Final Screening Assessment
Petroleum Sector Stream Approach

Low Boiling Point Naphthas
[Industry-Restricted]

Chemical Abstracts Service Registry Numbers

64741-42-0
64741-69-1
64741-78-2

Environment Canada
Health Canada

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Synopsis

The Ministers of the Environment and of Health have conducted a screening assessment of the following industry-restricted low boiling point naphthas (LBPNs):

CAS RN[a]	DSL[b] name
64741-42-0	Naphtha (petroleum), full-range straight-run
64741-69-1	Naphtha (petroleum), light hydrocracked
64741-78-2	Naphtha (petroleum), heavy hydrocracked

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[b] DSL, Domestic Substances List.

These substances were identified as high priorities for action during the categorization of substances on the Domestic Substances List (DSL) as they were determined to present greatest potential or intermediate potential for exposure of individuals in Canada, and were considered to present a high hazard to human health. These substances met the ecological categorization criteria for persistence or bioaccumulation potential and inherent toxicity to aquatic organisms. These substances were included in the Petroleum Sector Stream Approach (PSSA) because they are related to the petroleum sector and are considered to be of Unknown or Variable composition, Complex reaction products or Biological materials (UVCBs).

These LBPNs are complex combinations of petroleum hydrocarbons that serve as blending constituents in final gasoline products, as intermediate products of distillation or as residues derived from distillation or extraction units. The final fuel products usually consist of a combination of LBPNs, as well as high-quality hydrocarbons that have been produced from refinery or upgrader facilities. In order to predict the overall behaviour of these complex substances for the purposes of assessing the potential for ecological effects, representative structures have been selected from each chemical class in the substances.

The LBPNs considered in this screening assessment (CAS RNs 64741-42-0, 64741-69-1 and 64741-78-2) have been identified as industry restricted (i.e., they are a subset of LBPNs that may leave a petroleum sector facility and be transported to other industrial facilities). According to information submitted under section 71 of the *Canadian Environmental Protection Act, 1999* (CEPA 1999) and other sources of information, these LBPNs are transported from petroleum sector facilities to other industrial facilities by ship and truck.

Based on results of comparison of levels expected to cause harm to organisms with estimated exposure levels, releases to soil from loading, unloading and transport may cause harm to terrestrial organisms. However, given the conditions of such releases and the overall low frequency of spills to land, the risk of harm to the environment from these spills is low. As well, there is a low risk of harm to terrestrial organisms from releases of

LBPNs to air from ship and truck loading. As there were no recorded spills to marine waters during transport, these LBPNs have a low risk of harm to aquatic organisms.

Based on the information presented in this screening assessment on the frequency and magnitude of spills, there is low risk of harm to organisms or the broader integrity of the environment from these substances. It is concluded that the three industry-restricted LBPNs (CAS RNs 64741-42-0, 64741-69-1 and 64741-78-2) do not meet the criteria under paragraph 64(*a*) or 64(*b*) of the *Canadian Environmental Protection Act, 1999* (CEPA 1999) as they are not entering the environment in a quantity or concentration or under conditions that have or may have an immediate or long-term harmful effect on the environment or its biological diversity or that constitute or may constitute a danger to the environment on which life depends.

A critical effect for the initial categorization of industry-restricted LBPNs was carcinogenicity, based primarily on classifications by international agencies. Additionally, benzene, a component of LBPNs, has been identified by Health Canada and several international regulatory agencies as a carcinogen. In addition to LBPN-specific data, benzene was selected as a high-hazard component of LBPNs to characterize potential exposure and risk to the general population from evaporative emissions of LBPNs. Several studies also confirmed skin tumour development in mice following repeated dermal application of LBPNs. LBPNs demonstrated limited evidence of genotoxicity in *in vivo* and *in vitro* assays, as well as limited potential to adversely affect reproduction and development. Information on additional LBPNs in the PSSA that are similar from a processing and a physical-chemical perspective was considered for characterization of human health effects.

The physical-chemical properties of LBPNs indicate that these substances contain highly volatile components. Potential exposure to the industry-restricted LBPNs for individuals in proximity to shipping and trucking corridors results primarily from inhalation of vapours in ambient air due to evaporative emissions during transportation. The margins between the upper-bounding estimates of exposure to maximum air concentrations of total volatile organic compounds (VOCs) or benzene, based on the aromatic fraction of the LBPNs, and the critical inhalation effect levels are considered to be conservative and adequately protective to account for uncertainties related to health effects and exposure. General population exposure to industry-restricted LBPNs via the dermal and oral routes is not expected; therefore, risk to human health from the industry-restricted LBPNs via these routes is not expected.

Based on information presented in this screening assessment, it is concluded that the industry-restricted LBPNs (CAS RNs 64741-42-0, 64741-69-1 and 64741-78-2) do not meet the criteria under paragraph 64(*c*) of the *Canadian Environmental Protection Act, 1999* (CEPA 1999) as they are not entering the environment in a quantity or concentration or under conditions that constitute or may constitute a danger in Canada to human life or health.

It is therefore concluded that the three industry-restricted LBPNs listed under CAS RNs 64741-42-0, 64741-69-1 and 64741-78-2 do not meet any of the criteria set out in section 64 of CEPA 1999.

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Introduction

The *Canadian Environmental Protection Act, 1999* (CEPA 1999) (Canada 1999) requires the Minister of the Environment and the Minister of Health to conduct screening assessments of substances that have met the categorization criteria set out in the Act to determine whether these substances present or may present a risk to the environment or to human health.

Based on the information obtained through the categorization process, the Ministers identified a number of substances as high priorities for action. These include substances that

- met all of the ecological categorization criteria, including persistence (P), bioaccumulation potential (B) and inherent toxicity to aquatic organisms (iT), and were believed to be in commerce in Canada; and/or
- met the categorization criteria for greatest potential for exposure (GPE) or intermediate potential for exposure (IPE) and had been identified as posing a high hazard to human health based on classifications by other national or international agencies for carcinogenicity, genotoxicity, developmental toxicity or reproductive toxicity.

A key element of the Government of Canada's Chemicals Management Plan is the Petroleum Sector Stream Approach (PSSA), which involves the assessment of approximately 160 petroleum substances that are considered high priorities for action. These substances are primarily related to the petroleum sector and are considered to be of Unknown or Variable composition, Complex reaction products or Biological materials (UVCBs).

Screening assessments focus on information critical to determining whether a substance meets the criteria set out in section 64 of CEPA 1999. Screening assessments examine scientific information and develop conclusions by incorporating a weight-of-evidence approach and precaution.[1]

Grouping of Petroleum Substances

The high priority petroleum substances fall into nine groups of substances (Table A1.1 in Appendix 1) based on similarities in production, toxicity and physical-chemical

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[1] A determination of whether one or more of the criteria of section 64 are met is based upon an assessment of potential risks to the environment and/or to human health associated with exposures in the general environment. For humans, this includes, but is not limited to, exposures from ambient and indoor air, drinking water, foodstuffs and the use of consumer products. A conclusion under CEPA 1999 on the petroleum substances in the Chemicals Management Plan is not relevant to, nor does it preclude, an assessment against the hazard criteria specified in the *Controlled Products Regulations*, which are part of the regulatory framework for the Workplace Hazardous Materials Information System for products intended for workplace use. Similarly, a conclusion based on the criteria contained in section 64 of CEPA 1999 does not preclude actions being undertaken in other sections of CEPA 1999 or other Acts.

properties. In order to conduct the screening assessments, each high priority petroleum substance was placed into one of five categories (“streams”) depending on its production and uses in Canada:

> Stream 0: substances not produced by the petroleum sector and/or not in commerce;
> Stream 1: site-restricted substances, which are substances that are not expected to be transported off refinery, upgrader or natural gas processing facility sites;[2]
> Stream 2: industry-restricted substances, which are substances that may leave a petroleum sector facility and be transported to other industrial facilities (e.g., for use as a feedstock, fuel or blending component), but do not reach the public market in the form originally acquired;
> Stream 3: substances that are primarily used by industries and consumers as fuels;
> Stream 4: substances that may be present in products available to the consumer.

An analysis of the available data determined that 16 petroleum substances are evaluated under Stream 2, as described above. These occur within five of the nine substance groups: heavy fuel oils, gas oils, petroleum and refinery gases, low boiling point naphthas (LBPNs) and crude oils.

This screening assessment addresses three industry-restricted LBPNs described under Chemical Abstracts Service Registry Numbers (CAS RNs) 64741-42-0, 64741-69-1 and 64741-78-2. These substances were identified as GPE or IPE during the categorization exercise, and were considered to present a high hazard to human health. These substances met the ecological categorization criteria for persistence or bioaccumulation potential and inherent toxicity to aquatic organisms. According to information submitted under section 71 of CEPA 1999 (Environment Canada 2008, 2009), these substances can be consumed on-site or be transported from refineries and upgraders to other industrial facilities, but they are not sold directly to consumers. These substances were included in the PSSA because they are related to the petroleum sector and are all complex combinations of petroleum hydrocarbons.

Twenty site-restricted LBPNs were previously assessed under Stream 1, and an additional ten LBPNs are being assessed separately, as they belong to Streams 3 and 4 (as described above). The health effects of the industry-restricted LBPNs were assessed using health effects data pooled across all high priority LBPNs due to insufficient data specific to the industry-restricted LBPNs.

Included in this screening assessment is the consideration of information on chemical properties, uses, exposure and effects, including information submitted under section 71 of CEPA 1999 and voluntary submission of information from industry. Data relevant to the screening assessment of these substances were identified in original literature, review and assessment documents and stakeholder research reports and from recent literature searches, up to March 2010 for the human exposure and environmental sections of the

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[2] For the purposes of the screening assessment of PSSA substances, a site is defined as the boundaries of the property where a facility is located.

document and up to September 2011 for the health effects section of the document. Key studies were critically evaluated, and modelling results were used to reach conclusions.

Characterization of risk to the environment involves the consideration of data relevant to environmental behaviour, persistence, bioaccumulation and toxicity, combined with an estimation of exposure of potentially affected non-human organisms from the major sources of release to the environment. To predict the overall environmental behaviour and properties of complex substances such as these industry-restricted LBPNs, representative structures were selected from each chemical class contained within the substances. Conclusions regarding risk to the environment are based on an estimation of environmental concentrations resulting from releases and the potential for these concentrations to have a negative impact on non-human organisms. As well, other lines of evidence including fate, temporal/spatial presence in the environment and hazardous properties of the substance are taken into account. The ecological portion of the screening assessment summarizes the most pertinent data on environmental behaviour and effects and does not represent an exhaustive or critical review of all available data. Environmental models and comparisons with similar petroleum substances may have assisted in the assessment.

Evaluation of risk to human health involves consideration of data relevant to estimation of exposure (non-occupational) of the general population, as well as information on health effects. Health effects were assessed using toxicological data pooled across high priority LBPNs, as well as high-hazard components known to be present in LBPNs. Decisions for risk to human health are based on the nature of the critical effect and margins between conservative effect levels and estimates of exposure, taking into account confidence in the completeness of the identified databases on both exposure and effects, within a screening context. The screening assessment does not represent an exhaustive or critical review of all available data. Rather, it presents a summary of the critical information upon which the conclusion is based.

This screening assessment was prepared by staff in the Existing Substances Programs at Health Canada and Environment Canada and incorporates input from other programs within these departments. The human health and ecological portions of this assessment have undergone external written peer review/consultation. Comments on the technical portions relevant to human health were received from scientific experts selected and directed by Toxicology Excellence for Risk Assessment (TERA), including Dr. Michael Dourson (TERA), Dr. Stephen Embso-Mattingly (NewFields Environmental Forensics Practice, LLC), Dr. Michael Jayjock (The LifeLine Group) and Dr. Darrell McCant (Texas Center for Environmental Quality). Although external comments were taken into consideration, the final content and outcome of the screening assessment remain the responsibility of Health Canada and Environment Canada.

The critical information and considerations upon which the draft screening assessment is based are summarized below.

Substance Identity

LBPNs in general are complex, liquid, combinations of hydrocarbons containing volatile components and are produced by refining or upgrading crude oil or bitumen, or are produced in natural gas processing plants. These light petroleum fractions are commonly used as blending streams for gasoline, solvents and other industrial/residential components. They are substances of varying properties, with a typical boiling point range of 20–230°C, and predominantly fall in the C_4–C_{12} carbon range (CONCAWE 2005) (Table A2.1 in Appendix 2). LBPNs are composed of alkanes, isoalkanes, cycloalkanes, aromatics and, if subject to a cracking process, alkenes.

These UVCB substances are complex combinations of hydrocarbon molecules that originate in nature or are the result of chemical reactions and processes that take place during the upgrading and refining process. Given their complex and variable compositions, they could not practicably be formed by simply combining individual constituents.

Physical and Chemical Properties

The composition and physical-chemical properties of LBPNs vary depending on the sources of crude oil, bitumen and natural gas and the processing steps involved. A summary of the physical and chemical properties of the industry-restricted LBPNs is presented in Table 1.

Table 1. General physical-chemical properties of industry-restricted LBPNs

CAS RN	Ratio of aromatics to aliphatics[a]	Water solubility	Boiling point range (°C)	Relative density (g/cm^3)	Reference
64741-42-0	4:96	< 0.1 vol% at 20°C	20–220	0.70	ECB 2000a
64741-69-1	26:52	30–100 mg/L at 20°C	20–180		ECB 2000b
64741-78-2	20:80		65–230		CONCAWE 1992; ECB 2000c; API 2001a

[a] The composition of LBPNs is variable, and exact data on the ratio of components are not available. The data shown here are estimates based on data acquired from similar petroleum substances as reported in the references.

To predict the environmental behaviour and fate of complex petroleum substances such as these LBPNs, representative structures were chosen from each chemical class contained within the substances (Table A2.2 in Appendix 2). Nineteen structures were selected from the database in PETROTOX (2009) based on boiling point ranges for each LBPN, the number of data on each structure and the middle of the boiling point range of similar structures. As the composition of most LBPNs is not well defined, representative structures could not be chosen based on their proportion in the substance. This lack of general compositional data resulted in the selection of representative structures for alkanes, isoalkanes, alkenes, one- and two-ring cycloalkanes, and one- and two-ring aromatics ranging from C_4–C_{12}. Data on physical-chemical properties were assembled from the scientific literature and from the group of environmental models included in the United States Environmental Protection Agency's (U.S. EPA) Estimation Programs Interface Suite (EPI Suite 2008) (Table A2.2 in Appendix 2).

Water solubility ranges from very low (0.004 mg/L) for the longest chained alkanes to high (1790 mg/L) for the simplest monoaromatic structures. In general, aromatic compounds are more soluble than similar-sized alkanes, isoalkanes and cycloalkanes.

Experimental and modelled vapour pressures for representative structures are moderate to very high and decrease with increasing molecular size (Table A2.2 in Appendix 2). This indicates that losses to air from soil and water will likely be high and that the air will be the ultimate receiving environment for most of the components of LBPNs.

Sources

Industry-restricted LBPNs are produced in Canadian refineries and upgraders. Both CAS RN descriptions (NCI 2006) and typical process flow diagrams (Hopkinson 2008) indicate the origin of these LBPNs. Information submitted under section 71 of CEPA 1999 shows that these substances can be intermediate streams consumed within a facility, blended into a mixture leaving the facility under different CAS RNs or transported off-site for use as a feedstock in other industrial facilities (Environment Canada 2008, 2009).

CAS RN 64741-42-0 refers to a straight-run distillate directly from the atmospheric distillation of crude oils with a carbon range primarily from C_4–C_{11}.

CAS RNs 64741-69-1 and 64741-78-2 have slight differences in their dominant carbon ranges, but they each represent a distillate from the fractionation of hydrocracking effluents in a refinery or an upgrader.

CAS RN 64741-78-2 is a complex combination of hydrocarbons from distillation of the products from a hydrocracking process consisting largely of saturated hydrocarbons in the C_6–C_{12} range. In a refinery, it is used for gasoline blending. In an upgrader, it is fed either into the diluent naphtha stream or into the mid-distillate hydrotreating process.

Volumes transported in Canada, including shipments within Canada, imports and exports, were provided under section 71 of CEPA 1999 (Environment Canada 2009). The total annual amount transported via multiple transportation modes (ships and trucks) is less than 2 million tonnes (year 2006).

Uses

According to the information collected through the *Notice with respect to certain high priority petroleum substances* (Environment Canada 2008) and the *Notice with respect to potentially industry-limited high priority petroleum substances* (Environment Canada 2009) published under section 71 of CEPA 1999, these industry-restricted LBPNs have been identified as being consumed at the facility, blended into substances leaving the facility under different CAS RNs or transferred to another industrial facility as a feedstock. Although these substances were identified by multiple use codes established during the development of the Domestic Substances List (DSL), it has been determined from information submitted under section 71 of CEPA 1999, voluntary submissions from industry, an in-depth literature review and a search of material safety data sheets that these industry-restricted LBPNs (the CAS RNs identified in this screening assessment) may leave a petroleum facility and be transported to another industrial facility for use as a feedstock, but do not reach the public market in the form originally acquired.

Releases to the Environment

Potential releases of industry-restricted LBPNs consist of releases within facilities from activities associated with processing these substances, as well as releases related to transportation of these substances between industrial facilities.

Due to the complex nature of the petroleum industry and transportation industry, as well as the ambiguity in the literature in the use of the terminology that is critical to the understanding of the Stream 2 PSSA assessments, it is important that the definitions specific to the assessment of the industry-restricted petroleum substances are well understood. Table 2 lists the terminology specific to the present assessment.

Table 2. Definitions of terms specific to the PSSA assessments of industry-restricted petroleum substances

Terminology	**Definition**
Release	A generic term to define a leak, spill, vent, or other release of a gaseous or liquid substance, including controlled release and unintentional release, as defined below, but not including catastrophic events.
Controlled release	Any **planned** release for safety or maintenance purposes that is considered part of routine operations and occurs under controlled conditions.
Unintentional release	Any **unplanned** release of a petroleum substance. Causes can include equipment failure, poor maintenance, lack of proper operating practices, adverse weather-related events or other unforeseen factors, but can also be a routine part of normal operations. The following two categories are included under unintentional releases: (1) unintentional leaks or spills that occur from processing, handling and transport of a petroleum substance; such leaks or spills can be reduced or controlled by the industry; and (2) accidental releases that may not be controllable by the industry. Only unintentional leaks or spills (category 1 defined the

Terminology	Definition
	above) are considered in the assessment of the potential of industry-restricted petroleum substances to cause ecological harm.
Fugitive release	A specific type of unintentional release. It refers to an unintentional release, which occurs under normal operating conditions, of a gaseous substance into ambient air and which may occur on a routine basis. Fugitive releases can be reduced but may not be entirely preventable due to the substance's physical-chemical properties, equipment design and operating conditions. Evaporative emission during the transportation of petroleum substances is a fugitive release and is considered in the human exposure analysis for purposes of assessing the potential of the substance to cause harm to human health.

Potential On-site Releases

Potential releases of LBPNs from refineries or upgraders can be characterized as either controlled or unintentional releases. Controlled releases are planned releases from pressure relief valves, venting valves and drain systems for safety purposes or maintenance. Unintentional releases are typically characterized as spills or leaks from various equipment, valves, piping or flanges. Refinery and upgrader operations are highly regulated, and regulatory requirements are established under various jurisdictions. As well, voluntary non-regulatory measures implemented by the petroleum industry are in place to manage these releases (SENES 2009).

Controlled Releases

The industry-restricted LBPNs in this screening assessment originate from distillation columns as a distillate in a refinery or upgrader. Thus, the potential locations for the controlled release of these LBPNs include relief valves, venting valves and drain valves on the piping or vessels where these streams are generated.

Under typical operating conditions, controlled releases of industry-restricted LBPNs would be captured in a closed system,[3] according to defined procedures, and returned to the processing facility or to the facility's wastewater treatment plant. In both cases, exposure of the general population or the environment to these industry-restricted LBPNs is not expected.

Unintentional Releases

Unintentional releases (including fugitive releases) occur from equipment (e.g., pumps, storage tanks), valves, piping, flanges, etc. during processing and handling of petroleum substances and can be greater in situations of poor maintenance or operating practices. Regulatory and non-regulatory measures are in place to reduce these events at petroleum refineries and upgraders (Appendix 3) (SENES 2009). Rather than being specific to one

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[3] For the purposes of the screening assessment of PSSA substances, a closed system is defined as a system within a facility that does not have any releases to the environment and where evaporative emissions are collected and recirculated, reused or destroyed.

substance, these measures are developed to be more generic to limit non-routine releases of all substances in the petroleum sector.

Conclusion for Potential On-site Releases

Based on the information presented in this screening assessment and in the screening assessment of the Stream 1 (site-restricted) LBPNs, exposure of the general population or the environment to the on-site releases (controlled or unintentional) of industry-restricted LBPNs is not expected.

Potential Releases from Transportation

As these industry-restricted LBPNs can be transported between facilities, releases may also occur during transportation. In general, three operating procedures are involved during the process of transportation: loading, transit and unloading.

The on-site handling of petroleum substances for transportation is regulated at the federal and provincial/territorial levels by legislation covering loading and unloading (Appendix 3).

For those substances containing highly volatile components (e.g., LBPNs, gasoline), a vapour recovery system is generally implemented or recommended at loading terminals of Canadian petroleum facilities (SENES 2009). Such a system can significantly reduce evaporative emissions during handling procedures.

Storage of industry-restricted LBPNs may be required prior to transportation off-site. Potential releases during storage, such as leaks, spills and breathing loss (expulsion of vapour due to changes in temperature and pressure), are similar to other potential on-site releases, and are not separately addressed in this screening assessment.

Tanks or containers for transferring petroleum substances are typically dedicated vessels, and cleaning is therefore not required on a routine basis (U.S. EPA 2008; OECD 2009). As such, exposure of the general population and the environment to the LBPNs considered in this screening assessment from tank cleaning is not expected. Cleaning facilities require processing of grey-water to meet local and provincial release standards.

Release Estimation

Information on the transportation quantities and relevant transportation modes was collected under section 71 of CEPA 1999 (Environment Canada 2009) and from a literature review with respect to each CAS RN identified in this screening assessment. The total annual transport quantity of the three LBPNs considered in this report is less than 2 million tonnes (year 2006). The common transportation modes for these LBPNs are ships and trucks.

Two types of releases potentially occur during transportation and are considered in this screening assessment. These are regular fugitive evaporative emissions and unintentional releases (e.g., spills or leaks) during the handling and transit processes.

Evaporative emissions are similar to breathing loss of organic substances from storage tanks. The quantity lost depends on the volatility of a substance, temperature or pressure changes occurring during transportation and tightness of transport vessels and valve settings. Ambient air is the receiving medium for the evaporative emissions.

Evaporative emissions were considered in transportation by ships and trucks and were estimated based on empirical equations from the U.S. EPA (2008), physical-chemical properties of these LBPNs (e.g., vapour pressure, molecular weight and density of vapours) and their annual transported quantities (see sample calculations following Table A6.3 in Appendix 6). These estimated evaporative emission quantities for the various modes of transportation are used for determining the concentration of LBPN vapours in ambient air in the human health exposure assessment.

The estimated evaporative emission quantities for loading and unloading are considered in the human health exposure assessment for industry-restricted LBPNs insofar as the focus is on the potential for releases outside the facility, where the potential for exposure of the general population (non-occupational) is the greatest. Occupational exposures as a result of evaporative emission vapours are not considered.

Unintentional releases (i.e., spills) of these LBPNs were estimated through analysis of historical spill data (2000–2009) from the Environment Canada Spill Line database (Environment Canada 2011). The releases analyzed were generically categorized as “naphthas” (most naphthas transported in Canada are typically relatively light combinations of hydrocarbons used for blending gasoline and solvents); although they may not be specific to the LBPNs considered in this report, they did provide a conservative estimate of releases.

Some of the reports in the Environment Canada Spill Line database have no estimate of the volume released into the environment. In order to account for underestimation of the volume released, the estimated total volumes were extrapolated by assuming that the statistical distribution of reported volumes released was representative of all releases. Collisions, poor road conditions and/or adverse weather-related events listed as a source or cause of or reason for the spill were not included in the release estimate, as they were not considered preventable with regard to loading/unloading and transport of the naphthas. Releases where the source was pipeline or train were also not considered, as information submitted under section 71 of CEPA 1999 indicated that the LBPNs being assessed in this report are not transported using those modes of transportation. Furthermore, an extremely large spill (190 776 L) in Alberta in 2007 was removed in order to provide a better assessment of more common release scenarios. It should be noted, however, that although large spills are not common, they are still possible. Results are shown in Table A4.1 (Appendix 4).

The overall average spill volume for naphthas is 1966 L per year based on historical spill data (2000–2009). From 2000–2003, there was only one reported spill, with a volume of 2226 L. The lack of data during this time is believed to be due to differences in the reporting by several provinces rather than the absence of any releases. From 2005–2009, more releases were reported, as Alberta started to provide data in 2005, although there are still few releases per year. Release volumes by year for each province and the environmental compartment to which releases occurred (air, land, fresh water) are reported in Tables A4.2 and A4.3 (Appendix 4), respectively. No spills into salt water were reported, indicating that releases from ships during loading did not occur. From 2000–2009, there were eight spills to land, one spill to fresh water, one spill to an unknown media and five releases to air, for an average 1.5 releases per year for naphthas, as a whole. The average release to air is 407 L, whereas the average release to land is 3186 L. No release volume was available for the spill to fresh water. As these industry-restricted LBPNs are likely only a portion of naphthas released, then releases of the LBPNs under assessment in this report would be less than 1.5 per year.

The Environment Canada Spill Line database provides information on the sources and causes of and reasons for many of the naphtha releases. The data were analyzed to determine how and why the majority of naphtha releases occur (Tables A4.4a–c in Appendix 4). The majority of naphtha spills occur at industrial plants (45% by volume) and refineries (34% by volume), which implies that most spills do not occur during transportation, although they could potentially occur during loading and unloading (Table A4.4a in Appendix 4). Almost half of the naphtha releases are attributed to unnamed causes, but of those causes identified, leaks from valves (32% by volume) and pipes (12% by volume) are the most common (Table A4.4b in Appendix 4). Analyzing the reasons for the releases reveals that equipment failure is a primary reason (75% by volume), followed by unnamed reasons (12% by volume) and error (7% by volume) (Table A4.4c in Appendix 4).

Assessment of potential exposure of the environment from the transportation of industry-restricted LBPNs focuses on releases to water, soil and air due to unintentional spills. In comparison, assessment of potential exposure of the general population from transportation of industry-restricted LBPNs focuses on evaporative emission, which occurs during regular operating activities. Although spills to water or soil may occur during transit and in loading or unloading operations, such releases are considered to occur on a non-routine or unpredictable basis in distinct locations and are therefore not considered in the assessment of exposure of the general population.

Environmental Fate

When liquid phase petroleum substances are released into the environment, four major fate processes will take place: dissolution in water, volatilization, biodegradation and sorption. These processes will cause changes in the composition of these UVCB substances. In the case of spills on land or water surfaces, another fate process, photodegradation, can also be significant.

The rates of dissolution in water or volatilization of individual petroleum components are retarded by the complex nature of these petroleum mixtures. The solubility and volatility of individual components in petroleum hydrocarbon mixtures are proportional to the solubility or volatility of the component in its pure state and its concentration in the mixture. Solubility and volatility of a component decrease when the component is present in a mixture (Banerjee 1984; Potter and Simmons 1998).

Each of the fate processes affects hydrocarbon families differently. Aromatics tend to be more water soluble than aliphatics of the same carbon number, whereas aliphatics tend to be more volatile (Gustafson et al. 1997). Thus, when a petroleum mixture is released into the environment, the principal water contaminants are likely to be aromatics while aliphatics will be the principal air contaminants (Potter and Simmons 1998). The trend in volatility by component class is as follows: alkenes ≈ alkanes > aromatics ≈ cycloalkanes. The most soluble and volatile components have the lowest molecular weight; thus, there is a general shift to higher molecular weight components in residual materials.

Biodegradation is almost always operative when petroleum mixtures are released into the environment. It has been widely demonstrated that nearly all soils and sediments have populations of bacteria and other organisms that are capable of degrading petroleum hydrocarbons. Degradation occurs both in the presence and absence of oxygen. Two key factors that determine degradation rates are oxygen supply and molecular structure. In general, degradation is more rapid under aerobic conditions. Decreasing trends in degradation rates according to structure are (Potter and Simmons 1998):

(1) *n*-alkanes (especially in the C_{10}–C_{25} range which are degraded readily);
(2) isoalkanes;
(3) alkenes;
(4) benzene, toluene, ethylbenzene and xylenes (BTEX) (when present in concentrations that are not toxic to the microorganisms);
(5) monoaromatics;
(6) polynuclear (polycyclic) aromatic hydrocarbons (PAHs); and
(7) higher molecular weight cycloalkanes (which may degrade very slowly (Pancirov and Brown 1975)).

These trends typically result in the depletion of the more readily degradable components and the accumulation of the most resistant in residues.

Level III fugacity modelling of representative hydrocarbons contained in the LBPN group of substances was performed using EQC (2003) (Table A5.1 in Appendix 5), based on their physical-chemical properties as given in Table A2.2 (Appendix 2). Lower estimated water solubility may affect the amount of substance that will partition to the different environmental compartments; however, this was not considered in the model output due to its limitations.

If released solely to air, > 98% of most components in LBPNs are expected to remain in air (Table A5.1 in Appendix 5); the only exception would be in the two-ring aromatics, where roughly 14% would partition out of air to water and soil. Many components (C_4–C_6) are extremely volatile, with vapour pressures in excess of 13 000 Pa, and most other components are highly volatile, with vapour pressures above 165 Pa. The larger (C_{12}) alkanes, cycloalkanes and one- and two-ring alkylated aromatics are moderately volatile, with vapour pressures from 2–18 Pa. Due to these generally very high vapour pressures, air will be a major environmental compartment for LBPNs.

If released solely to water, the C_4–C_6 alkanes and isoalkanes, C_9 alkenes, C_6–C_9 one-ring cycloalkanes, C_9 two-ring cycloalkanes and the one- and two-ring aromatics will remain in water (Table A5.1 in Appendix 5). All of these components are moderately soluble in water, with solubility of the pure substances ranging from 5–1790 mg/L. According to the EQC model, those components that do not remain in water will partition to sediments (C_{12} alkanes, isoalkanes, and cycloalkanes). However, the behaviour of petroleum mixtures in the environment is much more complex than the current fugacity model can accommodate. All LBPNs are much less dense than water (API 2003a, b, c) so that, upon entering water, they will rise to the surface and spread out as a slick where most components, due to their high vapour pressures and Henry's Law constants, will likely volatilize. Some of these will also dissolve into water and some will sorb to suspended particles.

If released solely to soil, the C_4–C_6 alkanes and isoalkanes will partition to air; all other component groups will largely remain in soil (Table A5.1 in Appendix 5).

Fugacity estimations in soil do not take into account situations where large quantities of a hydrocarbon mixture enter the soil compartment. When soil organic matter and other sorption sites in soil are fully saturated, the hydrocarbons will begin to form a separate phase (a non-aqueous phase liquid or NAPL) in the soil. At concentrations below the retention capacity for the hydrocarbon in the soil (Arthurs et al. 1995), the NAPL will be immobile; this is referred to as residual NAPL (Brost and DeVaull 2000). Above the retention capacity, the NAPL becomes mobile and will move within the soil (Arthurs et al. 1995; Brost and DeVaull 2000). According to a study by Brost and DeVaull (2000), the NAPLs of fuel products in the density range of gasoline, such as LBPNs, will become mobile in the range of 3400–80 000 mg/kg dw depending on the type of soil. Above this range, they can move through the soil due to gravity.

Persistence and Bioaccumulation Potential

Environmental Persistence

No empirical data are available on the degradation of LBPNs as complex mixtures; however, estimates can be derived from analyzing the biodegradation of the components of LBPNs.

Aerobic biodegradation data for individual isoalkanes (C_9–C_{12}) from an Organisation for Economic Co-operation and Development (OECD) 301F ready biodegradation test indicate that they will be 22% degraded (ultimate biodegradation) over a period of 28 days (ECB 2000d). This equates to a degradation half-life of approximately 78 days in water, assuming that degradation follows first-order kinetics. Numerous researchers have found that the degree of branching in an isoalkane increases its resistance to biodegradation (Atlas 1981). However, Prince et al. (2007a, b) reported that C_6–C_{10} components (alkanes, isoalkanes, alkenes, cycloalkanes and one- and two-ring aromatics) in a formulated gasoline had relatively short median half-lives (primary biodegradation)—ranging from 3–17 days—in fresh water, salt water and sewage effluent (Table A5.2 in Appendix 5). They hypothesized that primary biodegradation half-lives were shorter for hydrocarbons in a gasoline mix than for individual components because indigenous micro-organisms degrade hydrocarbons most effectively when they are presented as a mixed suite of hydrocarbon substrates that allows microbes to use intermediates from different pathways to balance their overall metabolism.

