Derivation of Petroleum Hydrocarbon Wildlands Criteria for British Columbia

by

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Abstract

Environmental criteria for petroleum hydrocarbon contaminants do not currently exist for areas of British Columbia that are designated as wildlands. This study presents a process by which to derive criteria for a wildlands setting. A methodology is presented to derive criteria for petroleum hydrocarbon compounds with the derivation process being illustrated by deriving wildlands criteria for Northern British Columbia using the proposed methodology. The goal of study is to create a process that is focussed on being scientifically defensible, flexible and transparent process based on human and ecological toxicological effects endpoints that serve to protect human and ecological populations. The study presents toxicity reference values that can be used for the purposes of criteria development or risk assessment purposes to assess wildlands receptors as well as resulting soil, sediment, surface water and tissue residue (fish and wild game) criteria that can be used as a management tool.

Keywords: Hydrocarbons; Wildlands; Criteria; British Columbia; Criteria Derivation; Contaminated Sites

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Glossary

CCME	Canadian Council of Ministers of the Environment
CSR	Contaminated Sites Regulation
CSST	Contaminated Sites Soil Task Force
CYP	Cytochrome P450
EPH	Extractable Petroleum Hydrocarbons
HEPH	Heavy Extractable Petroleum Hydrocarbons
LEPH	Light Extractable Petroleum Hydrocarbons
LOAEL	Lowest Observable Adverse Effects Level
LOEC	Lowest Adverse Effects Concentration
MAH	Monocyclic Aromatic Hydrocarbon
NOAEL	No Observable Adverse Effects Level
NOEC	No Observable Adverse Effects Concentration
PAH	Polycyclic Aromatic Hydrocarbon
RfC	Reference Concentration
RfD	Reference Dose
TPHCWG	Total Petroleum Hydrocarbon Working Group
TRV	Toxicity Reference Value
USEPA	United Stated Environmental Protection Agency

1: Introduction

Wildlands account for the majority of the land base in British Columbia (British Columbia Ministry of Agriculture and Lands, 2007); however, currently environmental criteria do not exist for this land use. Wildlands, often consisting of crown lands, land owned by the government, may be allocated to industry for the purposes of resource extraction; such resource extraction can include the extraction of petroleum. After resource extraction is complete, the lands are returned to the government. Numerical human and ecological health effects-based criteria need to be established to ensure that the lands returned to the Crown do not contain concentrations of contaminants that are detrimental to the environment. Contaminants should be at concentrations that ensure that there is a low potential to adversely effect human health and to ensure that plant and animal communities can be sustained during and post resource extraction.

The lack of wildlands criteria leaves a gap in the management practices of contaminated sites. The British Columbia Ministry of Environment, Environmental Protection Division regulates contaminated sites in British Columbia under the Environmental Management Act's Contaminated Sites Regulations (CSR). The CSR categorizes areas by land uses, these categories consist of agricultural, urban park, residential, commercial and industrial; currently there is no land criteria specific to wildlands.

Soil standards in the CSR are based on the work of the Contaminated Sites Soil Task Group (CSST), which in 1995 developed criteria for soil contaminants found on contaminated sites (BC Ministry of Environment, 1996). The soil criteria developed by the CSST, and currently used in the CSR for management of contaminated sites, were not intended to be used for managing wildlands areas and other lands including forest, rangelands, wetlands and tundra areas (MacDonald Environmental Sciences Ltd., 2007). Due to the lack of criteria for wildlands, current policy is to apply urban parkland criteria to the top three meters of soil and commercial land use criteria applied to depths below three meters (BC Ministry of Environment, 1996).

The CSR contains environmental standards for each of these land use categories for soil and vapour. Standards for the protection of aquatic life are encapsulated by surface water standards and sediment criteria are based on the type of water body, freshwater or marine, and groundwater standards are based on based on the type of receiving water body. Sediment criteria are further designated into typical and sensitive habitat categories.

More recently, the Canadian Council of Ministers of the Environment (CCME) has developed criteria for petroleum hydrocarbons in soil (Canadian Council of Ministers of the Environment, 2008). The criterion is considered applicable to federal jurisdictions and provides a scientific basis for criteria development. Criteria for wildlands, however, have not been developed as a part of the criteria. The intent of this study is to develop a methodology to derive criteria that are specific to wildlands and present values resulting from the application of the methodology.

The methodology for the derivation of wildlands guidelines for petroleum hydrocarbons was developed to provide a progressive tool that incorporates current scientific knowledge of environmental fate and transport with scientific rationale to protect ecosystems. The approach uses a fugacity model to predict petroleum hydrocarbon concentrations in abiotic and biotic environmental media. A risk-based approach was then used to determine abiotic concentrations that are protective of ecological and human health. This was achieved by first determining acceptable exposure concentrations of chemicals to organisms and second using relationships between media to determine the environmental concentrations associated with those concentrations. The intent of this study is to illustrate the execution of a proposed conceptual framework for criteria development and to provide regulators, industry professionals and risk assessors with tools and rationales to manage wildlands.

1.1 Goals and Objectives

The objective of the project is to (i) develop a methodology for deriving wildland criteria for petroleum hydrocarbon criteria, (ii) apply the methodology to derive criteria and compare these developed criteria to available guidelines. The goal of methodology development is to create a process that is scientifically defensible, flexible and transparent process based on human and ecological toxicological effects endpoints that

serve to protect human and ecological populations. The guiding goal of this derivation methodology is to protect ecosystem health and viability and human health using the best available scientific knowledge. Protection goals for criteria are to be derived with the intent to protect ecological organisms on a community basis to a 95% protection level; humans are to be protected on the individual basis to a no adverse health effects level. The intent of this methodology is to integrate biota and soil, sediment, water and vapour concentration to provide a holistic set of management criteria.

2: Background

2.1 Physical Properties of Petroleum Hydrocarbons

Hydrocarbons consist of compounds that contain carbon and hydrogen atoms. Petroleum hydrocarbons are a group of compounds that are organic components of crude oil and other geologically sourced substances, such as coal and bitumen (Canadian Council of Ministers of the Environment, 2008). The fate and transport of petroleum hydrocarbons influence exposure. Some of the properties that influence petroleum hydrocarbon fate and transport are chemical structure, soil-water sorptive capacity, partitioning coefficients (organic carbon and octanol water), Henry's law and diffusion in air and water (Potter & Simmons, 1998). Because it would be unfeasible to include each hydrocarbon in this study, the derivation methodology was applied to hydrocarbon constituents found in crude oil and petroleum distillate products. This information was based on the findings of a literature search preformed by the Technical Advisory Committee for the Total Petroleum Hydrocarbon Criteria Working Group and is presented in Potter & Simmons, 1998.

2.1.1 Overview of Physical Properties

The structure and the physical properties of petroleum hydrocarbons influence how they are transported into the environment and how they will interact with organisms. Below is a summary of two physical properties of petroleum hydrocarbon compounds that were used to assess the environmental distribution and toxicity of petroleum hydrocarbons.

2.1.2 Chemical Structure

Compounds were classified according to chemical structure as a means to evaluate toxicity potential with the assumption that similar toxicity responses will result from chemicals with similar structures. This method is based on that previously used by the TPHCWG serves as the basis for grouping of hydrocarbons; this method is incorporated into the CCME Canada-Wide Standards for Petroleum Hydrocarbons in

Soil for the protection of human health. Furthermore, the Science Advisory Board For Contaminated Sites in British Columbia EPH/LEPH/HEPH Task Force recommended that the current hydrocarbon fractions used in petroleum hydrocarbon standards in the CSR (C10-19 and C19-32) be modified to be in agreement with the CCME Canada-Wide Standards for Petroleum Hydrocarbon fractions (Science Advisory Board For Contaminated Sites in British Columbia, 2004).

Petroleum hydrocarbons can be classified into one of two broad categories, aromatics (or aryls), molecules containing a benzene ring with alternating single and double bonds, and aliphatic, molecules that do not contain a benzene ring. The characteristics of each of these chemical structures result in different means of imposing toxic injury. Toxicological properties for each of these chemical groups are further presented in Section 2.

2.1.3 Equivalent Carbon

Equivalent carbon number groups (or fractions) were used to classify petroleum hydrocarbon components. Equivalent carbon values are determined by analysing the boiling point obtained in a boiling point gas chromatograph, the analytical procedure used by laboratories to determine carbon number, and normalizing it to the boiling point of n-alkanes obtained using the same method (Gustafson, Griffith, & Orem, 1997). The stability of the structure is taken into account using the equivalent carbon approach with substances with higher retention times in a boiling gas chromatograph having higher equivalent carbon numbers than their number of carbons. For example, hexane (an alkane) and benzene (an aromatic) each have six carbons, hexane has a boiling point of 69 C while benzene has a boiling point of 80°C, the resulting value (normalized to the hexane) results in hexane having an equivalent carbon value of six and benzene having an equivalent carbon of six point five (Gustafson, Griffith, & Orem, 1997). The method was chosen for the purposes of consistency with industry practice and for the accountment of structural stability into grouping.

2.1.4 Summary of Physical Properties for Hydrocarbons

The chemicals evaluated in this work are presented in Table 1 below.

Table 1. Composition of a Typical Crude Oil Hydrocarbon Mixture

Substance	Percent composition	Structure	Equivalent carbon	Carbon atoms
CRUDE OIL				
Straight Chain Alkanes				
n-Hexane	1.8	Aliphatic	6	6
n-Heptane	2.3	Aliphatic	7	7
n-Octane	1.9	Aliphatic	8	8
n-Nonane	1.9	Aliphatic	9	9
n-Decane	1.8	Aliphatic	10	10
n-Undecane	1.7	Aliphatic	11	11
n-Dodecane	1.7	Aliphatic	12	12
Branched Chain Alkanes				
2,2-Dimethylbutane	0.04	Aliphatic	5.37	6
2,3-Dimethylbutane	0.14	Aliphatic	5.68	6
2-Methylpentane	0.4	Aliphatic	5.72	6
3-Methylpentane	0.4	Aliphatic	5.85	6
3-Ethylpentane	0.05	Aliphatic	-	7
2,4-Dimethylpentane	0.05	Aliphatic	6.31	7
2,3-Dimethylpentane	0.6	Aliphatic	6.69	7
2,2,4-Trimethylpentane	0.004	Aliphatic	6.89	8
2,3,3-Trimethylpentane	0.006	Aliphatic	7.58	8
2,3,4-Trimethylpentane	0.005	Aliphatic	7.55	8
2-Methyl-3-ethylpentane	0.04	Aliphatic	7.66	8
2-Methylhexane	0.7	Aliphatic	6.68	7
3-Methylhexane	0.5	Aliphatic	6.76	7
2,2-Dimethylhexane	0.1	Aliphatic	7.25	8

Substance	Percent composition	Structure	Equivalent carbon	Carbon atoms
2,3-Dimethylhexane	0.16	Aliphatic	7.65	8
2,4-Dimethylhexane	0.06	Aliphatic	7.38	8
2,5-Dimethylhexane	0.06	Aliphatic	7.36	8
3,3-Dimethylhexane	0.03	Aliphatic	7.45	8
2,3-Dimethylheptane	0.05	Aliphatic	8.64	9
2,6-Dimethylheptane	0.25	Aliphatic	8.47	9
2-Methyloctane	0.4	Aliphatic	-	9
3-Methyloctane	0.4	Aliphatic	8.78	9
4-Methyloctane	0.1	Aliphatic	8.71	9
Cycloalkanes				
Cyclopentane	0.05	Aliphatic	5.66	5
Methylcyclopentane	0.9	Aliphatic	6.27	6
1,1-Dimethylcyclopentane	0.2	Aliphatic	6.72	7
1-trans-2-Dimethylcyclopentane	0.5	Aliphatic	6.87	7
1-cis-3-Dimethylcyclopentane	0.2	Aliphatic	6.82	7
1-trans-3-Dimethylcyclopentane	0.9	Aliphatic	6.85	7
1,1,2-Trimethylcyclopentane	0.06	Aliphatic	7.67	8
1,1,3-Trimethylcyclopentane	0.3	Aliphatic	7.25	8
1-trans-2-cis-3-Trimethylcyclopentane	0.4	Aliphatic	7.51	8
1-trans-2-cis-4-Trimethylcyclopentane	0.2	Aliphatic	-	8
1-trans-2-Dimethylcyclohexane	0.3	Aliphatic	7.94	8
Ethylcyclohexane	0.2	Aliphatic	8.38	8
Cyclohexane	0.7	Aliphatic	6.59	6
1-trans-2-trans-4-Trimethylcyclohexane	0.2	Aliphatic	-	8

Substance	Percent composition	Structure	Equivalent carbon	Carbon atoms
Alkyl Benzenes				
Benzene	0.4	Aromatic	6.5	6
Toluene	2.5	Aromatic	7.58	7
Ethylbenzene	0.31	Aromatic	8.5	8
o-Xylene	0.68	Aromatic	8.51	8
m-Xylene	2	Aromatic	8.6	8
p-Xylene	0.68	Aromatic	8.61	8
1-Methyl-4-ethylbenzene	0.13	Aromatic	9.57	9
1-Methyl-2-ethylbenzene	0.09	Aromatic	9.71	9
1-Methyl-3-ethylbenzene	0.4	Aromatic	9.55	9
1,2,3-Trimethylbenzene	0.1	Aromatic	10.06	9
1,2,4-Trimethylbenzene	0.69	Aromatic	9.84	9
1,3,5-Trimethylbenzene	0.18	Aromatic	9.62	9
1,2,3,4-Tetramethylbenzene	0.2	Aromatic	11.57	10
Aryl-Benzene				
Biphenyl	0.04	Aromatic	14.26	12
Naphtheno-Benzenes				
Indane	0.07	Aromatic	10.27	9
Tetralin (tetrahydronaphthalene)	0.03	Aromatic	11.7	10
5-Methyltetrahydronaphthalene	0.08	Aromatic	-	11
6-Methyltetrahydronaphthalene	0.09	Aromatic	-	11
Fluorene	0.06	Aromatic	16.55	13
Alkyl Naphthalenes				
Naphthalene	0.09	Aromatic	11.69	10

Substance	Percent composition	Structure	Equivalent carbon	Carbon atoms
Polynuclear Aromatics				
Phenanthrene	0.05	Aromatic	19.36	14

- Value not represented in Parkinson, 1995

(Parkinson, 1995)

2.2 Toxicological Properties of Petroleum Hydrocarbons

2.2.1 Overview

This section describes the processes by which petroleum hydrocarbons affect biological organisms at the biochemical and cellular levels. The current body of toxicological information for hydrocarbons is relatively limited to polyaromatic hydrocarbons in comparison to other hydrocarbon compounds, therefore, inferences about toxicity and toxicological effects are made. Hydrocarbon compounds are typically found in mixtures in the environment. The approach used to assess toxicity was based on a structure-activity relationship approach. The information pertinent to the rationale of grouping hydrocarbons as a means to evaluate potential toxicity is provided. Generalizations based on chemical structure are needed as toxicological information, including information on biochemical mode of toxic action, of specific components of petroleum hydrocarbons is limited to select chemicals.

Hydrophobic molecules, such as petroleum hydrocarbon components, enter cell membranes by processes of passive transport, such as simple diffusion by traversing membranes through aqueous pores (Klaassen, 1996). The rate of transport across a cell membrane is correlated to the octanol-water partitioning coefficient of the molecule with molecules with high octanol-water partitioning coefficients being able to penetrate cell membranes more readily than molecules with low octanol water partitioning coefficients.

2.2.2 Aromatics

Aromatics are a group of hydrocarbons that contain a carbon ring of alternating single and double bonds. The presence of the ring increases the stability of the

compound as electrons freely cycle the atomic ring. Aromatic compounds can be further classified into two groups, alkyl benzenes (a single benzene ring with the presence of an alkyl functional group) and polycyclic aromatic hydrocarbons (compounds containing fused benzene rings). The toxicity of each of these groups is discussed below.

2.2.2.1 Polycyclic Aromatic Hydrocarbons

Toxicological effects of exposure to polycyclic aromatic hydrocarbons in mammals, when exposed to doses of 0.03 to 0.3 mg/kg/day, include decreased body weight, nephrotoxicity and hepatotoxicity (Edwards, et al., 1997). Developmental toxicity (such as embryo deformaties and edema) in fish have been reported for chronic exposure to polycyclic aromatic hydrocarbons (Barron, 2003). A review of polycyclic aromatic hydrocarbon toxicity data is provided in Appendix A and B.

2.2.2.1.1 CYP1A1

The cytochrome P450 CYP1A1 enzyme (also known as aryl hydrocarbon hydroxylase) is induced by polycyclic aromatic hydrocarbons. For example, CYP1A1 catalyses the reaction in which benzo(a)pyrene, is oxidized [looses an electron] forming BP-7,8-epoxide (Parkinson, 1995). Epoxide hydrolase then hydrolyses BP-7,8-epoxide to form BP-7,8-dihydrodiol, the oxidation of this compound is then catalyzed by CYP1A1 to form the carcinogenic BP-7,8-dihydrodiol-9,10-epoxide (Parkinson, 1995). Note detoxification of benzo(a)pyrene oxides is performed by epoxide hydrolase as well (Parkinson, 1995).

The CYP1A1 enzyme is found in all mammalian species and is found in non-hepatic human tissues and in the non-hepatic tissues of most mammals (Parkinson, 1995).

2.2.2.1.2 Carcinogenesis of PAHs

The carcinogenicity of PAHs results from the covalent bonding of [reactive metabolites] (such as BP-7,8-dihydrodiol-9,10-epoxide) to nucleophillic DNA centres results in the initiation of tumourogenesis (Snyder & Andrews, 1995; Hu, Herzog, Zimniak, & Singh, 1999). There is conflicting information regarding whether genotoxicity of mixtures containing PAHs are additive or sub-additive (White, 2002); therefore, it is recommended that mixture toxicity for PAH containing substances be handled on a case

by case basis (White, 2002). As only one PAH, phenanthrene, is included in this study, and as it typically only comprises 0.003 to 0.05 % of crude oil mixtures by weight (Gustafson, Griffith, & Orem, 1997) carcinogenicity will not be assessed.

2.2.2.2 Benzene and Alkylbenzenes

Toxicological effects of benzene and alkylbenzene exposure in mammals include neurotoxicological effects (such as hyperactivity and convulsions) as well as hepatotoxicity, nephrotoxicity and hemotological changes (Edwards, et al., 1997). Developmental effects (such as reduced egg hachability and depressed growth) have resulted from chronic exposure to aquatic species (DeGraeve, et al., 1982). A review of toxicity data for benzene and alkyl benzenes is provided in Appendix A and B.

2.2.2.2.1 Inhibition of CYP2E1

Based on kinetic studies, of the two cytochrome P450 enzymes (CYP2E1 and CYP2B1) involved in benzene metabolism, CYP2E1 is considered to be predominant oxidase in benzene hydroxylation (Snyder & Andrews, 1995). Benzene is oxidized to benzene oxide/oxepin by the CYP enzyme. A non-enzymatic rearrangement of atoms results in the formation of phenol, which is hydroxylated, to either hydroquinone or catechol (which differ in the positioning of the hydroxyl groups). Hydroquinone and catechol are then hyroxylated to form 1,2,4-trihydroxybenzne forming p-benzoquinone and o-benzoquinone, respectively (Snyder & Andrews, 1995). An alternative formation of catechol arises from the hydrolysis of benzene oxide/oxepin by expoxide hydrolase to form dihydrodiol dehydrognase, which is oxidized to form catechol (Snyder & Andrews, 1995; Gregus & Klaassen, 1995).

2.2.2.2.2 Carcinogenesis of Alkylbenzenes

Benzene metabolites, p-benzoquinone and o-benzoquinone, form benzetheno adducts [covalent bonding of carcinogenic compounds with DNA] with deoxycytidine, deoxyadenosine, and deoxyguanosine [deoxyribose (the five carbon sugar backbone of DNA) covalently bonded to the nucleoside base of cytidene, adenosine and guanosine, respectively] (Chenna, et al., 1995). Genotoxicity results if the adduct is not repaired in the DNA, lethality results if replication is blocked by an adduct, mutagenesis results if the adduct leads to coding error (Chenna, et al., 1995). As benzene typically only comprises

0.4 % of crude oil mixtures by weight (Gustafson, Griffith, & Orem, 1997) carcinogenicity will not be assessed.

2.2.3 Aliphatics

Aliphatic hydrocarbons are defined as hydrocarbons that do not contain an aromatic ring. Aliphatics can be either straight chained, branched or cyclic. Substances belonging to this family can be further classified into alkanes (single bonds), alkenes (containing at least one double bond), alkynes (containing at least one triple bond) and alicyclics (containing a ring with any bond type). Toxicological effects of exposure to aliphatics in mammals include neurotoxicity, hepatotoxicity and developmental toxicity (Edwards, et al., 1997). Little information is available regarding toxicological effects involving aquatic organisms. A review of aliphatic toxicity data is provided in Appendix B. The toxicity of n-hexane, a well studied aliphatic in mammals, is discussed below.

