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Screening Assessment
Petroleum Sector Stream Approach

Asphalt and
Oxidized Asphalt

Chemical Abstracts Service Registry Numbers

8052-42-4

64742-93-4

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Synopsis

Pursuant to section 74 of the *Canadian Environmental Protection Act, 1999* (CEPA), the Minister of the Environment and the Minister of Health have conducted a screening assessment of two substances known as asphalt and oxidized asphalt.

CAS RN[a]	***Domestic Substances List*[b] name**
8052-42-4	Asphalt
64742-93-4	Asphalt, oxidized

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[b] Domestic Substances List.

Asphalt (CAS RN 8052-42-4) and oxidized asphalt (CAS RN 64742-93-4) were identified as priorities for assessment as they met the categorization criteria under subsection 73(1) of CEPA. 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).

Asphalt and oxidized asphalt are complex combinations of high molecular weight organic compounds containing a relatively high proportion of hydrocarbons; carbon (C) numbers are predominantly greater than C_{25} with high carbon-to-hydrogen ratios. They are residual substances derived from the high temperature vacuum distillation of petroleum. The composition and physical-chemical properties of asphalt and oxidized asphalt vary depending on the source(s) of crude oil, processing steps and blending stocks involved in their production.

Asphalt and oxidized asphalt are used primarily (approximately 99%) in the construction of roads and in roofing materials, but they may be found in adhesives and sealants, paints and coatings, and other miscellaneous products available to consumers. Other residual substances in the asphalt group with different CAS RNs might also be added into asphalt formulations if they meet the material specifications. As data on asphalt formulations (final formulated products) generally do not specify a CAS RN, and all residuals are expected to behave similarly when used in these products, the results of the analyses herein on these asphalt formulations for the purpose of assessing asphalt and oxidized asphalt may also apply to other substances in the asphalt group. For the purpose of this assessment the term “asphalts” is used to refer to different grades and types of asphalt (CAS RNs 8052-42-4) and oxidized asphalts (CAS RN 64742-93-4).

Release of hazardous components of asphalts to the environment is considered to be low based on experimental studies on polycyclic aromatic hydrocarbons (PAHs) leaching from paving and roofing asphalt formulations. In addition, asphalts can stabilize

and entrap contaminants or other petroleum substances mixed with asphalts, making them unavailable to organisms or the environment.

Based on physical-chemical properties such as high K_{OW}, high K_{OC}, very low vapour pressure and very low water solubility under environment-relevant conditions, asphalt components are not expected to disperse in the environment or to be bioavailable. Modelled ecotoxicity data, and results from ecological toxicity studies on bean and corn seeds exposed to oxidized asphalt indicate low toxicity. Use of hot-mix asphalt as lining for fish ponds, and low aquatic toxicity of runoff from unsealed asphalt pavement also indicate that the bioavailability and ecotoxicity of asphalts is very low. Although spills of asphalt materials are reported, asphalts are not likely to disperse in the environment because of their low mobility. The lack of bioavailability and low toxicity of asphalts also indicate that the ecological concern of spilled asphalt materials is considered to be low.

Considering all available lines of evidence presented in this screening assessment, there is a low risk of harm to organisms or the broader integrity of the environment from these substances. It is concluded that asphalt and oxidized asphalt (CAS RNs 8052-42-4 and 64742-93-4) do not meet the criteria under paragraphs 64(*a*) or (*b*) of CEPA 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 health effect for the initial categorization of asphalt and oxidized asphalt was carcinogenicity, based on classifications by international agencies. PAHs and benzene, minor components of asphalts, have been identified by Health Canada and several international regulatory agencies as carcinogens. PAHs (as represented by benzo[a]pyrene equivalents) and benzene were therefore selected as high-hazard components of asphalts for use in characterizing long-term exposure and risk to the general population in the vicinity of asphalt production and storage facilities. Naphthalene, a volatile PAH, was selected to characterize shorter term exposures to bystanders.

The potential for exposure of the general population to asphalts includes consideration of inhalation exposure in the vicinity of either asphalt-producing and refining industries or near paving operations. Dermal exposure can occur from use of consumer products, including as a result of application of asphalt-based pavement sealant in residential settings. Long-term oral exposure to components of asphalts (i.e., PAHs) can occur as components of asphalts may migrate from asphalt surfaces indoors and are found in house dust. Risks to human health were characterized for these potential exposures.

For short-term inhalation exposure in the vicinity of paving operations, a margin of exposure based on an a conservative exposure of a bystander to naphthalene, and the effect level in exposed animals causing a local effect on nasal olfactory epithelium, was considered adequate. Short-term dermal exposure to asphalts from asphalt-containing products is not considered to constitute a risk to human health due to a lack of toxicity

exhibited by asphalt in short-term animal studies, and due to low dermal absorption of asphalts.

For potential inhalation exposures to asphalts from fugitive and point releases from the production and refinement of asphalt in the vicinity of facilities, margins of exposure between conservative estimates of exposures to benzene and to PAHs, and estimates of cancer potency previously developed for inhalation exposure to benzene and to PAHs, are considered adequate to address uncertainties in health effects and exposure databases.

For potential long-term oral exposure to components of asphalts that may migrate indoors over time from asphalt driveway sealcoat (where exposure is through ingestion of house dust that contains asphalt-derived PAHs), margins of exposure between oral exposure to PAHs and a conservative point of departure for benzo[a]pyrene are considered adequate to address uncertainties in health effects and exposure databases.

Therefore, general population exposure to asphalt and oxidized asphalt is not considered to constitute a risk to human health.

Based on the information presented in this screening assessment, it is concluded that asphalt and oxidized asphalt (CAS RNs 8052-42-4 and 64742-93-4) do not meet the criteria under paragraph 64(*c*) of CEPA 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 concluded that asphalt and oxidized asphalt (CAS RNs 8052-42-4 and 64742-93-4) do not meet any of the criteria set out in section 64 of CEPA.

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Tables and Figures

1 Introduction

Pursuant to section74 of the *Canadian Environmental Protection Act, 1999* (CEPA) (Canada 1999), the Minister of the Environment and the Minister of Health conduct screening assessments of substances to determine whether these substances present or may present a risk to the environment or to human health.

A key element of the Government of Canada's Chemicals Management Plan (CMP) is the Petroleum Sector Stream Approach (PSSA), which involves the assessment of approximately 160 petroleum substances that are considered 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).

These petroleum substances fall into nine groups of substances based on similarities in production, toxicity and physical–chemical properties (see Table A-1 in Appendix A). In order to conduct the screening assessments, each 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;[1]
> Stream 2: industry-restricted substances, which are substances that may leave a petroleum sector facility and may be transported to other industrial facilities (for example, for use as a feedstock, fuel or blending component), but that 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 67 of these petroleum substances may be present in consumer products under Stream 4, as described above. These 67 substances were further sub-grouped as follows, based on their physical and chemical properties and potential uses: aromatic extracts, gas oils, heavy fuel oils (HFOs), low boiling point naphthas, natural gas condensates, solvents, petroleum and refinery gases (including liquefied petroleum gases), base oils, petrolatum and waxes, and asphalts.

[1]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.

This screening assessment addresses asphalt and oxidized asphalt described under Chemical Abstracts Service Registry Number (CAS RN) 8052-42-4 and CAS RN 64742-93-4, respectively. Asphalt and oxidized asphalt were identified as priorities for assessment as they met the categorization criteria under subsection 73(1) of CEPA (ECCC, HC 2007). Asphalt and oxidized asphalt were included in the PSSA because they are related to the petroleum sector and are complex combinations of hydrocarbons.

Included in this screening assessment is the consideration of information on chemical properties, health effects, uses, and exposure, including information submitted under section 71 of CEPA. Data relevant to the screening assessment of these substances were identified in original literature, review and assessment documents, and in stakeholder research reports, submissions and correspondence, as well as from recent literature searches, up to June 2015. Key studies, which were critically evaluated, and modelling results were used to inform conclusions.

This screening assessment was prepared by staff in the CEPA Risk Assessment Programs at Health Canada and Environment and Climate Change Canada and incorporates input from other programs within these departments. The ecological and human health portions of this assessment have undergone external review and/or consultation. Comments on the technical portions relevant to the environment were received from Dr. Lawrence Kapustka, Dr. Gladys Stephenson and Mr. Geoff Granville, while those relevant to human health were received from Dr. Simon Hesp (Queen's University, Kingston, ON) and Dr. E. Spencer Williams (Baylor University, Waco, TX). Additionally, the draft of this Screening Assessment was subject to a 60-day public comment period. While external comments were taken into consideration, the final content and outcome of the screening assessment remain the responsibility of Health Canada and Environment and Climate Change Canada.

This screening assessment focuses on information critical to determining whether a substance meets the criteria as set out in section 64 of CEPA, by examining scientific information and incorporating a weight–of-evidence approach and precaution.[1] The screening assessment presents the critical information and considerations on which the conclusion is based.

[1] A determination of whether one or more of the criteria of section 64 of CEPA are met is based upon an assessment of potential risks to the environment and/or 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 products used by consumers. A conclusion under CEPA is not relevant to, nor does it preclude, an assessment against the hazard criteria specified in the *Hazardous 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 does not preclude actions being taken under other sections of CEPA or other Acts.

2 Substance Identity

For the purposes of this document, the term “asphalt” refers to the substance with CAS RN 8052-42-4; the term “oxidized asphalt” refers to the substance with CAS RN 64742-93-4; the term “asphalts” refers to both. Various terms related to asphalt are used in this assessment, and their definitions are provided in Table A-3 in Appendix A for clarity. The asphalt considered in this screening assessment is a refined petroleum product imported into Canada or produced in Canadian refineries and upgraders, though natural asphalt deposits occur in various parts of the world due to mineral oil seepage from the ground (WHO 2004). Oxidized asphalt is also known as air-blown or air-refined asphalt, which is produced from asphalt during air-blown processes at elevated temperatures (WHO 2004). This process is principally one of oxidative condensation, which increases the molecular weight, melting point and asphaltene content (Kleinschmidt and Snoke 1958; CONCAWE 1992).

Asphalts are UVCBs rather than discrete chemicals and, as such, they have no specific chemical formula or structure. These UVCB substances are complex combinations of molecules that can originate in nature or are the result of chemical reactions and processes that take place during the distillation process. Given their complex and variable compositions, they could not practicably be formed by simply combining individual constituents.

Asphalts are complex combinations of high molecular weight organic compounds containing a relatively high proportion of hydrocarbons; carbon (C) numbers are predominantly greater than C_{25} with high carbon-to-hydrogen ratios (API 2009). Elemental analyses indicate that most asphalts contain approximately 79%–88% by weight (wt%) carbon, 7 wt%–13 wt% hydrogen, 2 wt%–8 wt% oxygen, trace to 8 wt% sulphur and trace to 3 wt% nitrogen (US EPA 2011).

Asphalts are among seven residual streams covered within the asphalt group as defined in the US Environmental Protection Agency High Production Volume (HPV) Challenge Program (API 2009). The asphalt group is typically made up of a group of heavy residual streams derived from the high temperature vacuum distillation of petroleum but may also be produced by thermal alteration during distillation, or from air blowing of residual streams (API 2009; Speight 2014). Substances in the asphalt group of the HPV program have high molecular weights (500–5000 g/mol) (API 2006, 2009).

Asphalt that has been refined to meet specifications for a particular use (e.g., paving, roofing, industrial or other uses) is termed asphalt cement (AI 1990b). Other synonyms for asphalt include asphaltic bitumen, asphaltum, petroleum asphalt, and, in Europe, bitumen (Speight 2014).

Asphalt is commercially available in a variety of types and grades. For example, there are different penetration grades of asphalt and hard asphalts (hard bitumens), which are described in detail in Section 5. The different grades of asphalt are still addressed by CAS RN 8052-42-4. Commercial asphalt has three main types: penetration grade, hard

asphalts and oxidized asphalts; therefore, it includes both non-oxidized asphalt (CAS RN 8052-42-4) and oxidized asphalt (CAS RN 64742-93-4) (API 2006, 2009). For the purpose of this assessment, the term “asphalts” is used to refer to both asphalt and oxidized asphalt.

The four major chemical classes present in asphalt, oxidized asphalt and other substances of the asphalt group from petroleum refineries are described in Table 2-1. Proportions of these chemical classes can vary because of significant differences in the compositions of crude oil sources and the manufacturing process. No two asphalts are chemically identical and chemical analysis cannot be used to define the exact chemical structure or chemical composition of asphalt (IARC 1998; CONCAWE 1992; API 2006, 2009).

Table 2-1. Summary of major chemical classes found in asphalts (API 2009)

Chemical Class	Description
Asphaltenes	“Brittle brown-black amorphous solids, which are highly condensed aromatic compounds with molecular weights of 2000–5000. The asphaltenes constitute 5%–25% wt%of asphalts. They are comprised of one or two chromophores each containing four to ten fused rings, with a significant number of alkyl substituents. A higher proportion of asphaltenes are present in hard asphalts.”
Resins	“Brown-black, adhesive, shiny solids or semi-solids. Comprised of heterogeneous polar aromatic compounds with small amounts of oxygen, nitrogen, and sulfur with molecular weights of 800-2000, they constitute 15 wt%–25 wt% of asphalts. They can be considered lower molecular weight asphaltenes and are dispersing agents for asphaltenes. The proportion of resin to asphaltenes governs to a degree the viscosity characteristic of the asphalt.”
Aromatics	“Viscous dark brown liquids containing mainly carbon, hydrogen and sulfur with minor amounts of oxygen and nitrogen. They have molecular weights of 500–900, and constitute 45 wt%–60 wt% of asphalt. They are compounds with aromatic and naphthenic-aromatic nuclei with side chain constituents.”
Saturates	“Viscous liquids or solids ranging from a straw to water-white colour, consisting mainly of long-chain, saturated hydrocarbons with some branched-chain compounds, alkyl aromatics with long side chains, and cyclic paraffins (naphthenes). They have molecular weights of 500–1000, and constitute 5 wt%–20 wt% of asphalt.”

Polycyclic aromatic hydrocarbons (PAHs) are part of the aromatic fraction of asphalt and oxidized asphalt. They are organic compounds comprised of two or more fused aromatic rings in various arrangements, and contain only carbon and hydrogen

molecules (Environment Canada, Health Canada 1994). The predominant PAHs present in asphalt and oxidized asphalt are of a high molecular weight. The presence of conventional PAHs (condensed three to seven ringed structures with no or few alkyl side chains) is limited in asphalt: 29 PAHs ranged from not detected to 28 mg/kg in one study (Kriech et al. 2002).

Commercial asphalt and oxidized asphalt can be treated further, processed or modified (e.g., by incorporating certain other substances) to produce asphalt formulations used as pavement and roofing materials, such as cutback asphalt, emulsified asphalt, foamed asphalt or polymer-modified asphalt. The term “asphalt materials” refers to Statistics-Canada-defined-asphalt, which is “produced by petroleum processing and includes asphalt flux, asphalt primers, asphaltic saturants, bitumals, cutback asphalts, liquid or solid asphalts, oxidized asphalt, paving compound and fluxes or primers” (Statistic Canada 2013); it is a general term that is inclusive of both asphalt group substances and asphalt formulations. Other residual stream substances in the asphalt group with different CAS RNs might also be used in asphalt formulations if they meet the material specifications. As data on asphalt formulations generally do not specify a CAS RN, and all residuals are expected to behave similarly when used in these products, the results of the analyses herein on such asphalt formulations for the purpose of assessing asphalt and oxidized asphalt may apply to all asphalt group substances that are used in such asphalt formulations. In addition, asphalt formulations can contain other petroleum substances (e.g., gas oils, kerosene, base oils, heavy fuel oils, distillate aromatic extracts, or naphtha). It is not possible to apportion releases of PAHs to individual substances when they are present in an asphalt formulation. Therefore, when characterizing environmental releases of PAHs from asphalt formulations, the contribution of components from any other petroleum substances present in the formulation is considered as part of the release scenario. Volatile organic compounds (VOCs) are also released during the application of asphalt formulations. The concern with VOCs is also from the cumulative loadings from numerous sources including asphalt formulations. VOCs are already listed on Schedule 1 of CEPA and actions are being taken to address releases of these compounds from different sources, including asphalt formulations. The environmental concerns of VOCs are not considered as part of this assessment.

3 Physical and Chemical Properties

The general physical and chemical properties for asphalt and oxidized asphalt are presented in Table 3-1 and Table 3-2, respectively. The physical and chemical properties of asphalts vary according to the primary components present which are determined in turn by factors such as its crude oil source and the processing steps involved in producing the asphalt substance (API 2006, 2009). Asphalts are complex mixtures with compositions that vary from batch to batch and in many cases only single values were found in the literature for physical–chemical properties that would be better represented by a range of values.

Table 3-1. Estimated physical and chemical properties for asphalt (CAS 8052-42-4)

Property	Value	Temperature (°C)	Reference
Physical state	Solid or semi-solid	-	CONCAWE 1992
Softening point (°C)[a]	30–75	-	API 2009
Boiling point (°C)	>450	-	API 2006
Density (kg/m^3)	950–1100	-	API 2009
Vapour pressure (Pa)	Negligible	-	API 2006
Vapour pressure (Pa)	100	120	Trumbore 1999
Vapour pressure (Pa)	4.6×10^7	315	Trumbore 1999
Vapour pressure (Pa)[b]	4.9×10^{-16} to 4.9×10^{-4}	-	Appendix B
Henry's law constant (Pa·m^3/mol)[b]	3.3×10^{-3} to 2.0×10^{11}	-	Appendix B
Log K_{ow}[c] (dimensionless)	≥10	-	API 2006
Log K_{ow}[b] (dimensionless)	8.8–25	-	Appendix B
Log K_{oc}[b] (dimensionless)	7.7–21.6	-	Appendix B

Water solubility (mg/L)	Extremely low water solubility at room temperature.	-	API 2006, 2009
Water solubility (mg/L)[b]	2.6×10^{-21} to 1.5×10^{-5}	25	Appendix B

Abbreviations: K_{oc}, organic carbon–water partition coefficient; K_{ow}, octanol–water partition coefficient.
[a]softening point is the temp at which asphalt softens enough to allow a steel ball to fall a distance of 25 mm (1 inch) through the asphalt (API 2006)
[b]based on representative structures with boiling point greater than 350°C and carbon number from C_{25} to C_{50}.
[c]log K_{ow} was estimated for various hydrocarbons having 25 carbon atoms using the computer program EPISuite (2000).

Table 3-2. Estimated physical and chemical properties for oxidized asphalt (CAS 64742-93-4)

Property	Value	Temperature (°C)	Reference
Physical state	Solid	-	CONCAWE 1992
Softening point (°C)	60–130	-	API 2009
Boiling point (°C)	>400	-	API 2009
Density (kg/m^3)	1000–1100	-	API 2009
Vapour pressure (Pa)	Negligible	-	API 2009

Water solubility (mg/L)	Extremely low water solubility at room temperature	-	API 2009

Twenty-two representative structures were selected (Appendix B) on the basis of two fundamental characteristics: a boiling point greater than 350 °C and a carbon number inclusively between C_{25} and C_{50}. Naphthalene (C_{10}) was included as a representative structure due to its presence in leachate of asphalt formulations, although it has a boiling point of 218 °C and would not be substantively present in untreated asphalt (i.e., asphalt not diluted or modified with other petroleum substances to reduce viscosity) (Kriech 1990, 1992a, 1992b). The presence of naphthalene in leachate is indicative of asphalt formulations containing other petroleum substances, or could simply be due to some carryover of naphthalene and its presence in leachate due to its high solubility. Asphaltenes and resin were not included within the representative structures because their complex structures are difficult to identify and their very high molecular weight (800–5000) makes them likely not bioavailable (Speight 2014; API 2009).

The physical and chemical behaviour of the individual representative structures will differ if these substances are present in a mixture. The vapour pressures of components in a mixture will be lower than their individual vapour pressures as explained by Raoult's law (i.e., the total vapour pressure of an ideal mixture is proportional to the sum of the vapour pressures of the mole fractions of each individual component). Similar to Raoult's law, the water solubilities of components in a mixture are lower than those for individual components alone (Banerjee 1984). Additionally, mixture components, which are normally solid under environmental conditions, might have lower melting points (and therefore be in a liquid state at lower temperatures), as well as increased vapour pressure and water solubility (Banerjee 1984). Nevertheless, the physical and chemical properties of the individual representative structures, as reported by Environment Canada (2014), indicate how the individual components of the mixture might behave in the environment.

Throughout this screening assessment, where information specific to asphalt was not available, information relating to its components was considered.

4 Sources

Asphalt (CAS RN 8052-42-4) is obtained as the non-volatile residue from crude oil distillation or as the raffinate following separation from a residual oil in a de-asphalting or de-carbonisation process (API 2009). In the refining process, starting with the residuum from atmospheric distillation, the maximum quantity of high value distillates are extracted from the residue until only the high boiling, high molecular weight

components are left. Oxidized asphalt (CAS RN 64742-93-4) is obtained by treating asphalt by blowing air at elevated temperatures (CONCAWE 1992).

Multiple sources of production quantities of asphalt materials (import and export statistics of asphalt, and related materials, including asphalt flux, asphalt primers, asphaltic saturants, bitumals, cutback asphalts, liquid or solid asphalts, oxidized asphalt, paving compound and fluxes or primers) for provincial and territorial production in 2012 are presented in Table C-1 (Appendix C). According to Statistics Canada, 4.35 million tonnes of all types of asphalt materials were produced in 2012 and 1.40 million tonnes were exported. After accounting for other adjustments, such as transfers and inventory changes, 4.04 million tonnes of asphalt materials were available for sale in Canada in 2012 (Statistics Canada 2013).

In Canada, an estimated 1.25 million tonnes of asphalt shingles and roofing materials are discarded every year into landfills (CCIC 2007). Asphalt shingles can be recycled into hot-mix asphalt for road or trail construction, or used as fuel in cement kilns (CCIC 2007). Asphalt is the most recycled substance in the USA with 80% of reclaimed asphalt pavement being reused in other roads (US EPA 1993). A similar reuse of asphalt is expected to occur in Canada. Up to 100% of existing asphalt used for the construction of roads can be recycled (Martec Recycling Corporation 2016).

Asphalt has been identified by the Organisation for Economic Co-operation and Development (OECD) as an HPV chemical, with 1000 tonnes or more produced per year (OECD 2004).The European Union (EU) and the United States Environmental Protection Agency (US EPA) have also identified asphalt as an HPV chemical (EC 2008a; US EPA 2011). HPV chemicals in the United States are produced or imported in quantities of one million pounds (approximately 450 000 kg) or more per year (US EPA 2011). In the EU, HPV chemicals are produced or imported in volumes of over 1000 tonnes per year (EC 2008a). Asphalt plants in the United States produce estimated 400 million tons of asphalt materials annually (NAPA c2016).

5 Uses

It has been determined from an in-depth literature review and a search of material safety data sheets (MSDS) that asphalt identified as CAS RN 8052-42-4 and oxidized asphalt identified as CAS RN 64742-93-4 are transported to other facilities where they are further refined for use in commercial and consumer products.

Information on the use for asphalt (non-oxidized; CAS RN 8052-42-4) and oxidized asphalt (CAS RN 64742-93-4) is often combined. There are three main types of commercial asphalt produced, as described in Table 5-1.

Table 5-1. Types of asphalt produced (CONCAWE 1992)

Type	Description
Penetration grade[a] (PG – asphalt	Residues produced from crude oil by atmospheric distillation followed by further processing such as

cements, viscosity-grade asphalts)	vacuum distillation (straight run asphalts), thermal conversion, partial oxidation (air rectification/semi-blowing) or solvent precipitation. These processes can be combined to meet application specifications for road surfacing or in roofing applications.
Hard asphalts (hard bitumens)	Manufactured using processes similar to penetration grades[a] but have lower penetration values and higher softening points. They are hard and more brittle than the PG, and are used primarily in the manufacture of asphalt paints and enamels.
Oxidized (air blown) asphalts	Produced by passing air through hot, soft asphalt feedstock under controlled conditions. They have a higher softening point with reduced susceptibility to changes in temperature and greater resistance to imposed stress than the PG. Applications include roofing materials, waterproof papers, electrical components, pipe coating, undersealing of concrete pavements, hydraulic applications, membrane envelopes, and paint manufacture.

[a]Penetration grade is a common asphalt binder classification (University of Washington 2005).

The United States High Production Volume (US HPV) Program reports that asphalt and oxidized asphalt represent over 99% of all asphalt material used in the United States and Europe, which is used primarily for road surface coating and roofing applications (API 2009). Based on an analysis of information from various sources, Cheminfo (2009) reports that, in the United States and Europe, 80-90% of asphalt is used in road surface coating, 10-15% in roofing applications, and 1-2% in other applications.

Cheminfo (2009) also estimated the proportion of asphalt and oxidized asphalt used in each of the above application types, though there is uncertainty in the values due to lack of quantitative data. It is estimated that 90% of the asphalt used in road surfacing and 50% of applications other than road surfacing or roofing is non-oxidized asphalt (Cheminfo 2009). Approximately 50% of the commercial asphalt in roofing applications is non-oxidized and about 50% is polymer-modified asphalt (CONCAWE 1992).

In Canada in 2012, 4.0 million cubic metres of asphalt and oxidized asphalt was sold (Statistics Canada 2013). Table 5-2 reports the use of these asphalt formulations in three broad sectors. The uses associated with manufacturing primarily correspond to the manufacture of roofing products, which represents about 20% of domestic use of asphalt formulations. Construction, commercial and other uses roughly correspond to road and driveway paving applications and represent approximately 80% of its total use (Statistics Canada 2013). These values are consistent with what has been reported globally.

Table 5-2. Use of asphalt formulations in Canada, 2012 (Statistics Canada 2013)

Sector	Use (million m^3)	Share (%)
Construction	2.5	63
Manufacturing	0.81	20
Commercial and other institutional	0.70	17
Total	4.0	100

Based on the data in Table 5-2, and assuming that 1% of domestic use is for applications other than road surfacing or roofing, it is estimated that approximately 80% of asphalt and oxidized asphalt is used for surfacing roads, 19% in roofing, and 1% in other applications. Table 5-3 provides the estimated proportion of asphalt and oxidized asphalt used for various uses in 2012 in Canada.

