life, derived from WQG_{CBR}

PAH	Non-polar Narcosis (CBR)type Water-Based Threshold (WBT_{CBR}) ($\mu g \cdot L^{-1}$)	Corresponding Soil-Based Threshold for Freshwater Life Protection (SBT_{FL}) ($mg \cdot kg^{-1}$)	Soil Quality Guideline for Freshwater Life Protection Calculated from Existing $CWQG_{FAL}$ (from Table 7-9) (SQG_{FL}) ($mg \cdot kg^{-1}$)
naphthalene	168*	2.5	0.013
acenaphthylene	**46**	**320**	
acenaphthene	56*	12	0.28
fluorene	33*	19	0.25
anthracene	17*	no value[1]	no value[1]
phenanthrene	17*	6.5	0.046
pyrene	6.8*	no value[1]	no value[1]
fluoranthene	5.8*	no value[1]	no value[1]
benz[a]anthracene	1.4*	no value[1]	no value[1]
chrysene	**1.4**	**no value[1]**	
benzo[b]fluoranthene	**0.48**	**no value[1]**	
benzo[k]fluoranthene	**0.48**	**no value[1]**	
perylene	**0.30**	**no value[1]**	
benzo[a]pyrene	0.59*	no value[1]	8800
indeno[1,2,3-c,d]pyrene	**0.21**	**no value[1]**	
dibenz[a,h]anthracene	**0.26**	**no value[1]**	
benzo[g,h,i]perylene	**0.17**	**no value[1]**	

* PAHs for which $CWQG_{FAL}$ are available.

1: A SQG_{FL} or WBT_{FL} could not be calculated based on the assumed generic site/soil properties and the K_{OC} of the PAH, since the concentration in groundwater at the point of leaching would need to far exceed the solubility limit to account for a concentration that approaches the toxicity threshold at a point 10 m down-gradient.

7.3 Offsite Migration Check

The offsite migration check value (SQG_{OM-E}, SQG_{OM-HH}) was not calculated, since the calculated soil quality guideline values for commercial and industrial land uses were in no cases ≥15-fold greater than for the more sensitive land uses that might comprise the surrounding lands. The risks of off-site soil transfer, therefore, were deemed to be negligible.

7.4 Data Gaps and Areas for Future Research

There were numerous data gaps encountered in the development of these guidelines. Some of the major gaps are outlined below, along with suggestions for future research that would help to improve our knowledge of the environmental fate and toxic effects of PAHs in soil.

For the effects on ecological receptors, both benzo[a]pyrene and fluoranthene have been well studied in both plants and invertebrates, and therefore a weight-of-evidence approach could be used to derive direct soil contact guidelines for these substances. A large amount of data was available on the toxicity of anthracene to plants, but little on its toxicity to invertebrates. Conversely, fluorene and pyrene were reasonably well-studied for their effects on invertebrates, but not on plants. However, for the majority of the PAHs, there was a general paucity of toxicity data for either type of taxonomic group. Even in the datasets for the two well-studied PAHs, most of the plant studies focused on agricultural crop species. Future toxicity studies on organisms that are found in boreal forest and arctic environments would help to improve our understanding of potential effects of PAHs in different Canadian ecosystems.

For all PAHs except naphthalene, there were insufficient data to derive soil and food ingestion guidelines. Further toxicity studies on avian and mammalian species (other than laboratory rodents) are needed.

Currently, the available PAH toxicity data for all potential receptors primarily focus on just a select list of unsubstituted PAHs. There are a much broader range of PAHs that may be contributing to the toxicity of PAH mixtures, but which have not assessed.

Many studies on the environmental fate and behaviour of PAHs, as well as their toxic effects, have focused on single PAHs, rather than looking at more complex mixtures containing multiple PAHs, as well as other hydrocarbons. When entrained within a hydrocarbon mixture, it is possible that PAHs could behave differently than when they are isolated. Another aspect that has not been well-studied is the effect of weathering of PAHs, or a hydrocarbon mixture, on bioavailability and toxicity; most toxicity tests are conducted with “fresh” product that has not been exposed to conditions that would be experienced in the field.

8. RECOMMENDED CANADIAN SOIL QUALITY GUIDELINES

Table 8-1 provides a collation of all soil quality guideline and check values developed in Chapter 7. The CCME (2006) protocol further specifies the nomination of the most sensitive risk-based guideline for the protection of each of human and environmental health, as the final CCME soil quality guidelines.

According to the formal CCME protocol, both environmental (SQG_E) and human health (SQG_{HH}) soil quality guidelines are developed for four land uses: agricultural, residential/parkland, commercial and industrial. The lowest value generated by the two approaches for each of the four land uses is recommended by CCME as the Canadian Soil Quality Guideline.

It is not possible to nominate a single SQG for each of human health and environmental health for the PAHs examined herein, since several compounds were examined, and the approach for dealing with PAH mixtures needed to be adapted for different exposure/receptor groups. Guideline values for some exposure pathways are based on the concentrations of the individual PAHs (e.g., mg fluoranthene·kg^{-1} soil) while for other pathways they are based on potency relative to benzo[a]pyrene (i.e., mg B[a]P TPE·kg^{-1} soil) and are intended to be considered together with any other PAHs present at the site. Consequently, it is recommended that the guidelines for all applicable pathways at a site be considered individually in determining whether there are exceedances. Figure 8-1 provides a conceptual framework for how to apply the PAH soil quality guidelines at a contaminated site. Figure 8-2 provides a case-study with hypothetical field data to provide a working example of the conceptual framework presented in Figure 8-1.

In practice, those assessing and remediating contaminated sites within Canada have tended to avail themselves of the broader range of developed preliminary SQG_{HH} and SQG_E when interpreting risks at a site, and developing risk management or risk reduction strategies. The larger set of preliminary SQG provide pathway-specific estimates of protective thresholds, which can be used along with an implicit or explicit qualitative environmental risk assessment approach.

It should be noted that, although petroleum hydrocarbons (PHCs) contain PAHs, the Canada-wide Standard for Petroleum Hydrocarbons was not developed to address the issue of potential carcinogenicity presented by the presence of PAHs in PHC mixtures. Remediation to meet the PHC standards will not necessarily mean that the PAH soil quality guidelines will be met, and vice versa.

Figure 8-1. How to apply Canadian Soil Quality Guidelines for PAHs at a contaminated site.

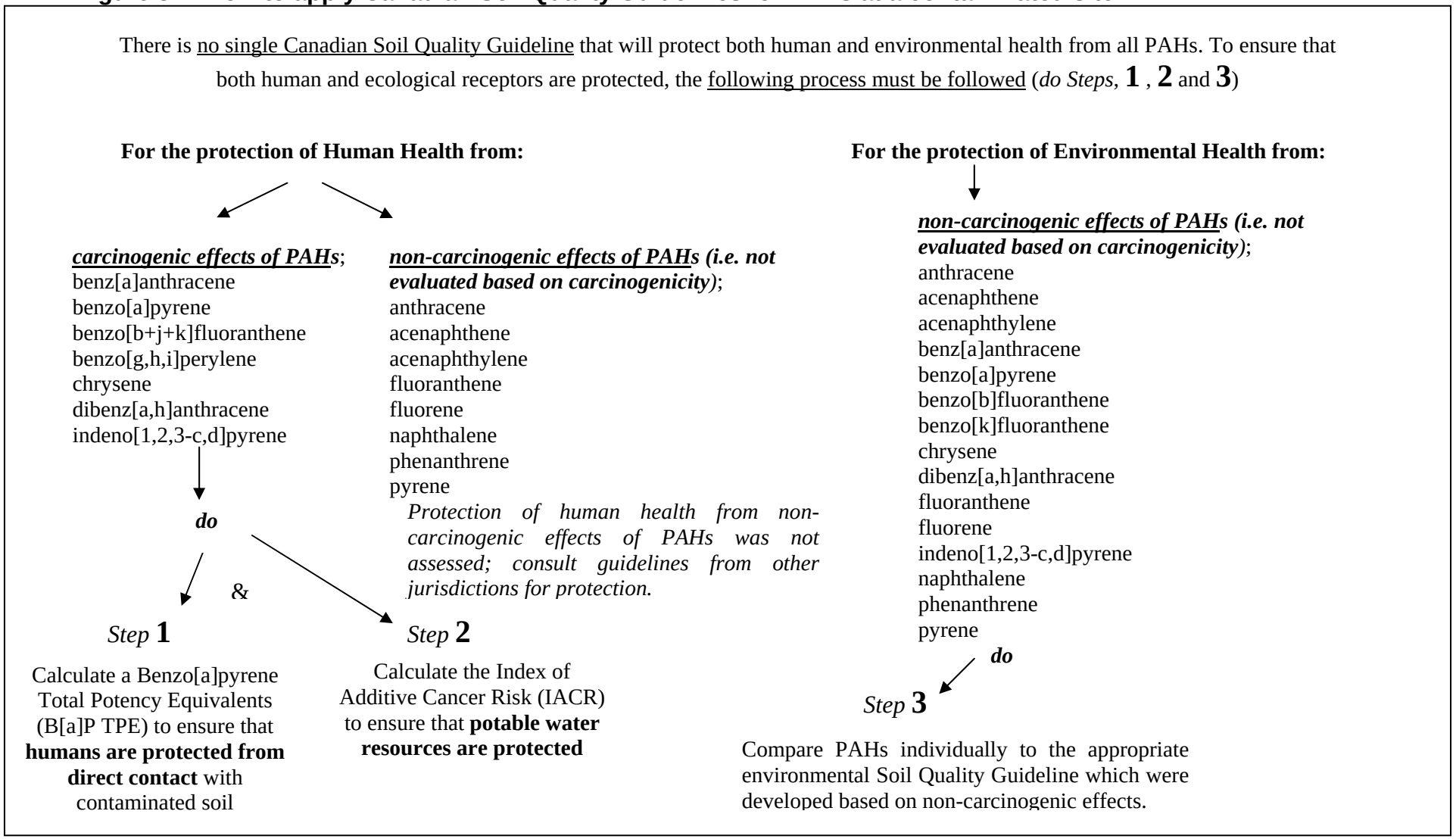


Table 8-1. Soil Quality Guidelines for Carcinogenic and Other PAHs (mg·kg^{-1})

IMPORTANT NOTE (1): There is no single final Soil Quality Guideline (SQG_F) for any of the PAHs included in this guideline that will protect both human and environmental health. To ensure that both human and ecological receptors are protected, the user must (1) calculate a Benzo[a]pyrene Total Potency Equivalents (B[a]P TPE) to ensure that humans are protected from direct contact with soil contaminated with carcinogenic PAHs, (2) calculate the Index of Additive Cancer Risk (IACR) to ensure that potable water resources are protected from carcinogenic PAHs, and (3) consider all relevant guidelines to protect ecological receptors from non-carcinogenic effects, in this table, for the land use in question.

IMPORTANT NOTE (2): For soil contaminated by coal tar or creosote mixtures, the calculated Benzo[a]pyrene Total Potency Equivalents (B[a]P TPE) concentration for soil samples should be multiplied by a safety factor of 3 prior to comparison with the SQG_{DH} to account for carcinogenic potential of alkylated and other PAHs present for which a Potency Equivalent Factor (PEF) does not currently exist, but which are likely to contribute to mixture carcinogenic potential.

		Land Use			
		Agricultural	**Residential/ Parkland**	**Commercial**	**Industrial**
	Guideline (SQG_F)–see table caption, IMPORTANT NOTE 1				
Human health guidelines/check values	**Human health guidelines/check values based on carcinogenic effects of PAHs** (potentially carcinogenic PAHs are Benz[a]anthracene, Benzo[a]pyrene, Benzo[b+j+k]fluoranthene[m], Benzo[g,h,i]perylene, Chrysene, Dibenz[a,h]anthracene, and Indeno[1,2,3-c,d]pyrene)				
	SQG_{HH}	NC	NC	NC	NC
	Direct contact[a] (SQG_{DH}) – ingestion, inhalation, and dermal exposures				
	1×10^{-6} incremental lifetime cancer risk	0.6 B[a]P TPE[b]	0.6 B[a]P TPE[b]	0.6 B[a]P TPE[b]	0.6 B[a]P TPE[b]
	1×10^{-5} incremental lifetime cancer risk	5.3 B[a]P TPE[b]	5.3 B[a]P TPE[b]	5.3 B[a]P TPE[b]	5.3 B[a]P TPE[b]
	Protection of indoor air quality (SQG_{IAQ})	NC	NC	NC	NC
	Off-site migration ($SQG_{OM\text{-}HH}$)	-	-	NC	NC
	Protection of potable water (SQG_{PW})	IACR≤1.0[c]	IACR≤1.0[c]	IACR≤1.0[c]	IACR≤1.0[c]
	Produce, meat, and milk (SQG_{FI})	NC	NC	-	-
Environmental health guidelines/check values	**Environmental health guidelines/check values based on non-carcinogenic effects of PAHs** (*do not use these values to protect humans; for carcinogenic PAHs consult the Human health guidelines above; to protect humans from non-carcinogenic effects of PAHs consult guidelines from other jurisdictions; if a PAH displays both cancer and non-cancer effects to humans, protect human health based on the threat from cancer*)				
	Environmental health guideline values for Acenaphthene				
	SQG_E[d]	NC	NC	NC	NC
	Soil contact (SQG_{SC})	NC	NC	NC	NC
	Soil and food ingestion (SQG_I)	21.5[e]	21.5[e]	-	-
	Protection of freshwater life[f] (SQG_{FL})	0.28[g]	0.28[g]	0.28[g]	0.28[g]
	Interim Soil Quality Criteria (CCME 1991)	no value	no value	no value	no value
	Environmental health guideline values for Acenaphthylene				
	SQG_E[d]	NC	NC	NC	NC
	Soil contact (SQG_{SC})	NC	NC	NC	NC
	Soil and food ingestion (SQG_I)	NC	NC	-	-
	Protection of freshwater life[f] (SQG_{FL})	320[h]	320[h]	320[h]	320[h]
	Interim Soil Quality Criteria (CCME 1991)	no value	no value	no value	no value

