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Canada

Screening Assessment

Coal Tars and Their Distillates

**Environment and Climate Change Canada
Health Canada**

June 2021

Cat. No.: En84-238/2021E-PDF

ISBN 978-0-660-38401-6

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Synopsis

Pursuant to section 68 or 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 six substances referred to collectively as coal tars and their distillates. These six substances were identified as priorities for assessment as they met categorization criteria under subsection 73(1) of CEPA or were considered a priority on the basis of other considerations. Data obtained on these six coal tars and their distillates were used to assess the risk from all coal tars and their distillates. The conclusions of this assessment are therefore considered to cover all coal tars and their distillates, including the six substances with Chemical Abstract Service Registry Numbers (CAS RN¹) and Domestic Substances List (DSL) names listed in the table below.

Table 1. The six substances representing coal tars and their distillates

CAS RN	Domestic Substances List name
8007-45-2	Tar, coal
65996-82-9 ^a	Tar oils, coal
65996-91-0 ^a	Distillates (coal tar), upper
65996-90-9	Tar, coal, low-temp.
65996-89-6 ^a	Tar, coal, high-temp.
65996-93-2	Pitch, coal tar, high-temp.

^a This substance was not identified under subsection 73(1) of CEPA but was included in this assessment as it was considered a priority on the basis of other health concerns.

Coal tars are the condensation products obtained by cooling, to approximately ambient temperature, the gases evolved in the destructive distillation (pyrolysis) of coal. This process occurs at integrated steel mills, with the resulting coal tars often delineated by the pyrolysis temperature (low or high). Coal tar distillates are various boiling point fractions derived from the distillation of coal tars at a coal tar refinery and include both the fractions obtained from the distillation tower and the residue (pitch) that remains following distillation. Coal tars and their distillates are considered to be substances of unknown or variable composition, complex reaction products or biological materials (UVCBs). They are complex mixtures of hydrocarbons (mainly aromatic), phenolics, and heterocyclic oxygen, sulphur and nitrogen compounds.

Coal tar is used as a feedstock in the production of coal-tar-based products, such as oils, creosote, naphthalene, carbon black and coal tar pitch. Following further refinements, coal tar is also an active ingredient present in human and veterinary drugs (therapeutic products), primarily in the form of shampoos used to treat skin conditions, such as psoriasis, eczema and seborrheic dermatitis.

¹ The Chemical Abstracts Service Registry Number (CAS RN) is the property of the American Chemical Society and any use or redistribution, except as required in supporting regulatory requirements and/or for reports to the Government of Canada when the information and the reports are required by law or administrative policy, is not permitted without the prior, written permission of the American Chemical Society.

Coal tar oils and upper distillates are used in industrial applications, such as a feedstock for carbon black and chemical manufacturing. Coal tar pitch is primarily used as a binder in anodes and electrodes, particularly in the aluminum industry, but may also be used as an adhesive/binder in clay pigeons and briquettes to strengthen and impregnate refractories for lining industrial furnaces and in pavement sealants and roofing systems. An estimated 165 to 220 kilotonnes (kt) of coal tar are produced annually in Canada. From this, about 82 to 100 kt of coal tar pitch and an unknown quantity of coal tar oils and coal tar upper distillates are produced.

Coal tars and their distillates may be released to air from activities associated with their production, transportation and storage, as well as to water and soil from product use and disposal. The results of toxicity studies conducted using coal tar products and coal-tar-based pavement sealants indicate that exposure to these products in the environment can lead to adverse effects in organisms. Adverse effects are attributed mainly, but not exclusively, to polycyclic aromatic hydrocarbons (PAHs) present as components in the coal tar substances. For this reason, PAHs have been considered in evaluating the ecological risk of coal tars and their distillates.

PAHs present as major coal tar components demonstrate acute and chronic toxicity to aquatic and terrestrial organisms and, based on this, these substances are considered to present a high hazard to the environment. A comparison of predicted PAH concentrations deposited to soil resulting from the release of PAHs into air at a coal tar refining facility, with no-effect toxicity levels taken from the Canadian Soil Quality Guidelines, determined that PAH concentrations in soil in the vicinity of such facilities have the potential to exceed levels causing adverse effects in organisms. Releases of coal-tar-related PAHs from the same refinery to water are not likely to be causing ecological harm as concentrations are expected to be below the Canadian Water Quality Guidelines. In addition, releases of coal tar substances to water from the application and use of coal-tar-based pavement sealants have the potential to exceed levels that elicit adverse effects in aquatic organisms based on estimated releases of PAHs from these products.

Considering all available lines of evidence presented in this screening assessment, there is a risk of harm to the environment from coal tars and their distillates. It is concluded that coal tars and their distillates meet the criteria under paragraph 64(a) of CEPA as they are entering or may enter 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. However, it is concluded that coal tars and their distillates do not meet the criteria under paragraph 64(b) of CEPA as they are not entering the environment in a quantity or concentration or under conditions that constitute or may constitute a danger to the environment on which life depends.

With respect to human health, PAHs and benzene are regarded as high-hazard components present in coal tar substances. There may be exposure to these high-hazard volatile constituents of coal tars and their distillates to the general population

living in the vicinity of coal tar producers and refineries. The margins of exposure between estimates of exposure to benzene and estimates of cancer potency previously developed for inhalation exposure to benzene are considered potentially inadequate to address uncertainties related to health effects and exposure databases. Additionally, the margins of exposure for the ingestion of house dust containing PAHs associated with the use of coal-tar-based sealants and estimates of cancer potency are considered potentially inadequate to address uncertainties related to health effects and exposure databases.

On the basis of the information presented in this screening assessment,, it is concluded that coal tars and their distillates meet the criteria under paragraph 64(c) of CEPA, as they are entering or may enter the environment in a quantity or concentration or under conditions that constitute or may constitute a danger in Canada to human life or health.

It is therefore concluded that all coal tars and their distillates, including the six substances listed in the table above, meet one or more of the criteria set out in section 64 of CEPA.

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1. Introduction

Pursuant to section 68 or 74 of the *Canadian Environmental Protection Act, 1999* (CEPA) (Canada 1999), the Minister of the Environment and the Minister of Health have conducted a screening assessment of six substances, referred to collectively as coal tars and their distillates, to determine whether they present or may present a risk to the environment or to human health. These substances were identified as priorities for assessment as they met categorization criteria under subsection 73(1) of CEPA or were considered a priority on the basis of other human health concerns (ECCC, HC [modified 2017]). Data obtained on these six coal tars and their distillates were used to assess the risk from all coal tars and their distillates. The conclusions of this assessment are therefore considered to cover all coal tars and their distillates, including these six substances.