Table 5-3. Estimated volumes of asphalt and oxidized asphalt used in Canada in 2012

Use	Total asphalt[a] (million m^3)	Asphalt (non-oxidized)[b] (million m^3)	Oxidized asphalt[b] (million m^3)
Road surfacing	3.2	2.9	0.33
Hot-mix asphalt	2.8	2.5	0.28
Emulsions	0.36	0.32	0.04
Cutbacks	0.13	0.12	0.01
Roofing	0.77	0.38	0.38
Other	0.04	0.02	0.02

[a] based on 4.04 million cubic metres of asphalt and oxidized asphalt sold in 2012 (Statistics Canada 2013)
[b] based on estimate of the ratio of non-oxidized asphalt:oxidized asphalt of 90:10 for road surfacing, 50:50 for roofing and 50:50 for other (Cheminfo 2009)

The main use of asphalts is in road surfacing. This can include paving (application of asphalt cement), application of sealants, and maintenance (e.g., crack filler, pothole repair). Three asphalt formulations (i.e., hot-mix asphalt, asphalt emulsions and cutback asphalt) are prominent in paved surface construction as outlined in Table 5-4 and the estimated volumes used in Canada in 2012 are reported in Table 5-3 assuming that Canadian use is similar to that in the United States (US NTP 1997). For road surfacing in Canada, about 11% emulsions and 4% cutbacks are used.

Table 5-4. Description of asphalt formulations used in paving applications (US NTP 1997; WHO 2004)

Type of Use	Description
Hot-mix asphalt (HMA)	This accounts for 85% of asphalt products used in road surfacing in the USA. Most asphalt formulations used in paving operations are not oxidized. The asphalts are heated to about 149–177 °C and

	mixed with heated (143–163 °C) mineral aggregate. Once transported to the worksite, the HMA is applied to the roadway. The temperature of application is generally between 112 and 162 °C. In HMA, the asphalt functions as a waterproof, and thermoplastic and viscoelastic adhesives.
Asphalt emulsions	These are used primarily for road sealing and maintenance, accounting for 11% of asphalt used in road surfacing in the USA. Emulsified asphalt is a suspension of small asphalt cement globules in water, which is assisted by an emulsifying agent (such as soap). Emulsions have lower viscosities than asphalt without additives designed to reduce their viscosity, and can thus be used in low temperature applications. After an emulsion is applied, the water evaporates and the asphalt cement remains. Emulsions are often used as prime coats and tack coats.
Cutback asphalts	These are used primarily for road sealing and maintenance, accounting for 4% of asphalt used in road surfacing in the USA. Cutback asphalts are combinations of asphalt cement and petroleum solvent. Like emulsions, cutback asphalts are used because their viscosity is lower than that of unmixed asphalts and can thus be used in low temperature applications. After cutback asphalts are applied, the solvent evaporates and the asphalt and aggregate mixture remains. Cutback asphalts are less common today because the petroleum solvents are more expensive than water and the concern of release of VOCs. Cutback asphalts are primarily used as prime coat, tack coat and dust palliative/suppressant, which are sprayed on; it's also used as plant and road mix, stock pile, cold patch and driveway sealer (Environmental Health Strategies 2010).

The second major use of asphalt is in roofing, including underlayments, asphalt shingles, roll roofing, modified bitumen roofing and built-up roofing systems (Statistic Canada 2013; Cheminfo 2009).

A very small proportion of asphalts is used in other diverse applications (IARC 2011), many of which need to be purchased directly from the distributor. These include, but are not limited to, damp-proofing and waterproofing; mastic asphalt for industrial flooring; building papers; primes; caulking compounds; acoustical blocks, compositions, felts; bricks; joint filler compounds; soundproofing; stucco base; lining irrigation canals, water reservoirs, dams, and dykes; protective coatings for walls, cars, and water mains; brake linings; clutch facings; rubber extenders; lubricating grease; printing inks; well drilling fluid; boat deck sealing compound; enamels; paints; adhesives for the building industry;

coal briquetting; electrical insulation; and battery making (US DHHS 2000, Inchem 1982, HSDB 2009, Al 1990a in API 2009).

Asphalt was first registered in Canada in 1973 as an active ingredient under the *Pest Control Products Act* (PCPA).The pesticides containing asphalt were registered for use in pruning paints with pest control claims as a protective coating against moisture, wood rot disease and wood-boring insects. Under PMRA's re-evaluation program, registrants agreed to remove pest control claims. As a result, registrations of products under the PCPA have expired and the re-evaluation for asphalt was not carried out. Asphalt is not listed as an approved food additive in the Lists of Permitted Food Additives as regulated under the *Food and Drugs Act* nor has it been identified as being used/present in formulations of food packaging materials or incidental additives that come in contact with foods (Health Canada 2013, November 2014 email from Food Directorate, Health Canada, to Risk Management Bureau, Health Canada; unreferenced).

Asphalt is listed in the Natural Health Products Ingredients Database (NHPID) with a non-NHP role as it is not a naturally occurring substance included in Schedule 1 of the *Natural Health Products Regulations*. It is not listed in the Licensed Natural Health Products Database (LNHPD) to be present in any currently licensed natural health products (NHPID 2015; Canada 2003; LNHPD 2015).

6 Releases to the Environment

Releases to the environment may either be of asphalts themselves or of components of asphalt formulations, such as the more volatile components that may be released to air when heated, or the relatively more soluble components that might leach.

Releases of asphalt and oxidized asphalt are not reportable to the National Pollutant Release Inventory (NPRI 2008). However, releases to air including volatile hydrocarbons such as toluene, xylenes, and benzene from Canada's asphalt paving formulations and block manufacturing are reported.

Releases of components of asphalts can also occur from their use in road pavement, roofing, adhesives and sealants, paints and coatings, and miscellaneous consumer products. Meridian (2009) considered the potential releases to be small because asphalt formulations are in solid form.

In general, three operating procedures are involved in the process of transportation: loading, transit and unloading. Loading and unloading of asphalts is normally conducted at sites where access by the general public is limited. The handling of asphalts at petroleum facilities, for the purpose of transportation, is regulated at both the federal and provincial levels, with measures covering both loading and unloading (SENES 2010). Collectively, these measures establish requirements for the safe handling of petroleum substances and are intended to minimize or prevent potential releases during loading, transportation and unloading operations (SENES 2010). Unintentional spills or

leaks during the handling and transport processes were considered in this screening assessment for the potential to cause ecological harm.

The Ontario Ministry of the Environment (OMOE) spill database (OMOE 2013) was used to evaluate the frequency and volume of accidental releases of asphalts. The Spills Action Center at the Ontario Ministry of the Environment is responsible for managing oil spill reporting in Ontario. All spills of asphalt materials must be reported, including spills from motor vehicles, although there are exceptions for spills smaller than 100 L unless they are likely to enter waters, or where remediation has not occurred immediately.

The Ontario database does not report spills by CAS RNs; therefore, the analysis captures spills of all asphalt materials as a group. Spills not considered in the analysis were those due to collisions and storms, as well as those occurring on industrial sites (e.g., refineries); the latter were excluded because releases at these locations are expected to be contained and recovered, so the probability of entry into the environment would be minimal.

Finally, landfills that do not collect and treat their leachate may potentially release substances to ground or surface water via leachate. However, release of components from asphalt formulations that are discarded into landfills is expected to be low based on the low water solubility.

7 Environmental Fate and Behaviour

7.1 Environmental Distribution

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

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 (Potter and Simmons 1998). It has also been widely demonstrated that nearly all soils and sediments have populations of bacteria and other organisms capable of degrading petroleum hydrocarbons (Pancirov and Brown 1975). However, these generalizations tend to be less relevant when dealing with hydrocarbons with carbon numbers greater than 25, such as asphalts, when water solubility and volatilization are minimal at normal temperatures.

Given that asphalt exists in a semi-solid state and oxidized asphalt exists in a solid state at most temperatures (API 2006), they are unlikely to be found outside of where they were initially applied or spilled. Asphalts are also unlikely to change from the semi-solid or solid state except when being heated for application (API 2006). When released into

the environment, regular asphalts (without further treatment) tend to harden and remain intact (CONCAWE 1992); hot asphalt will flow until it cools below its pour point, then it will solidify and not disperse any further; whereas cutback asphalts and emulsified asphalts are fluids at ambient temperatures and thus might be more dispersive. Asphalt formulations for paving will not flow due to the high proportion of gravel and sand. However, heating of asphalts to facilitate paving and roofing applications produces vapours and fumes composed of the lower molecular weight components, which may be inhaled or deposited on the skin or clothing (US EPA 2011, 2006). Additionally, asphalts in roofing and paving products will become oxidized with exposure to air over time (Robertson 1991).

Due to their high molecular weights and hydrophobic characteristics, asphalts have extremely low water solubility, so that if spilled into water they will sink, rapidly solidify and not disperse. Despite the low solubility of asphalt, however, some of its components such as PAHs, can leach from asphalts. Generally, leaching tests from various asphalt formulations found concentrations of PAHs below detection limits or in the parts per trillion range (ng/L) in the leachate (Brandt and de Groot 2001; Kriech 1990, 1992, Legret et al. 2005; Cooper and Kratz 1996).

Additionally, asphalt in roofing and paving products will become oxidized with exposure to air over time (Robertson 1991). When asphalt is oxidized, ketones and carboxylic anhydrides are formed in the oxidized region of the molecule (Petersen 2009). Oxidized asphalts are produced from asphalt during air-blown processes, during which the softening point and asphaltene content of the product increase linearly (Kleinschmidt and Snoke 1958).

Due to the complex interaction of components within a mixture, which impacts their physical–chemical properties and behaviour, it is difficult to predict the fate of a complex mixture. Therefore, as a general indication of the fate of asphalts, the physical–chemical properties of representative structures of asphalts (Appendix B) were examined.

Based on the physical–chemical properties of representative structures of asphalts, the majority of components are not soluble in water nor easily degraded, and are likely to sorb to soil. The C_{25}–C_{50} components have boiling points ranging from approximately 395 to 746 °C. The individual components of asphalts are characterized by low water solubilities (2.6×10^{-21} to 1.5×10^{-5} mg/L) and low vapour pressures (3.1×10^{-15} to 4.9×10^{-4} Pa). As noted previously, the solubility and vapour pressure of components within a mixture will differ from those of the components individually and these interactions are complicated for complex UVCBs such as asphalts. In addition, they have very low to very high Henry's law constants (3.3×10^{-3} to 2.0×10^{11} Pa·m^3/mol), high log K_{ow} values (10.9–25), and high log K_{oc} values (7.7–21.6) (Appendix B).

Based on its low vapour pressure (3.1×10^{-15} to 4.9×10^{-4} Pa), the majority of components of asphalts are not expected to be found in air (Appendix B). Therefore, releases of asphalts to air are unlikely unless it is heated to a high temperature (e.g., greater than 150 °C). In preparing asphalt, asphalt cement is mixed with either a

petroleum diluent, to produce cutback asphalt or with emulsifiers, water and sometimes a small quantity of petroleum diluent, to produce emulsified asphalt. Once the liquefied asphalt cement is applied to the surface of the road, the diluent (petroleum solvent in the case of cutback asphalt and primarily water in the case of asphalt emulsions) evaporates, leaving the residual asphalt cement to perform its function. VOCs are released during this process.

The density of asphalt and oxidized asphalt varies with composition (0.95–1.10 g/mL; Table 3-1), such that, upon entering water some asphalts will float, while others will sink (CONCAWE 2001). Based on the water solubility of these components (2.6×10^{-21} to 1.5×10^{-5} mg/L), if a release occurs to water most representative structures of asphalts are not expected to dissolve into water to any great extent, except for naphthalene, which is a minor component as it is outside the boiling range of asphalts. Additionally, asphalts show little tendency to disperse in the water column, but instead will sorb to sediments based upon the high estimated log K_{oc} values of its components (CONCAWE 2001; Appendix B).

Based on the high estimated log K_{oc} values of components of asphalts, if released to soil, most components are expected to sorb to soil. Volatilization from moist soil surfaces may occur based upon estimated Henry's law constant values of 3.3×10^{-3} to 2.0×10^{11} Pa·m^3/mol for lower molecular weight representative structures of asphalts (alkanes, isoalkanes, cycloalkanes and one-ring aromatics), which might also volatilize from dry soil surfaces based upon their moderate vapour pressures (Appendix B).

7.2 Persistence and Bioaccumulation

Due to the complex nature of petroleum substances such as asphalts, the persistence and bioaccumulation characteristics for a suite of petroleum hydrocarbon structures representative of components expected to occur in asphalts are assessed based on empirical and/or modelled data.

7.2.1 Environmental Persistence

Persistence was characterized based on empirical and/or modelled data for a suite of petroleum hydrocarbons expected to occur in asphalts. Model results and the weighing of information are reported in the supporting documentation for the persistence and bioaccumulation of petroleum substances (Environment Canada 2014). These data are summarized in Appendix D.

Empirical and modelled half-lives in the atmosphere for all components of asphalts are less than 2-d (Environment Canada 2014). A number of three- to six-ring PAHs can undergo long-range transport to remote regions due to sorption to particulate matter (Environment Canada 2014).

Considering biodegradation in water, soil and sediment, the following components are expected to have half-lives greater than 6 months in water and soils and greater than a

year in sediments: C_{30} isoalkanes, C_{50} monocycloalkanes, C_{30}–C_{50} dicycloalkanes, C_{30}–C_{50} monoaromatics, C_{25}–C_{50} diaromatics, C_{30}–C_{50} three-ring aromatics, and C_{25}–C_{30} five-ring aromatics. The C_{30} monocycloalkanes and C_{30}–C_{50} monoaromatics have half-lives greater than a year in sediments (Appendix D).

Asphaltenes and resins, which comprise up to 50% of asphalts, are not represented in Environment Canada (2014); however, their high molecular weight and structural complexity indicate that they likely persist in the environment for a long time.

7.2.2 Potential for Bioaccumulation

Bioaccumulation potential for a suite of petroleum hydrocarbons expected to occur in asphalts was characterized based on empirical and/or modelled data. Bioaccumulation factors (BAFs) are the preferred metric for assessing the bioaccumulation potential of substances, as the bioconcentration factor (BCF) may not adequately account for the bioaccumulation potential of substances via the diet, which predominates for substances with log K_{ow} greater than approximately 4.5 (Arnot and Gobas 2003). Components with carbon numbers greater than $>C_{20}$ have estimated log K_{ow} values of > 8 and were not included in the modelling because there were insufficient empirical bioaccumulation data for these components.

The aliphatic and aromatic components of asphalts have carbon numbers greater than C_{25} and estimated log K_{ow} values greater than 10 (Appendix B). Evidence suggests that substances with log K_{ow} greater than 8 are not likely highly bioaccumulative due to reduced bioavailability, and there is also evidence that metabolism occurs to some degree (Arnot and Gobas 2006); thus, these substances are not expected to be highly bioaccumulative. While log K_{ow} data were not available for asphaltenes and resins, their high molecular weights (greater than 1000) suggest that they will not easily be absorbed by organisms. Therefore, the asphaltenes and resins are also not expected to bioaccumulate.

8 Potential to Cause Ecological Harm

8.1 Ecological Effects Assessment

8.1.1 Water

Asphalt and oxidized asphalt are not expected to cause acute or chronic toxicity to aquatic organisms due to their extremely low water solubility. As the molecular weight of components of asphalts range from 500 to greater than 5000, and due to their low water solubility, the likelihood of components of asphalts migrating into the water column is small, as demonstrated by the results of toxicity studies as described below. Measured concentrations of asphalt components in leachate from asphalt formulations are presented in Section 8.2.1.

Hot-mix asphalt is used to line fish hatchery ponds that are operated by the Oregon Department of Fish and Wildlife and the Washington State Department of Fisheries,

where no apparent adverse impact to the culture and propagation of sport and food fish was observed (API 2009).This indicates that asphalts are not toxic to fish even with long-term exposure.

Mahler et al. (2015) investigated the toxicity of runoff from unsealed asphalt pavement to *Ceriodaphnia dubia* and *Pimephales promelas*. Runoff samples were collected from pavement test plots in areas of active parking and driving, and the samples were analyzed for the 16 US EPA priority PAH[2] pollutants, as well as 4*H*-cyclopenta-[*def*] phenanthrene, seven *N*-heterocycles (azaarenes) (i.e., quinoline, isoquinoline, acridine, phenanthridine, carbazole, benzo[*c*]-cinnoline, and 2,2′-biquinoline; 1-methylphenanthrene), and nitrobenzene (Mahler et al. 2015). Groups of ten organisms in each of four replicates were exposed to runoff samples for 48 h. Three treatments of exposure waters were tested for each runoff sample: 100% runoff, 10% runoff (1:10 dilution with control water), and 0% runoff (100% control water). Mean mortality for both species in 10% and 100% runoff concentrations were less than 5%, and that in the control was less than 2% (Mahler et al. 2015).

Another study demonstrated that asphalt emulsions could stabilize waste incinerator salt and ash and significantly decrease their toxicity: inhibition of the observed parameter (IC50) in organisms (*Pseudomonas putida, Lactaca sativa* and *Sinapis alba*) was not observed even with 100% leachate (Vondruska et al. 2002).

Since there were no empirical aquatic toxicity data for asphalt (CAS RN 8052-42-4) or oxidized asphalt (CAS RN 64742-93-4), aquatic toxicity was modeled. PetroTox is an aquatic toxicity model specific for petroleum hydrocarbon mixtures (PetroTox 2012). This model assumes narcosis is the chemical mode of action and accounts for additive effects according to the toxic unit approach. The PetroTox model can be used to predict petroleum hydrocarbon toxicity for C_4–C_{41} compounds dissolved in the water fraction. Substances smaller than C_4 are considered too volatile to remain in water, while compounds larger than C_{41} are considered too hydrophobic and immobile, to impart any significant aquatic toxicity. This model is very sensitive to the proportion of hydrocarbons considered to be sufficiently soluble to impart toxicity. Thus, the model was run assuming a 1:1 ratio of components within the C_{25}–C_{35} versus C_{36}–C_{41} carbon ranges for illustrative purposes. The model cannot be applied to asphaltenes or resins, which comprise up to 50% of asphalts. However, given the physical–chemical properties of asphaltenes and resins (i.e., high molecular weight and low water solubility), they are not expected to be bioavailable or toxic to aquatic organisms.

The PetroTox model generates estimates of toxicity with a median lethal loading concentration (LL_{50}) rather than a median lethal concentration (LC_{50}) due to the

[2]The 16 US EPA priority pollutant PAHs are naphthalene, acenaphthylene, acenaphthene, fluorene, phenanthrene, anthracene, fluoranthene, pyrene, benz[*a*]anthracene, chrysene, benzo[b]fluoranthene, benzo[k]fluoranthene, benzo[a]pyrene, dibenz[a,h]anthracene, benzo[ghi]perylene, and indeno[1,2,3-cd]pyrene.

insolubility of petroleum products in water. The LL_{50} value is the amount of petroleum product needed to generate a water-accommodated fraction (WAF) that is toxic to 50% of the test organisms. It is not a measure of the concentration of the petroleum components in the WAF. The modelled ecotoxicological data in Table E-1 (Appendix E) indicates that even if assuming that as much as half the asphalt components are in the more bioavailable range (i.e., carbon number less than C_{36}), the acute toxicity is still extremely low for all test organisms, as indicated by LL_{50} values greater than 1000 mg/L. While the proportion of components in asphalts within certain carbon ranges is unknown, the ratio used in the model is considered a reasonable conservative approximation and indicates low hazard.

The predicted toxicity of asphalts was comparable to empirical results from toxicity studies with aromatic extracts in the carbon number range of C_{20}–C_{50} (Table E-2 in Appendix E). Aromatic hydrocarbons are considered to contribute the most to petroleum toxicity (Verbruggen et al. 2008). Therefore, the predictions from the PetroTox model are considered relevant and reliable.

8.1.2 Terrestrial

Due to the low water solubility and poor volatilization of asphalt and oxidized asphalt, it is unlikely that these substances will be directly toxic to terrestrial organisms. A review of the available literature did not find any reports of toxicity to terrestrial organisms. For example, Miller et al. (1980) conducted a 56-d laboratory study using natural soil media. In the experiment, *Phasiolus vulgaris* (bean) seeds or *Zea mays* (corn) seeds were exposed to either 4.09 or 20.5 g of blown asphalt (oxidized asphalt) per 1.8 kg of soil for 56 d. Results indicated that the blown asphalt had no effects on the growth of either bean or corn seeds (Miller et al. 1980; WHO 2004). In addition, plants are observed to grow next to or through cracks in asphalt pavements without any apparent harmful effects, which suggests low leaching of components from asphalt pavement to the soil.

8.2 Ecological Exposure Assessment

The ecological exposure assessment of asphalts focuses on unintentional releases from transportation and use, as these releases potentially pose the greatest risk to the environment. Releases to air during the heating of asphalts tend to have less of an impact on the environment because they are typically either transient in nature or of a small quantity. Gaseous releases tend to disperse quickly to concentrations that do not typically present a threat to non-human organisms. As such, releases to air are not considered further in the ecological assessment.

8.2.1 Release from road surface and roofing materials

Asphalts used for road paving and roofing are subject to leaching. However, asphalts predominantly contain large hydrocarbons with molecular weights in the range of 500–15 000 (API 2009) with low solubility, which are generally considered to have low bioavailability and ecotoxicity (see Sections 7.2.2 and 8.1).

Extensive research has been conducted on the leaching of PAHs from asphalt formulations used as pavement and roofing materials. This assessment considers PAHs leaching from asphalt formulations (such as asphalt emulsion, cutback asphalt and hot-mix asphalt) that might contain other petroleum substances (e.g., aromatic extracts, base oils, gas oils, kerosene, naphtha and heavy fuel oils). Thus, the release of these other petroleum substances from asphalt formulations is addressed in this assessment.

Leaching tests conducted on asphalt formulations (e.g., hot-mix asphalt, asphalt concrete pavements and roofing asphalts) have shown that equilibrium concentrations of PAHs in the leachate are generally below detection limits or are typically in the parts per trillion range (ng/L); however, low concentrations of some PAHs (e.g., naphthalene and phenanthrene) are often found in leachate (Kriech 1990, 1992a; Brandt and de Groot 2001; Kriech et al. 2002). The individual PAH concentrations measured in those laboratory leaching studies are shown in Table E-3 (in Appendix E). Kriech (1990) measured 16 PAHs in extracts from leaching tests with new (uncontaminated) hot-mix asphalt and detected only naphthalene at a concentration of 0.25 µg/L. Similar results were reported for leaching tests using new hot-mix asphalt and field samples of hot-mix asphalt collected from various locations on a road (Kriech 1992a). Only naphthalene was detected in samples collected from the road with concentrations ranging from 0.26 to 0.31µg/L; naphthalene at 0.76 µg/L was measured in soil extracts of samples collected from the shoulder of the road (Kriech 1992a). With further development of laboratory analytical methodology, Brandt and de Groot (2001) detected low concentrations of PAHs (less than detection limit for benzo[a]pyrene (B[a]P) to 0.12 µg/L for naphthalene) in leachates from conventional penetration grade asphalt using high-performance liquid chromatography (HPLC) analysis. Mahler et al. (2004) found that of 17 PAHs (15 of the 16 US EPA priority PAHs plus 9h-fluorene and nitrobenzene) analyzed, only anthracene was detected in the laboratory simulated runoff from test plots and a used parking lot sealed with asphalt emulsion at an estimated concentration of 0.09–0.23 µg/L (depending on the sampling date as the concentration decreased over time) and 0.42 µg/L, respectively. In this and similar studies, the concentration of PAHs in runoff was initially small and decreased over a seven-week period following application (Mahler et al. 2004, 2005; Rowe and O'Connor 2011).

Except for those test samples of new asphalt formulations obtained directly following manufacture, some PAHs in leachate might be from other sources such as vehicle tire wear and contamination from transportation. Old or weathered asphalts, as demonstrated through leaching studies, are likely to contain multiple sources of petroleum products (e.g., vehicle emissions, lube oils, and tire wear). Therefore, leaching studies on fresh asphalts provide a better indication of what could be released from asphalts. In a study by Birgisdottir et al. (2007), PAH leached from samples collected on the surface of an old road built in 1980 were much more elevated compared to the samples from the base layer of a new road. The PAH concentrations from the base layer of a new road are considered more reliable for evaluating PAH leachability from asphalts because the top layer is likely contaminated.

Slightly higher release of PAHs from cutback asphalts than from asphalt emulsions and hot-mix asphalt was observed (Table E-3 in Appendix E). Kriech (1992b) found relatively higher PAHs in the leachate from new asphalt emulsion, gelled asphalt and cutback asphalt. Material safety data sheets from various companies for these three asphalt formulations identified the presence of solvents such as kerosene or fuel oil no. 2 in the formulations (MSDS 2006, 2007a,b), though asphalt emulsion does not usually contain solvent. The PAHs in the leachate mostly originate from the solvent components in the formulation. The solvent components are volatile; when exposed to air, they evaporate and leave the hard asphalt at the site of application after curing (Speight 2014). Rapid-curing (RC), medium-curing (MC) and slow-curing (SC) cutback asphalts are classified depending on the relative speed of evaporation, and they contain solvents with different boiling points (Asphalt Institute 2007). RC and MC asphalts generally contain solvents with a boiling point similar to naphtha and kerosene, respectively (Environmental Health Strategies 2010). These solvents are volatile, and if released to the environment, they will not likely persist for long in the air (Environment Canada 2014). SC asphalts generally contain solvents with a boiling point similar to fuel oils. Although the SC asphalt solvents are less volatile and more persistent than naphtha and kerosene, asphalt can entrap these solvents and limit their availability to the environment and the organisms (Meegoda 1999; Vondruska et al. 2002).