Continued…

		Land Use			
		Agricultural	**Residential/ Parkland**	**Commercial**	**Industrial**
Environmental health guidelines/check values	**Environmental health guideline values for Anthracene**				
	SQG_E[d]	2.5[p]	2.5[p]	32[p]	32[p]
	Soil contact (SQG_{SC})	2.5	2.5	32	32
	Soil and food ingestion (SQG_I)	61.5[e]	61.5[e]	-	-
	Protection of freshwater life[f] (SQG_{FL})	NA[g,i]	NA[g,i]	NA[g,i]	NA[g,i]
	Interim Soil Quality Criteria (CCME 1991)	no value	no value	no value	no value
	Environmental health guideline values for Benz[a]anthracene				
	SQG_E[d]	NC	NC	NC	NC
	Soil contact (SQG_{SC})	NC	NC	NC	NC
	Soil and food ingestion (SQG_I)	6.2[e]	6.2[e]	-	-
	Protection of freshwater life[f] (SQG_{FL})	NA[g,i]	NA[g, i]	NA[g, i]	NA[g, i]
	Interim Soil Quality Criteria (CCME 1991)	0.1[j]	1[j]	10[j]	10[j]
	Environmental health guideline values for Benzo[a]pyrene				
	SQG_E[d]	20[k]	20[k]	72[k]	72[k]
	Soil contact (SQG_{SC})	20	20	72	72
	Soil and food ingestion (SQG_I)	0.6[e]	0.6[e]	-	-
	Protection of freshwater life[f] (SQG_{FL})	8800[g]	8800[g]	8800[g]	8800[g]
	Provisional SQG_E (CCME 1997)	0.7	0.7	1.4	1.4
	Environmental health guideline values for Benzo[b]fluoranthene[r]				
	SQG_E[d]	NC	NC	NC	NC
	Soil contact (SQG_{SC})	NC	NC	NC	NC
	Soil and food ingestion (SQG_I)	6.2[e]	6.2[e]	-	-
	Protection of freshwater life[f] (SQG_{FL})	NA[h,i]	NA[h,i]	NA[h,i]	NA[h,i]
	Interim Soil Quality Criteria (CCME 1991)	0.1[j]	1[j]	10[j]	10[j]
	Environmental health guideline values for Benzo[k]fluoranthene[r]				
	SQG_E[d]	NC	NC	NC	NC
	Soil contact (SQG_{SC})	NC	NC	NC	NC
	Soil and food ingestion (SQG_I)	6.2[e]	6.2[e]	-	-
	Protection of freshwater life[f] (SQG_{FL})	NA[h,i]	NA[h,i]	NA[h,i]	NA[h,i]
	Interim Soil Quality Criteria (CCME 1991)	0.1[j]	1[j]	10[j]	10[j]
	Environmental health guideline values for Benzo[g,h,i]perylene				
	SQG_E[d]	NC	NC	NC	NC
	Soil contact (SQG_{SC})	NC	NC	NC	NC
	Soil and food ingestion (SQG_I)	NC	NC	-	-
	Protection of freshwater life[f] (SQG_{FL})	NA[h,i]	NA[h,i]	NA[h,i]	NA[h,i]
	Interim Soil Quality Criteria (CCME 1991)	no value	no value	no value	no value
	Environmental health guideline values for Chrysene				
	SQG_E[d]	NC	NC	NC	NC
	Soil contact (SQG_{SC})	NC	NC	NC	NC
	Soil and food ingestion (SQG_I)	6.2[e]	6.2[e]	-	-
	Protection of freshwater life[f] (SQG_{FL})	NA[h,i]	NA[h,i]	NA[h,i]	NA[h,i]
	Interim Soil Quality Criteria (CCME 1991)	no value	no value	no value	no value

Continued…

		Land Use			
		Agricultural	**Residential/ Parkland**	**Commercial**	**Industrial**
Environmental health guidelines/check values	**Environmental health guideline values for Dibenz[a,h]anthracene**				
	SQG_E[d]	NC	NC	NC	NC
	Soil contact (SQG_{SC})	NC	NC	NC	NC
	Soil and food ingestion (SQG_I)	NC	NC	-	-
	Protection of freshwater life[f] (SQG_{FL})	NA[h,i]	NA[h,i]	NA[h,i]	NA[h,i]
	Interim Soil Quality Criteria (CCME 1991)	0.1[j]	1[j]	10[j]	10[j]
	Environmental health guideline values for Fluoranthene				
	SQG_E[d]	50[p]	50[p]	180[p]	180[p]
	Soil contact (SQG_{SC})	50	50	180	180
	Soil and food ingestion (SQG_I)	15.4[e]	15.4[e]	-	-
	Protection of freshwater life[f] (SQG_{FL})	NA[g,i]	NA[g,i]	NA[g,i]	NA[g,i]
	Interim Soil Quality Criteria (CCME 1991)	no value	no value	no value	no value
	Environmental health guideline values for Fluorene				
	SQG_E[d]	NC	NC	NC	NC
	Soil contact (SQG_{SC})	NC	NC	NC	NC
	Soil and food ingestion (SQG_I)	15.4[e]	15.4[e]	-	-
	Protection of freshwater life[f] (SQG_{FL})	0.25[g]	0.25[g]	0.25[g]	0.25[g]
	Interim Soil Quality Criteria (CCME 1991)	no value	no value	no value	no value
	Environmental health guideline values for Indeno[1,2,3-c,d]pyrene				
	SQG_E[d]	NC	NC	NC	NC
	Soil contact (SQG_{SC})	NC	NC	NC	NC
	Soil and food ingestion (SQG_I)	NC	NC	-	-
	Protection of freshwater life[f] (SQG_{FL})	NA[h,i]	NA[h,i]	NA[h,i]	NA[h,i]
	Interim Soil Quality Criteria (CCME 1991)	0.1[j]	1[j]	10[j]	10[j]
	Environmental health guideline values for Naphthalene				
	SQG_E[d]	NC	NC	NC	NC
	Soil contact (SQG_{SC})	NC	NC	NC	NC
	Soil and food ingestion (SQG_I)	8.8	8.8	-	-
	Protection of freshwater life[f] (SQG_{FL})	0.013[g,l]	0.013[g,l]	0.013[g,l]	0.013[g,l]
	Provisional SQG_E (CCME 1997)	0.6[n]	0.6[n]	22[n]	22[n]
	Environmental health guideline values for Phenanthrene				
	SQG_E[d]	NC	NC	NC	NC
	Soil contact (SQG_{SC})	NC	NC	NC	NC
	Soil and food ingestion (SQG_I)	43.0[e]	43.0[e]	-	-
	Protection of freshwater life[f] (SQG_{FL})	0.046[g,l]	0.046[g,l]	0.046[g,l]	0.046[g,l]
	Interim Soil Quality Criteria (CCME 1991)	0.1[o]	5[o]	50[o]	50[o]
	Environmental health guideline values for Pyrene				
	SQG_E[d]	NC	NC	NC	NC
	Soil contact (SQG_{SC})	NC	NC	NC	NC
	Soil and food ingestion (SQG_I)	7.7[e]	7.7[e]	-	-
	Protection of freshwater life[f] (SQG_{FL})	NA[g,i]	NA[g,i]	NA[g,i]	NA[g,i]
	Interim Soil Quality Criteria (CCME 1991)	0.1[q]	10[q]	100[q]	100[q]

Continued…

	Land Use			
	Agricultural	**Residential/ Parkland**	**Commercial**	**Industrial**
The following guidelines/check pathways were evaluated for all PAHs appearing in the environmental section				
Livestock Watering (SQG_{LW})	NC	-	-	-
Irrigation Water (SQG_{IR})	NC	-	-	-
Nutrient and energy cycling check (SQG_{NEC})	NC	NC	NC	NC
Off-site migration check (SQG_{OM-E})	-	-	NC	NC

Notes: NA = not applicable; NC = not calculated; SQG_E = soil quality guideline for environmental health; SQG_{HH} = soil quality guideline for human health; SQG_I = soil quality guideline for protection of livestock and wildlife based on soil and food ingestion; SQG_{IR} = soil quality guideline for the protection of irrigation water; SQG_{IAQ} = soil quality guideline for the protection of indoor air quality; SQG_F = final soil quality guideline (for protection of environmental and human health); SQG_{LW} = soil quality guideline for protection of livestock based on water consumption; SQG_{NEC} = soil quality guideline check value for the protection of nutrient and energy cycling; SQG_{PW} = soil quality guideline for the protection of potable groundwater; SQG_{OM-E} = soil quality guideline check value for off-site migration of soils in consideration of environmental health risks; SQG_{OM-HH} = soil quality guideline check value for off-site migration of soils in consideration of human health risks; SQG_{SC} = soil quality guideline for soil contact by soil-dependent organisms (e.g., plants and invertebrates). The dash indicates a guideline/check value that is not part of the exposure scenario for this land use and therefore is not calculated.

a Guideline values for toddler pica soil ingestion have also been calculated for benzo[a]pyrene, acenaphthene, fluorene, anthracene and fluoranthene, but are several orders of magnitude higher than the direct contact guidelines. For more details on the pica guidelines, refer to section 7.1.4 of the scientific supporting document (CCME, 2008a).

b B[a]P TPE = Benzo[a]pyrene Total Potency Equivalents, which is the sum of estimated cancer potency relative to B[a]P for all potentially carcinogenic unsubstituted PAHs. The B[a]P TPE for a soil sample is calculated by multiplying the concentration of each PAH in the sample by its B[a]P Potency Equivalence Factor (PEF), given below, and summing these products (see Figure 2 for B[a]P TPE example calculation including PAH mixtures found in coal tar or creosote). B[a]P PEFs are order of magnitude estimates of carcinogenic potential and are based on the World Health Organization (WHO/IPCS 1998) scheme, as follows:

Benz[a]anthracene	0.1	Benzo[g,h,i]perylene	0.01	Indeno[1,2,3-c,d]pyrene	0.1
Benzo[a]pyrene	1	Chrysene	0.01		
Benzo[b+j+k]fluoranthene	0.1	Dibenz[a,h]anthracene	1		

c The Index of Additive Cancer Risk (IACR) assesses potential threats to potable groundwater water quality from leaching of carcinogenic PAH mixtures from soil. The IACR is calculated by dividing the soil concentration (numerator) of each carcinogenic PAH by its soil quality guideline for protection of potable water component value (denominator) to calculate a hazard index for each PAH, and then summing the hazard indices for the entire PAH mixture, as follows (see Figure 2 for IACR example calculation):

$$IACR = \frac{[Benz\,(a)anthracene\ ]}{0.33\ mg\cdot kg^{-1}} + \frac{[Benzo\,(b+j+k)\,fluoranthe\ ne\,]}{0.16\ mg\cdot kg^{-1}} + \frac{[Benzo\,(g,h,i)\,perylene\ ]}{6.8\ mg\cdot kg^{-1}} + \frac{[Benzo(a)pyrene]}{0.37 mg\cdot kg^{-1}} + \frac{[Chrysene]}{2.1 mg\cdot kg^{-1}} + \frac{[Dibenz(a,h)anthracene]}{0.23\ mg\cdot kg^{-1}} + \frac{[Indeno(1,2,3-c,d)pyrene]}{2.7 mg\cdot kg^{-1}}$$

The potable water component values were derived using a drinking water Maximum Allowable Concentration of 0.00001 mg/L for benzo(a)pyrene and the B[a]P PEFs listed in footnote b above, and the soil-to-groundwater model described in Appendix C of CCME (2006).

d The SQG_E is based on the lowest of the available environmental health guidelines (soil contact, soil and food ingestion, or protection of freshwater life). For PAHs where a soil contact guideline was not available, an overall SQG_E was not calculated.

e This guideline is considered provisional because minimum data requirements, as outlined in CCME (2006), were not met. The value is presented for users to consider applying at their own discretion, but it has not been used to determine the overall SQG_E recommended here.

f Modeling assumptions include the absence of biodegradation of PAHs in the subsurface environment, a highly conservative assumption.

g SQG_{FL} for freshwater life protection back-calculated based on CCME (2006) protocol, using pre-existing CCME Water Quality Guidelines (Freshwater Life) (CCME 1999).

h SQG_{FL} for freshwater life protection back-calculated from theoretically derived freshwater life thresholds based on baseline (narcosis-type) toxicity along with a Critical Body Residue (CBR) approach, assuming an internalized dose for aquatic life of 3.0 mmol PAH·kg^{-1} lipid is a threshold for chronic, non-lethal toxicity.

i A freshwater life protective guideline could not be calculated based on the assumed generic site/soil properties and the K_{OC} of the PAH, since the concentration in groundwater at the point of leaching would need to far exceed the solubility limit to account for a concentration that approaches the toxicity threshold at a point 10 m down-gradient.

[j] The interim soil quality criterion (CCME 1991) is retained as the environmental soil quality guideline for this land use because there was insufficient/inadequate data to calculate an SQG_E or provisional SQG_E. Consult the human health guidelines/check values to assess the human hazard of PAH mixtures containing this PAH.

[k] The SQG_E is based on the soil contact guideline value. The 2008 benzo[a]pyrene SQG_E replaces the 1997 provisional benzo[a]pyrene SQG_E. Consult the human health guidelines/check values to assess the human hazard of PAH mixtures containing this PAH.

[l] Users may wish to consider the application, on a site-specific basis, of the Soil Quality Guideline for the Protection of Freshwater Life where potential impacts on nearby surface water are a concern. This guideline value may be less than the common limit of detection in some jurisdictions. Consult appropriate jurisdiction for further guidance.