A quantitative structure–activity relationship (QSAR)-based weight-of-evidence approach was also applied using the BioHCWin (2008), BIOWIN 3, 4, 5, 6 (2009), CATABOL (c2004–2008), TOPKAT (2004) and AOPWIN (2008) degradation models. Primary biodegradation (estimated with BioHCWin and BIOWIN 4) is the transformation of a parent compound to an initial metabolite. Ultimate biodegradation (estimated with BIOWIN 3, 5 and 6, CATABOL and TOPKAT) is the transformation of a parent compound to carbon dioxide and water, mineral oxides of any other elements present in the test compound and new cell material (EPI Suite 2008). BIOHCWIN (2008) is a biodegradation model specific to petroleum hydrocarbons. Using an extrapolation ratio of 1:1:4 for water:soil:sediment biodegradation half-lives (Boethling et al. 1995), the half-lives in soil and sediment can be extrapolated from the half-life estimations in water. Modelled results that were out-of-domain were not considered when determining the persistence of components.

The results of the BioHCWin (2008) model indicate that the components of LBPNs have primary degradation half-lives ranging from 3–56 days (Table A5.3 in Appendix 5). Using an extrapolation ratio of 1:1:4 for water : soil : sediment biodegradation half-lives (Boethling et al. 1995), the half-life in soil for the heavy components is estimated to be < 182 days and the half-life in sediments is estimated to be < 365 days. These primary degradation results are in agreement with the ultimate biodegradation models, indicating that these structures will not likely persist in the environment. One exception was the C_{12} monoaromatics, which were found to be persistent based on the ultimate biodegradation models. However, BioHCWin (2008) indicates that this component has a primary degradation half-life of 5 days. The indication of a quick primary biodegradation suggests that the time to complete mineralization could be < 182 days.

In air, empirical data (Atkinson 1990) show that butane, iso-butane, pentane and iso-pentane are persistent (Table A5.4 in Appendix 5), with half-lives ranging from 2–3.4 days. Predicted atmospheric oxidation half-lives (AOPWIN 2008) for representative structures are consistent with these data. Additionally, AOPWIN (2008) predicted that butane, isobutane, benzene, hexane and isopentane would also be persistent in air, with half-lives of 4.1 days, 4.4 days, 5.5 days, 2 days and 2 days, respectively (Table A5.5 in Appendix 5).

Biodegradation modelling indicates that the industry-restricted LBPNs are unlikely to contain components that are persistent in water, soil or sediment; however, modelled and empirical data indicate that industry-restricted LBPNs contain C_4–C_6 components that meet the criterion for persistence in air as defined in the *Persistence and Bioaccumulation Regulations* of CEPA 1999 (Canada 2000).

Potential for Bioaccumulation

Bioconcentration Factors (BCF) and Bioaccumulation Factors (BAF)

Experimental Studies

Since LBPNs are complex combinations of hydrocarbons, empirical data on representative structures found in these LBPNs (using gasoline and Fuel Oil No. 2) in a read-across approach and a predictive approach using a bioconcentration/bioaccumulation factor model were applied (Arnot and Gobas 2003, 2004). According to the *Persistence and Bioaccumulation Regulations* (Canada 2000), a substance is bioaccumulative if its BCF or BAF is ≥ 5000; however, measures of BAF are the preferred metric for assessing the bioaccumulation potential of substances. This is because BCFs may not adequately account for the bioaccumulation potential of substances via the diet, which predominates for substances with log $K_{ow} > \sim 4.5$ (Arnot and Gobas 2003).

Correa and Venables (1985) studied the bioaccumulation of naphthalene in white mullet (*Mugil curema*). Naphthalene was accumulated into tissues at a much higher rate than it was depurated. BCFs for muscle tissue ranged from 81–561 L/kg wet weight (ww), declining with an increase in naphthalene concentration. BCFs measured over 96 hours were slightly higher than for 48 hours. Complete depuration was accomplished in two days.

Neff et al. (1976) exposed clams (*Rangia cuneata*), oysters (*Crassostrea virginica*) and fish (*Fundulus similus*) to the water-soluble fraction of Fuel Oil No. 2 (0.41 kg/L [2 ppm] total naphthalenes) for 2 hours, followed by depuration of hydrocarbons for 366 hours. All fish organs examined showed rapid accumulation of naphthalenes within the 2-hour exposure period, with the gallbladder and brain of fish accumulating the highest concentrations. BAFs of naphthalenes in clams ranged from 2.3–17.1 L/kg ww (Table A5.6 in Appendix 5). Depuration of naphthalenes by fish tested began immediately following transfer to fresh water, reaching undetectable levels after 366 hours (~15 days). Shrimp and fish depurated aromatic hydrocarbons more rapidly than clams and oysters because molluscs are unable to rapidly metabolize aromatic hydrocarbons, and accumulation can occur in stable tissue compartments with low hydrocarbon turnover and are not readily exchangeable (Stegeman and Teal 1973; Neff et al. 1976).

Invertebrates have also been shown to bioaccumulate petroleum hydrocarbons. Muijs and Jonker (2010) studied the bioaccumulation of petroleum hydrocarbons (total and divided into three different carbon ranges) over 49 days by the aquatic worm, *Lumbriculus variegatus,* after exposure to a series of 14 field-contaminated sediments with a known history of oil pollution. A maximum tissue concentration was reached for the C_{11}-C_{16} fraction after 14 days of exposure but then decreased; other fractions did not show any decrease in tissue concentration once a maximum was achieved. After 28 days of exposure, it was estimated that 70-90% of equilibrium was reached, though it was noted that it may take >90 days for hydrocarbons $>C_{34}$ to reach equilibrium. Characterization of the accumulated hydrocarbons was not determined, however, alkanes from C_{10} to C_{34} were identified in the aquatic worms. The accumulation of higher molecular weight alkanes may possibly be due to ingestion of organic matter to which the chemicals are sorbed. Depuration was not studied.

Two studies on BAFs of one- and two-ring aromatics in clam and Atlantic salmon were found. Experimental values of BAFs from the work of Neff et al. (1976) and Zhou et al. (1997) were compiled for comparison with modelled data (Arnot and Gobas 2003, 2004) (Tables A5.6 and 5.7 in Appendix 5). In general, the modelled values approximate those measured for the selected PAHs. None of the measured or modelled values for PAHs were shown to meet the bioaccumulation criterion (BAF ≥ 5000) defined in the *Persistence and Bioaccumulation Regulations* (Canada 2000).

Characterizing BCF/BAF

In characterizing bioaccumulation, the derivation of a BAF is preferred over a BCF where chemical exposure through the diet is not included in the latter (Barron 1990). According to Arnot and Gobas (2006), the BCF is a poor descriptor of biomagnification in food webs because it is typically derived from controlled laboratory experiments and does not include dietary exposure. Thus, BCFs based on laboratory studies have been shown to underestimate bioaccumulation potential for biomagnification of chemicals in the food web, as predators consume prey containing lipophilic compounds (U.S. EPA 1995). As hydrophobicity increases, dietary uptake is likely to be more important than absorption from water (Arnot and Gobas 2003). Further, laboratory BCFs have been shown to overestimate bioaccumulation potential when a chemical is bound or tightly sorbed to sediment (i.e., less bioavailable).

Due to the scarcity of measured BAFs available (Table A5.6 in Appendix 5), BCFs from various published works were used to help verify measured and modelled BAF values (Table A5.8a in Appendix 5). In contrast to the few available experimental BAFs on PAHs, a suite of BCFs for components of LBPNs were found, including alkanes, one-and two-ring cycloalkanes and one- and two-ring aromatics. Model estimates of these BCFs were also produced using a kinetic mass-balance model (Arnot and Gobas 2003) to fit the model kinetic elimination constants to agree with the observed BCF data in order to generate BAF predictions that reflect the known elimination rates.

A kinetic mass-balance model is, in principle, considered to provide the most reliable prediction method for determining bioaccumulation potential because it allows for correction of the kinetic rate constants and bioavailability parameters, when possible. BCF and BAF model predictions are considered "in domain" for this hydrocarbon assessment because it is based on first principles. As long as the mechanistic domain (passive diffusion), global parameter domain (range of empirical log K_{ow} and molecular weight), as well as metabolism domain (corrected metabolic rate [k_M]) are satisfied, predictions are considered valid (Arnot and Gobas 2003, 2006). The kinetic mass-balance model developed by Arnot and Gobas (2003, 2004) was employed using metabolic rate constants normalized to both conditions of the study and a representative middle trophic level fish as outlined in Arnot et al. (2008a, b) when the BCF or growth-corrected elimination rate constant is known. Both BCF and biomagnification factor (BMF) empirical data were used to correct default model uptake and elimination parameters, which are summarized in Table A5.8b (Appendix 5).

In Table A5.8b (Appendix 5), some metabolic rate constants calculated from the empirical BCF data were negative, suggesting that the metabolic rate is essentially zero and that other routes of elimination are more important. Accordingly, no metabolic rate correction was used when predicting the BCF and BAF for these structures. Gut and tissue metabolism is generally not regarded as an important elimination process for chemicals with a log K_{ow} less than ~4.5 (Arnot et al. 2008a, b; Arnot and Gobas 2006), but this can depend on the size and lipid content of fish used in testing.

In Table A5.8a (Appendix 5), only the C_{12} diaromatic (1,3-dimethyl naphthalene) and C_8 cycloalkane (ethyl cyclohexane) had measured or modelled BCFs or BAFs $\geq$ 5000. While, empirical data in minnows suggest that the C_{12} diaromatic (1,3-dimethylnaphthalene) is bioaccumulative, modelled data indicate that 1,3-dimethylnaphthalene does not bioaccumulate. The modelled BCF (4073 L/kg ww) and BAF (4073 L/kg ww) for 1,3-dimethylnaphthalene using the Arnot-Gobas mass-balance kinetic model indicates that they are not highly bioconcentrated or bioaccumulated. Furthermore, Neff et al. (1976) found that the C_{12} and C_{13} diaromatics (alkylated naphthalenes and biphenyls) were not highly bioaccumulative in clams upon exposure to an oil-in-water dispersion of Fuel Oil No. 2. Thus, the combined weight of evidence suggests that these C_{12} diaromatics are not likely to be highly bioaccumulative. For the C_8 cyclohexane (ethyl cyclohexane), the predicted BAF (Arnot and Gobas 2004) for the middle trophic level fish is 5495 L/kg ww, which just exceeds the criterion (BAF ≥ 5000), suggesting that it is bioaccumulative when all routes of uptake are considered. This prediction, however, was generated with a metabolic rate equal to zero because of the potential error associated with the estimate of metabolism rates (Table A5.7b in Appendix 5). Factoring in metabolism, it is expected that the BAF would be lower and likely below 5000. As well, the experimental BCF suggests this C_8 cycloalkane is not highly bioaccumulative (Table A5.7a in Appendix 5). Combining these lines of reasoning suggests that this C_8 cycloalkane is also not likely to be bioaccumulative according to the Canadian criteria.

BCF and BAF model estimates were also generated for an additional nineteen C_4–C_{12} linear and cyclic representative structures using the modified Arnot-Gobas three trophic level model (2004) (Table A5.7 in Appendix 5), as no empirical bioaccumulation data were identified for these substances. Metabolism and dietary assimilation efficiency kinetics were corrected for these predictions based on analogue BCF and BMF test data. From this analysis, two of the components are predicted to have a BCF or BAF ≥ 5000: a C_{12} one-ring cycloalkane (*n*-hexyl cyclohexane) and a C_{12} alkene (9-methyl-1-undecene). *n*-Hexyl cyclohexane has a modelled BCF of 6025 L/kg ww and a modelled BAF of 57 543 L/kg ww, while 9-methyl-1-undecene has a modelled BAF of 7079 L/kg ww. The log K_{ow} for these structures suggests that dietary uptake can predominate (up to 87% of total uptake) but will not be the sole route of exposure, as some substances are expected to have a 90% bioavailable fraction in the water column. BAF is therefore considered the most appropriate metric for assessing the bioaccumulation potential of these structures and represents a comparison of whole-body burdens compared with concentrations in water. The BCF and BAF predictions for these fractions are within the parametric, mechanistic and metabolic domains of the model and so are considered reliable.

Bioaccumulation Conclusion

As noted previously, of the parameters that have prescribed Canadian regulatory criteria, BAF values are preferred over BCF values because they represent the potential accumulation in biota from all exposure sources and thus represent a more complete picture of the total body burden of chemicals. Biomagnification (BMF), trophic or foodweb magnification (TMF) and biota-sediment accumulation factors (BSAF) are also considered very important for understanding the pattern of bioaccumulation and are used in a weight of evidence for characterizing the overall bioaccumulation potential of a chemical.

Overall, there are modelled and empirical bioaccumulation data to suggest that C_{12} alkenes and cycloalkanes meet the bioaccumulation criteria as defined in the *Persistence and Bioaccumulation Regulations* (Canada 2000). These components are associated with a slow rate of metabolism and are highly lipophilic. Exposures from water and the diet, when combined, suggests that the rate of uptake would exceed the total elimination rate. However, there is no evidence that these components would biomagnify, as a combination of metabolism, low dietary assimilation efficiency and growth dilution could allow the elimination rate to exceed the total uptake rate. Therefore, LBPNs may contain components that meet the bioaccumulation criteria in the *Persistence and Bioaccumulation Regulations.* Both empirical and modelled data suggest that none of the components analyzed are both highly persistent and bioaccumulative (Table A5.9 in Appendix 5).

Potential to Cause Ecological Harm

Ecological Effects Assessment

Aquatic Compartment

Experimental aquatic toxicity data were not available for the specific LBPNs considered in this assessment; however, toxicity data can be extrapolated from studies of similar types of LBPNs (Table A5.10 in Appendix 5). Empirical data for the water-accommodated fraction of naphthas and naphtha mixtures show moderate toxicity, with 96-hour median lethal loading / median lethal concentration (LL_{50}/LC_{50}) ranging from 2.6–18 mg/L in rainbow trout (*Oncorhynchus mykiss*), fathead minnow (*Pimephales promelas*) and amphipod (*Chaetogammarus marinus*), and median effects level / median effective concentration (EL_{50}/EC_{50}) ranging from 4.5–880 mg/L in water flea (*Daphnia magna*) and green algae (*Pseudokirchneriella subcapitata*). Empirical tests with water-accommodated fractions of LBPNs did not indicate that the substances tested were highly hazardous to aquatic organisms.

A range of moderate aquatic toxicity predictions were obtained from the PETROTOX model (PETROTOX 2009). These lethal loading (LL_{50}) predictions were in the same range as the empirical tests on similar substances, from 0.3–14.4 mg/L (Table A5.11 in Appendix 5). Predicted LL_{50} values for fish ranged from 0.6–14.4 mg/L, while values for invertebrates ranged from 0.3–7.2 mg/L.

To determine whether the modelled data from PETROTOX are suitable to use, a read-across approach was also conducted to compare the modelled toxicity of these LBPNs to empirical toxicity studies on leaded and unleaded gasoline, as well as other naphthas (Table A5.10 in Appendix 5). Aquatic 48-hour LC_{50}/EC_{50} values range from 1.2–51 mg/L, and 96-hour LC_{50}/EC_{50} values range from 0.3–182 mg/L. The range of values for fish is slightly higher than the range of values for invertebrates, but there is significant overlap. For example, the 96-hour LC_{50}/EC_{50} values for fish range from 2.7–182 mg/L, while the range for invertebrates is 0.3–171 mg/L. Comparison between the empirical data and the modelled data from PETROTOX shows that the modelled data are within the appropriate range of values for similar commercial products.

LBPNs are not known to be transported in fresh water and thus a CTV was not considered for the freshwater compartment.

The most sensitive marine organism from the collected empirical data is *Mysidopsis bahia* (mysid shrimp), with a 96-hour LC_{50} of 0.3 mg/L, and the most sensitive organism from the PETROTOX tests was *Rhypoxynius abronius* (amphipod), with an LL_{50} of approximately 0.3 mg/L. Therefore, the critical toxicity value (CTV) in the marine environment for this screening assessment will be 0.3 mg/L.

Terrestrial Compartment

The Canada-Wide Standards for Petroleum Hydrocarbons in Soil (CCME 2007) were used as a data source for effects of LBPNs on terrestrial ecosystems. These standards were developed based on consideration of four fractions of total petroleum hydrocarbons (TPH): F1 (C_6–C_{10}), F2 (> C_{10}–C_{16}), F3 (> C_{16}–C_{34}) and F4 (> C_{34}). Fraction 1 (F1) is most like LBPNs, although LBPNs have a lower starting carbon range of C_4. Standards were developed for four land-use classes (agriculture, residential, commercial, industrial) and two soil types (coarse grained and fine grained). The land-use and soil type with the lowest standard is typically agricultural coarse-grained soils. The F1 standard for soil contact by non-human organisms for agricultural coarse-grained soils is 210 mg/kg dw (Table A5.12 in Appendix 5; CCME 2008).

As most of these industry-restricted LBPNs under consideration are ultimately blended into gasoline, mammalian inhalation toxicity tests for unleaded gasoline were determined to be a reasonable surrogate for estimating the environmental toxicity of these LBPNs. Adult rats were tested for 6 hours/day, 5 days/week, for 13 weeks at up to 6570 mg/m^3 (1552 ppm) without any treatment-related mortality (Kuna and Ulrich 1984). MacFarland et al. (1984) found no treatment-related mortalities in rats exposed for 113 weeks to an unleaded gasoline vapour at concentrations up to 6170 mg/m^3. The value 6570 mg/m^3 is the highest no-observed-effect concentration (NOEC) for mortality from studies in animals identified, so it will be used as the CTV for acute inhalation effects in animals.

Ecological Exposure Assessment

All of the LBPNs in this report have been identified as industry-restricted, indicating that they leave petroleum facilities and are transported between facilities as substances with individual CAS RNs. Thus, potential for ecological harm during the movement and transportation of these various substances was evaluated.

Estimations of unintentional releases of these LBPNs were made with data from Environment Canada's Spill Line database (Environment Canada 2011). As industry-restricted LBPNs are not transported by fresh water, a freshwater scenario was not considered.

The environmental assessment of industry-restricted petroleum substances focuses on the unintentional releases of petroleum substances by the petroleum industry and the petroleum transportation industry, as these releases present the greatest potential for harm to the environment. Fugitive releases tend to pose less potential for harm to the environment as they are gaseous in nature. Such releases, when of low to moderate quantity, tend to disperse to concentrations that typically do not present a threat to non-human organisms.

Aquatic Compartment

Since the Environment Canada Spill Line database (Environment Canada 2011 did not have recorded releases to marine water from 2000-2009, an ecological exposure scenario for marine waters was not developed.

Atmospheric Compartment

Due to the volatile nature of LBPNs, releases can result in both liquid and evaporative loss. As a result, they can lead to air exposure. The highest transport quantity of industry-restricted LBPNs is by ships; therefore, the concentrations within 24 hours at 1000 m from evaporative emissions by ship transportation (i.e., 27 µg/m^3 or 0.02 mg/m^3) are used in evaluating ecological risk from this source (Table 5).

Evaporative emission scenarios for trucks and ships to air during loading and unloading were developed with the aid of emission factors developed for loading operations by the U.S. EPA (2008) and the use of a simple air dispersion model, SCREEN3 (1996), resulting in a predicted environmental concentration (PEC) of 0.02 mg/m^3.

Terrestrial Compartment

Historical data report less than 1 release per year of naphtha that may affect land, with an average spill volume of 3186 L (Table A4.3 in Appendix 4).

Due to the paucity of data available on the concentration of LBPNs in receiving soil following an average spill of LBPNs, the terrestrial scenario involves a read-across from data on gasoline to estimate the level of contamination following a spill. The terrestrial scenario does not provide an expected concentration of LBPNs in soil, but estimates the total potential area affected by a LBPN spill. This scenario is based on the retention capacity of gasoline in three soil types.

Arthurs et al. (1995) studied the volatilization of gasoline from three Canadian soils: Ottawa Sand, Delhi Loamy Sand and Elora Silt Loam. They provide the retention capacity of gasoline in dry soil thus enabling the determination of the approximate volume of soil that could reasonably be expected to be contaminated by an average gasoline spill. Information used to calculate the total potential volume affected, as well as the expected volume saturated with LBPNs can be found in Table A5.13 (Appendix 5).

An average reported LBPN spill per year to the terrestrial compartment is 3186 L (2230 kg). Based on the above information, if this volume of LBPN was released directly onto soil, it would contaminate between 6 and 19 m^3. This volume of soil is only that soil which would be saturated with LBPN (i.e., at the retention capacity, above which a mobile NAPL forms) and does not include any potential unsaturated areas or the movement of LBPN through soil after the spill. According to Arthurs et al. (1995), the retention capacity for gasoline in soil is 68 000 mg/kg dw for sand, 170 000 mg/kg dw for loamy sand, and 238 000 mg/kg dw for silt loam. These values will be used as the PECs for LBPN. It should be noted that these concentrations are greater than the concentrations required for LBPN to form a mobile NAPL (3400–80 000 mg/kg dw soil) according to Brost and DeVaull (2000).

Characterization of Ecological Risk

The approach taken in this ecological screening assessment was to examine available scientific information and develop conclusions based on a weight-of-evidence approach as required under CEPA 1999. Particular consideration has been given to risk quotient analyses based on estimates of exposure given that there are no empirical data for these LBPNs in the environment. For each endpoint organism, an estimate of the potential to cause adverse effects and a predicted no-effect concentration (PNEC) were determined. The PNEC is the lowest CTV for an appropriate species divided by an assessment factor. A risk quotient (RQ = PEC/PNEC) was calculated for each endpoint organism and is an important line of evidence in evaluating the potential risk to the environment.

Table 3 presents the summary of the risk quotients for the industry-restricted LBPNs.

Table 3. Risk quotients calculated for industry-restricted LBPNs

Compartment affected	Organism	PEC	CTV	Assessment factor	PNEC	Risk quotient
Air (ship and truck loading/unloading)	rat	0.02 mg/m^3	6570 mg/m^3	10	657 mg/m^3	3.0×10^{-5}
Terrestrial (loading/ unloading/transport – sand)	n/a	68 000 mg/kg	210 mg/kg	1	210 mg/kg[a]	324
Terrestrial (loading/ unloading/ transport – loamy sand)	n/a	170 000 mg/kg	210 mg/kg	1	210 mg/kg[a]	810
Terrestrial (loading/ unloading/ transport – silt loam)	n/a	238 000 mg/kg	210 mg/kg	1	210 mg/kg[a]	1133

[a] CCME Canada-wide Standard for Petroleum Hydrocarbon Fraction 1 by soil organisms in a coarse-grained residential or agricultural soil (mg/kg dw).
n/a: not applicable.

Releases to air during ship and truck loading/unloading are not expected to cause ecological harm to terrestrial organisms, as the RQ is substantially < 1 (Table 3). Thus, the estimated evaporative emissions are not of a sufficient concentration or duration to pose a risk to terrestrial animals.

Table 3 indicates that average volume LBPN releases to soil from loading, unloading and transport may cause harm to terrestrial organisms (RQ > 1). However, historical spills data indicate a low frequency of spills of naphtha, as a whole, that may affect land (less than 1 release per year). As industry-restricted substances are transported between petroleum facilities, the majority of releases of LBPNs during loading and unloading take place at these facilities, and data regarding the source of spills support this scenario (Table A4.4a in Appendix 4). Releases at such locations will typically be to hard surfaces, rather than to soil, and procedures for containment of such spills will generally be in place. Given the conditions of such releases and their frequency, the risk of harm to the terrestrial environment is low.

These LBPNs contain C_4–C_6 components that are considered to be persistent in air as defined in the *Persistence and Bioaccumulation Regulations* of CEPA 1999. Based on model results, two components of these industry-restricted LBPNs (C_{12} isoalkanes, alkenes and one-ring cycloalkanes) are considered to meet the criteria for bioaccumulation in the *Persistence and Bioaccumulation Regulations.* No components of these LBPNs were found to meet both the persistence and bioaccumulation criteria.

Based on the information presented in this screening assessment on the frequency and magnitude of spills, there is low risk of harm to organisms or the broader integrity of the environment from these substances. It is concluded that these industry-restricted LBPNs (CAS RNs 64741-42-0, 64741-69-1 and 64741-78-2) do not meet the criteria under paragraph 64(*a*) or (*b*) of the *Canadian Environmental Protection Act, 1999* (CEPA 1999) as they are not entering the environment in a quantity or concentration or under conditions that may have an immediate or long-term harmful effect on the environment or its biological diversity, or that constitute or may constitute a danger to the environment on which life depends.

Uncertainties in Evaluation of Ecological Risk

Uncertainty arises from the non-uniformity of spill data. The available data on spills generally do not report values for each specific transported substance by CAS RN. While Environment Canada has reported spills data for substances similar to these LBPNsspill data were used for naphthas in general as data specific to these industry-restricted LBPNs were not available.

The BAF model calculations were derived from a large database of measured BAF values from the Great Lakes for chemicals that are poorly metabolized (e.g., PCBs). With metabolic biotransformation, the BAF model predictions are in general agreement with measured BAFs in fish. Many petroleum hydrocarbons are readily metabolized, somewhat by invertebrates and at much higher levels in fish (Arnot and Gobas 2003; Arnot et al. 2008a, b). There is some uncertainty when estimating the biotransformation used by the model at the first trophic level. Thus, the BAF model predictions may be an overestimate in consideration of these factors.

The specific chemical compositions of the substances in this report are not well defined, given that low boiling point naphthas are UVCBs and cannot be represented by a single chemical structure. An LBPN under the same CAS RN but produced at different facilities can vary significantly in the number, identity and proportions of components, depending on feedstocks and operating conditions of processing units. Consequently, the data set presented here, which is based on representative structures and modelled physical-chemical properties, persistence, bioaccumulation and toxicity data, reflects this variability. For this reason, there is some uncertainty in the characterization of risk to the environment given that toxicological data derived from studies of a particular substance may not be totally representative of the spectrum of substances falling under the same CAS RN.

Potential to Cause Harm to Human Health

Exposure Assessment

The human health assessment of industry-restricted petroleum substances focuses on the fugitive releases that occur when the petroleum substances escape into ambient air. This includes evaporative emissions during the various modes of transportation of petroleum substances. The unintentional release (leaks or spills) data used in the ecological assessment are, for the purposes of assessing the potential to cause harm to human health, considered to be releases that occur on a non-routine or unpredictable basis in specific geographical locations. These unintentional releases (leaks or spills) typically do not contribute to the potential for exposure of the general population in Canada.

Evaporative emission of the industry-restricted LBPNs during the processes of loading and transportation from facilities by ships[4] or loading/unloading and transportation by trucks may result in exposure of the general population (living near ports or highways) via inhalation.

Loading and unloading of industry-restricted LBPNs for transportation are normally conducted on-site. A scenario to assess the potential for general population (non-occupational) exposure during the loading and unloading of industry-restricted LBPNs for truck transportation is described below. This scenario, based on reported loading procedures, considers the general population living in the vicinity of a loading facility. The assessment also focuses on the potential for releases of industry-restricted LBPNs during ship and truck transportation between facility sites.

Inhalation from Ambient Air

A description of the exposure scenarios considered for the assessment of industry-restricted LBPNs is presented in Table 4. Empirical monitoring data on LBPNs in the environment are not available. To estimate the contribution of LBPN vapours resulting from transport of these substances, SCREEN3 (1996), a screening-level Gaussian air dispersion model based on the Industrial Source Complex (ISC) model (for assessing pollutant concentrations from various sources in an industry complex), was used. It is designed to estimate maximum concentrations of chemicals at chosen receptor heights and at various distances from a release source for a given emission event. The driver for air dispersion in the SCREEN3 model is wind. The maximum calculated exposure concentration is selected based on a built-in meteorological data matrix of different combinations of meteorological conditions, including wind speed, turbulence and humidity. This model directly predicts concentrations resulting from point, area and

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[4] Unloading is not applicable to industry-restricted LBPNs transported by ship, as the substances are exported beyond the jurisdiction of Canada.

volume source releases. SCREEN3 also includes a mathematical model to give conservative estimates of emissions from line sources, such as truck and rail transit (SCREEN3 ISC Version 3 1995). Estimation of total releases along the line of transit is done using the sum of discrete volume sources along the highway up to distances far away from the receptor point (Figure A6.1 in Appendix 6). SCREEN3 gives the maximum exposure concentration in the direction downwind from the prevalent wind 1 hour after the release event. During a 24-hour period, for point emission sources, the maximum 1-hour exposure as assessed by the ISC Version 3, is multiplied by a factor of 0.4 to account for variable wind directions. This gives the maximum concentration within a 24-hour exposure (U.S. EPA 1992). Similarly, for exposure events happening over the span of a year, it can be expected that the direction of the prevalent winds will be even more variable and uncorrelated to the wind direction for a single event; thus, the maximum exposure concentration for one year is determined by multiplying the maximum 1-hour exposure by a factor of 0.08. Such scaling factors are not required for non–point source emissions. However, to prevent overestimation of the exposures, we use a scaling factor of 0.2 to obtain the yearly exposure concentration from the value of the maximum 1-hour exposure determined from SCREEN3 calculations. Input parameters are provided in Table A6.1 (Appendix 6).

Table 4. Description of defined exposure scenarios considered for the assessment of industry-restricted LBPNs

Scenario	Transportation mode	Type of emission source being modelled	Potential distance of bystander (m)[a]
I	Ships	Area (in port)	1000
IIa	Ships moving out of port at 10 km/h	Line (route parallel to port)	1000
IIb	Ships moving out of port at 10 km/h	Line (route perpendicular to port)	1000
III	Trucks idled for 1 h	Area (at rest stop)	200
IV	Trucks moving intra-city at 50 km/h	Line (highway)	200
V	Trucks moving inter-city at 100 km/h	Line (highway)	1000
VI	Evaporative emission from loading and unloading of trucks	Area (at refinery/loading dock)	1500

[a] The distances of bystanders were selected based on map analysis at different ports and highways, as well as industry verification of loading dock locations.

Estimated regular evaporative emission of industry-restricted LBPNs to air during transit is presented in Table A6.2 (Appendix 6) as a range to cover the losses from the various transportation modes involved. The emission rate (g/s) is derived based on the emission quantity of kilograms per day in Table A6.2. The estimated emission areas for different transportation modes and the speed of motion for line emissions are given in Table A6.1 (Appendix 6). This emission rate was used for determining the concentration of LBPN vapours in ambient air by SCREEN3 (1996).

In scenario I, a ship carrying an industry-restricted LBPN (CAS RN 64741-42-0) is assumed to release the total daily evaporative emission (kg/day) within a defined area (i.e., release from a stationary area source) in an urban setting. In scenarios IIa and IIb, the motion of the ship away from the port is considered a source of mobile evaporative transit emission. Schematic representations of scenarios IIa and IIb are shown in Figure A6.2 (Appendix 6).

For evaporative emission of industry-restricted LBPNs (CAS RNs 64741-42-0, 64741-69-1 and 64741-78-2) during truck transit, an average truck trip of 600 km is used, representing the distance travelled between industry facilities. This is subdivided into 2-hour travel in urban areas with a speed of 50 km/hour, 5-hour travel in rural areas with a speed of 100 km/hour and a 1-hour rest. The total travel time is thus 8 hours, and the daily evaporative emission is assumed to occur over these 8 hours. The exposures in these scenarios are amortized over a year. In scenario III, a tanker truck carrying industry-restricted LBPNs is assumed to remain idle for a 1-hour rest period (i.e., release from a stationary area source). Commonly, the locations at which a tanker truck would be idle are those that are shared with the general population in urban settings, although a rural setting scenario is also considered. A distance of 200 m for scenario III was confirmed to be a realistic minimum distance for the potential for general population exposure based on map analysis. In scenarios IV and V, it is assumed that the truck moves at 50 km/hour through local roadways in urban settings and 100 km/hour on the highway in rural settings, respectively (i.e., release from a line source; Appendix 6). Potential bystanders are considered to be located within distances of 200–1000 m from the highway, as determined by map analysis of the transportation routes. In scenario VI, the dispersion of vapours released during the loading and unloading of trucks at facilities to the general population living in the vicinity of these facility sites is considered. A distance of 1500 m for the closest proximity of the general population was based on analysis of the distance from the loading dock to the nearest residential area. Other assumptions were verified by industry.

SCREEN3 estimates the maximum concentrations for a given wind direction within 1 hour at various distances for a given population in the vicinity of the release source (i.e., from a ship or a truck loading in this screening assessment). The results of the modelled dispersion profile of the total VOCs in ambient air for the general population, for distances relevant for scenarios I–VI, as described above, are presented in Table 5. The estimated exposure from ship transportation for scenarios I, IIa and IIb relates to CAS RN 64741-42-0.

Table 5. Modelling results of industry-restricted LBPN dispersion in ambient air in Canada using SCREEN3[a]

Scenario	Maximum concentration within 1-h period (μg/m^3)		Maximum concentration with 24-h averaged wind direction (μg/m^3)[b]		Maximum concentration with 1-year averaged wind direction (μg/m^3)[c]	
	200 m	**1000 m**	**200 m**	**1000 m**	**200 m**	**1000 m**
I[d]	—[e]	68	—[e]	27	NA[f]	NA[f]
IIa[d]	—[e]	13	—[e]	5.2	NA[f]	NA[f]

IIb[d]	—[e]	16	—[e]	6.6	NA[f]	NA[f]
III[g]	14	0.92	5.6	0.37	2.8	0.18
IV[g]	0.0034	0.0015	0.0013	0.0006	0.0007	0.0003
V[g]	0.0089	0.0039	0.0035	0.0016	0.0018	0.0009
VI[g]	—[e]	213[h]	—[e]	85[h]	—[e]	43[h]

[a] The concentrations represent total VOCs.
[b] Concentration accounting for changing wind directions over a 24-h period.
[c] Concentration accounting for changing wind directions over a 1-year period.
[d] From CAS RN 64741-42-0; reported to be transported by ship.
[e] The distance is not applicable for this scenario.
[f] The exposure duration is not applicable for this scenario.
[g] From each of the three CAS RNs, which are transported in equal amounts.
[h] At 1500 m from the source; distance validated based on the location of the loading dock with respect to homes in the vicinity of the facility.