The slightly higher release of PAHs from cutback asphalts (as shown in Table E-3 in Appendix E) is not continuous. The risk of release is greatest following application and before it is fully cured. Cutback asphalt is primarily used as prime and tack coat (Environmental Health Strategies 2010). Prime and tack coats are used for preparing the surface and promoting bonding to the subsequent pavement layers; they are applied only when the road is constructed and are subsequently covered underneath after paving (US DOT 2005; TDOT 2017). Spray application of cutback asphalts might result in release of VOCs, which will quickly dissipate. In addition, cutback asphalts are less commonly used today due to the release of VOCs and the cost of petroleum solvents (US NTP 1997).

In preparing asphalt, asphalt cement is mixed with either a petroleum diluent, to produce cutback asphalt or with emulsifiers, water and sometimes a small quantity of petroleum diluent, to produce emulsified asphalt. Once the liquefied asphalt cement is applied to the surface of the road, the diluent (petroleum solvent in the case of asphalt cutbacks and primarily water in the case of asphalt emulsions) evaporates, leaving the residual asphalt cement to perform its function. VOCs are released during this process.

Studies with reclaimed asphalt pavement samples have found that the majority of PAHs are below detection limit ranges (Brantley and Townsend 1999; Townsend 1998; Legret et al. 2005). Legret et al. (2005) detected B[a]P and fluoranthene at concentrations greater than their respective detection limits in leachate from a reclaimed asphalt pavement sample.

Increased temperatures, the presence of road salts and high acidity can increase the rate of leaching from fresh asphalt. This was shown to be the case in a chemical oxygen

demand leaching test in which the amount of organic components in the leachate was determined by the oxygen consumption for complete chemical oxidization (Cai et al. 2009).

The amount of PAHs that can effectively leach from asphalt is dependent upon the amount of PAHs that are present. Roofing asphalt formulations contain lower concentrations of PAHs (range: 4–23 mg/kg) compared to road asphalt formulations (range: 1.9–66 mg/kg) (Kriech et al. 2002). Kriech et al. (2002) did not find any PAHs leaching from four roofing test samples. In tests on the aqueous leaching of PAHs from commercially available asphalt formulations, the level of PAHs leaching from roofing asphalts was minimal and generally lower than that from paving asphalts (Table E-3 in Appendix E; Brandt and de Groot 2001). Similarly, the PAH concentration of the asphalt-based pavement sealant (i.e., 50 mg/kg) (Mahler et al. 2012) is within the same range as asphalt formulations (Kriech et al. 2002) and is much lower than that of coal-tar-based pavement sealant (i.e., 66 000 mg/kg) (Mahler et al. 2012). The total PAH concentrations in runoff water from asphalt-based pavement sealant is low (i.e., 2 ug/L), compared to that of coal-tar-based pavement sealant (i.e., 71 ug/L) (Watt et al. 2010; Mahler et al. 2012).

Some studies show that asphalt emulsions can stabilize and solidify petroleum-contaminated soils to produce quality construction material (Meegoda 1999). Concrete made from cold-mix asphalt (CMA) consisted of asphalt emulsions mixed with six levels of petroleum-contaminated soils (PCSs). A uniform leaching test was designed to simulate the contaminant release rate from CMA made with PCSs when used as pavement and exposed to realistic environmental conditions. The structural integrity of the samples was kept intact during the test. Only the low molecular weight hydrocarbons (i.e., 2-methylpentane, heptane, benzene, toluene, ethylbenzene, *m*-xylene, *o*-xylene and 1,2,4-trimethylbenzene) found in the PCSs were analyzed. None of these compounds leached from any of the six mixtures (the detection limit was approximately 0.1 part per billion), except for iso-octane, which was present in two soils at 12.5 and 10 ppm (12.5 and 10 mg/L) (Meegoda 1999). While components from the cold-mix asphalt were not measured, the ability to entrain the low molecular weight and more soluble compounds within the cold-mix asphalt concrete indicates that the larger components of asphalts should be similarly entrained and have low leachability. Loss of the low molecular weight contaminants due to volatility is possible during the preparation of the cold-mix asphalt concrete, so the leaching test might have underestimated the leachability of the contaminants compared to the original amount in the contaminated soil. In addition, Vondruska et al. (2002) demonstrated that asphalt emulsions could stabilize salt and ash, which further supports the entrainment of low molecular weight substances in in asphalt.

Studies on metals leached from asphalts are also available. Aluminum and mercury were found at concentrations of 0.014 and 0.027 mg/L, respectively (Azizian et al. 2003). However, other studies identify metals as coming from the aggregate in the pavement and not from the asphalt itself (Kriech 1990, 1992; Brantley and Townsend 1999; Azizian et al. 2003). These metals include aluminum, barium, chromium, lead,

and mercury. Therefore, metals leaching from asphalt are not considered further in the assessment.

Leaching of other components of asphalts, such as aliphatics, resins and asphaltenes, is expected to be lower than PAHs due to their lower solubilities. Furthermore, the solubility of PAHs decreases with increased alkylation of the PAH (Gustafson et al. 1997). Overall, exposure of ecological receptors to asphalt components in leachates is considered limited based on the very low concentrations of PAHs leaching from asphalt paving and roofing formulations.

8.2.2 Exposure from spills

Spill data from the Ontario Ministry of the Environment databases were analyzed. The spill data reported under "asphalt" in this database included data from both liquid asphalt and asphalt found in shingles (roofing materials) or mixed with aggregates (pavements). However, it is unclear how many spills within the database were liquid asphalt, hot-mix asphalt, or asphalt shingles as this was not consistently recorded. The spills that include shingles or road debris are primarily composed of aggregate, typically gravel or sand, with only 5%–35% of the mixture being asphalt (Kreich et al. 2002; CCIC 2007).

There were 37 spills of asphalt materials reported to the Ontario Ministry of Environment from 2008 to 2012 (Table C-2 in Appendix C). Of the reported spills in the Ontario database between the years 2008 and 2012, the volume released was reported for 25 of these spills (Table C-2 in Appendix C). The average number of spills reported annually was less than eight (OMOE 2013). The total known spill volume was 154 017 L, with a median of 600 L. Most spills were to land (30 incidences), followed by five spills to water and one release to air; one incident did not report the spill medium (Table C-3 in Appendix C). Various causes for spills of asphalt materials were reported such as discharges or bypass to a watercourse, overflow, pipe, hose or valve leakage or failure, or process upset/malfunction. The causes for ten spills (25.4% by volume) were not specified (Table C-4 in Appendix C; OMOE 2013).

Once spilled, asphalts are generally not expected to disperse in the environment because of the high viscosity, high K_{ow} and K_{oc}, and low water solubility of their components (see Section 7.1). Regular and oxidized asphalts will tend to harden and remain intact upon release. Cutback asphalts and emulsified asphalts are liquids at ambient temperatures and will be more dispersive if released. However, the use of cutback asphalts is less common nowadays, which limits its potential release from spills.

8.2.3 Releases from industrial operations

The manufacture of hot-mix asphalt and roofing products is the primary industrial operation for asphalts (Cheminfo 2009). If water is used to clean production equipment and/or facilities, wastewater might be another source of potential release. As asphalt

components are highly hydrophobic, the majority of the components will be sorbed to sludge in the wastewater treatment system and end up in biosolids.

8.3 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 section 76.1 of CEPA. Multiple lines of evidence suggest that asphalts are not toxic to the terrestrial or aquatic environments as outlined below and in Table 8-1.

Table 8-1. Lines of evidence for risk characterization

Line of evidence	Key results	Reference
Hot-mix asphalt use as lining for fish ponds	No adverse effects observed over years	API 2009
Plant test	No growth effects on seeds	Miller et al. 1980
Modelled LL_{50}'s	LL_{50} > 1000 m g/L indicating low hazard	PETROTOX 2012
Aquatic toxicity of runoff from unsealed asphalt pavement	Mean mortality was not significant even in undiluted runoff samples	Mahler et al. 2015
Laboratory test of PAH leaching from paving and roofing formulations	Concentrations of PAHs in leachates were minimal. Most were lower than the detection limit and/or the CCME water quality guidelines.	See Table E-3 in Appendix E
PAHs in simulated runoff from asphalts	Concentrations of PAHs in simulated runoff from unsealed asphalt, asphalt emulsion sealed test plots and a used parking lot were not detected or were low; the concentrations decreased over time.	Mahler et al. 2004, 2005, 2012; Rowe and O'Connor 2011
Stabilizing and binding contaminants	None or very low concentrations of contaminants were detected in the leachate of petroleum-contaminated soils with asphalt emulsions. No acute aquatic toxicity (IC_{50}) of leachate was observed.	Meegoda 1999 Vondruska et al. 2002
Physical–chemical data	Low bioavailability, and thus bioaccumulation potential, for resins, asphaltenes and other components based on large molecular size, high K_{ow}, high K_{oc}, and low solubility.	See Sections 7.1,7.2.2 and 3

Abbreviations: LL_{50}, this refers to lethal loading, the amount of product necessary to kill 50% of test organisms; K_{oc}, organic carbon–water partition coefficient; K_{ow}, octanol–water partition coefficient

No adverse effects were observed in fish in aquaculture ponds with asphalt linings (API 2009). Oxidized asphalt also had no effect on the growth of either bean or corn seeds (WHO 2004). Modelled toxicity for asphalts indicates low toxicity (LL_{50}'s greater than 1000 mg/L assuming 50% of components are less than C_{36}) (PetroTox 2012).

Studies on the leaching of components from asphalt formulations reported low or undetectable concentrations of PAHs and inorganics from the examined asphalt road pavement and roofing formulations (API 2009; Kriech 1990, 1992a; Brandt and de Groot 2001; Kriech et al. 2002). Most PAH concentrations were lower than the detection limit or the CCME water quality guideline with a few exceptions (see Table E-3 in Appendix E). The relatively high PAH concentrations in leachates from aged asphalt road surfaces were attributed to contamination from other sources (see Section 8.2.1). Concentrations of PAHs in laboratory simulated runoff from unsealed asphalt pavement, asphalt emulsion sealed test plot and a used parking lots were not detected or were low; and the concentrations decreased over time (Mahler et al. 2004, 2012). The toxicity of runoff from unsealed asphalt did not show significant mortality in aquatic species exposed to diluted or undiluted runoff samples (Mahler et al. 2015). Although the particulate-phase PAHs are also present in the runoff besides the dissolved phase, it is of lower concern as the PAHs in the particulate fraction often have high molecular weight and low water solubility, hence they are not likely bioavailable to the aquatic organisms. The release of these PAHs will contribute to the loading of PAHs in the sediment that has been previously assessed (Environment Canada, Health Canada 1994). These findings indicate that exposure to the components leaching from asphalt formulations, whether used on paved surfaces, roofs, or disposed of in landfills, pose a low risk to the aquatic environment.

In addition to a low leaching of components of asphalts, asphalt formulations have been used to stabilize and solidify petroleum-contaminated soils and waste incinerator salts and ash. The ability of asphalts to bind hydrocarbons and other contaminants (e.g., metals) can significantly decrease the availability of these potential contaminants to leach out to the environment (Meegoda 1999; Vondruska et al. 2002). This process would also occur with hydrocarbons naturally present within the asphalts, limiting their availability and ability to leach from the cured asphalt formulations.

For reclaimed asphalt pavement (RAP), the origin of some substances measured in leaching studies is uncertain, because the recycled asphalt formulations might have been exposed to vehicle emissions, lube oils, gasoline and metals from brake pads during their original use (Brantley and Townsend 1999, Norin and Strömvaix 2004). However, since the asphalt formulations are recycled, any PAHs from these sources will be mixed into asphalt and likely entrapped.

For cutback asphalt, the slightly higher release of asphalt components in leachate than from asphalt emulsion and hot-mix asphalt is still of low significance (Table E-3 in Appendix E), because most of the PAHs in the leachate are below the CCME quality guideline, and will likely be further diluted by runoff and result in even lower concentrations in the environment. The major use of cutback asphalt as prime and tack

coat is infrequent and does not result in continuous release. Furthermore, the decreased use of cutback asphalts also limits the exposure of cutback asphalt (US NTP 1997). Therefore, the exposure from cutback asphalts is considered low.

Although the toxicity data of asphalts to terrestrial organisms are limited, asphalts are not considered to be toxic to terrestrial plants.

Very limited ecotoxicity data were available for asphalts or asphalt formulations overall. However, the observed low toxicity in the available studies, as well as the physical-chemical properties (e.g., high K_{ow}, high K_{oc} and low water solubility) of components of asphalts, and the very high molecular weight of asphaltenes and resins as major chemical classes in asphalts, indicate that asphalts are not likely to enter the environment in such a way as to be bioavailable or cause direct toxicity.

The use of asphalts in industrial operations may result in release of components in sludge and biosolids, if water is used to clean production equipment and/or facilities. However, because components of asphalts are generally not bioavailable, the risk is considered low.

A total of 37 spills of asphalt materials were reported in the Ministry of Ontario database. They are not likely to disperse in the environment once spilled, due to their low mobility. The lack of bioavailability and the low toxicity of asphalts indicate that the ecological concern is likely low when spilled.

8.3.1 Uncertainties in Evaluation of Ecological Risk

Modelling the physical and chemical properties, as well as the persistence and toxicity of asphalts, is based on representative structures potentially found in asphalts, which do not include some of the principal components, namely asphaltenes and resins, due to their extreme complexity and variability. The physicochemical properties of 22 representative structures were used to estimate the behaviour of asphalts. The reliance on this method for the physical and chemical characteristics, as well as for persistence and bioaccumulation potential, is a source of uncertainty in the prediction of partitioning to different environmental compartments and given the lack of empirical data for these processes. Given that a variety of different representative structures may be derived for the same petrochemical, it is recognized that structure-related uncertainties exist for these substances. The lack of specific proportions of representative structures in asphalts is also a source of considerable uncertainty. However, the physical-chemical properties (very low water solubility and very large molecular weight) of most components of asphalts, including asphaltenes and resins, indicate that they are not bioavailable and have low toxicity.

Leaching data for asphalt formulations are not specifically associated with a CAS RN, as leaching of PAHs from various types of asphalt formulations were considered. The

amount of PAHs in the leachate originating from asphalts was also uncertain. However, even when considering multiple sources of PAHs in the leachate, the overall release is insignificant, which was evident in the aquatic toxicity study on runoff from a parking lot (Mahler et al. 2015).

There were insufficient data to evaluate the release of petroleum substances from all types of use of asphalt formulations, (e.g., cutback asphalts used as stock pile mixes and cold patching). However, it is evident that once a substance in the formulations is mixed with asphalt, it is likely entrapped in asphalt and has decreased environmental availability (Meegoda 1999; Vondruska et al. 2002).

Large quantities of asphalt formulations are disposed of in landfill each year. Components of asphalts, such as PAHs, may contribute to the total concentration of these individual chemicals in the landfill leachate. While available evidence on the leaching of PAHs from asphalt formulations indicates low risk, the environmental conditions within a landfill could affect the leachability of components out of asphalt formulations. It is currently not possible to estimate what the contribution of asphalt formulations to landfill leachate might be. However, the physical-chemical properties of most components of asphalts indicate that asphalts are not bioavailable and have low toxicity.

9 Potential to Cause Harm to Human Health

9.1 Exposure Assessment

This assessment focuses on estimating general population exposures to volatile substances released during the production, refining and use of asphalt and oxidized asphalt, in areas surrounding industrial and application sites. Estimates are also derived for exposures to asphalts that may occur from the use of consumer products containing asphalts, as well as for ingestion of house dust that may contain substance components that originate from asphalt-based pavement sealant. Determining the potential for exposure to various substances found in asphalts from consumer products is difficult due to the variable nature of both the product formulations and the substance composition of asphalts.

Polycyclic aromatic hydrocarbons (PAHs), naphthalene and benzene are minor components of asphalts that are the basis of characterizing long-term exposure to asphalts, and are recognized as presenting a potential hazard to human health. These substances have been previously assessed by the Government of Canada and are included on the List of Toxic Substances in Schedule 1 of CEPA (Environment Canada, Health Canada 1993, 1994, 2008). Benzene, naphthalene and PAHs are organic compounds that contain only carbon and hydrogen atoms that form aromatic rings, where benzene contains a single ring and PAHs contain two or more fused rings.

9.1.1. Industrial Releases from Processing, Handling and Storage of Asphalts at Refineries and Asphalt Plants

Given that asphalts are predominantly composed of non-volatile substances, inhalation exposure of the general population to fugitive emissions of components of asphalt in the vicinity of industrial facilities that process, handle and store asphalt is expected to be low, even at elevated substance working temperatures. However, a Hot Mix Asphalt Plants Emissions Report (US EPA 2000) was prepared by the Office of Air Quality Planning and Standards at the U.S. Environmental Protection Agency that characterized asphalt emissions to air. These emissions data were therefore used as input parameters in SCREEN3 for dispersion modelling (SCREEN3 2011), in order to confirm the low potential for exposure to benzene and PAH components that may be released during the handling and storage of bulk quantities of asphalts (see Appendix F for input parameters) (US EPA 1992a). Emissions were considered to originate from an area source, given the size, potential points of emissions, and locations of the facilities involved. Total emission rates of benzene were reported to be 12.85 kg per year for a 100 000 tonne batch mix asphalt plant and 35.96 kg per year for a 200 000 tonne drum mix asphalt plant (US EPA 2000).

SCREEN3 is 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) (SCREEN3 2011). 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 volume source releases. SCREEN3 gives the maximum concentrations of a substance at chosen receptor heights and at various distances from a release source in the direction downwind from the prevalent wind one hour after a given 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 direction. This gives an estimate of the air concentration over a 24-hour exposure (US EPA 1992a; SCREEN3 2011). Similarly, for exposure events happening over the span of a year, it can be expected that the direction of the prevalent winds will be more variable and uncorrelated to the wind direction for a single event; thus, the maximum amortized exposure concentration for one year is determined by multiplying the maximum 1-hour exposure by a factor of 0.08 (US EPA 1992a; SCREEN3 2011). Such scaling factors are not used for non-point source emissions. However, to prevent overestimation of the exposures originating from area sources, a scaling factor of 0.2 was used to obtain the yearly amortized concentration from the value of the maximum 1-hour exposure concentration determined by SCREEN3(SCREEN3 2011).

The results of the dispersion modelling indicate a low level of benzene at a distance of 230 m from the fence-line of the facility, where this distance represents the maximum concentration of benzene found in air (Table F-2 of Appendix F). Map analysis suggests that residential homes may exist within this distance, which could lead to an incremental increased long-term exposure to benzene for those residing in the vicinity of such facilities. The annual concentrations of benzene at 230 m were estimated to range from 0.018 to 0.051 $\mu g/m^3$, a fraction of the 2009 Canadian average background

concentration of benzene in ambient air of 0.32-0.84 µg/m^3 for rural and urban areas (NAPS 2012).

Total emissions of six individual PAHs (benzo[a]pyrene, benzo[a]anthracene, benzo[b]flouranthene, benzo[k]flouranthene, dibenz[*a,h*]anthracene, indeno[1*,2,3*-cd]pyrene) from asphalt refining were also estimated by dispersion modelling using SCREEN3. PAHs with 5 or more rings have been shown to be predominantly particle-bound in the atmosphere, and are typically associated with particles less than 2.5 µm in diameter (Ontario 2011; EC 2001). During normal inhalation exposure to PAH-containing aerosols, the majority (likely greater than 80%) of inhaled PAHs are expected to be deposited on the thin alveolar epithelium and be rapidly absorbed into the blood (Ontario 2011). These considerations support the validity of conducting dispersion modelling for PAHs that originate from asphalt plants, where there could be inhalation exposure to PAHs for those residing in the vicinity.

Relevant parameters that were used in the modelling of PAH dispersion from asphalt plants are presented in Appendix H, Table F-3. The results of the modelling at 230 m from the site of the area release source are presented in Table F-4 for individual PAHs. The concentrations at 230 m were used to characterize risk of long-term general population exposure to PAHs originating from production of asphalts and handling at a facility. For individual PAHs, the estimated concentrations at 230 m range from 0.00016 ng/m^3 for dibenz[*a,h*]anthracene to 0.528 ng/m^3 for benz[*a*]anthracene based on the conservative release quantities associated with drum mix asphalt plants (US EPA 2000). These results corroborate occupational studies that indicate general population inhalation exposure to asphalt components are generally low, including for those down-wind of facilities that heat asphalt (refineries, terminals, hot mix plants, and roofing plants) (Cheminfo 2009).

An occupational exposure study was conducted at an asphalt plant in Germany that measured worker exposure to vapour and aerosols of asphalts (Rühl et al. 2006). During production of asphalts, air monitoring determined that a 95th percentile exposure to total vapour and aerosols of asphalt would be 33.2 mg/m^3 (arithmetic mean = 2.11 mg/m^3) in the plant. External to the asphalt plant, air monitoring showed that a 95th percentile exposure would be 0.88 mg/m^3 (arithmetic mean = 0.70 mg/m^3), showing that a substantial drop occurs within the facility grounds, over only a short distance from the emissions source. In the same study, B[a]P air concentrations were determined to range from 0.008 to 0.078 µg/m^3 in the plant (Rühl 2006). This direct air monitoring study therefore supports the results of air dispersion modelling with SCREEN3 in the estimation of potential long-term exposures of the general population to volatiles of asphalts in the vicinity of asphalt plants.

9.1.2 Releases from Transport of Asphalts

Releases of volatiles may also occur during the transport of asphalt products in open top trucks that carry rolled asphalt. Occupational exposure to vapour and aerosols during the transport of asphalts have been measured with air monitoring (Rühl 2006). A

value of 4.33 mg/m^3 for a 95th percentile exposure was found (arithmetic mean = 1.81 mg/m^3) for continuous exposure of a truck driver. A conservative estimate for transit losses may also be calculated by using stationary storage tank formulas adapted to typical dimensions of trucks (US EPA 2008). However, due to the low volatility of asphalts, the relatively small quantity transported in a truck, and the fact that it is an intermittent moving source (a line source of exposure), exposure is expected to be low for a bystander. Therefore, exposures that are estimated for the general population in the vicinity of asphalt facilities, as well as for estimates of exposure for bystanders near paving operations, are expected to cover bystander exposure to asphalt that occurs from open-top truck transport.

9.1.3 Exposures from Asphalt Paving and Roofing Operations

During paving and roofing operations asphalt is heated to a suitable working temperature and this produces vapours. These vapours cool and condense, producing a substance referred to as asphalt fume. Therefore, exposure can occur to both asphalt vapour and to asphalt fume. The properties of fumes have not been well characterized, but available studies tend to focus on asphalt fumes rather than vapours (Cheminfo 2009, CONCAWE 1992, API 2009). The data indicates that fumes contain low concentrations of PAHs (Van Metre 2012a, Van Metre 2012b, Diamond Environment Group 2011, Inchem 2004).

Application of asphalts to roads, roofs, and other surfaces may result in short-term inhalation exposures for the general population (Cheminfo 2009). No studies were identified which measured air concentrations of asphalt components in the vicinity of paving or roofing activities. However, a number of occupational inhalation exposure studies were identified in the literature. A recent study on personal breathing zone exposures among hot-mix asphalt paving workers measured air concentrations of individual PAHs (Osborn 2013). The highest breathing zone PAH concentration was identified for naphthalene at 1593 ng/m^3, exceeding other measured substances by an order of magnitude (Osborn 2013). This concentration was therefore selected as a conservative estimate of potential exposure of the general population in the vicinity of asphalt paving operations. It is noted, however, that exposures of the general population would be lower than the exposure derived from direct occupational exposure.

9.1.4 Environmental media, drinking water and food

Historically, asphalts were used to coat the interior of cast iron water pipes (Inchem 2004), but was replaced by cement in the 1970's (Blokker 2013). Thus, exposures via drinking water would be expected to be low.

Asphalt is not listed as an approved food additive in the Lists of Permitted Food Additives as regulated under the *Food and Drugs Act* nor has it been identified as being used/present in formulations of food packaging materials or incidental additives that come in contact with foods (Health Canada 2013, November 2014 email from Food Directorate, Health Canada, to Risk Management Bureau, Health Canada;

unreferenced).Migration into food and subsequent exposure is expected to be limited from petroleum asphalt when used as an indirect additive in food contact substances.

Exposure to asphalts from environmental media, drinking and food is expected to be low.

9.1.5 Consumer Products

Asphalt and oxidized asphalt are included on the List of Prohibited and Restricted Cosmetic Ingredients (more commonly referred to as the Cosmetic Ingredient Hotlist or simply the Hotlist). The Hotlist is an administrative tool that Health Canada uses to communicate to manufacturers and others that certain substances, when present in a cosmetic, may contravene (a) the general prohibition found in *Food and Drugs Act* or (b) a provision of the *Cosmetic Regulations*. As a result of this prohibition, exposure from cosmetic products is not expected.

Asphalts may be found in adhesives and sealants (e.g., cold-application cement, driveway sealer), paints and coatings (e.g., asphalt primer), and for miscellaneous use (e.g., wall insulation). Many of these products are considered commercial grade and need to be purchased directly from the distributor (Personal communication between Health Canada Risk Management Bureau and Industry, June 2010).

For professional roof resurfacing activities, the potential for inhalation exposure to asphalts for the general population is low. Roof resurfacing occurs outdoors and at variable locations. Any transient inhalation exposures are considered covered by the characterization of bystander inhalation exposure to naphthalene from road paving operations.

There is the potential for direct dermal contact with roofing shingles containing asphalt blends, and with dry asphalt coated surfaces such as driveways and roadways. The use of asphalts in these products is to act as a binder, and therefore by the very nature of this property it is expected there is limited transfer of asphalt components to the skin. Following these types of considerations in the assessment of the variety of products available, the sentinel products considered for estimating direct dermal exposure are driveway sealants and moisture/damping proofing paints, as these products exist in an uncured liquid state. Inhalation exposure to asphalt vapour from these products are expected to be negligible given the ambient application temperature for these products. If inhalation exposures occur, they are considered to be covered by the characterization of bystander inhalation exposure to naphthalene from road paving operations.