[m] Benzo[b]fluoranthene and benzo[j]fluoranthene tend to strongly co-elute under most gas chromatographic conditions. Furthermore, resolution between benzo[b]fluoranthene, benzo[j]fluoranthene, and benzo[k]fluoranthene is difficult to achieve when all three isomers are present in the soil matrix. Therefore, these three isomers have been considered together in deriving SQG_{HH} values.

[n] Data were insufficient/inadequate to update the 1997 provisional SQG_E and no attempt was made to calculate a SQG_{HH} or provisional SQG_{HH}, therefore the 1997 provisional SQG_E is retained as the soil quality guideline for the protection of environmental health for this land use. However, if there is concern for potential impacts to water bodies, the Soil Quality Guideline for the Protection of Freshwater Life (SQG_{FL}) should be applied. Consult other jurisdictions for the protection of human health from naphthalene.

[o] Data were insufficient/inadequate data to calculate an SQG_E or provisional SQG_E and no attempt was made to calculate a SQG_{HH}, or provisional $SQG_{HH,}$, therefore the interim soil quality criterion (CCME 1991) is retained as the environmental soil quality guideline for this land use. However, if there is concern for potential impacts to water bodies, the Soil Quality Guideline for the Protection of Freshwater Life (SQG_{FL}) should be applied. Consult other jurisdictions for the protection of human health from phenanthrene.

[p] The SQG_E is based on the soil contact guideline value.

[q] Data were insufficient/adequate data to calculate an SQG_E or provisional SQG_E and no attempt was made to calculate a SQG_{HH}, or provisional SQG_{HH}, therefore the interim soil quality criterion (CCME 1991) is retained as the environmental soil quality guideline for this land use. Consult other jurisdictions for the protection of human health from pyrene.

[r] Resolution between benzo[b]fluoranthene and benzo[k]fluoranthene gas chromatograph peaks may be difficult to achieve. When these two PAHs cannot be reported separately, report them as the sum of benzo[b+j+k]fluoranthene and compare this value to the guideline for benzo[b]fluoranthene.

Figure 8-2. Example of how to apply Canadian Soil Quality Guidelines for PAHs at a contaminated site

PAH concentration in soil (mg/kg dry weight) collected from a contaminated industrial site (fictitious data).

Acenaphthene	0.63	**Benzo[g,h,i]perylene**	**0.67**
Anthracene	1.4	**Chrysene**	**1.6**
Benz[a]anthracene	**4.5**	**Dibenz[a,h]anthracene**	**0.22**
Benzo[a]pyrene	**0.69**	**Indeno[1,2,3-c,d]pyrene**	**0.81**
Benzo[b]fluoranthene	**0.64**	Naphthalene	0.66
Benzo[k]fluoranthene	**0.62**	***(potentially carcinogenic PAHs are in bold)***	

Step 1 To ensure that humans are protected from direct contact with soil contaminated with carcinogenic PAHs, calculate the **Benzo[a]pyrene Total Potency Equivalents (B[a]P TPE)** using the following Equation (see factsheet text and Table 8-1, footnote b, for more details);

$$B[a]P\ TPE = \sum_{i=1}^{n}(C_i \times PEF_i)$$

where,
B[a]P TPE = concentration of the carcinogenic-PAH mixture, expressed as a total potency equivalent of B[a]P
n = number of carcinogenic PAHs (with an available PEF value)
C_i = concentration of carcinogenic-PAH compound *i*
PEF_i = potency equivalence factor for the carcinogenic-PAH compound *i* (unitless) (see Table 8-1, footnote b)

Take only the carcinogenic PAHs from the above table and calculate the B[a]P TPE as follows;

See Note 2 below for an explanation of this value

B[a]P TPE = (4.5 mg·kg^{-1} × 0.1) + (0.69 mg·kg^{-1} × 1) + (1.26 mg·kg^{-1} × 0.1) + (0.67 mg·kg^{-1} × 0.01) +
(1.6 mg·kg^{-1} × 0.01) + (0.22 mg·kg^{-1} × 1) + (0.81 mg·kg^{-1} × 0.1)
= 1.6 mg·kg^{-1}

Compare the B[a]P TPE of 1.6 mg·kg^{-1} to the SQG_{DH} in Table 8-1 with the desired level of acceptable risk. If the PAH mixture is found in soil co-contaminated with coal tar or creosote, the B[a]P TPE should be multiplied by a safety factor of 3 before comparison to the SQG_{DH}, as follows; B[a]P TPE = 1.6 mg·kg^{-1} × 3 = 4.8 mg·kg^{-1}

Step 2 To ensure that potable water resources are protected from carcinogenic PAHs, calculate the **Index of Additive Cancer Risk (IACR)** using the following equation (see factsheet text and Table 8-1, footnote c, for more details);

$$IACR = \frac{[Benz\,(a)anthracene]}{0.33\ mg\cdot kg^{-1}} + \frac{[Benzo\,(b+j+k)\,fluoranthene]}{0.16\ mg\cdot kg^{-1}} + \frac{[Benzo\,(g,h,i)\,perylene]}{6.8\ mg\cdot kg^{-1}} + \frac{[Benzo(a)pyrene]}{0.37 mg\cdot kg^{-1}} + \frac{[Chrysene]}{2.1 mg\cdot kg^{-1}} + \frac{[Dibenz(a,h)anthracene]}{0.23\ mg\cdot kg^{-1}} + \frac{[Indeno(1,2,3-c,d)pyrene]}{2.7 mg\cdot kg^{-1}}$$

Take only the carcinogenic PAHs from the above table and calculate the IACR as follows;

See Note 2 below for an explanation of this value

IACR = (4.5 mg·kg^{-1}/0.33 mg·kg^{-1}) + (1.26 mg·kg^{-1}/0.16 mg·kg^{-1}) + (0.67 mg·kg^{-1}/6.8 mg·kg^{-1}) +
(0.69 mg·kg^{-1}/0.37 mg·kg^{-1}) + (1.6 mg·kg^{-1}/2.1 mg·kg^{-1}) + (0.22 mg·kg^{-1}/0.23 mg·kg^{-1}) +
(0.81 mg·kg^{-1}/2.7 mg·kg^{-1})
= 25

The resulting IACR value, in this case = 25, is equivalent to a hazard index and should not exceed a value of 1.0 (Table 8-1 shows that the SQG_{PW} should be ≤ 1).

Note that for both the B[a]P TPE and IACR calculations;
1. no carcinogenic PAH should be left out of the calculations. If PAHs are suspected at a site, soil samples should be analyzed for the full suite of carcinogenic PAHs. If analysis returns non-detects, and until further guidance, enter ½

the detection limit into the formulas. Consult the appropriate jurisdiction to confirm that this advice does not conflict with program policy for dealing with non-detects at contaminated sites.

2. if concentrations of benzo[b]fluoranthene, benzo[j]fluoranthene, and benzo[k]fluoranthene are reported separately, they should be summed together and expressed as a single value for benzo[b+j+k]fluoranthene. In this example, benzo[b+j+k]fluoranthene = 0.64 $mg \cdot kg^{-1}$ + 0.62 $mg \cdot kg^{-1}$ = 1.26 $mg \cdot kg^{-1}$

→ *Step* **3**

To protect environmental health from non-carcinogenic effects of PAHs (i.e. hazard assessed based on non-carcinogenic modes of action), compare individual PAHs to the appropriate Soil Quality Guideline (SQG) in the bottom half of Table 8-1.

At this point, the cancer hazard to humans from potentially carcinogenic PAHs has been assessed by calculating the B[a]P TPE and IACR in Steps 1 and 2, respectively, above. Now the non-carcinogenic hazard of PAHs must be assessed. Note that the non-carcinogenic hazard posed by PAHs to humans was not assessed; consult guidelines from other jurisdictions for protection. Additionally, if a PAH displays both cancer and non-cancer effects to humans, protect human health based on the threat from cancer.

Take all PAH concentration data from the industrial site, and compare it to the appropriate environmental SQG from the Environmental health section of Table 8-1. Results of comparisonare presented below;

Acenaphthene	: There is no SQG for the protection of environmental health reported in Table 8-1 for this PAH (i.e. SQG_E was not calculated). However, Table 8-1 does provide value(s) for individual environmental soil pathway(s) that could be developed. Consult guidelines from other jurisdictions for the protection of humans from non-carcinogenic effects of this PAH. This conclusion would also apply to acenaphthylene and fluorene if it were present at the site.
Anthracene	: The SQG_E is valid for the protection environmental health from non-carcinogenic effects of this PAH. Consult guidelines from other jurisdictions for the protection of humans from non-carcinogenic effects of this PAH.
Benz[a]anthracene	: There is no SQG_E for this PAH, therefore Interim Soil Quality Criteria (CCME 1991) is retained for the protection of environmental health from non-carcinogenic effects of this PAH. For human health, the hazard posed by this PAH is assessed solely based on its carcinogenic potential (see Steps 1 and 2).
Benzo[a]pyrene	: Same conclusion as for anthracene.
Benzo[b]fluoranthene	: Same conclusion as for benz[a]anthracene. Additionally, if benzo[b]fluoranthene and benzo[k]fluoranthene cannot be reported separately, report them as the sum of benzo[b+j+k]fluoranthene and compare this value to the 1991 Interim guideline value for benzo[b]fluoranthene.
Benzo[k]fluoranthene	: Same conclusion as for benzo[b]fluoranthene.
Benzo[g,h,i]perylene	: There is no SQG reported for the protection of environmental health,or individual environmental soil pathways (Table 8-1) for this PAH. For human health, the hazard posed by this PAH is assessed solely based on its carcinogenic potential (see Steps 1 and 2).
Chrysene	: There is no SQG for the protection of environmental health reported in Table 8-1 for this PAH. However, Table 8-1 provides value(s) for individual environmental soil pathway(s) that could be developed. For human health, the hazard posed by this PAH is assessed solely based on its carcinogenic potential (see Steps 1 and 2).
Dibenz[a,h]anthracene	: Same conclusion as for benz[a]anthracene.
Indeno[1,2,3-c,d]pyrene	: Same conclusion as for benz[a]anthracene.
Naphthalene	: The SQG_{FL} is valid for the protection of environmental health from non-carcinogenic effects of this PAH. This conclusion would also apply to phenanthrene if it were present at the site. Consult guidelines from other jurisdictions for the protection of humans from non-carcinogenic effects of this PAH.

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APPENDICES

Appendix I: Calculation of CANCER RISK-BASED human health guidelines

This appendix provides calculations for the derivation of the direct human health-based soil guidelines (SQG_{DH}) presented in Section 7.1 that are based on cancer-type risks. Derivations are provided based on incremental lifetime cancer risks (ILCR) of both 10^{-6} and 10^{-5}.

A.1 Benzo[a]pyrene SQG_{DH} Based on Soil Ingestion and Inhalation

The preliminary soil quality guideline for benzo[a]pyrene, based on exposures via soil ingestion and inhalation, is calculated as follows:

$$PSQG_{DH} = \frac{RSD \times BW}{[(AF_G \times SIR) + (AF_S \times SR) + (AF_L \times IR_S)] \times ET} + BSC \qquad (1)$$

where -

$PSQG_{DH}$	=	preliminary direct human health-based soil quality guideline ($mg \cdot kg^{-1}$)		(calculated)
RSD	=	risk specific dose ($mg \cdot kg^{-1}$ per day)	=	0.00435 µg Benzo[a]pyrene·kg $bw^{-1} \cdot day^{-1}$ for an incremental cancer risk of 10^{-5}, based on a cancer slope factor of 2.3 $(mg \cdot kg^{-1} \cdot day^{-1})^{-1}$
			=	0.000435 µg Benzo[a]pyrene·kg $bw^{-1} \cdot day^{-1}$ for an incremental cancer risk of 10^{-6}, based on a cancer slope factor of 2.3 $(mg \cdot kg^{-1} \cdot day^{-1})^{-1}$
BW	=	body weight (kg)	=	70.7 kg for an average Canadian adult (CCME, 2006; HC, 2004a)
BSC	=	background soil concentration ($\mu g \cdot g^{-1}$)	=	0.07 $\mu g \cdot g^{-1}$ for benzo[a]pyrene (Randall Jones, Ontario Ministry of the Environment, personal communication)
AF_G	=	relative absorption factor for gut (unitless)	=	1.0, or 100% (assumed)
AF_L	=	relative absorption factor for lung (unitless)	=	1.0, or 100% (assumed)
AF_S	=	relative absorption factor for skin (unitless)	=	0.34 (Shatkin *et al.*, 2002)
SIR	=	soil ingestion rate ($g \cdot d^{-1}$)	=	2×10^{-2} $g \cdot day^{-1}$ (CCME, 2006; HC, 2004a)

IR_S	=	soil inhalation rate ($g \cdot d^{-1}$)	=	$15.8\ m^3 \cdot d^{-1}$ x $0.76\ \mu g \cdot m^{-3}$ ($=12\ \mu g \cdot d^{-1}$ or $1.2 \times 10^{-5}\ g \cdot d^{-1}$) (HC, 2004a)
SR	=	soil dermal contact rate ($g \cdot d^{-1}$)	=	$1.14 \times 10^{-1}\ g \cdot day^{-1}$ [CCME (2006): (hand surface area of 890 cm^2 x soil adherence factor of 1×10^{-4} $g \cdot cm^{-2} \cdot day^{-1}$)+(other surface area of 2500 cm^2 x soil adherence factor of $1 \times 10^{-5}\ g \cdot cm^{-2} \cdot day^{-1}$)
ET	=	exposure term (unitless)	=	1.0; i.e., individual is exposed continuously through lifetime and at all times (75 yr/75 yr; assumed)

The background soil concentration is based on the mean concentration of benzo[a]pyrene measured in soil samples from Ontario old urban parkland sites, as part of the Ontario Typical Range program (Randall Jones, Ontario Ministry of Environment, personal communication).