The SCREEN3 estimates derived for scenarios III–VI represent the potential for exposure to each of the three CAS RNs. All three of the substances being assessed were reported to be transported in equal amounts. Exposure estimates were modelled up to 3000 m or more, with results demonstrating that concentrations continue to decrease with increasing distance from the release source. It should be noted that CAS RN 64741-78-2 has a high boiling point (Table 1) and is expected to contribute to the VOC concentration only in the summer months.

With respect to scenarios I, IIa and IIb, evaporative emissions by ship transportation are represented by CAS RN 64741-42-0, which is the only industry-restricted LBPN reported to be transported by ship. With respect to scenarios III–VI, all three CAS RNs are transported by truck in approximately equal amounts. From Table 1, CAS RN 64741-78-2 is reported to have a high boiling point (65–230°C), and therefore this substance is expected to have the greatest potential for evaporative emission during the 3–4 hottest months of the year when temperatures near roads are high or light intensity becomes strong enough to dramatically heat up the truck storage tanks. CAS RNs 64741-42-0 and 64741-69-1 have lower boiling points and are more volatile. As such, these substances can be expected to contribute to the evaporative release of VOCs throughout the year.

Of the total VOC concentrations estimated in Table 5, the aromatic components are of greatest cause for concern regarding the potential for harm to human health. The percentage of aromatic compounds in each of the industry-restricted LBPNs is given in Table 1. Assuming that the compositions of the liquid and headspace gases of CAS RNs 64741-42-0, 64741-69-1 and 64741-78-2 are identical and that the aromatic fraction can be considered to be benzene, a high-hazard component of LBPNs, the estimated exposure concentrations of benzene that correspond to the values listed in Table 5 are presented in Table 6.

Table 6. Modelling results of the benzene dispersion profile from industry-restricted LBPNs in ambient air in Canada using SCREEN3[a]

Scenario		Maximum benzene concentration within 1-h period (μg/m^3)		Maximum benzene concentration with 24-h averaged wind direction (μg/m^3)[b]		Maximum benzene concentration with 1-year averaged wind direction (μg/m^3)[c]	
		200 m	1000 m	200 m	1000 m	200 m	1000 m
I[d]		—[e]	2.7	—[e]	1.1	NA[f]	NA[f]
IIa[d]		—[e]	0.52	—[e]	0.21	NA[f]	NA[f]
IIb[d]		—[e]	0.64	—[e]	0.26	NA[f]	NA[f]
III	64741-42-0	0.56	0.037	0.22	0.015	0.11	0.0072
	64741-69-1	3.6	0.24	1.4	0.096	0.73	0.047
	64741-78-2[g]	2.8	0.18	1.1	0.074	0.56	0.036
IV	64741-42-0	1.4×10^{-4}	6.0×10^{-5}	5.2×10^{-5}	2.4×10^{-5}	2.8×10^{-5}	1.2×10^{-5}
	64741-69-1	8.8×10^{-4}	3.9×10^{-4}	3.8×10^{-4}	1.6×10^{-4}	1.8×10^{-4}	7.8×10^{-5}
	64741-78-2[g]	6.8×10^{-4}	3.0×10^{-4}	2.6×10^{-4}	1.2×10^{-4}	1.4×10^{-4}	6.0×10^{-5}
V	64741-42-0	3.6×10^{-4}	1.6×10^{-4}	1.4×10^{-4}	6.4×10^{-5}	7.2×10^{-5}	7.8×10^{-5}
	64741-69-1	2.3×10^{-3}	1.0×10^{-3}	9.1×10^{-4}	4.2×10^{-4}	4.7×10^{-4}	2.3×10^{-4}
	64741-78-2[g]	1.8×10^{-3}	7.8×10^{-4}	7.0×10^{-4}	3.2×10^{-4}	3.6×10^{-4}	1.8×10^{-4}
VI	64741-42-0	—[e]	8.5[h]	—[e]	3.4[h]	—[e]	1.7[f]
	64741-69-1	—[e]	55[h]	—[e]	22[h]	—[e]	11[f]
	64741-78-2[g]	—[e]	43[h]	—[e]	17[h]	—[e]	8.6[f]

[a] Estimated ambient air concentrations represent the aromatic fractions reported for CAS RNs 64741-42-0, 64741-69-1 and 64741-78-2.
[b] Concentration accounting for changing wind directions over a 24-h period.
[c] Concentration accounting for changing wind directions over a 1-year period.
[d] From CAS RN 64741-42-0; reported to be transported by ship.
[e] The distance is not applicable for this scenario.
[f] The exposure duration is not applicable for this scenario.
[g] CAS RN 64741-78-2 has low volatility and is expected to release vapours only during warm summer months.
[h] At 1500 m from the source; distance validated based on the location of the loading dock with respect to homes in the vicinity of the facility.

As presented in Table 5, scenario I indicates that the total VOC concentration (from CAS RN 64741-42-0) at 1000 m from evaporative emissions by ship transportation is estimated to be 68 and 27 µg/m^3 for the 1-hour maximum and 24 hr average, respectively. This is the concentration that the general population, living at a distance of 1000 m from the port, could experience during the time that a ship is docked. As seen from the results of scenarios IIa and IIb, the concentration of LBPN vapours (from CAS RN 64741-42-0) in the vicinity of the moving release source (i.e., ship), for any given location, is considerably lower than that represented by the total daily release quantity from a stationary area release source. Furthermore, if there are two or more ports from which the LBPNs are shipped, the maximum exposure given in Table 5 would be further reduced in proportion to the total amount of LBPNs shipped from each site.

Although a larger amount of evaporative emissions (kg/hour) was estimated for marine transportation compared with transportation of LBPNs by truck (Table A6.3 in Appendix 6), the truck transportation scenarios represent a greater potential for exposure of the general population, because the roadways and highways that are used to move LBPNs between facility sites, as well as the rest locations where trucks are expected to idle for given periods of time, are shared with the general population and may be occupied by several trucks on a daily basis. Accordingly, additional focus for purposes of characterizing the risk to human health from the transportation of the industry-restricted LBPNs is on the truck scenarios; the potential locations for exposure of the general population through inhalation would be in closer proximity for road transportation than for marine transportation.

During truck transport of CAS RNs 64741-42-0, 64741-69-1 and 64741-78-2, an estimated evaporative emission of 140 kg/year was determined for each CAS RN (Table A6.3 in Appendix 6). Upper-bounding estimates for annual transportation of the reported volumes per CAS RN indicate that a total of 100–170 truck trips are required to transport each LBPN. The assumptions used in the estimations include an average of 100 truck trips per year and release occurring over an 8-hour travel time for each trip, 1 hour of which is considered to be a rest period. The assumption of 100 truck trips per year is considered to be the most conservative assumption, as the truck transport capacity will be larger and will give rise to greater evaporative emissions per trip.

In scenario III, the annualized emission rate ($g/s{\cdot}m^2$) from a stationary area (i.e., an idling truck) was estimated for a 1-hour rest period based on emission quantity (Table A6.2 in Appendix 6) and emission area (Table A6.1 in Appendix 6). The estimated exposures to total VOCs for scenario III are given in Table 5; they are based on the emission rate and other input variables given in Table A6.1. The LBPN concentrations in ambient air resulting from a truck idled for 1 hour, accounting for annual averaged wind directions, are estimated to be approximately 2.8 $\mu g/m^3$ at 200 m. The corresponding estimates of exposure concentrations of benzene from CAS RNs 6471-42-0, 64741-69-1 and 64741-78-2 are given in Table 6. Assuming a 26% benzene fraction for CAS RN 64741-69-1 as the upper-bounding estimate of exposure, the annual average benzene concentrations are estimated to be approximately 0.73 $\mu g/m^3$ at 200 m.

In scenarios IV and V, a line source calculation (input variables listed in Table A6.1 of Appendix 6) was applied to estimate the potential for exposure to these LBPNs from a moving truck travelling 50 km/hour (urban setting) or 100 km/hour (rural setting) on a highway for a 1-hour period. The air concentrations of total VOCs from these LBPNs at various distances due to the evaporative emissions from the truck were then estimated by SCREEN3; results are presented in Table 5. The annual concentrations were approximately 0.0007 µg/m^3 at 200 m in scenario IV, whereas the respective concentrations were estimated to be 0.0009 µg/m^3 at 1000 m in scenario V. The corresponding estimates of exposure concentrations of benzene from CAS RNs 64741-42-0, 64741-69-1 and 64741-78-2 are given in Table 6. Assuming a 26% benzene fraction for CAS RN 64741-69-1 as the upper-bounding estimate of exposure, the annual concentrations were approximately 1.8×10^{-4} µg/m^3 at 200 m in scenario IV, whereas the respective concentrations were estimated to be 2.3×10^{-4} µg/m^3 at 1000 m in scenario V.

With respect to scenario VI, the potential for exposure of the general population to CAS RNs 64741-42-0, 64741-69-1 and 64741-78-2 from evaporative emission during truck loading and unloading is considered. From Table A6.3 (Appendix 6), the estimated evaporative emission for each industry-restricted LBPN substance is 1050 kg/year. The upper-bounding estimates for annual transportation of the reported volume, as determined in scenario III, indicate that the estimated evaporative emission of 1050 kg/year corresponds to 100–170 truck loading/unloading events over the course of a year. Consistent with the number of truck trips assumed for the truck transportation scenarios, it was considered that 100 truck loading events was a conservative estimate for scenario VI. Additional parameters applied to this scenario include a vapour release area of 20×20 m^2 for the truck filling zone, a release height of 3 m and a time for completion of loading/unloading of 30 minutes (time required for loading verified by industry).

The emission rate during truck loading for scenario VI is calculated as follows:

$$\begin{aligned}\text{Emission rate} &= 1050 \times 10^3 \text{ g/year} \times 1 \text{ year/100 loading events} \times 1 \text{ loading event/}(30 \times 60 \text{ s}) \\ &= 5.83 \text{ g/s}\end{aligned}$$

Both urban and rural zone dispersions are considered in the SCREEN3 calculations for loading and unloading. Other details related to the SCREEN3 calculation for scenario VI are given in Table A6.1 (Appendix 6). For the urban zone dispersion, the maximum 1-hour and 24-hour release concentrations of total VOCs estimated by SCREEN3 during the loading and unloading of each of the three LBPNs are 213 µg/m^3 and 85 µg/m^3, respectively, at 1500 m (Table 5). Assuming a 26% benzene fraction for CAS RN 64741-69-1 as the upper-bounding estimate of exposure, the 1-hour and 24-hour average benzene concentrations are estimated to be approximately 55 µg/m^3 and 22 µg/m^3, respectively, at 1500 m (Table 6).

The estimated LBPN air concentrations described above and listed in Table 5 are considered to be conservative, as SCREEN3 is, by design, a conservative screening-level tool used as a rapid approach to estimate the air dispersion of various chemicals. Another consideration is that although a larger emission area is estimated for a moving truck, and thus the emission rate is significantly reduced, the potential air diffusion and vortex that occur around moving vehicles are not incorporated into the analysis by SCREEN3. The assumption that the receptor is located at various distances ranging from 200–1000 m from the release source adds an additional element of conservatism, as most Canadians are not expected to spend a significant amount of time in close proximity to these sources. This was verified for most rest stops along major highway corridors.

Health Effects Assessment

Given the lack of studies available that specifically evaluate the health effects of the industry-restricted LBPNs, an adequately representative toxicological dataset unique to these substances could not be obtained. Therefore, to characterize the health effects of these LBPNs, additional LBPNs in the PSSA that are similar from both a process and a physical-chemical perspective were also considered. Because both the industry-restricted and the additional LBPN substances have similar physical-chemical properties, their toxicological properties are likely similar. The health effects data were therefore pooled and used to construct a toxicological profile to represent all LBPNs. Accordingly, the health effects of LBPNs are represented as a group, not by individual CAS RNs. Despite pooling the data for similar LBPNs, there remained a limited number of identified studies for some health effects endpoints. Therefore, studies assessing unleaded gasoline (a highly-refined LBPN) were also reviewed and considered in this screening assessment. However, examination of data relevant to the composition of unleaded gasoline demonstrated that this is a highly regulated substance; it is expected to contain a lower percentage of benzene and has a discrete component profile when compared with other substances in the LBPN group. Given that gasoline will be reviewed separately in PSSA Stream 3, these studies were not used to characterize risk in the current screening assessment on industry-restricted LBPNs.

Appendix 7 contains a summary of available health effects information on LBPNs in laboratory animals. A summary of key studies is presented below.

LBPNs have low acute toxicity by the oral (median lethal dose [LD_{50}] in rats > 2000 mg/kg-bw), inhalation (LD_{50} in rats > 5000 mg/m^3) and dermal (LD_{50} in rabbits > 2000 mg/kg-bw) routes of exposure (CONCAWE 1992; Rodriguez and Dalbey 1994a, b, c, d; API 2008a). Most LBPNs are mild to moderate eye and skin irritants in rabbits, with the exception of heavy catalytic cracked and heavy catalytic reformed naphthas, which have higher primary skin irritation indices (API 1980a, 1985a, b, c, 1986a, b, c, d, 2008a; CONCAWE 1992; Rodriguez and Dalbey 1994e, f, g, h, i). LBPNs do not appear to be skin sensitizers, but a poor response in the positive control was also noted in these studies (API 1980a, 1985b, 1986a, b, c, d, e, f).

No short-term (2–89 days) or subchronic (> 90 days, < 2 years) studies were identified for industry-restricted LBPNs. The lowest-observed-adverse-effect concentration (LOAEC) and lowest-observed-adverse-effect level (LOAEL) values identified following short-term and subchronic exposure to additional LBPNs are listed in Table 7. These values were determined for a variety of endpoints after considering the health effects data for all LBPNs in the PSSA. Most of the studies were carried out by the inhalation route of exposure. Renal effects, including increased kidney weight, renal lesions (renal tubule dilatation, necrosis) and hyaline droplet formation, observed in male rats exposed to most LBPNs orally or by inhalation, were considered species and sex specific (Carpenter et al.

1975; Halder et al. 1984, 1985; Phillips and Egan 1984; Research and Environmental Division 1984; Gerin et al. 1988; Schreiner et al. 1998, 1999, 2000a; McKee et al. 2000; API 2005, 2008b, c). These effects were found to be due to an interaction between hydrocarbon metabolites and α-2-microglobulin, a protein not produced in substantial amounts in female rats, mice or other species, including humans. The resulting nephrotoxicity and subsequent carcinogenesis in male rats, as mediated through α-2-microglobulin, were therefore not considered in deriving LOAEC/LOAEL values, and are not applicable for the purpose of characterizing the risk to human health.

Table 7. LOAECs/LOAELs identified for a variety of endpoints in experimental animals following short-term or subchronic exposure to LBPNs

Route of exposure	Effects observed[a]	LOAEC/LOAEL	CAS RN	Reference
Inhalation	Inflammatory response of the respiratory tract	214 mg/m^3	8052-41-3	Riley et al. 1984
	Decreased survival	363 mg/m^3	8052-41-3	Rector et al. 1966
	Biochemical	575 mg/m^3	8052-41-3	Savolainen and Pfaffli 1982
	Decreased growth rate	1327 mg/m^3	64742-95-6	McKee et al. 1990
	Brain enzyme changes	1327 mg/m^3	Gasoline[b]	Chu et al. 2005
	Hematological	1800 mg/m^3	64742-95-6	Shell Research Ltd. 1980
	Oxidative stress in the liver	4679 mg/m^3	64742-48-9	Lam et al. 1994
Oral	Decreased growth rate; biochemical	500 mg/kg-bw per day	64742-95-6	Bio/Dynamics, Inc. 1991a
	Hematological	500 mg/kg-bw per day	64742-95-6	Bio/Dynamics, Inc. 1991b
Dermal	Skin irritation	30 mg/kg-bw per day	64741-55-5	Mobil 1988a
	Decreased growth rate	200 mg/kg-bw per day	64741-54-4	API 1986g
	Hematological	500 mg/kg-bw per day	64742-48-9	Zellers 1985
	Decreased survival	1000 mg/kg-bw per day	68955-35-1	API 1986h
	Biochemical	1500 mg/kg-bw per day	64742-48-9	Zellers 1985

[a] See Appendix 7 for additional details.

[b] Gasoline captures the following CAS RNs: 8006-61-9 and 86290-81-5.

No non-cancer chronic health effects studies (study duration ≥ 1 year) were found for industry-restricted LBPNs. Very few non-carcinogenic chronic health effects studies were identified for other LBPNs. A LOAEC of 200 mg/m^3 was noted in a chronic inhalation study that exposed mice and rats to unleaded gasoline (containing 2% benzene) at concentrations of 0, 200, 870 or 6170 mg/m^3. This inhalation LOAEC was based on ocular discharge and ocular irritation in rats. At the mid-exposure concentration of 870 mg/m^3, increased relative kidney weights were observed in male rats (MacFarland et al. 1984). A LOAEL of 694 mg/kg-bw was identified for dermal exposure based on local skin effects (inflammatory and degenerative skin changes) in mice following application of naphtha for 105 weeks. No systemic toxicity was reported (Clark et al. 1988).

Although no genotoxicity studies were identified for the industry-restricted LBPNs being assessed (CAS RNs 64741-42-0, 64741-69-1 and 64741-78-2), the genotoxic potential of several other LBPNs has been evaluated using a variety of *in vivo* and *in vitro* assays, as described below.

For *in vivo* genotoxicity tests, LBPNs exhibited negative results for chromosomal aberrations and micronuclei induction (Gochet et al. 1984; Khan 1984; Khan and Goode 1984; API 1985d, e, f, g, h, i, 1986i) but exhibited positive results in one sister chromatid exchange assay (API 1988a). Substances that were tested, which included a number of light naphthas, displayed mixed results (i.e., both positive and negative for the same assay) for chromosomal aberrations and negative results for the dominant lethal mutation assay (API 1977a). Unleaded gasoline (containing 2% benzene) was tested for its ability to induce unscheduled deoxyribonucleic acid (DNA) synthesis (UDS) and replicative DNA synthesis (RDS) in rodent hepatocytes and kidney cells. UDS and RDS were induced in mouse hepatocytes, and RDS was induced in rat kidney cells (Loury et al. 1986, 1987). Unleaded gasoline (benzene content not stated) exhibited negative results for chromosomal aberrations and the dominant lethal mutation assay (API 1977b, c, 1980b; Conaway et al. 1984; Dooley et al. 1988).

For *in vitro* genotoxicity studies, LBPNs were negative for seven out of eight Ames tests and were also negative for UDS and forward mutations (Blackburn 1981; Brecher 1984a; Brecher and Goode 1984a; Gochet et al. 1984; Papciak and Goode 1984; Blackburn et al. 1986, 1988; Riccio and Stewart 1991; Haworth 1978). LBPNs exhibited mixed and/or equivocal results for the mouse lymphoma and sister chromatid exchange assays, as well as for cell transformation (Jensen and Thilager 1978; Kirby et al. 1979; Roy 1981; Tu and Sivak 1981; Brecher 1984b; Brecher and Goode 1984b; Gochet et al. 1984; API 1985d, j, k, l, m, n, o, p, 1986j, k, l, 1987, 1988b). Substances that were tested, which included a number of light naphthas, displayed negative results for the Ames and mouse lymphoma assays (API 1977a). Gasoline exhibited negative results for the Ames test battery, the sister chromatid exchange assay and one mutagenicity assay (API 1977b; Farrow et al. 1983; Conaway et al. 1984; Richardson et al. 1986; Dooley et al. 1988). Mixed results were observed for UDS and the mouse lymphoma assay (API 1977b, 1988c; Farrow et al. 1983; Conaway et al. 1984; Loury et al. 1986, 1987; Dooley et al. 1988).

LBPNs appear to demonstrate limited evidence of genotoxicity, as the majority of *in vivo* and *in vitro* assays displayed negative results. However, the potential for genotoxicity cannot be discounted based on the mixed results observed for some assays, particularly from the *in vitro* data, as well as the incomplete test battery.

The European Commission, United Nations (UN) and International Agency for Research on Cancer (IARC) have classified the industry-restricted LBPNs as carcinogenic. The European Commission has classified these substances as Category 2 carcinogens (R45: *may cause cancer*; benzene content ≥ 0.1% by weight) (ESIS 2008; European Commission 2008a), and has since reclassified them as Category 1B carcinogens under the new UN Globally Harmonized System of Classification and Labelling of Chemicals in 2009 (H350: *may cause cancer*; benzene content ≥ 0.1% by weight) (European Commission 2008b, 2009). IARC has classified "occupational exposures in petroleum refining" as Group 2A carcinogens (*probably carcinogenic to humans*). In this classification, several LBPNs, including one industry-restricted CAS RN, were included: 64741-41-9, 64741-46-4, 64741-54-4, 64741-55-5, 64741-63-5, 64741-64-6, 64741-68-0, 64741-69-1, 64741-74-8, 64742-82-1, 68410-05-9 and 68919-37-9 (IARC 1989a).

No studies assessing the carcinogenicity of industry-restricted LBPNs via inhalation, the main route of exposure for the general population, were identified. Only unleaded gasoline has been examined for its carcinogenic potential in several inhalation studies (MacFarland et al. 1984; Short et al. 1989; Standeven and Goldsworthy 1993; Standeven et al. 1994, 1995). While there was an increased incidence of hepatocellular and renal tumours observed (Appendix 7), as stated above, the highly-refined nature of gasoline indicates that this substance is compositionally different than other LBPNs. Given that gasoline will be reviewed separately in PSSA Stream 3, these cancer studies were not used to characterize risk in the current screening assessment on industry-restricted LBPNs.

All LBPNs potentially contain the volatile component benzene. The most likely average benzene concentration in naphthas is approximately 1%, but measured benzene concentrations ranged from non-detectable in isomerized naphthas to 20% in reformates (UN 2009). Benzene was assessed by Health Canada (Canada 1993) and was determined to be harmful to human health based on carcinogenicity. Other organizations have drawn similar conclusions. For example, IARC has classified benzene as a Group 1 carcinogen (*carcinogenic to humans*) (IARC 2011), and the European Commission has recommended that all LBPNs containing ≥ 0.1% benzene by weight be classified as Category 1B carcinogens (classified as Category 2 carcinogens prior to 2009), even in the absence of stream-specific experimental animal data (ESIS 2008; European Commission 2008a, b, 2009). These conclusions are consistent with the approach used by Health Canada to categorize petroleum streams during the categorization exercise conducted for substances on the DSL under CEPA 1999 (Health Canada 2008).

Given the absence of LBPN-specific studies assessing carcinogenicity via inhalation, the potential for carcinogenicity can be assessed by considering the cancer risk associated with potential exposure to the high-hazard component benzene. The Government of Canada has previously developed estimates of carcinogenic potency associated with inhalation exposure to benzene. A tumorigenic concentration (TC_{05}) was calculated as 14.7×10^3 µg/m^3 from the epidemiological investigation of Rinsky et al. (1987) based on acute myelogenous leukemia and a linear-quadratic exposure-response model (Canada 1993). The U.S. EPA (2000) quantified the cancer potency from inhalation exposure to benzene using low-dose linearity maximum likelihood estimates based on the same epidemiological study of Pliofilm workers (Rinsky et al. 1981, 1987) that was the basis for the TC_{05} reported by the Government of Canada (U.S. EPA 2000).

Several studies were conducted using experimental animals to investigate the dermal carcinogenicity of LBPNs. The majority of these studies were conducted through exposure of mice to doses ranging from 694–1351 mg/kg-bw for durations ranging from 1 year to life or until a tumour persisted for 2 weeks. Given the route of exposure, the studies specifically examined the formation of skin tumours. Results for carcinogenicity via dermal exposure are mixed. Both malignant and benign skin tumours were induced with heavy catalytic cracked naphtha, light catalytic cracked naphtha, light straight-run naphtha and petroleum naphtha (Blackburn et al. 1986, 1988; Witschi et al. 1987; Clark et al. 1988; Broddle et al. 1996). Significant increases in squamous cell carcinomas were also observed when mice were dermally treated with Stoddard solvent (U.S. EPA 1984), but the latter was administered as a mixture (90% test substance), and the details of the study were not available. In contrast, insignificant increases in tumour formation or no tumours were observed when light alkylate naphtha, heavy catalytic reformed naphtha, sweetened naphtha or light catalytically cracked naphtha was dermally applied to mice (API 1986m, n, o; Skisak et al. 1994; Broddle et al. 1996). Negative results for skin tumours were also observed in male mice dermally exposed to sweetened naphtha using an initiation/promotion protocol (Skisak et al. 1994; API 1988d).

Therefore, after consideration of the carcinogenicity data set, there is limited evidence of carcinogenicity in rats and mice after dermal exposure to LBPNs.

No reproductive or developmental toxicity was observed for the majority of LBPNs evaluated. Most of these studies were carried out by inhalation exposure in rodents.

No-observed-adverse-effect concentration (NOAEC) values for reproductive toxicity following inhalation exposure ranged from 1701 mg/m^3 (CAS RN 8052-41-3) to 27 687 mg/m^3 (CAS 64741-63-5) for the LBPNs evaluated (API 1978, 2008a, b, c, d; Litton Bionetics 1978, 1980; Phillips and Egan 1981; Schreiner 1984; Dalbey et al. 1996; Dalbey and Feuston 1996; Bui et al. 1998; Schreiner et al. 1999, 2000b; Roberts et al. 2001). However, a decreased number of pups per litter and a higher frequency of post-implantation loss were observed following inhalation exposure of female rats to hydrotreated heavy naphtha (CAS RN 64742-48-9) at a concentration of 4679 mg/m^3 for 6 hours/day from gestation days 7–20 (Hass et al. 2001). For dermal exposures, no-observed-adverse-effect level (NOAEL) values of 694 mg/kg-bw per day (CAS RN 8030-30-6) and 1000 mg/kg-bw per day (CAS RN 68513-02-0) were noted (Clark et al. 1988; ARCO 1994). For oral exposures, no adverse effects on reproductive parameters were reported when rats were given light catalytic cracked naphtha at 2000 mg/kg-bw on gestation day 13 (Stonybrook Laboratories 1995).

For most LBPNs, no treatment-related developmental effects were observed by different routes of exposure (API 1977d, 1978, 2008a, b, c, d; Litton Bionetics 1978; Miller and Schardein 1981; Phillips and Egan 1981; Schreiner 1984; Mobil 1988b; ARCO 1994;

Stonybrook Laboratories 1995; Dalbey et al. 1996; Dalbey and Feuston 1996; Bui et al. 1998; Schreiner et al. 1999, 2000b; Roberts et al. 2001). However, developmental toxicity was observed for a few naphthas. Decreased fetal body weight and an increased incidence of ossification variations were observed when rat dams were exposed to light aromatized solvent naphtha by gavage at 1250 mg/kg-bw per day (Bio/Dynamics, Inc. 1991c). In addition, pregnant rats exposed by inhalation to hydrotreated heavy naphtha at 4679 mg/m^3 delivered pups with higher birth weights. Cognitive and memory impairments were also observed in the offspring (Hass et al. 2001).

While a number of older epidemiological studies have reported increases in the incidence of a variety of cancers, the majority of these studies are considered to contain incomplete or inadequate information. Limited data, however, are available for skin cancer and leukemia incidence and mortality among petroleum refinery workers (Hendricks et al. 1959; Lione and Denholm 1959; McCraw et al. 1985; Divine and Barron 1986; Nelson et al. 1987; Wong and Raabe 1989). IARC (1989a) therefore concluded that there is limited evidence supporting the view that working in petroleum refineries entails a carcinogenic risk (Group 2A carcinogen). The available evidence gathered from these epidemiological studies, while showing an association between working in a refinery and the noted health effect, is nevertheless considered to be inadequate to conclude that these effects in the general population are due solely to exposures to LBPNs.

Characterization of Risk to Human Health

Industry-restricted LBPNs were identified as high priorities for action during categorization of the DSL because they were determined to present greatest potential or intermediate potential for exposure of individuals in Canada, and were considered to present a high hazard to human health. A critical effect for the initial categorization of industry-restricted LBPNs was carcinogenicity, based primarily on classifications by international agencies. These substances are classified as Group 2A carcinogens (IARC 1989a) and are further classified as Category 1B carcinogens (classified as Category 2 carcinogens prior to 2009) when the concentration of benzene is ≥ 0.1% by weight (ESIS 2008; European Commission 2008a, b, 2009). However, given the absence of LBPN-specific studies assessing carcinogenicity via inhalation—the main route of exposure for the general population—the potential for carcinogenicity was assessed by considering the cancer risk associated with potential exposure to the high-hazard component benzene. Benzene can be present at higher concentrations in LBPNs than in highly refined gasoline, which is restricted to 1.5% benzene by volume (*Benzene in Gasoline Regulations*) (Canada 1997). Benzene has been assessed by Health Canada under CEPA 1999 (Canada 1993) and was determined to be carcinogenic. Benzene was added to the List of Toxic Substances in Schedule 1 of the Act.

The estimates of carcinogenic potency for benzene developed by the Government of Canada (Canada 1993) were used to calculate margins of exposure (MOEs) associated with chronic exposure to benzene from industry-restricted LBPNs. Information obtained indicates that ships spend a limited number of days per month in port or moving to and from port; therefore, estimating carcinogenic risk for scenarios I and II is not relevant.

With respect to scenarios III–V, the annual upper-bounding estimates of inhalation exposure (µg/m^3) to benzene in ambient air from truck transportation of industry-restricted LBPNs (specifically CAS RN 64741-69-1, reported as having the highest aromatic content of these substances, at 26%) between industry facilities were compared with the tumorigenic concentration (TC$_{05}$) of 14.7 × 10^3 µg/m^3 for benzene. The TC$_{05}$ value is the concentration of a substance in air associated with a 5% increase in incidence of or mortality from tumours (Health Canada 1996). The resulting MOEs were calculated for the general population for a defined distance from the release source. Table 8 lists the MOEs in the vicinity of trucking corridors as defined by each assessment scenario. Reasonably, the MOEs would be greater if the lower benzene concentrations of the other two CAS RNs were considered instead of CAS RN 64741-69-1.

Table 8. Margins of exposure based on air dispersion modelling of industry-restricted LBPNs during truck transport based on the aromatic content of CAS RN 64741-69-1

Scenario	Transportation mode	Distance of bystander from release source (m)	Annual upper-bounding estimates of exposure (µg/m^3)	MOE based on TC$_{05}$ of 14.7 × 10^3 µg/m^3 (Canada 1993)
III	Trucks idled for 1 h	200	0.73	20 100
IV	Trucks moving intra-city (50 km/h)	200	1.8×10^{-4}	81 700 000
V	Trucks moving inter-city (100 km/h)	1000	2.3×10^{-4}	63 900 000

Comparison of the annual upper-bounding estimate of exposure for a tanker truck carrying industry-restricted LBPNs, which is assumed to remain idle in proximity to the general population (i.e., scenario III; release from a stationary area source), with the inhalation tumorigenic concentration for benzene gives an MOE of approximately 20 000. This conservative assumption also considers that a tanker truck would be present in the vicinity of the general population chronically throughout the year. This MOE is considered adequately protective of human health. The calculated risks for scenarios IV and V, where trucks are considered to be in transit, range from 81 700 000–63 900 000 and are also considered adequately protective of human health.

With respect to non-cancer effects via inhalation exposure, scenarios I, II and VI can be considered. A LOAEC for short-term exposure to LBPNs was determined to be 214 mg/m^3 based on an inflammatory response of the respiratory tract in mice exposed to Stoddard solvent (CAS 8052-41-3) for 4 days (Riley et al. 1984). Regarding scenarios I and II, comparison of this LOAEC with the estimated 24-hour concentrations of total VOCs resulting from the transportation of industry-restricted LBPNs (specifically CAS RN 64741-42-0) by ship, including in port and travelling parallel or perpendicular to the port at a distance of 1000 m (27, 5.2 and 6.6 µg/m^3, respectively, for scenarios I, IIa and IIb), results in MOEs ranging from approximately 8000–41 000. These margins for short-term exposures are considered adequately protective of human health to address short-term or subchronic non-cancer effects, especially in light of the highly conservative nature of the estimated exposures and the fact that ships spend a limited number of days per month in port or moving to and from port. Assuming a 4% benzene fraction for CAS

RN 64741-42-0, the estimated 24-hour concentrations for ship transportation are 1.1, 0.21 and 0.26 µg/m^3, respectively, for scenarios I, IIa and IIb. Comparison of these concentrations with the critical non-neoplastic effect level of 32 mg/m^3 for short-term (6 hours/day for 6 days) inhalation exposure to benzene based on immunological effects in male mice (Canada 1993) results in MOEs ranging from 29 000–152 000, corroborating the conclusion that the short-term margins are adequately protective of human health.