Dermal Exposure to Asphalts from the use of Coating Products

For estimating dermal exposure to asphalts from the use of consumer products, a scenario was developed that considered an individual applying asphalt-based sealant or waterproofing product once per year as part of regular home maintenance. Spills, splashes and handling of the container or applicator could result in exposure; however,

for the purpose of this assessment, incidental contact with a small amount of product is assumed to occur via the palms of the hands. The mass of asphalts in contact with the skin as a result of such an exposure is derived using the EPA-Versar thin film approach (US EPA 2011c). This approach characterizes exposure from a mineral oil substance following a partial wipe of the hands to remove excess material. The substance thickness "thin film" estimated to remain on the skin after wiping was 2.00×10^{-3} cm. Given an density of 1.1 g/cm^3 of asphalts, with sealcoat containing 30% asphalt by weight (w/w) and waterproofing product containing 75% w/w, and with an exposed skin surface area of 227.5 cm^2 (one quarter of each hand), the mass of asphalt in contact with the skin was estimated to be 149.8 and 375.5 mg, respectively. Using the selected body weight of 70.9 kg (considered to be representative of an average Canadian adult; Health Canada 1998), the dermal dose was therefore estimated to be 2.1 and 5.3 mg/kg-bw, respectively. These estimates are considered to represent exposures that might occur under normal conditions of product use, and to occur yearly.

Oral Exposure to Components of Asphalts via House Dust

Asphalts are components of some pavement sealants in Canada that can be applied to residential driveways or small commercial or residential parking lots. A study conducted in Texas analyzed the PAH content in dust found inside 23 ground-floor apartments that had different parking lot surfaces (Mahler et al. 2010). Twelve of the lots were unsealed asphalt or concrete or asphalt sealed with an asphalt-based sealant, and the rest were surfaces that had been sealed with a coal tar-based sealant. The concentration of total PAHs found in the indoor dust samples were calculated as the sum of the 16 US EPA priority pollutant PAHs. The concentrations of PAHs were significantly lower in apartment dust associated with asphalts or concrete surface lots and those sealed with asphalt-based sealant compared with those lots sealed with a coal tar-based sealant (median concentrations of 5.1 vs. 129 µg/g, respectively; Table 9-1). Of 17 variables examined in the apartments, pavement surface type was found to be the most dominant single factor affecting the concentration of PAHs in indoor dust, accounting for 48% of the variance in PAH concentration.

Table 9-1. Median concentrations in dust (µg/g) from 23 ground-floor apartments in Austin, Texas (adapted from Mahler et al. 2010)

Substance	Parking lots with asphalt or concrete surface (µg/g)[a]	Parking lots with surface sealed with a coal tar-based sealant (µg/g)
Total PAH[b]	5.1	129
Benzo[a]pyrene	0.44	4.5

[a] This grouping includes unsealed asphalt, asphalt-based sealcoated asphalt and unsealed concrete.
[b] Determined as the sum of the 16 priority pollutant PAHs identified by the US EPA (see Table in Appendix H).

For characterizing risk from exposure to indoor dust associated with asphalt driveways/lots, the average concentration of each of the 16 PAH species identified in the asphalt/concrete samples in Mahler et al. (2010) were used (see Appendix I). It is assumed that each daily dust exposure would contain the same concentration of PAHs.

Supplementary material from Mahler et al. (2010) also shows that the PAH concentration varies depending on the type of asphalt used for the driveway/lot. In house dust, the PAH concentration ranged from 0.06 μg/g (for unsealed asphalt driveways) to 3.91 μg/g (for asphalt-based sealants). For samples taken from ground floor apartments with parking lots, PAH concentrations similarly ranged from 0.06 μg/g for unsealed asphalt to 3.36 μg/g for asphalt-based sealants (Mahler et al. 2010).

Research conducted with support by the US EPA at the University of New Hampshire on simulated seal-coated surfaces resulted in similar findings to those of Mahler et al. (2010). Pavement dust collected from coal tar-based seal-coated surfaces was found to contain up to 1192 mg/kg total PAHs, compared to less than 2 mg/kg collected from unsealed asphalt surfaces (UNHSC 2010).

9.2 Health Effects Assessment

9.2.1 Basis for Categorization

The International Agency for Research on Cancer (IARC) concluded that there was *sufficient evidence* for the carcinogenicity of extracts of steam-refined asphalts, air-refined bitumens (in Europe 'bitumen' describes 'asphalt cement' or 'asphalt binder') and pooled mixtures of steam- and air-refined asphalts in experimental animals, and extracts of steam-refined and air-refined bitumens were classified as possible human carcinogens (Group 2B); there was *limited evidence* for the carcinogenicity of undiluted steam-refined bitumens and for cracking-residue asphalts in experimental animals; there was *inadequate evidence* for the carcinogenicity of undiluted air-refined asphalts in experimental animals. There was *inadequate evidence* that asphalts alone were carcinogenic to humans (IARC 1998). In a recent evaluation of occupational exposures to asphalt IARC (2011) assigned Group 2A to "bitumens, occupational exposure to oxidized bitumens and their emissions during roofing" (for CAS RN 64742-93-4) and Group 2B to both "bitumens, occupational exposure to hard bitumens and their emissions during mastic asphalt work" (no CAS RN assigned) and "bitumens, occupational exposure to straight-run bitumens and their emissions during road paving" (for CAS RNs 8052-42-4 and 64741-56-6).

9.2.2 Summary of Health Effects

Appendix I summarizes health effects information for asphalts, including carcinogenicity, the no- and lowest-observed-(adverse)-effect levels/concentrations (N/LO(A)EL/Cs), and corresponding critical effects. Select studies are summarized below.

Asphalt has low acute toxicity, as an oral LD_{50} value was not established with exposures of 5000 mg/kg-bw in rats, an LC_{50} value was not established with inhalation exposure of 94.4 mg/m^3 over 4.5 hours, and a dermal LD_{50} was not established with exposure of 2000 mg/kg-bw in rabbits (API 1982; Fraunhofer 2000). In the latter study, a 24-hour occluded exposure on abraded skin with animal monitoring to 14 days did not reveal significant adverse effects (API 1982). Skin irritation was not observed with acute

dermal exposures to vacuum residues (CAS RN 64741-56-6) (API 1982). Slight eye irritation was observed for these samples, as well as for undefined asphalt dust (straight or mixed with street dust or inert dust) (API 1982, b; Truc and Fleig 1913).

Repeated dose inhalation studies have used asphalt fume condensate as a source for generating exposures. A LO(A)EC of 100 mg/m^3 was identified in rats after nose-only exposure for 28 days, based on changes in lung histology parameters (Fraunhofer 2009). The same authors showed a NOAEC for reproductive and developmental toxicity with exposures of 300 mg/m^3. A LO(A)EC of 149 mg/m^3 was identified in rats based on degenerative lesions in nasal and paranasal cavities after 14 weeks exposure (Fraunhofer 2001). In a chronic nose-only exposure study in rats, a LO(A)EC of 34.4 mg/m^3 was identified based on a statistically significant decrease in body weight (Fuhst et al. 2007).

In short-term dermal studies in rabbits, a LO(A)EL of 2000 mg/kg-bw per day was identified for CAS RN 64741-56-6 based on significantly reduced body weight gain in New Zealand White rabbits. Exposures to two different samples of vacuum residues were via abraded and occluded dorsal skin at 200, 1000 and 2000 mg/kg-bw, 6 hours per application, three times weekly for 4 weeks. Reduced body weight gain was seen for animals exposed to only one of the two similar test substances (API 1983a, b).

The International Agency for Research on Cancer (IARC) concluded there is *inadequate evidence* that asphalts are carcinogenic to humans (IARC 1998). However, there is *sufficient evidence* in animals for the carcinogenicity of extracts of steam-refined asphalts, air-refined bitumens and pooled mixtures of steam- and air-refined asphalts. Extracts of steam-refined and air-refined bitumens were classified as possible human carcinogens (Group 2B). There is *limited evidence* for the carcinogenicity of undiluted steam-refined bitumens and for cracking-residue asphalts in animals and there is *inadequate evidence* for the carcinogenicity of undiluted air-refined asphalts.

The potential for dermal carcinogenicity of asphalt has been investigated in multiple skin painting studies using whole asphalts of different grades, or asphalt fume condensates. Of 218 Swiss mice exposed to different samples of 10% whole asphalt in benzene, 6 developed skin tumours for an overall tumour incidence of 2.7% (Wallcave et al. 1971). Tumour incidences of 2.55% and 14.3% were observed in a study that exposed mice to either 40% of straight or cracked asphalt, respectively (Kireeva 1968). Road and roofing asphalts (diluted with acetone) showed carcinogenic activity in mice but not in rabbits (Hueper and Payne 1960). Another study exposed 50 mice to steam- or air-refined asphalts, resulting in 3 and 9 total skin tumours, respectively (Simmers 1965). A lack of carcinogenic activity was reported in a study that exposed male C3H mice to 30% whole asphalt in mineral oil (McGowan et al. 1992).

Asphalt fumes generated in laboratories under high temperature have been shown to produce skin tumours in mice (see Appendix I). However, it has been demonstrated that laboratory-generated asphalt fumes are compositionally different from the fumes generated by road workers (McCarthy et al. 1999; Reinke et al. 2000). For example, in

the lab, asphalts are heated to higher temperatures under vacuum and with continuous agitation for significantly longer periods of time in order to generate a sufficient yield of fumes for exposure experiments (Niemeier et al. 1988). These methods alter the chemical composition of the fumes, resulting in higher concentrations of volatiles, thus reducing the relevance of any testing results to exposure to fumes that are generated under different conditions by road workers (McCarthy et al. 1999).

Only limited evidence for carcinogenicity has been derived from studies assessing inhalation to "real world" asphalt fumes. SPF-Wistar rats did not exhibit an increase in tumours from nose-only exposure to 4, 20 and 100 mg/m^3 total hydrocarbons as derived from paving asphalt fume condensate (obtained from the overhead space of a hot storage tank of semi-blown paving asphalt) (Fuhst et al. 2007). However, a poorly differentiated adenocarcinoma of the nasal cavity was observed in a single male of the high-dose group, and therefore a link with asphalt fume exposure could not be ruled out (Fuhst et al. 2007). In another study, one bronchial adenoma was observed in C57 black mice exposed to fumes from a pooled asphalt sample heated to 120^{o}C (Simmer 1964). However, no lung cancer developed in Besthesda black rats and Strain-13 guinea pigs exposed to fumes from a roofing asphalt (Heuper and Payne 1960). Some animals did develop chronic fibrosing pneumonitis with peribronchial adenomatosis.

The available *in vivo* genotoxicity tests on asphalt and asphalt fumes had mixed results. Oral exposure to vacuum residuum did not cause chromosome damage in Sprague-Dawley rats (API 1984c, d). Nose-only exposure to asphalt fume condensates under realistic fume generation and exposure conditions did not induce cytogenic damage in the respiratory or hematopoetic systems of Wistar rats (Fraunhofer 2009). At the highest dose level, asphalt fume condensates (collected from the top of a paving storage tank at a temperature of 160^{o}C) caused increased micronuclei after intra-tracheal instillation, but also caused cytotoxicity at this dose (Ma et al. 2002). In another study, DNA adducts were seen in lung cells, but not in leukocytes obtained via cardiac puncture, in male CD rats exposed to condensed asphalt fumes that were generated at 316^{o}C (Qian et al. 1998). DNA adducts were not seen in transgenic mice and rats exposed to total particulate paving asphalt fumes that were generated at 170^{o}C (Micillino et al. 2002; Bottin et al. 2006). Single-strand DNA breaks were seen in Sprague Dawley female rat alveolar macrophages and lung tissue, but micronucleus formation was not apparent in bone marrow polychromatic erythrocytes (Zhao et al. 2004).

Whole asphalts were generally non-mutagenic or weakly mutagenic in *in vitro* mouse lymphoma and Ames assays (API 1984c, d; Robinson et al. 1984; Monarca et al. 1987). Asphalt fumes collected at approximately 146 to 157^{o}C from the headspace of an asphalt storage tank at a hot-mix asphalt production plant were not mutagenic in the modified Ames assay, whereas fume condensates generated in the laboratory at 149^{o}C and 316^{o}C were mutagenic (Reinke et al. 2000). The mutagenicity of asphalt fume condensates also varied with the origination of crude oil feedstock, but the effect of the generation temperature on mutagenicity could not be determined (Machado et al. 1993). DNA adducts have also been demonstrated *in vitro* with asphalt fume condensates generated using laboratory conditions (DeMéo et al 1996; Akkineni et al. 2001).

Epidemiology studies on asphalts provide limited evidence on the carcinogenicity of asphalts. Roofers have shown an excess of lung cancer, but it is uncertain whether this excess was related to asphalts or to known carcinogens such as coal tar or asbestos (Hammond et al. 1976; Menck and Handerson 1976; NIOSH 2000), although IARC has indicated that the occupation 'roofing' when using asphalt is probably carcinogenic to humans (Group 2A) (IARC 2011). Some epidemiologic studies of road pavers exposed to asphalt fumes reported an elevated risk for lung cancer. However, design limitations of these studies confound the results, and include smoking, and likely exposure to coal tar, traffic exhaust, diesel exhaust, silica and asbestos (Darby et al. 1986; Burgaz et al. 1988; Gunkel 1989; Pasquini et al. 1989; Hansen 1991; NIOSH 2000). There are several studies that show cytogenetic effects in blood cells of asphalt field and paving workers, including micronuclei, sister chromatid exchange and DNA strand breaks (Burgaz et al. 1998; Murray and Edwards 2005; Cavallo et al. 2005). Other studies do not reveal genotoxic effects including a negative result for sister chromatid exchange in paving workers (Cavallo et al. 2005). The results from these studies typically derive from small sample size populations, and are confounded by co-exposure to other materials such as coal tar fumes, diesel exhaust and asbestos, as well as lifestyle factors (e.g., grilled and smoked food intake).

9.2.3 Minor High-hazard Components of Asphalts

9.2.3.1 Polycyclic Aromatic Hydrocarbons

Although asphalts are known to contain only a low level of PAHs, PAH species have a wide carcinogenic potency range. IARC (2010) recently reviewed the carcinogenicity data in experimental animals for 60 PAHs: 13 have sufficient evidence, 16 have limited evidence and 31 have inadequate evidence. Some PAHs, including B[*a*]P are classified as Category 1B carcinogens 'may cause cancer' by the European Commission (2008a). PAH species that have sufficient toxicological information can be ranked according to toxicological potency relative to B[*a*]P (Appendix J).

The Government of Canada previously completed a human health risk assessment of certain PAHs, including B[*a*]P, under the Priority Substances List Program. Based primarily on the results of carcinogenicity bioassays in animal models, five PAHs were considered "probably carcinogenic to humans", substances for which there is believed to be some chance of adverse effects at any level of exposure (Environment Canada, Health Canada 1994). PAHs were added to the List of Toxic Substances in Schedule 1 of CEPA.

The US EPA previously identified PAHs that may be carcinogenic in animals and humans (US EPA 1992b), ultimately listing 16 substances that became known as the priority pollutant PAHs (Menzie et al. 1992; US EPA 2013). They are naphthalene, acenaphthylene, acenaphthene, fluorene, phenanthrene, anthracene, fluoranthene, pyrene, benz[*a*]anthracene, chrysene, benzo[b+*j*]fluoranthene, benzo[k]fluoranthene, benzo[a]pyrene, indeno[1,2,3-cd]pyrene, dibenz[*a,h*]anthracene and benzo[ghi]perylene.

Several authors have created toxic or potency equivalency factors (PEFs) for numerous PAH species (i.e., estimates of their carcinogenic potency relative to that of B[a]P). PEFs as developed by Nisbet and LaGoy (1992) were used in this assessment (Appendix J). The PEF approach to estimating carcinogenic potency is an accepted method for human health risk assessment of PAH-rich mixtures, and has been widely used by several international organizations (EC 2008b; WHO 1998, 2001). The PEF approach has been adopted herein as a method to characterize the theoretical carcinogenic risk from oral exposure to PAH-containing house dust. It is noted that the empirical evidence to date is inconclusive for the carcinogenicity of asphalts (CAS RN 8052-42-4 and 64742-93-4) as whole substances.

In 2015, Health Canada used the B[*a*]P-specific tumour data in mice provided in Culp et al. (1998) to derive a lower limit of a one-sided 95% confidence interval of a carcinogenic benchmark dose for B[*a*]P (Health Canada 2015). This reference value, referred to as an oral $BMDL_{10}$, was calculated to be 0.545 mg/kg-bw/day, and is based on forestomach papillomas and/or carcinomas in female B6C3F1 mice (Health Canada 2015). This is of the same order of magnitude as oral $BMDL_{10}$ values for B[a]P derived by other groups, including the WHO. Using tumour data based on exposure to coal tars in mice, JECFA calculated a $BMDL_{10}$ range for B[*a*]P of 0.1-0.23 mg/kg-bw/day (FAO/WHO 2006). Wester et al. (2012) recently proposed 3 mg/kg-bw/day based on hepatocellular carcinomas in B[*a*]P-exposed Wistar rats. Health Canada's Guidelines for Canadian Drinking Water Quality proposed a maximum acceptable daily intake concentration of 0.04 µg/L for B[a]P (Health Canada, 2015).

Additionally, the Goverrment of Canada previously developed estimates of carcinogenic potency associated with the inhalation of B[*a*]P. A tumourigenic dose (TD_{05}) was calculated to be 1.57 mg/m^3 based on respiratory tract tumours in laboratory animals (Thyssen et al. 1981), using a multi-stage model (Environment Canada, Health Canada 1994).

9.2.3.2 Benzene

In the initial stages of asphalt processing, it may contain low levels of the high-hazard component benzene; however, upon processing, handling and heating, benzene is lost through the vapour phase of asphalts. Benzene has been assessed by the Government of Canada (Environment Canada, Health Canada 1993), was determined to be a carcinogen and was added to the *List of Toxic Substances* in Schedule 1 of CEPA. International organizations have drawn similar conclusions; IARC classifies benzene as Group 1 (*carcinogenic to humans*) (IARC 2012). The Government of Canada has previously developed estimates of carcinogenic potency associated with inhalation exposure to benzene. A 5% tumourigenic concentration (TC_{05}) for benzene was calculated to be 14.7×10^3 µg/m^3 (Environment Canada, Health Canada 1993) from the epidemiological investigation of Rinsky et al. (1987), based on acute myelogenous leukaemia in Pliofilm workers. The TC_{05} value is the air concentration of a substance associated with a 5% increase in incidence or mortality due to tumours (Environment Canada, Health Canada 1993). Reference values for benzene from other international

agencies (US EPA 2000, WHO 2000) are similar to the TC_{05} used below in this screening assessment for the characterization of risk to human health.

9.2.3.3 Naphthalene

A non-cancer health effects database for naphthalene was available and considered. Naphthalene was previously assessed by the Government of Canada (Environment Canada, Health Canada 2008), and a LO(A)EC of 7.86 mg/m^3 was considered based on point-of-contact nasal olfactory epithelium injury in mice after a two-hour exposure (Phimister et al. 2004). This effect was also seen in repeated dose studies in rats at 5 and 10 mg/m^3 (EURAR 2003), and these effect levels have recently been supported by other studies (Dodd 2010, 2012; Cichocki 2014). Other low dose studies were also considered (Clewell 2014). As naphthalene is predominant in heated asphalt vapour (Osborn 2013) and has a low effect level, the short-term critical effect level of 5 mg/m^3 was selected to characterize risk of potential transient inhalation exposure of bystanders to volatiles from hot asphalt paving.

9.3 Characterization of Risk to Human Health

A critical health effect for the initial categorization of asphalts were carcinogenicity, based on classifications by international agencies. IARC classifies asphalts as having insufficient evidence for carcinogenicity, however asphalt extracts were considered to range from limited to sufficient evidence for carcinogenicity, depending on type of extract (i.e., undiluted air-refined asphalts, undiluted steam-refined asphalts, extracts of steam-refined, air-refined asphalts and pooled mixtures of steam- and air-refined asphalts).

The characterization of risk to human health involves the consideration of exposure scenarios that are considered to present the highest exposures of the general population to asphalts. This includes transient inhalation exposure of bystanders during paving operations, short-term dermal exposure from the use of coating products, long-term inhalation exposure from living in the vicinity of asphalt plants and refineries, and long-term oral exposure to house dust that contains asphalt components as derived from driveway sealant.

9.3.1 Short-term inhalation exposure from asphalt paving operations

Transient inhalation exposure to asphalts for the general population may occur during professional application of asphalts to roads, roofs, and other surfaces. The largest occupational breathing zone concentration from these activities was 1.59 µg/m^3 based on exposure to naphthalene, and this level exceeded those of other measured asphalt components by an order of magnitude (Osborn 2013). This concentration was therefore considered to be a worst-case exposure level for the general population in the vicinity of these types of activities, given the general public is further from the emissions source. Compared with a critical effect level for naphthalene of 5000 µg/m^3 (based on local effects in the nasal olfactory epithelium of rats), the resulting margin of exposure (MOE)

of 3140 is considered adequate to address uncertainties in exposure and health effects databases. This scenario is considered to cover other potential transient inhalation exposures to asphalt vapours including those that might occur for bystanders from the transportation of asphalts in open-top trucks, from roofing operations, and from consumer use of asphalt-containing consumer products.

9.3.2 Short-term dermal exposure to consumer products containing asphalt

Incidental dermal exposure to the asphalt component from the application of a driveway sealant was estimated using a "thin film" approach (US EPA 2011c). For an average exposure that is considered to occur under normal use conditions, the dermal exposure to asphalt was determined to be 5.33 mg/kg-bw. This type of exposure is considered to occur infrequently at once per year. In rabbit studies of dermal exposure to whole asphalts, no adverse effects were noted for a single exposure to 2000 mg/kg-bw, nor for exposures three times per week for four weeks to 200, 1000 or 2000 mg/kg-bw (reduced body weight gain was seen in the latter study at the highest dose). Given the low dose and infrequent nature of general population dermal exposures to asphalt, and the lack of toxicity of asphalts in short-term animal studies, incidental exposure to consumer products containing asphalts is not considered to constitute a risk to human health. Additionally, given the physical-chemical properties of asphalts (high molecular weight and high log K_{ow}), dermal absorption is expected to be low.

9.3.3 Long-term inhalation exposure in the vicinity of asphalt plants / refineries

For this scenario, the general population may be exposed to fugitive releases (i.e., volatile components of asphalts) that may occur during asphalt processing, handling and storage at asphalt plants and refineries. Exposures to component substances that are known to be present in asphalt in the initial stages of processing (benzene and PAHs), and that are released during asphalt production, were estimated using measured emissions data combined with dispersion modelling. Conservative assumptions were used in order to characterize risk for potential long term exposure to asphalt volatiles due to living in the vicinity of asphalt plants or refineries.

An estimate of the carcinogenic potency for benzene (TC_{05}), previously developed by the Government of Canada (Environment Canada, Health Canada 1993), was used to calculate MOEs associated with long term benzene exposure due to asphalt emissions at facilities that produce, handle and store asphalts. The resulting MOEs for this exposure scenario are presented in Table 9-2

Table 9-2. MOEs for long term benzene exposure in the vicinity of asphalts facilities

Plant Type	**Batch Mix**	**Drum Mix**
Benzene exposure concentrations ($\mu g/m^3$) (at 230 m^a)	0.0183	0.0505
TC_{05} for benzene ($\mu g/m^3$)	14 700	14 700
MOEs	803 000	291 000

[a] 230 metres was determined to be the distance associated with the highest exposures

The MOEs (291 000 and 803 000) for potential long-term inhalation exposure to benzene emissions from asphalt at asphalt plants are considered adequate to address uncertainties related to the health effects and exposure databases. The air concentrations attributed to asphalt-derived benzene at 230 metres from the facility are significantly below the background level of benzene found in ambient air in Canada (0.32-0.84 µg/m^3) (NAPS 2012).

Releases of PAHs to air are also associated with the production and refinement of asphalts. For the purposes of characterizing risk for potential long-term inhalation exposure to asphalt-derived PAHs, the PAH concentrations determined at 230 metres (the distance associated with the highest levels of exposure as determined by dispersion modelling) were converted into B[*a*]P equivalents using potency equivalence factors (PEFs) as developed by Nisbet and LaGoy (1992) (see Table Appendix H).

Based on measured emissions quantity data from the US EPA (2000) for asphalt plants, PAH concentrations in air at 230 metres were determined and their sum potency was 0.0105 ng/m^3 of B[*a*]P equivalents (see Table 9-3).

Table 9-3. Air concentrations of PAH species in the vicinity of an asphalt plant at 230 m from release source

Compound	**Annual Concentration at 230 m[a] (ng/m^3)**	**Potency Equivalency Factor**	**B[a]P Equivalents (ng/m^3)**
Benzo[a]anthracene	0.0528	0.1	0.00528
Benzo[a]pyrene	0.0023	1	0.0023
Benzo[b]fluoranthene	0.0193	0.1	0.00193
Benzo[k]fluoranthene	0.0005	0.1	0.00005
Dibenzo[a,h]anthracene	0.000159	5	0.000795
Indeno[1,2,3-c,d]pyrene	0.001204	0.1	0.0001204
Total Benzo[a]pyrene Equivalents (ng/m^3)			0.0105

[a] 230m was determined to be the distance at which maximum PAH concentration was achieved in air based on dispersion modelling using the exposure factors in Appendix G.

An estimation of potential long-term exposure to PAHs at 230 m from asphalt production, handling and storage was therefore determined to be 0.0105 ng/m^3 B[*a*]P equivalents. This value is lower, by over an order of magnitude, than the average Canadian background concentration of B[*a*]P in ambient air (0.14 ng/m^3) (NAPS 2012). Compared with the TD_{05} for B[*a*]P of 1.57 mg/m^3 (Environment Canada, Health Canada 1994), the resulting MOE is 149 500 000 for long-term exposure in the vicinity of asphalt plants. This margin is considered adequate to address uncertainties in the exposure and health effects databases.