Therefore –

For an ILCR of 10^{-5}:

$$PSQG_{DH} = \frac{0.00435\ \mu g \cdot kg\ bw^{-1} \cdot day^{-1} \times 70.7\ kg\ bw}{(1.0 \times 2.0 \times 10^{-2}\ g \cdot day^{-1}) + (0.34 \times 0.114\ g \cdot day^{-1}) + (1.0 \times 1.2 \times 10^{-5}\ g \cdot day^{-1})} + 0.07\ \mu g/g$$

$$PSQG_{DH} = \frac{0.00435\ \mu g \cdot kg\ bw^{-1} \cdot day^{-1} \times 70.7\ kg\ bw}{(2.0 \times 10^{-2}\ g \cdot day^{-1}) + (3.88 \times 10^{-2}\ g \cdot day^{-1}) + (1.2 \times 10^{-5}\ g \cdot day^{-1})} + 0.07\ \mu g/g$$

$= 5.3\ \mu g$ benzo[a]pyrene$\cdot g^{-1}$ soil or 5.3 $mg \cdot kg^{-1}$ B[a]P

For an ILCR of 10^{-6}:

$$PSQG_{DH} = \frac{0.000435\ \mu g \cdot kg\ bw^{-1} \cdot day^{-1} \times 70.7\ kg\ bw}{(1.0 \times 2.0 \times 10^{-2}\ g \cdot day^{-1}) + (0.34 \times 0.114\ g \cdot day^{-1}) + (1.0 \times 1.2 \times 10^{-5}\ g \cdot day^{-1})} + 0.07\ \mu g/g$$

$$PSQG_{DH} = \frac{0.000435\ \mu g \cdot kg\ bw^{-1} \cdot day^{-1} \times 70.7\ kg\ bw}{(2.0 \times 10^{-2}\ g \cdot day^{-1}) + (3.88 \times 10^{-2}\ g \cdot day^{-1}) + (1.2 \times 10^{-5}\ g \cdot day^{-1})} + 0.07\ \mu g/g$$

$= 0.6\ \mu g$ benzo[a]pyrene$\cdot g^{-1}$ soil, or 0.60 $mg \cdot kg^{-1}$

This preliminary soil quality guideline is interpreted to be applicable not just to residential land use sites, but also agricultural, commercial and industrial areas. Especially in the case of commercial and industrial area, humans are expected to be exposed less than 24 h/day over an entire year, and for less than the entire lifespan of an individual. Nonetheless, the available experimental data on cancer induction in mammalian species by B[a]P is for equivalent exposures that are much less than the entire life expectancy of the test animals, and there remains considerable uncertainty about whether incremental cancer risks decrease in direct proportion to any amortization of human exposures based on periods of time approaching years to decades.

The major sources of uncertainty in this estimate, as well as the SQG_{DH} based on soil ingestion and inhalation, are deemed to be as follows:

- oral cancer slope factor estimate: range of various proposed values for B[a]P of 0.2 to 11 $(mg \cdot kg^{-1} \cdot day^{-1})^{-1}$ relative to Health Canada value of 2.3 $(mg \cdot kg^{-1} \cdot day^{-1})^{-1}$:

- no consideration of benzo[a]pyrene exposures via diet.

This second point is deemed to be of only minor importance. CCME (1999b) provided an estimate of the average daily dietary intake (EDI: estimated daily intake) by Canadians of benzo[a]pyrene, which is 2.0×10^{-3} to 5.9×10^{-3} $\mu g \cdot kg\ bw^{-1} \cdot day^{-1}$, excluding additional PAH exposures through cigarette smoking. While this is in fact higher than the risk-specific dose used by Health Canada to develop a human health-based soil quality guideline (0.4×10^{-3} $\mu g \cdot kg\ bw^{-1} \cdot day^{-1}$, for an ILCR of 10^{-6}), it is irrelevant. For carcinogens, it is assumed that some level of risk exists at any level of exposure other than zero. Therefore, management of cancer-related human health risks is a policy decision based on incremental risks, including those beyond background exposures (CCME, 2006).

Appendix II: Collated PAH Toxicity for Ecologically Relevant Soil Microbial Processes

Organism	Effect	Endpoint	Effective Concentration ($mg \cdot kg^{-1}$)	Compound(s)	Exposure Period	Soil pH	Org C(%)	Soil Conditions/ Test Substrate	Spike or extraction methods	Reference
Soil bacteria	Nitrification	NOEC	79	Pyrene	4 w	6.2	1.6%	"Askov" soil - sieved through 2 mm. 38.4% coarse sand; 23.6% fine sand; 23.6% coarse silt; 10.0% fine silt; 13.0% clay. Density = 1.135 $g \cdot cm^{-3}$; CEC = 8.14 meq/100 g. Three replicates per conc. x 8 conc (1, 3, 10, 30, 300, 1,000, 3,000 $mg \cdot kg^{-1}$). Inoculum added after solvent evaporation and addition of nitrogen source. Nitrogen sourse was horn meal (9.1% nitrogen content, C:N ratio = 3.48 mol/mol. 57% water holding capacity, sealed containers.	Nominal, in acetone carrier	Sverdrup *et al.*, 2002c
Soil bacteria	Nitrification	EC10	130 (CI: 58-170)	Pyrene	4 w	6.2	1.6%	As above.	As above.	Sverdrup *et al.*, 2002c
Soil Nitrifying bacteria	Nitrification	NOEC	24	Fluoranthene	4 w	6.2	1.6%	As above.	As above.	Sverdrup *et al.*, 2002c
Soil bacteria	Nitrification	EC10	13 (CI: 1.5-66)	Fluoranthene	4 w	6.2	1.6%	As above.	As above.	Sverdrup *et al.*, 2002c
Heterotrophic flagellates	Total Number	EC5	2,200	Fluoranthene	4 w	6.2	1.6%	As above.	As above.	Sverdrup *et al.*, 2002c
Soil bacteria	Genetic Diversity	NOEC	3,000	Fluoranthene	4 w	6.2	1.6%	As above.	As above.	Sverdrup *et al.*, 2002c
Soil bacteria	Nitrification	NOEC	26	Phenanthrene	4 w	6.2	1.6%	As above.	As above.	Sverdrup *et al.*, 2002c
Soil bacteria	Nitrification	EC10	42 (CI: 0-70)	Phenanthrene	4 w	6.2	1.6%	As above.	As above.	Sverdrup *et al.*, 2002
Soil bacteria	Nitrification	EC50	250 (CI: 220-	Phenanthrene	4 w	6.2	1.6%	As above.	As above.	Sverdrup *et*

Organism	Effect	Endpoint	Effective Concentration ($mg \cdot kg^{-1}$)	Compound(s)	Exposure Period	Soil pH	Org C(%)	Soil Conditions/ Test Substrate	Spike or extraction methods	Reference
			380)							*al.*, 2002c
Protozoa	Total Number	EC5	2,400	Phenanthrene	4 w	6.2	1.6%	As above.	As above.	Sverdrup *et al.*, 2002c
Heterotrophic flagellates	Total Number	EC5	250	Phenanthrene	4 w	6.2	1.6%	As above.	As above.	Sverdrup *et al.*, 2002c
Soil bacteria	Nitrification	NOEC	72	Fluorene	4 w	6.2	1.6%	As above.	As above.	Sverdrup *et al.*, 2002c
Soil bacteria	Nitrification	EC10	33 (CI: 0-72)	Fluorene	4 w	6.2	1.6%	As above.	As above.	Sverdrup *et al.*, 2002c
Soil bacteria	Nitrification	EC50	190 (CI:160-220)	Fluorene	4 w	6.2	1.6%	As above.	As above.	Sverdrup *et al.*, 2002c

Appendix III: Collated Soil Invertebrate PAH Toxicity Data

Organism	Effect	Endpoint	Effective Concentration ($mg \cdot kg^{-1}$)	Compound(s)	Exposure Period	Soil pH	Org C (%)	Soil Conditions	Spike or extraction methods	Reference
Perionyx excavatus (India blue earthworm)	Mortality	LC50	170 (CI: 118-245)	9H-Fluorene in 2-propanone as a carrier	48 h	6.0	NA	Artificial Soil, 69% sand, 20% clay, 10% peat, direct application exposure route, 4 replicates per concentration. (35% moisture content)	Nominal	Neuhauser *et al.*, 1986
Allolobophora tuberculata (Earthworm)	Mortality	LC50	206 (NR: 164-260)	As above	48 h	6.0	NA	As above	Nominal	Neuhauser *et al.*, 1986
Eisenia fetida (Earthworm)	Mortality	LC50	173 (CI: 150-201)	As above	2 w	6.0	NR	As above	Nominal	Neuhauser *et al.*, 1985
Eudrilus eugeniae (African earthworm)	Mortality	LC50	197 (CI 100-384)	As above	48 h	6.0	NR	As above	Nominal	Neuhauser *et al.*, 1986
Eisenia fetida (Earthworm)	Growth	EC50	1410	9H-Fluorene in 2-propanone, water as a carrier	8 w	NR	NR	Artificial Soil, 20% moisture, (particle size not defined), soil slurry exposure route, exposure concentrations ($mg \cdot kg^{-1}$): 0, 250, 500, 750, 1000	Nominal	Neuhauser and Callahan, 1990
Eisenia fetida (Earthworm)	Reproduction	NOEC	500	As above	8 w	NR	NR	As above	Nominal	Neuhauser and Callahan, 1990
Eisenia fetida (Earthworm)	Reproduction	LOEC	750	As above	8 w	NR	NR	As above	Nominal	Neuhauser and Callahan, 1990
Eisenia fetida (Earthworm)	Reproduction	EC50	1180	As above	8 w	NR	NR	As above	Nominal	Neuhauser and Callahan, 1990
Folsomia fimetaria (Springtail)	Mortality	LC50	107 (on basis of initial measured conc.)	Acenaphthene	21 d	6.2 (H_2O method)	1.6	"Askov" soil - sieved through 2 mm. 38.4% coarse sand; 23.6% fine sand; 23.6% coarse silt;10.0% fine silt; 13.0% clay. Density = 1.135 $g \cdot cm^{-3}$; CEC = 8.14 meq/100 g. 4 repl/conc. X 6 concentrations (0, 10, 30, 100, 300, 1000 $mg \cdot kg^{-1}$ dw)@ 50% water holding capacity.Closed containers.	Nominal, in acetone carrier. Then measured conc. for 10, 100 and 1,000 $mg \cdot kg^{-1}$ substrates at days 0 and 21.	Sverdrup *et al.*, 2002a
Folsomia fimetaria (Springtail)	Reproduction	EC10	31 (95% CI = 0 - 42) (on basis of initial measured conc.)	Acenaphthene	21 d	6.2 (H_2O method)	1.6	As above.	As above.	Sverdrup *et al.*, 2002a
Folsomia fimetaria (Springtail)	Mortality	LC50	145 (on basis of initial measured conc.)	Acenaphthylene	21 d	6.2 (H_2O method)	1.6	As above.	As above.	Sverdrup *et al.*, 2002a
Folsomia fimetaria (Springtail)	Reproduction	EC10	23 (95% CI = 12 - 31) (on basis of initial measured conc.)	Acenaphthylene	21 d	6.2 (H_2O method)	1.6	As above.	As above.	Sverdrup *et al.*, 2002a

Organism	Effect	Endpoint	Effective Concentration ($mg \cdot kg^{-1}$)	Compound(s)	Exposure Period	Soil pH	Org C (%)	Soil Conditions	Spike or extraction methods	Reference
Folsomia fimetaria (Springtail)	Mortality	LC50	67 (95% CI = 44 - 98) (on basis of initial measured conc.)	Anthracene	21 d	6.2 (H_2O method)	1.6	As above.	As above.	Sverdrup *et al.*, 2002a
Folsomia fimetaria (Springtail)	Reproduction	EC10	5 (95% CI = 3.2 - 12) (on basis of initial measured conc.)	Anthracene	21 d	6.2 (H_2O method)	1.6	As above.	As above.	Sverdrup *et al.*, 2002a
Folsomia fimetaria (Springtail)	Mortality	LC50	>980 (on basis of initial measured conc.)	Benz[a]anthracene	21 d	6.2 (H_2O method)	1.6	As above.	Nominal, in acetone carrier. The measured conc. for 1,000 $mg \cdot kg^{-1}$ substrates at day 0 (initial).	Sverdrup *et al.*, 2002a
Folsomia fimetaria (Springtail)	Reproduction	EC10	>980 (on basis of initial measured conc.)	Benz[a]anthracene	21 d	6.2 (H_2O method)	1.6	As above.	As above.	Sverdrup *et al.*, 2002a
Eisenia fetida (Earthworm)	Mortality	NOEC	26,000	Benzo[a]pyrene	14 d	4.1	0.14	Artificial soil; sand 70%m silt 13%m clay 17%	Measured	Environment Canada, 1995 (as reported in Environment Canada, 1999)
Eisenia fetida (Earthworm)	Mortality	LC51	10	Benzo[a]pyrene in 2-propanone as a carrier	28 d	NR	2.3	Natural Soil (particle size not defined), direct application exposure route, exposure concentrations ($\mu mol \cdot kg^{-1}$): 0, 40, 400, 6-9 replicates (40% moisture content)	Nominal (in 2-Propanone as a carrier)	Achazi *et al.*, 1995
Eisenia fetida (Earthworm)	Mortality	LC59	100	Benzo[a]pyrene in 2-propanone as a carrier	28 d	NR	2.3	As above.	As above.	Achazi *et al.*, 1995
Eisenia fetida (Earthworm)	Growth	EC12	10	Benzo[a]pyrene in 2-propanone as a carrier	28 d	NR	2.3	As above.	As above.	Achazi *et al.*, 1995
Eisenia fetida (Earthworm)	Growth	EC18	100	Benzo[a]pyrene in 2-propanone as a carrier	28 d	NR	2.3	As above.	As above.	Achazi *et al.*, 1995
Eisenia fetida (Earthworm)	Reproduction	EC(91)	10	Benzo[a]pyrene in 2-propanone as a carrier	28 d	NR	2.3	As above.	As above.	Achazi *et al.*, 1995
Eisenia fetida (Earthworm)	Reproduction	EC(96)	100	Benzo[a]pyrene in 2-propanone as a carrier	28 d	NR	2.3	As above.	As above.	Achazi *et al.*, 1995
Enchytraeus crypticus (Earthworm)	Reproduction	EC(18) (LOEC)	40 $\mu mol \cdot kg^{-1}$	Benzo[a]pyrene in 2-propanone as a carrier	30 d	NR	2.3	As above.	As above.	Achazi *et al.*, 1995
Enchytraeus crypticus (Earthworm)	Reproduction	EC(35)	400 $\mu mol \cdot kg^{-1}$	Benzo[a]pyrene in 2-propanone as a carrier	30 d	NR	2.3	As above.	As above.	Achazi *et al.*, 1995
Folsomia fimetaria (Springtail)	Mortality	LC50	>840 (on basis of initial measured conc.)	Benzo[a]pyrene	21 d	6.2 (H_2O method)	1.6	"Askov" soil - sieved through 2 mm. 38.4% coarse sand; 23.6% fine sand; 23.6% coarse silt;10.0% fine silt;	Nominal, in acetone carrier. The measured conc. for	Sverdrup *et al.*, 2002a