During truck loading/unloading, the estimated 24-hour release concentration of total VOCs is 85 µg/m^3 at 1500 m from the release source, a distance validated based on the location of the loading dock with respect to homes in the vicinity of the facility. Comparison of this estimate of exposure with the short-term LOAEC for exposure to LBPNs of 214 mg/m^3 for Stoddard solvent (CAS 8052-41-3) (Riley et al. 1984), as described above, results in an MOE of approximately 2500. Taking into consideration a maximum 24-hour benzene exposure concentration of 22 µg/m^3 (assuming 26% aromatic fraction and a 30-minute exposure every 3 days) and comparing it with the critical non-neoplastic effect level of 32 mg/m^3 for short-term inhalation exposure to benzene (Canada 1993) gives a calculated MOE of approximately 1500. These values are both considered adequately protective of human health to address short-term or subchronic non-cancer effects and are conservative given that the potential for exposure during loading/unloading events is considered to occur in pulses.

Uncertainties in Evaluation of Human Health Risk

The PSSA screening assessments evaluate substances that are complex combinations of hydrocarbons (UVCBs) composed of a number of substances in various proportions due to the source of the crude oil, bitumen or natural gas and its subsequent processing. Monitoring information or provincial limits on releases from petroleum facilities target broad releases, such as oils and grease, to water or air. These widely encompassing release categories do not allow for detection of individual complex mixtures or production streams. As such, the monitoring of broad releases cannot provide sufficient data to associate a detected release with a specific substance identified by a CAS RN, nor can the proportion of releases attributed to individual CAS RNs be defined.

There is uncertainty regarding the characterization of human exposure to LBPNs because of the lack of atmospheric monitoring data and the use of estimated atmospheric concentrations derived using SCREEN3 modelling, which requires limited input parameters and non-site-specific meteorological data to produce estimates. This may introduce more uncertainty relative to complex dispersion models. Assumptions made in SCREEN3 (Tables A6.1 and A6.2 in Appendix 6) may also contribute to the uncertainty.

Uncertainty exists due to the paucity of data available regarding the physical-chemical properties of certain CAS RNs. The densities of several LBPNs were not provided in the health effects studies; thus, these properties were often obtained from alternative sources.

As the industry-restricted LBPNs are UVCBs, their specific compositions are not well defined. Across the industry, the composition of LBPN streams identified by the same

CAS RN can vary significantly in the number, identity and proportions of components, depending on operating conditions, feedstocks and processing units. Consequently, it is difficult to obtain a truly representative toxicological dataset for individual LBPNs. For this reason, all available health effects data on LBPNs were pooled across the CAS RNs to develop a comprehensive toxicological profile.

The lack of chronic toxicity and carcinogenicity studies for LBPNs (with the exception of unleaded gasoline) by the inhalation route, the principal route of exposure for the general population, also constitutes a source of uncertainty. In addition, no dose-response relationship could be evaluated in the dermal carcinogenicity studies, because typically only one dose of the test substance was used.

Uncertainty also exists because certain details of the laboratory animals (i.e., sex, strain, body weight and minute volume) were often not stated in the health effects studies and were obtained from laboratory standard data. Thus, those characteristics may not be entirely representative of the physical features of the test animals used in each particular study.

Uncertainty also exists because empirical equations were used to estimate evaporative emission. It is noted that during transit, evaporative emissions from the transport vessels also vary with physical conditions, such as tightness of containers or settings of valves. The screening estimation of evaporative emission does not account for these influences.

Conclusion

Based on results of comparison of levels expected to cause harm to organisms with estimated exposure levels, releases to soil from loading, unloading and transport may cause harm to terrestrial organisms. However, given the conditions of such releases and the overall low frequency of spills to land (on average <1 per year), the risk of harm to the environment from spills of these industry-restricted LBPNs is low. As well, the risk of harm to terrestrial organisms from releases of LBPNs to air from ship and truck loading is low. These LBPNs also pose a low risk of harm to aquatic organisms.

Based on the information presented in this screening assessment on the frequency and magnitude of spills, there is low risk of harm to organisms or the broader integrity of the environment from these substances. It is concluded that the three industry-restricted LBPNs (CAS RNs 64741-42-0, 64741-69-1 and 64741-78-2) do not meet the criteria under paragraph 64(a) or 64(b) of the *Canadian Environmental Protection Act, 1999* (CEPA 1999) as they are not entering the environment in a quantity or concentration or under conditions that have or may have an immediate or long-term harmful effect on the environment or its biological diversity or that constitute or may constitute a danger to the environment on which life depends.

Exposure of the general population to the industry-restricted LBPNs via the dermal and oral routes is not expected. The critical effect for characterization of risk to human health for the LBPNs considered in this assessment is carcinogenicity via the inhalation route of exposure, based on the presence of benzene, a high-hazard component. On the basis of the adequacy of the MOEs for the upper-bounding estimates of potential chronic exposures of the general population to benzene as a result of the transportation of the industry-restricted LBPNs by truck, as well as adequate margins for short-term exposures during ship transportation and truck loading/unloading, it is concluded that the three industry-restricted LBPNs (CAS RNs 64741-42-0, 64741-69-1 and 64741-78-2) do not meet the criteria under paragraph 64(*c*) of the *Canadian Environmental Protection Act, 1999* (CEPA 1999) as they are not entering the environment in a quantity or concentration or under conditions that constitute or may constitute a danger in Canada to human life or health.

It is therefore concluded that these three industry-restricted LBPNs listed under CAS RNs 64741-42-0, 64741-69-1 and 64741-78-2 do not meet any of the criteria set out in section 64 of CEPA 1999.

References

[AOPWIN] Atmospheric Oxidation Program for Microsoft Windows [Estimation Model]. 2008. Version 1.92a. 2008. Washington (DC): U.S. Environmental Protection Agency, Office of Pollution Prevention and Toxics; Syracuse (NY): Syracuse Research Corporation. Available from: www.epa.gov/oppt/exposure/pubs/episuite.htm

[API] American Petroleum Institute. 1977a. Letter from American Petroleum Institute to US Environmental Protection Agency regarding submission of 8(E) information on 3 commercial solvents with attachments. Washington (DC): API. EPA/OTS Document No. 88-7700010; NTIS/OTS0200393. [Abstract]. [cited in TOXLINE 2009].

[API] American Petroleum Institute. 1977b. Mutagenicity evaluation of unleaded gasoline (L5178Y mouse lymphoma assay and Ames test). Washington (DC): API. API Report No.: 28-30173. [cited in API 2001a, 2008a].

[API] American Petroleum Institute. 1977c. Rat bone marrow cytogenesis analysis, unleaded gasoline (5 daily intraperitoneal doses). Washington (DC): API. API Report No.: 26-60099. [cited in CONCAWE 1992].

[API] American Petroleum Institute. 1977d. Teratology study in rats. Stoddard solvent final report. Washington (DC): API. Report No.: FYI-AX-0183-0232 IN. [cited in ATSDR 1995a].

[API] American Petroleum Institute. 1978. Teratology study in rats, unleaded gasoline. Washington (DC): API. API Report No.: 26-60014. [cited in API 2008a].

[API] American Petroleum Institute. 1980a. Acute toxicity tests. API PS-6 unleaded motor gasoline. Washington (DC): API. API Report No.: 27-32130. [cited in CONCAWE 1992; API 2008a].

[API] American Petroleum Institute. 1980b. Mutagenicity evaluation of gasoline, API PS-6 fuel in the mouse dominant lethal assay. Washington (DC): API. API Report No.: 28-31344. [cited in CONCAWE 1992; API 2001a, 2008a].

[API] American Petroleum Institute. 1985a. Acute oral toxicity study in rats, acute dermal toxicity study in rabbits, primary dermal irritation study in rabbits, primary eye irritation study in rabbits, API 83-05 full range catalytic reformed naphtha (CAS #68955-35-1). Washington (DC): API. API Report No.: 32-31474. [cited in CONCAWE 1992; API 2008a].

[API] American Petroleum Institute. 1985b. Acute oral toxicity study in rats, acute dermal toxicity study in rabbits, primary dermal irritation study in rabbits, primary eye irritation study in rabbits, dermal sensitization study in guinea pigs. API sample 83-06 heavy catalytically reformed naphtha (CAS #64741-68-0). Washington (DC): API. API Health and Environmental Sciences Department Report No.: 32-32860. [cited in CONCAWE 1992].

[API] American Petroleum Institute. 1985c. Acute oral toxicity study in rats, acute dermal toxicity study in rabbits, primary dermal irritation study in rabbits and primary eye irritation study in rabbits of API sample 83-04 light catalytically reformed naphtha. Washington (DC): API. API Medical Research Publication No.: 32-31473. [cited in CONCAWE 1992].

[API] American Petroleum Institute. 1985d. Mutagenic evaluation studies in the bone marrow cytogenetics assay (inhalation) and in the mouse lymphoma forward mutation assay, light catalytic cracked naphtha 81-03. Washington (DC): API. API Report No.: 32-31300. [cited in CONCAWE 1992; API 2003b, 2008a].

[API] American Petroleum Institute. 1985e. Acute *in vivo* cytogenetics assay in male and female rats of API sample 83-19, light alkylate naphtha. Washington (DC): API. API Report No.: 32-32409. [cited in CONCAWE 1992; API 2003c, 2008a].

[API] American Petroleum Institute. 1985f. Activity of API 81-04, light catalytic cracked naphtha, in the acute (IP) *in vivo* cytogenetics assay in male and female rats. Washington (DC): API. API Report No.: 32-32288. [cited in CONCAWE 1992; API 2003b, 2008a].

[API] American Petroleum Institute. 1985g. Mutagenicity evaluation of 83-04 (light catalytic reformed naphtha) in the bone marrow cytogenetics assay (IP). Washington (DC): API. API Report No.: 33-31092. [cited in CONCAWE 1992; API 2003a, 2008a].

[API] American Petroleum Institute. 1985h. Mutagenicity evaluation of 83-06 (heavy catalytic reformed naphtha) in the bone marrow cytogenetics assay (IP). Washington (DC): API. API Report No.: 32-30494. [cited in API 2003a, 2008a].

[API] American Petroleum Institute. 1985i. Mutagenicity evaluation of 83-05 (full range catalytic reformed naphtha) in the bone marrow cytogenetics assay (IP). Washington (DC): API. API Report No.: 32-32289. [cited in CONCAWE 1992; API 2003a, 2008a].

[API] American Petroleum Institute. 1985j. L5178Y +/− mouse lymphoma assay, API 81-08 sweetened naphtha. Washington (DC): API. API Report No.: 32-31233. [cited in CONCAWE 1992; API 2003d, 2008a].

[API] American Petroleum Institute. 1985k. L5178Y +/− mouse lymphoma assay, API 83-19 light alkylate naphtha. Washington (DC): API. API Report No.: 32-32746. [cited in CONCAWE 1992; API 2003c, 2008a].

[API] American Petroleum Institute. 1985l. Mutagenicity evaluation of API sample 83-04 in the mouse lymphoma forward mutation assay. Final report. Washington (DC): API. API Medical Research Publication No.: 32-32168. [cited in CONCAWE 1992; API 2003a, 2008a].

[API] American Petroleum Institute. 1985m. L5178Y +/− mouse lymphoma assay, API 83-16 heavy catalytic reformed naphtha. Washington (DC): API. API Report No.: 32-32460. [cited in API 2008a].

[API] American Petroleum Institute. 1985n. Mutagenicity evaluation in the mouse lymphoma forward mutation assay, API 83-06 heavy catalytically reformed naphtha. Washington (DC): API. API Report No.: 33-32640. [cited in API 2003a].

[API] American Petroleum Institute. 1985o. L5178Y TK +/− mouse lymphoma assay of API 81-04. Washington (DC): API. API Medical Research Publication No.: 32-31710. [cited in CONCAWE 1992; API 2003b].

[API] American Petroleum Institute. 1985p. L5178Y +/− mouse lymphoma assay, API 83-05 full range catalytic reformed naphtha. Washington (DC): API. API Report No.: 32-32459. [cited in CONCAWE 1992; API 2003a, 2008a].

[API] American Petroleum Institute. 1986a. Acute oral toxicity in rats, acute dermal toxicity study in rabbits, primary dermal irritation study in rabbits, primary eye irritation study in rabbits, dermal sensitization study in guinea pigs on API 83-19, light alkylate naphtha (CAS #64741-66-8). Washington (DC): API. API Report No.: 33-30594. [cited in CONCAWE 1992; API 2008a].

[API] American Petroleum Institute. 1986b. Acute oral toxicity in rats, acute dermal toxicity study in rabbits, primary dermal irritation study in rabbits, primary eye irritation study in rabbits, dermal sensitization study in guinea pigs on API 83-20, light catalytic cracked naphtha (CAS #64741-55-5). Washington (DC): API. API Report No.: 33-32722. [cited in CONCAWE 1992; cited in API 2008a].

[API] American Petroleum Institute. 1986c. Acute oral toxicity in rats, acute dermal toxicity study in rabbits, primary dermal irritation study in rabbits, primary eye irritation study in rabbits, dermal sensitization study in guinea pigs on API 81-08, sweetened naphtha (CAS #64741-87-3). Washington (DC): API. API Report No.: 30-31990. [cited in CONCAWE 1992; API 2008a].

[API] American Petroleum Institute. 1986d. Acute oral toxicity in rats, acute dermal toxicity study in rabbits, primary dermal irritation study in rabbits, primary eye irritation study in rabbits, dermal sensitization study in guinea pigs. API sample 83-18 heavy catalytically cracked naphtha (CAS #64741-54-4). Final report. Washington (DC): API. API Health and Environmental Sciences Department Report No.: 33-30593. [cited in CONCAWE 1992].

[API] American Petroleum Institute. 1986e. Dermal sensitization study in guinea pigs with full range catalytic reformed naphtha (API 83-05). Washington (DC): API. API Report No.: 33-30497. [cited in CONCAWE 1992; API 2008a].

[API] American Petroleum Institute. 1986f. Dermal sensitization study in guinea pigs. API sample 83-04 light catalytically cracked reformed naphtha (CAS 64741-63-5). Final report. Washington (DC): API. API Health and Environmental Sciences Department Report No.: 33-30496. [cited in CONCAWE 1992].

[API] American Petroleum Institute. 1986g. 28-day dermal toxicity study in the rabbit. API sample 83-18 heavy catalytically cracked naphtha (CAS 64741-54-4). Study conducted by Tegeris Laboratories. Washington (DC): API. API Medical Research Publication No.: 32-32748. [cited in CONCAWE 1992].

[API] American Petroleum Institute. 1986h. 28-day dermal toxicity study in the rabbit. API sample 83-05 full range catalytically reformed naphtha (CAS 68955-35-1). Study conducted by Tegeris Laboratories. Washington (DC): API. API Health and Environmental Sciences Department Report No.: 33-30598. [cited in CONCAWE 1992].

[API] American Petroleum Institute. 1986i. Mutagenicity evaluation in the bone marrow cytogenetics assay with API 81-08 (IP). Washington (DC): API. API Report No.: 33-31093. [cited in CONCAWE 1992; API 2003d, 2008a].

[API] American Petroleum Institute. 1986j. L5178Y TK +/− mouse lymphoma assay. API 83-06 heavy catalytically reformed naphtha (CAS 64741-68-0). Washington (DC): API. API Report No.: 33-31641. [cited in API 2003a].

[API] American Petroleum Institute. 1986k. L5178Y +/− mouse lymphoma assay, API 81-04 light catalytic cracked naphtha. Washington (DC): API. API Report No.: 32-30498. [cited in API 2008a].

[API] American Petroleum Institute. 1986l. Mutagenicity of API sample 83-18 heavy catalytic cracked naphtha (petroleum) (CAS 64741-54-4) in a mouse lymphoma mutation assay. Final report. Washington (DC): API. API Health and Environmental Sciences Department Report No.: 33-32804. [cited in CONCAWE 1992].

[API] American Petroleum Institute. 1986m. Lifetime dermal carcinogenesis/chronic toxicity screening bioassay of refinery streams in C3H/HeJ mice. Twelve month toxicity evaluation report. API Study No. PS-36. PRI Study No. API-135r. Washington (DC): API.

[API] American Petroleum Institute. 1986n. Lifetime dermal carcinogenesis/chronic toxicity screening bioassay of refinery streams in C3H/HeJ mice. Twenty-fourth month progress report (weeks 101–104). API Study No. PS-36. PRI Study No. AP-135r. Washington (DC): API.

[API] American Petroleum Institute. 1986o. Lifetime dermal carcinogenesis/chronic toxicity screening bioassay of refinery streams in C3H/HeJ mice. Thirty-first month progress report (weeks 131–134). API Study No. PS-36. PRI Study No. AP-135r. Washington (DC): API.

[API] American Petroleum Institute. 1987. L5178Y +/− mouse lymphoma assay, API 83-20 light catalytic cracked naphtha. Washington (DC): API. API Report No.: 34-30633. [cited in CONCAWE 1992; API 2003b, 2008a].

[API] American Petroleum Institute. 1988a. *In vivo* sister chromatid exchange assay in B6C3F1 mice, with API 81-03 (light catalytic cracked naphtha). Washington (DC): API. API Report No.: 36-30044. [cited in CONCAWE 1992; API 2003b, 2008a].

[API] American Petroleum Institute. 1988b. Sister chromatid exchange assay in Chinese hamster ovary (CHO) cells with API 81-03 (light catalytic cracked naphtha). Washington (DC): API. API Report No.: 36-30045. [cited in CONCAWE 1992; API 2003b, 2008a].

[API] American Petroleum Institute. 1988c. *In vitro* unscheduled DNA synthesis in rat hepatocytes—gasoline. Washington (DC): API. API Report No.: 35-32431. [cited in API 2008a].

[API] American Petroleum Institute. 1988d. Short term dermal tumorigenesis study of selected petroleum hydrocarbons in male CD-1 mice initiation and promotion phases (draft final report) with attachments and cover letter 052688. Washington (DC): API. NTIS/OTS0000547-1.

[API] American Petroleum Institute. 2001a. Robust summary of information on paraffinic naphthas, gasoline, naphthenic naphthas, olefinic naphthas, aromatic naphthas. Washington (DC): API. Report No.: AR201-13409B. Available from: www.epa.gov/hpv/pubs/summaries/gasnecat/c13409rs.pdf

[API] American Petroleum Institute. 2001b. Robust summary of information on group 6: low benzene naphthas. Washington (DC): API. Report No.: AR201-13437B. Available from: www.epa.gov/chemrtk/pubs/summaries/lowbenze/c13437rs.pdf

[API] American Petroleum Institute. 2003a. Robust summary of information on aromatic naphthas. Washington (DC): API. Available from: www.petroleumhpv.org/docs/gasoline/052003_gasoline_robustsummary_anaphthas_revisedfinal.pdf

[API] American Petroleum Institute. 2003b. Robust summary of information on olefinic naphthas. Washington (DC): API. Available from: www.petroleumhpv.org/docs/gasoline/052003_gasoline_robustsummary_onaphthas_revisedfinal.pdf

[API] American Petroleum Institute. 2003c. Robust summary of information on paraffinic naphthas. Washington (DC): API. Available from: www.petroleumhpv.org/docs/gasoline/052003_gasoline_robustsummary_pnaphthas_revisedfinal.pdf

[API] American Petroleum Institute. 2003d. Robust summary of information on naphthenic naphthas. Washington (DC): API. Available from: www.petroleumhpv.org/docs/gasoline/052003_gasoline_robustsummary_nnaphthas_revisedfinal.pdf

[API] American Petroleum Institute. 2005. Baseline gasoline vapor condensate. Micronucleus assay in a 13 week whole-body inhalation toxicity study in rats with neurotoxicity assessments and 4-week *in vivo* genotoxicity and immunotoxicity assessments. HLS Study No. 00-6125, vol. IV, Appendix X. East Millstone (NJ): Huntington Life Sciences Laboratories; and Suffolk (GB): Huntington Eye Research Centre. [cited in API 2008a].

[API] American Petroleum Institute. 2008a. Gasoline blending streams category assessment document. Final 8-21-08. Submitted to the US Environmental Protection Agency. Consortium Registration No. 1100997. Washington (DC): API, Petroleum HPV Testing Group. Available from: www.petroleumhpv.org/docs/gasoline/2008_aug21_gasoline_catanalysis_final_category_assess_doc.pdf

[API] American Petroleum Institute. 2008b. OECD 422 inhalation combined repeated dose toxicity study with the reproductive/developmental toxicity screening test of heavy straight run naphtha (CAS # 64741-41-9). Project No. DuPont-18331. Wilmington (DE): Haskell Laboratories. [cited in API 2008a].

[API] American Petroleum Institute. 2008c. Baseline gasoline vapor condensate. A 2-generation whole-body inhalation reproductive study in rats. HLS Study No. 00-4207. East Millstone (NJ): Huntington Life Sciences Laboratories. [cited in API 2008a].

[API] American Petroleum Institute. 2008d. Baseline gasoline vapor condensate. Whole-body inhalation developmental toxicity study in rats with baseline gasoline vapour condensate. EMBSL No. MRD-00-695: 169534. Annandale (NJ): ExxonMobil Biomedical Sciences, Inc. [cited in API 2008a].

[ARCO] Atlantic Richfield Company. 1994. Developmental toxicity screen in rats administered test article F-250. ARCO Study No. ATX-93-0024 (Merox Feed); UBTL Study No. 66869. Los Angeles (CA): Atlantic Richfield Company. [cited in API 2008a].

Arnot J, Gobas F. 2003. A generic QSAR for assessing the bioaccumulation potential of organic chemicals in aquatic food webs. QSAR Comb Sci [Internet]. [cited 2007 Mar 15]; 22(3):337–345. Available from: www3.interscience.wiley.com/journal/104557877/home [restricted access].

Arnot J, Gobas F. 2004. A food web bioaccumulation model for organic chemicals in aquatic ecosystems.Environ Toxicol Chem 23:2343–2355.

Arnot J, Gobas F. 2006. A review of bioconcentration factor (BCF) and bioaccumulation factor (BAF) assessments for organic chemicals in aquatic systems. Environ Rev 14:257–297.

Arnot JA, Mackay D, Bonnell M. 2008a. Estimating metabolic biotransformation rates in fish from laboratory data. Environ Toxicol Chem 27(2):341–351.

Arnot JA, Mackay D, Parkerton TF, Bonnell M. 2008b. A database of fish biotransformation rates for organic chemicals. Environ Toxicol Chem 27(11): 2263–2270.

Arthurs P, Stiver WH, Zytner RG. 1995. Passive volatilization of gasoline from soil. J Soil Contam. 4: 1-13.

Atkinson R. 1990. Gas-phase tropospheric chemistry of organic compounds: a review. Atmos Environ 24A:1–41.

Atlas R. 1981. Microbial degradation of petroleum hydrocarbons: an environmental perspective. Microbiol Rev 45(1):180–209.

[ATSDR] Agency for Toxic Substances and Disease Registry. 1995a. Toxicological profile for Stoddard solvent. Atlanta (GA): US Department of Health and Human Services, Public Health Service. [cited 2009 Apr 17]. Available from: www.atsdr.cdc.gov/toxprofiles/tp.asp?id=416&tid=73

[ATSDR] Agency for Toxic Substances and Disease Registry. 1995b. Toxicological profile for automotive gasoline. Atlanta (GA): US Department of Health and Human Services, Public Health Service. [cited 2009 Apr 17]. Available from: www.atsdr.cdc.gov/toxprofiles/tp.asp?id=468&tid=83

Banerjee S. 1984. Solubility of organic mixtures in water. Environ Sci Technol 18:587–591.

Barron MG. 1990. Bioconcentration. Environ Sci Technol 24:1612–1618.

[BCFBAF] Bioconcentration Factor Program for Windows [Estimation Model]. 2008. Version 3.00. Washington (DC): U.S. Environmental Protection Agency, Office of Pollution Prevention and Toxics;

Syracuse (NY): Syracuse Research Corporation. Available from: www.epa.gov/oppt/exposure/pubs/episuite.htm

Bio/Dynamics, Inc. 1991a. A subchronic (3-month) oral toxicity study in the rat with LX-1006-01 via gavage (final report) with attachments and cover letter dated 042491 (sanitized). US Environmental Protection Agency Document No. 86-910000807S; NTIS/OTS0529439. [Abstract]. [cited in TOXLINE 2009].

Bio/Dynamics, Inc. 1991b. A subchronic (3-month) oral toxicity study in the dog with LX1106-01 via capsule administration (final report) with attachments and cover letters dated 042491 (sanitized). US Environmental Protection Agency Document No. 86-91000808S; NTIS/OTS0529440. [Abstract]. [cited in TOXLINE 2009].

Bio/Dynamics, Inc. 1991c. A teratology study in rats with LX1106-01 (final report) with attachments and cover letter dated 042491 (sanitized). US Environmental Protection Agency/Office of Toxic Substances Document No. 86-910000809S; NTIS/OTS0529441. [Abstract]. [cited in TOXLINE 2009].

[BIOHCWIN] Biodegradation of Petroleum Hydrocarbons Estimation Program for Windows [Estimation Model]. 2008. Version 1.01a. Washington (DC): U.S. Environmental Protection Agency, Office of Pollution Prevention and Toxics; Syracuse (NY): Syracuse Research Corporation. Available from: www.epa.gov/oppt/exposure/pubs/episuite.htm

[BIOWIN] Biodegradation Estimation Program for Windows [Estimation Model]. 2009. Version 4.10. Washington (DC): U.S. Environmental Protection Agency, Office of Pollution Prevention and Toxics; Syracuse (NY): Syracuse Research Corporation. Available from: www.epa.gov/oppt/exposure/pubs/episuite.htm

Blackburn GR. 1981. An Ames *Salmonella*/mammalian microsome mutagenesis assay for the determination of potential mutagenicity of rerun tower overheads from an olefins/aromatics plant. Study No. 1781-80. Princeton (NJ): Mobil Environmental and Health Science Laboratory. [cited in U.S. EPA 2004].

Blackburn GR, Deitch RA, Schreiner CA, Mackerer CR. 1986. Predicting carcinogenicity of petroleum distillation fractions using a modified *Salmonella* mutagenicity assay. Cell Biol Toxicol 2(1):63–84.

Blackburn GR, Deitch RA, Roy TA, Johnson SW, Schreiner CA, Mackerer CR. 1988. Estimation of dermal carcinogenic potency of petroleum fractions using a modified Ames assay. In: Cooke M, Dennis AJ, editors. Proceedings of the 10th annual symposium on polynuclear aromatic hydrocarbons: a decade of progress. Columbus (OH): Battelle Press. p. 83–97. [Abstract]. [cited in TOXLINE 2009].

Boethling RS, Howard PH, Beauman JA, Larosche ME. 1995. Factors for intermedia extrapolations in biodegradability assessment. Chemosphere 30(4):741–752.

Brecher S. 1984a. Hepatocyte primary culture/DNA repair test of hydrogenated pyrolysis gasoline. Project No. 2097. Prepared for Gulf Oil Chemicals Co., Houston, TX. Pittsburgh (PA): Gulf Life Sciences Center. [cited in U.S. EPA 2004].

Brecher S. 1984b. Transformation test of hydrogenated pyrolysis gasoline. Project No. 2098. Prepared for Gulf Oil Chemicals Co., Houston, TX. Pittsburgh (PA): Gulf Life Sciences Center. [cited in U.S. EPA 2004].

Brecher S. Goode JW. 1984a. Hepatocyte primary culture/DNA repair test of heavy aromatic distillate. Project No. 2056. Prepared for Gulf Oil Chemicals Co., Houston, TX. Pittsburgh (PA): Gulf Life Sciences Center. [cited in API 2001b].

Brecher S, Goode JW. 1984b. BALB/3T3 transformation test: heavy aromatic distillate. Project No. 2057. Prepared for Gulf Oil Chemicals Co., Houston, TX. Pittsburgh (PA): Gulf Life Sciences Center. [cited in API 2001b].

Broddle WD, Dennis MW, Kitchen DN, Vernot EH. 1996. Chronic dermal studies of petroleum streams in mice. Fundam Appl Toxicol 30(1):47–54.

Brost EJ, DeVaull GE. 2000. Non-aqueous phase liquid (NAPL) mobility limits in soil. API Soil and Groundwater Research Bulletin No. 9. Washington (DC): American Petroleum Institute (API).

Bui QQ, Burnett DM, Breglia RJ, Koschier FJ, Lapadula ES, Podhasky PI, Schreiner CA, White RD, Dalbey WE, Feuston MH. 1998. Toxicity evaluation of petroleum blending streams: reproductive and developmental effects of a distillate from light alkylate naphtha. J Toxicol Environ Health A 53(2):121–133.

Buryskova B, Bláha L, Vrskova D, Simkova K, Maršálek B. 2006. Effects and induction of glutathione S-transferase by short chain chlorinated paraffins (SCCPs) and C-12 alkane (dodecane) in*Xenopus laevis* frog embryo. Acta Veterinaria Brno 75(1):115–122.

Canada. 1993. Benzene [Internet]. Ottawa (ON): Environment Canada; Health Canada. (Priority substances list assessment report). Available from: www.hc-sc.gc.ca/ewh-semt/alt_formats/hecs-sesc/pdf/pubs/contaminants/psl1-lsp1/benzene/benzene-eng.pdf

Canada. 1997. *Canadian Environmental Protection Act, 1999: Benzene in Gasoline Regulations*. SOR/97-493; Canada Gazette, Part I, vol. 131, no. 18; Part II, no. 131, no. 24. [cited 2010 Apr 14]. Available from: www.ec.gc.ca/lcpe-cepa/eng/regulations/detailReg.cfm?intReg=1

Canada. 1999. *Canadian Environmental Protection Act, 1999*. S.C., 1999, c. 33. Canada Gazette, Part III, vol. 22, no. 3. Available from: www.gazette.gc.ca/archives/p3/1999/g3-02203.pdf

Canada. 2000. *Canadian Environmental Protection Act, 1999: Persistence and Bioaccumulation Regulations,* P.C. 2000-348, 23 March, 2000, SOR/2000-107. Canada Gazette. Part II, vol. 134, no. 7, p. 607−612. Available from: http://gazette.gc.ca/archives/p2/2000/2000-03-29/pdf/g2-13407.pdf

Canada. 2001. *Canada Shipping Act, 2001.* S.C., 2001, c. 26. Canada Gazette, Part III, vol. 24, no. 4, chapter 26. Available from: www.gazette.gc.ca/archives/p3/2001/g3-02404.pdf

Canada. 2010. *Petroleum Refinery Liquid Effluent Regulations* (C.R.C., c. 828) to the *Fisheries Act*. [cited 2010 Feb 19]. Available from: http://laws.justice.gc.ca/eng/C.R.C.-c.828/index.html

[CanLII] Canadian Legal Information Institute [databases on the Internet]. 2001– . Ottawa (ON): CanLII. [cited 2009]. Available from: www.canlii.org/en/index.php

Carpenter CP, Kinkead ER, Geary DL, Sullivan LJ, King JM. 1975. Petroleum hydrocarbon toxicity studies. III. Animal and human response to vapors of Stoddard solvent. Toxicol Appl Pharmacol 32(2):282–297.

[CATABOL] Probabilistic assessment of biodegradability and metabolic pathways [Computer Model]. c2004-2008. Version 5.10.3. Bourgas (BG): Bourgas Prof. Assen Zlatarov University, Laboratory of Mathematical Chemistry. Available from: http://oasis-lmc.org/?section= software&swid=1

[CCME] Canadian Council of Ministers of the Environment. 2008. Canada-wide standard for petroleum hydrocarbons (PHC) in soil. User Guidance. Winnipeg (MB): Canadian Council of Ministers of the Environment. Report number PN-1398. Available from: www.ccme.ca/publications/ceqg_rcqe.html

Chu I, Poon R, Valli V, Yagminas A, Bowers WJ, Seegal R, Vincent R. 2005. Effects of an ethanol–gasoline mixture: results of a 4-week inhalation study in rats. J Appl Toxicol 25(3):193–199.

[CITI] Chemicals Inspection and Testing Institute. 1992. Bioaccumulation and Biodegradation Data on Existing Chemicals Based on the CSCL Japan. Tokyo, Japan.

Clark CR, Walter MK, Ferguson PW, Katchen M. 1988. Comparative dermal carcinogenesis of shale and petroleum-derived distillates. Toxicol Ind Health 4(1):11–22.

Clark DG, Butterworth ST, Martin JG, Roderick HR, Bird MG. 1989. Inhalation toxicity of high flash aromatic naphtha. Toxicol Ind Health 5(3):415–428.

Conaway CC, Schreiner CA, Cragg ST. 1984. Mutagenicity evaluation of petroleum hydrocarbons. In: MacFarland HN, Holdsworth CE, MacGregor JA, Call RW, Lane ML, editors. Applied toxicology of petroleum hydrocarbons. Princeton (NJ): Princeton Scientific Publishers. p. 89–107. [cited in IARC 1989b].

[CONCAWE] CONservation of Clean Air and Water in Europe. 1992. Gasolines. Prepared by CONCAWE's Petroleum Products and Health Management Groups. Brussels (BE): CONCAWE. Product Dossier No. 92/103.

[CONCAWE] CONservation of Clean Air and Water in Europe. 1996. Acute aquatic toxicity of gasolines: report on CONCAWE test programme. Prepared by CONCAWE's Petroleum Products and Health Management Groups. Brussels (BE): CONCAWE. Product Dossier No. 96/57.