Long-term oral exposure to asphalt-derived PAHs found in house dust

For the purpose of characterizing risk for long-term exposure to asphalt-derived PAHs found in house dust (that are assumed to migrate indoors over time from the wearing of asphalt-based driveway/parking lot sealants), the concentration of each of 16 PAH species found in 10 samples of ground floor apartment dust with associated asphalt-based parking lots was considered (Mahler et al. 2010). It was conservatively assumed that these levels of PAHs were derived solely from asphalt surfaces (i.e., ignoring non-asphalt sources that might contribute to PAHs in house dust). The average PAH level found in the dust samples for each of the 16 PAHs was converted into a B[*a*]P equivalent using the Nisbet and LaGoy (1992) PEFs (Appendix H). The combined potency across the 16 PAH species resulted in a total average B[*a*]P equivalents in dust of 3.18 µg/g (see Table F-5 in Appendix F for more details). Therefore, exposure to house dust was considered to carry an associated oral exposure to B[*a*]P of 3.18 µg per gram of dust. For the purposes of conducting a combined dust and soil exposure risk characterization, this concentration of B[*a*]P was considered to exist in soil, and to be 100% bioavailable. Conservatism is built in from assuming 100% bioavailability for every exposure and by attributing all PAHs in the dust to asphalts.

Average combined soil and dust ingestion rates were adapted from the work of Wilson (2013) for each age group. A daily exposure estimate to B[*a*]P equivalents (i.e., representing asphalt-derived PAHs) was derived for each age group based on the ingestion of soil and indoor dust, and assuming that the soil and dust contains 3.18 µg/g of B[*a*]P. Soil and dust and B[*a*]P intakes for each age group are presented in Table 9-4

Table 9-4. Soil and dust and B[a]P-equivalent intakes by age group

Age Group (year)	0 - 0.5	0.5 - 4	5 - 11	12 - 19	20 - 59	60+
Soil and Dust Intake Rates (mg/day)	38	55	52	3.6	4.1	4.0
Intake mass of B[a]P equivalents[a] (µg/day)	0.12	0.17	0.17	0.01	0.01	0.01
Body weights (kg)	7.5	15.5	31.0	59.4	70.9	72.0
Dose of B[a]P equivalents (µg/kg-bw/day)	0.01611	0.01128	0.00533	0.00019	0.00018	0.00018
Time Weighted Intake relative to lifespan (µg/kg-bw/day)	0.00011	0.00056	0.00053	0.00002	0.00010	0.00003
Lifetime Average Daily Dose	0.00135					

(µg/kg-bw/day)	

[a] Average concentration of B[*a*]P equivalents in dust considered to be 3.18 ng/mg

The average daily dose of asphalt-derived PAHs (i.e., in B[*a*]P equivalents) over a lifetime was determined to be 0.00135 µg/kg-bw. Young children ingest the greatest mass of soil and dust due to touching surfaces that accumulate dust and their extensive hand-to-mouth action (Van Metre 2013).

In characterizing risk for a lifetime of exposure, US EPA age-dependent adjustment factors (US EPA 2011c) were adjusted to the Health Canada age bins and applied to each age group (see Appendix F). These factors were used to account for the sensitivity of each life stage; an MOE for long term exposure was then determined (see Appendix F). The point of departure for determining a lifetime MOE from a lifetime of daily exposure to soil and dust containing asphalt-derived PAHs was the lower limit of a one-sided 95% confidence interval ($BMDL_{10}$) for B[*a*]P. This was calculated by Health Canada to be 0.545 mg/kg-bw/day (Health Canada 2015). The daily B[*a*]P intakes for each age group were determined, were compared with this endpoint, and the MOE for each age group was adjusted based on age sensitivity using the adjustment factors fit to Health Canada's age bins. The resulting lifetime MOE is 99 150, considering a 70 year lifespan (equaling a sum total of 71 years of exposure), and factors in age group sensitivities and differences in daily exposure per age group. This MOE is considered adequate to address uncertainties in the exposure and health effects databases.

Given the possibility of a higher 'one-time' oral exposure to soil for infants and toddlers, the short-term risk from a potential higher oral exposure to asphalt-derived PAHs from the ingestion of soil was considered for toddlers. Based on the considerations outlined above, ingestion of 1 gram of soil for a toddler would result in an oral exposure to 3.18 µg B[*a*]P equivalents (0.21 µg B[*a*]P/kg-bw). Given that the ingested dose is low (on the order of low parts per billion relative to body weight), that PAHs are only partially bioavailable when adsorbed to soil, the short-term nature of the exposure, and the limited health endpoints available for characterizing short-term oral exposures to PAHs, risk to human health is considered to be low.

9.4 Uncertainties in Evaluation of Human Health Risk

Because asphalt and oxidized asphalt are UVCBs, their specific compositions are only broadly defined and vary from batch to batch. The proportion of component substances can change depending on operating conditions, feedstocks and processing units; however, given the nature by which asphalts are produced, volatile and light molecular weight substances such as benzene and 2-3 ring PAHs are expected to be consistently present only at very low levels.

Also, due to limited detailed information on the composition of asphalts, characterization of exposure and risk focussed on the known component substances that pose the highest hazard to human health, as well as on the component substance with the

potential for highest short-term inhalation exposure (i.e., naphthalene). Additionally, dermal exposure to whole asphalts was assessed. However, exposure to other component substances of asphalts may occur that were not considered in the inhalation and oral ingestion scenarios, and these substances may have associated health effects. The margins developed for the high exposure and high hazard substances are considered to account for potential exposure to other less well-defined component substances of asphalts.

There is uncertainty in the level of B[*a*]P equivalents contained in home dust and soil, as the concentration is based on ten dust samples from ground floor apartments in Texas. Additional PAH compositional analysis of dust and soil from Canadian homes would result in a better understanding of the range of PAH concentrations found here, and could be assessed across a variety of housing types and ages. Also, refining the percent attribution of PAHs that are found in house dust (from 100%) that originate from asphalts would result in more precise estimates of contribution of asphalts to PAHs in dust, but assuming 100% attribution is conservative for the purpose of assessing risk to human health.

10 Conclusion

Considering all available lines of evidence presented above including studies of releases from asphalt formulations used for paving and roofing, there is low risk of harm to organisms or the broader integrity of the environment from these substances. It is concluded that asphalt and oxidized asphalt (CAS RNs 8052-42-4 and 64742-93-4) do not meet the criteria under paragraphs 64(*a*) or (*b*) of CEPA as they are not entering the environment in quantities or concentrations 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.

The general population may be exposed to asphalts through product use, or may be exposed to minor components of asphalts that are found in soil and house dust, or that are released during professional asphalt paving, roofing or transportation activities, or by asphalt facilities. However, exposure to asphalts and to minor high-hazard components of asphalts are low and therefore risk is considered to be low.

Based on the information presented in this screening assessment, it is concluded that asphalt and oxidized asphalt (CAS RNs 8052-42-4 and 64742-93-4) do not meet the criteria under paragraph 64(*c*) of CEPA 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 concluded that asphalt and oxidized asphalt (CAS RNs 8052-42-4 and 64742-93-4) do not meet any of the criteria set out in section 64 of CEPA.

References

Akkineni LK, Zeisig MB, Baranczewski P, et al. 2001. Formation of DNA adducts from oil-derived products analyzed by 32P-HPLC. Arch. Toxicol. 74: 720-731.

[API] American Petroleum Institute, 1983a. 28-day dermal toxicity study in the rabbit. Vacuum residuum API sample 81-13. Med. Res. Publ. 30-32852. Washington (DC): American Petroleum Institute.(cited in IUCLID 2000; API 2006)

[API] American Petroleum Institute, 1983b. 28-day dermal toxicity study in the rabbit. Vacuum residuum API sample 81-14. Med. Res. Publ. 30-32853 Washington (DC): American Petroleum Institute.. (cited in IUCLID 2000; API 2006)

[API] American Petroleum Institute. 1982. Acute studies of vacuum residuum. API 81-13. Study conducted by Hazleton Raltech Inc. Report No. 30-31987 Washington (DC): American Petroleum Institute.. (cited in API 2006)

[API] American Petroleum Institute. 1984a. Dermal sensitization study in guinea pigs. Closed patch technique. Vacuum residuum. API 81-13. Study conducted by Hazleton Laboratories America Inc. Report No. 31-31415. Washington (DC): American Petroleum Institute. (cited in IUCLID 2000)

[API] American Petroleum Institute. 1984b. Dermal sensitization study in guinea pigs. Closed patch technique. Vacuum residuum. API 81-14. Study conducted by Hazleton Laboratories America Inc. Report No. 31-31416. Washington (DC): American Petroleum Institute. (cited in IUCLID 2000)

[API] American Petroleum Institute. 1984c. Mutagenicity evaluation studies in the rat bone marrow cytogenetic assay and in the mouse lymphoma forward mutation assay. Vacuum residuum PAP Sample 81-13. Washington (DC): American Petroleum Institute.

[API] American Petroleum Institute. 1984d. Mutagenicity evaluation studies in the rat bone marrow cytogenetic assay and in the mouse lymphoma forward mutation assay. Vacuum residuum PAP Sample 81-14. American Petroleum Institute Res. Rep. 31-30615. Washington (DC): American Petroleum Institute.. (cited in IUCLID 2000)

[API] American Petroleum Institute. 2006. Robust summary of information on asphalt. Washington (DC): American Petroleum Institute. 52 p. Available from: http://www.petroleumhpv.org/pages/asphalt.html

[API] American Petroleum Institute. 2009. Asphalt Category Analysis and Hazard Characterization. Washington (DC): American Petroleum Institute. 54 p. Consortium Registration # 1100997. Submitted to the US EPA by the American Petroleum Institute Petroleum HPV Testing Group. Available from: http://www.petroleumhpv.org/pages/asphalt.html

Arnot JA, Gobas FA. 2003. A generic QSAR for assessing the bioaccumulation potential of organic chemicals in aquatic food webs. QSAR Comb Sci. 22:337-345.

Arnot JA, Gobas FA. 2006. A review of bioconcentration factor (BCF) and bioaccumulation factor (BAF) assessments for organic chemicals in aquatic organisms. Environ Rev 14(4):257-298.

Asphalt Institute. 2007. Asphalt handbook, MS-4. 7th ed. Lexington (KY): Asphalt Institute.

Azizian MF, Nelson PO, Thayumanavan P, Williamson KJ. 2003. Environmental impact of highway construction and repair materials on surface and ground waters: Case study: crumb rubber asphalt concrete. Waste Manag. 23:719-728.

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

Birgisdottir H, Gamst J, Christensen TH. 2007. Leaching of PAHs from hot mix asphalt pavements. Environ Eng Sci. 24(10):1409-1421.

Blackburn GR, Kriech AJ. 1990. Status report on industry-sponsored toxicology and chemical testing of asphalts and asphalt fume condensates. Indianapolis (IN): Heritage Research Group. (cited in IUCLID 2000)

Blokker EL, van de Ven BM, de Jongh CM, Slaats PGG. 2013. Health implications of PAH release from coated cast iron drinking water distribution systems in the Netherlands. Environ. Health Perspect. 121: 600-606.

Bottin MC, Gate L, Rihn B, Micillino JC, Nathalie M, Martin H, Nunge G, Wrobel R, Ayi-Fanou L, Champmartin C, Keith G, Binet S. 2006. Genotoxic effects of bitumen fumes in Big Blue(R) transgenic rat lung. Mutation Research/Fundamental and Molecular Mechanisms of Mutagenesis 596: 91-105.

Brandt HCA, de Groot PC. 2001. Aqueous leaching of polycyclic aromatic hydrocarbons from bitumen and asphalt. Water Res. 35(17):4200-4207.

Brantley AS, Townsend TG. 1999. Leaching of pollutants from reclaimed asphalt pavement. Environ Eng Sci. 16(2):105-116.

Burgaz S, Bayhan A, Karakaya AE. 1988. Thioether excretion of workers exposed to bitumen fumes. Int Arch Occup Environ Health 60: 347-349.

Burgaz S, Erden O, Karahalil, Karakaya AE. 1998. Cytogenetic biomonitoring of workers exposed to bitumen fumes. Mutat Res, 419:123-30.

Cai H, Huang X, Wang P, Zhang Y. 2009. Factors influencing the leaching of asphalt components. J Test Eval. 37(5):16.

Health Canada. 1998. Exposure Factors for Assessing Total Daily Intake of Priority Substances by the General Population of Canada. Unpublished Report. Ottawa (ON): Health Canada, Environmental Health Directorate.

Canada. 1999. *Canadian Environmental Protection Act, 1999*. S.C. 1999, c. 33. Act current to 2012-10-02 and last amended on 2012-09-20. Available from: http://laws-lois.justice.gc.ca/eng/acts/C-15.31/index.html

Cavallo D, Ursini CL, Bavazzano P, et al. 2005. Sister chromatid exchange and oxidative DNA damage in paving workers exposed to PAHs. Ann Occup Hyg 50: 211-218.

[CCIC] Canadian Construction Innovation Council. 2007. Enhanced recovery of roofing materials. Prepared by the Athena Sustainable Materials Institute.Ottawa (ON):Canadian Construction Innovation Counci..1–57.

[CCME] Canadian Council of Ministers of the Environment. 1999. Canadian water quality guidelines for the protection of aquatic life: polycyclic aromatic hydrocarbons (PAHs). Winnipeg (MB): Canadian

Council of Ministers of the Environment. 13 p. Available on-line [retrieved July 13, 2015]: http://ceqg-rcqe.ccme.ca/download/en/201

[Cheminfo] Cheminfo Services Inc. 2009. Background technical study on the use, exposure and release potential of certain high priority petroleum substances under the Chemicals Management Plan, in sectors other than the petroleum sector – asphalt. Final confidential unpublished report. Gatineau (QC): Environment Canada. 132 p. Solicitation Number: K2A15-08-0018-C.

Cichocki JA, Smith GJ, Mendoza R, Buckpitt AR, Van Winkle LS, Morris JB. 2014. Sex differences in the acute nasal antioxidant/antielectrophilic response of the rat to inhaled naphthalene. Toxicological Sciences 139: 234-244.

Clewell HJ, Efremenko A, Campbell JL, Dodd DE, Thomas RS. 2014. Transcriptional responses in the rat nasal epithelium following subchronic inhalation of naphthalene vapor. Toxicology and Applied Pharmacology 280: 78-85.

[CONCAWE] CONservation of Clean Air and Water in Europe. 1992. Bitumens and bitumen derivatives. Brussels (BE): CONCAWE Petroleum Products and Health Management Group. 44 p. Report No.: 92/104.

[CONCAWE] CONservation of Clean Air and Water in Europe. 2001. Environmental classification of petroleum substances - summary data and rationale. Brussels (BE): CONCAWE Petroleum Products Ecology Group. 138 p. Report No.: 01/54.

Cooper SD, Kratz KW. 1996. The impact of runoff from asphaltic products on stream communities in California. Santa Barbara (CA): Marine Science Institute, University of California. 113 p. Report No.: FHWA/CA/TL-96-24.

Culp SJ, Gaylor DW, Sheldon WG, Goldstein LS, Beland FA. 1998. A comparison of the tumors induced by coal tar and benzo[*a*]pyrene in a 2-year bioassay. Carcinogenesis. 19: 117-124.

Darby FW Willis AF, Winchester RV. 1986. Occupational health hazards from road construction and sealing work. Ann Occup Hyg. 30: 445-454.

De Méo M, Genevois C, Brandt H, Laget M, Bartsch H, Castegnaro M. 1996. In vitro studies of the genotoxic effects of bitumens and coal tar fumes condensates: comparison of data obtained by mutagenicity testing and DNA adduct analysis by ^{32}P-postlabeling. Chemico-Biological Interactions 101: 75-88.

Diamond Environmental Group. 2011. Reconnaisance study of coal tar sealcoat application in Toronto and an estimate of release. Toronto (ON): Diamond Environmental Group, University of Toronto. 32 p. Prepared for Environment Canada, Environmental Protection Operations Division, Ontario.

Dietz WA, Dudenbostel BF, Priestley W.. 1956. Properties of high boiling petroleum products: carcinogenicity studies. Ind Eng Chem 44:1818–1827. [cited in CONCAWE 1999; US EPA 2006].

Dodd DE, Gross EA, Miller RA, Wong BA. 2010. Nasal olfactory epithelial lesions in F344 and SD rats following 1 and 5-day inhalation exposure to naphthalene vapor. International Journal of Toxicology 29(2): 175-184.

Dodd DE, Wong BA, Gross EA, Miller RA. 2012. Nasal epithelial lesions in F344 rats following a 90-day inhalation exposure to naphthalene. Inhalation Toxicology 24: 70-79.

[EC] European Commission. 2001. Ambient air pollution by polycyclic aromatic hydrocarbons (PAH). Position Paper. Luxembourg (LU): Office for Official Publications of the European Communities. Available from: http://ec.europa.eu/environment/air/pdf/pp_pah.pdf

[EC] European Commission. 2008a. Details on substances classified in Annex VI to Regulation (EC) No 1272/2008 [Database on the Internet]. Ispra (IT): European Commission, Joint Research Centre, Institute for Health and Consumer Protection. [cited 2014] Available from: https://osha.europa.eu/en/legislation/directives/regulation-ec-no-1272-2008-classification-labelling-and-packaging-of-substances-and-mixtures

[EC] European Commission. 2008b. European Union risk assessment report. Coal-tar pitch, high temperature. CAS No.: 65996-93-2. EINECS No.:266-028-2. Risk assessment. Environment. May 2008. Luxembourg (LU): Office for Official Publications of the European Communities. [cited 2014] Available from: http://echa.europa.eu/documents/10162/433ccfe1-f9a5-4420-9dae-bb316f898fe1

Environment Canada, Health Canada. 1993.*Canadian Environmental Protection Act:* Priority Substances List assessment report: benzene. Ottawa (ON): Environment Canada; Health Canada. Available from: www.hc-sc.gc.ca/ewh-semt/alt_formats/hecs-sesc/pdf/pubs/contaminants/psl1-lsp1/benzene/benzene-eng.pdf

Environment Canada, Health Canada. 1994. *Canadian Environmental Protection Act*: Priority Substances List assessment report: polycyclic aromatic hydrocarbons. Ottawa (ON): Environment Canada; Health Canada. Report No.: C94-980182-8.

[ECCC, HC] Environment and Climate Change Canada, Health Canada. [modified 2007 Apr 20]. Categorization. Ottawa (ON): Government of Canada. [accessed 2015 Jul 02]. http://www.chemicalsubstanceschimiques.gc.ca/approach-approche/categor-eng.php.

Environment Canada, Health Canada. 2008. Screening assessment for the Challenge: naphthalene: Chemical Abstracts Service Registry Number 91-20-3. Ottawa (ON): Environment Canada; Health Canada. Available from: http://www.ec.gc.ca/ese-ees/default.asp?lang=En&n=F212515C-1#a11

Environment Canada. 2014. Technical document on the persistence and bioaccumulation potential of petroleum hydrocarbons. Gatineau (QC): Environment Canada, Ecological Assessment Division.

Environmental Health Strategies Inc. 2010. Technical and economic study on VOC (volatile organic compound) emissions from emulsified and cutback asphalt use in Canada. CBI confidential. Final unpublished report. Gatineau (QC): Environment Canada. 83 p.

[EPI Suite] Estimation Program Interface Suite [estimation model]. 2000. Version 3.10. Washington (DC): US Environmental Protection Agency, Office of Pollution Prevention and Toxics; Syracuse (NY): Syracuse Research Corporation. [as cited in API 2006]

[EPI Suite] Estimation Program Interface Suite for Microsoft Windows [estimation model]. c2000-2010. Version 4.10. Washington (DC): US Environmental Protection Agency, Office of Pollution Prevention and Toxics; Syracuse (NY): Syracuse Research Corporation. Available from: www.epa.gov/oppt/exposure/pubs/episuite.htm

[EURAR] European Union risk assessment report: CAS: 91-20-3: naphthalene. 2003. Luxembourg: Office for Official Publications of the European Communities. Report No.: EUR 20763 EN. On the cover, European Commission Joint Research Centre. On the cover, European Commission Joint Research Centre. [European Communities, European Commission, Joint Research Centre, Institute for Health and

Consumer Protection, European Chemicals Bureau]. Available from: https://echa.europa.eu/documents/10162/4c955673-9744-4d1c-a812-2bf97863906a.

[FAO/WHO] Food and Agriculture Organization of the United Nations / World Health Organization. 2006. Evaluation of Certain Food Contaminants: sixty-fourth report of the Joint FAO/WHO Expert Committee on Food Additives. WHO Technical Report Series: 930. Geneva: World Health Organization. [cited 2014] Available from: http://whqlibdoc.who.int/trs/WHO_TRS_930_eng.pdf

Fraunhofer ITA. 2000. Acute inhalation toxicity study of 100 mg/m bitumen fumes in Wistar (WU) rats Fraunhofer ITA Study No. 02G00012. (cited in API 2006)

Fraunhofer ITA. 2001. 13 week inhalation toxicity study of bitumen fumes in Wistar (WU) rats. Fraunhofer ITA study No. 02G01005. (cited in API 2009)

Fraunhofer ITA. 2009. Combined repeated dose toxicity study with the reproduction/developmental toxicity screening test and mammalian erythrocyte micronucleus test via inhalation with roofing asphalt fume condensate. Fraunhofer ITEM Study No. 02N07533. Sponsored by American Petroleum Institute. R. Fuhst, Study Director.

Fraunhofer Institute of Toxicology, and Experimental Medicine, Hannover, Germany. (cited in API 2009). Available from: http://www.petroleumhpv.org/~/media/petroleumhpv/documents/publications/range%20finding%20testing%20for%20a%20combined%20repeated%20dose%20toxicity%20study%20with%20the%20reproduction%20developmental%20toxicity%20screening%20test.pdf

Fuhst R, Creutzeberg O, Ernst H. Hansen T, Pohlmann G, Preiss A, Rittinghausen S. 2007. 24 Month inhalation carcinogenicity study of bitumen fumes in Wistar (WU) rats. J Occup Environ Hygiene 4: 20-43.

Gunkel KO'C. 1989. Study of paving asphalt fumes. Sponsored by US National Asphalt Paving Association. (cited in IUCLID 2000)

Gustafson JB, Griffith Tell J, Orem D. 1997. Total petroleum hydrocarbon criteria working group series vol. 3: Selection of representative TPH fractions based on fate and transport considerations. Amherst (MA): Amherst Scientific Publications.

Hammond EC, Selikoff IJ, Lawther PL, Seidman H. 1976. Inhalation of benzpyrene and cancer in man. Ann NY Acad Sci. 271: 116-124. Available from: http://onlinelibrary.wiley.com/doi/10.1111/j.1749-6632.1976.tb23100.x/abstract

Hansen ES. 1991. Cancer incidence in an occupational cohort exposed to bitumen fumes. Scand J Work Environ Health 15:101-105.

Health Canada. 2015. Benzo[a] pyrene in Drinking Water. Ottawa (ON): Health Canada, Federal-Provincial-Territorial Committee on Drinking Water. Available from: https://www.canada.ca/content/dam/hc-sc/migration/hc-sc/ewh-semt/alt_formats/pdf/consult/_2015/bap/consult-eng.pdf

Heuper WC, Payne WW. 1960. Carcinogenic studies on petroleum asphalt. Cooling oil and coal tar. Arch Pathol 70: 372-384.

[IARC] IARC Working Group on the Evaluation of Carcinogenic Risks to Humans. 1998. Polynuclear aromatic compounds, Part 4, bitumens, coal-tars and derived products, shale-oils and soots. Summary of data reported and evaluation. IARC Monogr Eval Carcinog Risks Hum. 35: 1-276.

[IARC] IARC Working Group on the Evaluation of Carcinogenic Risks to Humans. 2010. Some Non-Heterocyclic Polycyclic Aromatic Hydrocarbons and Some Related Exposures. IARC Monogr Eval Carcinog Risks Hum. 92: 1-868.

[IARC] IARC Working Group on the Evaluation of Carcinogenic Risks to Humans. 2011. Evaluation of occupational exposures to bitumen and bitumen emissions. IARC Monogr Eval Carcinog Risks Hum. 103: 1-361.

[IARC] IARC Working Group on the Evaluation of Carcinogenic Risks to Humans. 2012. Chemical Agents and Related Occupations. Benzene. IARC Monogr Eval Carcinog Risks Hum. 100F: 249-285.

[IPCS] INCHEM. IPCS International Programme On Chemical Safety. 2004. Concise International Chemical Assessment Document 59. ASPHALT (BITUMEN). Cincinnati (OH): World Health Organization. Available from: http://www.inchem.org/documents/cicads/cicads/cicad59.htm#1.0

Kireeva IS. 1968. Carcinogenic properties of coal-tar pitch and petroleum asphalts used as binders for coal briquettes. Hyg Sanit 33:180-186. (cited in IUCLID 2000)

Kleinschmidt LR. Snoke HR. 1958. Changes in the properties of an asphalt during the blowing operation. Journal of Research of the National Bureau of Standard. Volume 6 (3): 169-172.

[KOWWIN] Octanol-Water Partition Coefficient Program for Microsoft Windows [estimation model]. 2010. Version 1.68. Washington (DC): US Environmental Protection Agency, Office of Pollution Prevention and Toxics; Syracuse (NY): Syracuse Research Corporation. Available from: www.epa.gov/oppt/exposure/pubs/episuite.htm

Kriech AJ, Kurek JT, Osborn LV, Wissel HL, Sweeney BJ. 2002. Determination of polycyclic aromatic compounds in asphalt and in corresponding leachate water. Polycyclic Aromatic Compounds 22: 517-535.

Kriech AJ. 1990. Evaluation of hot mix asphalt for leachability. Indianapolis (IND): Heritage Research Group.

Kriech AJ. 1992a. Leachability of asphalt and concrete pavements. Indianapolis (IND): Heritage Research Group.

Kriech AJ. 1992b. Leachability of cold mix asphalts. Indianapolis (IND): Heritage Research Group.Kriech AJ, Kurek JT, Osborn LV, Wissel HL, Sweeney BJ. 2002. Determination of polycyclic aromatic compounds in asphalt and in corresponding leachate water. Polycyclic Aromat Compd. 22(3):517-535.

Legret M, Odie L, Demare D, Jullien A. 2005. Leaching of heavy metals and polycyclic aromatic hydrocarbons from reclaimed asphalt pavement. Water Res. 39:3675-3685.