Organism	Effect	Endpoint	Effective Concentration ($mg \cdot kg^{-1}$)	Compound(s)	Exposure Period	Soil pH	Org C (%)	Soil Conditions	Spike or extraction methods	Reference
								13.0% clay. Density = 1.135 g/cm^3; CEC = 8.14 meq/100 g. 4 repl/conc. X 6 concentrations (0, 16, 32, 64, 128, 1000 $mg \cdot kg^{-1}$ dw)@ 50% water holding capacity.Closed containers.	1,000 $mg \cdot kg^{-1}$ substrates at day 0 (initial).	
Folsomia fimetaria (Springtail)	Reproduction	EC10	>840 (on basis of initial measured conc.)	Benzo[a]pyrene	21 d	6.2 (H_2O method)	1.6	As above.	As above.	Sverdrup *et al.*, 2002a
Folsomia fimetaria (Springtail)	Mortality	LC50	>360 (on basis of initial measured conc.)	Benzo[b]fluoranthene	21 d	6.2 (H_2O method)	1.6	As above.	As above.	Sverdrup *et al.*, 2002a
Folsomia fimetaria (Springtail)	Reproduction	EC10	>360 (on basis of initial measured conc.)	Benzo[b]fluoranthene	21 d	6.2 (H_2O method)	1.6	As above.	As above.	Sverdrup *et al.*, 2002a
Folsomia fimetaria (Springtail)	Mortality	LC50	>560 (on basis of initial measured conc.)	Benzo[k]fluoranthene	21 d	6.2 (H_2O method)	1.6	As above.	As above.	Sverdrup *et al.*, 2002a
Folsomia fimetaria (Springtail)	Reproduction	EC10	>560 (on basis of initial measured conc.)	Benzo[k]fluoranthene	21 d	6.2 (H_2O method)	1.6			Sverdrup *et al.*, 2002a
Folsomia fimetaria (Springtail)	Mortality	LC50	>1030 (on basis of initial measured conc.)	Chrysene	21 d	6.2 (H_2O method)	1.6	As above.	As above.	Sverdrup *et al.*, 2002a
Folsomia fimetaria (Springtail)	Reproduction	EC10	>1030 (on basis of initial measured conc.)	Chrysene	21 d	6.2 (H_2O method)	1.6	As above.	As above.	Sverdrup *et al.*, 2002a
Folsomia fimetaria (Springtail)	Mortality	LC50	>780 (on basis of initial measured conc.)	Dibenz[a,h]anthracene	21 d	6.2 (H_2O method)	1.6	As above.	As above.	Sverdrup *et al.*, 2002a
Folsomia fimetaria (Springtail)	Reproduction	EC10	>780 (on basis of initial measured conc.)	Dibenz[a,h]anthracene	21 d	6.2 (H_2O method)	1.6	"Askov" soil - sieved through 2 mm. 38.4% coarse sand; 23.6% fine sand; 23.6% coarse silt;10.0% fine silt; 13.0% clay. Density = 1.135 g/cm^3; CEC = 8.14 meq/100 g. 4 repl/conc. X 6 concentrations (0, 10, 30, 100, 300, 1000 $mg \cdot kg^{-1}$ dw)@ 50% water holding capacity.Closed containers.	Nominal, in acetone carrier. The measured conc. for 1,000 $mg \cdot kg^{-1}$ substrates at day 0 (initial).	Sverdrup *et al.*, 2002a
Eisenia fetida (Earthworm)	Mortality	LC10	1,000	Fluoranthene	28 d	NR	NR	Artificial Soil (particle size not defined)	NR	Kordel *et al.*, 1984
Eisenia fetida (Earthworm)	Mortality	NOEC	100	Fluoranthene	28 d	NR	NR	Artificial Soil (particle size not defined)	NR	Kordel *et al.*, 1984
Eisenia veneta (Earthworm)	Mortality	LC50	416 (CI: 308-710)	Fluoranthene in acetone carrier	28 d	6.2	1.6	"Askov" soil - sieved through 2 mm. 38.4% coarse sand; 23.6% fine sand; 23.6% coarse silt;10.0% fine silt; 13.0% clay. Density = 1.135 g/cm^3; CEC = 8.14 meq/100 g. 4 repl/conc.	Nominal, in acetone carrier. Then measured conc. for 10, 100 and 1,000 $mg \cdot kg^{-1}$ substrates at	Sverdrup *et al.*, 2002e

Organism	Effect	Endpoint	Effective Concentration ($mg \cdot kg^{-1}$)	Compound(s)	Exposure Period	Soil pH	Org C (%)	Soil Conditions	Spike or extraction methods	Reference
								X 6 concentrations (0, 10, 30, 100, 300, 1000 $mg \cdot kg^{-1}$ dw)@ 50% water holding capacity.Closed containers.	days 0 and 21.	
Eisenia veneta (Earthworm)	Growth	EC50	166 (CI: 153 - 178)	Fluoranthene in acetone carrier	28 d	6.2	1.6	As above.	As above.	Sverdrup *et al.*, 2002e
Eisenia veneta (Earthworm)	Growth	EC10	113 (CI: 101-124)	Fluoranthene in acetone carrier	28 d	6.2	1.6	As above.	As above.	Sverdrup *et al.*, 2002e
Enchytraeus crypticus (Earthworm)	Mortality	LC50	>2,500	Fluoranthene in acetone carrier	21 d	6.2	1.6	"Askov" soil - sieved through 2 mm. 38.4% coarse sand; 23.6% fine sand; 23.6% coarse silt;10.0% fine silt; 13.0% clay. Density = 1.135 g/cm^3; CEC = 8.14 meq/100 g. 4 repl/conc. X 6 concentrations (0, 10, 30, 100, 300, 1000 $mg \cdot kg^{-1}$ dw)@ 50% water holding capacity.Closed containers.	Nominal, in acetone carrier. Then measured conc. for 10, 100 and 1,000 $mg \cdot kg^{-1}$ substrates at days 0 and 21.	Sverdrup *et al.*, 2002d
Enchytraeus crypticus (Earthworm)	Reproduction	EC50	61 (CI: 20 - 85)	Fluoranthene in acetone carrier	21 d	6.2	1.6	As above.	As above.	Sverdrup *et al.*, 2002d
Enchytraeus crypticus (Earthworm)	Reproduction	EC10	15 (CI: 11 - 33)	Fluoranthene in acetone carrier	21 d	6.2	1.6	As above.	As above.	Sverdrup *et al.*, 2002d
Enchytraeus crypticus (Earthworm)	Reproduction	NOEC	38	Fluoranthene	21 d	6.2	1.6	As above.	As above.	Sverdrup *et al.*, 2002d
Enchytraeus crypticus (Earthworm)	Reproduction	LOEC	6 $mmol \cdot kg^{-1}$	Fluoranthene (in 2-propanone as a carrier)	30 d	NR	2.3	Natural Soil (particle size not defined), 6-9 replicates	Nominal (in 2-Propanone as a carrier)	Achazi *et al.*, 1995
Folsomia fimetaria (Springtail)	Mortality	LC50	81 (95% CI = 73 - 90)(on basis of initial measured conc.)	Fluoranthene	21 d	6.2 (H_2O method)	1.6	"Askov" soil - sieved through 2 mm. 38.4% coarse sand; 23.6% fine sand; 23.6% coarse silt;10.0% fine silt; 13.0% clay. Density = 1.135 g/cm^3; CEC = 8.14 meq/100 g. 4 repl/conc. X 6 concentrations (0, 10, 30, 100, 300, 1000 $mg \cdot kg^{-1}$ dw)@ 50% water holding capacity.Closed containers.	Nominal, in acetone carrier. Then measured conc. for 10, 100 and 1,000 $mg \cdot kg^{-1}$ substrates at days 0 and 21.	Sverdrup *et al.*, 2001, 2002a
Folsomia fimetaria (Springtail)	Reproduction	EC10	37 (95% CI = 26 - 48)(on basis of initial measured conc.)	Fluoranthene	21 d	6.2 (H_2O method)	1.6	"Askov" soil - sieved through 2 mm. 38.4% coarse sand; 23.6% fine sand; 23.6% coarse silt;10.0% fine silt; 13.0% clay. Density = 1.135 g/cm^3; CEC = 8.14 meq/100 g. 4 repl/conc. X 6 concentrations (0, 10, 30, 100, 300, 1000 $mg \cdot kg^{-1}$ dw)@ 50% water holding capacity.Closed containers.	As above.	Sverdrup *et al.*, 2001, 2002a
Folsomia fimetaria (Springtail)	Reproduction	EC50	51	Fluoranthene	21 d	6.2	1.6	"Askov" soil - sieved through 2 mm. 38.4% coarse sand; 23.6% fine sand; 23.6% coarse silt;10.0% fine silt;	As above.	Sverdrup *et al.*, 2001

Organism	Effect	Endpoint	Effective Concentration ($mg \cdot kg^{-1}$)	Compound(s)	Exposure Period	Soil pH	Org C (%)	Soil Conditions	Spike or extraction methods	Reference
								13.0% clay. Density = 1.135 g/cm^3; CEC = 8.14 meq/100 g. 4 repl/conc. X 6 concentrations (0, 10, 30, 100, 300, 1000 $mg \cdot kg^{-1}$ dw)@ 50% water holding capacity.Closed containers.		
Folsomia fimetaria (Springtail)	Reproduction	NOEC	47	Fluoranthene	21 d	6.2	1.6	"Askov" soil - sieved through 2 mm. 38.4% coarse sand; 23.6% fine sand; 23.6% coarse silt;10.0% fine silt; 13.0% clay. Density = 1.135 g/cm^3; CEC = 8.14 meq/100 g. 4 repl/conc. X 6 concentrations (0, 10, 30, 100, 300, 1000 $mg \cdot kg^{-1}$ dw)@ 50% water holding capacity.Closed containers.	As above.	Sverdrup *et al.*, 2001
Eisenia veneta (Earthworm)	Mortality	LC50	69 (CI: 42 - 83)	Fluorene in acetone carrier	28 d	6.2	1.6	As above.	As above.	Sverdrup *et al.*, 2002e
Eisenia veneta (Earthworm)	Growth	EC50	50 (CI: 45 - 56)	Fluorene in acetone carrier	28 d	6.2	1.6	As above.	As above.	Sverdrup *et al.*, 2002e
Eisenia veneta (Earthworm)	Growth	EC10	31 (CI: 28 - 36)	Fluorene in acetone carrier	28 d	6.2	1.6	As above.	As above.	Sverdrup *et al.*, 2002e
Enchytraeus crypticus (Earthworm)	Mortality	LC50	1,600 (CI: 1200 - 2500)	Fluorene in acetone carrier	21 d	6.2	1.6	As above.	As above.	Sverdrup *et al.*, 2002d
Enchytraeus crypticus (Earthworm)	Reproduction	EC50	55 (CI: 46 - 59)	Fluorene in acetone carrier	21 d	6.2	1.6	As above.	As above.	Sverdrup *et al.*, 2002d
Enchytraeus crypticus (Earthworm)	Reproduction	EC10	25 (CI: 0 - 39)	Fluorene in acetone carrier	21 d	6.2	1.6	As above.	As above.	Sverdrup *et al.*, 2002d
Enchytraeus crypticus (Earthworm)	Reproduction	NOEC	27	Fluorene	21 d	6.2	1.6	As above.	As above.	Sverdrup *et al.*, 2002d
Folsomia fimetaria (Springtail)	Mortality	LC50	39 (95% CI = 36 - 43) (on basis of initial measured conc.)	Fluorene	21 d	6.2 (H_2O method)	1.6	"Askov" soil - sieved through 2 mm. 38.4% coarse sand; 23.6% fine sand; 23.6% coarse silt;10.0% fine silt; 13.0% clay. Density = 1.135 g/cm^3; CEC = 8.14 meq/100 g. 4 repl/conc. X 6 concentrations (0, 10, 30, 100, 300, 1000 $mg \cdot kg^{-1}$ dw)@ 50% water holding capacity.Closed containers.		Sverdrup *et al.*, 2001
Folsomia fimetaria (Springtail)	Reproduction	EC10	7.7 (95% CI = 5.2 - 10)(on basis of initial measured conc.)	Fluorene	21 d	6.2 (H_2O method)	1.6	As above.	As above.	Sverdrup *et al.*, 2001
Folsomia fimetaria (Springtail)	Reproduction	NOEC	14	Fluorene	21 d	6.2	1.6	As above.	As above.	Sverdrup *et al.*, 2001