[CONCAWE] CONservation of Clean Air and Water in Europe. 2005. Classification and labelling of petroleum substances according to the EU dangerous substances directive (CONCAWE recommendations July 2005). Prepared by CONCAWE's Petroleum Products and Health Management Groups. Brussels (BE): CONCAWE. Product Dossier No. 6/05.

Correa M, Venables B. 1985. Bioconcentration of naphthalene in tissues of the white mullet (*Mugil curema*). Environ Toxicol Chem 4:227–231.

Curry P, Kramer G, Newhook R, Sitwell J, Somers D, Tracy B, Oostdam JV. 1993. Reference values for Canadian populations. Prepared by the Environmental Health Directorate Working Group on Reference Values. Ottawa (ON): Health Canada. [update of 1988 version].

Dalbey W and Feuston M. 1996. Partially vaporized full range catalytic reformed naphtha: subchronic and developmental toxicity studies in rats. [Abstract]. Inhal Toxicol Apr 8(3): 271-284. [cited in DART 2009].

Dalbey WE, Feuston MH, Yang JJ, Kommineni CV, Roy TA. 1996. Light catalytically cracked naphtha: subchronic toxicity of vapors in rats and mice and developmental toxicity screen in rats. J Toxicol Environ Health 47(1):77–91.

[DART] Developmental and Reproductive Toxicology Database [database on the Internet]. 1989 - . Bethesda (MD): National Library of Medicine (U.S.) [revised 2009 April 23; cited 2009 April 21]. Available from: http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?DARTETIC

Divine BJ, Barron V. 1986. Texaco mortality study. II. Patterns of mortality among white males by specific job groups. Am J Ind Med 10:371–381. [cited in IARC 1989a].

Dooley JF, Skinner MJ, Roy TA, Blackburn GR, Schreiner CA, Mackerer CR. 1988. Evaluation of the genotoxicity of API reference unleaded gasoline. In: Cooke M, Dennis AJ, editors. Proceedings of the 10th annual symposium on polynuclear aromatic hydrocarbons: a decade of progress. Columbus (OH): Battelle Press. p. 179–194. [cited in IARC 1989b; ATSDR 1995b].

[ECB] European Chemicals Bureau. 2000a. IUCLID dataset for naphtha (petroleum), full-range straight-run. CAS No. 64741-42-0. European Commission, European Chemicals Bureau. [cited 2008 Mar]. Available from: http://esis.jrc.ec.europa.eu/doc/IUCLID/data_sheets/64741420.pdf
[ECB] European Chemicals Bureau. 2000b. IUCLID dataset for naphtha (petroleum), light hydrocracked. CAS No. 64741-69-1. European Commission, European Chemicals Bureau. [cited 2008 Mar]. Available from:
http://esis.jrc.ec.europa.eu/doc/IUCLID/data_sheets/64741691.pdf

[ECB] European Chemicals Bureau. 2000c. IUCLID dataset for naphtha (petroleum), heavy hydrocracked. CAS No. 64741-78-2. European Commission, European Chemicals Bureau. [cited 2008 Mar]. Available from: http://esis.jrc.ec.europa.eu/doc/IUCLID/data_sheets/64741782.pdf
[EETD] Environmental Emergencies Technology Division. 1989. Experimental data. Ottawa (ON): Environment Canada.

Environment Canada. 2008. Data for petroleum sector stream substances collected under the *Canadian Environmental Protection Act, 1999*, section 71: Notice with respect to certain high priority petroleum substances. Data prepared by: Environment Canada, Oil, Gas, and Alternative Energy Division.

Environment Canada. 2009. Data for petroleum sector stream substances collected under the *Canadian Environmental Protection Act, 1999*, section 71: Notice with respect to potentially industry-limited high priority petroleum substances. Data prepared by: Environment Canada, Oil, Gas, and Alternative Energy Division.

Environment Canada. 2011. Spill Line data report on petroleum product spills 2000–2009. Gatineau (QC): Environment Canada, Environmental Emergencies Division. Unpublished information.

[EPI Suite] Estimation Programs Interface Suite for Microsoft Windows [Estimation Model]. 2008. Version 3.4. Washington (DC): U.S. Environmental Protection Agency, Office of Pollution Prevention and Toxics; Syracuse (NY): Syracuse Research Corporation. Available from: www.epa.gov/oppt/exposure/pubs/episuite.htm

[EQC] Equilibrium Criterion Model. 2003. Version 2.02. Peterborough (ON): Trent University, Canadian Environmental Modelling Centre. Available from: www.trentu.ca/cemc/

[ESIS] European Chemical Substances Information System [database on the Internet]. 2008. Database developed by the European Chemicals Bureau (ECB). [cited 2011 Aug 29]. Available from: http://esis.jrc.ec.europa.eu/

European Commission. 2008a. Commission Directive 2008/58/EC of 21 August 2008. Official Journal of the European Union. 15.9.2008. L246, vol. 51. European Commission. [cited 2011 Jun 16]. Available from: http://eur-lex.europa.eu/LexUriServ/LexUriServ.do?uri=OJ:L:2008:246:0001:0191:EN:PDF

European Commission. 2008b. Details on substances classified in Annex VI to Regulation (EC) No 1272/2008 [database on the Internet]. Database developed by the European Commission. [cited 2011 Aug 29]. Available from: http://esis.jrc.ec.europa.eu/index.php?PGM=cla

European Commission. 2009. Commission Regulation (EC) No 790/2009 of 10 August 2009. Official Journal of the European Union. 5.9.2009. L235, vol. 52. European Commission. [cited 2011 Jun 16]. Available from: http://eur-lex.europa.eu/LexUriServ/LexUriServ.do?uri=OJ:L:2009:235:FULL:EN:PDF

Farrow MG, McCarroll N, Cortina T, Draus M, Munson A, Steinberg M, Kirwin C, Thomas W. 1983. *In vitro* mutagenicity and genotoxicity of fuels and paraffinic hydrocarbons in the Ames, sister chromatid exchange, and mouse lymphoma assays. Toxicologist 3(1):36. [cited in IARC 1989b].

Gerin M, Viau C, Talbot D, Greselin E. 1988. Aviation gasoline: comparative subchronic nephrotoxicity study in the male rat. Toxicol Lett 44:13–19.

Gochet B, De Meester C, Leonard A, DeKnudt G. 1984. Lack of mutagenic activity of white spirit. Int Arch Occup Environ Health 53(4):359–364.

Golder Associates Ltd. 2006. Final Draft Report on Lakewater and Aquatics Long-term Monitoring – 2005–2006 Interpretive Report, Wabamun Lake Derailment Site, Canadian National Railway Company. PIN 2401257. Wabamun, Alberta.

Gustafson JB, Tell JG, Orem D. 1997. Selection of representative TPH fractions based on fate and transport considerations. Vol. 3. Total Petroleum Hydrocarbon Criteria Working Group Series, Association for Environmental Health and Sciences Foundation. 109 p. Available from: www.aehsfoundation.org/publications.aspx

Halder CA, Warne TM, Hartoum NS. 1984. Renal toxicity of gasoline and related petroleum naphthas in male rats. Adv Mod Environ Toxicol 7:73–88.

Halder CA, Holdsworth CE, Cockrell BY, Piccirillo VJ. 1985. Hydrocarbon nephropathy in male rats: identification of the nephrotoxic components of unleaded gasoline. Toxicol Ind Health 1(3):67–87. [cited in CONCAWE 1992].

Hansch C, Leo A, Hoekman D. 1995. Exploring QSAR: hydrophobic, electronic, and steric constants. ACS Professional Reference Book. Washington (DC): American Chemical Society.

Hass U, Ladefoged O, Lam HR, Ostergaard G, Lund SP, Simonsen L. 2001. Behavioural effects in rats after prenatal exposure to dearomatized white spirit. Pharmacol Toxicol 89(4):201–207.

Haworth SR. 1978. Bacterial DNA repair assay of Mobil Chemical Company compound MCTR-125-78 (MRI #110). Prepared for Mobil Chemical Co., Edison, NJ. Rockville (MD): E.G. and G. Mason Research Institute. [cited in U.S. EPA 2004].

Health Canada. 1996. Health-based tolerable daily intakes/concentrations and tumorigenic doses/concentrations for priority substances [Internet]. Ottawa (ON): Health Canada, Environmental Health Directorate. Available from: http://dsp-psd.pwgsc.gc.ca/Collection/H46-2-96-194E.pdf

Health Canada. 1998. Exposure factors for assessing total daily intake of priority substances by the general population of Canada. Ottawa (ON): Health Canada, Environmental Health Directorate. Unpublished report.

Health Canada. 2008. Results of the health-related components of categorization of the Domestic Substances List under CEPA 1999. [cited 2010 Feb 11]. Available from: www.hc-sc.gc.ca/ewh-semt/contaminants/existsub/categor/_result_substance/index-eng.php

Hendricks NV, Berry CM, Lione JG, Thorpe JJ. 1959. Cancer of the scrotum in wax pressman. I. Epidemiology. Arch Ind Health Occup Med 19:524–529. [cited in IARC 1989a].

[HENRYWIN] Henry's Law Constant for Windows [Estimation Model]. 2008. Version 3.20. Washington (DC): U.S. Environmental Protection Agency, Office of Pollution Prevention and Toxics; Syracuse (NY): Syracuse Research Corporation. Available from: www.epa.gov/oppt/exposure/pubs/episuite.htm

Hopkinson R. 2008. Priority substances under Environment Canada's Chemical Management Plan for the petroleum sector. Richmond (BC): Levelton Consultants Ltd.

Howard P, Boethling R, Jarvis W, Meylan W, Michalenko E. 1991. Handbook of environmental degradation rates. Syracuse Research Corp. for U.S. EPA. Boca Raton (FL): Lewis Publishers.

[IARC] IARC Working Group on the Evaluation of Carcinogenic Risks to Humans. 1989a. Occupational exposures in petroleum refining. IARC Monogr Eval Carcinog Risks Hum 45:39–117.

[IARC] IARC Working Group on the Evaluation of Carcinogenic Risks to Humans. 1989b. Gasoline. IARC Monogr Eval Carcinog Risks Hum 45:159–201.

[IARC] IARC Working Group on the Evaluation of Carcinogenic Risks to Humans. 2011. Agents classified by the IARC monographs, Volumes 1–102 (alphabetical order). Lyon (FR): International Agency for Research on Cancer. Available from: http://monographs.iarc.fr/ENG/Classification/ClassificationsAlphaOrder.pdf

[IPCS] International Programme on Chemical Safety. 1996. White spirit (Stoddard solvent). Geneva (CH): World Health Organization. (Environmental Health Criteria 187). Jointly sponsored by the United Nations Environment Programme, the International Labour Organization and the World Health Organization. [cited 2009 Aug 23]. Available from: www.inchem.org/documents/ehc/ehc/ehc187.htm

Jensen EM, Thilager AK. 1978. C3H 10T1/2 cell transformation assay. Mobil Chemical Co. compound MCTR-125-78 (MRI #110). Rockville (MD): E.G. and G. Mason Research Institute. [cited in U.S. EPA 2004].

[JNITE] Japanese National Institute of Technology and Evaluation. 2010. Official Bulletin of Economy, Trade and Industry [database on the Internet]. [accessed 2010 Sept]. Available from: http://www.nite.go.jp/index-e.html

Jonsson G, Bechmann RK, Bamber SD, Baussant T. 2004. Bioconcentration, biotransformation, and elimination of polycyclic aromatic hydrocarbons in Sheepshead minnows (*Cyprinodon variegates*) exposed to contaminated seawater. Environ Toxicol Chem 23:1538–1548. [cited in the Golder Associates Ltd. 2006 report: Lakewater and aquatic long-term monitoring 2005-2006 interpretive report, Wabamum Lake derailment site].

Khan SH. 1984. Micronucleus test of hydrogenated pyrolysis gasoline. Project No. 2096. Prepared for Gulf Oil Chemicals Co., Houston, TX. Pittsburgh (PA): Gulf Life Sciences Center. [cited in U.S. EPA 2004].

Khan SH, Goode JW. 1984. Micronucleus test in mouse bone marrow: heavy aromatic distillate administered orally for 2 days. Project No. 2005. Prepared for Gulf Oil Chemicals Co., Houston, TX. Pittsburgh (PA): Gulf Life Sciences Center. [cited in API 2001b].

Kirby PE et al. 1979. An evaluation of mutagenic potential of MCTR-125-78 (MRI #110) employing the L5178Y TK+/− mouse lymphoma assay. Prepared for Mobil Chemical Co., Edison, NJ. Rockville (MD): E.G. and G. Mason Research Institute. [cited in U.S. EPA 2004].

[KOCWIN] Organic Carbon Partition Coefficient Program for Windows [Estimation Model]. 2009. Version 2.00. Washington (DC): U.S. Environmental Protection Agency, Office of Pollution Prevention and Toxics; Syracuse (NY): Syracuse Research Corporation. Available from: www.epa.gov/oppt/exposure/pubs/episuite.htm

[KOWWIN] Organic Carbon Partition Coefficient Program for Windows [Estimation Model]. 2008. Version 1.67a. Washington (DC): U.S. Environmental Protection Agency, Office of Pollution Prevention and Toxics; Syracuse (NY): Syracuse Research Corporation. Available from: www.epa.gov/oppt/exposure/pubs/episuite.htm

Kuna RA, Ulrich CE. 1984. Subchronic inhalation toxicity of two motor fuels. J Am Coll Toxicol 3(4):217–229. [cited in API 2001a].

Lam HR, Ostergaard G, Guo SX, Ladefoged O, Bondy SC. 1994. Three weeks' exposure of rats to dearomatized white spirit modifies indices of oxidative stress in brain, kidney, and liver. Biochem Pharmacol 47(4):651–657.

Lampi M, Paumen ML, Parkerton T. 2010. An Evaluation of the Persistence, Bioaccumulation and Toxicity of Petroleum Hydrocarbons. Report Prepared for CONCAWE, Brussels, Belgium by ExxonMobil Biomedical Sciences, Inc, Annandale NJ, USA and ExxonMobil Petroleum and Chemical, Machelen, Belgium.

Lione JG, Denholm JS. 1959. Cancer of the scrotum in wax pressman. II. Clinical observations. Arch Ind Health Occup Med 19:530–539. [cited in IARC 1989a].

Litton Bionetics. 1978. Teratology study in rats: unleaded gasoline. Final report submitted to American Petroleum Institute, Washington, DC. Kensington (MD): Litton Bionetics, Inc. [cited in ATSDR 1995b].

Litton Bionetics. 1980. Mutagenicity evaluation of gasoline, API PS-6 fuel in the mouse dominant lethal assay. Final report submitted to American Petroleum Institute, Washington, DC. Kensington (MD): Litton Bionetics, Inc. [cited in ATSDR 1995b].

Lockhart WL, Danelle RW, Murray DAJ. 1987. Acute toxicity bioassays with petroleum products: influence of exposure conditions. In: Vandermeulen JH, Hurdy SE, editors. Oil in fresh water: chemistry, biology and counter-measure technology. Oxford (UK): Pergamon Press. [cited in CONCAWE 1992].

Loury DJ, Smith-Oliver T, Strom S, Jirtle R, Michalopoulos G, Butterworth BE. 1986. Assessment of unscheduled and replicative DNA synthesis in hepatocytes treated *in vivo* and *in vitro* with unleaded gasoline or 2,2,4-trimethylpentane. Toxicol Appl Pharmacol 85(1):11–23.

Loury DJ, Smith-Oliver T, Butterworth BE. 1987. Assessment of unscheduled and replicative DNA synthesis in rat kidney cells exposed *in vitro* or *in vivo* to unleaded gasoline. Toxicol Appl Pharmacol 87(1):127–140.

MacFarland HN, Ulrich CE, Holdsworth CE, Kitchen DN, Halliwell WH, Blum SC. 1984. A chronic inhalation study with unleaded gasoline vapour. Int J Toxicol 3(4):231–248.

MacLean MM, Doe KG. 1989. The comparative toxicity of crude and refined oils to *Daphnia magna* and *Artemia*. Environment Canada. Environmental Protection Directorate. Not published EE-111.

McAuliffe C. 1963. Solubility in water of C_1-C_9 hydrocarbons. Nature (London) 200:1092–1093.

McAuliffe C. 1966. Solubility in water of paraffin, cycloparaffin, olefin, acetylene, cycloolefin and aromatic hydrocarbon. J Phys Chem 70:1267–1275.

McCraw DS, Joyner RE, Cole P. 1985. Excess leukemia in a refinery population. J Occup Med 27:220–222. [cited in IARC 1989a].

McKee RH, Wong ZA, Schmitt S, Beatty P, Swanson M, Schreiner CA, Schardein JL. 1990. The reproductive and developmental toxicity of high flash aromatic naphtha. Toxicol Ind Health 6(3/4):441–460.

McKee RH, Trimmer GW, Whitman FT, Nessel CS, Mackerer CR, Hagemann R, Priston RAJ, Riley AJ, Cruzan G, Simpson BJ, Urbanus JH. 2000. Assessment in rats of the reproductive toxicity of gasoline from a gasoline vapor recovery unit. Reprod Toxicol 14:337–353.

Miller LG, Schardein JL. 1981. Rerun tower overheads: teratology study in rabbits (MCTR-171-79). IRDC Study No. 450-011a. Prepared for Mobil Oil Corporation, Princeton, NJ. Mattawan (MI): International Research and Development Corporation. [cited in U.S. EPA 2001, 2004].

[Mobil] Mobil Oil Corporation. 1988a. Thirteen week dermal administration of light catalytically cracked naphtha (LCCN) to rats. Study No. 50381. Princeton (NJ): Mobil Oil Corporation, Environmental and Health Science Laboratory. [cited in API 2008a].

[Mobil] Mobil Oil Corporation. 1988b. Developmental toxicity screen in rats exposed dermally to light catalytically cracked naphtha (LCCN). Study No. 50341. Princeton (NJ): Mobil Oil Corporation, Environmental and Health Science Laboratory. [cited in API 2008a].

[MPBPWIN] Melting Point Boiling Point for Windows [Estimation Model]. 2008. Version 1.43. Washington (DC): U.S. Environmental Protection Agency, Office of Pollution Prevention and Toxics; Syracuse (NY): Syracuse Research Corporation. Available from: www.epa.gov/oppt/exposure/pubs/episuite.htm

[NCI] National Chemical Inventories [database on a CD-ROM]. 2006. Columbus (OH): American Chemical Society, Chemical Abstracts Service. Available from: http://www.cas.org/products/other-cas-products/nci-on-cd

Neff JM, Cox BA, Anderson JW. 1976. Accumulation and release of petroleum derived aromatic hydrocarbons by four species of marine animals. Mar Biol 38(3):279–289.

Nelson NA, Van Peenen PFD, Blanchard AG. 1987. Mortality in a recent oil refinery cohort. J Occup Med 29:610–612. [cited in IARC 1989a].

[OECD] Organisation for Economic Co-operation and Development. 2009. Emission scenario document on transport and storage of chemicals. OECD series on emission scenario documents, No. 24. Available from: http://www.oecd.org/officialdocuments/displaydocumentpdf?cote=env/jm/mono(2009)26&doclanguage=en

Paixão JF, Nascimento IA, Pereira SA, Leite MBL, Carvalho GC, Silveira Jr. JSC, Rebouças M, Matias GRA, Rodrigues ILP. 2007. Estimating the gasoline components and formulations toxicity to microalgae (*Tetraselmis chuii*) and oyster (*Crassostrea rhizophorae)* embryos: An approach to minimize environmental pollution risk. Environ Res 103:365–374.

Pancirov R, Brown R. 1975. Analytical methods for polynuclear aromatic hydrocarbons in crude oils, heating oils, and marine tissues. Proceedings of a Conference on Prevention and Control of Oil Pollution, San Francisco, California. Washington (DC): American Petroleum Institute. p. 103–113. [cited in Potter and Simmons 1998].

Papciak MS, Goode JW. 1984. CHO/HGPRT test: heavy aromatic distillate. Project No. 2054. Prepared for Gulf Oil Chemicals Co., Houston, TX. Pittsburgh (PA): Gulf Life Sciences Center. [cited in API 2001b].

[PETROTOX] Spreadsheet model to calculate toxicity of petroleum hydrocarbons to aquatic organisms. 2009. Version 3.01. Brussels (BE): CONservation of Clean Air and Water in Europe (CONCAWE). Available from: www.concawe.be/content/default.asp?PageID=778

Phillips RD, Egan GF. 1981. Teratogenic and dominant lethal investigation of two hydrocarbon solvents. Toxicologist 1(1):15. [Abstract]. [cited in IPCS 1996; U.S. EPA 1998].

Phillips RD, Egan GF. 1984. Subchronic inhalation exposure of dearomatized white spirit and C10–C11 isoparaffinic hydrocarbon in Sprague-Dawley rats. Fundam Appl Toxicol 4(5):808–818.

Potter T, Simmons K. 1998. Composition of petroleum mixtures. Vol 2. Total Petroleum Hydrocarbon Criteria Working Group Series. Amherst (MA): Amherst Scientific Publishers.

[PPSC] Petroleum Product Stewardship Council. 1995a. Static-renewal 96-hour acute toxicity study of the water accommodated fraction (WAF) of whole light alkylate product to fathead minnow. Study conducted by Stonybrook Laboratories, Inc. Study No. 65908. Washington (DC): Petroleum Product Stewardship Council.

[PPSC] Petroleum Product Stewardship Council. 1995b. Static-renewal 48-hour acute toxicity study of the water accommodated fraction (WAF) of whole light alkylate product to *Daphnia magna*. Study conducted by Stonybrook Laboratories, Inc. Study No. 65907. Washington (DC): Petroleum Product Stewardship Council.

[PPSC] Petroleum Product Stewardship Council. 1995c. Static-renewal 96-hour actue toxicity study of the water accommodated fraction (WAF) of whole light alkylate product to mysid shrimp. Study conducted by Stonybrook Laboratories, Inc. Study No. 65910. Washington (DC): Petroleum Product Stewardship Council.

Prince R, Parkerton T, Lee C. 2007a. The primary aerobic biodegradation of gasoline hydrocarbons. Environ Sci Technol 41:3316–3321.

Prince R, Parkerton T, Lee C. 2007b. The primary aerobic biodegradation of gasoline hydrocarbons: supplementary information. Environmental Science and Technology supporting information file available from: http://pubs.acs.org/doi/suppl/10.1021/es062884d

PubMed [database on the Internet]. 1997– . Bethesda (MD): National Library of Medicine (US). [revised 2009 Apr 23; cited 25 Apr 2009]. Available from: www.ncbi.nlm.nih.gov/pubmed/

Rector DE, Steadman BL, Jones RA, Siegel J. 1966. Effects on experimental animals of long-term inhalation exposure to mineral spirits. Toxicol Appl Pharmacol 9(2):257–268.

Research and Environmental Division. 1984. Follow-up to TSCA Section 8(E) on Isopar C and Varsol 40 with cover letter dated 011884. US Environmental Protection Agency Document No. 88-8400586; NTIS/OTS0200630. [Abstract]. [cited in TOXLINE 2009].

Riccio ES, Stewart KR. 1991. *Salmonella–Escherichia coli*/microsome plate incorporation assay of hydrogenated pyrolysis gasoline. SRI Study No. 2545-A03-91. Sponsor Study No. 91-66. Prepared for Chevron Environmental Health Center, Richmond, CA. Menlo Park (CA): SRI International. [cited in U.S. EPA 2004].

Richardson KA, Wilmer JL, Smith-Simpson D, Skopek TR. 1986. Assessment of the genotoxic potential of unleaded gasoline and 2,2,4-trimethylpentane in human lymphoblasts *in vitro*. Toxicol Appl Pharmacol 82(2):316–322. [cited in IARC 1989b].

Riley AJ, Collings AJ, Browne NA, Grasso P. 1984. Response of the upper respiratory tract of the rat to white spirit vapour. Toxicol Lett 22(2):125–131.

Rinsky RA, Young RJ, Smith AB. 1981. Leukemia in benzene workers. Am J Ind Med 2:217–245.

Rinsky RA, Smith AB, Hornung R, Filloon TG, Young RJ, Okun AH, Landrigan PJ. 1987. Benzene and leukemia: an epidemiological risk assessment. N Engl J Med 316:1044–1050.

[RMRI] Risk Management Research Institute. 2007. Quantitative assessment of oil spill risk for the south coast of Newfoundland and Labrador. Prepared for Transport Canada. St. John’s (NL): RMRI. Draft Reference No.: CAN/0179/R003.

Roberts L, White R, Bui Q, Daughtrey W, Koschier F, Rodney S, Schreiner C, Steup D, Breglia R, Rhoden R, Schroeder R, Newton P. 2001. Developmental toxicity evaluation of unleaded gasoline vapour in the rat. Reprod Toxicol 15(5):487–494.

Rodrigues RV, Miranda-Filho KC, Gusmão EP, Moreira CB, Romano LA, Sampaio LA. 2010. Deleterious effects of water-soluble fraction of petroleum, diesel and gasoline on marine pejerrey *Odontesthes argentinensis* larvae. Sci Total Environ 408:2054–2059.

Rodriguez SC, Dalbey WE. 1994a. Acute oral toxicity of dripolene in Sprague-Dawley rats. Study No. 65642. Prepared for Mobil Chemical Co., Edison, NJ. Princeton (NJ): Stonybrook Laboratories. [cited in U.S. EPA 2004].

Rodriguez SC, Dalbey WE. 1994b. Acute oral toxicity of pyrolysis gasoline in Sprague-Dawley rats. Study No. 65636. Prepared for Mobil Chemical Co., Edison, NJ. Princeton (NJ): Stonybrook Laboratories. [cited in U.S. EPA 2004].

Rodriguez SC, Dalbey WE. 1994c. Dermal toxicity of dripolene in the New Zealand White rabbit. Study No. 65643. Prepared for Mobil Chemical Co., Edison, NJ. Princeton (NJ): Stonybrook Laboratories. [cited in U.S. EPA 2004].

Rodriguez SC, Dalbey WE. 1994d. Dermal toxicity of pyrolysis gasoline in the New Zealand White rabbit. Study No. 65637. Prepared for Mobil Chemical Co., Edison, NJ. Princeton (NJ): Stonybrook Laboratories. [cited in U.S. EPA 2004].

Rodriguez SC, Dalbey WE. 1994e. Acute dermal irritation/corrosion of dripolene in the New Zealand White rabbit. Study No. 65644. Prepared for Mobil Chemical Co., Edison, NJ. Princeton (NJ): Stonybrook Laboratories. [cited in U.S. EPA 2004].

Rodriguez SC, Dalbey WE. 1994f. Acute dermal irritation/corrosion of pyrolysis gasoline in the New Zealand White rabbit. Study No. 65639. Prepared for Mobil Chemical Co., Edison, NJ. Princeton (NJ): Stonybrook Laboratories. [cited in U.S. EPA 2004].

Rodriguez SC, Dalbey WE. 1994g. Ocular irritation of dripolene in the New Zealand White rabbit. Study No. 65644. Prepared for Mobil Chemical Co., Edison, NJ. Princeton (NJ): Stonybrook Laboratories. [cited in U.S. EPA 2004].

Rodriguez SC, Dalbey WE. 1994h. Acute dermal irritation/corrosion of dripolene in the New Zealand White rabbit. Study No. 65645. Prepared for Mobil Chemical Co., Edison, NJ. Princeton (NJ): Stonybrook Laboratories. [cited in U.S. EPA 2004].

Rodriguez SC, Dalbey WE. 1994i. Ocular irritation of pyrolysis gasoline in the New Zealand White rabbit. Study No. 65638. Prepared for Mobil Chemical Co., Edison, NJ. Princeton (NJ): Stonybrook Laboratories. [cited in U.S. EPA 2004].

Roy TA. 1981. Analysis of rerun tower bottom oil by combined capillary gas chromatography/mass spectrometry. Study No. 1272-81. Princeton (NJ): Mobil Oil Corporation, Toxicology Division. [cited in U.S. EPA 2004].

[RTECS] Registry of Toxic Effects of Chemical Substances. 2008a. Stoddard Solvent. RTECS No. WJ8925000. CAS No. 8052-41-3. [updated 2008 Nov; cited 2009 Nov 31]. Hamilton (ON): Canadian Centre for Occupational Health and Safety.

[RTECS] Registry of Toxic Effects of Chemical Substances. 2008b. Ligroine. RTECS No. OI6180000. CAS No. 8032-32-4. [updated 2008 Nov; cited 2009 Nov 31]. Hamilton (ON): Canadian Centre for Occupational Health and Safety.

[RTECS] Registry of Toxic Effects of Chemical Substances. 2008c. Benzin. RTECS No. DE3030000. CAS No. 8030-30-6. [updated 2008 Nov; cited 2009 Nov 31]. Hamilton (ON): Canadian Centre for Occupational Health and Safety.

Salem H, Katz SA, editors. 2006. Inhalation toxicology. 2nd ed. Boca Raton (FL): CRC Press, Taylor & Francis Group.

Savolainen H, Pfaffli P. 1982. Neurochemical effects of extended exposure to white spirit vapour at three concentration levels. Chem Biol Interact 39(1):101–110.

Schreiner CA. 1984. Petroleum and petroleum products: a brief review of studies to evaluate reproductive effects. In: Christian MS, Galbraith WM, Voytek P, Mehlman MA, editors. Advances in modern environmental toxicology, vol. III. Assessment of reproductive and teratogenic hazards. Princeton (NJ): Princeton Scientific Publishers. p. 29–45.

Schreiner CA, Lapadula E, Breglia R, Bui Q, Burnett D, Koschier F, Podhasky P, White R, Mandella R, Hoffman G. 1998. Toxicity evaluation of petroleum blending streams: inhalation subchronic toxicity/neurotoxicity study of a light alkylate naphtha distillate in rats. J Toxicol Environ Health A 55(4):277–296.

Schreiner C, Bui Q, Breglia R, Burnett D, Koschier F, Podhasky P, Lapadula E, White R, Schroeder RE. 1999. Toxicity evaluation of petroleum blending streams: reproductive and developmental effects of light catalytic cracked naphtha distillate in rats. J Toxicol Environ Health A 58(6):365–382.

Schreiner C, Bui Q, Breglia R, Burnett D, Koschier F, Lapadula E, Podhasky P, White R. 2000a. Toxicity evaluation of petroleum blending streams: inhalation subchronic toxicity/neurotoxicity study of a light catalytic reformed naphtha distillate in rats. J Toxicol Environ Health A 60(7):489–512. [abstract in PubMed 2009].

Schreiner C, Bui Q, Breglia R, Burnett D, Koschier F, Podhasky P, White R, Hoffman G, Schroeder R. 2000b. Toxicity evaluation of petroleum blending streams: reproductive and developmental effects of light catalytic reformed naphtha distillate in rats. J Toxicol Environ Health A 60(3):169–184.

[SCREEN3] Screening Tool Program for Windows [Screening Model]. 1996. Version 4.10. Research Triangle Park (NC): U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards, Emissions, Monitoring, and Analysis Division. Available from: www.epa.gov/scram001/dispersion_screening.htm

[SCREEN3 ISC3] SCREEN 3 Industrial Source Complex Dispersion Model. 1995. User's guide for the Industrial Source Complex (ISC3) dispersion models, vol. II. Description of model algorithms. Research Triangle Park (NC): U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards, Emissions, Monitoring, and Analysis Division. Report No.: EPA-454/B-95-003b. Available from: www.epa.gov/scram001/userg/regmod/isc3v2.pdf

[SENES] SENES Consultants Limited. 2009. Review of current and proposed regulatory and non-regulatory management tools pertaining to selected petroleum substances under the Chemicals Management Plan. Report to Health Canada. Richmond Hill (ON): SENES.

Shell Research Ltd. 1980. The inhalation toxicity of SHELLSOL A to rats following 13 weeks' exposure. Unpublished data submitted to US Environmental Protection Agency, Washington, DC, July 8, 1988 (OPTS-41009). Group Research Report No.: TLGR.79.141. [cited in Clark et al. 1989].

Short BG, Steinhagen WH, Swenberg JA. 1989. Promoting effects of unleaded gasoline and 2,2,4-trimethylpentane on the development of atypical cell foci and renal tubular cell tumors in rats exposed to *N*-ethyl-*N*-hydroxyethylnitrosamine. Cancer Res 49(22):6369–6378.

Simpson BJ. 2005. Analysis of petroleum hydrocarbon streams on the Health Canada CEPA/DSL Draft Maximal List. Report to the Canadian Petroleum Products Institute.

Skisak CM, Furedi-Machacek EM, Schmitt SS, Swanson MS, Vernot EH. 1994. Chronic and initiation/promotion skin bioassays of petroleum refinery streams. Environ Health Perspect 102(1):82–87.

Standeven AM, Goldsworthy TL. 1993. Promotion of preneoplastic lesions and induction of CYP2B by unleaded gasoline vapour in female B6C3F1 mouse liver. Carcinogenesis 14(10):2137–2141.

Standeven AM, Wolf DC, Goldsworthy TL. 1994. Interactive effects of unleaded gasoline and estrogen on liver tumor promotion in female B6C3F1 mice. Cancer Res 54(5):1198–1204.

Standeven AM, Wolf DC, Goldsworthy TL. 1995. Promotion of hepatic preneoplastic lesions in male B6C3F1 mice by unleaded gasoline. Environ Health Perspect 103(7–8):696–700.