[LNHPD] 2015. Licensed Natural Health Products Database. [database on the Internet]. Ottawa (ON): Health Canada. [cited 2015 June]. Available from: http://webprod5.hc-sc.gc.ca/lnhpd-bdpsnh/index-eng.jsp

Ma JYC, Yang HM, Barger MW, Siegel PD, Zhong BZ, Kiech AJ, Castranova V. 2002. Alteration of pulmonary cytochrome P-450 system, effects of asphalt fume condensate exposure. J Tox and Env Health, Part A, 65:1247-1260.

Machado ML, Beatty PW, Fetzer JC, Glickman AH, MCGinnis EL. 1993. Evaluation of the relationship between PAH content and mutagenic activity of fumes from roofing and paving asphalts and coal tar pitch. Fund. Appl. Toxicol. 21:492-499.

Mahler BJ, Ingersoll CG, Van Metre PC, Kunz JL, Little EE. 2015. Acute toxicity of runoff from sealcoat pavement to *Ceriodaphnia dubia* and *Pimephales promelas*. Environ Sci Technol. 49(8):5060-5069.

Mahler BJ, Van Metre PC, Bashara TJ, Wilson JT, Johns DA. 2005. Parking lot sealcoat: an unrecognized source of urban polycyclic aromatic hydrocarbons. Environ Sci Technol. 39:5560-5566.

Mahler BJ, Van Metre PC, Crane JL., Watts AW, Scoggins M, Williams ES. 2012. Coal-tar-based pavement sealcoat and PAHs: implications for the environment, human health, and stormwater management. Environ Sci Technol. 46: 3039-3045

Mahler BJ, Van Metre PC, Wilson JT, Musgrove M. 2010. Coal-tar-based parking lot sealcoat: an unrecognized source of PAH to settled house dust. Environ. Sci. Technol. 44: 894-900.

Mahler BJ, Van Metre PC, Wilson JT. 2004. Concentrations of polycyclic aromatic hydrocarbons (PAHs) and major and trace elements in simulated rainfall runoff from parking lots. Austin (TX): US Geological Survey.

Martec Recycling Corporation [internet]. 2016. Vancouver (BC): Martec Recycling Corporation. [cited 2016 Dec 09]. Available from: http://martec.ca/benefit/

McCarthy BM, Blackburn GR, Kriech AJ, Kurek JT, Wissel HL, Osborn LV. 1999. Comparison of field- versus laboratory-generated asphalt fumes. Transportation Research Board, 1661:54-59.

McGowan C, Daughtrey WC, Freeman JJ, McKee RH. 1992. Lack of carcinogenic and mutagenic activity with asphalt products, Abstract 1485. The Toxicologist 12:379. (cited in API 2006)

Meegoda JN. 1999. Stabilization/solidification of petroleum-contaminated soils with asphalt emulsions. Pract Period Hazard Toxic Radioact Waste Manage. 3:46-55.

Menck HR and Henderson BE. 1976. Occupational differences in rates of lung cancer. J Occup Med. 18: 797-801.

Menzie CA, Potocki BB, Santodonato J. 1992. Exposure to carcinogenic PAHs in the environment. Environ Sci Technol 26:1278-1284.

[Meridian] Meridian Environmental Inc. 2009. Development of MSDS database for petroleum products. Calgary (AB): Meridian Environmental Inc. Standing offer No. HT301-062111/001/XSB.

Micillino JC, Coulais C, Binet S, et al. 2002. Lack of genotoxicity of bitumen fumes in transgenic mouse lung. Toxicology 170: 11-20.

Miller RW, Honarvar S, Hunsaker B. 1980. Effects of drilling fluids on soils and plants: I. Individual fluid components. J Environ Qual. 9(4):547-552.

Monarca S, Pasquini R, Scassellati Sforzolini G, Savino A, Bauleo FA, Angeli G. 1987. Environmental monitoring of mutagenic/carcinogenic hazards during road paving operations with bitumens. Int. Arch. Occup. Environ. Health. 59: 393-402.

[MSDS] Material Safety Data Sheet. 2006. Marathon Anionic Emulsified Asphalt. Findley (OH): Marathon Petroleum Company LLC. [accessed 2016 Dec 12]. Available from: https://www.tdoteco.com/content/Guidance/MSDS/Marathon-Petroleum-Emuls-Asphalt-MSDS.pdf

MSDS] Material Safety Data Sheet. 2007a. CM-300, CM-200, CM-150, CM-90, HPCM-300. Indianapolis (IN): Asphalt Materials, Inc. [accessed 2015 Jan 14]. Available from: http://ebpaving.com/wp-content/uploads/2013/09/CM-90-CM-150-CM-300.pdf

[MSDS] Material Safety Data Sheet. 2007b. MC-30, MC-70, MC-250, MC-800, MC-3000. Indianapolis (IN): Asphalt Materials, Inc. [accessed 2016 Dec 12]. Available from: http://www.ebpaving.com/media/msds/asphalts/MC-30,%20MC-70,%20MC-250,%20ETC.pdf

Murray EB, Edwards JW. 2005. Differential induction of micronuclei in peripheral lymphocytes and exfoliated urothelial cells of workers exposed to 4,4'-methylenebis-(2-chloroaniline)(MOCA) and bitumen fumes, Rev. Environ. Health. 20:163-176.

[NAPA] National Asphalt Pavement Association—Engineering Overview [internet]. c2016. Lanham (MD): National Asphalt Pavement Association. [accessed 12 Dec 2016]. Available from: http://www.asphaltpavement.org/index.php?option=com_content&view=article&id=14&Itemid=33

[NAPS] National Air Pollution Surveillance Network. 2012. Data for PAHs in 2012. Gatineau (QC): Environment Canada.

[NAPS] National Air Pollution Surveillance Network. 2012. NAPS annual raw data for 2009. Gatineau (QC): Environment Canada.

[NHPID] Natural Health Products Ingredients Database. 2015. [database on the internet]. Ottawa (ON): Health Canada. [cited 2015 June]. Available from: http://webprod.hc-sc.gc.ca/nhpid-bdipsn/search-rechercheReq.do?lang=eng

Niemeier RW, Thayer PS, Menzies KT, vonThuna P, Moss CE. 1988. A comparison of the skin carcinogenicty of condensed roofing asphalt and coal tar pitch fumes. In: Cooke M, Dennis AJ, eds, Polynuclear aromatic Hydrocarbons: A decade of progress. 10th International Symposium on Polynuclear Aromatic Hydrocarbons. Battelle Press, Columbus, OH. 609-647. (cited in API 2009)

[NIOSH] National Institute for Occupational Safety and Health. 1981. Roofing asphalts, pitch and UVL carcinogenesis.. Cincinnati (OH): NIOSH, US Department of Health and Human Services. Publication No. 00122345

[NIOSH] National Institute for Occupational Safety and Health. 2000. Hazard Review: Health Effects of Occupational Exposure to Asphalt. Cincinnati (OH): NIOSH, US Department of Health and Human Services. Publication No. 2001-110.

Nisbet ICT, LaGoy PK. 1992. Toxic equivalency factors (TEFs) for polycyclic aromatic hydrocarbons (PAHs). Regulatory Tox. Pharmacol. 16:290–300

[NPRI] National Pollutant Release Inventory. [database on the Internet]. 2008. Gatineau (QC): Environment Canada. Available from: https://www.ec.gc.ca/inrp-npri/default.asp?lang=En&n=4A577BB9-1

Norin M, Strömvaix A-M. 2004. Leaching of organic contaminants from storage of reclaimed asphalt pavement. Environ Technol. 25:323-340.

[OMOE] Ontario Ministry of the Environment. 2013. Ontario Ministry of the Environment Spill Database, 2008-2012. Toronto (ON): Ontario Ministry of the Environment. Unpublished.

[Ontario] Ontario Standards Development Branch, Ministry of the Environment. 2011. Ontario Air Standards for Benzo[a]pyrene as a Surrogate for Polycyclic Aromatic Hydrocarbons. Toronto (ON): Ministry of the Environment. Available from: https://www.ontario.ca/page/ontarios-ambient-air-quality-criteria-sorted-contaminant-name#section-0

Osborn LV, Snawder JE, Kriech AJ, Cavalleri JM, McClean MD, Herric RF, Blackburn GR, Olsen LD. 2013. Personal breathing zone exposures among hot-mix asphalt paving workers; preliminary analysis for trends and analysis or work practices that resulted in the highest exposure concentrations. J. Occupational Environ. Hygiene 10:663-673.

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

Pasquini R, Monarca S, Sforzolini GS, Savino A, Bauleo FA, Angelil G. 1989. Urinary excretion of mutagens, thioethers and d-glucaric acid in workers exposed to bitumen fumes. Int Arch Occup Environ Health 61:335-349.

Petersen JC. 2009. A review of the fundamentals of asphalt oxidation. Chemical, physiochemical, physical property and durability relationships. Washington (DC): Characteristics of Bituminous Materials Committee Transportation Research Board.

PetroTox [estimation model]. 2012. Version 3.06. Brussels (BE): Conservation of Clean Air and Water in Europe (CONCAWE). Available from: https://www.concawe.eu/reach/petrotox/

Phimister AJ, Lee MG, Morin D, Buckpitt AR, Plopper CG. 2004. Glutathione depletion is a major determinant of inhaled naphthalene respiratory toxicity and naphthalene metabolism in mice. Toxicol Sci 82: 268-278. [cited in Canada 2008].

Potter TL, Simmons KE. 1998. Total petroleum hydrocarbon criteria working group series vol. 2: Composition of petroleum mixtures. Amherst (MA): Amherst Scientific Publications.

Qian HW, Ong T, Nath G, Whong WZ. 1998. Induction of DNA adducts in vivo in rat lung cells by fume condensates of roofing asphalt. Teratogenesis, Carcinogenesis and Mutagenesis, 18:131-140.

Robertson RE. 1991. Chemical properties of asphalts and their relationship to pavement performance. Laramie (WY): Western Research Institute; Washington (DC): Strategic Highway Research Program, National Research Council.

Robinson M, Bull RJ, Munch J, Meier J. 1984. Comparative carcinogenic and mutagenic activity of coal tar and petroleum asphalt paints used in potable water supply systems. J App Toxicol 4: 49-56.

Rowe AA, O'Connor TP. 2011. Assessment of water quality of runoff from sealed asphalt surfaces. Cincinnati (OH): US EPA, National Risk Management Research Laboratory. EPA/600/R-10/178.

Rühl R, Musanke U, Kolmsee K et al. Vapours and aerosols of bitumen: exposure data obtained by the German Bitumen Forum. 2006. Ann. Occup. Hyg., 50: 459-468.

SCREEN3 [computer model]. 2011. Ver. 3.5.0. Research Triangle Park (NC): US Environmental Protection Agency, Office of Air Quality Planning and Standards, Emissions, Monitoring, and Analysis Division. http://www.epa.gov/scram001/dispersion_screening.htm.

Simmers MH, Podolak E, Kinosita R. 1959. Carcinogenic effects of petroleum asphalt. Proc Soc Exp Biol Med 101: 266-268. Available from: http://ebm.sagepub.com/content/101/2/266.full.pdf+html

Simmers MH. 1965. Cancers from air-refined and steam-refined asphalt. Ind Med Surg 34: 255-261.

Simpson BJ. 2005. Analysis of petroleum hydrocarbon streams on the Health Canada CEPA/DSL Draft Maximal List. Calgary (AB): the Canadian Petroleum Products Institute. Report prepared for the Canadian Petroleum Products Institute.

Speight GJ. 2014. The chemistry and technology of petroleum. Boca Raton (FL): CRC Press.

Statistics Canada. 2013. The supply and disposition of refined petroleum products in Canada. Ottawa (ON): Statistics Canada. p.1-88. Catalogue No.: 45-004-X. Available from: http://www5.statcan.gc.ca/olc-cel/olc.action?objId=45-004-X&objType=2&lang=en&limit=0

[SENES] SENES Consultants Limited. 2010. Review of current and proposed regulatory and non-regulatory management tools pertaining to selected petroleum substances under the Chemicals Management Plan. Ottawa (ON): Health Canada, Healthy Environments and Consumer Safety Branch.

[TDOT] Texas Department of Transportation. 2017. Pavement Manual. Austin (TX): Texas Department of Transportation. [cited 2017 Aug 15]. Available from: http://onlinemanuals.txdot.gov/txdotmanuals/pdm/pdm.pdf

Townsend TG. 1998. Leaching characteristics of asphalt road waste. Gainesville (FL): University of Florida.

Truc H, Fleig C. 1913. Occular lesions produced by the dust and vapour of asphalt, Arch. Ophthalmol, 133:593-606. (cited in IUCLID 2000)

[UNHSC] University of New Hampshire Stormwater Center. 2010. Polycyclic Aromatic hydrocarbons Released from Sealcoated Parking Lots – A Controlled Field Experiment to Determine if Sealcoat is a Significant Source of PAHs in the Environment – Final Report. Durham (NH): United States Environmental Protection Agency.

[US DOT] United States Department of Transport (2005). Guidelines for using prime and tack coats. Lakewood (CO): Central Federal Lands Highway Division. Publication No.: FHWA-CFL/TD-05-002.

[US DHHS] United States Department of Health and Human Services. (2000), Hazard review - health effects of occupational exposure to asphalt. Cincinnati (OH): National Institute for Occupational Safety and Health

[US EPA] United States Environmental Protection Agency. 1992a. Screening procedures for estimating the air quality impact of stationary sources, revised. Washington (DC): US EPA, Office of Air Quality.

[US EPA] United States Environmental Protection Agency. 1992b. Peer consultation workshop on approaches to polycyclic aromatic hydrocarbon (PAH) health assessment. Washington (DC): US EPA. Available from: http://cfpub.epa.gov/ncea/iris_drafts/recordisplay.cfm?deid=54787

[US EPA] United States Environmental Protection Agency. 1993. A study of the use of recycled paving material: report to Congress. Washington (DC): Federal Highway Administration and US EPA. Report Nos.: FHWA-RD-93-147, EPA/600/R-3/095.

[US EPA] United States Environmental Protection Agency. 2000. Hot Mix Asphalt Plants Emission Assessment Report. Washington (DC): US EPA, Office of Air Quality Planning and Standards. EPA-454/R-00-019. [US EPA] United States Environmental Protection Agency. 2008. AP 42. 5th ed. Vol. 1 Chapter 5: Petroleum Industry, Section 5.2. Transportation and marketing of petroleum liquids. Washington (DC): US EPA.

[US EPA] United States Environmental Protection Agency. 2011. Screening-level hazard characterization. Asphalt category.
http://www.petroleumhpv.org/~/media/PetroleumHPV/Documents/Category_Asphalts_March_2011.pdf

U.S. Environmental Protection Agency Hazard Characterization Document. Washington (DC): US EPA.].

[US EPA] United States Environmental Protection Agency. 2011c. Exposure factors handbook 2011. National Center for Environmental Assessment. Office of Research and Development. Washington (DC): US EPA.

[US EPA] United States Environmental Protection Agency. 2013. Priority Pollutants [Webpage]. Washington (DC): US EPA. [Accessed 2015 May 21]. Available from: http://water.epa.gov/scitech/methods/cwa/pollutants.cfm

[US NTP] US National Toxicology Program. 1997. Literature review of health effects caused by occupational exposure to asphalt fumes: interim review produced by NIOSH in support of nomination to the National Toxicology Program. Research Triangle Park (NC): US NTP.

Van Metre PC, Mahler BJ, Williams ES. 2013. Cancer Risk from Ingestion Exposures to PAHs Associated with Coal-Tar-Sealed Pavement. Environ Sci Technol 47:1101-1109.

Van Metre PC, Majewski MS, Mahler BJ, William TF, Braun CL, Wilson JT, Burbank TL. 2012a. PAH volatilization following application of coal-tar-based pavement sealant. Atmosph. Environ 51: 108-115.

Van Metre PC, Majewski M, Mahler BJ, Foreman WT, Braun CL, Wilson JT, Burbank TL. 2012b. Volatilization of polycyclic aromatic hydrocarbons from coal-tar-sealed pavement. Chemosphere 88: 1-7.

Verbruggen EMJ, Beek M, Pijnenburg J, Traas TP. 2008. Ecotoxicological environmental risk limits for total petroleum hydrocarbons on the basis of internal lipid concentration. Environ Toxicol Chem. 27:2436-2448.

Vondruska M, Sild M, Koutny M, Bednarik V. 2002. Stabilization-solidification of waste incinerator salt and ash by asphalt emulsions. Environ Prot Eng. 28:129-142.

Wallcave L, Garicia H, Feldman R, Lijisky W, Shubik P. 1971. Skin tumorigenesis in mice by petroleum asphalts and coal tar pitches of known polynuclear aromatic hydrocarbon content. Toxicol Appl Pharmacol 18:41-52.

Wester PW, Muller JJA, Slob W, Mohn GR, Dortant PM, Kroese ED. 2012. Carcinogenic activity of benzo[*a*]pyrene in a 2 year oral study in Wistar rats. Food Chem Toxicol 50:927–935.

[WHO] World Health Organization. 1998. Selected non-heterocyclic polycyclic aromatic hydrocarbons. Environmental Health Criteria 202. Geneva (CH): International Programme on Chemical Safety. [cited 2014] Available from: http://www.inchem.org/documents/ehc/ehc/ehc202.htm

[WHO] World Health Organization. 2001. Polycyclic aromatic hydrocarbons. In: Background material to WHO Regional Publications, European series no. 91. , Copenhagen (DE): World Health Organization 91: 1-24.

Wilson R., Jones-Otazo H, Petrovic S, Mitchell I, Bonvalot Y. Williams D, Richardson GM. 2013. Revisiting dust and soil ingestion rates based on hand-to-mouth transfer. Human Ecolog. Risk Assessment: Inter. J. 19:158–88.

Appendices

Appendix A: Petroleum Substance Grouping

Table A-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 sea floor	Crude oil
Petroleum and refinery gases	Complex combinations of light hydrocarbons primarily from C_1 to C_5	Propane
Low boiling point naphthas	Complex combinations of hydrocarbons primarily from C_4 to C_{12}	Gasoline
Gas oils	Complex combinations of hydrocarbons primarily from C_9 to C_{25}	Diesel fuel
Heavy fuel oils	Complex combinations of heavy hydrocarbons primarily from C_{20} to C_{50}	Fuel oil No. 6
Base oils	Complex combinations of hydrocarbons primarily from C_{15} to C_{50}	Lubricating oils
Aromatic extracts	Complex combinations of primarily aromatic hydrocarbons from C_{15} to C_{50}	Feedstock for benzene production
Waxes, slack waxes and petrolatum	Complex combinations of primarily aliphatic hydrocarbons from C_{12} to 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 the CONservation of Clean Air and Water in Europe (CONCAWE) and a contractor's report presented to the Canadian Petroleum Products Institute (CPPI) (Simpson 2005).

Table A-2: Substance identity of asphalts

Property	Asphalt	Oxidized Asphalt
CAS RN	8052-42-4	64742-93-4
DSL name	Asphalt	Asphalt, oxidized
Chemical group (DSL category)	UVCB–organic	UVCB–organic
Major chemical class or use	Refinery streams	Refinery streams
Carbon range[a]	Mostly > C_{25}[b]	Mostly > C_{25}[b]
Aromatic content (%)	45–60[b]	45–60[b]
Aliphatic content (%)	5–20[b]	5–20[b]
Aliphatic : aromatic ratio when heated	99:1[c]	99:1[c]
Molecular weight	500–2000[c] 83–97 for asphalt vapour[a]	500-15 000[a]

Abbreviations: CAS RN, Chemical Abstracts Service Registry Number; DSL, Domestic Substances List; UVCB, Unknown or Variable composition, Complex reaction products or Biological materials.
[a]CONCAWE 1992; Trumbore 1999.
[b]IARC 1985; API 2009.
[c]API 2006, 2009.

Table A-3: Definitions for asphalts and related terms

Terms	Definitions
Asphalt:	Refers to asphalt the CAS RN 8052-42-4 in this assessment
Oxidized asphalt:	Refers to asphalt, oxidized, CAS RN 64742-93-4 in this assessment
Asphalts:	Refers to different penetration grades and types of asphalts and oxidized asphalt (CAS RNs 8052-42-4 and 64742-93-4) in this assessment
Asphalt group:	Typically made up of a group of heavy residual streams derived from the high temperature vacuum distillation of petroleum but may also be produced by thermal alteration during distillation, or from air blowing of residual streams. Asphalt group refers to the seven substances in US EPA defined "asphalt category" including asphalt and oxidized asphalt (US EPA 2011)
Commercial asphalt:	There are three main types of commercial asphalt, which are penetration grade, hard asphalts and oxidized asphalts, so it includes both non-oxidized and oxidized asphalt (API 2006)
Asphalt formulations:	Refers to asphalt products, which may contain other petroleum substances (e.g., aromatic extracts, base oils, gas oils, kerosene, naphtha and heavy fuel oils) that

	are not described by the CAS RNs considered in this assessment.
Asphalt materials:	A term refers to Statistics-Canada-defined asphalt, which is “produced by petroleum processing and includes asphalt flux, asphalt primers, asphaltic saturants, bitumals, cutback asphalts, liquid or solid asphalts, oxidized asphalt, paving compound and fluxes or primers” (Statistics Canada 2013)

Appendix B: Physical and chemical properties of representative substances for asphalts (experimental and modelled values, EPI Suite c2000-2010)[a]

***n*-Alkanes**

Carbon number, name (CAS RN)	BP (°C)	MP (°C)	VP (Pa)[b]	HLC (Pa·m^3/mol)	Log K_{ow}	Log K_{oc}	Water solubility (mg/L at 25°C)[c]
C_{30} triacontane (638-68-6)	449.7 (exp.)	65.8 (exp.)	3.64×10^{-9}	6.8×10^{8}	15.1	13.1	8.9×10^{-11}
C_{50} (6596-40-3)	548 (exp.)	87 (exp.)	2×10^{-7}	3.6×10^{10}	25	21.6	2.6×10^{-21}

Mono-cycloalkanes

Carbon number, name (CAS RN)	BP (°C)	MP (°C)	VP (Pa)[b]	HLC (Pa·m^3/mol)	Log K_{ow}	Log K_{oc}	Water solubility (mg/L at 25°C)[c]
C_{30} 1,5-dimethyl-1-(3,7,11,15-tetramethyl-octadecyl)-cyclohexane	420.9	103.2	1.5×10^{-4}	2.9×10^{8}	14.5	12.5	4.2×10^{-7}
C_{50}	674.2	294.0	5.6×10^{-13}	2.0×10^{11}	24.4	21.2	1.4×10^{-20}

Dicycloalkanes

Carbon number, name (CAS RN)	BP (°C)	MP (°C)	VP (Pa)[b]	HLC (Pa·m^3/mol)	Log K_{ow}	Log K_{oc}	Water solubility (mg/L at 25°C)[c]
C_{30} 2,4,6,10,14-	420.3	105.9	1.4×10^{-4}	3.9×10^{7}	13.6	11.8	1.7×10^{-9}

pentamethyl-dodecyl-2-decalin							
C_{50}	663.8	289.1	1.2×10^{-12}	5.7×10^{10}	23.2	20.2	1.4×10^{-19}

Polycycloalkanes

Carbon number, name (CAS RN)	**BP (°C)**	**MP (°C)**	**VP (Pa)[b]**	**HLC (Pa·m³/ mol)**	**Log K_{ow}**	**Log K_{oc}**	**Water solubility (mg/L at 25°C)[c]**
C_{26} 2,6-dimethyldecyl-hydrophenanthrene	394.6	112	4.9×10^{-4}	2.3×10^{5}	10.9	9.5	7.8×10^{-7}
C_{41} 2-(3,6-dimethylheptyl) -6-butyl-10-(2methylhexyl) hydrochrysene	554.6	236.6	4.3×10^{-9}	4.8×10^{7}	17.1	14.8	1.9×10^{-13}

Monoaromatics

Carbon number, name (CAS RN)	**BP (°C)**	**MP (°C)**	**VP (Pa)[b]**	**HLC (Pa·m³/ mol)**	**Log K_{ow}**	**Log K_{oc}**	**Water solubility (mg/L at 25°C)[c]**
C_{25} *n*-nonyldecyl benzene	407	124.4	2.8×10^{-4}	3.2×10^{5}	11.4	9.9	1.3×10^{-6}
C_{30} 1-benzyl-4,8,12,16-tetramethyl-eicosane	437.0	131.3	1.1×10^{-5}	3.8×10^{6}	13.5	11.8	6.8×10^{-9}
C_{50}	697.1	304.6	2.0×10^{-14}	1.0×10^{9}	23.8	20.7	1.7×10^{-19}

Cycloalkane monoaromatics

Carbon number, name (CAS RN)	**BP (°C)**	**MP (°C)**	**VP (Pa)[b]**	**HLC (Pa·m³/ mol)**	**Log K_{ow}**	**Log K_{oc}**	**Water solubility (mg/L at 25°C)[c]**
C_{26} 1-ethyl 4-2,6,10 trimethyldodec	399.6	126.8	1.1×10^{-4}	5.3×10^{4}	11.6	10	7.3×10^{-7}

ylindane							
C_{32} 2,6-dimethyloctyl-hexadecahydropicene	483	187.6	2.0×10^{-7}	2.9×10^{3}	12.7	11.0	2.9×10^{-8}

Di-aromatics

Carbon number, name (CAS RN)	**BP (°C)**	**MP (°C)**	**VP (Pa)[b]**	**HLC (Pa·m³/mol)**	**Log K_{ow}**	**Log K_{oc}**	**Water solubility (mg/L at 25°C)[c]**
C_{10} naphthalene (91-20-3)	217.9 (exp.)	80.2 (exp.)	13.1 (exp.)	44.6 (exp.)	3.3 (exp.)	3.0 (exp.)	31 (exp.)
C_{30} 2-(4,8,14,18-tetramethyl-hexadecyl)-naphthalene	468.5	170.6	7.1×10^{-7}	5.4×10^{4}	12.8	11.1	3.0×10^{-8}
C_{50}	721.5	316.1	3.1×10^{-15}	8.6×10^{6}	23.3	20.2	5.6×10^{-19}

Cycloalkane diaromatics

Carbon number, name (CAS RN)	**Boiling point (°C)**	**Melting point (°C)**	**Vapour pressure (Pa)[b]**	**HLC (Pa*m³/mol)**	**Log K_{ow}**	**Log K_{oc}**	**Water solubility (mg/L at 25°C)[c]**
C_{30} trimethylundecyl-1,2,3,6,7,8-hexahydropyrene	475.8	183.1	3.3×10^{-7}	4.2×10^{4}	12.7	11	3.2×10^{-9}

Three-ring aromatics

Carbon number, name (CAS RN)	**BP (°C)**	**MP (°C)**	**VP (Pa)[b]**	**HLC (Pa·m³/mol)**	**Log K_{ow}**	**Log K_{oc}**	**Water solubility (mg/L at 25°C)[c]**
C_{30} 2-(2,4,10-trimethyl-tridecyl)-phenanthrene	493.0	191.6	9.8×10^{-8}	942	12.0	10.5	1.2×10^{-8}
C_{50}	746.0	327.5	4.9×10^{-16}	3.1×10^{5}	23.0	19.3	3.5×10^{-19}

Four-ring aromatics

Carbon number, name (CAS RN)	BP (°C)	MP (°C)	VP (Pa)[b]	HLC (Pa·m^3/mol)	Log K_{ow}	Log K_{oc}	Water solubility (mg/L at 25°C)[c]
C_{30} 2-(3,6-dimethyldecyl) chrysene	524.4	211.4	9.2×10^{-9}	68.1	11.3	9.8	5.3×10^{-8}

Five-ring aromatics

Carbon number, name (CAS RN)	BP (°C)	MP (°C)	VP (Pa)[b]	HLC (Pa·m^3/mol)	Log K_{ow}	Log K_{oc}	Water solubility (mg/L at 25°C)[c]
C_{30} dimethyl-octylbenzo[*a*]-pyrene	544.8	231.2	1.6×10^{-9}	0.8	10.9	9.5	1.2×10^{-7}

Six-ring aromatics

Carbon number, name (CAS RN)	BP (°C)	MP (°C)	VP (Pa)[b]	HLC (Pa·m^3/mol)	Log K_{ow}	Log K_{oc}	Water solubility (mg/L at 25°C)[c]
C_{27} propyl benzo-perylene	548.5	278.2	1.2×10^{-9}	3.3×10^{-3}	8.8	7.7	1.5×10^{-5}

Abbreviations: BP, boiling point; CAS RN, Chemical Abstracts Service Registry Number; HLC, Henry's law constant; K_{oc}, organic carbon–water partition coefficient; K_{ow}, octanol–water partition coefficient MP, melting point; VP, vapour pressure.