Data relevant to the screening assessment of these substances were identified from original literature, review and assessment documents, stakeholder research reports, and recent literature searches up to April 2018. Key studies were critically evaluated, and modelling results were used to inform conclusions.

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

Evaluation of risk to human health involves consideration of data relevant to the estimation of exposure of the general population, as well as information on health effects. Health effects were assessed using toxicological data for coal tars and their distillates, as well as for high-hazard components expected to be present in these substances. Decisions for risk to human health are based on the nature of the critical effect and margins between conservative effect levels and estimates of exposure, taking into account confidence in the completeness of the identified databases on both exposure and effects, within a screening context. The screening assessment does not represent an exhaustive or critical review of all available data. Rather, it presents a summary of the critical information on which the conclusions are based.

This screening assessment was prepared by the CEPA Risk Assessment Program at Health Canada and Environment and Climate Change Canada and incorporates input from other programs within these departments. The human health and ecological portions of this assessment have undergone external written peer review/consultation. Comments on the technical portions relevant to human health were received from Dr. Spencer Williams of Baylor University, Dr. Miriam Diamond of the University of Toronto and officials from the Ontario Ministry of the Environment. Comments on the technical portions relevant to ecological health were received from Dr. Miriam Diamond of the University of Toronto and Geoff Granville of GCGranville Consulting Corp. Additionally, the draft of this screening assessment published on June 10, 2016 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 substances meet the criteria as set out in section 64 of CEPA, by examining scientific information and incorporating a weight-of-evidence approach and precaution.² The screening assessment presents the critical information and considerations on which the conclusions are based.

2. Identity of substances

Coal tars are the condensation products obtained by cooling, to approximately ambient temperature, the gas evolved in the destructive distillation (pyrolysis) of coal (Betts 2000). This process occurs at integrated steel mills, with the resulting coal tars often delineated by the pyrolysis temperature (low or high). Coal tar distillates are various boiling point fractions derived from the distillation of coal tars at a coal tar refinery and include both the fractions obtained from the distillation tower and the residue (pitch) that remains following distillation.

All coal tars and their distillates are complex mixtures of hydrocarbons (mainly aromatic), phenolics, and heterocyclic oxygen, sulphur and nitrogen compounds. As the nature and proportions of the various components are mixed and variable, coal tars and their distillates are considered to be substances of unknown or variable composition, complex reaction products or biological materials (UVCBs) rather than discrete chemicals and, as such, 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

² A determination of whether one or more of the criteria of section 64 of CEPA are met is based on an assessment of potential risks to the environment and/or to human health associated with exposures in the general environment. For humans, this includes, but is not limited to, exposures from ambient and indoor air, drinking water, foodstuffs, and products available to 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.

process. Given their complex and variable compositions, they could not practicably be formed by simply combining individual constituents.

Information on six coal tars and distillates was obtained from stakeholders during the categorization exercise. For the purposes of this document, the common names that will be used for these six substances are listed in Table 2-1. Although information was collected on six substances, this assessment addresses coal tars and their distillates as a group, as other CAS RNs may be described by the substance identities consistent with the coal tar and their distillates definitions given below.

Coal tar (CAS RN 8007-45-2) is a broad term that can include both low- and high-temperature coal tars. The plural form, coal tars, is used as a collective term to represent any coal tar, including CAS RN 8007-45-2 (coal tar), CAS RN 65996-90-9 (low-temperature coal tar) and CAS RN 65996-89-6 (high-temperature coal tar). In addition, the term “coal tar substances” is a generic term for coal tars and their distillates.

Low-temperature coal tar (CAS RN 65996-90-9) is the condensation product obtained under low-temperature (below 700 °C) pyrolysis conditions, while high-temperature coal tar (CAS RN 65996-89-6) is the distillation product formed from the pyrolysis of coal at temperatures above 700 °C (European Commission 1976; NCI 2010).

Coal tar oils (CAS RN 65996-82-9), coal tar upper distillates (CAS RN 65996-91-0) and high-temperature coal tar pitch (HTCTP; CAS RN 65996-93-2) are distilled from high-temperature coal tar (Blümer and Sutton 1998; personal communication, email from Ruetgers Canada Inc., to Risk Management Bureau, Health Canada, dated August 2013, unreferenced). Coal tar oils have an approximate distillation range of 130 to 250 °C, while the upper distillates have an approximate distillation range of 220 to 450 °C (NCI 2010). HTCTP (CAS RN 65996-93-2) is the solid distillation residue obtained from high-temperature coal tar and has an approximate softening point of 30 to 180 °C (NCI 2010). HTCTP can be further distilled into various pitch subfractions with different softening points for different uses (PCTC 2018; IARC 1985). However, each subfraction is described by the same CAS RN (65996-93-2).

The composition and properties of coal tars depend on the carbonization temperature and, to a lesser extent, on the nature of the coal used as feedstock (IARC 1985). The importance of the carbonization temperature in determining coal tar composition was demonstrated by Novotny et al. (1981), who found a high degree of similarity in the proportions of major components measured in crude coal tar samples obtained from four very different mining sites and process plants, but that were coked at similar temperatures.

Table 2-1 summarizes the main components of the six coal tars and distillates.

Table 2-1. Composition of select coal tars and their distillates

CAS RN and DSL name^a	Common name	Composition
8007-45-2 Tar, coal	Coal tar	Complex combination of aromatic hydrocarbons, phenolic compounds, nitrogen bases and thiophene.
65996-90-9 Tar, coal, low-temp.	Low-temperature coal tar	Composed primarily of polycyclic aromatic hydrocarbons, phenolic compounds, aromatic nitrogen bases and their alkyl derivatives.
65996-89-6 Tar, coal, high-temp.	High-temperature coal tar	Composed primarily of a complex combination of polycyclic aromatic hydrocarbons. May contain minor amounts of phenolic compounds and aromatic nitrogen bases.
65996-82-9 Tar oils, coal	Coal tar oils	Composed primarily of naphthalene, alkylnaphthalenes, phenolic compounds and aromatic nitrogen bases.
65996-91-0 Distillates (coal tar), upper	Coal tar upper distillates	Composed primarily of three- to four-membered polycyclic aromatic hydrocarbons and other hydrocarbons.
65996-93-2 Pitch, coal tar, high-temp.	High-temperature coal tar pitch (HTCTP)	Composed primarily of a complex combination of three- or more-membered polycyclic aromatic hydrocarbons.