2.2.3.1 n-hexane

The n-hexane metabolite, 2,5-hexanedione, is a neurofilament toxin that destroys the cytoskeleton resulting in neurotoxicity (Gregus & Klaassen, 1995). The 2,5-hexanedione compound causes loss of protein function resulting from the formation of dimethylpyrrole adducts due to the cross-linking covalent bonding of 2,5-hexanedione and amino groups (Carden, Lee, & Schlaepfer, 1986); this also results in the enlargement of the neurofilament containing axon which progresses to peripheral axon degeneration (Sayre, Shearson, Wongmongkolrit, Medori, & Gambetti, 1986).

Gamma diketone metabolites are formed by each n-hexane (2,5-hexanedione) and n-heptane (2,5-heptanedione) (Edwards, et al., 1997). While n-hexane exposure has been demonstrated to produce peripheral nervous system damage in animal studies, similar effects have been observed at relatively high concentrations (greater than 1000 mg/kg) of n-heptane (Edwards, et al., 1997).

2.2.4 Baseline Toxicity

Although aliphatic compounds constitute the majority of hydrocarbon mixtures, their toxicity is less studied; therefore, based on the lack of data available on many

aliphatic compounds, the mode of toxic action is unknown. Narcosis is the basic mode of toxic action for any given compound and is further discussed below.

2.2.4.1 Narcosis

Narcosis is referred to as baseline toxicity, or as the minimum toxicity expressed in an organism due to chemical exposure. Narcosis is further classified as polar-narcosis and non-polar narcosis, referring to the polarity of the chemical exhibiting the toxic effect. Petroleum hydrocarbon components are lipiphillic or hydrophobic, and thus exhibit non-polar narcosis effects. Narcosis is an acute manifestation of petroleum hydrocarbon poisoning. The mechanism of narcosis remains unclear, however it can be defined as a disruption of membrane function (Oberg, 2004). Interference with membrane function results in non-specific effects (Mayer & Reichenberg, 2006). Information regarding internal effects concentrations associated with aquatic non-polar narcosis is available (McCarty & MacKay, 1993). It has been demonstrated that the toxicity of hydrocarbons by way of non-polar narcosis follows an additive mode of toxic action (Vaes, Ramos, Verhaar & Hermens, 1998). It should be noted that the baseline toxicity effect exhibited on organisms is not the only effect, with more ecologically relevant effects, such as growth, reproduction and survival, taking precedence in importance. Information pertaining to baseline toxicity was not assessed in this study due in favour of selecting ecologically relevant toxicological effects endpoints, such as those listed above.

3: Theory

The key objective in this study was to develop a methodology to derive wildlands criteria for petroleum hydrocarbons. In order for criteria to be derived, a process by which to determine exposure and a process by which to determine toxicity threshold concentrations for organisms needed to be developed.

The derivation process followed in this study follows the steps in risk assessment theory. The exposure assessment phase involves determining routes and levels of exposure taking into account the interaction of species with the abiotic and biotic environment. To determine exposure, consideration must be given to which organisms to consider and how these organisms are exposed to contaminants. The effects assessment phase includes the determination of tolerable or acceptable contaminant concentrations to which an organism can be exposed without experiencing an effect in excess of that considered to be acceptable. Given the limited toxicological data available for hydrocarbons and the limited test organisms studied in the available data, a methodology to derive acceptable tolerable concentrations needed to be derived. Finally the risk characterization phase combines the results of the exposure and effects assessment to determine risk, in this study, determines the soil, sediment, water and tissue concentrations that result in acceptable risk effect levels.

This section presents the underlying theory that was used to derive criteria for petroleum hydrocarbons for wildlands.

3.1 Criteria Development

3.1.1 Overview of Approach for Derivation of Wildlands Criteria

The methodology for the derivation of petroleum hydrocarbons criteria for wildlands is based on a three step approach: First to derive toxicity reference values that correspond to the established protection levels or select existing values that are considered appropriate, Second to determine exposure of hydrocarbon compounds to organisms in a wildlands setting, and Third to use a fugacity based environmental fate

model to determine relationships between media to derive criteria that correspond to the established protection levels.

In the first step, acceptable concentrations, toxicity reference values, are derived to correspond to the established protection levels. Multiple types of toxicity data are considered in the toxicity reference value derivation methodology; following the process results in the most chemical and organism specific data being used first with inferences and default values being used when data is unavailable.

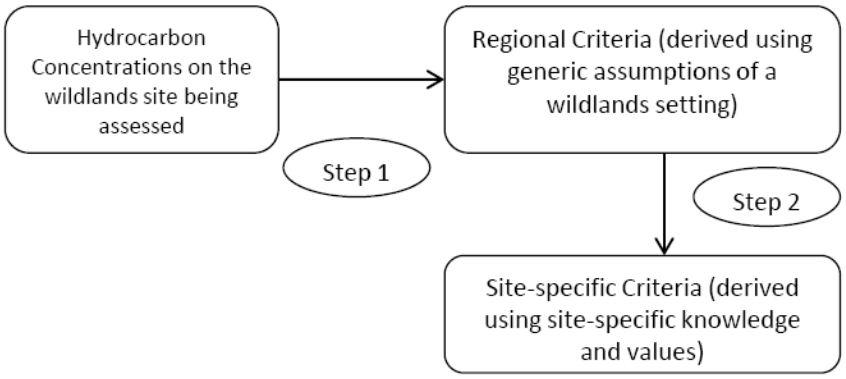
In the second step, the approach to derive criteria includes the use of an environmental fate and transport model to characterize the concentration of petroleum hydrocarbon components in multiple media via their predominant exposure pathway using the principles of fugacity. The concentration of crude oil or concentrations of its components may be entered into the model to determine concentrations in several environmental compartments including soil, sediment and surface water. The concentrations at which biota are exposed to chemicals, the bioavailability of the chemical to the organism, and the biological characteristics of the organism, determines the amount of chemical that the organism will uptake. The fugacity based contaminant fate model determines the concentration of substances at different trophic levels in representative aquatic and terrestrial food webs.

The third step is to determine the acceptable concentrations in the biota to the corresponding concentrations in the abiotic environment to determine acceptable concentrations of hydrocarbon components in soil, sediment, air and water that will be protective of ecological and human health.

Relating concentrations in multiple-media to one another means that knowledge of concentrations in one compartment can be used to predict concentrations in the other compartments. By considering multiple types of toxicological data, the best available scientific knowledge can be applied to protection of ecological and human health. The use of the presented methodology allows for proactive management of resources by determining concentrations in one media to resulting concentration in another.

3.1.2 Multiple Criteria Tiers

Criteria may be set on an area wide basis using broad assumptions or may be set on a site by site basis using site-specific data. Both types of criteria are useful, with generic or regional criteria being helpful to use as a screening tool to determine if contaminant concentration will present unacceptable adverse effects using generic assumptions of what is present in a typical wildlands setting while site-specific criteria are useful to allow for site-specific incorporation of data to allow for a more site-specific risk assessment approach based on what is present on the site. Therefore, this study will present a methodology to derive both region wide and site-specific criteria. Results will be generated for a Northern British Columbia regional setting (parameterization presented in Appendix D).



In Step 1 hydrocarbon concentrations are compared directly to regional criteria, if concentrations meet criteria, these values are considered to be acceptable, if values exceed either the concentrations are remediated to regional criteria or Step 2 is carried out where a site-specific risk assessment is conducted using the methodology used to derive regional criteria

Figure 1. ***Overview of Tiered Criteria Proposed for Assessing Hydrocarbon Concentrations in Wildlands***

3.1.3 Protection Levels

In order for criteria to be developed, a level of protection must be selected. This level of protection is needed to determine the concentrations of contaminants that are considered acceptable. Protection levels are to be incorporated into the toxicity reference values used in the study to which exposure levels are compared. One of the basic principles on which this study was based is that wild land uses need a greater

degree of protection for ecological receptors than that currently offered by the existing land use categories in the management of contaminated sites. Therefore, 95% protection levels for ecological organisms were used as a projection level for criteria development.

3.2 Exposure Analysis

3.2.1 Receptors

To establish protection levels, organisms that represent the wildlands ecosystems needed to be selected. The approach to select receptors was based on the different media that are present in a wildlands setting and to find receptors that reflect the organisms found in that media. The overall theory in receptor selection is that receptors need to reflect species that are likely to be found in the wildlands setting. Each receptor is to be characterized on the basis of feeding behaviour (e.g. what it consumed and in what quantity) and on a biochemical composition basis (e.g. lipid and protein content). By parameterizing each receptor evaluated, it is possible to use different receptors in the model and to adapt this approach to incorporate different species depending on their relevance. The media to be evaluated in the criteria derivation process included soil, sediment and surface water. Therefore, representative species that reflect organisms that are exposed to these media were selected.

3.2.2 Interactions

To determine the quantity of chemical an organism is exposed to, the interactions between the environmental media and within ecological communities must be established. As the intent of the criteria derivation process is to develop criteria on a site-specific and regional basis, the approach was designed to be adaptable on a case-by-case basis. For example, certain media may not be impacted on certain sites or differences may exist between organisms present on different sites. Therefore, to allow for flexibility in determining interactions between media and receptors, a modelling approach was used that parameterized media into compartments (e.g. soil, sediment, surface water and organism tissue).

3.3 Effects Analysis

3.3.1 Toxicity Reference Values

Toxicity reference values are contaminant concentrations that correspond to an expected toxicological effect. With limited toxicological data available for petroleum hydrocarbons (including that specific to the species evaluated in this study), a methodology to derive toxicity reference values needed to be formulated for the purposes of criteria development.

As discussed in Section 2.2, the toxic mode of action of the majority of hydrocarbon compounds that comprise crude oil is unknown. It was assumed that the lipid acts as the target site for the hydrocarbon contaminants to exert their toxicity as the mode of toxic action. Lipid content differences amongst study organisms and those being evaluated in this study become important to consider. Therefore, when possible, lipid content differences will be adjusted for when the test organism used in the reference study differs from that being evaluated.

3.4 Corresponding Media Concentrations

3.4.1 Derivation of Criteria from Exposure and Effects Levels

By establishing tolerable concentrations (toxicity reference values) and knowing receptor exposure levels, contaminant concentrations that correspond to the desired or acceptable effects concentration can be established and used as criteria values. This is achieved by determining the highest concentrations in media that result in exposure concentrations that are either at or below the tolerable concentration for each receptor being considered. This concentration in the media is then selected as the criteria concentration which compounds must not exceed. In order for these concentrations to be protective of the organism that are being evaluated, relationships between the organisms and the media must be understood and parameterized.

4: Methodology

Toxicity reference values were not available for the evaluated compounds for the receptors of interest nor were TRVs available for the desired protection levels. Therefore, a methodology to derive toxicity reference values to meet the needs of this study, including a 95% level of ecological protection, and the use of scientifically sound principles, was developed. This section presents the methodology developed to derive toxicity reference values for use in the wildlands criteria derivation process.

4.1 Endpoints to Assess

Many toxicological endpoints may be assessed during toxicity testing. Such endpoints include those related to population level effects (e.g. growth, reproduction and mortality) and those related to cellular level effects (e.g. enzymatic and genetic alterations). In order to apply a risk-based approach to determine appropriate toxicological reference values, endpoints that are protective of populations of species must be selected. Ecological endpoints were to protect the population level with human endpoints to protect at the individual level.

4.1.1 Ecological Health Endpoints

As ecological protection was considered at a community or population level, endpoints that are considered critical to ecological life function were assessed; these endpoints include survival/mortality, reproduction, and growth. At minimum these endpoints are to be considered for the derivation of ecological toxicity reference values. Other endpoints may be taken into consideration in an effort to offer a higher degree of protection of ecological receptors.

4.1.2 Human Health Endpoints

As human receptors were to be protected to the no adverse effects levels, toxicity reference values derived for human health protection were to consider endpoints for known adverse effects that may be detrimental to health.

4.2 Data Selection

To ensure that the most appropriate data is used to derive criteria the sources of data that have undergone the rigour of scientific review were to be used. These sources may include information from regulatory bodies (e.g. USEPA, CCME), data from peer-reviewed documents (e.g. scientific journals, peer-reviewed reports, databases containing peer-reviewed information). Preference is given to data that considers chemical properties of hydrocarbons presented in Section 1.

The use of uncertainty factors in criteria development was avoided. Therefore, emphasis will be given to incorporation of methods that reduce uncertainty in the application of toxicological information over use of uncertainty factors.

4.2.1 Receptor Specificity and Extrapolation Across Species

Surrogate species for multiple trophic levels in each of the aquatic and terrestrial food chain were selected based on relevance and prevalence to wildlands. Species sensitivity distribution studies were given preference, if available, over the surrogate approach to ensure protection of all ecological receptors. Selecting toxicologically relevant endpoints for each of these receptors presented a challenge, as the chosen receptors are not widely studied for toxicological effects.

Due to the nature of toxicity testing, toxicity data are not available for each species, or trophic level; therefore, inferences from one species to another must be made. In order to extrapolate across species, in an effort to reduce uncertainty, values were to be normalized for organism compositional characteristics (lipid and/or protein content) to account for differences amongst species.

Differences in lipid content amongst species or which toxicity data were taken into account for both aquatic and terrestrial ecological receptors. Hydrocarbons are substances with high octanol-water partitioning coefficients. The uptake of hydrophobic chemicals into tissue is often determined based on lipid content (DeBruyn & Gobas, 2007). It is well recognized that octanol can serve as a surrogate for the lipid partitioning ability of a compound (McGrath & Di Toro, 2009). In the case of aquatic species, the lipid tissues are often the target site for hydrophobic organic chemical uptake from water and diet (Russell, Gobas, & Haffner, 1999); furthermore, evidence supports that

concentrations of compounds in target organisms are similar (McGrath & Di Toro, 2009), therefore the normalization for lipid, the assumed target area, is substantiated. Therefore, lipid content across organisms was taken into account. This was achieved by converting toxicity reference values to a per lipid basis by dividing the toxicity reference value by a literature value for the test species. The lipid normalized toxicity reference value was then multiplied by the lipid content for the study species to represent a toxicity reference value that took lipid content into account (the method used to normalize toxicity reference values is presented in Section 4.5). Reference values were normalized for the lipid content of the test species to the species being evaluated. Assumptions on lipid content and body weight are presented later in Table 6.

Protein content differences amongst species were also taken into account for terrestrial ecological receptors. Sorption of hydrophobic compounds into tissue proteins may account for 1 to 10% of the sorption into lipid content, with a value of 5% being recommended for modelling purposes (DeBruyn & Gobas, 2007). The intent for using normalization in the development of toxicity reference values is to reduce uncertainty in the extrapolation of reference doses across species. The incorporation of protein normalization into the derivation of terrestrial toxicity reference was therefore used in wildlands criteria development. The approach used to normalize toxicity reference values is described in Section 4.5.

As limited toxicological data are available for human subjects, use of established toxicity reference values that have undergone the scrutiny of scientific review were considered for use in criteria derivation. Due to the protection of human populations to the individual no adverse effects level, use of established values were used in this study.

4.2.2 Chemical Specificity and Extrapolation Across Chemicals

Hydrocarbon toxicological endpoint data is often only available for mixtures of chemicals with limited data available for specific compounds. Similarities in chemical structures may be used to predict effects using the assumption that similar physical-chemical properties result in similar modes of toxicity (Escher & Hermens, 2002).

In an effort to increase the specificity of toxicological data, preference should be given to toxicological data that is based on the physical-chemical properties of hydrocarbons to reduce uncertainty in inferences made when extrapolating data across

chemicals. Extrapolation of effects across chemicals was limited to chemicals with similar chemical structures (e.g. aromatics to aromatics, aliphatics to aliphatics).

4.3 Determination of Exposure

Chemical concentrations to which an organism is exposed must be determined. These concentrations can then be compared to chemical concentrations that are considered tolerable to an organism to see if exposure will result in an unacceptable effect. To relate chemical concentrations that do not result in unacceptable effects to concentrations in environmental media (e.g. soil, sediment, water) exposure concentrations must be determined. The principles of an exposure assessment were used to determine the population of interest and the exposure pathways to be considered in order to determine exposure concentrations. This section presents the determination of exposure of petroleum hydrocarbons in a wildlands setting.

4.3.1 Population of Interest

Wildlands comprise a large landmass in the province and as such provide potential ecological habitat for numerous species. In an attempt to ensure that potential ecological receptors were not excluded from consideration in protection efforts, three representative food webs were assessed, two terrestrial and one aquatic. The approach uses surrogate species to be representative receptors for their respective trophic guild. The characteristics of each receptor within each of the food webs may be altered to be reflective of site-specific receptors to ensure that potentially rare and/or sensitive species are considered as needed. Human populations that consume foods used in these areas were also evaluated with intake rates being adaptable to take into consideration site-specific consumption. The use of a surrogate food web approach allows for flexibility in assessment of site-specific species to ensure that relevant communities, populations and individual species are considered in evaluation of effects resulting from chemical exposure. Each of the trophic guilds used in this study are presented in detail below.

4.3.1.1 Aquatic Biota

The aquatic trophic guilds considered in the derivation process were based on the aquatic food web presented in the wildlands environmental fate model (Taylor,

2010). The food web consists of five representative guilds, phytoplankton, zooplankton, benthos, forage fish and piscivorous fish as follows:

1. The phytoplankton food guild represents autotrophic (plant) plankton species (e.g. *Pseudokirchneriella subcapitata*).
2. The zooplankton food guild represents heterotrophic (animal) plankton species (e.g. *Ceriodaphnia dubia, Daphnia magna*).
3. The benthos food guild represents sediment dwelling species (e.g. *Hyalella azteca, Chironomus tentans*).
4. The forage fish food guild represents prey fish that are consumed by higher order predators (e.g. *Pimephales promelas*).
5. The piscivorous food web represents higher order fish that consume prey fish (e.g. *Oncorhynchus mykiss, Oncorhynchus gorbuscha*).

The aquatic food web is illustrated in Figure 2 below.

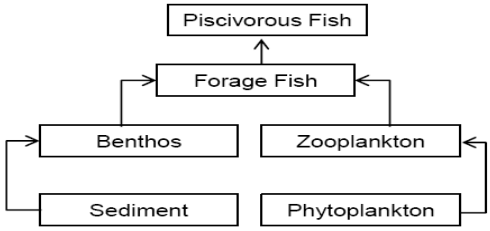


Figure 2. ***Schematic Diagram of Aquatic Food Web based on Taylor, 2010***

4.3.1.2 Terrestrial Biota

The terrestrial trophic guilds considered in the derivation process were based on two terrestrial food web presented in the wildlands environmental fate model (Taylor, 2010). The inclusion of two food webs allows for flexibility in selecting representative species. One terrestrial food web consists of three trophic guilds based on a plant based primary food source with the other being based off of an animal source. The plant based terrestrial food web consists of three representative guilds, lichen, herbivore and carnivore as follows:

1. The lichen food guild represents a food source to herbivorous animals in British Columbia

2. The herbivore food guild represents herbivorous species that may be present within wildlands habitats (surrogate herbivore species: caribou).
3. The carnivore food guild represents carnivorous species that may be present within wildlands habitats that consume herbivores (surrogate carnivore species: wolf).

The plant based terrestrial food web is illustrated in Figure 3 below.

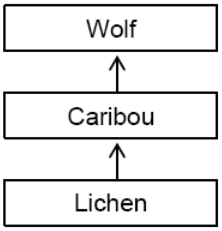


Figure 3. ***Plant Based Terrestrial Food Web based on Taylor, 2010***

The soil based terrestrial food web consists of three representative guilds i.e., worm, small mammal and avian as follows:

1. The worm food guild represents a soil based food source to small mammals that may be present within wildlands habitats.
2. The small mammal food guild represents carnivorous mammals that may be present within wildlands habitats that consume worms (surrogate small mammal species: shrew).
3. The avian food guild represents carnivorous avian species that may be present within wildlands habitats that consume small mammals (surrogate carnivore species: owl).

The soil based terrestrial food web is illustrated in Figure 4 following.

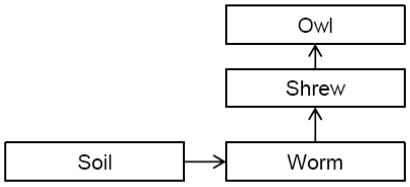


Figure 4. ***Soil Based Terrestrial Food Web based on Taylor, 2010***

4.3.1.3 Human Populations

Human populations were taken into consideration in the derivation of criteria. Although wildlands are non-residential lands, the use of these lands by groups such as first nations, recreational users, campers and so forth were assessed. A surrogate approach was used assessing risks to both toddlers and adults to take into account life stage sensitivities to wildlands users.