[a]All values are modelled unless denoted with an (exp.) for experimental data.

[b]This is the maximum vapour pressure of the representative substance; the actual vapour pressure as a component of a mixture will be lower due to Raoult's law (the total vapour pressure of an ideal mixture is proportional to the sum of the vapour pressures of the mole fractions of each individual component). The lightest C_{15} representative substances were chosen to estimate a range of vapour pressures from the minimum to maximum values.

[c]Maximum water solubility was estimated for each representative substance based on its individual physical–chemical properties. The actual water solubility of a component in a mixture will decrease, as the total water solubility of an ideal mixture is proportional to the sum of the water solubilities of the mole fractions of each individual component (Banerjee 1984).

Appendix C: Sources and Releases of Asphalts in Canada

Table C-1. Canadian supply and demand characteristics in million liters, 2012 (Statistics Canada 2013)

Parameter[a]	Ontario	Alberta	Other provinces and territories[b]	Total for Canada
Production	736	1491	NA	**4349**
Exports	169	494	NA	**1398**
Imports	0.8	0.4	NA	**7.4**
Inter-regional transfers[c]	98.2	- 472	NA	**0**
Domestic sales	679	535	1884	**3098**

Abbreviation: NA, data not available.
[a]Data reported from refining companies in Canada and major wholesalers/distributors (Statistics Canada 2013).
[b]Data are suppressed by Statistics Canada to meet the confidentiality requirements of the *Statistics Act*.
[c]Negative values (–) denote transfers out of province/territory. Positive values denote transfers into provinces/territories.

Table C-2. Reported release volumes and spill numbers of asphalts spilled in Ontario from 2008 to 2012 (OMOE 2013)[a]

Year	Average spill volume (L)	Maximum single spill volume (L)	Median spill volume (L)	Number of spills reported	Spills with unknown volume (%)	Total known volume spilled (L)
2008	4661	25 000	500	9	22	32 628
2009	16 686	41 720	12 500	4	0	66 745
2010	1862	10 000	275	11	45	11 174
2011	1941	4720	70	5	20	7764
2012	8926	20 000	7850	8	50	35706
Total	-	-	-	37	-	154 017

[a]This does not include releases due to aircraft crashes, collisions, weather, ice/frost, road conditions, fires, petroleum refineries, subsidence, and vandalism.

Table C-3. Reported number of spills of asphalt materials affecting air, land, and freshwater in Ontario from 2008 to 2012 (OMOE 2013)

Year	Land	Freshwater	Air	Unknown	Total[a]
2008	8	1	0	0	9
2009	3	1	0	0	4
2010	9	3	0	0	12
2011	3	0	1	0	4
2012	7	0	0	1	8
Total	30	5	1	1	37
Median spill volume	1112	15 000	-	-	-

[a]This represents the total spills per year. Spills might have been released to multiple media and therefore the total is not a sum of the columns.

Table C-4. Causes of reported spills of asphalt materials in Ontario from 2008 to 2012 (OMOE 2013)

Cause	Total number of releases	Total volume of releases (L)	Proportion of volume (%)	Average release (L)
Discharge or bypass to a watercourse	1	432.3	0.3	432.3
Other	1	12 700.6	8.5	12 700.6
Other discharges	14	10 991	7.1	1099
Other transport accident	6	68 314	44.4	17 078.5
Overflow (tanks, lagoons)	1	5000	3.3	5000
Pipe or hose leak	2	619	0.4	309.5
Process upset/malfunction	1	-	-	-
Unknown	1	-	-	-
Valve/fitting leak or failure	2	30 000	19.5	15 000
Not listed	8	25 960	16.9	6490
Grand total	37	154 017	100	6161

Appendix D: Persistence of specific groups of petroleum hydrocarbons potentially found in asphalts

Table D-1. Persistence of petroleum hydrocarbon groups (based on Environment Canada (2014)

C#	C_{25}	C_{30}	C_{50}
n-Alkane	n/a	-	-
i-Alkane	n/a	S, W, Sd	-
n-Alkene	n/a	n/a	n/a
Diene	n/a	n/a	n/a
Monocycloalkane	n/a	Sd	S, W, Sd
Dicycloalkane	S, W, Sd	S, W, Sd	S, W, Sd
Polycycloalkane	n/a	n/a	n/a
Monoaromatic	n/a	Sd	Sd
Cycloalkane monoaromatic	n/a	n/a	n/a
Diaromatic	n/a	S, W, Sd	S, W, Sd
Cycloalkane diaromatic	n/a	n/a	n/a
Three-ring PAH	n/a	S, W, Sd	S, W, Sd
Four-ring PAH	n/a	n/a	n/a
Five-ring PAH	n/a	S, W, Sd	n/a
Six-ring PAH	n/a	n/a	n/a

Abbreviation: n/a – Indicates that no such carbon numbers exist within the group or the structure was not modelled.; PAH, polycyclic aromatic hydrocarbon; -, indicates that these structures are not considered to persist for long periods of time in air, soil, water, or sediment; S, a predicted half-life in soil of sux months or greater; W, a predicted half-life in water of six months or greater; Sd, a predicted half-life in sediment of one year or greater.

Appendix E: Ecotoxicological Information

Table E-1. Modelled acute aquatic toxicity data for asphalts (PETROTOX 2012)[a]

Test organism	Common name	LL_{50} (mg/L) 75:25[b] Ar:Al
Palaemonetes pugio	Grass shrimp	>1000
Daphnia magna	Water flea	>1000
Onchorynchus mykiss	Rainbow trout	>1000
Selenastrum capricornutum	Freshwater algae	>1000
Menidia beryllina	Inland silverside	>1000

Abbreviations: LL_{50}, this refers to lethal loading, the amount of product necessary to kill 50% of test organisms; Ar:Al, aromatic: aliphatic ratio.

[a]PETROTOX was run in the low resolution mode that only requires an aromatic to aliphatic ratio and a boiling point range for each hydrocarbon block, assuming a 1:1 ratio of components fall in the carbon range of C25–C35 (vs C_{36}–C_{50}).

[b]A 75:25 aromatic to aliphatic ratio was assumed based on a 5%–20% composition of saturates and 45%–60% composition of aromatics as asphalt components (IARC 1985; CONCAWE 1992; API 2006, 2009).

Table E-2. Aquatic toxicity of aromatic extracts (C_{20}–C_{50}) (US EPA 2011)

Species	Endpoint	Value (mg/L)	Reference[a]
O. mykiss	96-h LL_{50}	>1000	BP 1994
Scenedesmus subspicatus	96-h LL_{50}	>1000	BP 1994
	96-h LL_{50}	>1000	BP 1994
D. magna	48-h EL_{50}	>1000	BP 1994
	21-d EL_{50}	>1000	BP 1995
	21-d EL_{50}	>1000	BP 1995

Abbreviations: LL_{50}, this refers to lethal loading, the amount of product necessary to kill 50% of test organisms; EL_{50}, this refers to effect loading, the amount of product necessary to cause an effect on 50% of test organisms.

[a]Transcribed from US EPA 2011.

Table E-3. PAH concentrations in various leaching tests with a comparison to CCME water quality guidelines

PAH (ug/L)[a]	ACN	ANTHR	B[a]A	B[a]P
WQG (CCME 1999)	5.8	0.012	0.018	0.015
HMA (new asphalt) (Kriech 1990, 1992a)	<0.194, <0.16	<0.015, <0.16	<0.048, <0.013	<0.023, <0.023
Hot mix asphalt (road HMA (Kriech 1992a)	<0.16	<0.16	<0.013	<0.023
Cutback asphalt (Medium curing (Kriech 1992b)	<0.16	<0.021	<0.13	<0.23
CPG (paving) (Brandt and de Groot 2001)	0.017	0.012	0.0014	0.0003
HMA (Birgisdottir et al. 2007)	0.07	<0.007	0.012	<0.05
RAP (Legret et al. 2005)	<0.050	<0.02	<0.025	0.020
Roofing (Brandt and de Groot 2001)	0.0027	0.0061	0.0005	<0.00002

Table E-3. PAH concentrations in various leaching tests with a comparison to CCME water quality guidelines (continued)

PAH (ug/L)	FLUOR	NAPH	PHEN	PYR
WQG (CCME 1999)	0.04	1.1	0.4	0.025
HMA (new asphalt) (Kriech 1990, 1992a)	<0.037, <0.021	0.25, <0.16	<0.033, <0.16	0.04, <0.075
Hot mix asphalt (road HMA) (Kriech 1992a)	<0.021	0.76	0.30	<0.075
Cutback asphalt (Medium curing (Kriech 1992b)	<0.021	8.0	0.74	<0.075
CPG (paving) (Brandt and de Groot 2001)	0.0017	0.371	0.180	0.0039
HMA (Birgisdottir et al. 2007)	0.027	0.32	0.12	0.022
RAP (Legret et al. 2005)	0.05	<0.100	0.300	<0.025
Roofing (Brandt and de Groot 2001)	0.0017	0.120	0.0159	0.0043

Abbreviations: ACN, acenaphthene; ANTHR, anthracene; B[a]A, benzo[a]anthracene; B[a]P, benzo[a]pyrene; FLUOR, fluoranthene; NAPH, naphthalene; PHEN, phenanthrene; PYR, pyrene; CPG, conventional penetration grade; HMA, hot-mix asphalt; RAP; reclaimed asphalt pavement; WQG, water quality guideline.
[a]PAH concentrations from leaching tests presented here are the maximum measured concentrations.

Appendix F. Exposure from Industrial Release from Asphalt Processing, Handling and Storage for Asphalt Refineries and Dust Ingestion

Table F-1 Variable inputs to SCREEN3 for Hot Mix Asphalt Plants benzene dispersion

Variables	Input
Source type	Area
Effective emission area[a]	200 m × 275 m
Emission rates ($g/m^2 \cdot s$) [b]	7.41×10^{-9} (batch mix) 2.07×10^{-8} (drum mix)
Receptor height[c]	1.74 m
Source release height[a]	10 m
Adjustment factor for annual exposure[d]	0.2
Urban–rural option	Urban
Meteorology[e]	1 (full meteorology)
Minimum and maximum distance to use	1–3000 m

[a] Professional judgement.
[b] US EPA (2000). Annual emissions within plant obtained for beznene. Typical batch mix plant load-out operations used to estimate annual emissions within plant (Uncontrolled emissions from 100,000 tons of hot mix asphalt per year and 200,000 tonnes for drum mix asphalt per year).
[c] Curry et al. (1993)
[d] US EPA (1992a)
[e] Default value in SCREEN3 (1996).

Table F-2. Concentrations of benzene at hot mix asphalt plants form dispersion modelling

Distance (m)	Batch Mix Plant 1 Hour Concentration (µg/m3)	Batch Mix Plant Annual Concentration (µg/m3)	Drum Mix Plant 1 Hour Concentration (µg/m3)	Drum Mix Plant Annual Concentration (µg/m3)
1	0.0378	0.0076	0.1059	0.0212
100	0.0625	0.0125	0.1748	0.0350
200	0.0866	0.0173	0.2422	0.0484
230	0.0917	0.0183	0.2523	0.0505
300	0.0800	0.0160	0.2240	0.0448
400	0.0623	0.0125	0.1744	0.0349
500	0.0500	0.0100	0.1398	0.0280
600	0.0413	0.0083	0.1156	0.0231
700	0.0349	0.0070	0.0976	0.0195
800	0.0300	0.0060	0.0837	0.0167
900	0.0260	0.0052	0.0727	0.0145
1000	0.0228	0.0046	0.0637	0.0127
1100	0.0202	0.0040	0.0565	0.0113
1200	0.0181	0.0036	0.0505	0.0101
1300	0.0162	0.0032	0.0455	0.0091
1400	0.0148	0.0030	0.0413	0.0083
1500	0.0135	0.0027	0.0377	0.0075
1600	0.0124	0.0025	0.0347	0.0069
1700	0.0115	0.0023	0.0320	0.0064
1800	0.0106	0.0021	0.0297	0.0059
1900	0.0099	0.0020	0.0277	0.0055
2000	0.0093	0.0019	0.0259	0.0052
2100	0.0087	0.0017	0.0243	0.0049
2200	0.0082	0.0016	0.0229	0.0046
2300	0.0077	0.0015	0.0216	0.0043
2400	0.0073	0.0015	0.0204	0.0041
2500	0.0069	0.0014	0.0194	0.0039
2600	0.0066	0.0013	0.0184	0.0037
2700	0.0063	0.0013	0.0176	0.0035
2800	0.0060	0.0012	0.0168	0.0034
2900	0.0057	0.0011	0.0160	0.0032
3000	0.0055	0.0011	0.0154	0.0031

Table F-3 Variable inputs to SCREEN3 for hot mix asphalt plant total PAHs and individual PAH dispersion

Variables	Input
Source type	Area
Effective emission area[a]	200 m × 275 m
Emission rate benzo[a]anthracene[b] ($g/m^2 \cdot s$)	2.17×10^{-11}
Emission rate benzo[a]pyrene[b] ($g/m^2 \cdot s$)	9.34×10^{-13}
Emission rate benzo[b]fluoranthene[b] ($g/m^2 \cdot s$)	7.92×10^{-12}
Emission rate benzo[k]fluoranthene[b] ($g/m^2 \cdot s$)	2.14×10^{-12}
Emission rate dibenz[a,h]anthracene[b] ($g/m^2 \cdot s$)	6.54×10^{-14}
Emission rate indeno[1,2,3-cd]pyrene[b] ($g/m^2 \cdot s$)	4.95×10^{-13}
Receptor height[c]	1.74 m
Source release height[a]	10 m
Adjustment factor for annual[d]	0.2
Urban–rural option	Urban
Meteorology[d]	1 (full meteorology)
Minimum and maximum distance to use	1–3000 m

[a] Professional judgement.
[b] US EPA (2000) Estimates for annual emissions within plant obtained for hot mix asphalt plants
[c] Curry et al. (1993)
[d] Default value in SCREEN3 (2011).

Table F-4a. Dispersion modelling (in ug/m^3) results for PAHs (total and individual species) in the vicinity of drum mix asphalt plants

Distance (m)	Batch Mix 1 Hour	Batch Mix Annual	Drum Mix 1 Hour	Drum Mix Annual
1	0.1200	0.0240	0.0048	0.0010
100	0.1830	0.0366	0.0787	0.0157
200	0.2536	0.0507	0.0109	0.0022
230	0.2641	0.0528	0.0114	0.0023
300	0.2346	0.0469	0.0101	0.0020
400	0.1826	0.0365	0.0079	0.0016
500	0.1464	0.0293	0.0063	0.0013
600	0.1210	0.0242	0.0052	0.0010
700	0.1022	0.0204	0.0044	0.0009
800	0.0877	0.0175	0.0038	0.0008
900	0.0667	0.0133	0.0033	0.0007
1000	0.0591	0.0118	0.0029	0.0006
1100	0.0591	0.0118	0.0025	0.0005
1200	0.0529	0.0106	0.0023	0.0005
1300	0.0477	0.0095	0.0020	0.0004
1400	0.0433	0.0087	0.0019	0.0004
1500	0.0395	0.0079	0.0017	0.0003
1600	0.0363	0.0073	0.0016	0.0003
1700	0.0335	0.0067	0.0014	0.0003
1800	0.0311	0.0062	0.0013	0.0003
1900	0.0290	0.0058	0.0012	0.0002
2000	0.0271	0.0054	0.0012	0.0002
2100	0.0254	0.0051	0.0011	0.0002
2200	0.0239	0.0048	0.0010	0.0002
2300	0.0226	0.0045	0.0010	0.0002
2400	0.0214	0.0043	0.0009	0.0002
2500	0.0203	0.0041	0.0009	0.0002
2600	0.0193	0.0039	0.0008	0.0002
2700	0.0184	0.0037	0.0008	0.0002
2800	0.0176	0.0035	0.0008	0.0002
2900	0.0168	0.0034	0.0007	0.0001
3000	0.0161	0.0032	0.0007	0.0001

Table F-4b. Dispersion modelling (in ug/m^3) results for PAHs (total and individual species) in the vicinity of drum mix asphalt plants

Distance (m)	benzo[b]fluoranthene		benzo[k]fluoranthene	
	1 Hour Concentration (ng/m^3)	Annual Concentration (ng/m^3)	1 Hour Concentration (ng/m^3)	Annual Concentration (ng/m^3)
1	0.0405	0.0081	0.0110	0.0022
100	0.0668	0.0134	0.0181	0.0036
200	0.0926	0.0185	0.0251	0.0050
230	0.0964	0.0193	0.0026	0.0005
300	0.0856	0.0171	0.0232	0.0046
400	0.0666	0.0133	0.1803	0.0361
500	0.0534	0.0107	0.0145	0.0029
600	0.0442	0.0088	0.0120	0.0024
700	0.0373	0.0075	0.0101	0.0020
800	0.0320	0.0064	0.0087	0.0017
900	0.0278	0.0056	0.0075	0.0015
1000	0.0244	0.0049	0.0066	0.0013
1100	0.0216	0.0043	0.0058	0.0012
1200	0.0193	0.0039	0.0052	0.0010
1300	0.0174	0.0035	0.0047	0.0009
1400	0.0158	0.0032	0.0043	0.0009
1500	0.0144	0.0029	0.0039	0.0008
1600	0.0133	0.0027	0.0036	0.0007
1700	0.0122	0.0024	0.0033	0.0007
1800	0.0114	0.0023	0.0031	0.0006
1900	0.0106	0.0021	0.0029	0.0006
2000	0.0099	0.0020	0.0027	0.0005
2100	0.0093	0.0019	0.0025	0.0005
2200	0.0087	0.0017	0.0024	0.0004
2300	0.0082	0.0016	0.0022	0.0004
2400	0.0078	0.0016	0.0021	0.0004
2500	0.0074	0.0015	0.0020	0.0004
2600	0.0070	0.0014	0.0019	0.0004
2700	0.0067	0.0013	0.0018	0.0004
2800	0.0064	0.0013	0.0017	0.0003
2900	0.0061	0.0012	0.0017	0.0003
3000	0.0059	0.0012	0.0016	0.0003

Table F-4c. Dispersion modelling (in ug/m^3) results for PAHs (total and individual species) in the vicinity of drum mix asphalt plants

Distance (m)	dibenz(a,h)anthracene		indeno(1,2,3-cd)pyrene	
	1 Hour Concentration (ng/m^3)	Annual Concentration (ng/m^3)	1 Hour Concentration (ng/m^3)	Annual Concentration (ng/m^3)
1	0.000334	0.000067	0.002528	0.000506
100	0.000551	0.000110	0.004172	0.000834
200	0.000764	0.000153	0.005783	0.001157
230	0.000796	0.000159	0.006022	0.001204
300	0.000706	0.000141	0.005348	0.001070
400	0.000550	0.000110	0.004163	0.000833
500	0.000441	0.000088	0.003338	0.000668
600	0.000364	0.000073	0.002758	0.000552
700	0.000308	0.000062	0.002331	0.000466
800	0.000264	0.000053	0.001999	0.000400
900	0.000229	0.000046	0.001734	0.000347
1000	0.000210	0.000042	0.001522	0.000304
1100	0.000178	0.000036	0.001348	0.000270
1200	0.000159	0.000032	0.001205	0.000241
1300	0.000144	0.000029	0.001086	0.000217
1400	0.000130	0.000026	0.000986	0.000197
1500	0.000119	0.000024	0.000901	0.000180
1600	0.000109	0.000022	0.000828	0.000166
1700	0.000101	0.000020	0.000764	0.000153
1800	0.000094	0.000019	0.000709	0.000142
1900	0.000087	0.000017	0.000661	0.000132
2000	0.000082	0.000016	0.000618	0.000124
2100	0.000077	0.000015	0.000580	0.000109
2200	0.000072	0.000014	0.000546	0.000103
2300	0.000068	0.000012	0.000515	0.000098
2400	0.000064	0.000013	0.000488	0.000093
2500	0.000061	0.000012	0.000463	0.000093
2600	0.000058	0.000012	0.000440	0.000088
2700	0.000055	0.000011	0.000419	0.000084
2800	0.000053	0.000011	0.000400	0.000080
2900	0.000051	0.000010	0.000383	0.000077
3000	0.000048	0.000010	0.000367	0.000073

Table F-5a. PAH concentrations (µg/g) and benzo[a]pyrene equivalents in house dust (includes unsealed asphalt and asphalt-based sealcoated asphalt pavement types)

Compound[a]	Anthra-cene	Benzo[a]-pyrene	Fluor-anthene	Naph-thalene	Benzo [k] fluor-anthene
Sample Conc.	0.14	1.36	3.44	<0.03	1.10
Sample Conc.	0.42	3.91	8.13	0.27	2.80
Sample Conc	0.08	0.58	1.00	<1.25	0.40
Sample Conc.	0.20	1.50	5.82	<0.23	0.95
Sample Conc	0.28	2.05	6.43	<0.42	1.38
Sample Conc	0.02	0.06	0.17	0.02	0.06
Sample Conc	0.04	0.26	0.88	0.04	0.23
Sample Conc	0.05	0.23	0.71	0.11	0.18
Sample Conc	<0.23	0.30	0.70	<0.23	0.25
Sample Conc	<0.03	0.25	0.61	0.05	0.18
Average	0.149	1.05	2.789	0.265	0.753
Potency Factor[b]	0.001	1	0.001	0.001	0.1
Equivalent BaP Conc.	0.000149	1.05	0.002789	0.000265	0.0753

Table F-5b. PAH concentrations (µg/g) and benzo[a]pyrene equivalents in house dust (includes unsealed asphalt and asphalt -based sealcoated asphalt pavement types)

Compound[a]	Benz [a] anthracene	Pyrene	Fluorene	Dibenzo [a,h] anthracene	Indeno [1,2,3-cd] pyrene
Sample Conc.	0.95	2.89	<2.94	<0.31	1.05
Sample Conc.	1.8	6.77	0.19	<0.75	2.22
Sample Conc	0.35	0.83	<1.25	<1.25	0.45
Sample Conc.	1.19	4.89	<0.23	<0.30	0.96
Sample Sample Conc	1.86	5.68	0.14	<0.42	1.36
Sample Conc	0.05	0.15	<0.01	<0.01	0.05
Sample Conc	0.18	0.70	<0.02	<0.05	0.20
Sample Conc	0.20	0.58	<0.03	<0.04	0.14
Sample Conc	<0.2	0.58	<0.23	0.23	0.30
Sample Conc	0.17	0.50	<0.02	<0.05	0.19
Average	0.695	2.357	0.506	0.341	0.692
Potency Factor[b]	0.1	0.001	0.001	5	0.1
Equivalent B[a]P Conc.	0.0695	0.002	0.00051	1.705	0.069

Table F-5c. PAH concentrations (µg/g) and benzo[a]pyrene equivalents in house dust (includes unsealed asphalt and asphalt–based sealcoated asphalt pavement types)

Compound[a]	Phen-anthrene	Acenaph-thene	Acenaph-thalene	Benzo [b] fluor-anthene	Benzo [g,h,i] perylene	Chry-sene
Sample Conc.	1.35	<0.31	<0.31	2.91	1.15	1.98
Sample Conc.	3.44	<0.42	0.24	6.48	1.64	5.02
Sample Conc.	0.55	<1.25	<1.25	0.89	0.42	0.51
Sample Conc.	1.94	<0.23	<0.23	2.33	0.85	1.61
Sample Conc.	1.60	<0.42	<0.42	4.00	1.26	1.09
Sample Conc.	0.18	<0.02	<0.01	0.14	0.06	0.11
Sample Conc.	0.36	<0.02	0.02	0.55	0.27	0.38
Sample Conc.	0.47	<0.05	0.02	0.48	0.20	0.45
Sample Conc.	0.25	<0.23	<0.23	0.69	0.31	0.50
Sample Conc.	0.33	<0.02	0.01	0.48	0.25	0.32
Average (ug/g)	1.047	0.297	0.274	1.895	0.641	1.197
Potency Factor[b]	0.001	0.001	0.001	0.1	0.01	0.01
Equivalent BaP Conc.	0.00105	0.0003	0.00027	0.1895	0.00641	0.012
Sum of 16 B[a]P Equivalent Conc.'s (ug/g^3)[c]	3.18					

[a] Concentration data from Mahler (2010).
[b] Potency Equivalency Factors from Nisbet and LaGoy (1992).
[c] Total B(a)P concentration from the sum of the potency equivalent PAH concentrations.