Source: ESIS c1995-2011.

^a DSL, *Domestic Substances List*.

Data obtained on these six coal tars and their distillates were considered representative of the characteristics of all coal tars and their distillates.

Polycyclic aromatic hydrocarbons (PAHs) are the major components of coal tar substances. PAHs are organic compounds comprising two or more fused aromatic rings in various arrangements and containing only carbon and hydrogen (Environment Canada, Health Canada 1994). High-temperature coal tars have a higher PAH content than coal tars formed under low-temperature conditions, while low-temperature coal tars contain a higher proportion of phenolic and heterocyclic compounds and have lower pitch content (Kleffner et al. 1981). Coal tar oils, coal tar upper distillates and HTCTP are all derived from high-temperature coal tars and are therefore expected to contain a high proportion of PAHs. For example, the European Commission (2008) reported that HTCTP contains approximately 80% PAHs, including 14 PAHs that are listed as priority pollutants by the United States Environmental Protection Agency (U.S. EPA 2013).

No data were found on the composition of coal tar oils and coal tar upper distillates. Coal tar oils, which are also called tar acid oil and naphthalene oil (personal communication, email from Ruetgers Canada Inc. to Risk Management Bureau, Health Canada, dated August 2013; unreferenced), are expected to contain only one PAH (i.e., naphthalene), as their boiling point range of 168 to 202 °C (Table 3-2) is too low to include any other PAHs. They are also expected to contain tar acids, such as phenols, xylenols and cresols. Coal tar upper distillates, which are also called heavy aromatic oil (personal communication, email from Ruetgers Canada Inc. to Risk Management Bureau, Health Canada, dated August 2013, unreferenced), contain the following substances based on their boiling point range of 307 to 365 °C (Table 3-3): phenanthrene, anthracene, benzo[e]pyrene, acridine, carbazole and dibenzothiophene.

Alkylated homologues of the parent PAHs are likely to be present as minor components in high-temperature coal tars (Wise et al. 1988, 2010). Wise et al. (1988, 2010) identified alkylated PAHs, including methylated naphthalenes, phenanthrenes, pyrenes, chrysenes and fluoranthenes, as minor components of a high-temperature coal tar. The proportion of PAHs, as well as the presence and distribution of alkylated PAHs, can be used to identify the source of PAH-containing substances in the environment. Non-alkylated parent PAHs are primarily formed under high-temperature conditions and indicate pyrogenic sources, while alkylated PAHs are indicative of carbon compounds exposed for extended periods to lower temperatures, such as petroleum sources (LaFlamme and Hites 1978; Sporstøl et al. 1983). However, methyl- and/or dimethyl-substituted naphthalenes have been shown to be formed by the pyrolysis of coal and coal tar at 600 °C and 1000 °C (Ledesma et al. 2000). As coal tars and their distillates are products of combustion, the parent PAHs are expected to predominate over their alkylated homologues.

Further compositional information on coal tars and their distillates is available in the supporting document (Environment Canada 2015a).

3. Physical and chemical properties

The physical and chemical properties of coal tars and their distillates vary according to the primary constituents present, which are in turn determined by factors such as the origin and composition of the coal and the carbonization temperature used.

Lewis (2001) characterized coal tar as an almost black liquid or semisolid that is heavier than water and slightly alkaline, with a characteristic naphthalene-like odour. The substance is only slightly soluble in water, but is soluble in organic solvents, such as ether, benzene, carbon disulfide and chloroform, and partially soluble in alcohol, acetone and methanol (Lewis 2001).

Limited information was found on the physical and chemical properties of coal tars, coal tar oils and coal tar upper distillates. However, some data are available for HTCTP. A read-across approach was used to estimate the properties of coal tars and their

distillates based on the properties of related substances as provided by the coal sector under the European Union's Registration, Evaluation, Authorisation and Restriction of Chemicals (REACH) regulation (R4CC 2011; ECHA 2013). These are summarized in Tables 3-1 through 3-4. It should be noted that coal tars and their distillates are complex mixtures with compositions that vary from batch to batch, and in many cases only single values were found for physical-chemical properties that would be better represented by a range of values. This variability of the physical-chemical properties and the lack of data on them are a source of uncertainty in this assessment.

Table 3-1. Estimated physical and chemical properties of coal tars (CAS RNs 8007-45-2, 65996-90-9 and 65996-89-6) (R4CC 2011; ECHA c2007-2017a)^a

Property	Value	Temperature (°C)
Physical state	Viscous liquid	20
Melting point (°C)	-9	-
Boiling point (°C)	> 215 to < 350	-
Density (g/cm ³)	1.18	-
Vapour pressure (Pa)	1091	20
Water solubility (mg/L)	≤ 1.7	20
Flash point (°C)	77	-
Auto flammability (°C)	> 560	-

^a Based on read-across from high-temperature coal tar (CAS RN 65996-89-6).

Table 3-2. Estimated physical and chemical properties of coal tar oils (CAS RN 65996-82-9) (R4CC 2011; ECHA c2007-2017b)^a

Property	Value	Temperature (°C)
Physical state	Liquid	20
Melting point (°C)	-16	-
Boiling point (°C)	168 to 202	-
Distillation range (°C)	130 to 250	-
Density (g/cm ³)	0.99	20
Vapour pressure (Pa)	1700	21
Water solubility (mg/L)	2 to 60	20
Flash point (°C)	51.5	-
Auto flammability (°C)	595	-

¹ Based on read-across from related substance, distillates (coal tar) light oils (CAS RN 84650-03-3).

Table 3-3. Estimated physical and chemical properties of coal tar upper distillates (CAS RN 65996-91-0) (R4CC 2011; ECHA c2007-2017c; NCI 2010)

Property	Value	Temperature (°C)
Physical state	Liquid	20
Melting point (°C)	46	-
Boiling point (°C)	258 to 409	-
Distillation range (°C)	220 to 450	-
Density (g/cm ³)	1.104	15
Vapour pressure (Pa)	38.5 ^a	20
Vapour pressure (Pa)	52 ^a	25

Vapour pressure (Pa)	203 ¹	50
Vapour pressure (Pa)	1300 ¹	103
Water solubility (mg/L)	37	20
Flash point (°C)	125	-
Auto flammability (°C)	> 500	-

^a Data for ECHA key study (reliability 1 without restrictions) using acceptable surrogate substance, No. 5 Wash oils ATE-No. 10441 (no CAS RN provided) as read-across (ECHA c2007-2017c).