Stegeman JJ, Teal JM. 1973.Accumulation, release and rention of petroleum hydrocarbons by the Oyster *Crassostrea virginica*. Mar Biol 22:37–44.

Stonybrook Laboratories. 1995. Teratogenicity study in rats exposed orally to a single dose of a refinery stream. Study No. 65371. Princeton (NJ): Stonybrook Laboratories, Inc. [cited in API 2008a].

Stubblefield WA, McKee RH, Kapp RW Jr., Hinz JP. 1989. An evaluation of the acute toxic properties of liquids derived from oil sands. J Appl Toxicol 9(1): 59-65.

Tolls J, van Dijk J. 2002. Bioconcentration of n-dodecane and its highly branched isomer 2,2,4,6,6-pentamethylheptane in fathead minnows. Chemosphere 47:1049–1057.

[TOPKAT] TOxicity Prediction by Komputer Assisted Technology [Internet]. 2004. Version 6.1. San Diego (CA): Accelrys Software Inc. Available from: http://accelrys.com/mini/toxicology/predictive-functionality.html

[TOXLINE] Toxicology Literature Online [database on the Internet]. 1974– . Bethesda (MD): National Library of Medicine (US). [revised 2009 Apr 18; cited 2009 Apr 21]. Available from: http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?toxadv.htm

Tu AS, Sivak A. 1981. BALB-c/3T3 neoplastic transformation assay on 0818802, 08188003 and 08188005 (rerun tower overheads). ALD Reference No. 86374. Study No. 1771-80. Prepared for Mobil Oil Corporation, Princeton, NJ. Cambridge (MA): Arthur D. Little, Inc. [cited in U.S. EPA 2004].

[UN] United Nations. 2009. Development of guidance on the application of GHS criteria. Geneva (CH): UN Committee of Experts on the Transport of Dangerous Goods and on the Globally Harmonized System of Classification and Labelling of Chemicals.

[U.S. EPA] United States Environmental Protection Agency. 1984. Estimating concern levels for concentrations of chemical substances in the environment. Washington (DC): U.S. EPA, Office of Pollution Prevention and Toxics, Economics, Health and Environmental Review Division, Environmental Effects Branch. [cited in U.S. EPA 1998].

[U.S. EPA] United States Environmental Protection Agency. 1992. Screening procedures for estimating the air quality impact of stationary sources, revised. Research Triangle Park (NC): U.S. EPA, Office of Air and Radiation, Office of Air Quality Planning and Standards. Report No.: EPA-454/R-92-019. Available from: http://www.epa.gov/oppt/exposure/presentations/efast/usepa_1992b_sp_for_estim_aqi_of_ss.pdf

[U.S. EPA] United States Environmental Protection Agency. 1995. Great Lakes Water Quality Initiative Technical Support Document for the Procedure to Determine Bioaccumulation Factors, Report No. EPA-820-B-95-005

[U.S. EPA] United States Environmental Protection Agency. 1998. Cleaner technologies substitutes assessment: professional fabricare processes. Prepared for the Economics, Exposure and Technology

Division, Office of Pollution Prevention and Toxics, U.S. Environmental Protection Agency, Washington, DC. Cambridge (MA): Abt Associates, Inc. Contract Nos.: 68-W-9805 and 68-W6-0021. Report No.: EPA 744-B-98-001. Available from: www.epa.gov/dfe/pubs/garment/ctsa/fabricare.pdf

[U.S. EPA] United States Environmental Protection Agency. 2000. Benzene (CASRN 71-43-2). Washington (DC): U.S. EPA, Integrated Risk Information System (IRIS). Available from: www.epa.gov/iris/subst/0276.htm

[U.S. EPA] United States Environmental Protection Agency. 2001. High benzene naphthas robust summaries. Prepared for the High Production Volume (HPV) Chemical Challenge Program, U.S. Environmental Protection Agency, Washington, DC, by Olefins Panel HPV Work Group of the American Chemistry Council. EPA Reference No. AR201-13436B. Available from: www.epa.gov/HPV/pubs/summaries/hibenznp/c13436rs.pdf

[U.S. EPA] United States Environmental Protection Agency. 2004. High benzene naphthas robust summaries: mammalian toxicity (Attachment 1C). Prepared for the High Production Volume (HPV) Chemical Challenge Program, U.S. Environmental Protection Agency, Washington, DC, by Olefins Panel HPV Work Group of the American Chemistry Council. EPA Reference No. 201-15727B. Available from: www.epa.gov/HPV/pubs/summaries/hibenznp/c13436rr2.pdf

[U.S. EPA] United States Environmental Protection Agency. 2008. Transportation and marketing of petroleum liquids. In: AP 42, Compilation of air pollutant emission factors, vol. 1. 5th ed. Washington (DC): U.S. EPA. Available from: www.epa.gov/ttn/chief/ap42/ch05/final/c05s02.pdf

Verschueren K. 2001. Handbook of environmental data on organic chemicals, 4th ed. New York (NY): John Wiley & Sons.

Whiticar S, Bobra M, Fingas M, Jokuty P, Liuzzo P, Callaghan S, Ackerman F, Cao J. 1993. A catalogue of crude oil and oil product properties (1992 edition). EE-144. Unpublished report. Ottawa (ON): Environment Canada, Environmental Protection Directorate.

Witschi HP, Smith LH, Frome EL, Pequet-Goad ME, Griest WH, Ho C-H, Guerin MR. 1987. Skin tumorigenic potential of crude and refined coal liquids and analogous petroleum products. Fundam Appl Toxicol 9(2):297–303.

Wong O, Raabe GK. 1989. Critical review of cancer epidemiology in petroleum industry employees, with a quantitative meta-analysis by cancer site. Am J Ind Med 15:283–310. [cited in IARC 1989a].

[WSKOWWIN] Water Solubility for Organic Compounds Program for Microsoft Windows [Estimation Model]. 2008. Version 1.41a. Washington (DC): U.S. Environmental Protection Agency, Office of Pollution Prevention and Toxics; Syracuse (NY): Syracuse Research Corporation. Available from: www.epa.gov/oppt/exposure/pubs/episuite.htm

Zellers JE. 1985. Four week repeated dose dermal toxicity study in rats using heavy aromatic distillate. Project No. 2063. Prepared for Gulf Oil Chemicals Co., Houston, TX. Pittsburgh (PA): Gulf Life Sciences Centre. [cited in API 2001b].

Zhou S, Heras H, Ackman RG. 1997. Role of adipocytes in the muscle tissue of Atlantic salmon (*Salmo salar*) in the uptake, release and retention of water-soluble fraction of crude oil hydrocarbons. Mar Biol 127:545–553.

Appendix 1. Petroleum substance grouping

Table A1.1. Description of the nine groups of petroleum substances

Group[a]	**Description**	**Example**
Crude oils	Complex combinations of aliphatic and aromatic hydrocarbons and small amounts of inorganic compounds, naturally occurring under the earth's surface or under the seafloor	Crude oil
Petroleum and refinery gases	Complex combinations of light hydrocarbons primarily from C_1–C_5	Propane
Low boiling point naphthas	Complex combinations of hydrocarbons primarily from C_4–C_{12}	Gasoline
Gas oils	Comples combinations of hydrocarbons primarily from C_9–C_{25}	Diesel
Heavy fuel oils	Complex combinations of heavy hydrocarbons primarily from C_{11}–C_{50}	Fuel Oil No. 6
Base oils	Complex combinations of hydrocarbons primarily from C_{15}–C_{50}	Lubricating oils
Aromatic extracts	Complex combinations of primarily aromatic hydrocarbons from C_{15}–C_{50}	Feedstock for benzene production
Waxes, slack waxes and petrolatum	Complex combinations of primarily aliphatic hydrocarbons from C_{12}–C_{85}	Petrolatum
Bitumen or vacuum residues	Complex combinations of heavy hydrocarbons having carbon numbers greater than C_{25}	Asphalt

[a] These groups were based on classifications developed by Conservation of Clean Air and Water in Europe (CONCAWE) and a contractor's report presented to the Canadian Petroleum Products Institute (Simpson 2005).

Appendix 2. Physical and chemical data tables for industry-restricted LBPNs

Table A2.1. Substance identity of industry-restricted LBPNs

CAS RN and DSL Name	64741-42-0 Naphtha (petroleum), full-range straight-run	NCI 2006	
	64741-69-1 Naphtha (petroleum), light hydrocracked	NCI 2006	
	64741-78-2 Naphtha (petroleum), heavy hydrocracked	NCI 2006	
Chemical group	Petroleum – LBPNs		
Major components	Aliphatic and aromatic hydrocarbons		
Carbon range	64741-42-0	C_4–C_{11}	ECB 2000a
	64741-69-1	C_4–C_{10}	ECB 2000b
	64741-78-2	C_6–C_{12}	ECB 2000c
Approximate ratio of aromatics to non-aromatics	64741-42-0	4:96	ECB 2000a
	64741-69-1	26:52	ECB 2000b
	64741-78-2	20:80	ECB 2000c; CONCAWE 1992; API 2001a

Table A2.2. Physical-chemical properties for representative structures contained in LBPNs[a]

Chemical class, name and CAS RN	**Boiling point (°C)**	**Melting point (°C)**	**Vapour pressure (Pa)**	**Henry's Law constant (Pa·m^3/mol)**	**Log K_{ow}**	**Log K_{oc}**	**Aqueous solubility (mg/L at 25°C unless otherwise stated)**
Alkanes							
C_4 butane (106-97-8)	−0.5 (expt.)	−138.2 (expt.)	2.43×10^5 (expt.)	9.63×10^4 (expt.)	2.89[b] (expt.)	3.00	61[c]

C_6 hexane (110-54-3)	68.7[d]	−95.3[e] (expt.)	2.0×10^{4} (expt.)	1.8×10^{5}	3.90[b] (expt.)	2.17	fresh water: 9.5–13 (20°C); salt water: 75.5 (20°C)[e]
C_9 nonane (111-84-2)	150.8[c] (expt.)	−53.5[c] (expt.)	5.93×10^{2} (expt.)	3.4×10^{5} (expt.)	5.65[c] (expt.)	2.97	0.22 (expt.)
C_{12} dodecane (112-40-3)	216.3[c] (expt.)	−9.6[c] (expt.)	18[b] (expt.)	8.29×10^{5} (expt.)	6.10[c] (expt.)	3.77	0.0037[e]
Isoalkanes							
C_4 isobutane (75-28-5)	−11.7[f]	−138.3 (expt.)	3.48×10^{5} (expt.)	1.21×10^{5} (expt.)	2.76[f]	1.55	49[c]
C_6 2-methylpentane (43133-95-5)	60.2 (expt.)	−153.7 (expt.)	2.8×10^{4} (expt.)	1.7×10^{5} (expt.)	3.21	2.10	14 (expt.)
C_9 2,2-dimethylheptane (1071-26-7)	133 (expt.)	−113 (expt.)	1.4×10^{3}	6.4×10^{4}	4.61	2.85	0.700
C_{12} 2,3-dimethyldecane (17312-44-6)	181.36	−43	165.3	2.5×10^{5}	6.09	3.64	0.113
***n*-Alkenes**							
C_9 nonene (27215-95-8)	149.5	−56.7	500 (expt.)	2.4×10^{4}	4.55	2.97	3.62
C_{12} 9-methyl-1-undecene	192.2	−33	99.8	1.3×10^{5}	6	5.2	0.13
One-ring cycloalkanes							

C_6 cyclohexane (110-82-7)	80.7 (expt.)	6.6 (expt.)	1.3×10^4 (expt.)	1.52×10^4 (expt.)	3.44[f]	2.22	55 (expt.)
C_9 1,2,3-trimethylcyclohexane (1678-97-3)	144[g] (expt.)	−66.9[g] (expt.)	650	1.7×10^4	4.43	2.86	4.56
C_{12} *n*-hexylcyclohexane (4292-75-5)	224[g] (expt.)	−43[g] (expt.)	15.2[g] (expt.)	2.9×10^4	6.05	3.77	0.12
Two-ring cycloalkanes							
C_9 *cis*-bicyclo[4.3.0]nonane (4551-51-3)	167[g] (expt.)	−53[g] (expt.)	320	2.0×10^3	3.71	3.00	19.3
C_{12} dicyclohexyl (92-51-3)	177.9[g] (expt.)	−51.4[g] (expt.)	196[g] (expt.)	20.4 (expt.)	3.18[g] (expt.)	3.00	109 (expt.)
One-ring aromatics							
C_6 benzene (71-43-2)	80[g] (expt.)	5.5 (expt.)	1.2×10^4	562	2.13[d] (expt.)	2.22	1790[d] (expt.)
C_9 1-methyl-2-ethylbenzene (611-14-3)	165.2[g] (expt.)	−80.8[g] (expt.)	348	560	3.53[g] (expt.)	2.93	74.6[g] (expt.)
C_{12} 1,2,3-triethylbenzene (42205-08-3)	229.59	11.85	10.6	595.2	5.11	3.72	1.8
Two-ring aromatics							
C_{12} biphenyl (92-52-4)	256.1[g] (expt.)	69[g] (expt.)	1.19 (expt.)	31.2 (expt.)	3.98[g] (expt.)	3.8	6.94 (expt.)

[a] All values are modelled unless denoted with an (expt.) for experimental data. Models used were: melting and boiling points and vapour pressure, MPBPWIN (2008); Henry's Law constant, HENRYWIN (2008); log K_{ow}, KOWWIN (2008); log K_{oc}, KOCWIN (2009); water solubility, WSKOWWIN (2008).

[b] McAuliffe 1966

[c] McAuliffe 1963

[d] PETROTOX 2009

[e] Verschueren 2001

[f] Hansch et al. 1995

[g] EPI Suite 2008

Appendix 3. Measures designed to prevent, reduce or manage unintentional releases

For the Canadian petroleum industry, requirements at the provincial/territorial level typically prevent or manage the unintentional releases of petroleum substances and streams within a facility through the use of operating permits (SENES 2009).

At the federal level, unintentional releases of some petroleum substances are addressed under the *Petroleum Refinery Liquid Effluent Regulations* and guidelines in the *Fisheries Act* (Canada 2010). These regulations set the discharge limits of oil and grease, phenol, sulfides, ammonia nitrogen and total suspended matter, and lay out testing requirements for acute toxicity in the final petroleum effluents entering Canadian waters.

Additionally, existing occupational health and safety legislation specifies measures to reduce occupational exposures of employees, and some of these measures also serve to reduce unintentional releases (CanLII 2009).

Non-regulatory measures (e.g., guidelines, best practices) are also in place at petroleum sector facilities to reduce unintentional releases. Such control measures include appropriate material selection during the design and setup processes; regular inspection and maintenance of storage tanks, pipelines and other process equipment; the implementation of leak detection and repair or other equivalent programs; the use of floating roofs in above-ground storage tanks to reduce the internal gaseous zone; and the minimal use of underground tanks, which can lead to undetected leaks or spills (SENES 2009).

Under the *Canada Shipping Act, 2001* (Canada 2001), releases of petroleum substances from marine loading and unloading and transportation are managed by pollution prevention and response provisions (Parts 8 and 9), including the establishment of pollution prevention plans and pollution emergency plans for any discharges during loading or unloading activities.

For those substances containing highly volatile components (e.g., LBPNs, gasoline), a vapour recovery system is generally implemented or recommended at loading terminals of Canadian petroleum facilities (SENES 2009). Such a system is intended to reduce evaporative emissions during handling procedures.

Appendix 4. Release estimation of industry-restricted LBPNs during transportation

Table A4.1. National naphtha spills information, 2000–2009, from Environment Canada's Spill Line database (Environment Canada 2011)[a]

Year	Minimum spill volume (litres)	Maximum single spill volume (litres)	Median spill volume (litres)	Total number of spills reported	Number of spills with unknown volume	Total known volume spilled (litres)	Extrapolated total volume spilled (litres)
2009		5500		1	0	5500	5500
2008		600		4	2	600	1800
2007[b]		1590		2	1	1590	3180
2006	200	6400	3300	4	2	6600	13 200
2005	318	1260	789	2	0	1578	1578
2004		40		1	0	40	40
2003	0	0	0	0	0	0	0
2002	0	0	0	0	0	0	0
2001	0	0	0	0	0	0	0
2000		2226		1	0	2226	2226
Total volume spilled						18 133	27 524
Average volume spilled (Estimated total volume spilled/total number spills)							1966

[a] Collisions, poor road conditions and/or adverse weather-related events listed as a source, cause or reason of spill were not included in the release estimate. Releases where the source was pipeline or train were also not considered.

[b] An extremely large spill (190 776 L) in Alberta in 2007 was not included.

Table A4.2. Known volume (L) of naphtha spills in Canada, 2000–2009 (Environment Canada 2011)

Province	Year										Total
	2000	2001	2002	2003	2004	2005	2006	2007	2008	2009	
AB							6600				6600
SK										5500	5500
MB						1260					1260
QC					40	318			600		958
NL	2226							1590			3816

Table A4.3. Naphtha releases to air, land and freshwater reported in the Environment Canada Spill Line database (Environment Canada 2011)

	Air	Land	Fresh water
2000	0	1	0
2001	0	0	0
2002	0	0	0

2003	0	0	0
2004	0	1	0
2005	1	1	0
2006	1	3	0
2007	1	1	0
2008	2	1	1
2009	0	1	0
Total	5	8	1
Total volume (L)	1018	17 115	N/A
Total estimated volume (L)	2036	25 488	N/A
Average volume (L)	407	3186	N/A

N/A – Not available; the release to fresh water did not have a reported volume.

Table A4.4a. Sources of naphtha spills in Canada, 2000–2009 (Environment Canada 2011)

Source	**Total number of releases**	**Total volume of releases (L)**	**Proportion of volume**	**Average release (L)**
Other industrial plant	5	8178	0.45	2044
Refinery	5	6100	0.34	3050
Other	1	2226	0.12	2226
Unknown	1	1590	0.09	1590
Tank truck	1	40	0.00	40
Service station	1	N/A	0.00	N/A
Total	**14**	**18 134**	**1.00**	**2014**

N/A – not available.

Table A4.4b. Causes of naphtha spills in Canada, 2000–2009 (Environment Canada 2011)

Cause	**Total number of releases**	**Total volume of releases (L)**	**Proportion of volume**	**Average release (L)**
Other	5	8826	0.49	2942
Valve, fitting leak	3	5818	0.32	2909
Pipe leak	3	2190	0.12	1095
Overturn	1	1260	0.07	1260
Discharge	1	40	0.00	40
Process upset	1	N/A	0.00	N/A
Total	**14**	**18 134**	**1.00**	**2014**

N/A – not available.

Table A4.4c. Reasons for naphtha spills in Canada, 2000–2009 (Environment Canada 2011)

Reason	Total number of releases	Total volume of releases (L)	Proportion of volume	Average release (L)
Equipment failure	7	13 690	0.75	3738
Other	1	2226	0.12	2226
Error	2	1300	0.07	650
Material failure	2	600	0.03	600
Fire, explosion	1	318	0.02	318
Intentional	1	N/A	0.00	N/A
Total	**14**	**18 134**	**1.00**	**2014**

N/A – not available.

Appendix 5. Modelling results for environment properties of industry-restricted LBPNs

Table A5.1. Results of the Level III fugacity modelling (EQC 2003)

Compartment of release (100%)	**Percentage of substance partitioning into each compartment**			
***n*-Alkanes**	**Air**	**Water**	**Soil**	**Sediment**
C_4 butane				
Air	100	0	0	0
Water	9.3	90.4	0	0.3
Soil	93.5	0	6.5	0
C_6 hexane				
Air	100	0	0	0
Water	5.8	92.5	0	1.7
Soil	66.5	0	33.5	0
C_9 nonane				
Air	99.5	0.03	0.5	0.02
Water	1.5	48	0	50.5
Soil	0.1	0	99.9	0
C_{12} dodecane				
Air	99.6	0	0.4	0
Water	0.4	23.6	0	76.0
Soil	3.0	0	97.0	0
Isoalkanes				
C_4 isobutane				
Air	100	0	0	0
Water	9.7	90.1	0	0.2
Soil	94.8	0	5.2	0
C_6 methylpentane				
Air	100	0	0	0
Water	5.9	93.8	0	0.3
Soil	89.8	0.01	10.2	0
C_9 2,3-dimethylheptane				
Air	99.8	0	0.2	0
Water	3.3	85.7	0	11
Soil	6.2	0	93.7	0
C_{12} 2,3-dimethyldecane				
Air	99.4	0	0.6	0
Water	0.4	23.3	0	76.3
Soil	0.9	0	99.0	0
***n*-Alkenes**				
C_9 nonene				
Air	99.8	0	0.2	0
Water	0.7	93.5	0	5.8
Soil	0.8	0	99.2	0

C_{12} 9-methyl-1-undecene				
Air	99.4	0	0.6	0
Water	0.4	27.6	0	72
Soil	0.5	0	99.5	0
One-ring cycloalkanes				
C_6 cyclohexane				
Air	99.9	0.02	0.06	0
Water	4.1	91.2	0	4.7
Soil	33.0	0.2	66.8	0
C_9 1,2,3-trimethylcyclohexane				
Air	99.8	0	0.2	0
Water	2.8	93.4	3.8	0
Soil	3.2	0	96.8	0
C_{12} *n*-hexylcyclohexane				
Air	99.0	0	0.9	0.04
Water	0.3	20.1	0	79.6
Soil	0.07	0	99.9	0
Two-ring cycloalkanes				
C_9 *cis*-bicyclo[4.3.0]nonane				
Air	99.0	0.2	0.8	0.01
Water	2.7	88.8	0.02	8.5
Soil	2	0.1	97.9	0.01
C_{12} dicyclohexyl				
Air	98.3	0.02	1.6	0.1
Water	0.2	16.1	0	83.7
Soil	0.05	0	99.9	0.01
One-ring aromatics				
C_6 benzene				
Air	99.7	0.2	0.1	0
Water	10.4	89.4	0	0.2
Soil	37.7	1.0	61.2	0
C_9 1-methyl-2-ethylbenzene				
Air	99.4	0.3	0.3	0
Water	4.4	94.6	0.01	0.9
Soil	1.0	0.1	98.9	0
C_{12} 1,2,3-triethylbenzene				
Air	99.4	0.2	0.4	0.04
Water	1.6	76.1	0	22.3
Soil	0	0	100	0
Two-ring aromatics				
C_{12} biphenyl				
Air	85.6	9.9	3.4	1.1

Water	1.6	88.2	0.06	10.1
Soil	0	0.1	99.9	0

Table A5.2. Empirical biodegradation half-lives of hydrocarbons from a formulated gasoline (Prince et al. 2007b)

Class and compound	Median half-life (days)	Mean half-life (days)
Aromatics		
benzene	3.2	4.6
1-methylethylbenzene	3.2	5.2
2-ethyl-1,3-dimethylbenzene	3.2	4.9
Two-ring aromatics		
naphthalene	3.2	4.4
***n*-Alkanes**		
butane	15.0	31.8
hexane	6.5	10.2
nonane	3.2	4.4
dodecane	2.8	3.8
Isoalkanes		
2-methylpropane (isobutane)	17.1	41.7
2-methylpentane	10.4	16.7
3-methylpentane	10.1	21.3
2-methylheptane	4.8	6.0
4-methylnonane	3.2	4.8
Cycloalkanes		
1,1,3-trimethylcyclohexane	8.5	14.2
Alkenes		
cis-3-hexene	6.5	8.4
Cycloalkenes		
cyclopentene	8.1	11.5
4-methylcyclopentene	8.1	12.5

Table A5.3. Modelled data for primary (BIOHCWIN 2008; BIOWIN 4 2009) and ultimate (BIOWIN 3, 5, 6 2009; CATABOL c2004-2008; TOPKAT 2004) degradation of LBPNs

	Primary Biodegradation	
	BioHCWin (2008)[a]	**BIOWIN 4 (2009) Expert Survey[b]**
Alkanes		
C_4 butane	4	4.0
C_6 hexane	5	3.99
C_9 *n*-nonane	7	4.20
C_{12} dodecane	12	4.14
Isoalkanes		
C_4 isobutane	3	3.76
C_6 2-methylpentane	4	3.72
C_9 2,3-dimethylheptane	8	3.93
C_{12} 2,3-dimethyldecane	12	3.87
Alkenes		
C_9 nonene	4	4.2
C_{12} 9-methyl-1-undecene	11	3.60
One-ring cycloalkanes		
C_6 cyclohexane	55.4 (28–182)[c]	3.73
C_9 1,2,3-trimethylcyclohexane	4	3.67
C_{12} *n*-hexylcyclohexane	16	3.87
Two-ring cycloalkanes		
C_9 cis-bicyclononane	56	3.67
C_{12} dicyclohexyl	27	3.61
One-ring aromatics		
C_6 benzene	4.6(5–16)[c]	3.39
C_9 1-methyl-2-ethylbenzene	5	3.54
C_{12} 1,2,3-triethylbenzene	5	3.41

	Primary Biodegradation	
	BioHCWin (2008)[a]	**BIOWIN 4 (2009) Expert Survey[b]**
Two-ring aromatics		
C_{12} biphenyl	31.0 (1.5–7)[c]	3.64

Table A5.3 cont. Modelled data for primary (BioHCWin 2008; BIOWIN 4 2009)[a] andultimate (BIOWIN 3, 5, 6 2009; CATABOL c2004–2008; TOPKAT 2004) degradation of LBPNs

	Ultimate Biodegradation					
	BIOWIN 3 (2009) Expert Survey[b]	**BIOWIN 5 (2009) MITI linear probability[d]**	**BIOWIN 6 (2009) MITI non-linear probability[d]**	**CATABOL (2008) % BOD**	**TOPKAT (2004) Probability of biodegradability**	**Extrapolated half-life compared with criteria (days)**
Alkanes						
C_4 butane	3.4	0.64	0.85	98	1	< 182
C_6 hexane	3.3	0.65	0.86	98	1	< 182
C_9 *n*-nonane	3.51	0.68	0.87	99.95	1	< 182
C_{12} dodecane	3.42	0.70	0.87	100	1	< 182
Isoalkanes						
C_4 isobutane	3.07	0.49	0.69	10.6	0.98	< 182
C_6 2-methylpentane	0.71	0.51	0.70	16.7	1	< 182
C_9 2,3-dimethyl-heptane	3.21	0.38	0.50	7.8	1	< 182
C_{12} 2,3-dimethyldecane	3.12	0.40	0.52	60.2	1	< 182
Alkenes						
C_9 nonene	3.52	0.60	0.75	43.9	0.32	< 182
C_{12} 9-methyl-1-undecene	2.83	0.53	0.67	27.8	1	< 182
One-ring cycloalkanes						
C_6 cyclohexane	3.01	0.58	0.82	100	0	< 182
C_9 1,2,3-	2.92	0.43	0.32	2.64	0.011[e]	< 182

	Ultimate Biodegradation					
	BIOWIN 3 (2009) Expert Survey[b]	**BIOWIN 5 (2009) MITI linear probability[d]**	**BIOWIN 6 (2009) MITI non-linear probability[d]**	**CATABOL (2008) % BOD**	**TOPKAT (2004) Probability of biodegradability**	**Extrapolated half-life compared with criteria (days)**
trimethyl-cyclohexane						
C_{12} *n*-hexyl-cyclohexane	3.13	0.57	0.71	4.3	1	< 182
Two-ring cycloalkanes						
C_9 cis-bicyclo-nonane	2.92	0.51	0.58	0	0.001	< 182
C_{12} dicyclohexyl	2.83	0.44	0.46	0	1	< 182
One-ring aromatics						
C_6 benzene	2.44	0.53	0.73	7.5	1	< 182
C_9 1-methyl-2-ethylbenzene	2.78	0.37	0.44	10.7[e]	0.09	< 182
C_{12} 1,2,3-triethyl benzene	2.62	0.09	0.11	5.7	0	≥ 182
Two-ring aromatics						
C_{12} biphenyl	2.90	0.34	0.33	12.8	0.57	< 182

Abbreviations: BOD, biological oxygen demand; MITI, Ministry of International Trade & Industry, Japan

[a] Half-life estimations are for non-specific media (i.e., water, soil and sediment).

[b] Output is a numerical score from 0–5.

[c] Howard et al. (1991)

[d] Output is a probability score.

[e] Modelled results were found to be out of domain and therefore not considered for persistence. For modelled results of CATABOL that were found to be out of domain, it was assumed that results for TOPKAT, BIOWIN 5, 6 were also out of domain because these models use the same dataset. In these cases, only BIOWIN 3, 4 and BioHCWin were considered when determining the persistence of the component.

Table A5.4. Empirical data for photodegradation of components of LBPNs (Atkinson 1990)

Substance	Half-lives in air (days)
butane	3.4
isobutane	3.2

pentane	2.0
isopentane	2.0

Table A5.5. Modelled atmospheric degradation of representative structures for LBPNs (AOPWIN 2008)

	Half-lives (days)	
	Oxidation	**Ozone**[a]
Alkanes		
C_4 butane	4.1	N/A[b]
C_6 hexane	2	N/A
C_9 nonane	1.1	N/A
C_{12} dodecane	0.8	N/A
Isoalkanes		
C_4 isobutane	4.4	N/A
C_6 methylpentane	2	N/A
C_9 2,3-dimethylheptane	1.1	N/A
C_{12} 2,3-dimethyldecane	0.8	N/A
***n*-Alkenes**		
C_9 nonene	0.1	0.1
C_{12} 9-methyl-1-undecene	0.28	0.96
One-ring cycloalkanes		
C_6 cyclohexane	1.3	N/A
C_9 1,2,3-trimethylcyclohexane	0.8	N/A
C_{12} *n*-hexylcyclohexane	0.6	N/A
Two-ring complex rings		
C_9 cis-bicyclo[4.3.0]nonane	0.8	N/A
C_{12} dicyclohexyl	1.3	N/A
One-ring aromatics		
C_6 benzene	5.5 (2–20)[a]	N/A
C_9 1-methyl-2-ethylbenzene	1.4	N/A
C_{12} 1,2,3-triethylbenzene	0.6	N/A
Two-ring aromatics		
C_{12} biphenyl	1.6	N/A

[a] Howard et al. (1991)

[b] N/A: not available.