Organism	Effect	Endpoint	Effective Concentration ($mg \cdot kg^{-1}$)	Compound(s)	Exposure Period	Soil pH	Org C (%)	Soil Conditions	Spike or extraction methods	Reference
Folsomia fimetaria (Springtail)	Mortality	LC50	>910 (on basis of initial measured conc.)	Indeno[1,2,3-c,d]pyrene	21 d	6.2 (H_2O method)	1.6	"Askov" soil - sieved through 2 mm. 38.4% coarse sand; 23.6% fine sand; 23.6% coarse silt;10.0% fine silt; 13.0% clay. Density = 1.135 g/cm^3; CEC = 8.14 meq/100 g. 4 repl/conc. X 6 concentrations (0, 10, 30, 100, 300, 1000 $mg \cdot kg^{-1}$ dw)@ 50% water holding capacity.Closed containers.	Nominal, in acetone carrier. The measured conc. for 1,000 $mg \cdot kg^{-1}$ substrates at day 0 (initial).	Sverdrup *et al.*, 2002a
Folsomia fimetaria (Springtail)	Reproduction	EC10	>910 (on basis of initial measured conc.)	Indeno[1,2,3-c,d]pyrene	21 d	6.2 (H_2O method)	1.6	As above.	As above.	Sverdrup *et al.*, 2002a
Folsomia fimetaria (Springtail)	Mortality	LC50	167 (95%CI: 129 - 196)(on basis of initial measured conc.)	Naphthalene	21 d	6.2 (H_2O method)	1.6	As above.	Nominal, in acetone carrier. Then measured conc. for 10, 100 and 1,000 $mg \cdot kg^{-1}$ substrates at days 0 and 21.	Sverdrup *et al.*, 2002a
Folsomia fimetaria (Springtail)	Reproduction	EC10	20 (95%CI: 0 - 39)(on basis of initial measured conc.)	Naphthalene	21 d	6.2 (H_2O method)	1.6	As above.	As above.	Sverdrup *et al.*, 2002a
Folsomia fimetaria (Springtail)	Mortality	LC50	>560 (on basis of initial measured conc.)	Perylene	21 d	6.2 (H_2O method)	1.6	As above.	Nominal, in acetone carrier. The measured conc. for 1,000 $mg \cdot kg^{-1}$ substrates at day 0 (initial).	Sverdrup *et al.*, 2002a
Folsomia fimetaria (Springtail)	Reproduction	EC10	>560 (on basis of initial measured conc.)	Perylene	21 d	6.2 (H_2O method)	1.6	As above.	As above.	Sverdrup *et al.*, 2002a
Eisenia veneta (Earthworm)	Mortality	LC50	134	Phenanthrene in acetone carrier	28 d	6.2	1.6	As above.	Nominal, in acetone carrier. Then measured conc. for 10, 100 and 1,000 $mg \cdot kg^{-1}$ substrates at days 0 and 21.	Sverdrup *et al.*, 2002e
Eisenia veneta (Earthworm)	Growth	EC50	94 (CI: 64 - 125)	Phenanthrene in acetone carrier	28 d	6.2	1.6	As above.	As above.	Sverdrup *et al.*, 2002e
Eisenia veneta (Earthworm)	Growth	EC10	25 (CI: 9.0-41)	Phenanthrene in acetone carrier	28 d	6.2	1.6	As above.	As above.	Sverdrup *et al.*, 2002e
Enchytraeus crypticus (Earthworm)	Mortality	LC50	>2,500	Phenanthrene in acetone carrier	21 d	6.2	1.6	As above.	As above.	Sverdrup *et al.*, 2002d
Enchytraeus crypticus (Earthworm)	Reproduction	EC50	87 (CI: 61 - 100)	Phenanthrene in acetone carrier	21 d	6.2	1.6	As above.	As above.	Sverdrup *et al.*, 2002d

Organism	Effect	Endpoint	Effective Concentration ($mg \cdot kg^{-1}$)	Compound(s)	Exposure Period	Soil pH	Org C (%)	Soil Conditions	Spike or extraction methods	Reference
Enchytraeus crypticus (Earthworm)	Reproduction	EC10	40 (CI: 0 - 78)	Phenanthrene in acetone carrier	21 d	6.2	1.6	As above.	As above.	Sverdrup *et al.*, 2002d
Enchytraeus crypticus (Earthworm)	Reproduction	NOEC	34	Phenanthreme	21 d	6.2	1.6	As above.	As above.	Sverdrup *et al.*, 2002d
Folsomia candida (Springtail)	Mortality	LOEC	380	Phenanthrene in 2-propanone as a carrier	33-34 d	6	NR	Artificial Soil, 70% sand, 20% clay, 10% peat, 6-7 replicates, polluted soils	Nominal (in 2-Propanone as a carrier)	Crouau, 1999
Folsomia candida (Springtail)	Mortality	NOEC	220	Phenanthrene in 2-propanone as a carrier	33-34 d	6.0	NR	As above.	As above.	Crouau *et al.*, 1999
Folsomia candida (Springtail)	Reproduction	EC50	175 (CI 148-192)	Phenanthrene in 2-propanone as a carrier	33-34 d	6.0	NR	As above.	As above.	Crouau *et al.*, 1999
Folsomia candida (Springtail)	Reproduction	LOEC	220	Phenanthrene in 2-propanone as a carrier	33-34 d	6	NR	As above.	As above.	Crouau *et al.*, 1999
Folsomia candida (Springtail)	Reproduction	NOEC	140	Phenanthrene in 2-propanone as a carrier	33-34 d	6.0	NR	As above.	As above.	Crouau *et al.*, 1999
Folsomia fimetaria (Springtail)	Mortality	LC50	41 (95% CI = 38 - 45)(on basis of initial measured conc.)	Phenanthrene	21 d	6.2 (H_2O method)	1.6	"Askov" soil - sieved through 2 mm. 38.4% coarse sand; 23.6% fine sand; 23.6% coarse silt;10.0% fine silt; 13.0% clay. Density = 1.135 g/cm^3; CEC = 8.14 meq/100 g. 4 repl/conc. X 6 concentrations (0, 10, 30, 100, 300, 1000 $mg \cdot kg^{-1}$ dw)@ 50% water holding capacity.Closed containers.		Sverdrup *et al.*, 2001, 2002b
Folsomia fimetaria (Springtail)	Reproduction	EC10	23 (95% CI = 9.1 - 38)(on basis of initial measured conc.)	Phenanthrene	21 d	6.2 (H_2O method)	1.6	As above.	As above.	Sverdrup *et al.*, 2001, 2002b
Folsomia fimetaria (Springtail)	Reproduction	NOEC	21	Phenanthrene	21 d	6.2	1.6	"Askov" soil - sieved through 2 mm. 38.4% coarse sand; 23.6% fine sand; 23.6% coarse silt;10.0% fine silt; 13.0% clay. Density = 1.135 g/cm^3; CEC = 8.14 meq/100 g. 4 repl/conc. X 6 concentrations (0, 10, 30, 100, 300, 1000 $mg \cdot kg^{-1}$ dw)@ 50% water holding capacity.Closed containers.	Nominal, in acetone carrier. Then measured conc. for 10, 100 and 1,000 $mg \cdot kg^{-1}$ substrates at days 0 and 21.	Sverdrup *et al.*, 2001
Eisenia veneta (Earthworm)	Mortality	LC50	155 (CI: 120-218)	Pyrene in acetone carrier	28 d	6.2	1.6	As above.	As above.	Sverdrup *et al.*, 2002e
Eisenia veneta (Earthworm)	Growth	EC50	71 (CI: 59 - 83)	Pyrene in acetone carrier	28 d	6.2	1.6	As above.	As above.	Sverdrup *et al.*, 2002e
Eisenia veneta	Growth	EC10	38 (CI: 28 - 46)	Pyrene in acetone	28 d	6.2	1.6	As above.	As above.	Sverdrup *et al.*,

Organism	Effect	Endpoint	Effective Concentration ($mg \cdot kg^{-1}$)	Compound(s)	Exposure Period	Soil pH	Org C (%)	Soil Conditions	Spike or extraction methods	Reference
(Earthworm)				carrier						2002e
Enchytraeus crypticus (Earthworm)	Mortality	LC50	>2,300	Pyrene in acetone carrier	21 d	6.2	1.6	As above.	As above.	Sverdrup *et al.*, 2002d
Enchytraeus crypticus (Earthworm)	Reproduction	EC50	42 (CI: 25 - 56)	Pyrene in acetone carrier	21 d	6.2	1.6	As above.	As above.	Sverdrup *et al.*, 2002d
Enchytraeus crypticus (Earthworm)	Reproduction	EC10	11 (CI: 2.3 - 27)	Pyrene in acetone carrier	21 d	6.2	1.6	As above.	As above.	Sverdrup *et al.*, 2002d
Enchytraeus crypticus (Earthworm)	Reproduction	NOEC	18	Pyrene	21 d	6.2	1.6	As above.	As above.	Sverdrup *et al.*, 2002d
Folsomia fimetaria (Springtail)	Mortality	LC50	53 (95% CI = 47 - 58)(on basis of initial measured conc.)	Pyrene	21 d	6.2 (H_2O method)	1.6	As above.		Sverdrup *et al.*, 2001, 2002b
Folsomia fimetaria (Springtail)	Reproduction	EC50	24	Pyrene	21 d	6.2 (H_2O method)	1.6	As above.		Jensen and Sverdrup, 2001
Folsomia fimetaria (Springtail)	Reproduction	NOEC	15	Pyrene	21 d	6.2 (H_2O method)	1.6	As above.		Jensen and Sverdrup, 2001
Folsomia fimetaria (Springtail)	Reproduction	EC10	10 (95% CI = 7.3 - 13)(on basis of initial measured conc.)	Pyrene	21 d	6.2 (H_2O method)	1.6	As above.		Sverdrup *et al.*, 2001, 2002b
Folsomia fimetaria (Springtail)	Reproduction	NOEC	15	Pyrene	21 d	6.2 (H_2O method)	1.6	As above.		Jensen and Sverdrup, 2001

Appendix IV: Collated Plant PAH Toxicity Data

Organism	Effect	Endpoint	Effective Concentration ($mg \cdot kg^{-1}$)	Compound(s)	Exposure Period	Soil pH	Org C(%)	Soil Conditions	Spike or extraction methods	Reference
Triticum aestivum (Bread wheat)	Growth (above-ground biomass)	LOEC ($EC_{9.1}$)	1.0	Anthracene	8 d	7.8	3.3	Natural Soil (28.7% sand, 57.6% silt, 13.7% clay), dosed 1 time, direct application exposure route, 5 replicates, Concentration/Dose 1 $mg \cdot kg^{-1}$).	nominal No sig difference from controls at 16 d; therefore this endpoint was not used.	Cervelli *et al.*, 1995
Avena sativa (Common oat)	Growth (above-ground biomass)	EC50	30 (C.I. = 20 - 45)	Anthracene (in ethanol/ 2-propanone as a carrier)	32 d	5.5	2	Natural Soil (sandy loam, silt/clay fraction 12.4%), dosed 1 time, direct application exposure route, exposure concentrations: 0, 10, 100 and 1,000 $mg \cdot kg^{-1}$, four replicates of each treatment.	nominal;(in ethanol or 2-propanone as a carrier)	Mitchell *et al.*, 1988
Avena sativa (Common oat)	Seedling Emergence	LC50	525 (C.I. = 385 - 665)	Anthracene (in ethanol/ 2-propanone as a carrier)	32 d	5.5	2	As above.	As above.	Mitchell *et al.*, 1988
Banksia ericifolia (Heath banksia)	Growth (above-ground biomass)	EC50	>1,000	Anthracene (in ethanol/ 2-propanone as a carrier)	32 d	5.5	2	As above.	As above.	Mitchell *et al.*, 1988
Banksia ericifolia (Heath banksia)	Seedling Emergence	LC50	>1,000	Anthracene (in ethanol/ 2-propanone as a carrier)	32 d	5.5	2	As above.	As above.	Mitchell *et al.*, 1988
Casuarina distyla (She-oak)	Growth (above-ground biomass)	EC50	>1,000	Anthracene (in ethanol/ 2-propanone as a carrier)	56 d	5.5	2	As above.	As above.	Mitchell *et al.*, 1988
Casuarina distyla (She-oak)	Seedling Emergence	LC50	>1,000	Anthracene (in ethanol/ 2-propanone as a carrier)	56 d	5.5	2	As above.	As above.	Mitchell *et al.*, 1988
Cucumis sativus (Cucumber)	Growth (above-ground biomass)	EC50	720 (C.I. =225 - 1655)	Anthracene (in ethanol/ 2-propanone as a carrier)	32 d	5.5	2	As above.	As above.	Mitchell *et al.*, 1988
Cucumis sativus (Cucumber)	Seedling Emergence	LC50	>1,000	Anthracene (in ethanol/ 2-propanone as a carrier)	32 d	5.5	2	As above.	As above.	Mitchell *et al.*, 1988
Eucalyptus eximia (Yellow Bloodwood)	Growth (above-ground biomass)	EC50	>1,000	Anthracene (in ethanol/ 2-propanone as a carrier)	32 d	5.5	2	As above.	As above.	Mitchell *et al.*, 1988
Eucalyptus eximia (Yellow Bloodwood)	Seedling Emergence	LC50	>1,000	Anthracene (in ethanol/ 2-propanone as a carrier)	32 d	5.5	2	As above.	As above.	Mitchell *et al.*, 1988
Glycine Max (Soybean)	Growth (above-ground biomass)	EC50	>1,000	Anthracene (in ethanol/ 2-propanone as a carrier)	32 d	5.5	2	As above.	As above.	Mitchell *et al.*, 1988
Glycine Max (Soybean)	Seedling Emergence	LC50	>1,000	Anthracene (in ethanol/ 2-propanone as a carrier)	32 d	5.5	2	As above.	As above.	Mitchell *et al.*, 1988