4.3.2 Exposure Pathways Considered

All potential routes by which a receptor may be exposed to a chemical considered were the ingestion, dermal contact and inhalation routes. Exposure can include that from direct contact with petroleum compounds in media (via dermal and inhalation exposure routes) and the ingestion of contaminate containing media (including trophic level transfer and ingestion of compounds in soil, sediment, water and biota). Soil criteria were developed with the intent to protect terrestrial organisms, water criteria were developed with the intent to protect aquatic organisms and tissue residue criteria were developed with the intent to protect human populations. The pathways that were considered to be the most relevant were used for the purposes of criteria derivation. This section examines the exposure pathways considered in criteria development.

4.3.2.1 Aquatic Ecological Exposure Pathways

Pathways to protect aquatic ecological receptors were evaluated in the development of water criteria. For aquatic receptors, both ingestion and dermal pathways were taken into consideration. Exposure to contaminants via ingestion of water and biota can be assessed, as BCFs and BAFs are available for these substances. The BCFs and BAFs presented in Taylor, 2010 may be used to assess contaminant uptake through ingestion and dermal exposure to aquatic receptors.

4.3.2.2 Terrestrial Ecological Exposure Pathways

Pathways to protect terrestrial ecological receptors were evaluated in the development of soil criteria. For terrestrial receptors, only the ingestion pathway was considered. Ingestion of contaminants in media includes exposures due to ingestion of

soil, sediment and biota were taken into consideration. Although, soil and sediment may be ingested by terrestrial receptors, such ingestion will typically be incidental. As ingestion is the main route of exposure for terrestrial organisms with both dermal and inhalation exposures being less significant exposure routes. While these receptors may come into contact with chemicals through dermal contact, the coats of many terrestrial organisms limit dermal contact to many substances reducing the potential for exposure.

4.3.2.3 Human Populations Exposure Pathways

Pathways to protect human populations consuming country foods were evaluated in the development of human health criteria. The ingestion pathway was assessed in the determination of potential risks arising from consumption of petroleum hydrocarbon compounds contained within wildlands organisms. The ingestion pathway considered consists of ingestion of contaminated aquatic and terrestrial biota.

4.3.2.4 Determination of Exposure Concentrations

Exposure concentrations may be determined using a combination of sources, including site measurements and observations, environmental fate models, and peer-reviewed sources. The process by which exposure concentrations may be determined is presented below.

4.3.2.5 Site-specific Intake Values

If needed, site-specific observations can be used to perform a risk assessment on the site to determine site-specific criteria. If available, site-specific data, including relevant studies for species occurring on-site and field observations should be used to estimate intake. The values used in this study represent peer-reviewed data for use in the derivation process in the absence of site-specific information.

4.3.2.6 Steady State Fugacity Model

Contaminant concentrations in media were assessed using the results of a fugacity based environmental fate model. The steady-state model (Taylor, 2010) was used to evaluate exposure concentrations in the environment, including water, soil, sediment, surface water and biota. The use of a steady-state model is supported by the need to assess chronic exposure levels in the environment and the time required to

reach equilibrium with organisms. The steady-state scenario is further supported by length of resource extraction occurring over longer time periods with the need to assess environmental concentrations in the environment post-extraction.

4.3.2.7 Terrestrial Ecological Exposure Concentrations

Chemical concentrations in earthworm and lichen tissue were based on BAFs determined in the Taylor (2010) model. Bioaccumulation factors are based on a comparison of the concentration of a chemical in the tissue of an organisms to the concentration of that chemical in media.

$$C_{tissue} = C_{media} \times BAF$$

Where:

C_{tissue} = concentration in organism ($mg \cdot kg^{-1}$)

C_{media}= concentration in media (e.g. soil) ($mg \cdot kg^{-1}$)

BAF = bioaccumulation factor (untiless)

Mammalian tissue transfer factors, which estimate the concentration of chemical transferred from feed to mammalian tissue, were not available for the species and compounds evaluated in the study. However, bioavailability is taken into account using the fugacity approach in the Taylor (2010) model.

4.3.2.7.1 Terrestrial Food Web Structure

The terrestrial ecological food web structure used in the determination of exposure of aquatic receptors to hydrocarbon compounds is presented in Table 2 below:

Table 2. Summary of Terrestrial Ecological Dietary Ingestion and Feeding Preference from Taylor, 2010

Exposure medium	Consumer		
Species	**Lichen**	**Caribou**	**Wolf**
Air	100%	0%	0%
Lichen	0%	100%	0%
Caribou		0%	100%
Wolf			0%
Species	**Worm**	**Shrew**	**Owl**
Soil	100%	0%	0%
Worm	0%	100%	0%
Shrew		0%	100%
Owl			0%

4.3.2.7.2 Daily Dietary Intake (Mammals and Birds)

Daily intake concentrations obtained from Sample, Opresko, & Sutter (1996) were used and compared to concentrations used in toxicological dosing studies for terrestrial organisms. As intake was compared to another intake parameter, the approach is assumed be conservative. Until adequate scientific data are available to assess internal ADME processes in terrestrial organisms, as are available in aquatic organisms, the dietary intake approach was considered to be an appropriate method to use in development of criteria. Intake parameters used in the derivation of criteria are presented in Table 3 following.

Table 3. Body Weight and Dietary Consumption Rates of Receptors used in Criteria Derivation

Guild	Shrew	Owl	Caribou	Wolf
Representative species	Short-tailed shrew (*Blarina brevicauda*)	Barred Owl (*Strix varia*)	White-tailed Deer (*Odocoileus virginianus*)	Red Fox (*Vulpes fulva*)
Body weight (kg)	0.015 kg (Schlesinger and Potter, 1974)	0.72 kg (Dunning, 1984)	57 kg (Smith, 1991)	4.5 kg (Storm et al., 1976)
Food intake (kg/d)	0.009 kg/d (Barrett and Stueck, 1976, Buckner, 1964)	0.084 kg/d (Craighead and Craighead, 1969)	1.74 kg/d (Mautz et al, 1976)	0.45 kg/d (Sargent, 1978, Vogtsberer and Barrett, 1973)

4.3.2.7.3 Direct Exposure (Invertebrates)

Invertebrates are exposed to chemicals in soil via both the ingestion route and the dermal contact route due to the dwelling nature of these organisms. Available toxic effects concentrations for invertebrates are measured in soil and not on a dietary intake or absorption basis. As well, earthworms contain substantial soil content in their guts which would be subsequently ingested by a consumer of earthworms. Therefore, an implicit soil concentration and a BAF of one was used in determining invertebrate contaminant uptake and subsequent earthworm ingestion.

4.3.2.8 Aquatic Ecological Exposure Concentrations

Tissue concentrations in aquatic ecological biota were obtained from the Taylor (2010) model. The bioconcentration factors determined in the Taylor (2010) model that were associated with aquatic tissue concentrations used in this study are presented in Appendix D.

4.3.2.8.1 Aquatic Food Web Structure

The aquatic food web structure used in the determination of exposure of aquatic receptors to hydrocarbon compounds is presented in Table 4 following.

Table 4. Summary of Aquatic Dietary Ingestion Exposure Routes from Taylor, 2010

Species	Phyto-plankton	Zoo-plankton	Benthos	Forage fish A	Forage fish B	Piscivorous fish
Sediment	n/a	n/a	100%	n/a	n/a	n/a
Phyto-plankton	0%	100%	0%	n/a	n/a	n/a
Zooplankton		0%	0%	50%	50%	0%
Benthos			0%	50%	50%	0%
Forage fish A				0%	0%	50%
Forage fish B					0%	50%
Piscivorous fish						0%

4.3.2.9 Human Exposure Concentrations

The consumption of country foods was determined by evaluating both aquatic and terrestrial organisms. The piscivorous fish and the caribou were chosen as the consumption items as both represent higher trophic level organisms that are most likely to be consumed.

4.3.2.9.1 Daily Chemical Uptake

Dietary intake of country foods were determined using human characteristics recommended by Health Canada (2009). Fish consumption rates were based on those for the general Canadian population (Health Canada, 2009); wild game consumption rates were unavailable for the general Canadian population, therefore, ingestion rates for First Nations populations (Health Canada, 2009) were used. The uptake concentrations were used to determine exposures are presented in Table 5 following.

Table 5. Uptake Parameters for the Consumption of Country Foods obtained from Health Canada, 2009

Receptor characteristic	Toddler (7 months to 4 years)	Adult (20 years or older)	Reference
Body weight	16.5 kg	70.7 kg	Health Canada, 2009; Richardson, 1997
Fish ingestion (g/d)	56 g/d	110 g/d	Health Canada, 2009; Richardson, 1997
Wild Game ingestion (g/d)	85 g/d	270 g/d	Health Canada, 2009; Richardson, 1997

The Health Canada (2009) formula for determining intake via ingestion of non-carcinogenic contaminated foods was used to determine a chronic daily intake associated with each compound being evaluated. The formula used is presented below:

$$Dose = (Cfood \times IRfood \times RAForal \times Di) \div (BW \times 365)$$

(Health Canada, 2009)

Where:

Dose = daily intake ($mg \cdot kg^{-1} \cdot d^{-1}$)

C_{food} = concentration of contaminant in food (mg/kg)

IRfood = ingestion rate of food (kg/d)

RAForal = relative absorption factor from GI tract (unitless) – assumed to be one

Di = days per year in which consumption will occur (assumed to be 365 as per Health Canada, 2009 wildlands/recreational use setting)

BW = body weight (kg)

365 = days per year

4.3.2.9.2 Chemical Concentration in Aquatic Tissue

Aquatic tissue concentrations for the piscivorous fish receptor were determined from the Taylor (2010) model. Absorption of chemicals in tissue was assumed to be 100% as a conservative measure in the absence of chemical specific absorption data.

4.3.2.9.3 Chemical Concentration in Terrestrial Tissue

Terrestrial tissue concentrations for the caribou receptor were determined from the Taylor (2010) model. Absorption of chemicals in tissue was assumed to be 100% as a conservative measure in the absence of chemical specific absorption data.

4.4 Derivation of Toxicity Reference Values

Toxicity reference values establish acceptable effects concentrations for chemicals in media. These values are used as the basis for developing criteria as exposure concentrations are compared to these values in order to determine acceptable concentrations of contaminants in environmental media.

Toxicity reference values were developed for the following media, soil, surface water, sediment, and tissue for human health country food consumption. The methodology and resulting values are presented in this section.

The objectives governing TRV development were to first protect to a higher degree than current criteria established for non-wildlands land uses and to second use the best available science (Gobas and Taylor, 2010) in an effort to reduce the reliance of uncertainty factors to compensate for scientific rigour. Ecological TRVs were to establish protection a 95% protection level with human health protection levels set to no observable effects concentrations to protect for non-carcinogenic health effects in consistency with current BC MOE risk policy. As carcinogenic compounds comprised less than one percent of the typical crude oil mixture (Parkinson, 1995), carcinogenicity was not assessed as a part of this study. An attempt was made to use the best available science to minimize uncertainty with the use of laboratory toxicological data; this is consistent with recommended efforts to reduce the uncertainty with the extrapolation of laboratory effects to field effects (Chapman, Fairbrother, & Brown, 2009). Therefore, toxicological studies and methods that have been placed under rigor were used, if available, to develop toxicity reference values.

4.4.1 Soil Toxicity Reference Values

A search for available peer-reviewed sources was conducted to find relevant toxicological effects studies for petroleum hydrocarbons conducted for terrestrial guilds presented prior. It is well established that use of acute studies for the protection of

chronic exposures is not recommended (Allard et al, 2009). Preference was given to chronic and sub-chronic data when evaluating data to be used in the establishment of criteria. This section outlines the approach used to derive these values and the resulting toxicity reference values used in criteria derivation.

4.4.1.1 Derivation of Mammalian Toxicological Reference Values

Toxicity reference values for the protection of mammalian guilds were derived for soil using NOAEL and LOAEL data obtained from sub-chronic and chronic studies in Edwards et al, 1997. Effects doses were normalized for lipid and protein content differences amongst species.

4.4.1.1.1 Toxicological Studies Evaluated

Mammalian effects concentrations were established using toxicological data recommended by the TPHCWG for the intent of protecting for human health. The stringency of use of data established to protect to no observable effects levels and lowest observable effects levels was considered appropriate for the 95% protection level established for wildlands in Section 1.

The Total Petroleum Hydrocarbon Criteria Working Group conducted a comprehensive review of toxicological studies for hydrocarbon compounds and evaluated the findings. The intent of the TPHCWG was to establish soil remedial targets to be protective of human health that were scientifically defensible (Edwards, et al., 1997). Resulting toxicity reference values recommended by the TPHCWG have been incorporated into current CCME PHC soil guidelines established for the protection of human health. The recommended TPHCWG TRVs were used directly for human health protection in this study.

The endpoint concentrations established by the CCME for petroleum hydrocarbon guidelines for the protection of ecological health were not considered appropriate for use for establishing wildlands criteria as the CCME guidelines are based on a species sensitivity distribution ranked percentile approach; toxicological effects data for multiple species and endpoints are first pooled and then ranked with the 20% and 50% percentiles of the pooled data set being used to establish criteria. The ranked approach is not associated with a specific protection endpoint, therefore, as one of the

objectives of wildlands criteria development is to establish a 95% protection level, the CCME approach was not considered appropriate for the purposes of establishing wildlands criteria because it accepts toxicological damage to an ecologically significant number of animal species. Consequently, the endpoint concentrations recommended in TPHCWG were used as the basis of ecological TRVs derived in this study.

4.4.1.1.2 Daily Dietary Intake

Toxicological endpoints assessed by Edwards, et al, 1997 were presented in a daily dose based amount of chemical per body weight per day (mg/kg/day). Given that internal concentrations for exposure cannot be developed at this time and the limited availability of tissue residue concentrations associated with no observable effects levels, daily doses of contaminant uptake (i.e. RfDs) were used to determine toxicological reference values for organisms of terrestrial mammalian guilds.

4.4.1.1.3 Lipid and Protein Normalization

To account for differences across species, toxicological effect endpoint concentrations presented in TPHCWG were normalized for lipid and protein content to account for differences in sorptive capacities across ecological organisms. Lipid and protein normalization are applied based on the assumption that the biological make-up of all organisms includes the same or similar lipids and proteins. By assuming that chemicals, in particular hydrophobic compounds such as petroleum hydrocarbons, act on the same target site across species, normalizing effects concentrations to an organism's lipid and protein content takes differences in quantity of available target sites (lipid and protein molecules) into account. Lipid comprises a significant portion of cell membranes and normalization supports the assumption of non-polar narcosis as a mode of toxic action of hydrocarbons, as described in Section 2. DeByurn and Gobas (2007) have demonstrated sorption of hydrophobic compounds by lipid and proteins, 2007. Lipid and protein contents used in the derivation of toxicity reference values were based those presented in Taylor, 2010 and those obtained from peer-reviewed sources as stated in Table 6. It was assumed that protein sorption was 5% of that of lipid sorption as per DeBruyn and Gobas, 2007 using the following formula to normalize original dose associated with the reference study to the desired receptor in the food web:

$$\frac{TRV_a}{L_a + 0.05P_a} \times L_b + 0.05P_b = TRV_b$$

Where: TRV_a = Toxicity Reference Value in Original Study (mg/kg-d)

TRV_b = Normalized Toxicity Reference Value (mg/kg-d)

L_a = Lipid Content of Test Organism in Original Study (% total weight)

L_b = Lipid Content of Desired Receptor (% total weight)

P_a = Protein Content of Test Organism in Original Study (% total weight)

P_b = Protein Content of Desired Receptor (% total weight)

The lipid and protein contents used in the derivation of toxicity reference values are presented below in Table 6.

Table 6. Lipid and Protein Content of Organisms used in Toxicity Testing and Organisms evaluated in Study

Guild	Rat	Mouse	Shrew	Caribou	Wolf
Lipid Content (%)	22 (Papakon-strantinou, 2003)	26 (Larsson, 1966)	12.6 (Taylor, 2010)	8 (Taylor, 2010)	9 (Taylor, 2010)
Protein Content (%)	24 (Papakons trantinou, 2003)	20 (Larsson, 1966)	12.9 (Taylor, 2010)	22.7 (Taylor, 2010)	20 (Taylor, 2010)

4.4.1.1.4 Resulting Mammalian Toxicity Reference Values

The lipid and protein content above were used in the DeByurn and Gobas (2007) formula to extrapolate the TPHCWG toxicity reference values (which were based on rat and mouse species) to the animals being evaluated in this study (i.e. caribou, wolf and shrew). The resulting mammalian toxicity values are summarized in Table 7.

Table 7. Mammalian Toxicity Reference Values

Substance	Caribou	Wolf	Shrew
	$mg \cdot kg^{-1} \cdot d^{-1}$	$mg \cdot kg^{-1} \cdot d^{-1}$	$mg \cdot kg^{-1} \cdot d^{-1}$
CRUDE OIL			
Straight Chain Alkanes			
n-Hexane	2.07E+02	2.26E+02	3.00E+02
n-Heptane	7.88E+01	8.62E+01	1.14E+02
n-Octane	1.97E+02	2.16E+02	2.85E+02
n-Nonane	1.97E+02	2.16E+02	2.85E+02
n-Decane	1.97E+02	2.16E+02	2.85E+02
n-Undecane	3.94E+01	4.31E+01	5.71E+01
n-Dodecane	3.94E+01	4.31E+01	5.71E+01
Branched Chain Alkanes			
2,2-Dimethylbutane	2.07E+02	2.26E+02	3.00E+02
2,3-Dimethylbutane	2.07E+02	2.26E+02	3.00E+02
2-Methylpentane	2.07E+02	2.26E+02	3.00E+02
3-Methylpentane	2.07E+02	2.26E+02	3.00E+02
3-Ethylpentane	2.07E+02	2.26E+02	3.00E+02
2,4-Dimethylpentane	2.07E+02	2.26E+02	3.00E+02
2,3-Dimethylpentane	2.07E+02	2.26E+02	3.00E+02
2,2,4-Trimethylpentane	2.07E+02	2.26E+02	3.00E+02
2,3,3-Trimethylpentane	1.97E+02	2.16E+02	2.85E+02
2,3,4-Trimethylpentane	1.97E+02	2.16E+02	2.85E+02
2-Methyl-3-ethylpentane	1.97E+02	2.16E+02	2.85E+02
2-Methylhexane	1.97E+02	2.16E+02	2.85E+02
3-Methylhexane	1.97E+02	2.16E+02	2.85E+02
2,2-Dimethylhexane	1.97E+02	2.16E+02	2.85E+02
2,3-Dimethylhexane	1.97E+02	2.16E+02	2.85E+02
2,4-Dimethylhexane	1.97E+02	2.16E+02	2.85E+02
2,5-Dimethylhexane	1.97E+02	2.16E+02	2.85E+02
3,3-Dimethylhexane	1.97E+02	2.16E+02	2.85E+02
2,3-Dimethylheptane	1.97E+02	2.16E+02	2.85E+02
2,6-Dimethylheptane	1.97E+02	2.16E+02	2.85E+02
2-Methyloctane	1.97E+02	2.16E+02	2.85E+02
3-Methyloctane	1.97E+02	2.16E+02	2.85E+02
4-Methyloctane	1.97E+02	2.16E+02	2.85E+02
Cycloalkanes			
Cyclopentane	2.07E+02	2.26E+02	3.00E+02
Methylcyclopentane	2.07E+02	2.26E+02	3.00E+02
1,1-Dimethylcyclopentane	2.07E+02	2.26E+02	3.00E+02
1-trans-2-Dimethylcyclopentane	2.07E+02	2.26E+02	3.00E+02
1-cis-3-Dimethylcyclopentane	2.07E+02	2.26E+02	3.00E+02
1-trans-3-Dimethylcyclopentane	2.07E+02	2.26E+02	3.00E+02
1,1,2-Trimethylcyclopentane	1.97E+02	2.16E+02	2.85E+02
1,1,3-Trimethylcyclopentane	1.97E+02	2.16E+02	2.85E+02
1-trans-2-cis-3-Trimethylcyclopentane	1.97E+02	2.16E+02	2.85E+02
1-trans-2-cis-4-	1.97E+02	2.16E+02	2.85E+02

Substance	Caribou	Wolf	Shrew
Trimethylcyclopentane			
1-trans-2-Dimethylcyclohexane	1.97E+02	2.16E+02	2.85E+02
Ethylcyclohexane	1.97E+02	2.16E+02	2.85E+02
Cyclohexane	2.07E+02	2.26E+02	3.00E+02
1-trans-2-trans-4-Trimethylcyclohexane	2.07E+02	2.26E+02	3.00E+02
Alkyl Benzenes			
Benzene	1.10E-01	1.20E-01	1.59E-01
Toluene	8.78E+01	9.61E+01	1.27E+02
Ethylbenzene	3.82E+01	4.19E+01	5.54E+01
o-Xylene	7.05E+01	7.72E+01	1.02E+02
m-Xylene	7.05E+01	7.72E+01	1.02E+02
p-Xylene	5.57E+01	6.10E+01	8.07E+01
1-Methyl-4-ethylbenzene	4.33E+01	4.74E+01	6.28E+01
1-Methyl-2-ethylbenzene	4.33E+01	4.74E+01	6.28E+01
1-Methyl-3-ethylbenzene	4.33E+01	4.74E+01	6.28E+01
1,2,3-Trimethylbenzene	4.33E+01	4.74E+01	6.28E+01
1,2,4-Trimethylbenzene	4.33E+01	4.74E+01	6.28E+01
1,3,5-Trimethylbenzene	4.33E+01	4.74E+01	6.28E+01
1,2,3,4-Tetramethylbenzene	1.41E+01	1.54E+01	2.04E+01
Aryl-Benzene			
Biphenyl	1.97E+01	2.16E+01	2.85E+01
Naphtheno-Benzenes			
Indane	4.33E+01	4.74E+01	6.28E+01
Tetralin (tetrahydronaphthalene)	1.41E+01	1.54E+01	2.04E+01
5-Methyltetrahydronaphthalene	1.41E+01	1.54E+01	2.04E+01
6-Methyltetrahydronaphthalene	1.41E+01	1.54E+01	2.04E+01
Fluorene	4.23E+01	4.63E+01	6.13E+01
Alkyl Naphthalenes			
Naphthalene	2.80E+01	3.06E+01	4.05E+01
Polynuclear Aromatics			
Phenanthrene	2.54E+01	2.78E+01	3.68E+01

4.4.1.2 Derivation of Invertebrate Toxicological Reference Values

4.4.1.2.1 Toxicological Studies Evaluated

Toxicity reference values for the protection of invertebrates were not derived. Instead, the NOEC data for un-weathered crude oil in a sand clay mixture obtained (Visser, 2003; CCME, 2008) was used directly. An EcoTox search conducted for toxicological studies using invertebrates resulting in limited results, as presented in

Appendix B. CCME 2008 presents NOEC studies conducted for un-weathered and weathered oil on E. foetida (CCME, 2008). The no observable effects concentration is consistent with the 95% protection level established in Section 1. As information for specific hydrocarbon compounds is limited, whole oil data studies were preferred as mixture toxicity is taken into account and reliance on large degrees of extrapolation of effects across chemicals is avoided. The lowest NOEC, that for the un-weathered oil, was used in the derivation of criteria for the protection of invertebrates for the purposes of conservatism. Either value, however, may be considered appropriate for use for the protection of invertebrate populations.