Table 3-4. Physical and chemical properties of HTCTP (CAS RN 65996-93-2) (ECHA c2007-2017d; European Commission 2008)

Property	Value	Temperature (°C)
Physical state	Solid	20
Melting point (°C)	116 to 150	-
Boiling point (°C)	> 360	-
Density (g/cm ³)	> 1.15 to < 1.4	20
Vapour pressure (Pa)	0.0000058	20
Vapour pressure (Pa)	0.00026	50
Vapour pressure (Pa)	2900	294
Water solubility (mg/L)	0.0008 to 0.091	21
Flash point (°C)	> 200	-
Auto flammability (°C)	> 560	-

The data presented in Tables 3-1 to 3-4 on the six coal tars and their distillates are considered representative of the characteristics of all coal tars and their distillates.

To predict the physical-chemical properties and ecological fate of coal tars and their distillates, representative structures were chosen from each chemical class contained within the substance (Environment Canada 2015a). As the composition of these substances is variable and not well defined, representative structures could not be chosen based on their proportion in the mixture. Representative structures for PAHs, one-ring aromatics, phenols and heterocyclic substances were selected.

It should be noted that the physical and chemical behaviour of the individual representative structures will differ if these substances are present in a mixture, such as in coal tars or their distillates. The vapour pressures of components of a mixture will be lower than their individual vapour pressures based on 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 when they are present individually (Banerjee 1984; Di Toro et al. 2007). However, when an individual petroleum hydrocarbon chemical that is normally solid under environmental conditions is part of a petroleum mixture (or a UVCB) such as in this instance, components that are normally solid under environmental conditions may be found in a liquid state due to the lowering of its melting point when in a mixture (Di Toro et al. 2007). Presence of the hydrocarbon that is normally solid in a mixture thus results in an increase in its vapour pressure and water solubility, as determined by the subcooled vapour pressure

(Staikova et al. 2005) and subcooled solubility (Di Toro et al. 2007) of the hydrocarbon that is normally solid. The subcooled vapour pressure or solubility is used to determine the contribution of a normally solid component to the overall vapour pressure or solubility of a petroleum substance. Nevertheless, the physical and chemical properties of the individual representative structures, as reported in Environment Canada (2015b), give an indication of how these individual components of the mixture may behave in the environment.

As noted, PAHs are major components of most coal tars and their distillates. PAHs may be divided into two groups based on molecular weight, with low-molecular-weight (LMW) PAHs having two or three benzene rings and molecular weights of 152 to 178 g/mol, and high-molecular-weight (HMW) PAHs having four to seven rings and molecular weights in the range of 202 to 278 g/mol (ATSDR 1995; Burgess et al. 2003). The two groups exhibit very different physical and chemical properties, which in turn influence environmental fate and toxicity. Most major coal tar constituent PAHs are in the HMW group and have low water solubilities, vapour pressures and Henry's law constants, and high partition coefficients (K_{ow} , K_{oc}). The major LMW PAHs present in coal tar substances, especially coal tar upper distillates (i.e., naphthalene, phenanthrene and anthracene), display somewhat higher water solubilities and vapour pressures. Naphthalene, the PAH with the lowest molecular weight, which is the major PAH constituent of coal tar oils, has moderate water solubility and vapour pressure and low to moderate partition coefficient values.

Much greater variability is evident in the non-PAH coal tar components, which display a range of physical and chemical properties. In general, substances with a single aromatic ring and no or few alkyl-based side groups (e.g., benzene, toluene, styrene, ethylbenzene and xylenes) exhibit high vapour pressure, moderate to high water solubility and low partition coefficients. Single-ring structures with phenolic side groups, such as phenol, cresol, methylresorcinol and xylenol, exhibit lower vapour pressure, increased water solubility and low partition coefficients. With the addition of benzene rings or increased alkylation to the molecule (e.g., pyridine, quinoline, carbazole and acridine), both the vapour pressure and water solubility decrease, and partition coefficients increase.

The physical-chemical properties of major PAH and non-PAH coal tar components are available in Environment Canada (2015a). Throughout this screening assessment, where information specific to the coal tar substance is not available, information relating to its major components, and in particular PAHs, will be used to infer the behaviour of the substance as a whole.

4. Sources

Coal tars are produced in Canada as a by-product of the coke-making operations of integrated iron and steel mills and are also imported into Canada (Sutton 2008, ECCC 2019). Coal tar distillates, including coal tar oils (CAS RN 65996-82-9), coal tar upper

distillates (CAS RN 65996-91-0) and high-temperature coal tar pitch (CAS RN 65996-93-2), are produced by the further distillation of high-temperature coal tars at a coal tar refining facility. There are integrated iron and steel mills in Canada, which manufacture coke and thus coal tars. Coal tar distillates are also imported into Canada (ECCC 2019).

Coal tars that are produced in Canada are classified as either CAS RN 8007-45-2 (the generic coal tar descriptor) or CAS RN 65996-89-6 (high-temperature coal tar), but not CAS RN 65996-90-9 (low-temperature coal tar) because the temperatures used in coke-making operations of integrated iron and steel mills are well above 700 °C (WCI 2007). A temperature of 700 °C was specified in the CAS RN definition as the delineator between high-temperature and low-temperature coke-making operations (NCI 2010).

Information on the manufacture and import of low-temperature coal tar (CAS RN 65996-90-9), coal tar oils (CAS RN 65996-82-9) and coal tar upper distillates (CAS RN 65996-91-0) during the 2011 calendar year was collected by means of a survey issued pursuant to section 71 of CEPA (Canada, Dept. of the Environment 2012). Canadian manufacture and import of coal tar oils was reported by steel mills and coal tar refineries to be in the range of 100 to 1000 kt in 2011. No manufacture or import of low-temperature coal tar (65996-90-9) was reported above the reporting threshold of 100 kg. Coal tar upper distillates were not reported to be manufactured or produced in Canada in 2011, but were reported to be imported by coal tar refineries in the range of 1 to 100 kt (Environment Canada 2013a). An additional survey issued pursuant to section 71 of CEPA (Canada 2018) for the calendar year 2017 for all six substances indicated similar manufacture and import activities.