Organism	Effect	Endpoint	Effective Concentration ($mg \cdot kg^{-1}$)	Compound(s)	Exposure Period	Soil pH	Org C(%)	Soil Conditions	Spike or extraction methods	Reference
Lolium perenne (Perennial ryegrass)	Growth (above-ground biomass)	LOEC ($EC_{40.8}$)	226	Anthracene in trichloromethane as a carrier	40d	6.6	1.5	Natural Soil (22% sand, 52% silt, 26% clay), dosed 1 time per study period, direct application exposure route, 5 replicates, Concentration/Dose 0.1 $g \cdot kg^{-1}$.	nominal, in trichloromethane as a carrier	Leyval and Binet,1998
Lupinus albus (Lupin)	Emergence	LOEC	>155	Benz(a)anthracene	30 d	5.9	NR	Agricultural soil: 57% sand, 32% silt, 11%clay. C 1.1%. N 0.11%. C/N 10, P 0.094%.	nominal	Henner *et al.*, 1999
Lupinus albus (Lupin)	Growth (above-ground biomass)	LOEC	>155	Benz(a)anthracene	30 d	5.9	NR	As above.	As above.	Henner *et al.*, 1999
Cannabis sativa (Hemp)	Emergence	EC50	89	Benzo(a)pyrene	45 d	NR	NR	Leileihau soil (Wahiawa ser.): silty clay (clayey, kaolinitic, isothermic, tropeptic, eutrustox)	nominal	Campbell *et al*, 2002 (data for 25, 50, 75 $mg \cdot kg^{-1}$ exposures used for linear regression)
Lupinus albus (Lupin)	Emergence	LOEC	>155	Benzo(a)pyrene	30 d	5.9	NR	Agricultural soil: 57% sand, 32% silt, 11%clay. C 1.1%. N 0.11%. C/N 10, P 0.094%.	nominal	Henner *et al.*, 1999
Lupinus albus (Lupin)	Growth (above-ground biomass)	LOEC	>155	Benzo(a)pyrene	30 d	5.9	NR	As above.	As above.	Henner *et al.*, 1999
Lupinus albus (Lupin)	Emergence	LOEC	>155	Dibenz(a,h)anthracene	30 d	5.9	NR	As above.	As above.	Henner *et al.*, 1999
Lupinus albus (Lupin)	Growth (above-ground biomass)	LOEC	>155	Dibenz(a,h)anthracene	30 d	5.9	NR	As above.	As above.	Henner *et al.*, 1999
Avena sativa (Common oat)	Growth (above-ground biomass)	EC50	>1,000	Fluoranthene	14 d	0	NR	Unknown Soil (particle size not defined)	NR	Kordel *et al.*, 1984
Brassica rapa (Bird rape)	Growth (above-ground biomass)	EC50	>1,000	Fluoranthene	14 d	0	NR	As above.	NR	Kordel *et al.*, 1984
Avena sativa (Common oat)	Growth (above-ground biomass)	EC50	>1,000	Perylene	14 d	0	NR	As above.	NR	Kordel *et al.*, 1984
Brassica rapa (Bird rape)	Growth (above-ground biomass)	EC50	>1,000	Perylene	14 d	0	NR	As above.	NR	Kordel *et al.*, 1984

Appendix V: Collated Amphibian and Reptile PAH Toxicity Data

Organism	Latin Name/Life Stage	Effect	Endpoint	Effective Conc.	Compound(s)	Exposure Period	Exposure Route	Methods	Reference
Bullfrog	*Rana catesbeiana* tadpoles	teratogenesis	LOEC	11 ug/L	Fluoranthene	96 h	water-borne	0, 10.97, 37.97, 59.48 $\mu g \cdot L^{-1}$ with simulated solar UV radiation (20/group)	Walker *et al.*, 1998
Southern Leopard Frog	*Rana utricularia* tadpoles	mortality	LC100	5% (with 17 $\mu W \cdot cm^{-2}$; no mortality without photoactivation)	Weathered Petroleum (from and abandoned mine in California)	NR	water-borne	Tadpoles were exposed to UV-A and UV-B (365 and 313 nm light; 14 h/day and 5 h/day respectively). Tadpoles exposed to 0% (0 $mg \cdot L^{-1}$), 5.0% (1.52 $mg \cdot L^{-1}$), and 10% (2.83 mg/l) solutions of the water-soluble fraction of the petroleum at 0.002, 2, and 17 $\mu W \cdot cm^{-2}$.	Little *et al.*, 2000

Appendix VI: Collated Avian PAH Toxicity Data

Organism	Latin Name/Life Stage	Effect	Endpoint	Effective Concentration	Compound(s)	Exposure Period	Exposure Route	Methods	Reference
Mallard duck	*Anas platyrhynchos*	reprod. and development (embryo dev.)	LOEC	0.2 $mg \cdot kg^{-1}$ egg	Benzo[k]-fluoranthene	24 d	injection	control, 0.2, 2.0 mg benzo[k]fluoranthene/kg egg; injected into egg yolks (20/dose)	Brunström *et al.*, 1990
Mallard duck	*Anas platyrhynchos*	systemic tox. (liver wt., blood flow)	LOEC	180 $mg \cdot kg^{-1}$ diet	Naphthalene	NR	oral		Eisler, 1987
Northerm Bobwhite	*Colinus virginianus*	mortality-subchronic	LD50	2,690 $mg \cdot kg^{-1}$ org. C	Naphthalene	14 d	oral via capsule		Office of Pesticide Programs, 2000
Northerm Bobwhite	*Colinus virginianus*	mortality-subchronic	LC50	> 5,620 ppm	Naphthalene	8 d	oral via capsule		Office of Pesticide Programs, 2000
American kestrels	*Falco sparverius*	growth	LOEC	0.3% of BW	Crude oil	28 d	diet	0, 0.3, 3.0% diet (16 juveniles/group)	Pattee and Franson. 1982
Mallard duck	*Anas platyrhynchos*	length of reproductive cycle; delaued gonad maturation	LOEC	3 mL oil/100 g dw diet	Crude oil	96 d	diet	control, 3 ml South Louisiana crude oil/100 g diet, dry weight (17 pairs/dose)	Cavanaugh *et al.*, 1983
Mallard duck	*Anas platyrhynchos* juveniles	growth rate	LOEC	5% by weight of diet	Crude oil	8 weeks	diet	0, 0.025, 0.25, 2.5, 5% south Louisiana crude oil (50/dose)	Szaro *et al.*, 1978
Mallard duck	*Anas platyrhynchos*	immunocompetence	LOEC	4.0 $ml \cdot kg^{-1}$	Crude oil	28 d	gavage	control, 2.5, 4.0 ml/kg South Louisiana crude oil, daily (10-32 ducks/dose)	Rocke, *et al.*,1984
Herring gull	*Larus argentatus* chicks	growth (weight-gain)	LOEC	0.2 mL	Crude oil	8-9 d	oral	single oral dose of 0.2-ml **Kuwait crude** (**KC**) **oil** containing 22% aromatics or **South Louisiana crude** (**SLC**) **oil** containing 17% aromatics, and maintained on 100% seawater	Miller *et al.*, 1978
Herring gull	*Larus argentatus* nestlings	hematopoetic pathologies	LOEC	10 ml **oil**/kg/day	Crude oil	NR	oral (intubation)	daily oral doses of 0, 1, 4, 5, 10 or 20 ml **Prudhoe Bay crude oil**/kg body weight/day by intubation	Leighton, 1985

Appendix VII: Collated Mammalian PAH Toxicity Data (Non-carcinogenic endpoints)

Organism	Latin Name/ Life Stage	Effect	Endpoint	Effective Concentration	PAH	Exposure Period	Exposure Route	Notes	Reference
Mouse		**Hepatotoxicity**	**NOAEL**	**175 mg·kg^{-1} bw**	**Acenaphthene**	**90 d**	**Oral**		**USEPA 1989a**
Mouse		Hepatotoxicity	LOAEL	350 mg·kg^{-1} bw	Acenaphthene	90 d	Oral		USEPA 1989a
Mouse		Immunocompetence	TDLo	180 mg·kg^{-1}	Acenaphthene	12 d		Immunological including allergic - uncharacterized	RTECS (1999: Toxicologist, 48:13)
Rat		Nephrotoxicity	LOAEL	2,000 mg·kg^{-1}	Acenaphthene	32 d	oral		Knobloch *et al*., 1969
Rat		Mortality	LD50	600 mg·kg^{-1}	Acenaphthene	NR	intraperitoneal		Reshetyuk *et al*., 1970.
Mouse		**Immunocomp.**	**TDLo**	**180 mg·kg^{-1}**	**Acenaphthylene**	**12 d**			**RTECS (1999: Toxicologist. 48:13)**
Mouse		Sublethal	NOAEL	1,000 mg·kg^{-1} bw	Anthracene	90 d	oral		USEPA, 1989c
Mouse		Mortality	LD50	430 mg·kg^{-1}	Anthracene	NR	intraperit.		Salamone, 1981
Rat		Mortality	LD50	8,100 mg·kg^{-1}	Anthracene	NR	oral		Mellon Institute, 1977
Rat		Mortality	LD50	>4,000 mg·kg^{-1}	Anthracene	24 h	dermal		Mellon Institute, 1977
Mouse		**Immunocompet.**	**NOAEL**	**10 mg·kg^{-1}**	**Benzo(b)-fluoranthene**	**immunized 12 h after single dose**	**oral**	**Little or no effect**	**Silkworth *et al*., 1995**
Mouse		Immunocompet.	LOAEL	100 mg·kg^{-1}	Benzo(b)-fluoranthene	immunized 12 h after single dose	oral	>50% suppression	Silkworth *et al*., 1995
Mouse		**Immunocompet.**	**NOAEL**	**10 mg·kg^{-1}**	**Benzo(k)-fluoranthene**	**immunized 12 h after single dose**	**oral**	**Little or no effect**	**Silkworth *et al*., 1995**
Mouse		Immunocompet.	LOAEL	100 mg·kg^{-1}	Benzo(k)-fluoranthene	immunized 12 h after single dose	oral	>50% suppression	Silkworth *et al*., 1995
Mouse		Immunocomp.	NOEC	10 mg·kg^{-1}	Benz[a]anthracene	NR	oral	Little or no effect	Silkworth *et al*., 1995
Mouse		Immunocomp.	LOEC	100 mg·kg^{-1}	Benz[a]anthracene	NR	oral	> 50% suppression	Silkworth *et al*., 1995

Organism	Latin Name/ Life Stage	Effect	Endpoint	Effective Concentration	PAH	Exposure Period	Exposure Route	Notes	Reference
Rat		Mortality	LD50	>200 $mg \cdot kg^{-1}$	Benz[a]anthracene	NR	intraven.		Uematsu and Huggins, 1968
Rat		Systemic	NOAEL	150 $mg \cdot kg^{-1}$ bw	Benz[a]anthracene	4 d	oral		Nousiainen *et al*., 1984
Mouse	pregant dams	Reproduction	LOAEL	10 $mg \cdot kg^{-1}$ bw	Benzo[a]pyrene	10 d (days 7 to 16 of gestation)	oral	Reduced fertility in F1 generation	Mackenzie and Angevine, 1981
Mouse		Reproduction	EC_{97}	40 $mg \cdot kg^{-1}$	Benzo[a]pyrene	10 d (days 7 to 16 of gestation)	oral	Sterility in F1 generation	Mackenzie and Angevine, 1981
Mouse	female	Reproduction	TDLo	100 $mg \cdot kg^{-1}$	Benzo[a]pyrene	9 d.	oral	female 7-16 days after conception	Mackenzie and Angevine, 1981
Mouse		Immunocomp.		10 $mg \cdot kg^{-1}$	Benzo[a]pyrene	single dose	oral	immunized 12 h after single dose	Silkworth *et al*., 1995
Mouse		Immunocomp.		100 $mg \cdot kg^{-1}$	Benzo[a]pyrene	single dose	oral	immunized 12 h after single dose (> 50% suprression)	Silkworth *et al*., 1995
Mouse	pregnant CD1 mice	Reproduction		10 $mg \cdot kg^{-1}$ $bw \cdot d^{-1}$	Benzo[a]pyrene	NR	oral	Females: reduction in gonadal weight but no effect on body weight of male or female offsping. Reduction in fertility and reproductive capacity	WHO, 1983
Mouse	pregnant CD1 mice	Reproduction		40 $mg \cdot kg^{-1}$ $bw \cdot d^{-1}$	Benzo[a]pyrene	NR	oral	Females: almost complete sterility of offspring of both sexes	WHO, 1983
Mouse	pregnant	Reproduction		100-150 $mg \cdot kg^{-1}$ $bw \cdot d^{-1}$	Benzo[a]pyrene	admin over 4 generations	dermal	sensitization of offspring to the effects of B[a]P, so that an increase in rate of appearance of papillomas and carcinomas was observed	WHO, 1983
Mouse	female	Reproduction	TDLo	200 $mg \cdot kg^{-1}$	Benzo[a]pyrene	NR	intraperiton.	female, 7 days after conception	Shum *et al.*, 1979
Mouse	female	Reproduction	TDLo	250 $mg \cdot kg^{-1}$	Benzo[a]pyrene	4 d.	intraperiton.	female, 13-17 days after conception	Holladay and Smith, 1994