4.4.1.2.2 Estimation of Chemical Uptake

Invertebrate uptake is based on both ingestion of contaminated soil and dermal absorption of chemicals in soil. As the toxicological study (Visser, 2003) was based on a soil dosing study that would take into account both chemical exposure routes, daily intake values were not used in the establishment of protection levels for invertebrates. A BAF of one (Taylor, 2010) was used to estimate chemical uptake from soil, which results in the soil concentration being equivalent to the concentration in the tissue.

4.4.1.2.3 Resulting Invertebrate Toxicity Reference Values

The NOECs established in the reference study by Visser, 2003 were not modified and were used directly to establish protection levels for the soil invertebrate trophic guild. Both the un-weathered oil endpoint and the weathered oil endpoint are presented in Table 8 below. The appropriate value should be used based on sound judgement with the fresh crude oil being used by default as a conservative measure.

Table 8. Invertebrate Toxicity Reference Concentrations

Substance	Worm (mg/kg)	Endpoint	Source
Fresh Crude Oil - Sand/Clay	1.5E+03	NOEC	Visser, 2003
Residual Oil - Sand	7.3E+03	NOEC	Visser, 2003

4.4.1.3 Derivation of Avian Toxicological Reference Values

4.4.1.3.1 Toxicological Studies Evaluated

Insufficient toxicological data were available that evaluated toxicological effects of hydrocarbons to avian species. Results of an EcoTox search resulted in limited studies, as presented in Appendix B.

4.4.1.3.2 Recommendation for Avian Toxicity Reference Values

Toxicity reference values for the protection of avian species could not be established due to the lack of toxicological data available for petroleum hydrocarbons. Because extrapolation of toxicological effects concentrations across animal kingdoms is not advised (Science Advisory Board For Contaminated Sites in British Columbia, 2008); available toxicological data was considered insufficient to establish toxicity reference values for the protection of avian species. Consequently, avian species were not included in the final derivation of criteria.

4.4.2 Water Toxicity Reference Values

A search of available peer-reviewed sources was conducted to find relevant toxicological effects studies for petroleum hydrocarbons conducted for aquatic guilds presented in Section 3. Preference was given to chronic and sub-chronic data when evaluating data to be used in the establishment of criteria. This section outlines the approach used to derive these values and the resulting toxicity reference values used in criteria derivation.

4.4.2.1 Derivation of Aquatic Toxicological Reference Values

Toxicity reference values for the protection of aquatic guilds were derived for surface water using target lipid models that have previously been validated for protection of 95% of species tested for growth, reproduction and survival endpoints (McGrath and DiToro, 2009).

4.4.2.1.1 Toxicological Studies Evaluated

An EcoTox search was conducted for the endpoints of growth, reproduction and survival for aquatic species for petroleum hydrocarbon compounds as presented in

Appendix A. Results were largely limited to 50% lethality concentrations and PAH compounds. Peer-data was also reviewed for internal body burdens associated with no observable adverse effects concentrations, however, such data was not found. The majority of the reviewed internal body burden concentrations were associated with lethality and were considered inappropriate for use in criteria derivation. The approach selected for derivation of aquatic protection values was based on a validated target lipid model following the principle of use of scientifically defensible data for the purposes of this study.

Acceptable aquatic effects concentrations (those associated with a 95% protection level of species) for multiple aquatic species were established using models created by McGrath and DiToro (2009) based on the endpoints of growth, reproduction and survival. Two models were developed, one for mono-aromatic hydrocarbons and the other for poly-aromatic hydrocarbons. The models were developed based on the primary assumption that the critical internal body burden for lipids is consistent across aquatic species and non-polar chemicals with an acute to chronic ratio used to estimate concentrations associated with chronic exposures (McGrath and DiToro, 2009). The internal concentration presented (119 μmol/g octanol; McGrath and DiToro, 2009) itself was not validated across species. The model presented in McGrath and DiToro (2009) using the octanol-water partitioning co-efficient as an input was validated with available toxicological data. Therefore, the validated model for determining water levels to protect to this internal value were preferred based on the objective to use the best available science for criteria development.

The mono-cyclic aromatic hydrocarbon (MAH) model was based on toxicological studies representing 28 aquatic species with the PAH model based on toxicological studies representing 20 aquatic species (McGrath and DiToro, 2009). Acute to chronic ratios were then applied to data to determine chronic concentrations with validation of the models consisting of evaluation of the resulting models to NOECs levels for the species investigated (McGrath and DiToro, 2009). The models used to determine water concentrations associated with 95% protection of multiple aquatic species are presented below.

Model for PAHs:

$$\log(HC_5) = (-0.936)\log(K_{ow}) + \log(52.9) - \log(3.83) - 2.3\sqrt{(0.000225[\log(K_{ow})^2]) + 0.105 + 0.112}$$

(McGrath and DiToro, 2009)

Where: HC_5 = Water concentration associated with protection of 95% of aquatic species (mmol/L)

K_{ow} = Octanol-water partitioning co-efficient for chemical (unitless)

Model for MAHs:

$$\log(HC_5) = (-0.936)\log(K_{ow}) + \log(92.7) - \log(3.83) - 2.3\sqrt{(0.000225[\log(K_{ow})^2]) + 0.105 + 0.112}$$

(Recreated from McGrath and DiToro, 2009)

Where: HC_5 = Water concentration associated with protection of 95% of aquatic species (mmol/L)

K_{ow} = Octanol-water partitioning co-efficient for chemical (unitless)

4.4.2.1.2 Application of Lipid Target Model

The models presented above for mono-aromatic hydrocarbons and poly-aromatic hydrocarbons (McGrath and DiToro, 2009) were applied to compounds in the 2010 Taylor hydrocarbon model; log Kow values presented in the 2010 hydrocarbon model were used to determine the HC5 for each compound. As alkanes are present in the composition of crude oil the toxicological effects of these compounds was determined using the MAH model. The toxicity of alkanes is not as well known (as discussed in Section 2) and therefore it is assumed that use of a model that protects for chemicals exhibiting more toxic potential such as MAHs, is conservative for application to the alkanes evaluated in this study.

4.4.2.1.3 Resulting Aquatic Toxicity Reference Values

The resulting aquatic toxicity values using the McGrath and DiToro (2009) model for each compound are summarized in Table 9 below.

Table 9. Aquatic Toxicity Reference Concentration in Water

Substance	Surface water (µg/L)
CRUDE OIL	
Straight Chain Alkanes	
n-Hexane	3.88E+01
n-Heptane	8.70E+00
n-Octane	3.21E+00
n-Nonane	2.14E+00
n-Decane	3.93E-01
n-Undecane	2.45E-01
n-Dodecane	1.42E-01
Branched Chain Alkanes	
2,2-Dimethylbutane	4.62E+01
2,3-Dimethylbutane	1.10E+02
2-Methylpentane	5.49E+01
3-Methylpentane	7.43E+01
3-Ethylpentane	6.81E+01
2,4-Dimethylpentane	8.10E+01
2,3-Dimethylpentane	8.10E+01
2,2,4-Trimethylpentane	4.55E+00
2,3,3-Trimethylpentane	3.41E+01
2,3,4-Trimethylpentane	3.72E+01
2-Methyl-3-ethylpentane	3.20E+01
2-Methylhexane	6.81E+01
3-Methylhexane	6.81E+01
2,2-Dimethylhexane	2.93E+01
2,3-Dimethylhexane	3.20E+01
2,4-Dimethylhexane	3.20E+01
2,5-Dimethylhexane	3.20E+01
3,3-Dimethylhexane	2.93E+01

Substance	Surface water (µg/L)
2,3-Dimethylheptane	1.24E+01
2,6-Dimethylheptane	1.24E+01
2-Methyloctane	1.04E+01
3-Methyloctane	1.04E+01
4-Methyloctane	1.04E+01
Cycloalkanes	
Cyclopentane	2.22E+02
Methylcyclopentane	1.19E+02
1,1-Dimethylcyclopentane	9.23E+01
1-trans-2-Dimethylcyclopentane	1.01E+02
1-cis-3-Dimethylcyclopentane	1.01E+02
1-trans-3-Dimethylcyclopentane	1.01E+02
1,1,2-Trimethylcyclopentane	4.34E+01
1,1,3-Trimethylcyclopentane	4.34E+01
1-trans-2-cis-3-Trimethylcyclopentane	4.64E+01
1-trans-2-cis-4-Trimethylcyclopentane	4.64E+01
1-trans-2-Dimethylcyclohexane	3.98E+01
Ethylcyclohexane	1.21E+01
Cyclohexane	1.03E+02
1-trans-2-trans-4-Trimethylcyclohexane	1.79E+01
Alkyl Benzenes	
Benzene	1.62E+03
Toluene	5.22E+02
Ethylbenzene	2.42E+02
o-Xylene	2.59E+02
m-Xylene	2.18E+02
p-Xylene	2.42E+02
1-Methyl-4-ethylbenzene	9.71E+01
1-Methyl-2-ethylbenzene	1.21E+02
1-Methyl-3-ethylbenzene	5.65E+01
1,2,3-Trimethylbenzene	9.71E+01
1,2,4-Trimethylbenzene	8.35E+01
1,3,5-Trimethylbenzene	1.53E+02
1,2,3,4-Tetramethylbenzene	5.08E+01
Aryl-Benzene	
Biphenyl	5.47E+01
Naphtheno-Benzenes	
Indane	1.04E+02
Tetralin (tetrahydronaphthalene)	3.95E+01
5-Methyltetrahydronaphthalene	1.02E+01
6-Methyltetrahydronaphthalene	1.02E+01
Fluorene	2.33E+01

Substance	Surface water (µg/L)
Alkyl Naphthalenes	
Naphthalene	1.21E+02
Polynuclear Aromatics	
Phenanthrene	1.36E+01

4.4.3 Vapour Toxicity Reference Values

Toxicity reference values were not derived for vapour as it was considered that the TRVs reported by TPHCWG were considered appropriate for direct application at wildlands settings. This section outlines the approach used to arrive at this conclusion.

4.4.3.1 Toxicological Studies Evaluated

Toxicological reference values for petroleum hydrocarbon mixtures are limited (CCME, 2010) with most studies focussing on poly-aromatic hydrocarbons that only comprise a small fraction of petroleum mixtures (as presented in Section 2). Available data reviewed from regulatory sources (the USEPA's IRIS database and Health Canada 2009) was limited to a few chemicals being evaluating in this study; the IRIS database contained TRVs for benzene, toluene, ethylbenzene, xylenes, n-hexane and naphthalene with Health Canada having TRVs available for additional chemicals (Appendix B). The TPHCWG document recommending RfCs for hydrocarbon fractions contains toxicity reference values for the substances evaluated in the Taylor (2010) model. The TPHGWG document (Edwards et al, 1997) was determined to be the most comprehensive and appropriate source of toxicity reference values for the protection of human health. Therefore, derivation of TRVs for inhalation was not considered necessary as these values were already available from other regulatory sources.

4.4.3.2 Inhalation Toxicity Reference Values Used

The inhalation reference values used in criteria development for the protection of human health for each compound are summarized in Table 10.

Table 10. Summary of Inhalation Toxicity Reference Values Recommended for Criteria Development

Chemical / chemical structure	Carbon fraction	Recommended TRV	Source
Benzene	5-8	0.03 mg/m^3	IRIS, 2010
Ethylbenzene	>8-10	1.0 mg/m^3	IRIS, 2010
Naphthalene	>10-12	0.003 mg/m^3	IRIS, 2010
Toluene	>8-10	5 mg/m^3	IRIS, 2010
n-hexane	5-6	0.7 mg/m^3	IRIS, 2010
Xylene	>8-10	0.1 mg/m^3	IRIS, 2010
Aromatic Hydrocarbons	5-8	0.4 mg/m^3	Edwards et al, 1997
Aromatic Hydrocarbons	9-16	0.2 mg/m^3	Edwards et al, 1997
Aromatic Hydrocarbons	17-35	Non-recommended due to lack of volatility of fraction	Edwards et al, 1997
Aliphatic Hydrocarbons	5-8	18.4 mg/m^3	Edwards et al, 1997
Aliphatic Hydrocarbons	9-16	1.0 mg/m^3	Edwards et al, 1997
Aliphatic Hydrocarbons	17-35	Non-recommended due to lack of volatility of fraction	Edwards et al, 1997

Reference values in this study were categorized by chemical structure and equivalent carbon fraction with the corresponding TRV presented in Table 10 above assigned to each chemical accordingly.

4.4.4 Tissue Residue Toxicity Reference Values

Toxicity reference values were needed for tissue of aquatic (piscivourous fish) and terrestrial (caribou) biota. These values are associated with acceptable dietary

intake of chemicals to be of human populations consuming aquatic and terrestrial biota from exposed habitats. The RfDs reported by TPHCWG were considered appropriate for the basis of human health TRVs. This section outlines the approach used to arrive at this conclusion and the method used to determine appropriate tissue residue concentrations for use as criteria.

4.4.4.1 Toxicological Studies Evaluated

Toxicological reference values evaluating petroleum hydrocarbon mixtures are limited (CCME, 2010) with most studies focussing on poly-aromatic hydrocarbons that only comprise a small fraction of petroleum mixtures (as presented in Section 2). Available data reviewed from regulatory sources (the USEPA's IRIS database and Health Canada 2009) was limited to a few chemicals being evaluating in this study; the IRIS database contained TRVs for relatively few hydrocarbon compounds (benzene, toluene, biphenyl, fluorene and naphthalene) and Health Canada also has TRVs available for a few hydrocarbons (ethylbenzene, fluorene, n-hexane, naphthalene, toluene and xylene) (Appendix B). As both of these jurisdictions contain values for limited compounds peer-reviewed sources for remaining compounds were needed. The TPHCWG document recommending RfDs for hydrocarbon fractions contains toxicity reference values for the substances evaluated in the Taylor 2010 model. Edwards et al, 1997 is likely to be the most comprehensive and appropriate source of toxicity reference values for the protection of human health. Similarly, reviews of toxicity reference value data for petroleum hydrocarbons conducted by both the CCME and the RIVM have adopted reference values recommended by Edwards et al, 1997 into petroleum hydrocarbon criteria in their respective jurisdictions (CCME, 2008).

4.4.4.1.1 Application of Reference Doses to Tissue Residue Reference Values

Oral toxicity reference values were applied giving precedence to values from IRIS, Health Canada and TPHWG in descending order. Values from the IRIS database were given the highest priority due to it being frequently updated (BC MOE, 2007) with Health Canada being given second priority and the TPHWG being used as a default source of information when unavailable from other sources. The Edwards et al, 1997 document provides toxicity reference values for hydrocarbon compounds by equivalent

carbon fraction. Table 11 summarizes the reference doses recommended by IRIS, Health Canada and the TPHWG.

Table 11. Summary of Toxicity Reference Doses Used in Criteria Development

Chemical / chemical structure	Carbon fraction	Recommended TRV	Source
Benzene	5-8	0.04 mg/kg-d	IRIS, 2010
Biphenyl	>12-16	0.05 mg/kg-d	IRIS, 2010
Ethylbenzene	>8-10	0.1 mg/kg-d	IRIS, 2010
Fluorene	>12-16	0.04 mg/kg-d	IRIS, 2010
Naphthalene	>10-12	0.02 mg/kg-d	IRIS, 2010
Toluene	>8-10	0.08 mg/kg-d	IRIS, 2010
n-hexane	5-6	0.1 mg/kg-d	Health Canada, 2009
Xylene	>8-10	1.5 mg/kg-d	Health Canada, 2009
Aromatic Hydrocarbons	5-8	0.2 mg/kg-d	Edwards et al, 1997
Aromatic Hydrocarbons	>8-10	0.04 mg/kg-d	Edwards et al, 1997
Aromatic Hydrocarbons	>10-12	0.04 mg/kg-d	Edwards et al, 1997
Aromatic Hydrocarbons	>12-16	0.04 mg/kg-d	Edwards et al, 1997
Aromatic Hydrocarbons	>16-21	0.03 mg/kg-d	Edwards et al, 1997
Aromatic Hydrocarbons	>21-34	0.03 mg/kg-d	Edwards et al, 1997

Chemical / chemical structure	Carbon fraction	Recommended TRV	Source
Aromatic Hydrocarbons	>34	0.03 mg/kg-d	Edwards et al, 1997
Aliphatic Hydrocarbons	5-8	5.0 mg/kg-d	Edwards et al, 1997
Aliphatic Hydrocarbons	>8-10	0.1 mg/kg-d	Edwards et al, 1997
Aliphatic Hydrocarbons	>10-12	0.1 mg/kg-d	Edwards et al, 1997
Aliphatic Hydrocarbons	>12-16	0.1 mg/kg-d	Edwards et al, 1997
Aliphatic Hydrocarbons	>16-21	2.0 mg/kg-d	Edwards et al, 1997
Aliphatic Hydrocarbons	>21-34	2.0 mg/kg-d	Edwards et al, 1997
Aliphatic Hydrocarbons	>34	20.0 mg/kg-d	Edwards et al, 1997

Reference values in this study were categorized by chemical class and equivalent carbon fraction (as defined in Section 2) with the corresponding RfD presented in Table 11 assigned to each chemical accordingly.