Table F-6. Age-dependent adjustment factors (ADAF) and determination of dust ingestion MOE.

Life Stage	Ages (year)	ADAF[a]
Child	0 – < 2	10
Child	2 – < 6	3
Adult	6 – < 16	3
Adult	16 – < 30	1

Table F-7. Age-dependent adjustment factors (ADAF) and determination of dust ingestion MOE.

Factors Adjusted to Health Canada Age Bins Life Stage	Ages (year)	ADAF
Infant	0 – 0.5	10
Toddler	0.5 – 4	5[b]
Children	5 – 11	3
Teenager	12 – 19	2[c]
Adult	20+	1

[a] US EPA (2011)

[b] $ADAF_{0.5-4} = (ADAF_{0 \text{ to } <2} \times D_{0.5-1}/D_{0.5-4}) + (ADAF_{2-4} \times D_{2-4}/D_{0.5-4})$
$= (10 \times 1.5/4.5) + (3 \times 3/4.5) = 5$, D_i = exposure duration (years)

[c] $ADAF_{12-19} = (ADAF_{12 \text{ to } <16} \times D_{12-15}/D_{12-19}) + (ADAF_{16+} \times D_{16-19}/D_{12-19})$
$= (3 \times 4/8) + (1 \times 4/8) = 2$, D_i = exposure duration (years)

Age group-specific adjusted MOEs =

$$MOE_{ADJ\ age\ group} = (BMDL_{10}/Dose_{age\ group}) \times (1/ADAF_{age\ group})$$

Lifetime adjusted MOE =

$$MOE_{ADJ\ lifetime} = 1 / \Sigma[1/MOE_{ADJ\ age\ group} \times Averaging\ Time_{age\ group}]$$

$$MOE_{ADJ\ lifetime} = 1 / [(1/MOE_{ADJ\ 0\ to\ 0.5yrs} \times 0.5/71) + (1/MOE_{ADJ\ 0.5\ to\ 4yrs} \times 4.5/71) + (1/MOE_{ADJ\ 5\ to\ 11yrs} \times 7/71) + (1/MOE_{ADJ\ 12\ to\ 19yrs} \times 8/71) + (1/MOE_{ADJ\ 20\ to\ 59yrs} \times 40/71) + (1/MOE_{ADJ\ 60+yrs} \times 11/71)]$$

$$MOE_{ADJ\ lifetime} = 1 / [(1/3488 \times 0.5/71) + (1/9961 \times 4.5/71) + (1/35119 \times 7/71) + (1/1458019 \times 8/71) + (1/3056128 \times 40/71) + (1/3181132 \times 11/71)]$$

$$MOE_{ADJ\ lifetime} = 99\ 150$$

Appendix G: Summary of health effects information for asphalts (CAS RNs 8052-42-4 and 64742-93-4)

Endpoint	Effect levels[a]/Results
Acute	**Dermal** LD_{50} >2000 mg/kg bw (rabbit). Test substance was warmed overnight and applied at 2000 mg/kg-bw to abraded or non-abraded skin of New Zealand White rabbits (2 per sex per condition) with occlusion for 24 hours. Animals were monitored for 14 days. No clinical signs of adversity were noted, and there were no visible lesions at the application site. Two animals developed diarrhea, one on day 1 and one on days 6 and 7 (API 1982).
Acute	**Inhalation** LC_{50} >94.4 mg/m^3 (rat). Ten Wistar rats (5 per sex) were nose-only exposed for 4.5 hours to vapourized fume from a sample of bitumen condensate. Study was according to the OECD 403 guideline and followed GLP. No adverse effects were noted, although exposed animals exhibited significantly lower body temperature (lower by 1.4^{o}C on average relative to control animals) (Fraunhofer 2000).
Acute	**Oral** LD_{50} >5000 mg/kg bw (rat). Ten Sprague-Dawley rats were administered 5000 mg/kg-bw of test substance in corn oil and observed for 14 days. The study was conducted according to GLP. No adverse effects were noted. Clinical signs included hypoactivity and diarrhea. No significant abnormalities at necropsy (API 1982).
Short-term Repeated-exposure	**Dermal** **LO(A)EL = 2000 mg/kg-bw** per day, based on significantly reduced body weight gain in New Zealand White rabbits. Rabbits (5 per sex per group) were exposed to vacuum residues (API samples 81-13 and 81-14) via abraded and occluded dorsal skin at 200, 1000 and 2000 mg/kg-bw, 6 hours per application, three times weekly for 4 weeks. Reduced body weight gain was seen for animals exposed to test substance API 81-13 but not for those exposed to API 81-14. Studies were conducted according to GLP (API 1983a,b).
Short-term Repeated-exposure	**Inhalation** **LO(A)EC = 100 mg/m^3**, based on changes in lung histology parameters, in male and female Wistar (Crl:WU) rats exposed

	to asphalt fume condensate at target concentrations of 0, 30, 100 and 300 mg/m^3 total hydrocarbons (THC) for 28 days (or 42 days for pregnant females) (Fraunhofer 2009).
Subchronic Repeated-exposure	**Inhalation** **LO(A)EC = 149.17 mg/m^3**, based on degenerative lesions in nasal and paranasal cavities (slight to moderate eosinophilic cytoplasmic inclusions), in groups of 16 Wistar rats of each sex, exposed either to clean air or asphalt fumes at concentrations of 5.53, 28.17, or 149.17 mg/m^3, 5 days per week for 14 weeks (Fraunhofer 2001).
Chronic (non-cancer)	**Inhalation** **LO(A)EC = 34.4 mg/m^3**, based on a statistically-significant decrease in body weight gain, in groups of 50 male and 50 female SPF-Wistar rats exposed to concentrations of 4, 20 and 100 mg/m^3 total hydrocarbons paving asphalt fume condensate (actual concentrations were determined to be 0, 6.8, 34.4 and 172.5 mg/m^3) via nose-only inhalation, 6 hours per day, 5 days per week for 2 years. No statistically significant differences were observed in mortality and no clinical signs of intoxication were seen. Statistically significant decreases in body weight gain in the mid and high exposure groups were seen but not at all time points of observation. Slight increases in the transitional zone of respiratory to olfactory epithelium were seen only in 172.5 mg/m^3 males. Histopathology identified slight dose-related degenerative, inflammatory and proliferative lesions in the nasal cavity and slight dose-related alveolar bronchiolization and mononuclear/inflammatory cell infiltrations in lungs (Fuhst et al. 2007).
Chronic (non-cancer)	A total of 30 male and female C57 black mice were exposed (whole body) to a mix of fumes, generated from steam-refined and oxidized asphalt at 120^{o}C, for 5 days per week for 21 months. Histological pulmonary changes noted included bronchitis, loss of cilia, epithelial atrophy and necrosis, and pneumonitis. One bronchial adenoma was found in treated animals (Simmer 1964).
Chronic (non-cancer)	One group of 65 Bethesda black rats and one group of 13 guinea pigs were exposed to an unknown concentration of roofing asphalt fumes (derived from oxidized asphalt; fumes were generated by heating a dish containing air-blown asphalt to 120^o-135^{o}C) for 5 hours per day, 4 days per week for 2 years. None of the animals developed lung cancer but some rats had chronic fibrosing pneumonitis with peribronchical adenomatosis (Heuper and Payne 1960).

Carcinogenicity	**Dermal** Swiss mice (15 per sex per group) were exposed to 8 samples of 10% whole asphalt diluted in benzene. Test samples were applied to approximately 6.5 cm^2 shaved areas on the back, twice per week for at least 81 weeks. Tumour incidence ranged from 0 to 7% across the 8 samples, with an average incidence of 2.7%. Overall, there were 6 tumours in 218 mice, including 1 squamous cell carcinoma (Wallcave et al. 1971).
Carcinogenicity	Groups of male C3H mice (group size unknown) were dermally exposed to 37.7 µl of a 30% weight per volume (w/v) solution of AC 20 asphalt (whole asphalt) (CAS RN 8052-42-4 or CAS RN 64741-56-6) dissolved in mineral oil solvent, twice per week for 24 weeks. Results for both test substances were negative (McGowan et al. 1992).
Carcinogenicity	A total of 348 mice (of unknown strain and sex) were exposed to unknown doses of 6 grades of whole asphalt (40% of test substance in benzene). Test substances were painted onto the animals once per week for 19 months. The study concluded that the straight asphalt was not a dermal carcinogen (4 tumours in 163 animals, incidence of 2.55%), but that cracked asphalt was a dermal carcinogen (13 tumours in 91 animals (incidence of 14.3%) (Kireeva 1968).
Carcinogenicity	C57BL mice (25 per sex per group; 100 animals in control), or 6 New Zealand White rabbits, were dermally exposed twice per week for two years to unknown doses of 4 steam-distilled road asphalts (CAS RNs 8052-42-4 and 64741-56-6) and roofing asphalt (CAS RN 64742-93-4). For the study conducted in mice, test substances were diluted in acetone and applied to the nape of the neck. For the study in rabbits, test substances were applied undiluted to the ear and to a 2.0 cm^2 shaved area of the back. For mice exposed to road asphalts, there were 0/100, 2/50, 1/50 and 0/50 with skin tumours (in total, 4 skin tumours developed, including 2 carcinomas and 2 papillomas). Six leukemias were observed in two groups of mice exposed to road asphalt. Rabbits did not develop any tumours with exposure to road asphalts. For 50 mice exposed to roofing asphalt, one skin tumour (potentially a carcinoma) was observed. No skin tumours were seen in the control group (Hueper and Payne 1960).
Carcinogenicity	C57BL mice (25 per sex per group) were exposed to 75 to 100 mg (2500 to 3300 mg/kg-bw[b]; specific dose unknown) of oxidized (air-refined) asphalt (CAS RN 64742-93-4) with or without toluene dilution, or steam-refined asphalt (CAS RN 64741-56-6) without toluene dilution. Test substances were

	painted onto the shaved skin of the mice one to three times per week, for 21 months. One lung adenoma and one papilloma was observed in the group exposed to undiluted air-refined asphalt. Nine epidermoid cancers of the skin occurred at the site of application, as well as two lung adenomas, in the group exposed to air-refined asphalt in toluene. Three epidermoid cancers, one lung adenoma and two papillomas were seen in the group exposed to steam-refined asphalt (Simmers 1965a).
Carcinogenicity	Groups of 50 C3H/HeJ and CD-1 mice (sex unknown) were exposed to condensed fumes from two asphalt (CAS RN 64742-93-4) samples (each generated under low and high temperatures, resulting in four different samples), in the presence and absence of UV radiation, twice per week for 72 weeks. Test substances were applied as 50% solutions dissolved in a 1:1 cyclohexane/acetone solvent to the shaven interscapular regions of the test animals. The condensates were carcinogenic both in the presence and absence of UV radiation. The C3H/HeJ mouse strain was more sensitive than the CD-1 strain (per strain, in the absence of UV radiation, tumour incidences were 89-96% with mean latencies of 40-52 weeks, and 16-43% with mean latencies of 52-60 weeks, respectively). The two test substances generated under lower temperature were less active in the CD-1 strain (tumour incidence of 18% and 29%) compared to the two samples generated under higher temperature (tumour incidence of 47% and 43%) (NIOSH 1981).
Carcinogenicity	Groups of 31-36 (actual group sizes unknown) of male and female C57BL mice were dermally exposed to a pooled sample containing steam-refined asphalt (CAS RN 8052-42-4 or 64741-56-6), and oxidized asphalt (CAS RN 64742-93-4) in benzene, twice per week for more than 54 weeks (actual exposure period not specified). The dose and proportion of each CAS RN in the test substance was not provided. The test substance mixture was considered a dermal carcinogen (12 epidermoid carcinomas and 5 papillomas were observed in 68 animals) (Simmers et al. 1959).
Carcinogenicity	Groups of 50 male CD-1 and C3H/HeJ mice were dermally exposed to fume condensates (dose unknown) generated from Type I and Type III roofing asphalt at 232^{o}C and 316^{o}C. Test substances were applied to shaved backs biweekly for 78 weeks. Tumours were induced by fume condensates from both types of asphalts (incidences unknown). The tumorigenic response of both types of asphalt was greater from fumes generated at 315^{o}C compared to fumes generated at 232^{o}C (Niemeier et al. 1988).

Carcinogenicity	**Inhalation** Groups of 50 male and 50 female SPF-Wistar rats (control groups contained 86 rats of each sex) were nose-only exposed to 4, 20 and 100 mg/m^3 total hydrocarbons from paving asphalt (fumes were collected from a hot asphalt storage tank), for 6 hours per day, 5 days per week for 2 years. No increases in the number of tumour-bearing animals were observed relative to controls (incidence in male groups: 28/50, 30/50, 27/50 and 30/50 and in female groups: 42/50, 34/50, 39/50 and 33/50 for control, low, medium and high dose groups, respectively). No statistically significant increases in organ-specific tumors were seen in exposed animals (e.g., for lung adenomas in males: 0/50, 0/50, 0/50 and 1/50 as above; and zero incidence was observed in all female groups). A poorly differentiated adenocarcinoma of the nasal cavity was observed in a single male of the high dose group (for adenocarcinoma of nasal and paranasal cavities in males: 0/50, 0/50, 0/50 and 1/50 as above; and zero incidence was observed in all female groups) (Fuhst et al. 2007).
Carcinogenicity	One group of 65 Bethesda black rats and one group of 13 guinea pigs were exposed to an unknown concentration of roofing asphalt fumes (derived from oxidized asphalt; fumes were generated by heating a dish containing air-blown asphalt to 120^o-135^{o}C) for 5 hours per day, 4 days per week for 2 years. None of the animals developed lung cancer but some rats had chronic fibrosing pneumonitis with peribronchical adenomatosis (Heuper and Payne 1960).
Carcinogenicity	A total of 30 male and female C57 black mice were exposed (whole body) to a mix of fumes, generated from steam-refined and oxidized asphalt at 120^{o}C, for 5 days per week for 21 months. Histological pulmonary changes noted included bronchitis, loss of cilia, epithelial atrophy and necrosis, and pneumonitis. One bronchial adenoma was found in exposed animals (Simmer 1964).
Genotoxicity – *in vivo*	**Chromosomal aberrations** Groups of 10 male and 10 female Sprague-Dawley rats were administered a vacuum residue in corn oil (API sample 81-13) by gavage at 300, 1000 and 3000 mg/kg-bw per day for five days (API 1984c). No effect was noted.
Genotoxicity – *in vivo*	Groups of 10 male and 10 female Sprague-Dawley rats were administered vacuum residue in corn oil (API sample 81-14) by gavage at 400, 1300 and 4000 mg/kg-bw per day for five days (API 1984d). No effect was noted.

Genotoxicity – *in vivo*	**Micronucleus assay** Groups of male Sprague-Dawley rats (group size unknown) were exposed to an asphalt fume condensate (AFC) (collected from the top of a paving storage tank) intra-tracheally at 0.45, 2.22 and 8.88 mg/kg-bw for 3 consecutive days, and were sacrificed the following day. A significant increase in the frequency of micronuclei formation in bone marrow polychromatic erythroctyes (PCEs) was seen in the high-dose AFC-exposed group compared with controls (Ma et al. 2002).
Genotoxicity – *in vivo*	Groups of Wistar [Crl:WU] rats (group size unknown) were exposed to oxidized roofing asphalt fume condensate by nose-only inhalation at target concentrations of 30, 100 or 300 mg/m^3 for 28 days. No increase in micronucleated polychromatic erythrocytes in the bone marrow of treated rats was observed (Fraunhofer 2009).
Genotoxicity – *in vivo*	**DNA adduct formation** Groups of three male CD rats were exposed to condensed asphalt fumes (type I and type III roofing asphalts, obtained by heating small pieces of the asphalt to 316 ± 10^{o}C in round bottomed flasks) via intratracheal instillation at 500, 1000 and 2250 mg/kg-bw every 8 hours for a total of 3 instillations per treatment group. Significant increases in DNA adducts occurred in lung cells after instillation of the medium and high doses of type I asphalt sample, and of all dose levels of type III asphalt sample. No elevation in DNA adduct levels in leukocytes (collected by cardiac puncture from the same rats) was observed (Qian et al. 1998).
Genotoxicity – *in vivo*	Five transgenic male *LacI* mice C57BL/6 (lambda *LIZ* Big Blue®), were nose-only exposed to 100 mg/m^3 total particulate paving asphalt fumes [50/70 penetration grade of asphalt (pen)] (that were generated at 170^{o}C; a road paving temperature) 6 hours per day for 5 days (Micillino et al. 2002). No effect was noted.
Genotoxicity – *in vivo*	A total of 12 transgenic Big Blue® male rats were nose-only exposed to 100 mg/m^3 total particulate paving asphalt fumes (that were generated at 170^{o}C; a road paving temperature) 6 hours a day for five consecutive days (Bottin et al. 2006). No effect was noted.
Genotoxicity – *in vivo*	**DNA strand breaks** Groups of Sprague Dawley female rats were exposed to

	asphalt fumes generated under road paving conditions (120-170^{o}C), 6 hours per day for 1-5 days. Dose-dependent DNA single strand breaks in alveolar macrophages and lung tissue were observed in the Comet assay. No micronucleus formation was seen in bone marrow polychromatic erythrocytes (Zhao et al. 2004).
Genotoxicity – *in vivo*	***Human cytogenetic Effects*** **Micronucleus assay** Twelve male asphalt road layers, between 25 and 63 years old, exhibited increases in micronucleus formation in peripheral blood cells compared to 18 control hospital workers (Information on exposure concentration and condition of the twelve road layers were unavailable) (Murray and Edwards 2005).
Genotoxicity – *in vivo*	Twenty-one male asphalt field workers in road paving operations and 7 workers that prepared hot mixtures of stone chips and asphalt in an asphalt plant (workers were non-smokers) exhibited a statistically-significant increase in micronucleus formation in peripheral blood compared with 28 men from university and hospital staff (Information on exposure concentration and condition of the workers were unavailable) (Burgaz et al. 1998).
Genotoxicity – *in vivo*	**Sister chromatid exchange** Twenty-one male asphalt field workers in road paving operations and 7 workers that prepared hot mixtures of stone chips and asphalt in an asphalt plant (workers were non-smokers) exhibited sister chromatid exchange in peripheral blood compared with 28 men from university and hospital staff (Information on exposure concentration and condition of the workers were unavailable) (Burgaz et al. 1998).
Genotoxicity – *in vivo*	Paving workers (9 smokers and 10 non-smokers) were compared to 22 healthy subjects that worked in administration (11 smokers and 11 non-smokers) (Information on exposure concentration and condition of the workers were unavailable) (Cavallo et al. 2005). No differences in SCE were noted.
Genotoxicity – *in vivo*	**DNA strand breaks** Paving workers (9 smokers and 10 non-smokers) were compared to 22 healthy subjects working in administration (11 smokers and 11 non-smokers). DNA strand breaks were higher in paving workers (Information on exposure concentration and condition of the workers were unavailable) (Cavallo et al. 2005).

Genotoxicity – *in vitro*	**Ames Assay** *S. typhimurium* strains TA98 and TA100 were exposed to 80/100 penetration grade of asphalt collected during paving operations (CAS RN 8052-42-4 or CAS RN 64741-56-6), dissolved in dimethyl sulphoxide (DMSO), with and without rat-liver S9 (Monarca et al. 1987). No effect was noted.
Genotoxicity – *in vitro*	*S. typhimurium* strain TA98 was exposed to asphalt fume condensate collected from the head space of a hot (temperature range of 147^{o}C to 157^{o}C) mix asphalt storage tank, with S9 (Reinke et al. 2000). No effect was noted.
Genotoxicity – *in vitro*	Other *S. typhimurium* strains (TA1535, TA1537, TA1538, TA98 and TA100, with and without S9) produced negative results (Robinson et al. 1984).
Genotoxicity – *in vitro*	*S. typhimurium* strain TA98, with laboratory-generated fumes from two Type III roofing asphalts derived from different crudes (fumes were generated at 232^{o}C or 316^{o}C), with S9, exhibited weak to moderately mutagenic activity (Machado et al. 1993).
Genotoxicity – *in vitro*	*S. typhimurium* strain TA98 with laboratory-generated fumes at 149^{o}C and 316^{o}C, with S9, exhibited mutagenic activity (Reinke et al. 2000).
Genotoxicity – *in vitro*	DMSO extracts of roofing and paving asphalts (CAS RN 8052-42-2 or CAS RN 64741-56-6), in an optimized Ames Test [ASTM method E1687-95] using elevated levels of S9 exhibited marginally positive mutagenic activity (Blackburn and Kriech 1990).
Genotoxicity – *in vitro*	**Mouse Lymphoma Assay** L5178Y TK+/- mouse lymphoma cell line was exposed to 62.5 to 1000 ug/ml of vacuum residues API 81-13 and API 81-14 (both CAS No. 64741-56-6), with metabolic activation. Positive results were seen only with metabolic activation (API 1984c,d).
Genotoxicity – *in vitro*	**DNA Adducts** Asphalt fume condensates were generated at temperatures of 160^{o} or 200^{o}C and metabolically activated with Aroclor-induced rat liver homogenate, and calf thymus DNA was used in a ^{32}P- post-labeling assay. Positive results were obtained with metabolic activation (but not without activation) from Aroclor-induced rat liver homogenate at levels much lower than that required for coal tar fume condensates to induce DNA adducts. The adducts also exhibited a qualitative difference compared with coal tar-induced adducts (DeMéo et al 1996).

Genotoxicity – *in vitro*	Asphalt fume condensates (BFCs), obtained from hot asphalt tanks and having a chemical composition similar to that found for human exposures (McCarthy et al. 1999), was positive when tested on calf thymus DNA (Akkineni et al. 2001).
Reproductive	**Inhalation** **NOAEC (reproductive) = 300 mg/m^3**. Groups of male and female Wistar (Crl: WU) rats (group size unknown) were exposed to RAFC fumes via nose-only inhalation at target concentrations of 0, 30, 100 and 300 mg/m^3 total hydrocarbons (THC) for 28 days.
Reproductive	**LOAEC (maternal) = 300 mg/m^3**, based on minimal histopathological effects in the lungs, in which a slight increase of alveolar macrophage accumulation was observed in combination with minimal mononuclear/inflammatory cell infiltration and minimal to slight (adaptive) alveolar hyperplasia of the bronchioles. Rats (7/12) of the high dose group showed multi-focal very slight to slight mucosal mononuclear/inflammatory cell infiltration compared to one female control. Pup viability indices, sex ratio, body weight and body weight gain over lactation days 0-4 were comparable to controls (Fraunhofer 2009).
Irritation	**Skin irritation** Not irritating (rabbit) (API 1982).
Irritation	**Eye irritation** Slightly irritating (rabbit) (API 1982).
Irritation	**Eye irritation** Slightly irritating / irritating (rabbit) (Truc and Fleig 1913).
Sensitization	**Sensitization** Not sensitizing (guinea pig) (API 1984a,b).
Human study	A cohort of 679 male Danish mastic asphalt workers who were heavily exposed to asphalt fumes was followed from 1959 to 1984 and observed for cancer incidence. Among these individuals, 75 cases of cancer were found. The cancer incidence significantly exceeded that of the total Danish male population, with a standardized morbidity ratio (SMR) of 195. Significant increases were seen for cancer of the mouth, the rectum and the lung. It was suggested that exposure to cracking products in the fumes of heated asphalt contributed to the elevated cancer incidence (Hansen 1989).
Human study	Death certificates mentioning lung cancer in 2161 white males aged 20 to 64, for the period 1968-1970, and 1777 incident cases of lung cancer in white males of the same age group reported to the Los Angeles County Cancer Surveilance Program in 1972-1973 were reviewed. These

	mortality and morbidity data were pooled because of the high mortality rate of lung cancer, and the relatively high accuracy of death certification regarding lung cancer. Of the 3938 subjects, 689 had no reported occupation, and 1222 had no reported industry of employment. One occupational title, and one industry of employment were obtained per subject. The occupation and industry of each subject were coded into one of 417 occupational codes, and one of 215 industry codes, pertaining to the 1970 U.S. Census occupational classification system. The occupations with statistically significant increased Standard Mortality for lung cancer included roofers (SMR = 496%) (Herman and Henderson 1976).

[a] LD_{50}, median lethal dose; LC_{50}, median lethal concentration; LO(A)EL, lowest-observed-(adverse-) effect level; LO(A)EC, lowest-observed-(adverse-) effect concentration; NOAEL, no-observed-adverse-effect level; NOAEC, no-observed-adverse-effect concentration

[b] Assuming 30 grams average body weight

Appendix H. Potency equivalence factors for 16 PAH species

Table H-1. Potency equivalence factors for 16 PAH species

PAH Species	Relative B[*a*]P potency[a]
naphthalene	0.001
acenaphthylene	0.001
acenapthene	0.001
fluorene	0.001
phenanthrene	0.001
anthracene	0.01
fluoranthene	0.001
pyrene	0.001
benz[a]anthracene	0.1
chrysene	0.01
benzo[b+j]fluoranthene	0.1
benzo[k]fluoranthene	0.1
benzo[a]pyrene	1
indeno[1,2,3-cd]pyrene	0.1
dibenz[a,h]anthracene	5
benzo[ghi]perylene	0.01

[a] adapted from Nisbet and LaGoy, 1992