Table A5.6. Experimental BAFs for aromatic hydrocarbons

	Reference **Species; Study details**	**Log K_{ow}**	**BAF experimental (L/kg ww)**
One-ring aromatics			
C_6 benzene	Zhou et al.1997 Atlantic salmon (white muscle); 96-hour (WSF of crude oil)	2.13 (expt.)	4

C_7 toluene	Zhou et al. 1997 Atlantic salmon (white muscle); 96-hour (WSF of crude oil)	2.73 (expt.)	11
C_8 ethylbenzene	Zhou et al. 1997 Atlantic salmon (white muscle); 96-hour (WSF of crude oil)	3.15 (expt.)	26
C_9 xylenes	Zhou et al. 1997 Atlantic salmon (white muscle); 96-hour (WSF of crude oil)	3.12 (expt.)	47
C_9 isopropyl-benzene	Zhou et al. 1997 Atlantic salmon (white muscle); 96-hour (WSF of crude oil)	3.66 (expt.)	20
C_9 propylbenzene	Zhou et al. 1997 Atlantic salmon (white muscle); 96-hour (WSF of crude oil)	3.69 (expt.)	36
C_9 ethylmethyl-benzene	Zhou et al. 1997 Atlantic salmon (white muscle); 96-hour (WSF of crude oil)	3.98 (expt.)	51
C_{12} trimethyl-benzene	Zhou et al. 1997 Atlantic salmon (white muscle); 96-hour (WSF of crude oil)	3.66 (expt.)	74
Two-ring aromatics			
C_{10} naphthalene	Neff et al. 1976 Clam; 24-hour (oil-in-water dispersion of No. 2 fuel oil) lab study	3.30 (expt.)	2.3
C_{11} methyl naphthalenes	Zhou et al. 1997 Atlantic salmon (white muscle); 96-hour (WSF of crude oil) lab study	3.87 (expt.)	230
C_{11} 1-methyl-naphthalene	Neff et al. 1976 Clam; 24-hour (oil-in-water dispersion of No. 2 fuel oil) lab study	3.87 (expt.)	8.5
C_{11} 2-methyl-naphthalene	Neff et al. 1976 Clam; 24-hour (oil-in-water dispersion of No. 2 fuel oil) lab study	3.86 (expt.)	8.1
C_{12} dimethyl-naphthalene	Neff et al. 1976 Clam; 24-hour (oil-in-water dispersion of No. 2 fuel oil) lab study	4.31 (expt.)	17.1

Abbreviation: (expt.), experimental log K_{ow} data

Table A5.7. Fish BAF and BCF predictions for representative structures of LBPNs using the Arnot-Gobas three trophic level model (2004) with corrections for metabolism rate (k_m) and dietary assimilation efficiency (E_d)

	Log K_{ow}	**Metabolic rate constant for MTL fish (day^{-1})[a]**	**BCF[b] MTL fish (L/kg ww)**	**BAF[b] MTL fish (L/kg ww)**
Alkanes				
C_4 butane	2.9	0.6	47	47
C_6 hexane	3.9	0.3	302	302
C_9 nonane	5.7	0.09	1905	4074
C_{12} dodecane	6.1	2.2 (expt.)[c]	126	155
Isoalkanes				
C_4 isobutane	2.8	0.7	38	38
C_6 methylpentane	3.2	0.5	85	85
C_9 2,3-dimethyl-heptane	4.6	0.02 (expt.)	2138	2754
C_{12} 2,3-dimethyl-decane	6.1	1.22[d]	794	1950
***n*-Alkenes**				
C_9 nonene	4.6	0.1	955	1000
C_{12} 9-methyl-1-undecene	6.0	0.08	1995	**7079**[f]
One-ring cycloalkanes				
C_6 cyclohexane	3.0	1.6 (expt.)	44	44
C_9 1,2,3-trimethyl-cyclohexane	4.4	0.09	966	1000
C_{12} *n*-hexyl-cyclohexane	6.1	0.023[e](expt.)	6025	**57 543**
Two-ring cycloalkanes				
C_9 cis-bicyclo[4.3.0]nonane	3.7	0.08	272	280
C_{12} dicyclohexyl	5.9	0.1 (expt.)	1175	2512
One-ring aromatics				
C_6 benzene	2.2	0.2	11	11
C_9 1-methyl-2-ethylbenzene	2.9	0.3	51	51
C_{12} 1,2,3-triethylbenzene	3.7	0.2	257	257
Two-ring aromatics				
C_{12} biphenyl	3.8	0.2	295	302

[a] Metabolic rate constant normalized to middle trophic level (MTL) fish in Arnot-Gobas three trophic level model (2004) at W = 184 g, T = 10°C, L = 6.8%) based on estimated QSAR vaues from BCFBAF v3.01 unless otherwise indicated

[b] Arnot-Gobas BCF and BAF predictions for midde trophic level fish using three trophic level model (Arnot and Gobas 2004) using normalized rate constant and correcting for observed or estimated dietary assimilation efficiency reported in Table A5.8b (Appendix 5).
[c] (expt.) – experimental half-life used.
[d] Based on calculated metabolic rate constant for *n*-dodecane.
[e] Based on calculated metabolic rate for C_{14} *n*-octylcyclohexane.
[f] Bolded values refer to BAFs ≥ 5000 based on the *Persistence and Bioaccumulation Regulations* (Canada 2000a)

Table A5.8a. Experimental and predicted BCFs and BAFs for selected representative structures

Substance	Log K_{ow}	BCF Measured (L/kg ww)	Predicted BCF[a] (L/kg ww)		Predicted BAF[a] (L/kg ww)		Reference; species
			Study conditions[b]	MTL fish[c]	Study conditions[b]	MTL fish[c]	
Alkanes							
C_8 octane	5.18 (expt.)	530	537	490	560	537	JNITE 2010; carp
C_{12} *n*-dodecane	6.10 (expt.)	240	240	794	251	1950	Tolls and van Dijk 2002; fathead minnow
One-ring cycloalkanes							
C_6 cyclohexane	3.44 (expt.)	77	77	89	77	89	CITI 1992; carp
C_7 1-methyl-cyclohexane	3.61 (expt.)	240	190[f]	275[f]	229[f]	426[f]	CITI 1992; carp
C_8 ethylcyclohexane	4.56 (expt.)	2529	1622[f]	2344[f]	4467[f]	5495[f]	CITI 1992; carp
Two-ring cycloalkanes							
C_{10} trans-decalin	4.20	2200	724[f]	1072[f]	1288[f]	1660[f]	CITI 1992; carp
C_{10} cis-decalin	4.20	2500	724[f]	1072[f]	1288[f]	1660[f]	CITI 1992; carp
One-ring aromatics							
1,2,3-trimethyl-benzene	3.66 (expt.)	133[d]	135	155	135	155	CITI 1992; carp
C_{10} 1,2-diethyl-benzene	3.72 (expt.)	516[d]	245[f]	355[f]	309[f]	427[f]	CITI 1992; carp
C_{11} 1-methyl-4-tertbutylbenzene	3.66 (expt.)	< 1.0	214[f]	309[f]	263[f]	263[f]	JNITE 2010; carp
Cycloalkane monoaromatics							

C_{10} tetralin	3.49 (expt.)	230	145 [f]	214 [f]	166 [f]	562 [f]	CITI 1992; carp
Two-ring aromatics							
C_{10} naphthalene	3.30 (expt.)	94	95 [f]	138 [f]	105 [f]	148 [f]	JNITE 2010; carp
C_{11} 2-methylnapthalene	3.86 (expt.)	2886[d] 3930[e]	2884[f]	N/A	2884 [f]	N/A	Jonsson et al. 2004; sheepshead minnow
C_{12} 1,3-dimethyl-naphthalene	4.42 (expt.)	4039[d] 5751[e]	4073	N/A	4073	N/A	Jonsson et al. 2004; sheepshead minnow
Cycloalkane diaromatics							
C_{12} acenaphthene	3.92 (expt.)	991[d]	389	562	977	741	CITI 1992; carp

[a] BCF and BAF predictions were performed using the Arnot-Gobas mass-balance kinetic model normalizing the metabolic rate constant according to fish weight, lipid content and temperature reported in study or protocol.

[b] Fish weight, lipid content and water temperature used when specified in study. For CITI/NITE tests when conditions not known, fish weight =30 g, lipid = 4.7%, temperature = 22°C for carp in accordance with MITI BCF test protocol. When more than one study was reported, the geomean of study values was used for model normalization inputs.

[c] Kinetic mass-balance predictions made for middle trophic level fish (W = 184 g, T = 10°C, L = 6.8%) in Arnot-Gobas three trophic level model (Arnot and Gobas 2004).

[d] Geometric mean of reported steady-state values.

[e] Geometric mean of reported kinetic values.

[f] Predictions generated with metabolism rate equal to zero due to negative predicted metabolism rate constant. Metabolism rate constant deemed erroneous or not applicable given log k_{ow} and BCF result (see kinetic rate constants table).

N/A – not applicable; study details could not be obtained to determine predicted BCFs and BAFs.

(e) – experimental data.

Table 5.8b. Calculated kinetic rate constants for selected representative structures

Substance	**Study endpoint**	**Gill elimination rate constant day^{-1} (k_2)**	**Metabolic rate constant day^{-1} (k_M)[a]**	**Growth rate constant day^{-1} (k_G)**	**Fecal egestion rate constant day^{-1} (k_E)[c]**
Alkanes					
C_8 octane[g]	BCF_{ss}[f]	0.077	0.657	0.001	0.007
C_{12} *n*-dodecane	BCF_{ss}[f]	0.035	4.95	0.002	0.013
One-ring cycloalkanes					
C_6 cyclohexane	BCF_{ss}[f]	3.031	2.050	0.001	0.008

C_7 1-methylcyclohexane[g]	BCF_{ss}[f]	2.072	-0.429	0.001	0.008
C_8 ethylcyclohexane[g]	BCF_{ss}[f]	0.238	-0.087	0.001	0.008
Two-ring cycloalkanes					
C_{10} trans-decalin[g]	BCF_{ss}[f]	0.510	-0.336	0.001	0.008
C_{10} cis-decalin[g]	BCF_{ss}[f]	0.542	-0.390	0.001	0.008
One-ring aromatics					
C_9 1,2,3-trimethylbenzene[g]	BCF_{ss}[f]	1.852	1.128	0.001	0.008
C_{10} 1,2-diethylbenzene[g]	BCF_{ss}[f]	1.617	-0.854	0.001	0.008
C_{11} 1-methyl-4-tertbutyl-benzene[g]	BCF_{ss}[f]	1.852	395.6	0.001	0.008
Cycloalkane monoaromatics					
C_{10} tetralin[g]	BCF_{ss}[f]	2.711	-1.009	0.001	0.008
Two-ring aromatics					
C_{10} naphthalene[g]	BCF_{ss}[f]	4.129	-0.020	0.001	0.008
C_{11} 2-methylnaphthalene[g]	BCF_{ss}[f]				
	$BCF_{kinetic}$[f]	0.607	0.000	0.002	0.001
C_{12} 1,3-dimethylnaphthalene[g]	BCF_{ss}[f]	N/A	N/A	N/A	N/A
	$BCF_{kinetic}$[f]	0.403	0.000	0.002	0.001
Cycloalkane diaromatics					
C_{12} acenaphthene[g]	BCF_{ss}[f]	1.028	-0.632	0.001	0.008

Table A5.8b cont. Calculated kinetic rate constants for selected representative structures

Substance	**Study endpoint**	**Total elimination rate constant day^{-1} (k_T)[b]**	**Uptake rate constants day^{-1} (k_1)**	**Dietary assimilation efficiency (α, E_D)**	**Reference, species**
Alkanes					
C_8 octane[g]	BCF_{ss}[f]	0.742	406		JNITE 2010; carp

C_{12} *n*-dodecane	BCF_{ss}[f]	5.00	1525		Tolls and van Dijk 2002; fathead minnow
One-ring cycloalkanes					
C_6 cyclohexane	BCF_{ss}[f]	5.090	392		CITI 1992; carp
C_7 1-methylcyclohexane[g]	BCF_{ss}[f]	2.081	397		CITI 1992; carp
C_8 ethylcyclohexane[g]	BCF_{ss}[f]	0.247	405		CITI 1992; carp
Two-ring cycloalkanes					
C_{10} trans-decalin[g]	BCF_{ss}[f]	0.519	404		CITI 1992; carp
C_{10} cis-decalin[g]	BCF_{ss}[f]	0.551	404		CITI 1992; carp
One-ring aromatics					
C_9 1,2,3-trimethylbenzene[g]	BCF_{ss}[f]	2.989	398		CITI 1992; carp
C_{10} 1,2-diethylbenzene[g]	BCF_{ss}[f]	1.679	398		CITI 1992; carp
C_{11} 1-methyl-4-tertbutyl-benzene[g]	BCF_{ss}[f]	398.2	398		JNITE; carp
Cycloalkane monoaromatics					
C_{10} tetralin[g]	BCF_{ss}[f]	2.720	394		CITI 1992; Carp
Two-ring aromatics					
C_{10} naphthalene[g]	BCF_{ss}[f]	4.138	387		JNITE 2010; carp
C_{11} 2-methylnaphthalene[g]	BCF_{ss}[f] $BCF_{kinetic}$[f]	0.610[d] 0.610	 1089	 3.2%[e]	Jonsson et al. 2004; sheepshead minnow
C_{12} 1,3-dimethylnaphthalene[g]	BCF_{ss}[f] $BCF_{kinetic}$[f]	0.406[d] 0.406	2322[d] 1100	N/A 3.2%[e]	Jonsson et al. 2004 (cited in Lampi et al. 2010); sheepshead minnow
Cycloalkane					

diaromatics					
C_{12} acenaphthene[g]	BCF_{ss}[f]	1.037	401		CITI 1992; carp

[a] Negative values of k_M indicate possible kinetic model error, as the estimated rate of metabolism exceeds the total of all other elimination rate constants combined. Observed BCFs may thus not be explained by kinetic modelling of metabolic rate (e.g., steric hindrance, low bioavailability) and could also point to study exposure error. Negative values of k_M are not included in the estimate of k_T.

[b] Calculated using kinetic mass-balance BCF or BAF model based on reported rate kinetics of empirical study and correcting for log K_{ow}, fish body weight, temperature and lipid content of fish from cited study.

[c] $k_T = (k_2 + k_M + k_E + k_G)$ or when depuration rate constant is known $k_T = (k_2 + k_G)$

[d] As reported in empirical study (geomean used when multiple values reported).

[e] Based on assimilation efficiency data for 6-*n*-butyl-2,3-dimethylnaphthalene.

[f] BCF steady state (tissue conc./water conc.).

[g] Structures that are included as analogues for the chosen representative structures.

N/A – not applicable; study details could not be obtained to determine predicted BCFs and BAFs.

Table A5.9. An analysis of modelled persistence and bioaccumulation data on petroleum hydrocarbons with respect to the Canadian *Persistence and Bioaccumulation Regulations*

C#	**C_4**	**C_6**	**C_9**	**C_{12}**
***n*-alkane**	P[a]	P[a]		
***i*-alkane**	P[a]	P[a]		
alkene				B
monocycloalkane				B
dicycloalkane	(-)			
monoaromatic	(-)	P[a]		
diaromatic	(-)	(-)		

P[a] – Predicted persistence in air based on data from AOPWIN (2008).

P – Predicted persistence in soil, water and sediment based on data from BioHCWin (2008), BIOWIN (2008), CATABOL (c2004-2008) and TOPKAT (2004).

B – Predicted fish BCFs and/or BAFs using kinetic mass-balance model (Arnot and Gobas 2003).

Blank cell – representative structures are neither persistent nor bioaccumulative.

(-) – No such carbon numbers exist within the group.

Table A5.10. Aquatic toxicity of LBPNs naphthas and gasoline

Organism	**Common name**	**Substance**	**Endpoint**	**Duration (hours)**	**Toxicity value (mg/L)**	**Reference**
Fish						
Cyprinodon variegatus	Sheepshead Minnow	Gasoline (API PS-6)	LC_{50}	96	8.3	CONCAWE 1992
		Synthetic gasoline	LC_{50}	96	5.3	CONCAWE 1992
Lepomis macrochirus	Bluegill Sunfish	Gasoline (API PS-6)	LC_{50}	96	6.3	CONCAWE 1992
		Synthetic gasoline	LC_{50}	96	6.4	CONCAWE 1992
Oncorhynchus mykiss	Rainbow Trout	Gasoline (API PS-6)	LC_{50}	96	2.7	CONCAWE 1992

		Synthetic gasoline	LC$_{50}$	96	5.1	CONCAWE 1992
		Unleaded/low-lead gasoline	LC$_{50}$	48	5.4–6.8	CONCAWE 1992
			LC$_{50}$	96	125.0–182.0	CONCAWE 1992
			LC$_{50}$	168	96.0–182.0	CONCAWE 1992
			LL$_{50}$	96	10–18	CONCAWE 1996
			NOEL	96	4.5–10	CONCAWE 1996
		Naphtha mixtures	LL$_{50}$	96	10-18	CONCAWE 1996
	Larvae	Unleaded/low-lead gasoline	LC$_{50}$	48	7	Lockhart 1987
			LC$_{50}$	48	5	Lockhart 1987
			EC$_{50}$ closed container	48	6.80	Whiticar et al. 1993
			EC$_{50}$ open container	48	5.40	Whiticar et al. 1993
Alburnus alburnus	Common Bleak	Unleaded/low-lead gasoline	LC$_{50}$	24	47.0	CONCAWE 1992
Alosa sapidissima	American Shad	Gasoline (unspecified)	TLM	24	90–91	CONCAWE 1992
			TLM	48	91	CONCAWE 1992
Odontesthes argentinensis	Marine Pejerrey larvae	Gasoline (unspecified)	LC$_{50}$	96	54.8	Rodrigues et al. 2010
Pimephales promelas	Fathead Minnow	Naphtha mixtures	LC$_{50}$	96	8.3	PPSC 1995a
Freshwater invertebrates						
Daphnia magna	Water Flea	Gasoline (API PS-6)	EC$_{50}$	48	3	CONCAWE 1992
		Synthetic gasoline	EC$_{50}$	48	1.2	CONCAWE 1992
		Unleaded/low-lead gasoline	EC$_{50}$	24	260	CONCAWE 1992
			EC$_{50}$	24	345	CONCAWE 1992
			EC$_{50}$	48	6.3	MacLean and Doe 1989
			EC$_{50}$	48	4.9	MacLean and Doe 1989

			LC_{50}	48	6.8	Lockhart et al. 1987
			LC_{50}	48	5.4	Lockhart et al. 1987
			LC_{50}	48	50	MacLean and Doe 1989
			LC_{50}	48	18	EETD 1989
			EC_{50}	48	4.5–13	CONCAWE 1996
			NOEL	48	0.1–4.5	CONCAWE 1996
			LC_{50}	48	18.4–50.3	Whiticar et al. 1993
			EC_{50}	48	1.79–4.91	Whiticar et al. 1993
		Naphtha mixtures	EL_{50}	48	4.5–32	PPSC 1995b; CONCAWE 1996
Marine invertebrates						
Artemia sp.	Brine Shrimp	Unleaded/low-lead gasoline	EC_{50}	48	25.1	CONCAWE 1992
			LC_{50}	48	51	MacLean and Doe 1989
			LC_{50}	48	18	EETD 1989
			LC_{50}	48	17.7–51.4	Whiticar et al. 1993
			EC_{50}	48	8.6–25.1	Whiticar et al. 1993
Mysidopsis bahia	Mysid Shrimp	Gasoline (API PS-6)	LC_{50}	96	1.8	CONCAWE 1992
		Synthetic gasoline	LC_{50}	96	0.3	CONCAWE 1992
		Naphtha mixtures	EL_{50}	96	13.8	PPSC 1995c
Strongylocentrotus droebachiensis eggs	Green Sea Urchin	Gasoline (unspecified)	Cytolysis		> 38	CONCAWE 1992
Strongylocentrotus pallidus eggs	Pale Sea Urchin	Gasoline (unspecified)	Irregular cleavage		28	CONCAWE 1992
Nitocra spinipes	Copepod	Unleaded/low-lead gasoline	LC_{50}	96	171.0	CONCAWE 1992
Crangon crangon	Common Shrimp	Gasoline (unspecified)	LC_{50}	96	15	CONCAWE 1992
		Naphtha (64742-73-0)	LC_{50}	96	4.3	ECB 2000a
Tigriopus californicus	Copepod	Gasoline (unspecified)	85% mortality	24	1	CONCAWE 1992

Tretraselmis chuii	Microalgae	14 gasoline formulations	IC_{50}	96	4.93–96.52	Paixão et al. 2007
Crassostrea rhizophorae	Oyster embryos	14 gasoline formulations	EC_{50}	24	8.25–41.37	Paixão et al. 2007
Chaetogammarus marinus	Marine gammarid	Naphtha (64742-73-0)	LC_{50}	96	2.6	ECB 2000a
Algae						
Pseudokirchnerie lla subcapitata	Green alga	Catalytically cracked naphtha	EC_{50} growth	72	880	ECB 2000b
			NOEL	72	0.1	ECB 2000b
Other						
Xenopus sp.	Frog	*n*-dodecane	Mortality	96	500	Buryskova et al. 2006

Definitions: EL_{50}: the loading concentration of a substance that is estimated to cause some toxic effect on 50% of the test organisms; EC_{50}: the concentration of a substance that is estimated to cause a defined effect on 50% of the test organisms; LC_{50}: the concentration of a substance that is estimated to be lethal to 50% of the test organisms; LL_{50}: the loading concentration of a substance that is estimated to be lethal to 50% of the test organisms; NOEC/L: no-observed-effect concentration/level.

Table A5.11. Modelled data for toxicity to aquatic organisms (PETROTOX 2009)[a]

Organism	**64741-42-0 Acute LL_{50}[b] (mg/L)**	**64741-69-1 Acute LL_{50} (mg/L)**	**64741-78-2 Acute LL_{50} (mg/L)**
Daphnia magna	1.29	3.58	1.94
Oncorhynchus mykiss	0.61	1.61	0.94
Pseudokirchneriella subcapitatum[c]	1.48	1.60	2.17
Rhepoxynius abronius	0.26	0.76	0.34
Palaemonetes pugio	0.50	1.40	0.77
Menidia beryllina	4.25	14.40	6.28
Neanthes arenaceodentata	2.33	7.22	3.47

[a] All results are from PETROTOX (2009) version 3.04 using the low resolution procedure. These tests used a 90:10 water to headspace ratio to allow for evaporative emissions.

[b] Median lethal loading concentration (LL_{50}) was used in place of median lethal concentration (LC_{50}) due to the insolubility of petroleum substances in water.

[c] Default particulate organic carbon (POC) concentration for algae: 2.0 mg/L.

Table A5.12. Canada Wide Standards for Petroleum Hydrocarbons in coarse-grained agricultural soils (mg/kg dw) (CCME 2008)

Exposure pathways	**F1**[a]	**F2**	**F3**	**F4**
	(C_6–C_{10})	**(> C_{10}–C_{16})**	**(> C_{16}–C_{34})**	**(> C_{34})**
Protection of groundwater for aquatic life	970	380	N/A[b]	N/A
Protection of groundwater for livestock watering	5300	14 000	N/A	N/A
Nutrient cycling	NC[c]	NC	NC	NC
Eco soil contact	210	150	300	2800
Eco soil ingestion	NC	NC	NC	NC

[a] F: fraction.

[b] N/A: not available.

[c] NC: not calculated.

Table A5.13. Total volume of soil expected to be contaminated after a spill of 2230 kg of LBPNs to various Canadian soils based on a read-across from data on gasoline

Soil Type	Retention capacity[a,b] ($mg_{gasoline}/kg_{soil}$)	Bulk density of soil[b] (g/cm^3)	Area affected by average spill of LBPNs at soil saturation (m^3)
Ottawa Sand	68 000	1.7	19.3
Delhi Loamy Sand	170 000	1.5	8.7
Elora Silt Loam	238 000	1.5	6.2

[a] After 24 hours of free drainage.

[b] From Arthurs et al. 1995

Appendix 6. Modelling results for human exposure to industry-restricted LBPNs

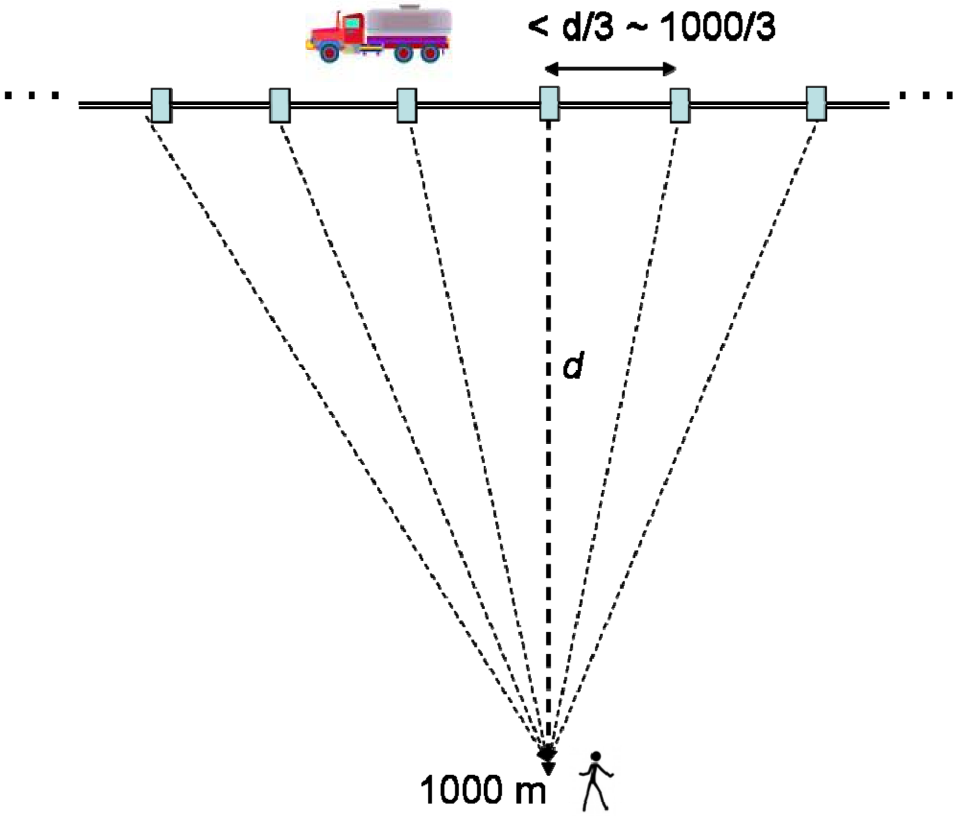

Figure A6.1. Schematic of volume sources spaced at regular intervals (blue squares) along the trajectory of motion to mimic emissions from moving truck line source. The ISC3 User's Guide (SCREEN3 ISC3 1995: p. 1–47) suggests the use of a set of volume sources spaced at regular intervals along the trajectory of motion to mimic the effect of emissions from line sources (e.g., train lines or highways). Protocols for choosing the minimum distances between the discrete volume sources are given. For example, the distance between adjacent volume sources, *d*, should be approximately one third or less of the distance between the line source and the receptor. The estimation of the rate of release of emissions from each volume source is as follows. The total emission rate (kg/h) is known. If the truck is moving at a speed of 100 km/h and the volume sources are spaced at 300-m intervals, the truck emission can be approximated as emission from 100 000 m/300 m = 333 volume sources. The emission rate for each volume source is determined by (total emission rate)/333. For a truck moving at 50 km/h, the number of volume sources is 50 000/300 = 167, and the emission rate for each source is higher than the latter case.

Table A6.1. Variable inputs to SCREEN3

Variables	**Input**				
Source type	Area	Area	Line	Line	Area
Effective emission area or speed for line source[a]	Scenarios I, IIa, IIb	Scenario III	Scenario IV	Scenario V	Scenario VI
	50×10 m^2	10×2 m^2	50 km/h	100 km/h	20×20 m^2
Emission rate (g/s·m^2)	2.0×10^{-3} [b]	2.3×10^{-4} [b]	2.3×10^{-4} [b]	2.3×10^{-4} [b]	1.46×10^{-2} [c]
Receptor height[d]	1.74 m (humans)				
Source release height[a]	3 m (I, IIa, IIb, IV, V, VI) and 1 m (III)				
Variable wind adjustment factor[e]	0.4 (from maximum 1 hour to 24 hour) 0.2 (from maximum 1 hour to annual)				
Urban–rural option	Urban (scenarios I, IIa, IIb, IV and VI) Rural (scenarios III and V)				
Meteorology[f]	1 (Full meteorology)				
Minimum and maximum distance to use	200–3000 m (scenarios I, IIa, IIb, III, IV, V) 500–3000 m (scenario VI)				

[a] Professional judgement.

[b] Emission rate from transit (g/s) is available in Table A6.2 (Appendix 6).

[c] Calculated using the formula for evaporative emission to air during loading shown after Table A6.3 (Appendix 6).

[d] Curry et al. (1993)

[e] U.S. EPA (1992).

[f] Default value in SCREEN3 (1996).

Table A6.2. Estimated regular evaporative emission of industry-restricted LBPNs to air in transit process[a]

Substance	**Estimated regular evaporative emission to air**		
	kg/year	**kg/day[b]**	**g/s**
Industry-restricted LBPNs	140[c]–30 000[d]	0.40[c]–85[d]	4.6×10^{-3}–0.98

[a] Numbers are presented as a range to cover evaporative emissions from the various transportation modes involved.

[b] 350 days/year for transportation period: The Risk Management Research Institute (RMRI 2007) summarized the industry-related shipping traffic in Placentia Bay, Newfoundland and Labrador, during 2004–2005, showing approximately 3900 transits per year from tankers, bulk cargo, tugboat and other means. For the Come By Chance refinery only, approximately over 230 tanker transits per year are related to shipping petroleum substances. Thus, it is reasonable to assume an average 350 days/year of transportation period. Information on transport frequency by trucks and trains is not available.

[c] From truck transport of each of CAS RNs 64741-42-0, 64741-69-1 and 64741-78-2.

[d] From ship transport of CAS RN 64741-42-0.

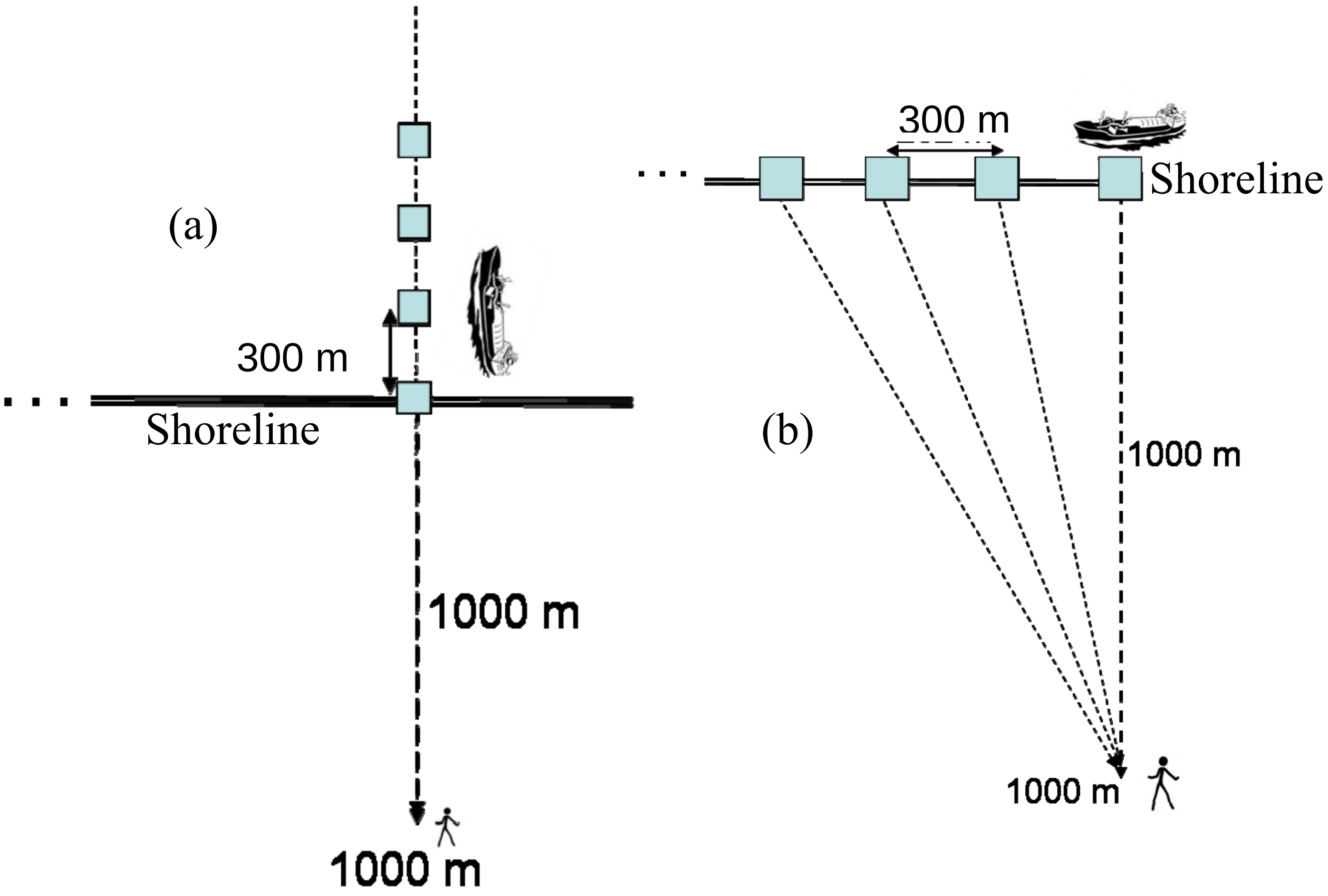

Figure A6.2. Exposure scenarios for ships leaving port. Ship motion perpendicular to the shoreline (a) and parallel to the shoreline (b) is considered. The ship is assumed to move at a speed of 10 km/h as it is leaving port. Separation of adjacent volume sources is taken as *d*/3. Exposures at *d* = 200, 500 and 1000 m from the shoreline are calculated.

Table A6.3. Estimated evaporative emissions of industry-restricted LBPNs (CAS RNs 64741-42-0, 64741-69-1, and 64741-78-2) to air

Category	**Releases to air due to evaporative emission (kg/year)**
By pipelines	Not involved[a]
By ships[b]	
Loading	40 000[c]
Transport	30 000[c]
Unloading	N/A[d]
By trucks	
Loading	1050[e]
Transport	140[e]
Unloading	1050[e]
By trains	Not involved[a]

[a] No releases expected as these LBPNs assessed in this report are not transported via this mode of transportation based on information submitted under section 71 of CEPA 1999 (Environment Canada 2009).

[b] Calculated based on a one week transit period in a port and in Canadian waters.

[c] Evaporative emission from CAS RN 64741-42-0.

[d] N/A: not applicable as they are exported beyond the jurisdiction of Canada.

[e] Evaporative emission of each CAS RN 64741-42-0, 64741-69-1, and 64741-78-2. Note that CAS RN 64741-78-2 has a high boiling point and low volatility and this value will apply only to summer months. A factor of 0.7 is used for vapour recovery during loading/unloading (U.S. EPA 2008).