Tissue residue concentrations were derived from the RfDs presented in Table 11 above by applying a modified version of the USEPA 2001 equation originally used to determine fish residue concentrations presented below. The formula takes into account intake parameters for each the human receptor, body weight and prey intake rates for which those presented in Section 4 were used as default values. The USEPA (2001 formula used to determine acceptable tissue reference concentrations is as follows:

$$TRC = \frac{[BW \times (RfD - RSC)]}{\sum_{i=1}^{n} PI_i}$$

Where:

TRC = acceptable tissue residue concentration (mg/kg)

BW = body weight of receptor (kg)

RSC = relative source contribution/consumption from other dietary sources (mg/kg-d)

RfD = reference dose (mg/kg-d)

FI*i* = fish intake rate summed for aquatic trophic levels fish (kg/d)

The formula presented above (USEPA, 2001), was modified to account for unknown background exposure to compounds being evaluated. A total of 20% of the RfD was applied to each fish and wild game tissue residue concentrations to allow for exposure of chemicals from background sources to be accounted for. The allocation of 20% of the TRV to a specific exposure route is consistent with CSST (1996) methodology as the group notes that this methodology should ensure adequate protection in the absence of site specific background data being available (CSST, 1996). The use of the 20% value was based on five sources of contaminants being available for exposure, with each source or media being given equal allocation of the final reference dose. The relative source contribution term (RSC), originally used to account for other dietary sources of the compound being evaluated, was removed and replaced with a default 20% allocation term. If additional sources of exposure are known, the allocation term should be modified to reflect relative contributions of other sources of the compounds being evaluated. The following equation (modified from USEPA 2001), was used to derive acceptable fish and wild game tissue residue concentrations:

$$TRC = \frac{[BW \times (RfD \times A)]}{\sum_{i=1}^{n} PI_i}$$

Where:

TRC = acceptable tissue residue concentration (mg/kg)

BW = body weight of receptor (kg)

RfD = reference dose (mg/kg-d)

A = allocation term (%)

PI*i* = prey intake rate summed for tissue consumption of diet items, i=1 though n (kg/d)

4.5 Derivation of Criteria

4.5.1 Soil Criteria

Soil criteria were based on concentrations in the terrestrial receptors diet relating to the hydrocarbon concentration in soil. A soil criterion was developed for each trophic guild. For soil invertebrates, soil criterion was based directly on those values presented in Section 5. For the remaining terrestrial guilds, criteria were derived using the following formula:

$$Criteria = \frac{TRV \times BW}{DI \times BA}$$

Where:

Criteria = soil criteria (mg/kg)

TRV = oral toxicity reference value (mg/kg-d)

BW = body weight (kg)

DI = daily intake (kg/d)

BA = bioavailability based on dietary uptake efficiency (unitless)

4.5.2 Surface Water Criteria

Surface water criteria were derived for each hydrocarbon compound directly from the models presented in Section 4.5.2 using the Kows represented in Section 2.1.

4.5.3 Sediment Criteria

Sediment criteria to protect for aquatic organisms were based on the surface water to sediment concentration ratios presented in Taylor, 2010. The following formula was used to develop sediment criteria to protect for aquatic species:

$$Criteria_{sed} = \frac{S}{SW} \times Criteria_{sw} \times CF$$

Where:

$Criteria_{sed}$ = sediment criteria (µg/g)

S/SW = ratio of concentration of compound in sediment (µg/g) to the concentration of compound in surface water (µg/g)

$Criteria_{sw}$ = surface water criteria (µg/L)

CF = conversion factor (1 L/1000 g)

4.5.4 Vapour Criteria

Vapour criteria to protect for human and terrestrial animal populations life were not produced as a part of this study. The intent of vapour criteria is to protect for the inhalation pathway from soil gas releasing into the breathing zone. Vapour criteria may be set that are equal to the tolerable reference concentrations recommended by the TPHHCWG (Edwards, et al., 1997). These values are used by the CCME to derive vapour inhalation guidelines with recommended guidelines based on an indoor setting (CCME, 2008). As structures representing indoor air scenarios are unlikely to be significant in a wildlands setting, these underlying reference concentrations used in the derivation of the criteria would be a more appropriate comparison characteristic. As the underlying reference concentrations are the same, these values are considered to be representative of meeting the guiding principle of protection of human health for populations using wildlands.

4.5.5 Tissue Criteria

The intake parameters presented in Section 3 were applied to the formula presented in Section 4.4.4.4 in the derivation of specific tissue concentrations considered to be acceptable for fish and wild game tissue.

5: Results and Discussion

5.1 Soil Criteria

Criteria for each trophic guild are presented in Table 13. However, for use in a site-specific setting, only those values corresponding to the receptors that are present/ or may be on-site should be used.

Table 13. Calculated Soil-Based Criteria for Petroleum Hydrocarbons for the Protection of Variety of Possible Target Organisms

Substance	Overall (mg/kg)	Caribou (mg/kg)	Wolf (mg/kg)	Shrew (mg/kg)	Owl (mg/kg)	Worm (mg/kg)
CRUDE OIL						
Straight Chain Alkanes						
n-Hexane	5.00E+02	6.71E+03	2.26E+03	5.00E+02	1.93E+03	1.50E+03
n-Heptane	1.90E+02	2.56E+03	8.62E+02	1.90E+02	7.36E+02	1.50E+03
n-Octane	4.76E+02	6.39E+03	2.16E+03	4.76E+02	1.84E+03	1.50E+03
n-Nonane	4.76E+02	6.39E+03	2.16E+03	4.76E+02	1.84E+03	1.50E+03
n-Decane	4.76E+02	6.39E+03	2.16E+03	4.76E+02	1.84E+03	1.50E+03
n-Undecane	9.52E+01	1.28E+03	4.31E+02	9.52E+01	3.68E+02	1.50E+03
n-Dodecane	9.52E+01	1.28E+03	4.31E+02	9.52E+01	3.68E+02	1.50E+03
Branched Chain Alkanes						
2,2-Dimethyl-butane	5.00E+02	6.71E+03	2.26E+03	5.00E+02	1.93E+03	1.50E+03
2,3-Dimethyl-butane	5.00E+02	6.71E+03	2.26E+03	5.00E+02	1.93E+03	1.50E+03
2-Methyl-pentane	5.00E+02	6.71E+03	2.26E+03	5.00E+02	1.93E+03	1.50E+03
3-Methylpentane	5.00E+02	6.71E+03	2.26E+03	5.00E+02	1.93E+03	1.50E+03
3-Ethylpentane	5.00E+02	6.71E+03	2.26E+03	5.00E+02	1.93E+03	1.50E+03
2,4-Dimethyl-pentane	5.00E+02	6.71E+03	2.26E+03	5.00E+02	1.93E+03	1.50E+03
2,3-Dimethyl-pentane	5.00E+02	6.71E+03	2.26E+03	5.00E+02	1.93E+03	1.50E+03
2,2,4-Trimethyl-pentane	5.00E+02	6.71E+03	2.26E+03	5.00E+02	1.93E+03	1.50E+03
2,3,3-Trimethyl-pentane	4.76E+02	6.39E+03	2.16E+03	4.76E+02	1.84E+03	1.50E+03

Substance	Overall (mg/kg)	Caribou (mg/kg)	Wolf (mg/kg)	Shrew (mg/kg)	Owl (mg/kg)	Worm (mg/kg)
2,3,4-Trimethyl-pentane	4.76E+02	6.39E+03	2.16E+03	4.76E+02	1.84E+03	1.50E+03
2-Methyl-3-ethyl-pentane	4.76E+02	6.39E+03	2.16E+03	4.76E+02	1.84E+03	1.50E+03
2-Methylhexane	4.76E+02	6.39E+03	2.16E+03	4.76E+02	1.84E+03	1.50E+03
3-Methylhexane	4.76E+02	6.39E+03	2.16E+03	4.76E+02	1.84E+03	1.50E+03
2,2-Dimethyl-hexane	4.76E+02	6.39E+03	2.16E+03	4.76E+02	1.84E+03	1.50E+03
2,3-Dimethyl-hexane	4.76E+02	6.39E+03	2.16E+03	4.76E+02	1.84E+03	1.50E+03
2,4-Dimethyl-hexane	4.76E+02	6.39E+03	2.16E+03	4.76E+02	1.84E+03	1.50E+03
2,5-Dimethyl-hexane	4.76E+02	6.39E+03	2.16E+03	4.76E+02	1.84E+03	1.50E+03
3,3-Dimethylhexane	4.76E+02	6.39E+03	2.16E+03	4.76E+02	1.84E+03	1.50E+03
2,3-Dimethyl-heptane	4.76E+02	6.39E+03	2.16E+03	4.76E+02	1.84E+03	1.50E+03
2,6-Dimethyl-heptane	4.76E+02	6.39E+03	2.16E+03	4.76E+02	1.84E+03	1.50E+03
2-Methyloctane	4.76E+02	6.39E+03	2.16E+03	4.76E+02	1.84E+03	1.50E+03
3-Methyloctane	4.76E+02	6.39E+03	2.16E+03	4.76E+02	1.84E+03	1.50E+03
4-Methyloctane	4.76E+02	6.39E+03	2.16E+03	4.76E+02	1.84E+03	1.50E+03
Cycloalkanes						
Cyclopentane	5.00E+02	6.71E+03	2.26E+03	5.00E+02	1.93E+03	1.50E+03
Methylcyclopentane	5.00E+02	6.71E+03	2.26E+03	5.00E+02	1.93E+03	1.50E+03
1,1-Dimethylcyclo-pentane	5.00E+02	6.71E+03	2.26E+03	5.00E+02	1.93E+03	1.50E+03
1-trans-2-Dimethylcyclo-pentane	5.00E+02	6.71E+03	2.26E+03	5.00E+02	1.93E+03	1.50E+03
1-cis-3-Dimethylcyclo-pentane	5.00E+02	6.71E+03	2.26E+03	5.00E+02	1.93E+03	1.50E+03
1-trans-3-Dimethylcyclo-pentane	5.00E+02	6.71E+03	2.26E+03	5.00E+02	1.93E+03	1.50E+03
1,1,2-Trimethylcyclo-pentane	4.76E+02	6.39E+03	2.16E+03	4.76E+02	1.84E+03	1.50E+03
1,1,3-Trimethylcyclo-pentane	4.76E+02	6.39E+03	2.16E+03	4.76E+02	1.84E+03	1.50E+03
1-trans-2-cis-3-Trimethylcyclo-pentane	4.76E+02	6.39E+03	2.16E+03	4.76E+02	1.84E+03	1.50E+03

Substance	Overall (mg/kg)	Caribou (mg/kg)	Wolf (mg/kg)	Shrew (mg/kg)	Owl (mg/kg)	Worm (mg/kg)
1-trans-2-Dimethylcyclo-hexane	4.76E+02	6.39E+03	2.16E+03	4.76E+02	1.84E+03	1.50E+03
Ethylcyclohexane	4.76E+02	6.39E+03	2.16E+03	4.76E+02	1.84E+03	1.50E+03
Cyclohexane	5.00E+02	6.71E+03	2.26E+03	5.00E+02	1.93E+03	1.50E+03
1-trans-2-trans-4-Trimethylcyclo-hexane	5.00E+02	6.71E+03	2.26E+03	5.00E+02	1.93E+03	1.50E+03
Alkyl Benzenes						
Benzene	2.65E-01	3.56E+00	1.20E+00	2.65E-01	1.02E+00	1.50E+03
Toluene	2.12E+02	2.85E+03	9.61E+02	2.12E+02	8.20E+02	1.50E+03
Ethylbenzene	9.24E+01	1.24E+03	4.19E+02	9.24E+01	3.57E+02	1.50E+03
o-Xylene	1.70E+02	2.29E+03	7.72E+02	1.70E+02	6.59E+02	1.50E+03
m-Xylene	1.70E+02	2.29E+03	7.72E+02	1.70E+02	6.59E+02	1.50E+03
p-Xylene	1.35E+02	1.81E+03	6.10E+02	1.35E+02	5.20E+02	1.50E+03
1-Methyl-4-ethylbenzene	1.05E+02	1.41E+03	4.74E+02	1.05E+02	4.05E+02	1.50E+03
1-Methyl-2-ethylbenzene	1.05E+02	1.41E+03	4.74E+02	1.05E+02	4.05E+02	1.50E+03
1-Methyl-3-ethylbenzene	1.05E+02	1.41E+03	4.74E+02	1.05E+02	4.05E+02	1.50E+03
1,2,3-Trimethyl-benzene	1.05E+02	1.41E+03	4.74E+02	1.05E+02	4.05E+02	1.50E+03
1,2,4-Trimethyl-benzene	1.05E+02	1.41E+03	4.74E+02	1.05E+02	4.05E+02	1.50E+03
1,3,5-Trimethyl-benzene	1.05E+02	1.41E+03	4.74E+02	1.05E+02	4.05E+02	1.50E+03
1,2,4-Tetramethyl-benzene	3.40E+01	4.56E+02	1.54E+02	3.40E+01	1.31E+02	1.50E+03
Aryl-Benzene						
Biphenyl	4.76E+01	6.39E+02	2.16E+02	4.76E+01	1.84E+02	1.50E+03
Naphtheno-Benzenes						
Indane	1.05E+02	1.41E+03	4.74E+02	1.05E+02	4.05E+02	1.50E+03
Tetralin (tetrahydro-naphthalene)	3.40E+01	4.56E+02	1.54E+02	3.40E+01	1.31E+02	1.50E+03
5-Methyltetrahydro-naphthalene	3.40E+01	4.56E+02	1.54E+02	3.40E+01	1.31E+02	1.50E+03
6-Methyltetrahydo-naphthalene	3.40E+01	4.56E+02	1.54E+02	3.40E+01	1.31E+02	1.50E+03
Fluorene	1.02E+02	1.37E+03	4.63E+02	1.02E+02	3.95E+02	1.50E+03
Alkyl Naph-thalenes						
Naphthalene	6.76E+01	9.08E+02	3.06E+02	6.76E+01	2.61E+02	1.50E+03
Poly-nuclear Aromatics						
Phenanthrene	6.13E+01	8.24E+02	2.78E+02	6.13E+01	2.37E+02	1.50E+03

The final soil concentration for each compound was based on the lowest value to ensure protection of all trophic guilds. Soil criteria for petroleum hydrocarbon mixtures are available from both the CCME and BC CSR. The CSR approach is based on a corrected extractable hydrocarbon method that cannot be compared directly with the equivalent carbon Edwards et al, 1997 method (Science Advisory Board for Contaminated Sites in British Columbia, 2004). The CCME uses the equivalent carbon fraction approach that is based on the Edwards et al, 1997 methodology which was also used in the Taylor 2010 model. Criteria for the ecological soil ingestion pathway, however, have not been produced by the CCME. The CCME states that although guidelines were not derived to protect for this pathway due to the possibility of limited data to evaluate, soil ingestion is an important pathway to consider (CCME, 2008). As guidelines based on ingestion using higher order food web interactions guidelines were not used in the CCME approach, for the purposes of comparison, the criteria produced in this study were compared to the existing CCME guidelines for the protection of ecological health in the agricultural/residential land use category (based on lower trophic level protection) as it is the most protective land use considered by the CCME.

A mass fraction approach was used to determine F1, F2 and F3 fraction criteria from the criteria concentrations presented in Table 13 above. The overall soil criteria presented in Table 13 was multiplied by the fraction of mass that compound contributes to the total mass of its fraction group using the following formula.

$$\sum_{i=1}^{n} Csb = \frac{Mass_i}{Mass_{sb}} \times C_i$$

Where:

C_{sb} = Criteria representing the sub-fraction (mg/kg)

C_i = Criteria (overall) for the compound (mg/kg)

$Mass_i$ = Molecular Mass of the compound (g/mol)

$Mass_{sb}$ = Total molecular mass of the sub-fraction (g/mol)

The hydrocarbon fraction groups were based on the CCME sub-fractions with F1 consisting of compounds that have equivalent hydrocarbons of 6-10, F2 >10-16, and F3 >16-34. Resulting study criteria for each sub-fraction are presented in Table 14.

Table 14. Comparison of Resulting Soil Criteria to Existing CCME Guidelines for the Protection of Ecological Health

Hydro-carbon Fraction	Criteria from this study (mg/kg)	CCME 2008 Ag/Res value (mg/kg)	Rational for CCME value	Comments	Reference
F1 (C6-10)	368	210	Based on ranked percentile (25^{th}%ile) distribution to be protective of soil invertebrates and plants.	Study value averaged overall criteria for all available carbon fractions in the study.	(CCME, 2008)
F2 (C>10-16)	62	150	Based on ranked percentile (25^{th}%ile) distribution to be protective of soil invertebrates and plants.	Study value averaged overall criteria for all available carbon fractions in the study.	(CCME, 2008)
F3 (C>16-34)	61	300-1300	Based on ranked percentile (25^{th}%ile) distribution to be protective of soil invertebrates and plants. Value of 300 mg/kg for coarse and 1300 mg/kg for fine grained soil.	Study value corresponds to phenanthrene criteria as this was the only compound within the C>16-34 range.	(CCME, 2008)

Resulting study values were within the same order of magnitude as the CCME 2008 values for the F1 fraction with the study values being higher than those presented by the CCME. Study values for the F2 and F3 fractions were lower than the CCME values by one order of magnitude. It should be noted that the CCME values are based on a ranked percentile approach that is not associated with a specific protection level and that the study values are protecting for the terrestrial organisms selected in Section 4 (e.g. caribou, wolf, and shrew) and that the CCME values are protecting for soil invertebrates and plants. Therefore, as the study values are either in the same order of magnitude or lower than the CCME values, the study values are generally consistent with a similar or higher degree of protection than the CCME values.

5.1.1 Worked Example of Soil Criteria Derivation

Sample Substance: n-heptane

- Unit Conversions: 1000 (g/kg)
- Bioavailability: 0.8 (unitless)
    - Lipid and Protein Normalized TRVs developed in this study ($mg \cdot kg^{-1} \cdot d^{-1}$):
        - Shrew = 1.14E+02 $mg \cdot kg^{-1} \cdot d^{-1}$
        - Wolf = 8.62E+01 $mg \cdot kg^{-1} \cdot d^{-1}$
        - Caribou = 7.88E+01 $mg \cdot kg^{-1} \cdot d^{-1}$
    - Daily Intakes (g/day):
        - Shrew = 9 g/day
        - Wolf = 450 g/day
        - Caribou = 1740 g/day
    - Body Weight (kg):
        - Shrew = 0.015 kg
        - Wolf = 4.5 kg
        - Caribou = 57 kg

Using the formula:

$$Criteria = \frac{TRV \times BW}{DI}$$

Soil Criteria for Protection of Shrew:

$$Soil_{\substack{criteria \\ forshrew}} = \frac{(1.14E+02 \times 0.015)}{(9)} \times 1000 = 1.90E+02$$

Soil Criteria for Protection of Wolf:

$$Soil_{\substack{criteria \\ forwolf}} = \frac{(8.62E+01 \times 4.5)}{(450)} \times 1000 = 8.62E+02$$

Soil Criteria for Protection of Caribou:

$$Soil_{\substack{criteria \\ forcaribou}} = \frac{(7.88E+01 \times 57)}{(1740)} \times 1000 = 2.56E+03$$

5.2 Surface Water and Sediment Criteria

5.2.1 Surface Water

Surface water criteria were developed based on a species sensitivity distribution target lipid model developed by McGrath and DiToro (2010). The resulting criteria are presented in Table 15.