According to Sutton (2008), an estimated 165 to 220 kt of coal tar are produced and refined annually in Canada. This estimate is higher than the quantity reported from the survey conducted pursuant to section 71 of CEPA for the 2017 calendar year (ECCC 2019). Given the uncertainty in the 2017 data resulting from the fact that the survey focused mainly on paving and roofing applications, the values from Sutton (2008) are still considered relevant for the purpose of this assessment. Actual production varies from year to year, driven primarily by increases and decreases in the demand for steel (Sutton 2008). Changes in the production of steel lead directly to changes in the production of coke and coal tars as by-products. Distillation of coal tar produces about 50% by weight of coal tar pitch (European Commission 2008). Therefore, about 82 to 110 kt of coal tar pitch are produced annually in Canada, based on the Sutton's estimate of Canadian coal tar production (Sutton 2008).

In Canada, approximately 307 kt per year of coal tar pitch are used as binder in anodes by the aluminum industry (Sutton 2008). Therefore, a quantity of coal tar pitch is imported to meet the demand.

5. Uses

The dominant use of coal tar is in the production of substances such as creosote, crude naphthalene, carbon black feedstock and coal tar pitch, after undergoing distillation (NTP 2005). Distillation of coal tar produces several oil fractions and pitch in proportions of approximately 1% light oil, 2% carbolic oil, 10% naphthalene oil, 10% wash oil, 10% anthracene oil, 12% base oil and 50% pitch (European Commission 2008). Note that coal tar oils (CAS RN 65996-82-9) include tar acid oil and naphthalene oil and that coal tar upper distillates (CAS RN 65996-91-0) include wash oil and heavy aromatic oil (ECCC 2018). Coal tar oils and coal tar upper distillates are used as feedstocks for industrial processes. Coal tar oils are used in organics recovery and to produce naphthalene as well as naphthalene sulphonates for use in superplasticizers (personal communication, email from Ruetgers Canada Inc. to Risk Management Bureau, Health Canada, dated August 2013, unreferenced). Coal tar upper distillates are also used in the production of creosote for use in wood preservation and in carbon black production. Coal tar is listed in the Drug Product Database as an active ingredient present in human and veterinary drugs (therapeutic products), primarily in the form of shampoos, and is used to treat skin conditions, such as psoriasis, eczema and seborrheic dermatitis (DPD 2014).

Coal tar pitch is primarily used by aluminum smelters as a binder for aluminum smelting anodes (ATSDR 2002). It is also used as a binder in graphite electrodes and as an adhesive/binder in clay pigeons and briquettes. Additionally, it can be used to strengthen and impregnate refractory brick (for lining industrial furnaces) and in pavement sealants and built-up roofing systems for flat and low-slope roofs (NTP 2005; European Commission 2008; EHS 2010). Coal tar pitch can also be used as fuel in blast furnaces in the steel industry and in surface coatings for industrial applications, such as pipe linings and harsh climate protection (European Commission 2008).

In Canada, coal tar pitch may be used in roofing systems for buildings with flat roofs (Conestoga 2014). One HTCTP-containing roofing product material safety data sheet was identified, which states that the product contains 100% HTCTP (SDS 2013). However, there appears to be very low usage of coal tar pitch in built-up roofing systems (BUR) used for low-slope roofs in Canada. According to a survey by the Canadian Roofing Contractors' Association (CRCA ca. 2001), coal-tar-pitch-based BUR is not being used in new construction and only represented 0.1% of re-roofing sales in 2000. The CRCA believes that usage of coal tar pitch in BUR is currently even lower (e-mail from CRCA to Products Division, Chemicals Sector Directorate, Environment Canada, dated 2014, unreferenced). Information reflecting imports during the 2017 calendar year confirms use of at least one coal tar substance in roofing applications (ECCC 2019).

Coal-tar-pitch-based epoxy coatings, containing 10% to 30% w/w coal tar pitch, are typically used in locations that require corrosion protection and/or chemical resistance in sub-surface, underwater or chemical immersion applications, such as heavy-duty structural coatings for steel or concrete piles, ship hulls, petroleum storage tanks, water treatment facilities, and non-potable water storage tanks and pipe coatings (Cloverdale Paint 2013). Most coal tar epoxy coating applications appear to be industrial in nature,

and any potential exposure of the general population is expected to be minimal. Coal-tar-pitch-based epoxy coatings are manufactured in Canada by one company, from imported coal tar pitch (e-mail from the Canadian Paint and Coatings Association to Environment Canada, dated 2013, unreferenced). The impact on the environment from these coatings is anticipated to be minimal, as the volumes used are expected to be relatively low given that their use is limited to specialized industrial applications.

Coal-tar-based pavement sealant (CTPS) constitutes one of several types of pavement sealants and contains coal tars and/or their distillates. CTPS is generally described as containing HTCTP (EHS 2010; personal communication, email from Ruetgers Canada Inc. to Risk Management Bureau, Health Canada, dated August 2013, unreferenced; personal communication, correspondence from Pavement Coatings Technology Council, Alexandria, VA, to Ecological Assessment Division, Environment and Climate Change Canada, dated August 2016, unreferenced). However, some manufacturers identify other coal tar substances, such as coal tar (CAS RN 8007-45-2), as an ingredient (Neyra Industries Inc. 2011, 2014). In the pavement sealant industry, sealants containing HTCTP are referred to as refined coal-tar-based pavement sealants and utilize a specific distillation fraction of HTCTP that meets the product specifications (e.g., specific gravity, softening point, etc.) for road tar RT-12, a medium-soft pitch, as given in ASTM D490 (ASTM 2011; PCTC 2018). CTPS generally contains about 15% to 30% coal tar substance emulsified in water (EHS 2010).

CTPS is available in the Canadian retail market and has limited availability in stores across Canada in do-it-yourself products used by consumers (EHS 2010). Pavement sealing contractors are known to use CTPS, but detailed information is limited (EHS 2010). Pavement sealants are mainly applied to residential driveways and small commercial or residential parking lots; they are not applied to roadways or airport tarmacs in Canada (EHS 2010). It is estimated that $10\,500 \pm 50\%$ tonnes/year of CTPS are sold in Canada by a small number of companies, mostly in the retail sector (EHS 2010). This range is considered reasonable as it was derived using best estimates following a market review of pavement sealants used in Canada (EHS 2010) and is corroborated by quantity information for this application collected for the 2017 calendar year through a section 71 survey (ECCC 2019). Diamond Environmental Group (2011) found that 23% of 92 driveways and parking lots sampled in February and March of 2011 in the greater Toronto area were sealed with CTPS. Of the 22 sites sealed with CTPS, 21 were driveways and only 1 was a parking lot.