Organism	Latin Name/ Life Stage	Effect	Endpoint	Effective Concentration	PAH	Exposure Period	Exposure Route	Notes	Reference
Mouse	(Ah(d)/Ah(d) type), nonresponsive mice	Immunocomp.		500 mg·kg^{-1}	Benzo[a]pyrene	NR	intraperiton.		WHO, 1983
Mouse	(Ah(b)/Ah(b) type), responsive mice	Immunocomp.		500 mg·kg^{-1}	Benzo[a]pyrene	NR	intraperiton.	survival time significantly shorter than nonresponsive mice	WHO, 1983
Mouse	C3H/Anf mice; mid-late pregnancy	Immunocomp.		100-150 mg·kg^{-1} bw·d^{-1}	Benzo[a]pyrene	NR	intraperiton.		WHO, 1983
Mouse		Mortality	LD50	250 mg·kg^{-1}	Benzo[a]pyrene	NR	intraperiton.		WHO, 1973
Mouse		Mortality	LDLo	500 mg·kg^{-1}	Benzo[a]pyrene	NR	intraperiton.		Epstein *et al.*, 1972
Mouse	female	Reproduction	TDLo	160 mg·kg^{-1}	Benzo[a]pyrene	NR	subcutan.	female 12 days after conception (live birth index affected)	Sokya, 1980
Mouse		Reproduction	TDLo	12,000 mg·kg^{-1}	Benzo[a]pyrene	NR	subcutan.	multigenerations	Turusov *et al.*, 1990
Rat	**female**	**Reproduction**	**TDLo**	**2,000 mg·kg^{-1}**	**Benzo[a]pyrene**	**49 d.**	**oral**	**female - 28 days premating, 1-22 days after conception**	**Rigdon and Rennels, 1964**
Rat	female	Reproduction	TDLo	150 mg·kg^{-1}	Benzo[a]pyrene	2 d.	subcutan.	6-8 days after conception	Bui *et al.*, 1986
Rat	female	Reproduction	TDLo	2.1 mg·kg^{-1}	Benzo[a]pyrene	4 d.	intramuscular	15-19 days after conception	Csaba *et al*., 1993
Rat	pregnant	Reproduction		5 mg·d^{-1}	Benzo[a]pyrene	NR	subcutan.	Females: Death of all fetuses.	WHO, 1983
Rat		Nephrotoxicity	TDLo	2,250 mg·kg^{-1}	Benzo[a]pyrene	5 wk.	oral		Vandebriel *et al.*, 1998
Rat		Mortality- Acute	LD50	50 mg·kg^{-1} bw	Benzo[a]pyrene	NR	subcutan.	NR	Eisler, 1987
Mouse		**Immunocompetence**	**TDLo**	**180 mg·kg^{-1}**	**Chrysene**	**12 d.**	**NR**		**RTECS (1999: Toxicologist 48:13)**
Mouse		Immunocompetence	NOAEL	10 mg·kg^{-1}	Chrysene	immunized 12 h after single dose	oral		Silkworth *et al*., 1995
Mouse		Immunocompetence	LOAEL	100 mg·kg^{-1}	Chrysene	immunized 12 h after single dose	oral		Silkworth *et al*., 1995

Organism	Latin Name/ Life Stage	Effect	Endpoint	Effective Concentration	PAH	Exposure Period	Exposure Route	Notes	Reference
Mouse		Mortality	LD50	>320 $mg \cdot kg^{-1}$	Chrysene	NR	intraperit.		WHO, 1983
Deer mice	**juvenile males**	**Immuno-suppression**	**ED50**	**0.048 $mg \cdot kg^{-1} \cdot d^{-1}$**	**Dibenz[a,h]-anthracene**	**11 d**	**intraperitoneal**	**NR**	**Dickerson *et al*, 1994.**
Mouse		Immunocompetence	NOAEL	100 $mg \cdot kg^{-1}$	Fluoranthene	immunized 12 h after single dose	oral		Silkworth *et al*., 1995
Rat		Mortality -Acute	LD50	2,000 $mg \cdot kg^{-1}$ bw	Fluoranthene	NR	oral	NR	Eisler, 1987
Rat		Mortality	LD50	2,000 $mg \cdot kg^{-1}$	Fluoranthene		oral		Smyth *et al*., 1962
Rat		Growth		30 mg	Fluoranthene	single dose, in sesame oil	intraperiton.		Haddow *et al*., 1937
Rabbit		Mortality	LD50	3,180 $mg \cdot kg^{-1}$	Fluoranthene		dermal		Smyth *et al.*, 1962
Rat		Nephrotoxicity, liver weight	LOAEL	250 $mg \cdot kg^{-1}$	Fluoranthene	13 wk	oral (gavage)		USEPA, 1988
Rat		Hematological	NOAEL	150 $mg \cdot kg^{-1} \cdot d^{-1}$	Fluoranthene	90 d	oral (diet)		Knuckes *et al*., 2003
Mouse		Hematological	LOAEL	250 $mg \cdot kg^{-1}$ bw	Fluorene	13 wk	Oral (gavage)		USEPA, 1989b
Mouse		Mortality	LD50	>2,000 $mg \cdot kg^{-1}$	Fluorene	NR	intraperit.		Vatulina *et al*., 1985
Mouse		**Immunocompetence**	**NOAEL**	**100 $mg \cdot kg^{-1}$**	**Fluorene**	**immunized 12 h after single dose**	**oral**		**Silkworth *et al*., 1995**
Cat		Mortality	LDLo	1,000 $mg \cdot kg^{-1}$	Naphthalene		oral		HBAMAK, 1935
Dog		Mortality	LDLo	400 $mg \cdot kg^{-1}$	Naphthalene		oral		HBAMAK, 1935
Mouse		Weight gain	TDLo	3,700 $mg \cdot kg^{-1}$	Naphthalene	14 d.	oral	Continuous	Shopp *et al.*, 1984
Mouse		Reprod.	TDLo	2,400 $mg \cdot kg^{-1}$	Naphthalene	8 d.	oral	7-14 day(s) after conception	Plasterer *et al.*, 1985
Mouse		Immunocompetence	NOAEL	100 $mg \cdot kg^{-1}$	Naphthalene	immunized 12 h after single dose	oral		Silkworth *et al*., 1995
Mouse	males	Mortality	LD50	533 $mg \cdot kg^{-1}$	Naphthalene	5 d	oral		Shopp *et al.*, 1984
Mouse		**Reprod.**		**300 $mg \cdot kg^{-1}$**	**Naphthalene**	**56 d.**	**gavage**	**Females: began on day 7 of gestation**	**USEPA, 1990**

Organism	Latin Name/ Life Stage	Effect	Endpoint	Effective Concentration	PAH	Exposure Period	Exposure Route	Notes	Reference
Mouse		Mortality	LD50	710 $mg \cdot kg^{-1}$	Naphthalene		gavage		USEPA, 1990
Rabbit		**Reprod.**	**NOAEL**	**16 $mg \cdot kg^{-1}$**	**Naphthalene**	**3 times - days 20, 22, 24 of gestation**	**gavage**	**pregnant females: days 20,22 and 24 of gestation**	**Shepard, 1986**
Rabbit, New Zealand White	Adult	Reproduction	EC50	630 $mg \cdot kg^{-1}$	Naphthalene	6 - 18 d	oral gavage	5 groups of 4 rabbits dosed 0, 50, 250, 630, and 10000 $mg \cdot kg^{-1}$ per day by gavage gestation days (GD) 6-18	Naismith and Matthews, 1985
Rabbit		Mortality	LDLo	3,000 $mg \cdot kg^{-1}$	Naphthalene		oral		HBAMAK, 1935
Rabbit		Mortality	LD50	>20,000 $mg \cdot kg^{-1}$	Naphthalene		dermal		RTECS (1National Technical Information Service, AD691-490)
Rabbit		Mortality	LD50	>2,000 $mg \cdot kg^{-1}$	Naphthalene		dermal		Papciak and Mallory, 1990
Rabbit, New Zealand White	Adult	Maternal weight gain	LOEC	630 $mg \cdot kg^{-1}$	Naphthalene	6 - 18 d	oral gavage	5 groups of 4 rabbits dosed 0, 50, 250, 630, and 10000 $mg \cdot kg^{-1}$ per day by gavage gestation days (GD) 6-18	Naismith and Matthews, 1985
Rabbit, New Zealand White	Adult	Mortality-Subchronic	LOEC	630 $mg \cdot kg^{-1}$	Naphthalene	6 - 18 d	oral gavage	5 groups of 4 rabbits dosed 0, 50, 250, 630, and 10000 $mg \cdot kg^{-1}$ per day by gavage gestation days (GD) 6-18	Naismith and Matthews, 1985
Rabbit, New Zealand White	Adult	Mortality-Subchronic	LD100	1,000 $mg \cdot kg^{-1}$	Naphthalene	6 - 18 d	oral gavage	5 groups of 4 rabbits dosed 0, 50, 250, 630, and 10000 $mg \cdot kg^{-1}$ per day by gavage gestation days (GD) 6-18	Naismith and Matthews, 1985
Rabbit, New Zealand White	Offspring	Teratogenicity	LOEC	400 $mg \cdot kg^{-1}$	Naphthalene	6 - 18 d	oral gavage	4 groups of 18 rabbits, dosed 0, 40, 200, and 400 $mg \cdot kg^{-1}$/day by gavage gestation days (GD) 6-18.	Naismith and Matthews, 1986

Organism	Latin Name/ Life Stage	Effect	Endpoint	Effective Concentration	PAH	Exposure Period	Exposure Route	Notes	Reference
Rat		Reprod.	TDLo	5,900 mg·kg^{-1}	Naphthalene	15 d.	intraperit.	females: 1-15 day(s) after conception	Harris *et al.*, 1979
Rat		Mortality	LD50	490 mg·kg^{-1}	Naphthalene		oral		Izmerov *et al.*, 1982
Rat		Reprod.	TDLo	4,500 mg·kg^{-1}	Naphthalene	10 d.	oral	females: 6-15 day(s) after conception	NTP, 1991
Rat		Pulmonary/Rena.	NOAEL	1,600 mg·kg^{-1}	Naphthalene		intraperit.		O'Brien *et al.*, 1985
Rat		Reprod.		395 mg·kg^{-1}	Naphthalene		intraperit.		USEPA, 1990
Rat		Mortality	LD50	2,600 mg·kg^{-1}	Naphthalene		oral		Papciak and Mallory, 1990
Rat		Mortality	LD50	2,200 mg·kg^{-1}	Naphthalene		oral		USEPA, 1990
Rat		Mortality	LD50	2,400 mg·kg^{-1}	Naphthalene		oral		USEPA, 1990
Rat		Mortality	LD50	>2,500 mg·kg^{-1}	Naphthalene		dermal		Gaines, 1969
Rats		Mortality - Acute	LD50	1,780 mg·kg^{-1} bw	Naphthalene	NR	oral	NR	Eisler, 1987
Rats		**Mortality-chronic**	**NOAEL**	**41 mg·kg^{-1} bw**	**Naphthalene**	**23 mo.**	**oral**		**Schmahl, 1955**
Rats, Albino, Sherman	Female Adult	Mortality -Acute	LD50	9,430 mg·kg^{-1} (95% CI: 7,000-12,700 mg·kg^{-1})	Naphthalene	single dose	oral gavage	Not reported	Union Carbide, 1949
Rats, Sprague Dawley	Adult	Mortality -Acute	LD50	2649 mg·kg^{-1} (95% CI: 2079-3376 mg·kg^{-1})	Naphthalene	single dose	oral (in corn oil)	5 groups of 10 rats (5 m: 5 f) administered single doses of naphthalene in corn oil by oral gavage at levels of 1000, 1600, 2500, 3200 and 4000 mg·kg^{-1} of body weight.	Pharmakon, 1985
Rats, Sprague Dawley	Adult	Mortality-Subchronic	LD50	>1000 mg·kg^{-1} body weight	Naphthalene	28 d	dermal	(5 m: 5 f) receiving one occluded, 6 hour application of naphthalene 5 days/week for 4 weeks at dose levels of 0, 50, 125, 250, 500, and 1000 mg·kg^{-1} body weight. Mortality	Pharmakon, 1985

Organism	Latin Name/ Life Stage	Effect	Endpoint	Effective Concentration	PAH	Exposure Period	Exposure Route	Notes	Reference
								was not observed.	
Rat, Fischer 344	6-wk old	Reduced body weight	LOAEL	200 $mg \cdot kg^{-1}$ (143 $mg \cdot kg^{-1} \cdot d^{-1}$ time-adjusted)	Naphthalene	13 wks: administered 5 days/week	Oral (gavage)		BCL, 1980
Mouse		Mortality	LD50	700 $mg \cdot kg^{-1}$	Phenanthrene	NR	oral		RTECS (1964:Hygiene and Sanitation 29(6):19)
Mouse		Mortality	LD50	700 $mg \cdot kg^{-1}$	Phenanthrene	NR	intraperit.		Simmon *et al*., 1979
Mouse		Mortality	LD50	514 $mg \cdot kg^{-1}$	Pyrene		intraperiton.		Salamone, 1981
Mouse		**Immunocompetence**	**TDLo**	**180 $mg \cdot kg^{-1}$**	**Pyrene**	**12 d.-intermittent**	**NR**		**RTECS (1999: Toxicologist 48:13)**
Mouse		Immunocompetence	NOAEL	100 $mg \cdot kg^{-1}$	Pyrene	immunized 12 h after single dose	oral		Silkworth *et al*., 1995
Mouse		Dermal lesions	NOAEL	0.30%	Pyrene	680 d: applied in benzene twice weekly	dermal		WHO, 1983
Mouse		Dermal lesions	NOAEL	5%	Pyrene	1 yr: applied three times weekly	dermal		WHO, 1983
Mouse		Nephrotoxicity	NOAEL	75 $mg \cdot kg^{-1}$	Pyrene	90 d	gavage		USEPA, 1989d
Mouse		Nephrotoxicity	LOAEL	125 $mg \cdot kg^{-1}$	Pyrene	90 d	gavage		USEPA, 1989d