Table 15. Surface Water Criteria

Substance	Surface water (µg/L)
CRUDE OIL	
Straight Chain Alkanes	
n-Hexane	3.88E+01
n-Heptane	8.70E+00
n-Octane	3.21E+00
n-Nonane	2.14E+00
n-Decane	3.93E-01
n-Undecane	2.45E-01
n-Dodecane	1.42E-01
Branched Chain Alkanes	
2,2-Dimethylbutane	4.62E+01

Substance	Surface water (µg/L)
2,3-Dimethylbutane	1.10E+02
2-Methylpentane	5.49E+01
3-Methylpentane	7.43E+01
3-Ethylpentane	6.81E+01
2,4-Dimethylpentane	8.10E+01
2,3-Dimethylpentane	8.10E+01
2,2,4-Trimethylpentane	4.55E+00
2,3,3-Trimethylpentane	3.41E+01
2,3,4-Trimethylpentane	3.72E+01
2-Methyl-3-ethylpentane	3.20E+01
2-Methylhexane	6.81E+01
3-Methylhexane	6.81E+01
2,2-Dimethylhexane	2.93E+01
2,3-Dimethylhexane	3.20E+01
2,4-Dimethylhexane	3.20E+01
2,5-Dimethylhexane	3.20E+01
3,3-Dimethylhexane	2.93E+01
2,3-Dimethylheptane	1.24E+01
2,6-Dimethylheptane	1.24E+01
2-Methyloctane	1.04E+01
3-Methyloctane	1.04E+01
4-Methyloctane	1.04E+01
Cycloalkanes	
Cyclopentane	2.22E+02
Methylcyclopentane	1.19E+02
1,1-Dimethylcyclopentane	9.23E+01
1-trans-2-Dimethylcyclopentane	1.01E+02
1-cis-3-Dimethylcyclopentane	1.01E+02
1-trans-3-Dimethylcyclopentane	1.01E+02
1,1,2-Trimethylcyclopentane	4.34E+01
1,1,3-Trimethylcyclopentane	4.34E+01
1-trans-2-cis-3-Trimethylcyclopentane	4.64E+01
1-trans-2-cis-4-Trimethylcyclopentane	4.64E+01
1-trans-2-Dimethylcyclohexane	3.98E+01
Ethylcyclohexane	1.21E+01
Cyclohexane	1.03E+02
1-trans-2-trans-4-Trimethylcyclohexane	1.79E+01
Alkyl Benzenes	
Benzene	1.62E+03
Toluene	5.22E+02
Ethylbenzene	2.42E+02
o-Xylene	2.59E+02
m-Xylene	2.18E+02
p-Xylene	2.42E+02
1-Methyl-4-ethylbenzene	9.71E+01
1-Methyl-2-ethylbenzene	1.21E+02

Substance	Surface water (µg/L)
1-Methyl-3-ethylbenzene	5.65E+01
1,2,3-Trimethylbenzene	9.71E+01
1,2,4-Trimethylbenzene	8.35E+01
1,3,5-Trimethylbenzene	1.53E+02
1,2,3,4-Tetramethylbenzene	5.08E+01
Aryl-Benzene	
Biphenyl	5.47E+01
Naphtheno-Benzenes	
Indane	1.04E+02
Tetralin (tetrahydronaphthalene)	3.95E+01
5-Methyltetrahydronaphthalene	1.02E+01
6-Methyltetrahydronaphthalene	1.02E+01
Fluorene	2.33E+01
Alkyl Naphthalenes	
Naphthalene	1.21E+02
Polynuclear Aromatics	
Phenanthrene	1.36E+01

Neither the CCME nor the BC Water Quality Guidelines have surface water guidelines for mixture hydrocarbon compounds to compare to the criteria derived in this study. The BC Water Quality Guidelines contain criteria and/or interim criteria for the mono aromatic hydrocarbons, benzene, ethylbenzene, toluene and xylene as well as the polycyclic aromatic hydrocarbon, naphthalene. These values were used for comparison with the values resulting from the study. The BC WQG and the basis for each criteria (BC MOE, 2010) are presented in Table 16 following.

Table 16. Comparison of Resulting Surface Water Criteria to Existing BC WQG

Substance	Criteria used in this study (µg/L)	BC WQG value (µg/L)	Rational for BC WQG	Comments	Reference
Benzene	1618	40 (interim value)	Based on a 7-day LOAEL of 400 µg/L (corresponding to a 20% effects level for R. pipiens) with a safety factor of 0.1	This guideline is considered to be interim due to lack of toxicological data.	(BC MOE, 2003)
Ethyl-benzene	242	200	Based on multiple EC50s ranging from 1800 to 2200 µg/L on D. magna species with a safety factor of 0.1.	Daphnia magna were determined to be the most sensitive species.	(BC MOE, 1999)
Toluene	522	0.5	Based on an EC20 of 5 µg/L of O. mykiss species with a safety factor of 0.1.	O. mykiss were determined to be the most sensitive species.	(BC MOE, 2007)
Xylene	218-259	30	Based on a EC20 of 310 µg/L of R. pipiens with a safety factor of 0.1.	R. pipiens were determined to be the most sensitive species, the application of a safety factor is stated to protect for R. pipiens.	(BC MOE, 2007)
Naphtha-lene	121	1	Based on a EC20 of 11 µg/L for chronic O mykiss with a safety factor of 0.1.	Large distribution of toxicological endpoint concentrations reported by BC MOE.	(BC MOE, 2007)

The resulting values from the McGrath and DiToro (2009) method result in higher concentrations than those criteria set by the BC MOE, as demonstrated in Table 15. The 2009 method is based on species sensitivity distributions for a 95% protection level; the method uses Kow values to account for differences in lipid action potential of chemicals (McGrath and DiToro, 2009). The BC MOE criteria are based on the most sensitive species offering 20% effects levels with safety factors of 0.1, or 10 times lower

modifier, applied (BC MOE, 1999, 2003, 2007a, 2007b, 2007c). Validation of the McGrath and DiToro (2009) method includes comparison to chronic no observable effects concentrations including trophic guilds represented in this study. However, the toxicity values presented in the BC Water Quality Guidelines indicate lower concentrations for toxicity than those reported in McGrath and DiToro (2009) and those resulting from an EcoTox search (Appendix A). The BC MOE (2007c) acknowledges the variation in toxicity concentrations considered in guideline development of the volatiles compared above. As the McGrath and DiToro (2009) incorporates no observable effects concentrations and multiple species in the derivation methodology, therefore limiting the reliance the use of safety factors, and that the 2009 model can be applied to all MAH and PAH compounds, the use of the 2009 McGrath and DiToro lipid target model is considered appropriate for wildlands criteria derivation as it follows the guiding principles of use of the best available science (through use of a species sensitivity model, consideration of mode of toxic action of hydrocarbons and model validation by comparing results to know no observable adverse effects concentrations) and a 95% degree of protection. If more no observable adverse effects concentrations, or EC5s become available for hydrocarbon compounds these concentrations should be compared to the 2009 model to ensure that species are not under protected.

5.2.2 Sediment

The resulting values derived for sediment criteria using the methodology presented in Section 4.6.3 are presented in Table 17.

Table 17. Sediment Criteria Determined in this Study using Surface Water to Sediment Ratios

Substance	Sediment (µg/g)
CRUDE OIL	
Straight Chain Alkanes	
n-Hexane	1.64E+01
n-Heptane	2.56E+01
n-Octane	1.85E+02
n-Nonane	3.01E+01
n-Decane	1.29E+01
n-Undecane	8.99E+00
n-Dodecane	9.18E+00

Substance	Sediment (µg/g)
Branched Chain Alkanes	
2,2-Dimethylbutane	1.59E+01
2,3-Dimethylbutane	1.41E+01
2-Methylpentane	1.55E+01
3-Methylpentane	1.48E+01
3-Ethylpentane	1.78E+01
2,4-Dimethylpentane	1.74E+01
2,3-Dimethylpentane	1.74E+01
2,2,4-Trimethylpentane	2.38E+02
2,3,3-Trimethylpentane	2.36E+01
2,3,4-Trimethylpentane	2.32E+01
2-Methyl-3-ethylpentane	2.39E+01
2-Methylhexane	1.78E+01
3-Methylhexane	1.78E+01
2,2-Dimethylhexane	2.43E+01
2,3-Dimethylhexane	2.39E+01
2,4-Dimethylhexane	2.39E+01
2,5-Dimethylhexane	2.39E+01
3,3-Dimethylhexane	2.43E+01
2,3-Dimethylheptane	3.23E+01
2,6-Dimethylheptane	3.23E+01
2-Methyloctane	3.30E+01
3-Methyloctane	3.30E+01
4-Methyloctane	3.30E+01
Cycloalkanes	
Cyclopentane	1.04E+01
Methylcyclopentane	1.35E+01
1,1-Dimethylcyclopentane	1.64E+01
1-trans-2-Dimethylcyclopentane	1.62E+01
1-cis-3-Dimethylcyclopentane	1.62E+01
1-trans-3-Dimethylcyclopentane	1.62E+01
1,1,2-Trimethylcyclopentane	2.18E+01
1,1,3-Trimethylcyclopentane	2.18E+01
1-trans-2-cis-3-Trimethylcyclopentane	2.16E+01
1-trans-2-cis-4-Trimethylcyclopentane	2.16E+01
1-trans-2-Dimethylcyclohexane	2.22E+01
Ethylcyclohexane	2.76E+01
Cyclohexane	1.42E+01
1-trans-2-trans-4-Trimethylcyclohexane	2.37E+01
Alkyl Benzenes	
Benzene	3.68E+02
Toluene	5.97E+02
Ethylbenzene	6.37E+02
o-Xylene	7.23E+02
m-Xylene	4.23E+01
p-Xylene	1.92E+01

Substance	Sediment (µg/g)
1-Methyl-4-ethylbenzene	1.41E+01
1-Methyl-2-ethylbenzene	2.11E+02
1-Methyl-3-ethylbenzene	1.88E+01
1,2,3-Trimethylbenzene	1.55E+01
1,2,4-Trimethylbenzene	2.50E+01
1,3,5-Trimethylbenzene	3.97E+01
1,2,3,4-Tetramethylbenzene	1.89E+01
Aryl-Benzene	
Biphenyl	4.97E+01
Naphtheno-Benzenes	
Indane	5.39E+00
Tetralin (tetrahydronaphthalene)	8.61E+00
5-Methyltetrahydronaphthalene	1.66E+01
6-Methyltetrahydronaphthalene	1.66E+01
Fluorene	1.68E+01
Alkyl Naphthalenes	
Naphthalene	1.71E+01
Polynuclear Aromatics	
Phenanthrene	1.04E+00

Limited sediment criteria for the protection of aquatic life are available for petroleum hydrocarbon substances. The BC CSR standards are limited to naphthalene, fluorene and combined criteria for total PAHs. Similarly, CCME interim sediment guidelines for the protection of aquatic life exist for naphthalene and fluorene. Comparisons to both criteria are presented following in Table 18. Note that the CSR sediment criteria are categorized into sensitive and typical uses, for the purposes of comparison to a wildlands setting, the sensitive use category was taken into consideration. Additionally, total PAHs were not compared due to the differing compounds comprising the CSR total PAH mixture [2-methylnaphthalene, acenaphthene, acenaphthylene , anthracene , fluorene, naphthalene, phenanthrene, benz(a)anthracene, benzo(a)pyrene, chrysene, dibenz(a,h)anthracene, fluoranthene, and pyrene] from those evaluated in this study (naphthalene and phenanthrene).

Table 18. Comparison of Resulting Calculated Sediment Criteria to Existing BC CSR and CCME Sediment Guidelines

Substance	Study value (µg/g)	Guideline value (µg/g)	Rationale for guideline	Comments	Reference
Naphthalene	17.1	CSR - 0.24	Not provided	None	(BC MOE, 1996)
Naphthalene	17.1	CCME - 34.6	Specific rationale not provided	None	(CCME, 1999)
Fluorene	16.8	CSR - 0.086	Not provided	None	(BC MOE, 1996)
Fluorene	16.8	CCME – 21.2	Specific rationale not provided	None	(CCME, 1999)
Total PAHs	18.1	CSR - 10	Not provided	The study evaluates two PAHs while the CSR value 13 PAH compounds.	

The values obtained from the study are consistent with the CCME guidelines for both naphthalene and fluorene with the wildlands values being within the same order of magnitude and slightly lower than the CCME values consistent with the intent to produce criteria to a higher degree of protection for wildlands. The individual hydrocarbon concentrations produced in the study are higher than those presented in the BC CSR by two orders of magnitude while the total PAH mixture is in the same order of magnitude as the BC CSR values. However, it should be noted that the BC CSR value takes into account several PAHs not included in this study to arrive at the total PAH value which theoretically results in the study value being even higher than presented if additional PAHs were taken into account. Sufficient background information is not available from either the BC MOE or the CCME to speculate on the discrepancy between these values. As well, the values are limited to two substances and are unable to serve as validation points for the proposed wildlands methodology. Therefore, the underlying assumption of surface water to sediment concentration ration used to derive the wildlands criteria should be further evaluated to validate the study values.

5.2.3 Groundwater

Groundwater criteria to protect for aquatic life were not produced as a part of this study. The intent of groundwater criteria is to protect for the groundwater to surface water pathway to ensure that aquatic life is protected at point of discharge (potential contact with aquatic organisms). It is recommended that groundwater to surface water interactions be further evaluated before groundwater criteria are derived.

5.3 Tissue Residue Criteria

The tissue residue concentrations derived for fish and wild game for the protection of human populations with values derived for each a toddler and an adult as presented in Table 19.

Table 19. Tissue Residue Criteria for Select Animal Tissue for the Protection of Human Health

Substance	**Tissue residue value (mg/kg)**			
	Toddler	**Toddler**	**Adult**	**Adult**
	Fish tissue	Caribou tissue	Fish tissue	Caribou tissue
CRUDE OIL				
Straight Chain Alkanes				
n-Hexane	5.89E+00	3.88E+00	1.27E+01	5.24E+00
n-Heptane	2.95E+02	1.94E+02	6.37E+02	2.62E+02
n-Octane	2.95E+02	1.94E+02	6.37E+02	2.62E+02
n-Nonane	5.89E+00	3.88E+00	1.27E+01	5.24E+00
n-Decane	5.89E+00	3.88E+00	1.27E+01	5.24E+00
n-Undecane	5.89E+00	3.88E+00	1.27E+01	5.24E+00
n-Dodecane	5.89E+00	3.88E+00	1.27E+01	5.24E+00
Branched Chain Alkanes				
2,2-Dimethylbutane	2.95E+02	1.94E+02	6.37E+02	2.62E+02
2,3-Dimethylbutane	2.95E+02	1.94E+02	6.37E+02	2.62E+02
2-Methylpentane	2.95E+02	1.94E+02	6.37E+02	2.62E+02
3-Methylpentane	2.95E+02	1.94E+02	6.37E+02	2.62E+02
3-Ethylpentane	2.95E+02	1.94E+02	6.37E+02	2.62E+02
2,4-Dimethylpentane	2.95E+02	1.94E+02	6.37E+02	2.62E+02
2,3-Dimethylpentane	2.95E+02	1.94E+02	6.37E+02	2.62E+02
2,2,4-Trimethylpentane	2.95E+02	1.94E+02	6.37E+02	2.62E+02

Substance	Toddler	Toddler	Adult	Adult
	Fish tissue	Caribou tissue	Fish tissue	Caribou tissue
2,3,3-Trimethylpentane	2.95E+02	1.94E+02	6.37E+02	2.62E+02
2,3,4-Trimethylpentane	2.95E+02	1.94E+02	6.37E+02	2.62E+02
2-Methyl-3-ethylpentane	2.95E+02	1.94E+02	6.37E+02	2.62E+02
2-Methylhexane	2.95E+02	1.94E+02	6.37E+02	2.62E+02
3-Methylhexane	2.95E+02	1.94E+02	6.37E+02	2.62E+02
2,2-Dimethylhexane	2.95E+02	1.94E+02	6.37E+02	2.62E+02
2,3-Dimethylhexane	2.95E+02	1.94E+02	6.37E+02	2.62E+02
2,4-Dimethylhexane	2.95E+02	1.94E+02	6.37E+02	2.62E+02
2,5-Dimethylhexane	2.95E+02	1.94E+02	6.37E+02	2.62E+02
3,3-Dimethylhexane	2.95E+02	1.94E+02	6.37E+02	2.62E+02
2,3-Dimethylheptane	2.95E+02	1.94E+02	6.37E+02	2.62E+02
2,6-Dimethylheptane	2.95E+02	1.94E+02	6.37E+02	2.62E+02
2-Methyloctane	2.95E+02	1.94E+02	6.37E+02	2.62E+02
3-Methyloctane	2.95E+02	1.94E+02	6.37E+02	2.62E+02
4-Methyloctane	2.95E+02	1.94E+02	6.37E+02	2.62E+02
Cycloalkanes				
Cyclopentane	2.95E+02	1.94E+02	6.37E+02	2.62E+02
Methylcyclopentane	2.95E+02	1.94E+02	6.37E+02	2.62E+02
1,1-Dimethylcyclopentane	2.95E+02	1.94E+02	6.37E+02	2.62E+02
1-trans-2-Dimethylcyclopentane	2.95E+02	1.94E+02	6.37E+02	2.62E+02
1-cis-3-Dimethylcyclopentane	2.95E+02	1.94E+02	6.37E+02	2.62E+02
1-trans-3-Dimethylcyclopentane	2.95E+02	1.94E+02	6.37E+02	2.62E+02
1,1,2-Trimethylcyclopentane	2.95E+02	1.94E+02	6.37E+02	2.62E+02
1,1,3-Trimethylcyclopentane	2.95E+02	1.94E+02	6.37E+02	2.62E+02
1-trans-2-cis-3-Trimethylcyclopentane	2.95E+02	1.94E+02	6.37E+02	2.62E+02
1-trans-2-cis-4-Trimethylcyclopentane	2.95E+02	1.94E+02	6.37E+02	2.62E+02
1-trans-2-Dimethylcyclohexane	2.95E+02	1.94E+02	6.37E+02	2.62E+02
Ethylcyclohexane	5.89E+00	3.88E+00	1.27E+01	5.24E+00
Cyclohexane	2.95E+02	1.94E+02	6.37E+02	2.62E+02
1-trans-2-trans-4-Trimethylcyclohexane	2.95E+02	1.94E+02	6.37E+02	2.62E+02
Alkyl Benzenes				
Benzene	1.18E+01	7.76E+00	2.55E+01	1.05E+01
Toluene	4.71E+00	3.11E+00	1.02E+01	4.19E+00
Ethylbenzene	5.89E+00	3.88E+00	1.27E+01	5.24E+00
o-Xylene	8.84E+01	5.82E+01	1.91E+02	7.86E+01
m-Xylene	8.84E+01	5.82E+01	1.91E+02	7.86E+01
p-Xylene	8.84E+01	5.82E+01	1.91E+02	7.86E+01
1-Methyl-4-ethylbenzene	2.36E+00	1.55E+00	5.10E+00	2.09E+00
1-Methyl-2-ethylbenzene	2.36E+00	1.55E+00	5.10E+00	2.09E+00
1-Methyl-3-ethylbenzene	2.36E+00	1.55E+00	5.10E+00	2.09E+00
1,2,3-Trimethylbenzene	2.36E+00	1.55E+00	5.10E+00	2.09E+00
1,2,4-Trimethylbenzene	2.36E+00	1.55E+00	5.10E+00	2.09E+00
1,3,5-Trimethylbenzene	2.36E+00	1.55E+00	5.10E+00	2.09E+00
1,2,3,4-Tetramethylbenzene	2.36E+00	1.55E+00	5.10E+00	2.09E+00
Aryl-Benzene				
Biphenyl	2.95E+00	1.94E+00	6.37E+00	2.62E+00

Substance	Toddler	Toddler	Adult	Adult
	Fish tissue	Caribou tissue	Fish tissue	Caribou tissue
Naphtheno-Benzenes				
Indane	2.36E+00	1.55E+00	5.10E+00	2.09E+00
Tetralin (tetrahydronaphthalene)	2.36E+00	1.55E+00	5.10E+00	2.09E+00
5-Methyltetrahydronaphthalene	2.36E+00	1.55E+00	5.10E+00	2.09E+00
6-Methyltetrahydronaphthalene	2.36E+00	1.55E+00	5.10E+00	2.09E+00
Fluorene	2.36E+00	1.55E+00	5.10E+00	2.09E+00
Alkyl Naphthalenes				
Naphthalene	1.77E+00	1.16E+00	3.82E+00	1.57E+00
Polynuclear Aromatics				
Phenanthrene	2.36E+00	1.55E+00	5.10E+00	2.09E+00

Tissue residue criteria have not been developed for purposes of protection of consumption of country foods by either the CCME or the CSR for hydrocarbon mixtures. Therefore, the tissue residue concentrations resulting from the study could not be compared to similar guidelines due to limitations in obtaining human consumption criteria for fractionated compounds.

6: Conclusions

Both data gaps and uncertainty lead to limitations in the derivation process developed and applied in this project. This section presents the applicability of this process along with the uncertainties in the risk-based approach (specifically the relationships between media, the development of toxicity reference values and exposure assumptions) and presents ways by which uncertainty can be reduced.

6.1 Uncertainty in Risk-based Approach

6.1.1 Assumptions in Relationships between Media

The methodology developed to derive of wildlands criteria presented in this study relies on the relationships between environmental media (e.g. surface water to sediment concentrations). The relationships used to develop criteria were based on the 2010 Taylor fugacity based wildlands model. Validation of the model to measure and mitigate uncertainty in the relationships between compartments would be helpful to reduce uncertainty with the assumptions made in the model. Methods to validate the model include validation of a 'generic' site or validation on a site-specific basis as a part of the tiered structure of the criteria application process.

6.1.2 Uncertainty in Toxicity Reference Values

Ecological toxicity reference values were derived for both aquatic and terrestrial receptors with tissue residue concentrations derived for human populations. Remaining values were based on existing peer-reviewed sources. An effort was made to provide ecological receptors with a 95% protection level on a community basis.

Aquatic criteria were based on a corrected species sensitivity distribution based model that was developed to provide 95% protection level for critical endpoints (survival, growth and reproduction) therefore the uncertainty with the toxicity reference values for aquatic species remains low.

Toxicity data was not available for the 95% protection level of terrestrial receptors. Thus, use of no adverse effects level data incorporating more sensitive endpoints was used when available to reduce the uncertainty with terrestrial protection levels. Furthermore, values were protein and lipid normalized to reduce uncertainty with extrapolation across species.