An additional use of HTCTP is as an asphalt rejuvenator, a product used on roadways, tarmacs and other asphalt surfaces, that penetrates the asphalt surface to replace tars and bitumens lost during the aging of the asphalt. This differs from CTPS, which coats the asphalt only (RejuvaSeal c2018). One product containing both HTCTP (CAS RN 65996-93-2; 40% to 60%) and coal tar upper distillates (CAS RN 65996-91-0; 5% to 15%) was identified for this use (Pavement Rejuvenation International 2014).

None of the six priority coal tars and distillate substances in this assessment are listed as approved food additives in the Lists of Permitted Food Additives as regulated under

the *Food and Drugs Act* (Health Canada 2013). Based on the nature of these substances, it is unlikely that any of these substances would be used in food flavours or components of fruit and vegetable coatings (e-mail from Food Directorate, Health Canada, to Risk Management Bureau, Health Canada, May 2010, unreferenced). None of the substances are identified as being used in food packaging applications or incidental additives (e-mail from Food Directorate, Health Canada, to Risk Management Bureau, Health Canada, May 2010, unreferenced).

Coal tars, crude and refined, are described on the Cosmetic Ingredient Hotlist as prohibited as ingredients in cosmetic products in Canada. 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 under the *Food and Drugs Act* or (b) a provision of the *Cosmetic Regulations* (Health Canada 2008).

Coal tar is listed in the Natural Health Products Ingredients Database (NHPID) as a non-natural health product substance not falling under Schedule 1 of the *Natural Health Products Regulations* and thus not listed in the Licensed Natural Health Products Database (LNHPD) as being present in any currently licensed natural health products (NHPID 2014; Canada 2003; LNHPD 2014). None of the six coal tars and distillate substances identified in this assessment are listed in the NHPID or LNHPD as medicinal or non-medicinal ingredients present in licensed natural health products in Canada (NHPID 2014; LNHPD 2014).

In addition to these six substances, other coal tars and their distillates are expected to have similar use patterns.

6. Releases to the environment

Coal tars and their distillates may be released from activities associated with their production, processing, handling, transportation and storage, as well as during use and disposal of consumer and industrial products that contain them. Within this screening assessment, releases of all coal tars and their distillates from integrated steel mills and coal tar refining facilities that process and/or handle and store these substances are considered together. Releases of coal tars or distillates during their transport and/or use are also assessed.

Coal tars are produced as by-products of coke production at integrated steel mills in Canada, and there is potential for release of coal tar or its components during production, transportation and storage. Coal tar is produced by the heating of coal at high temperatures; thus, there is potential for release of coal tars or their components from relief valves, venting valves or drain valves on the piping or equipment (e.g., vessels).

The primary use of coal tars in Canada is in the production of substances such as creosote, naphthalene, carbon black and coal tar pitch. The distillation and processing

of coal tars to produce these substances takes place at coal tar refineries in the case of creosote or pitch, or at processing facilities, which obtain coal tar distillates to use as feedstocks for products such as refined naphthalene and carbon black (European Commission 2008). Coal tar oil, upper distillates and pitch are formed during the processing of coal tars. All processing activities take place within an industrial setting with control systems that reduce releases of coal tars and their distillates to the environment. However, there is still potential for release of the coal tars and their distillates, both at the plant and during transport of these substances to other processing facilities. Information provided by industry has indicated that the exact substance (coal tars, distillates or coal tar pitch) from which emissions are originating cannot be identified due to extensive vapour capture and emissions control systems that are interlinked throughout the facility (personal communication, email from Ruetgers Canada Inc. to Risk Management Bureau, Health Canada, dated August 2013, unreferenced). Thus, releases from the coal tar refinery are considered to apply to all coal tars and their distillates in this assessment.

Both coal tar oils and coal tar upper distillates are industry-restricted substances that can be either further processed on-site at the coal tar processing facility or transported to other industrial facilities where they are either consumed or transformed into different substances. These substances are not marketed to the general public. Coal tar oils are used as industrial fuel, in organics recovery, and in naphthalene refining and the production of naphthalene sulphonates (superplasticizers) (personal communication, email from Ruetgers Canada Inc. to Risk Management Bureau, Health Canada, dated August 2013, unreferenced). Coal tar upper distillates are used to produce creosote and carbon black feedstock (personal communication, email from Ruetgers Canada Inc. to Risk Management Bureau, Health Canada, dated August 2013, unreferenced). Releases of coal tar oils and upper distillates may occur during loading, unloading and transport between industrial facilities or during storage at these facilities. Releases from washing or cleaning transportation vessels are not considered in this assessment, as tanks or containers for transferring coal tar substances are typically dedicated vessels and therefore washing or cleaning is not required on a routine basis (U.S. EPA 2008).

Releases may also occur from the use of therapeutic products that contain coal tar, such as some creams, lotions and shampoos. Potential ecological exposure to the PAHs present in these therapeutic products is expected to be minimal as these products are used in the treatment of specific skin conditions and represent a small fraction of sales in their respective product categories in Canada. Most coal-tar-containing therapeutics are formulated to contain 1% to 10% coal tar (Health Canada 2006), thereby limiting the amounts entering receiving water. PAH levels from therapeutics will be further reduced as a result of wastewater treatment, environmental biodegradation and/or drinking water treatment prior to consumption (Pham and Proulx 1997).

Coal tar pitch is used primarily as a binder in anodes for aluminum smelting (ATSDR 2002) and in graphite electrodes (European Commission 2008), which can result in the release of coal tar components to air. Releases of PAHs from the use of coal tar pitch in

anodes at aluminum smelters were examined in Environment Canada, Health Canada (1994), and risk management actions to address this source have been implemented. Therefore, anodes as a release source of pitch are not considered further in this assessment. As for its use as a binder in graphite electrodes, the sole manufacturer of graphite electrodes in Canada closed in the first quarter of 2014 (Steel Times International 2013; 2015 telephone communication, SGL Inc. and Mining and Processing Division, Industrial Sectors Directorate, Environment Canada, unreferenced).

Coal tar pitch is also used in CTPS and in some built-up roofing systems that employ coal-tar-pitch-based products. Releases from the use of CTPS are discussed in detail in the Ecological Exposure and Potential to Cause Harm to Human Health sections of this report. As the use of coal tar pitch in built-up roofing systems in Canada is expected to be lower than other applications and would contribute incrementally to overall releases (see Uses section), releases of substances from such roofing systems are not evaluated in this report. Also not evaluated here is the use of coal-tar-pitch-based epoxy surface coatings. The impact on the environment from these surface coatings is anticipated to be minimal. Their usage volume is expected to be relatively low, as their use is limited to specialized industrial applications (see Uses section).