A generic example of the calculation for release quantities by evaporative emission in Table A6.3 is given as follows:

For evaporative emission to air (kg per year):

$L_L = 12.46 \times S \times P \times M/T$ (Equation 1 in Chapter 5 of U.S. EPA 2008 for estimating evaporative emission from loading or unloading)

$L_T = 0.1 \times P \times W$ (Equation 5 in Chapter 5 of U.S. EPA 2008 for estimating evaporative emission during transit by ships)

$L_S = 365 \times V_V \times W_V \times K_E \times K_S$ (Equation 1-2 in Chapter 7 of U.S. EPA 2008 for estimating evaporative emission during transit by trains and trucks)

$K_S = 1/(1 + 0.053 \times P \times H_V)$ (Equation 1-20 in Chapter 7 of U.S. EPA 2008 for estimating vented vapour saturation factor)

where,
L_L = evaporative emission during loading or unloading, lb/10^3 gal
S = saturation factor, dimensionless
P = vapour pressure of the substance, psia
M = molecular weight of vapours, lb/lb-mole
T = temperature of bulk liquid loaded or unloaded, °R = 460 + °F
L_T = evaporative emission from transit by ships, lb/week-10^3 gal transported
W = density of the condensed vapours, lb/gal
L_S = standing storage loss, lb/year
V_V = vapour space volume, ft^3, based on tank size and loading volume
W_V = vapour density, lb/ft^3
K_E = vapour space expansion factor, dimensionless, 0.07
K_S = vapour saturation factor, dimension less
H_V = vapour space outage, ft, estimated as half of an effective height for a horizontal tank

Appendix 7. Summary of health effects information from pooled health effects data for LBPNs

Table A7.1. Critical health effects information on LBPNs

Endpoints	**CAS RN (or specific substance)**	**Effect levels[a]/results**
Acute health effects		**Oral exposure**
	Dripolene; Pyrolysis gasoline	**LD_{50}:** > 2000 mg/kg-bw (rat) (Rodriguez and Dalbey 1994a, b).
	68955-35-1	**LD_{50}:** 3500 mg/kg-bw (rat) (API 2008a).
		Inhalation exposure
	8 CAS RNs	**LC_{50}:** > 5 mg/L (> 5000 mg/m^3)[b] (rat) (CONCAWE 1992; API 2008a).
	8052-41-3	**LC_{50}:** > 1400 ppm (> 7936 mg/m^3)[c,d] (RTECS 2008a)
	8032-32-4	**LC_{50}:** 3400 ppm (9025 mg/m^3)[c,e] (rat) (RTECS 2008b).
		Dermal exposure
	9 CAS RNs	**LD_{50}:** > 2000 mg/kg-bw (rabbit) (CONCAWE 1992; Rodriguez and Dalbey 1994c, d; API 2008a).
	8030-30-6	**LD_{50}:** > 3000 mg/kg-bw (rabbit) (RTECS 2008c).
	Untreated naphtha	**LD_{50}:** > 3160 mg/kg-bw (rabbit) (Stubblefield et al. 1989).
Short-term and subchronic repeated-exposure health effects		**Inhalation exposure**
	64742-95-6	**LOAEC:** 500 ppm (1327 mg/m^3) for decreased growth rate. Concentrations of 0, 102, 500 or 1514 ppm (0, 271, 1327 or 4019 mg/m^3)[c,f] were administered to pregnant CD-1 mice (30 per concentration), 6 hours/day, from GD 6–15; surviving females were sacrificed on GD 18 (systemic effects of developmental health effects study described below). *≥ 1327 mg/m^3*: Significant decrease in body weight gain; one unexplained mortality. *4019 mg/m^3*: Maternal mortality (44%). Decreased percent hematocrit and mean corpuscular volume. Abnormal gait, laboured breathing, hunched posture, weakness, inadequate grooming, circling and ataxia (McKee et al. 1990). **LOAEC:** 1800 mg/m^3 for hematological changes. Concentrations of 0, 1800, 3700 or 7400 mg/m^3 were administered to rats for 13 weeks. *≥ 1800 mg/m^3*: Low-grade anemia (females). *≥ 3700 mg/m^3*: Increased liver and kidney weights (females) (Shell Research Ltd. 1980).
	64742-48-9	**LOAEC:** 800 ppm (4679 mg/m^3) for hepatic effects. Concentrations of 0, 400 or 800 ppm (0, 2339 or 4679 mg/m^3) were administered to male Wistar rats (28 per concentration), 6 hours/day, 7 days/week, for 3 weeks. *All concentrations*: Increased glutathione levels in the

Endpoints	CAS RN (or specific substance)	Effect levels[a]/results
		hemisphere (brain). Mucous membrane irritation. Increased relative kidney weight (concentration-dependent) and body weight. *4679 mg/m³*: Oxidative stress induction in the brain, kidney and liver. Reactive oxygen species increased in the liver and hippocampus, but decreased in the kidney. Decreased hepatic glutamine synthetase activity. Decreased feed consumption and increased water consumption (Lam et al. 1994).
	Gasoline[g]	**LOAEC:** 500 ppm (1327 mg/m³) for changes in brain enzyme levels. Concentrations of 0 or 500 ppm (0 or 1327 mg/m³)[c,h] were administered to male and female Sprague-Dawley rats (15 of each sex per concentration), 6 hours/day, 5 days/week, for 4 weeks. Included are 5 of each sex per concentration that were allowed 4 weeks of recovery. Increased kidney weight and hepatic ethoxyresorufin *O*-deethylase activity (males). Elevated lymphocyte counts and serum phosphate (males). Increased heart weight and glucose levels (females). Decreased hemoglobin levels (females). Altered brain biogenic amine levels (dependent on brain region and sex). Increased urinary ascorbic and hippuric acid levels. Most effects returned to control levels after recovery (Chu et al. 2005).
	8052-41-3	**LOAEC:** 363 mg/m³ for increased mortality. Concentrations of 114–1271 mg/m³ administered to Long-Evans or Sprague Dawley rats (*n* = 106), guinea pigs (*n* = 217), albino New Zealand rabbits (*n* = 20), male squirrel monkeys (*n* = 18) and male Beagle dogs (*n* = 12), continuously for 90 days. *≥ 363 mg/m³*: Mortality in guinea pigs (4/15, most susceptible). *1271 mg/m³*: Congested lungs, bronchitis and mixed inflammatory cell infiltration in the lungs of all species (Rector et al. 1966). **Lowest inhalation LOAEC:** 214 mg/m³ for an inflammatory response of the respiratory tract. Concentrations of 0 or 214 mg/m³ were administered to female CD-1 rats (6 per concentration) by head-only exposure, 4 hours/day for 4 consecutive days. *214 mg/m³*: Inflammatory cell infiltrate in nasal cavity, trachea and larynx; loss of cilia, hyperplasia of basal cells and squamous metaplasia of trachea and nasal cavity (Riley et al. 1984). **LOAEC:** 575 mg/m³ for biochemical changes. Concentrations of 0, 575, 2875 or 5750 mg/m³ were administered to male Wistar rats (20 per concentration), 6 hours/day, 5 days/week, for 4, 8, 12 or 17 weeks.

Endpoints	CAS RN (or specific substance)	Effect levels[a]/results
		≥ *575 mg/m³*: Decreased serum creatine kinase at 17 weeks. Decreased cerebellar succinate dehydrogenase activity from weeks 8–17 (concentration-dependent). ≥ *2875 mg/m³*: Changes in cerebellar glutathione levels and creatine kinase activity. Muscle membrane effects were suggested, as muscle membrane sialic and uronic acid residue levels were decreased (Savolainen and Pfaffli 1982).
		Oral exposure
	64742-95-6	**Lowest oral LOAEL:** 500 mg/kg-bw per day for biochemical changes (both sexes) and decreased growth rate (males). Doses of 500, 750 or 1250 mg/kg-bw per day were administered to male and female rats (10 of each sex per dose) for 3 months. ≥ *500 mg/kg-bw per day*: Decreased body weight (males). Dose-related increases in liver and kidney weights and relative weights, as well as increased serum glutamic pyruvic transaminase (males and females). *1250 mg/kg-bw per day*: Increased alkaline phosphatase (males) (Bio/Dynamics, Inc. 1991a). **Lowest oral LOAEL:** 500 mg/kg-bw per day for hematological changes. Doses of 125, 250 or 500 mg/kg-bw per day were administered to male and female Beagle dogs (4 of each sex per dose), 7 days/week for 90 days. *500 mg/kg-bw per day*: Borderline anemia (Bio/Dynamics, Inc. 1991b).
		Dermal exposure
	64741-54-4	**LOAEL:** 200 mg/kg-bw for decreased growth rate. Doses of 200, 1000 or 2000 mg/kg-bw were applied to the shaven skin of male and female rabbits, 3 times/week for 28 days (12 applications total). *200 mg/kg-bw*: Slight to moderate and slight skin irritation in males and females, respectively; reduced growth rate (males). *1000 mg/kg-bw*: Moderate skin irritation; reduced growth rate (male and female). *2000 mg/kg-bw*: Moderate skin irritation; weight loss (females), before reduced growth weight (males) (API 1986g).
	64742-48-9	**LOAEL:** 500 mg/kg-bw per day for hematological changes (males) and 1500 mg/kg-bw per day for biochemical changes (males and females). Doses of 0, 500, 1000 or 1500 mg/kg-bw per day were administered to male and female F344 rats (10 of each sex per group), 6 hours/day, 5 days/week, for 4 weeks. *500 mg/kg-bw per day*: Dose-dependent increase in white blood cells (due to increase in neutrophils and

Endpoints	CAS RN (or specific substance)	Effect levels[a]/results
		lymphocytes) in males. *1000 mg/kg-bw per day*: Significant decrease in feed consumption (females). *1500 mg/kg-bw per day*: Severe erythema, moderate eschar formation, dose-dependent increase in white blood cells (due to increase in neutrophils and lymphocytes) in females, significant decrease in feed consumption (males), mild anemia, decreased serum albumin (9–25%), total serum protein (10–13%) and blood urea nitrogen (9–25%) and increased platelet counts (10–20%) (Zellers 1985).
	64741-55-5	**Lowest dermal LOAEL:** 30 mg/kg-bw per day for skin irritation. Doses of 0, 30, 125 or 3000 mg/kg-bw per day were applied to the clipped backs of male and female Sprague-Dawley rats (15 of each sex per dose), 5 days/week for 90 days. *All doses*: Dose-related increase in skin irritation, erythema and edema at treated sites and histopathological correlates of hyperplasia, inflammation and ulceration. No other effects reported (Mobil 1988a).
	68955-35-1	**LOAEL:** 1000 mg/kg-bw per day for increased mortality. Doses of 200, 1000 or 2000 mg/kg-bw per day applied to shaven skin of male and female rabbits, 3 times/week for 28 days (12 applications total). *200 mg/kg-bw per day*: Moderate skin irritation. *1000 mg/kg-bw per day*: Moderate skin irritation; mortality in 1/5 males. *2000 mg/kg-bw per day*: Severe skin irritation; decreased body weight gain and body weight; mortality in 2/5 males with tubular degeneration; granulopoiesis of bone marrow (API 1986h).
Chronic repeated-exposure health effects (non-cancer)	Gasoline[g]	**Lowest inhalation LOAEC:** 67 ppm (200 mg/m^3). Male and female B6C3F1 mice and Fischer 344 albino rats (approximately 6 weeks of age; 100 mice or rats of each sex per group) exposed to 0, 67, 292 or 2056 ppm (0, 200, 870 or 6170 mg/m^3, as cited in IARC 1989b) of the test substance (containing 2% benzene) via inhalation, 6 hours/day, 5 days/week, for 103–113 weeks. *All concentrations*: Ocular discharge and irritation (rats). *870 mg/m^3*: Increased relative kidney weight (male rats). *6170 mg/m^3*: Increased absolute and relative kidney weights (male rats) and increased relative kidney weight (female rats). Decreased body weight (rats and male mice). Decreased absolute heart weight (rats) (MacFarland et al. 1984).
	8030-30-6	**Lowest dermal LOAEL:** 25 mg (neat) (694 mg/kg-bw).

Endpoints	**CAS RN (or specific substance)**	**Effect levels[a]/results**
		Male and female C3H/HeN mice (25 of each sex) exposed to 25 mg (694 mg/kg-bw)[i,j] of the test substance (neat), applied to the shaved skin of the dorsal thoracic region, 3 times/week for 105 weeks. Dermal irritation after 10–15 days. Inflammatory and degenerative skin changes after 6 months (Clark et al. 1988).
Reproductive and developmental health effects		**Inhalation exposure**
	64742-48-9	**LOAEC:** 800 ppm (4679 mg/m^3) for reproductive and developmental toxicity and developmental neurotoxicity. Pregnant Wistar rats exposed to 800 ppm (4679 mg/m^3)[c,k] of the test substance, via inhalation, 6 hours/day from GD 7–20. *4679 mg/m^3*: Decreased number of pups per litter and higher frequency of post-implantation loss. Increased birth weight of pups. *4679 mg/m^3*: Decreased motor activity (non-significant). No effect observed for neuromotor activity. For learning ability, exposed rats showed behaviour comparable to that of controls at 1 month of age. At 2 months of age, impaired cognitive function (females) and impaired memory (males) were observed. At 5 months of age, learning and memory deficits were observed in both sexes (Hass et al. 2001).
	64741-63-5	**Highest NOAEC:** 7480 ppm (27 687 mg/m^3) for developmental and reproductive toxicity. Female Sprague-Dawley rats (10 per concentration) exposed to 0, 750, 2490 or 7480 ppm (0, 2776, 9217 or 27 687 mg/m^3) of the test substance via inhalation, 6 hours/day, 7 days/week, from 2 weeks before mating through to GD 19; and male Sprague-Dawley rats (10 per concentration) exposed to same concentrations, 6 hours/day, 7 days/week, from 2 weeks before mating for 46 consecutive days. Rats sacrificed on postnatal day 4. *All concentrations*: No effect on reproductive organs (testes, epididymides, ovaries), reproductive performance or fetal development (Schreiner et al. 2000b; API 2008a).
		Oral exposure
	64742-95-6	**LOAEL:** 1250 mg/kg-bw per day for developmental toxicity. Pregnant Sprague-Dawley CD rats (24 per dose) exposed to 0, 125, 625 or 1250 mg/kg-bw per day of the test substance, via gavage, from GD 6–15. Rats sacrificed on GD 20. *1250 mg/kg-bw per day*: Reduced fetal body weight and increased incidence of ossification variations. Retardation in ossification of vertebral elements and sternebrae (Bio/Dynamics, Inc. 1991c).
	64741-55-5	**NOAEL:** 2000 mg/kg-bw for reproductive toxicity and teratogenicity. Pregnant Sprague-Dawley rats exposed to

Endpoints	CAS RN (or specific substance)	Effect levels[a]/results
		2000 mg/kg-bw of the test substance, via oral exposure, on GD 13 (other refinery streams also tested in separate experiments) to identify and compare any potential direct teratogenic effects that might be obscured by maternal or fetal toxicity resulting from repetitive exposure. Moderate to severe toxicity observed in the first rats treated (although none perished, fetal viability may have been compromised); thus, the test group was limited to five animals. Caesarean sections performed on GD 20 (Stonybrook Laboratories 1995).
		Dermal exposure
	68513-02-0	**Highest NOAEL:** 1000 mg/kg-bw per day for reproductive and developmental toxicity. Pregnant Sprague-Dawley rats (12 per dose, 15 for control) exposed to 0, 100, 500 or 1000 mg/kg-bw per day of the test substance (neat), applied to the shaved skin of the back (not occluded), from GD 0–20. Observation until lactation day 4. Reproductive and developmental effects examined include number of females delivering live litters, gestation length, number of implantation sites, number of litters with live pups, offspring survival at lactation days 0–4, pup sex ratio and pup body weight (ARCO 1994).
	8030-30-6	**NOAEL:** 25 mg (694 mg/kg-bw per day) for reproductive toxicity. Male and female C3H/HeN mice (25 of each sex) exposed to 25 mg (694 mg/kg-bw per day)[i,j] of the test substance (neat), applied to the shaved skin of the dorsal thoracic region, 3 times/week for 105 weeks. No effects observed in gonads (Clark et al. 1988).
Carcinogenicity		**Dermal exposure (*chronic*)**
	8030-30-6	**Lowest dermal effect level:** 25 mg (694 mg/kg-bw per day). Male and female C3H/HeN mice (42–50 days of age, 25 of each sex) were exposed to 25 mg (694 mg/kg-bw per day)[i,j] of the test substance (neat) applied to the shaved skin of the dorsal thoracic region, 3 times/week for up to 105 weeks. Increased incidence of skin tumours (21%). Tumour incidence: 10/47 in test group (3 squamous cell carcinomas and 7 fibrosarcomas); 0/46 in the negative control group; 49/49 in the positive control group (49 squamous cell carcinomas). Tumours appeared after 94 weeks in the test group and 28 weeks in the positive control group (Clark et al. 1988).
	64741-46-4	**Highest dermal effect level:** 50 mg (1351 mg/kg-bw per day). 50 male C3H/HeJ mice (6–8 weeks of age) were exposed to 50 mg (1351 mg/kg-bw per day)[i,j] of the test substance (neat) applied to the shaved skin of the interscapular region of the back, 2 times/week, until a papilloma > 1 mm^3 appeared. Increased incidence of skin

Endpoints	CAS RN (or specific substance)	Effect levels[a]/results
		tumours. Tumour incidence: 11/44 in the test group; 0/50 in the negative control group; 46/48 in the positive control group. Tumours appeared after 85 weeks in the test group and after 46 weeks in the positive control group (Blackburn et al. 1986).
		Dermal exposure (*initiation/promotion*)
	64741-87-3	**Initiation:** 30 male CD-1 mice (7–9 weeks of age) administered 50 µL (917 mg/kg-bw per day)[j,l,m] of the test substance (neat) for 5 consecutive days. After a 2-week rest period, 50 µL of the promoter PMA was administered 2 times/week for 25 weeks. Both substances applied to the shaved dorsal intrascapular skin. Insignificant increase in skin tumours. Tumour incidence: 3/29 in the test group (squamous cell papillomas); 3/30 in the negative control group; 30/30 in the positive control group. Tumours appeared after 20 weeks in the test group and 16 weeks in the negative control group. **Promotion:** 30 male CD-1 mice (7–9 weeks of age) administered 50 µL of DMBA as a single dose. After a 2-week rest period, 50 µL (917 mg/kg-bw per day)[j,l,m] of the test substance was administered, 2 times/week for 25 weeks. Both substances applied to the shaved dorsal intrascapular skin. No increase in skin tumours. Tumour incidence: 0% in the test and negative control groups; 30/30 in the positive control group (Skisak et al. 1994).
		Inhalation exposure (*chronic*)
	Gasoline[g]	0, 67, 292 or 2056 ppm (0, 200, 870 or 6170 mg/m^3, as cited in IARC 1989b) of the test substance (containing 2% benzene content) administered to male and female B6C3F1 mice and Fischer 344 albino rats (approximately 6 weeks of age, 100 animals of each sex per group), via inhalation, 6 hours/day, 5 days/week, for 103–113 weeks. Increased incidence of hepatocellular tumours (adenomas and carcinomas) in female mice (14%, 19%, 21% and 48%, respectively; final group was statistically significantly different from controls). Increased incidence of renal tumours in female mice (2/100 at the highest concentration). Concentration-related increased incidence of primary renal neoplasms in male rats (*n* = 0, 1, 5 and 7, respectively). Appearance of tumours not considered statistically significant in male mice and female rats, and renal tumours in male rats are not considered relevant to humans (MacFarland et al. 1984). 0, 10, 69 or 298 ppm (0, 27, 183 or 791 mg/m^3)[c,h] of the test substance (PS-6 blend) administered to F344 rats (31

Endpoints	CAS RN (or specific substance)	Effect levels[a]/results
		animals of each sex per group) or to a positive control (50 ppm TMP), via inhalation, 6 hours/day, 5 days/week, until sacrifice at 65–67 weeks. Appropriate controls present. No significant increase in number of animals with atypical cell foci for any exposure group. No animals with renal cell tumours observed (part of the initiation/promotion study mentioned below) (Short et al. 1989).
		Inhalation exposure (*initiation/promotion*)
	Gasoline[g]	**Extended promotion:** Male and female F344 rats (8–9 weeks of age, 30 animals of each sex per group) administered EHEN at 170 mg/L in the drinking water for 2 weeks. After a 4-week rest period, 10, 69 or 298 ppm (27, 183 or 791 mg/m^3)[c,h] of the test substance (PS-6 blend) or a positive control (50 ppm TMP) was administered, via inhalation, 6 hours/day, 5 days/week, until sacrifice at 65–67 weeks. Appropriate controls present. In males, significant linear trend in number of animals with atypical cell foci, however no significant increase in number of animals with renal cell tumours observed for any exposure group (1, 0, 1 and 2 animals developed tumours, respectively). In females, no significant increase in number of animals with atypical cell foci or renal cell tumours observed for any exposure group (1, 0, 2 and 2 animals developed tumours, respectively) (Short et al. 1989). **Promotion:** 36 female B6C3F1 mice (12 days of age, 12 animals per concentration) administered DEN at 5 mg/kg-bw, via intraperitoneal injection. At 5–7 weeks of age, mice then exposed to the test substance (PS-6 blend), via inhalation, at concentrations of 0, 283 or 2038 ppm (0, 751 or 5410 mg/m^3)[c,h], 6 hours/day, 5 days/week, for 16 weeks. Alternatively, the test substance was administered to initiated mice at 2038 ppm (5410 mg/m^3) in addition to 1 mg/kg of EE2 in the diet. Significant increases in focal size and volume fraction of altered hepatic foci, as well as the incidence of macroscopic hepatic neoplasms, observed in mice exposed to 2038 ppm of the test substance alone and also for co-exposure to EE2 (10.3-fold and 60-fold increases in tumour incidence, respectively, over control group) (Standeven et al. 1994).

Endpoints	**CAS RN (or specific substance)**	**Effect levels[a]/results**
Genotoxicity (*in vivo*)	Gasoline[g]	**Lowest oral LOAEL:** 135 mg/kg-bw per day. **Positive for RDS:** Male and female Fischer 344 rats (3 of each sex per group) exposed to 200 mg/kg-bw per day (for 4 days) or 135 mg/kg-bw per day (for 18 days) of the test substance (PS-6 containing 2% benzene), via oral gavage. Induction of RDS in kidney cells after 4 and 18 days (males only; changes in females not statistically significant) (Loury et al. 1987). **Highest oral NOAEL:** 5000 mg/kg-bw per day. **Negative for UDS:** Male Fischer 344 rats (3 per group) exposed to 2000 mg/kg-bw (cells isolated 2 or 12 hours after exposure), 5000 mg/kg-bw (cells isolated 12 or 24 hours after exposure) or 5000 mg/kg-bw per day (for 1–4 days) of the test substance (PS-6), via oral gavage. No induction of UDS in kidney cells (Loury et al. 1987). **Inhalation LOAEC:** 2000 ppm (5309 mg/m^3). **Positive for RDS:** Male and female Fischer 344 rats (3 of each sex per group) exposed to 2000 ppm (5309 mg/m^3)[c,h] of the test substance (PS-6 containing 2% benzene), via inhalation, 6 hours/day for 4 and 18 days (male) or 18 days (female). Induction of RDS in kidney cells after 18 days (males only; changes in females not statistically significant) (Loury et al. 1987).
	64741-55-5	**Intraperitoneal injection LOAEL:** 200 mg/kg-bw.

Endpoints	CAS RN (or specific substance)	Effect levels[a]/results
		Positive for sister chromatid exchange: Male and female mice (5 of each sex per group) were administered 200, 1200 or 2400 mg/kg-bw of the test substance (API 81-03), as a single dose, via intraperitoneal injection. Pairwise comparisons, by sex, of sister chromatid exchanges in bone marrow cells from each treatment group with its vehicle control were significantly different. *Reviewers note that although interaction between the test substance and DNA was demonstrated, it was not considered definitive for clastogenic activity since no genetic material was unbalanced or lost (API 1988a).
	8052-41-3	**Highest inhalation NOAEC:** 5 g/m^3 (50 000 mg/m^3). **Negative for micronuclei induction:** Four male BALB/c mice exposed to 50 g/m^3 (50 000 mg/m^3) of white spirit, via inhalation, for five periods of 5 min, spaced by 5 min intervals. No induction of micronuclei in the polychromatic erythrocytes from bone marrow cells in mice (Gochet et al. 1984). **Highest intraperitoneal injection NOAEL:** 0.1 mL (3710 mg/kg-bw). **Negative for micronuclei induction:** Male and female BALB/c mice (5 of each sex per group) administered 0.01, 0.05 or 0.1 mL (371, 1855 or 3710 mg/kg-bw)[j,l,n] of white spirit, as a single dose, via intraperitoneal injection (sacrificed after 30 h). No induction of micronuclei in the polychromatic erythrocytes from bone marrow cells in mice (Gochet et al. 1984).
Genotoxicity (*in vitro*)[o]	64741-46-4	**Negative for mutagenicity (reverse mutations):** *Salmonella typhimurium* TA98 exposed to DMSO extracts of the test substance at concentrations of 0–50 µl/plate, with and without exogenous metabolic activation, using a modified Ames assay (Blackburn et al. 1986). **Positive for mutagenicity (reverse mutations):** *Salmonella typhimurium* (strains not identified) exposed to extracts of the test substance (concentrations not identified), with and without exogenous metabolic activation, using a modified Ames assay. Data analysis conducted using non-linear regression (Blackburn et al. 1988).
	64741-55-5	**Negative for mutagenicity (forward mutations):** L5178Y TK+/− mouse lymphoma cells exposed to test substance (API 83-20) at concentrations of 0.05–0.15 µl/mL without exogenous metabolic activation (S9) and 0.2–0.3 µl/mL with S9 (API 1987).
	64741-54-4	**Positive for mutagenicity (forward mutations):** L5178Y TK+/− mouse lymphoma cells exposed to test substance

Endpoints	CAS RN (or specific substance)	Effect levels[a]/results
		(API 83-18). Details of study not provided (API 1986l).
	68410-97-9	**Negative for mutagenicity (reverse mutations):** *Salmonella typhimurium* TA98, TA100, TA1535 and TA1537 and *Escherichia coli* WP2(uvrA) were exposed to the test substance (hydrogenated pyrolysis gasoline) at concentrations of 0, 33, 100, 333, 1000, 3333 or 10 000 µg/plate (3 plates per concentration ± S9), with and without exogenous metabolic activation (male Sprague-Dawley rat liver S9), using the Ames assay (Riccio and Stewart 1991). **Negative for UDS:** Primary rat hepatocyte cultures derived from male Fischer 344 rats (10 weeks old) exposed to the test substance (hydrogenated pyrolysis gasoline) at concentrations of 8, 16, 32, 64, 128, 256, 512 or 1024 µg/mL for 18 hours, without exogenous metabolic activation. Toxicity observed at 512 and 1024 µg/mL (insufficient cells for UDS analysis); UDS not evident at lower concentrations (Brecher 1984a). **Positive for cell transformation:** BALB/3T3-A31-1-1 mouse embryo cells exposed to the test substance at concentrations of 100, 250, 500 or 1500 µg/mL (15 cultures per concentration) for 2 days, without exogenous metabolic activation (S9). Toxicity observed at all concentrations (cloning efficiencies of 53.7% at 100 µg/mL to 0% at 1500 µg/mL). Transformation observed at 1500 µg/mL (frequency of 0.36) (Brecher 1984b).
	64742-48-9	**Negative for cell transformation:** BALB/3T3-A31-1-1 mouse embryo cells exposed to the test substance at concentrations of 16, 32, 64 or 200 µg/mL (15 cultures per concentration) for 2 days, without exogenous metabolic activation (S9). Toxicity observed at ≥ 32 µg/mL (cloning efficiencies of 67.2% at 32 µg/mL to 28.8% at 200 µg/mL) (Brecher and Goode 1984b).
	8052-41-3	**Negative for sister chromatid exchange:** Lymphocytes derived from 1 human (male; 2 cultures per concentration) were exposed to the test substance (white spirit) at ratios of 1:1, 1:2, 1:4 and 1:8 for 1 and 24 hours (Gochet et al. 1984).
	Gasoline[g]	**Positive for UDS:** Hepatocytes derived from 3 male Fischer 344 rats, 2 male B6C3F1 mice and 1 human were exposed to the test substance (PS-6 containing 2% benzene) at concentrations of 0.01–0.33% by volume (rats) and 0.01–0.05% by volume (mice and humans). Maximum induction of UDS occurred at 0.10% by volume for rats (concentration-dependent) (cytotoxicity occurred at higher concentrations). Induction of UDS occurred at 0.01% by

Endpoints	CAS RN (or specific substance)	Effect levels[a]/results
		volume for mice and humans (cytotoxicity occurred at higher concentrations; thus, a concentration-response trend could not be established) (Loury et al. 1986).
Skin irritation	64741-55-5	Primary irritation index: 1.7/8.0 (Draize 24-hour occluded patch test in rabbit skin); moderate skin irritant in rabbits (API 1986b).
	64741-54-4	Primary irritation index: 6.9/8.0 (Draize 24-hour occluded patch test in rabbit skin) (API 1986d).
	64741-63-5	Primary irritation index: 2.0/8.0 (Draize 24-hour occluded patch test in rabbit skin) (API 1985c).
	64741-68-0	Primary irritation index: 5.4/8.0 (Draize 24-hour occluded patch test in rabbit skin) (API 1985b).
	68955-35-1	Primary irritation index: 3.1/8.0 (Draize 24-hour occluded patch test in rabbit skin); moderate skin irritant in rabbits (API 1985a).
	64741-87-3	Primary irritation index: 1.2/8.0 (Draize 24-hour occluded patch test in rabbit skin); mild skin irritant in rabbits (API 1986c).
	64741-66-8	Primary irritation index: 3.9/8.0 (Draize 24-hour occluded patch test in rabbit skin); moderate skin irritant in rabbits (API 1986a).
	Gasoline[g]	Primary irritation index: 0.98/8.0 (Draize 24-hour occluded patch test in rabbit skin); mild skin irritant in rabbits (API 1980a). Unleaded gasoline with or without 3% methanol slightly irritating to rabbit skin in 4-hour semi-occluded patch test (CONCAWE 1992).
	Dripolene; Pyrolysis gasoline	Non-corrosive after 1- and 4-hour occlusions and 48 hours post-dose and non-irritant (Draize method) in New Zealand White rabbits (three of each sex) when 0.5 mL of test substance applied (Rodriguez and Dalbey 1994e, f).
Eye irritation Draize test (rabbit)	64741-55-5	Slight (API 1986b); non-irritant (API 1986b).
	64741-54-4	Slight (API 1986d).
	64741-63-5	Slight (API 1985c).
	64741-68-0	Slight (API 1985b).
	68955-35-1	Slight (API 1985a); irritant within 1 hour of instillation, gradually resolved over 7 days and not apparent at 14 days (API 1985a).
	64741-87-3	Slight (CONCAWE 1992); non-irritant (API 1986c, 2008a).
	64741-66-8	Non-irritant (API 1986a).
	Gasoline[g]	Non-irritant (API 1980a).
	Dripolene; Pyrolysis gasoline	Irritant in New Zealand White rabbits (3 of each sex), 0.1 mL of test substance administered to conjunctival sac of left eye; 4/6 rabbits had corneal ulceration, conjunctival

Endpoints	**CAS RN (or specific substance)**	**Effect levels[a]/results**
		redness and swelling, and 2 of these 4 rabbits had corneal opacity and iritis (Rodriguez and Dalbey 1994f, g, h, i).
Sensitization[p] Closed patch technique (guinea pigs)	64741-55-5	Negative (API 1986b).
	64741-54-4	Negative (API 1986d).
	64741-63-5	Negative (API 1986f).
	64741-68-0	Negative (API 1985b).
	68955-35-1	Negative (API 1986e).
	64741-87-3	Negative (API 1986c).
	64741-66-8	Negative (API 1986a).
	Gasoline[g]	Negative (applied as 50% dilution in mineral oil to reduce irritancy) (API 1980a).

Abbreviations: bw, body weight; DEN, *N*-nitrosodiethylamine; DMBA, 7,12-dimethylbenzanthracene; DNA, deoxyribonucleic acid; EE2, ethinyl estradiol; EHEN, *N*-ethyl-*N*-hydroxyethylnitrosamine; GD, gestation day; PMA, phorbol-12-myristate-13-acetate; RDS, replicative DNA synthesis; TMP, 2,2,4-trimethylpentane; UDS, unscheduled DNA synthesis.

[a] LC_{50}, median lethal concentration; LD_{50}, median lethal dose; LOAEC, lowest-observed-adverse-effect concentration; LOAEL, lowest-observed-adverse-effect level; NOAEC, no-observed-adverse-effect concentration; NOAEL, no-observed-adverse-effect level.

[b] 1 m^3 = 1000 L.

[c] The following formula was used for conversion of provided values into mg/m^3: (x ppm × MM)/24.45.

[d] Molar mass (MM) of CAS RN 8052-41-3 reported to be 138.6 g/mol (Carpenter et al. 1975).

[e] The MM of CAS RN 8032-32-4 was not available; therefore, a MM of 64.9 g/mol (gasoline) was used (Roberts et al. 2001).

[f] The MM of CAS RN 64742-95-6 was not available; therefore, a MM of 64.9 g/mol (gasoline) was used (Roberts et al. 2001).

[g] Gasoline captures the following CAS RNs: 8006-61-9 and 86290-81-5.

[h] MM of gasoline reported to be 64.9 g/mol (Roberts et al. 2001).

[i] The following formula was used for conversion of provided values into mg/kg bw: x mg/kg bw.

[j] Body weight not provided; thus, laboratory standards from Salem and Katz (2006) were used.

[k] MM of CAS RN 64742-48-9 reported to be 143 g/mol (Hass et al. 2001).

[l] The following formula was used for conversion of provided values into mg/kg bw: x mL/kg bw × ρ.

[m] Density (ρ) of CAS RN 64741-87-3 reported to be 678.2 mg/mL (API 2003d).

[n] Density (ρ) of CAS RN 8052-41-3 reported to be 779 mg/mL (Gochet et al. 1984).

[o] Negative result studies described in table correspond to studies with the highest dose/concentration used.

[p] Poor response in positive control noted.