Uncertainty with extrapolation of toxicological reference data across chemicals is present as data specific to each compound assessed in the study was unavailable. To reduce uncertainty in extrapolation across chemicals, extrapolation was limited to compounds with similar chemical structures to take into account proposed modes of toxic action and to chemicals in similar physical characteristics (e.g. equivalent carbon fraction ranges) to account for similarities in compound stability.

6.1.3 Uncertainty in Exposure Assessment

Determination of chemical exposure by receptors was largely limited to use of peer-reviewed references and assumptions on exposure pathways. The use of peer-reviewed exposure data was used as a means to reduce uncertainty although the degree of differences in exposure variations across geographic distributions is unknown increasing the uncertainty associated with the exposure assessment presented in this study.

The use of surrogate receptors to assess exposure may add to uncertainty in the exposure assessment. Aquatic exposures were based on a species sensitivity distribution therefore selection of surrogate receptors was not used. The surrogate approach, however, was used in the assessment of terrestrial ecological receptors. Methods to reduce uncertainty with terrestrial receptor selection included use of two terrestrial food webs in an effort to incorporate different species and feeding patterns, use of lipid and protein content to account for differences amongst species. Sensitivities among life stages were considered in the assessment of human exposures with both a toddler and an adult being evaluated to reduce exposure uncertainty amongst potential wildlands human receptors. Uncertainty in both the terrestrial ecological and human exposure assessments may be reduced by incorporation of site-specific data including observations regarding species present, feeding patterns and consumption rates.

6.2 Summary

A methodology for the derivation of petroleum hydrocarbon criteria for wildlands was developed and applied. Currently environmental criteria do not exist for wild land areas. The intent of the project was to provide ecological populations with a 95% protection level. The goal of the project was to illustrate the execution of a proposed conceptual framework for criteria development and to provide regulators, industry professionals and risk assessors with tools and rationales to manage wildlands. A risk-based approach was applied to derive criteria. However, a review of each regulatory and peer-reviewed data revealed that toxicological reference values associated with 95% protection levels were not available for terrestrial ecological organisms. Therefore, a methodology to derive toxicity reference values for the compounds evaluated in this study was developed and applied.

6.3 Findings

The methodology presented in the study may be used for the purposes of generic regulatory criteria or may be adapted for an area wide or site-specific basis.

The methodology incorporated methods to extrapolate across species and methods to extrapolate across compounds. Both extrapolation techniques, lipid and protein normalization in the case of species to species extrapolation, and use of physical-chemical properties in the case of compound to compound extraction, were presented and applied in an effort to reduce uncertainty in the derivation process. Lipid and protein normalization are applied on the assumption that the biological make-up of all organisms includes the same or similar lipids and proteins. By assuming that chemicals, in particular hydrophobic compounds such as petroleum hydrocarbons, act on the same target site across species, normalizing effects concentrations to an organism's lipid and protein content takes differences in quantity of available target sites into account. The incorporation of these methods was given preference to the use of uncertainty factors on the basis of scientific merit.

A methodology to develop soil criteria based on the protection of terrestrial ecological organisms was derived and executed. Preference was given to use of data that offered a higher degree of protection, that of the no observable adverse effects level

originally intended for use for protection of human populations, over the use of uncertainty factors.

Aquatic criteria were derived for surface water using target lipid models that have previously been validated for protection of 95% of species tested for growth, reproduction and survival endpoints using a species sensitivity distribution. This method was favoured over the use of studies reviewed (e.g. internal body burdens based on lethality) as the target lipid model had been validated for extrapolation across chemicals and was developed to provide the same degree of protection as intended for wildlands criteria.

A methodology to derive tissue residue criteria to protect human populations whom consume aquatic and terrestrial biota from exposed habitats was developed and illustrated.

6.4 Recommendations

6.4.1 Tiered Use

The methodology presented in the study was developed for use on a regional or site-specific basis. The tools and methods presented in the study may be used on a site-specific basis to increase applicability of criteria. The methodology to derive toxicological reference values may be modified to include specific species present on-site or alternatively, criteria may be modified to reduce the receptors evaluated to those present on-site. It is recommended that this option should be exercised when considered appropriate.

6.4.2 Proactive Use, Monitoring and Clean-up Targets

It is recommended that criteria be used as a proactive measure, during resource extraction to support environmental monitoring and to increase protection of wildlands habitats before extraction is complete. Use of criteria for monitoring purposes allows for proactive management and may reduce clean-up efforts post-extraction activity. The incorporation of site-specific information during the monitoring process would increase the relevance of clean-up targets post-resource extraction.

6.4.3 Model Validation

The methodology developed to derive of sediment criteria presented in this study relies on the relationships between surface water and sediment. Validation of the ratios considered in this study would decrease the uncertainty associated with the resulting sediment criteria. It is recommended that these ratios be validated.

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Appendices

Appendix A:

Aquatic Toxicity Reference Value Search Results

Aquatic Toxicity Reference Values Obtained from ECOTOX Database

(U.S. Environmental Protection Agency, 2007)

Benzene
Zooplankton: Insufficient Data
Benthic Invertebrates: Insufficient Data
Forage Fish: Fathead Minnow, *Pimephales promelas* Lowest LOEC: 17 200 µg/L - 7 Day Growth and Survival Endpoint (Marchini, Tosato, Norberg-King, Hammermeister, & Hoglund, 1992) Highest NOEC below LOEC: 10 200 µg/L - 7 Day Growth and Survival Endpoint (Marchini, Tosato, Norberg-King, Hammermeister, & Hoglund, 1992)
Piscivorous Fish: Insufficient Data

Biphenyl
Zooplankton: *Daphnia magna* Lowest LOEC: 330 µg/L - 21 Day Survival Endpoint (Dow Chem. Co., 2002) Highest NOEC below LOEC: 250 µg/L - 2 Day Survival Endpoint (USEPA, 1982)
Benthic Invertebrates: Insufficient Data
Forage Fish: Insufficient Data
Piscivorous Fish: Rainbow Trout, *Oncorhynchus mykiss* Lowest LOEC: 332 µg/L - 87 Day Survival Endpoint (Dow Chem. Co., 2000) Highest NOEC below LOEC: 229 µg/L - 87 Day Survival Endpoint (Dow Chem. Co., 2000)

Ethylbenzene

Zooplankton: Insufficient Data

Benthic Invertebrate: Opossum Shrimp, *Bermudamysis speluncola*
Lowest LOEC: 2700 µg/L - 4 Day Survival Endpoint
(Maten, Boeri, & Walker, 1994)
Highest NOEC below LOEC: 1000 µg/L - 4 Day Survival Endpoint
(Maten, Boeri, & Walker, 1994)

Forage Fish: Insufficient Data

Piscivorous Fish: Atlantic Silverside, *Menidia menidia*
Lowest LOEC: 5900 µg/L - 4 Day Survival Endpoint
(Maten, Boeri, & Walker, 1994)
Highest NOEC below LOEC: 3300 µg/L - 4 Day Survival Endpoint
(Maten, Boeri, & Walker, 1994)

Fluorene

Zooplankton: *Daphnia magna*
Lowest LOEC: 125 µg/L - 14 Day Reproduction Endpoint
(Finger, Little, Henry, Fairchild, & Boyle, 1985)
Highest NOEC below LOEC: 62.5 µg/L - 14 Day Reproduction Endpoint
(Finger, Little, Henry, Fairchild, & Boyle, 1985)

Benthic Invertebrates: Insufficient Data

Forage Fish: Insufficient Data

Piscivorous Fish: Bluegill, *Lepomis macrochirus*
Lowest LOEC: 250 µg/L - 30 Day Growth Endpoint
(Finger, Little, Henry, Fairchild, & Boyle, 1985)
Highest NOEC below LOEC: 125 µg/L - 30 Day Growth Endpoint
(Finger, Little, Henry, Fairchild, & Boyle, 1985)

Naphthalene

Zooplankton: Insufficient Data

Benthic Invertebrates: Insufficient Data

Forage Fish: Insufficient Data

Piscivorous Fish: Coho/Silver Salmon, *Oncorhynchus kisutch*
Lowest LOEC: 3200 µg/L - 3 Day Mortality Endpoint
(Holland, Lasater, Neumann, & Eldridge, 1960)
Highest NOEC below LOEC: 1800 µg/L - 3 Day Mortality Endpoint
(Holland, Lasater, Neumann, & Eldridge, 1960)

Phenanthrene
Zooplankton: *Daphnia pulex* Lowest LOEC: 60 µg/L - 16 Day Reproduction and Growth Endpoint (Savino & Tanabe, 1989)
Zooplankton: *Daphnia magna* Highest NOEC below LOEC: 57 µg/L - 21 Day Growth Endpoint (Call, Brooke, Harting, Poirier, & McCauley, 1986)
Benthic Invertebrate: Polychaete Worm, *Neanthes arenaceodentata* Lowest LOEC: 20 µg/L - 56 Day Reproduction and Growth Endpoint (Emery, 1993) Highest NOEC below LOEC: 10 µg/L - 56 Day Reproduction and Growth Endpoint (Emery, 1993)
Forage Fish: Medaka, High-Eyes, *Oryzias latipes* Lowest LOEC: 200 µg/L - 18 Day Growth Endpoint (Rhodes, Farwell, Hewitt, MacKinnon, & Dixon, 2005) Highest NOEC below LOEC: 100 µg/L - 18 Day Growth Endpoint (Rhodes, Farwell, Hewitt, MacKinnon, & Dixon, 2005)
Piscivorous Fish: Rainbow Trout, *Oncorhynchus mykiss* Lowest LOEC: 8 µg/L - 87 Day Growth and Mortality Endpoint (Call, Brooke, Harting, Poirier, & McCauley, 1986) Highest NOEC below LOEC: 5 µg/L - 87 Day Growth and Mortality Endpoint (Call, Brooke, Harting, Poirier, & McCauley, 1986)

Toluene
Zooplankton: Insufficient Data
Benthic Invertebrates: Insufficient Data
Forage Fish: Fathead Minnow, *Pimephales promelas* Lowest LOEC: 6000 µg/L - 32 Day Growth Endpoint (Devlin, 1982) Highest NOEC below LOEC: 5440 µg/L - 7 Day Growth and Mortality Endpoint (Marchini, Tosato, Norberg-King, Hammermeister, & Hoglund, 1992)
Piscivorous Fish: Insufficient Data

Xylene
Zooplankton: Rotifier, *Brachionus calyciflorus* Lowest LOEC: 40 000 µg/L – 2 Day Reproduction Endpoint (Snell & Moffat, 1992) Highest NOEC below LOEC: 20 000 µg/L – 2 Day Reproduction Endpoint (Snell & Moffat, 1992)
Benthic Invertebrates: Insufficient Data
Forage Fish: Insufficient Data
Piscivorous Fish: Insufficient Data

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Appendix B:

Terrestrial Toxicity Reference Values Search Results

Terrestrial Toxicity Values Obtained from ECOTOX Database

(U.S. Environmental Protection Agency, 2007)

1,2-Dimethylbenzene (o-Xylene)
Norway Rat, *Rattus norvegicus*
Lowest LOAEL: 200 mg/kg-d – 10 Day Growth Endpoint (Condie, Hill, & Borzelleca, 1988)
Highest NOAEL below LOAEL: 10 mg/kg-d – 10 Day Growth Endpoint (Condie, Hill, & Borzelleca, 1988)

1,4-Dimethylbenzene (p-Xylene)
Norway Rat, *Rattus norvegicus*
Lowest LOAEL: 200 mg/kg-d – 10 Day Growth Endpoint (Condie, Hill, & Borzelleca, 1988)
Highest NOAEL below LOAEL: 100 mg/kg-d – 10 Day Growth Endpoint (Condie, Hill, & Borzelleca, 1988)

Fluorene
Earthworm, *Eisenia fetida*
Lowest LOAEL: 750 mg/kg – 56 Day Reproduction Endpoint (Neuhauser & Callahan, 1990)
Highest NOAEL below LOAEL: 500 mg/kg – 56 Day Reproduction Endpoint (Neuhauser & Callahan, 1990)

Napthalene
Earthworm, *Enchytraeus crypticus*
Lowest LOEAL: 2045 µmol/kg-soil – 28 Day Mortality Endpoint (Droge, Paumen, Bleeker, Kraak, & Van Gestel, 2006)
Highest NOEAL below LOEAL: 220 µmol/kg-soil – 28 Day Mortality Endpoint (Droge, Paumen, Bleeker, Kraak, & Van Gestel, 2006)

Terrestrial Toxicity Values Obtained from Total Petroleum Hydrocarbon Criteria Working Group Series

(Edwards, et al., 1997)

Aromatics Compounds

Toluene, C_7

Norway Rat, *Rattus norvegicus*

0.2 mg/kg-d based on a 13-week subchronic study with endpoint effect of weights changes in liver and kidney of male rats. The value was derived from a NOAEL of 223 mg/kg-d with an uncertainty factor of 1000 applied (10 sub-chronic, 10 sensitivity, 10 animal to human).

(National Research Program, 1989)

Ethylbenzene, C_8

Rat, species unknown, original document unavailable

0.1 mg/kg-d based on a 182 day subchronic study with endpoint effect of liver and kidney histopathologic changes. The value was derived from a NOAEL of 97.1 mg/kg-d with an uncertainty factor of 1000 applied (10 sub-chronic, 10 sensitivity, 10 animal to human).

(Wolfe, Rowe, McCollister, Hollingsworth, & Oyen, 1956)

Styrene, C_8

Beagle Dog, *canis lupus familiaris*

0.2 mg/kg-d based on a 560 day subchronic study with an endpoint of decreased hemiglobin and RBC counts and other changes in the blood of specimens. The value was derived from a NOAEL of 200 mg/kg-d with an uncertainty factor of 1000 applied (10 sub-chronic, 10 sensitivity, 10 animal to human).

(Quast, Humiston, & Kalnins, 1979)

Xylene, mixture of m-,o-,p-, C_8

Norway Rat, *Rattus norvegicus*, and House Mouse, *Mus musculus*

2 mg/kg-d based on a 103 week chronic study with an endpoint of mortality in rats and CNS hyperactivity in mice. The value was derived from a NOAEL of 179 mg/kg-d with an uncertainty factor of 100 applied (10 sensitivity, 10 animal to human).

(National Research Program, 1986)

Isopropylbenzene C_9

Wistar Albino Rat, *Rattus norvegicus*

0.04 mg/kg-d based on a 194 day subchronic study with an endpoint of increased kidney weight. The value was derived from a NOAEL of 110 mg/kg-d with an uncertainty factor of 3000 applied (3 inadequate database, 10 subchronic, 10 sensitivity, 10 animal to human).

(Wolfe, Rowe, McCollister, Hollingsworth, & Oyen, 1956)

Naphthalene C_{10}

Rat, *species unknown,* unpublished study

0.04 mg/kg-d based on a 13 week subchronic study with an endpoint of decreased body weight. The value was derived from a NOAEL of 35.7 mg/kg-d with an uncertainty factor of 1000 applied (10 subchronic, 10 sensitivity, 10 animal to human).

(National Research Program, 1980)

Methylnapthalene C_{11}

House Mouse, *Mus musculus*

0.06 mg/kg-d based on a 90 day subchronic study with an endpoint of changes in liver weight. The value was derived from a NOAEL of 175 mg/kg-d with an uncertainty factor of 3000 applied (3 inadequate database, 10 subchronic, 10 sensitivity, 10 animal to human).

(Hazelton Laboratories Inc., 1989)

Biphenyl C_{12}

Weanling Albino Rat, *Rattus norvegicus*

0.05 mg/kg-d based on a chronic study of unknown duration with an endpoint of damage to the kidney, reduced hemoglobin and decreased food intake. The value was derived from a NOAEL of 50 mg/kg-d with an uncertainty factor of 100 (10 sensitivity, 10 animal to human) and a modifying factor of 10 (NOAEL derivation from percentage in diet).

(Ambrose, Booth, & Cox, 1960)

Flourene C_{13}

House Mouse, *Mus musculus*

0.04 mg/kg-d based on a 13 week subchronic study with an endpoint of histopathological increases in the liver and the spleen. The value was derived from a NOAEL of 125 mg/kg- with an uncertainty factor of 1000 (10 subchronic, 10 sensitivity, 10 animal to human) and a modifying factor of 3 (inadequate toxicity data for reproduction and development endpoints).

(Toxicity Research Laboratories, 1989)

Anthracene C_{14}

House Mouse, *Mus musculus*

0.3 mg/kg-d based on a 90 day subchronic study with an endpoint of various mortality, growth and food intake endpoint evaluated, none observed that were believed to be associated with dosing. The value was derived from a NOAEL of 1000 mg/kg-d with an uncertainty factor of 3000 applied (3 inadequate database, 10 subchronic, 10 sensitivity, 10 animal to human).

(Hazelton Laboratories America Inc., 1989)

Fluoranthene C_{16}

House Mouse, *Mus musculus*

0.04 mg/kg-d based on a 13 week subchronic study with an endpoint of increased body weight and food intake, changes in SGPT, kidney and liver pathology and hematological changes. The value was derived from a NOAEL of 125 mg/kg-d with an uncertainty factor of 3000 applied (3 inadequate database, 10 subchronic, 10 sensitivity, 10 animal to human).

(Toxicity Research Laboratories Ltd., 1988)

Pyrene C_{16}

House Mouse, *Mus musculus*

0.03 mg/kg-d based on a 13 week subchronic study with an endpoint of kidney changes including decreased kidney weight and renal tubular pathological changes. The value was derived from a 75 mg/kg-d with an uncertainty factor of 1000 applied (10 subchronic, 10 sensitivity, 10 animal to human) and a modifying factor of 3 applied (3 inadequate database).

(Toxicity Research Laboratories, 1989)

Fractions, $C_{>16-21}$

Oral reference doses were not available, therefore, Edwards, et al., 1997 suggest using the pyrene data, summarized above, as a surrogate. The value of 0.03 mg/kg-d is considered to be conservative pyrene has a lower carbon fraction [and therefore is considered to be more volatile] than compounds within this carbon range (Edwards, et al., 1997).

Aliphatic Compounds

n-hexane, C_6, commercial

Rat and mice, *species unknown*

5 mg/kg-d based on a chronic inhalation study of unknown duration for the endpoint of neurotoxicity, reproduction and development

Value based on a NOAEL of 1840 mg/m3 with an uncertainty factor of 100 (10 sensitivity/intraspecies variation, 10 animal to human) obtained by Edwards, et al., 1997 using multiple studies. The value was converted from an RfC to an RfD using the assumption of 70 kg body weight and 20 m3/d inhalation rate.

Resulting NOAEL = 526 mg/kg-d

(Kelley, Duffy, Daughtrey, Keenan, Newton, & Rhoden, 1994), (Daughtrey, et al., 1994), (Keenan, Neeper-Bradley, Dodd, Kirwin, Duffy, & Soiefer, 1991)

n-heptane, C_6

Human subjects

2 mg/kg-d based on an n-hexane RfC value. Edwards, et al., 1997 evauated multiple n-heptane studies and concluded that n-heptane toxicity was approximatley 38 times less than that found in a n-hexane RfD of 0.06 mg/kg/d

NOAEL = 526 mg/kg-d (n-hexane)*0.38 = 200 mg/kg-d

The n-hexane value was based on a neurotoxic endpoint in humans evaluated from the n-heptane metabolite gamma diketone 2,5-heptane-dione and the n-hexane metabolite gamma diketone 2,5-heptane-dione which are believed to produce neurotoxic effects in humans

(Edwards, et al., 1997)

Fractions, C_{5-8}

5 mg/kg-d was determined to be a suitable RfD by Edwards, et al., 1997 based on the n-hexane value summarized above using the rationale of conservatism in in RfD derivation and the composition of n-hexane in total petroluem hydrocarbon mixtures being less than 53% of that found in commercial n-hexane

(Edwards, et al., 1997)

Fractions, C_{9-12}

Sprague Dawley Norway Rats, *Rattus norvegicus*

0.1 mg/kg-d based on a 90 day subchronic study with an endpoint of decreased body weight, increased food intake, irritation of GI tract and increased enzyme levels (alanine aminotransferase and glutamyl trans- ferase). The value was derived from a LOAEL of 500 mg/kg-d with an uncertainty factor of 5000 applied (5 LOAEL to NOAEL, 10 subchronic, 10 sensitivity and 10 animal to human).

Unpublished data reported by Edwards, et al., 1997

Fractions, C_{10-13}

Sprague Dawley Norway Rats, *Rattus norvegicus*

0.1 mg/kg-d based on a 13 week subchronic study with an endpoint of increased liver weight. The value was derived from a NOAEL of 100 mg/kg-d with an uncertainty factor of 1000 applied (10 subchronic, 10 sensitivity, 10 animal to human).