Spills data for the five years from 2008 to 2012 were obtained for the province of Ontario, where the only coal tar refinery in Canada and all Canadian integrated steel mills are located (Ontario 2013). At all industrial sites, a total of 16 spills of coal tars and their distillates (average of 3.2 spills/year) were reported, for a reported total of 39 862 L over five years (average of 7 972 L/year). All of the industrial spills were to land, except one to air and one 115 L spill of wash oil on the pier at a port.

There were also a total of 29 spills (average of about 6 spills/year) reported in the categories of “tar,” “tar and water mixture,” “driveway sealer,” “asphalt sealer,” “sealant (not otherwise specified (N.O.S.)),” “tar base caulking,” and “coal tar distillates (N.O.S.),” totalling 22 556 L over five years (average of about 4 500 L/year), which are products that may, but do not necessarily, contain coal tar pitch. These spills occurred at non-industrial sites. Tar identified as being released by roadway maintenance companies or during road maintenance activities was not included in this total, as coal-tar-pitch-based pavement sealants are not applied on roads (see Uses section).

Historically, coal tars were produced as a by-product of coal gasification to provide fuel for heating and lighting; coal tars therefore exist as contaminants at many former coal gasification sites in Canada. A national inventory carried out in 1987 recorded over 150 coal gasification sites across the country. These sites are located in all provinces except Prince Edward Island, with the greatest site densities found near major urban centres in Quebec (Montreal), Ontario (Toronto) and British Columbia (Vancouver) (RDRC 1987). Site assessments have been conducted on the majority of coal gasification sites and most are subject to remediation and/or risk management activities (MENVIQ 1988; RDRC 1987). As well, environmental protection measures have been implemented at many coal-tar-related industries in Canada, particularly for steel plants equipped with

coke ovens (SLV 1996; EMA 1997, 2000; Environment Canada 2001). Historical contamination at such sites is not considered further in this assessment.

7. Environmental fate and behaviour

When coal tar or its distillates are released into the environment, four major fate processes will take place: dissolution in water, volatilization, biodegradation and adsorption. These processes will cause changes in the composition of these UVCB substances over time. In the case of spills on land or water surfaces, photodegradation—another fate process—can also be significant. Three of the processes—dissolution in water, volatilization and biodegradation—typically result in the depletion of the more readily soluble, volatile and degradable compounds and the accumulation of those most resistant to these processes in residues. The gradual compositional changes and accompanying changes in physicochemical and fate properties that occur over time are referred to as aging or weathering of the coal tar substance.

As noted previously, the solubility and vapour pressure of components within a mixture will differ from those of the component alone. These interactions are complex for complex UVCBs, such as coal tars.

Due to the complex interaction of components within a mixture that 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 coal tars and their distillates, the physical-chemical properties of representative structures of these substances (Appendix 3 of Environment Canada 2015a) were examined.

When coal tars are released into the environment, more volatile components, such as benzene and naphthalene, may volatilize into the air, while water soluble components, such as cresols, may dissolve in water over time. Components entering air or water are subject to transport away from the source. As coal tars are substances that are denser than water, they behave as dense non-aqueous phase liquids (DNAPLs). Coal tar released into water is expected to sink through the water column, settling onto or into the bed sediment. More soluble components present in the coal tar will dissolve into the surrounding aqueous medium or seep into the sediment bed via sediment pore water. Coal tar released into soil will likely remain within this medium, with volatile components possibly moving upward through the soil matrix via interstitial air spaces until they reach the surface and dissipate into the air. More water soluble components may dissolve into soil pore water and, in this way, may be transported away from the coal tar.

The coal tar oils, which have a boiling point range of 168 to 202 °C (Table 3-2), are expected to contain the representative structures naphthalene, phenols, cresols and xlenols. The coal tar oils are liquid at ambient temperatures and have a density slightly less than that of water (0.99 g/cm³), a low to moderate water solubility range of 2 to 60 mg/L and a high vapour pressure of 1700 Pa (Table 3-2). Therefore, they will volatilize

and also dissolve in water to some extent, especially the more soluble components, such as phenols, cresols and xylenol, which have high to very high water solubilities. Since these representative structures have low to moderate log K_{oc} values, they will mainly be found in the water column and will not be heavily sorbed to sediment. They have moderate vapour pressures, meaning that they will volatilize to some extent.

Based on the boiling point range of 307 to 365 °C (Table 3-3), it is expected that the upper distillates will contain the representative structures phenanthrene, anthracene, benzo[e]pyrene, acridine, carbazole and dibenzothiophene, which have boiling points within this range. The upper distillates, which are solids at ambient temperatures (Table 3-3), have a density slightly greater than that of water, a moderate vapour pressure and a low water solubility, which means that they will sink in water and volatilize to some extent. Of the representative structures contained in the upper distillates, most have low vapour pressures and water solubilities, so they will not volatilize or dissolve quickly. Most have high log K_{oc} values, meaning that they will tend to sorb to organic matter, such as that contained in soil or sediment.

Coal tar oils and coal tar upper distillates contain a significant proportion of more volatile components, including naphthalene and other low molecular weight PAHs, monoaromatics such as benzene and ethylbenzene, and phenolic compounds such as phenol and cresols. When released into the air or soil, coal tar oils and upper distillates are expected to release these components into air through volatilization. High-temperature coal tar pitch is composed primarily of HMW PAHs. These PAHs have low volatilities and will not undergo significant volatilization when the pitch is released into air or soil. HMW PAHs also have low water solubilities and will not distribute significantly into water. For this reason, high-temperature coal tar pitch is not expected to undergo significant volatilization or dissolution of its major components when released into the environment. The high density of coal tar pitch indicates that when released into water, it will tend to settle out of the water column onto bed sediment. The high log K_{oc} values, as well as the planarity of the HMW PAHs which are its primary components, suggest that high-temperature coal tar pitch will tend to adsorb to the organic fraction of soil and sediment.

High log K_{oc} values for the HMW PAHs (a range of 4.8 to 6.3) (Appendix 3 of Environment Canada 2015a) indicate that when released into water, these substances will readily sorb to suspended solids and bed sediment. Because they have low Henry's law constants, volatilization from water surfaces is not expected to be an important fate process for HMW PAHs. While most HMW PAHs have very low water solubilities (a range of 0.00026 to 0.26 mg/L at 25 °C; Appendix 3 of Environment Canada 2015a), some limited dissolution may occur and, therefore, a proportion of the total quantity released is predicted to reside in the water column. This was confirmed in research conducted by Rostad et al. (1985), who reported the presence of poorly soluble HMW PAHs at low concentrations in the aqueous fraction of coal-tar-contaminated groundwater samples.