Unpublished data reported by Edwards, et al., 1997

Fractions, C_{11-17}, isoparaffinic solvent composed of 22% naphthenes; and <0.05% aromatics

Rats, species unknown

0.1 mg/kg-d based on a 90 day subchronic study with an endpoint of increased liver weight. The value was derived from a NOAEL of 100 mg/kg-d with an uncertainty factor of 1000 applied (10 subchronic, 10 sensitivity, 10 animal to human).

Unpublished data reported by Edwards, et al., 1997

Terrestrial Toxicity Values Obtained from IRIS Database

(United States Environmental Protection Agency, 2010)

Benzene

Human subjects

0.004 mg/kg-d based on a epidemiological study with 0.7-16 years (mean exposure 6.3 years with standard deviation of 4.4 years) with the endpoint of haematological changes including red and white blood cell counts, hematocrit, ALC, platelet count, and absolute lymphocyte count. The value was derived from a BMDL of 1.2 mg/kg-d with an uncertainty factor of 300 applied (3 inadequate database, 3 effect level extrapolation, 3 subchronic, 10 sensitivity).

(Rothman, et al., 1996)

Biphenyl

Weanling Albino Rat, *Rattus norvegicus*

0.05 mg/kg-d based on a chronic study of unknown duration with an endpoint of damage to the kidney, reduced hemoglobin and decreased food intake. The value was derived from a NOAEL of 50 mg/kg-d with an uncertainty factor of 100 (10 sensitivity, 10 animal to human) and a modifying factor of 10 (NOAEL derivation from percentage in diet).

(Ambrose, Booth, & Cox, 1960)

Ethylbenzene

Wistar Albino Rat, *Rattus norvegicus*

0.1 mg/kg-d based on a 182 day subchronic study with an endpoint of liver and kidney changes. The value was derived from a NOAEL of 136 mg/kg-d with an uncertainty factor of 1000 applied (10 sensitivity, 10 subchronic and 10 animal to human).

(Wolfe, Rowe, McCollister, Hollingsworth, & Oyen, 1956)

Fluorene

House Mouse, *Mus musculus*

0.04 mg/kg-d based on a 13 week subchronic study with the endpoint of decreased red blood count, packed cell volume and hemoglobin. The value was derived from a NOAEL of 125 mg/kg-d with an uncertainty factor of 3000 applied (3 inadequate database, 10 sensitivity, 10 subchronic, 10 animal to human).

(Toxicity Research Laboratories, 1989)

Napthalene

Fisher 344 Rat, *Rattus norvegicus*

0.02 mg/kg-d based on a 13 week subchronic study with an endpoint of decreased body weight. The value was derived from a NOAEL of 71 mg/kg-d with an uncertainty factor of 3000 applied (3 inadequate database, 10 sensitivity, 10 subchronic, 10 animal to human).

Unpublished data (Battelle's Columbus Laboratories, 1980)

Toluene

Fisher 344 Rat, *Rattus norvegicus*

0.08 mg/kg-d based on a 13 week subchronic study with the endpoint of increased kidney weight. The value was derived from a BMDL of 238

mg/kg-d with an uncertainty factor of 3000 applied (3 inadequate database, 10 sensitivity, 10 subchronic, 10 animal to human).

(National Research Program, 1990)

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Appendix C:

EcoTox Search Strategy

Toxicity data was obtained from the US EPA EcoTox database (U.S. Environmental Protection Agency, 2007). The EcoTox database contains peer-reviewed toxicity data for aquatic and terrestrial life acquired by literature searches preformed by the US EPA Mid-Continent Ecology Division that is updated quarterly (U.S. Environmental Protection Agency, 2007). The database was used to ensure that relevant available data was captured for data selection.

An advanced database query was preformed for each substance by both chemical name and by CASN. Relevant animals groupings were selected (crustaceans, molluscs, other invertebrates and fish for aquatic searches and mammals, birds, worms and other invertebrates for terrestrial searches) for each search. Endpoints selected included LC/LD and EC/ED (all % values) were selected in addition to LOEC, NOEC, LOEL, NOEL and MATC endpoints. Effects measurements included the growth (development and growth), reproduction (reproduction and avian/reptile egg) and mortality group; the morphology category in the growth group was not selected, as it was not considered a critical endpoint for growth based on adverse effects due to morphology effects being accounted for in the growth endpoint.

Behavioural, biochemical, and cellular group endpoints were not selected as either cause or effect relationships cannot be established between these endpoints and the critical (growth, reproduction and mortality) endpoints. If sufficient data becomes available linking these endpoints, they should be included in future studies and if possible, pooled together to determine sensitivity distributions for trophic levels. As this information was considered insufficient at the current time, it was not taken into consideration in determination of toxicological effects endpoints values.

Appendix D:

Fugacity Model Parameters

Values used in the fugacity model are based on assumptions made in Taylor, 2010 and from values obtained from the ChemCan Version 6.00 for Northern British Columbia as presented by Taylor, 2010. The input parameters presented in the Taylor (2010) model are as follows:

Table D1 ***Primary Environmental Compartment (from Taylor, 2010)***

Compartment	Value	Units	Value entered or calculated by model
Total surface area	3.17E+08	m^2	ENTER
Surface covered by water	2.10E+00	% of total	ENTER
Water surface area	6.65E+06	m^2	CALCULATED
Water depth	2.00E+01	m	ENTER
Water volume	1.33E+08	m^3	CALCULATED
Ave air height	2.00E+03	m	ENTER
Air surface area	3.17E+08	m^2	CALCULATED
Air volume	6.33E+11	m^3	CALCULATED
Groundwater depth	0.00E+00	m	ENTER
Soil depth	1.00E-01	m	ENTER
Soil area	3.10E+08	m^2	CALCULATED
Soil volume	3.10E+07	m^3	CALCULATED
Sediment depth	1.00E-02	m	ENTER
Sediment surface area	6.65E+06	m^2	CALCULATED
Sediment Volume	6.65E+04	m^3	CALCULATED

Table D2 ***Water Compartment Specific Parameters (from Taylor, 2010)***

Parameter	Value	Units	Value entered or calculated by model
water in- and out-flow	9.60E+04	L/day	ENTER
dissolved oxygen saturation	9.00E-01	%	ENTER
Disequilibrium factor POC	1.00E+00		ENTER
Disequilibrium factor DOC (DOC)	1.00E+00		ENTER
POC-octanol proportionality constant (alphapoc)	3.50E-01		ENTER
DOC-octanol proportionality constant (alphadoc)	8.00E-02		ENTER
pH of water	7.00E+00		ENTER
Sediment OC octanol proportionality constant	3.50E-01		ENTER
Concentration of particulate organic carbon (Xpoc)	4.80E-01	kg/L	ENTER

Table D3 ***Volume Fractions (from Taylor, 2010)***

Volume Fractions	Value	Units	Value entered or calculated by model
particles in air	2.00E-11	NA	ENTER
particles in water	5.00E-06	NA	ENTER
air in soil	2.00E-01	NA	ENTER
water in soil	3.00E-01	NA	ENTER
soil solids	5.00E-01	NA	ENTER
sediment pore water	7.00E-01	NA	ENTER
sediment solids	3.00E-01	NA	ENTER
air-vapour	1.00E+00	NA	CALCULATED
water-liquid	1.00E+00	NA	CALCULATED

Table D4 *Sub-compartment Values (from Taylor, 2010)*

Sub-compartment volumes	Value	Units	Value entered or calculated by model
air-vapour	6.33E+11	m^3	CALCULATED
air-solid	1.27E+01	m^3	CALCULATED
water-liquid	1.33E+08	m^3	CALCULATED
water-solid	6.65E+02	m^3	CALCULATED
soil-vapour	6.20E+06	m^3	CALCULATED
soil-liquid	9.30E+06	m^3	CALCULATED
soil-solid	1.55E+07	m^3	CALCULATED
sediment-liquid	4.65E+04	m^3	CALCULATED
sediment-solid	1.99E+04	m^3	CALCULATED

Table D5 *Densities (from Taylor, 2010)*

Densities	Value	Units	Value entered or calculated by model
Air-air	1.29E+00	kg/m^3	CALCULATED
Air-aerosol	2.40E+03	kg/m^3	ENTER
Water-water	1.00E+03	kg/m^3	ENTER
DOC in water (dDOC)	1.20E-06	kg/L	ENTER
Water-sus.particles	2.40E+03	kg/m^3	ENTER
Water-sus.particles	2.40E+00	kg/L	CALCULATED
Soil-air	1.29E+00	kg/m^3	CALCULATED
Soil-water	1.00E+03	kg/m^3	ENTER
Soil-solid	2.40E+03	kg/m^3	ENTER
Sediment-water	1.00E+03	kg/m^3	ENTER
Sediment-solid	2.40E+03	kg/m^3	ENTER
Sediment-solid	2.40E+00	kg/L	CALCULATED
Organic Carbon	2.00E+00	kg/L	ENTER
Air-bulk	1.29E+00	kg/m^3	CALCULATED
Water-bulk	1.00E+03	kg/m^3	CALCULATED
Soil-bulk	1.50E+03	kg/m^3	CALCULATED
Sediment-bulk	1.42E+03	kg/m^3	CALCULATED
Groundwater-bulk	1.00E+03	kg/m^3	CALCULATED

Table D6 ***Temperatures (from Taylor, 2010)***

Temperature Conditions	Value	Units	Value entered or calculated by model
Environmental Temperature (envTc)	9.00E-01	deg C	ENTER
Environmental Temperature (envTk)	2.74E+02	deg K	CALCULATED

Table D7 ***Organic Carbon Fractions (from Taylor, 2010)***

Organic Carbon Fraction	Value	Units	Value entered or calculated by model
Particles in water	2.00E-01	g/g	ENTER
soil solids	2.00E-02	g/g	ENTER
sediment solids	4.00E-02	g/g	ENTER

Table D8 ***Bioconcentration Factors at Freely Dissolved Steady-State for Piscivorous Fish (from Taylor, 2010)***

Parameter	BCF (Freely dissolved at steady-state)
Units	L/kg
Parameter Name	BCF
CRUDE OIL	
Straight Chain Alkanes	
n-Hexane	2.4E+02
n-Heptane	5.3E+02
n-Octane	8.5E+02
n-Nonane	1.2E+03
n-Decane	7.6E+02
n-Undecane	1.2E+03
n-Dodecane	1.6E+03

Parameter	BCF (Freely dissolved at steady-state)
Branched Chain Alkanes	
2,2-Dimethylbutane	2.5E+02
2,3-Dimethylbutane	1.2E+02
2-Methylpentane	1.5E+02
3-Methylpentane	1.6E+02
3-Ethylpentane	1.8E+02
2,4-Dimethylpentane	1.6E+02
2,3-Dimethylpentane	1.6E+02
2,2,4-Trimethylpentane	4.6E+02
2,3,3-Trimethylpentane	3.4E+02
2,3,4-Trimethylpentane	2.7E+02
2-Methyl-3-ethylpentane	2.9E+02
2-Methylhexane	1.9E+02
3-Methylhexane	1.8E+02
2,2-Dimethylhexane	3.9E+02
2,3-Dimethylhexane	2.9E+02
2,4-Dimethylhexane	2.9E+02
2,5-Dimethylhexane	2.9E+02
3,3-Dimethylhexane	3.7E+02
2,3-Dimethylheptane	4.9E+02
2,6-Dimethylheptane	4.6E+02
2-Methyloctane	5.2E+02
3-Methyloctane	5.2E+02
4-Methyloctane	5.2E+02
Cycloalkanes	
Cyclopentane	5.8E+01
Methylcyclopentane	1.2E+02
1,1-Dimethylcyclopentane	1.4E+02
1-trans-2-Dimethylcyclopentane	1.7E+02
1-cis-3-Dimethylcyclopentane	1.7E+02
1-trans-3-Dimethylcyclopentane	1.7E+02
1,1,2-Trimethylcyclopentane	2.9E+02
1,1,3-Trimethylcyclopentane	2.9E+02
1-trans-2-cis-3-Trimethylcyclopentane	3.8E+02
1-trans-2-cis-4-Trimethylcyclopentane	3.8E+02
1-trans-2-Dimethylcyclohexane	3.8E+02
Ethylcyclohexane	5.7E+02
Cyclohexane	1.3E+02
1-trans-2-trans-4-Trimethylcyclohexane	5.8E+02
Alkyl Benzenes	
Benzene	1.3E+01
Toluene	3.2E+01

Parameter	BCF (Freely dissolved at steady-state)
Ethylbenzene	6.0E+01
o-Xylene	8.8E+01
m-Xylene	1.0E+02
p-Xylene	9.3E+01
1-Methyl-4-ethylbenzene	2.0E+02
1-Methyl-2-ethylbenzene	1.7E+02
1-Methyl-3-ethylbenzene	2.9E+02
1,2,3-Trimethylbenzene	1.6E+02
1,2,4-Trimethylbenzene	1.7E+02
1,3,5-Trimethylbenzene	1.2E+02
1,2,3,4-Tetramethylbenzene	1.7E+02
Biphenyl	3.4E+02
Naphtheno-Benzenes	
Indane	7.7E+01
Tetralin (tetrahydronaphthalene)	2.8E+02
5-Methyltetrahydronaphthalene	5.2E+02
6-Methyltetrahydronaphthalene	5.2E+02
Fluorene	3.3E+02
Alkyl Naphthalenes	
Naphthalene	1.7E+02
Polynuclear Aromatics	
Phenanthrene	7.6E-01

Table D9 ***Dietary Chemical Uptake Efficiencies based on Kelley, Gobas and McLachlan (2004) (from Taylor, 2010)***

Parameter	Dietary chemical uptake efficiency (unitless)
CRUDE OIL	
Straight Chain Alkanes	
n-Hexane	8.3E-01
n-Heptane	8.3E-01
n-Octane	8.3E-01
n-Nonane	8.3E-01
n-Decane	8.1E-01
n-Undecane	7.9E-01
n-Dodecane	7.5E-01
Branched Chain Alkanes	

Parameter	Dietary chemical uptake efficiency (unitless)
2,2-Dimethylbutane	8.3E-01
2,3-Dimethylbutane	8.3E-01
2-Methylpentane	8.3E-01
3-Methylpentane	8.3E-01
3-Ethylpentane	8.3E-01
2,4-Dimethylpentane	8.3E-01
2,3-Dimethylpentane	8.3E-01
2,2,4-Trimethylpentane	8.3E-01
2,3,3-Trimethylpentane	8.3E-01
2,3,4-Trimethylpentane	8.3E-01
2-Methyl-3-ethylpentane	8.3E-01
2-Methylhexane	8.3E-01
3-Methylhexane	8.3E-01
2,2-Dimethylhexane	8.3E-01
2,3-Dimethylhexane	8.3E-01
2,4-Dimethylhexane	8.3E-01
2,5-Dimethylhexane	8.3E-01
3,3-Dimethylhexane	8.3E-01
2,3-Dimethylheptane	8.3E-01
2,6-Dimethylheptane	8.3E-01
2-Methyloctane	8.3E-01
3-Methyloctane	8.3E-01
4-Methyloctane	8.3E-01
Cycloalkanes	
Cyclopentane	8.3E-01
Methylcyclopentane	8.3E-01
1,1-Dimethylcyclopentane	8.3E-01
1-trans-2-Dimethylcyclopentane	8.3E-01
1-cis-3-Dimethylcyclopentane	8.3E-01
1-trans-3-Dimethylcyclopentane	8.3E-01
1,1,2-Trimethylcyclopentane	8.3E-01
1,1,3-Trimethylcyclopentane	8.3E-01
1-trans-2-cis-3-Trimethylcyclopentane	8.3E-01
1-trans-2-cis-4-Trimethylcyclopentane	8.3E-01
1-trans-2-Dimethylcyclohexane	8.3E-01
Ethylcyclohexane	8.3E-01
Cyclohexane	8.3E-01
1-trans-2-trans-4-Trimethylcyclohexane	8.3E-01
Alkyl Benzenes	
Benzene	8.3E-01
Toluene	8.3E-01
Ethylbenzene	8.3E-01
o-Xylene	8.3E-01

Parameter	Dietary chemical uptake efficiency (unitless)
m-Xylene	8.3E-01
p-Xylene	8.3E-01
1-Methyl-4-ethylbenzene	8.3E-01
1-Methyl-2-ethylbenzene	8.3E-01
1-Methyl-3-ethylbenzene	8.3E-01
1,2,3-Trimethylbenzene	8.3E-01
1,2,4-Trimethylbenzene	8.3E-01
1,3,5-Trimethylbenzene	8.3E-01
1,2,3,4-Tetramethylbenzene	8.3E-01
Biphenyl	8.3E-01
Naphtheno-Benzenes	
Indane	8.3E-01
Tetralin (tetrahydronaphthalene)	8.3E-01
5-Methyltetrahydronaphthalene	8.3E-01
6-Methyltetrahydronaphthalene	8.3E-01
Fluorene	8.3E-01
Alkyl Naphthalenes	
Naphthalene	8.3E-01
Polynuclear Aromatics	
Phenanthrene	8.3E-01

Table D10 ***Sediment to Surface Water Ratios used to determine Sediment Criteria (from Taylor, 2010)***

Substance	Csediment / Cwater
CRUDE OIL	
Straight Chain Alkanes	
n-Hexane	4.23E+02
n-Heptane	2.94E+03
n-Octane	5.77E+04
n-Nonane	1.40E+04
n-Decane	3.29E+04
n-Undecane	3.66E+04
n-Dodecane	6.44E+04
Branched Chain Alkanes	
2,2-Dimethylbutane	3.45E+02
2,3-Dimethylbutane	1.28E+02
2-Methylpentane	2.82E+02

Substance	Csediment / Cwater
3-Methylpentane	1.99E+02
3-Ethylpentane	2.62E+02
2,4-Dimethylpentane	2.14E+02
2,3-Dimethylpentane	2.14E+02
2,2,4-Trimethylpentane	5.25E+04
2,3,3-Trimethylpentane	6.91E+02
2,3,4-Trimethylpentane	6.23E+02
2-Methyl-3-ethylpentane	7.47E+02
2-Methylhexane	2.62E+02
3-Methylhexane	2.62E+02
2,2-Dimethylhexane	8.29E+02
2,3-Dimethylhexane	7.47E+02
2,4-Dimethylhexane	7.47E+02
2,5-Dimethylhexane	7.47E+02
3,3-Dimethylhexane	8.28E+02
2,3-Dimethylheptane	2.60E+03
2,6-Dimethylheptane	2.60E+03
2-Methyloctane	3.16E+03
3-Methyloctane	3.16E+03
4-Methyloctane	3.16E+03
Cycloalkanes	
Cyclopentane	4.67E+01
Methylcyclopentane	1.13E+02
1,1-Dimethylcyclopentane	1.78E+02
1-trans-2-Dimethylcyclopentane	1.61E+02
1-cis-3-Dimethylcyclopentane	1.61E+02
1-trans-3-Dimethylcyclopentane	1.61E+02
1,1,2-Trimethylcyclopentane	5.03E+02
1,1,3-Trimethylcyclopentane	5.03E+02
1-trans-2-cis-3-Trimethylcyclopentane	4.66E+02
1-trans-2-cis-4-Trimethylcyclopentane	4.66E+02
1-trans-2-Dimethylcyclohexane	5.57E+02
Ethylcyclohexane	2.28E+03
Cyclohexane	1.39E+02
1-trans-2-trans-4-Trimethylcyclohexane	1.32E+03
Alkyl Benzenes	
Benzene	2.27E+02
Toluene	1.14E+03
Ethylbenzene	2.63E+03
o-Xylene	2.80E+03
m-Xylene	1.94E+02
p-Xylene	7.93E+01
1-Methyl-4-ethylbenzene	1.45E+02
1-Methyl-2-ethylbenzene	1.75E+03
1-Methyl-3-ethylbenzene	3.32E+02
1,2,3-Trimethylbenzene	1.60E+02
1,2,4-Trimethylbenzene	2.99E+02

Substance	Csediment / Cwater
1,3,5-Trimethylbenzene	2.59E+02
1,2,3,4-Tetramethylbenzene	3.71E+02
Biphenyl	9.08E+02
Naphtheno-Benzenes	
Indane	5.17E+01
Tetralin (tetrahydronaphthalene)	2.18E+02
5-Methyltetrahydronaphthalene	1.62E+03
6-Methyltetrahydronaphthalene	1.62E+03
Fluorene	7.22E+02
Alkyl Naphthalenes	
Naphthalene	1.42E+02
Polynuclear Aromatics	
Phenanthrene	7.63E+01

Appendix E:

Models used in Criteria Derivation

Models attached in electronic format.