LMW PAHs and most non-PAH coal tar components are more soluble in water than HMW PAHs and, when released into this medium, are expected to primarily remain within the water column. Sediments are the major environmental reservoir for PAHs with four or more rings released into water (Environment Canada, Health Canada 1994). Some distribution into sediment may also occur with substances having lower solubilities and higher partition coefficients (e.g., phenanthrene). As well, some volatilization from the water surface is expected for those substances having moderate to high vapour pressures and Henry's law constants (e.g., monoaromatics such as benzene, naphthalene).

Hyun et al. (2010) reported that coal tar reaching a water body will separate into lighter components (e.g., LMW PAHs), which form a pool on the top of the water table, and denser components (e.g., HMW PAHs), which then migrate downwards in the water column to form coal-tar-sediment mixtures. As aging of coal tar in the contaminated sediment takes place, various organic solutes present in the tar are released at differing rates, resulting in the coal-tar-contaminated sediment acting as a long-term source of contamination to the aquatic environment (Hyun et al. 2010).

8. Persistence and bioaccumulation potential

Due to the complex nature of coal tars and their distillates, persistence and bioaccumulation potential of components of these substances is characterized based on empirical and/or modelled data for a suite of representative structures expected to be present within them. These representative structures do not represent all possible structures in coal tar substances and it is recognized that their proportion in each individual coal tar or coal tar distillate substance will differ substantially depending on the boiling point range of the substance. These representative structures also do not necessarily represent the full range of persistence or bioaccumulation potential present in any given chemical class of structures (e.g., phenols and PAHs). Thus, the modelling results give a general indication of the persistence and bioaccumulation potential of coal tars and their distillates.

8.1 Environmental persistence

The persistence of a suite of representative structures expected to occur in coal tars and their distillates was characterized based on empirical and modelled data. Empirical study descriptions, model results and the weighing of information are reported in Environment Canada (2015a).

Most coal tar constituent substances, such as PAHs, phenolics (i.e., phenol, cresols, xylenols, naphthol) and most heterocyclics (i.e., nitrogen-, oxygen- and sulphur-substituted aromatics, such as quinoline and dibenzothiophene) are rapidly degraded in air and have atmospheric half-lives of less than two days (Environment Canada 2015a). Exceptions are benzene, toluene, ethylbenzene, pyridine and dibenzofuran, which have estimated atmospheric half-lives of greater than two days and thus may undergo long-

range atmospheric transport to regions far from their source. As well, many three- to six-ring PAHs sorb to particulate matter in the atmosphere and may undergo long-range atmospheric transport by being carried from their source to remote areas along with the particles to which they are sorbed (Arey and Atkinson 2003; Peters et al. 1995; AMAP 2004; Becker et al. 2006; Wang et al. 2010a, 2010b).

Considering biodegradation in water, soil and sediment, the following representative structures are expected to have half-lives greater than six months in water and soil and greater than one year in sediment: carbazole, dibenzothiophene, dibenzofuran and all PAHs, with the exceptions of naphthalene and phenanthrene (Environment Canada 2015a). Quinoline is also expected to have a half-life longer than one year in sediment.

8.2 Potential for bioaccumulation

Bioaccumulation potential of a suite of representative structures of coal tar and their distillates 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} values greater than about 4.5 (Arnot and Gobas 2003). As most components of coal tar substances have log K_{ow} values greater than 4.5, BAFs are considered to more accurately reflect the potential for bioaccumulation. Empirical BAF data are limited, and therefore most BAFs were modelled using the BCFBAF (2010) program in EPI Suite (2000-2010).

Empirical and modelled bioaccumulation data for major coal tar components, as well as the weighing of information, can be found in Environment Canada (2015a). In addition to fish BCF and BAF data, bioaccumulation data for aquatic invertebrate species were also considered. Biota-sediment/soil accumulation factors (BSAFs), trophic magnification factors (TMFs) and biomagnification factors (BMFs) were also considered in characterizing bioaccumulation potential.

Overall, there is consistent empirical and predicted evidence to indicate that some components of coal tars and their distillates have the potential to be highly bioaccumulative. Empirical fish BCFs greater than 5000 L/kg wet weight (ww) have been determined for the PAHs phenanthrene, anthracene, fluoranthene and pyrene, while invertebrate BCFs exceeding 5000 L/kg ww have been measured for phenanthrene, anthracene, fluoranthene, pyrene, benzo[*a*]anthracene, benzo[*k*]fluoranthene, benzo[*a*]pyrene (B[*a*]P), benzo[*g,h,i*]perylene and dibenz[*a,h*]anthracene (Environment Canada 2015a). Non-PAH coal tar components, including monocyclic aromatic hydrocarbons, phenolics and heterocyclics, are not highly bioaccumulative. Most PAH and non-PAH components of coal tars and their distillates are not expected to biomagnify in aquatic or terrestrial food webs, largely because a combination of metabolism, low dietary assimilation efficiency and growth dilution allows the elimination rate to exceed the uptake rate from their diet (Environment Canada 2015a). However, one study (Harris et al. 2011) suggests that some alkyl PAHs may

biomagnify in the sea otter food web. Median fish BSAFs for 16 PAHs were all less than 1, which is consistent with evidence for efficient metabolism in these organisms. It is possible that BSAFs will be greater than 1 for invertebrates given that they do not have the same metabolic competency as fish.

9. Potential to cause ecological harm

9.1 Ecological effects assessment

Ecotoxicity data are available for coal tar, coal tar pitch, and coal-tar-based pavement sealants. No information was found on the toxicity of coal tar oils and upper distillates. However, several related substances were identified by ECHA (c2007-2017) as suitable surrogates for read-across, and ecotoxicity values for these substances are considered. The ecological effects information is considered representative of the characteristic of all coal tars and their distillates, including, but not limited to the six substances identified in Table 2-1.

9.1.1 Coal tar studies

The acute toxicity of coal tar and coal tar pitch was determined for killifish, *Oryzias latipes*, and the water flea, *Daphnia magna* (Tadokoro et al. 1991). Ninety-six hour median lethal (LC₅₀) values with coal tar for killifish were 7.33 to 12.1 mg/L (nominal), and 48-hour LC₅₀ values for the water flea were 4.44 to 11.2 mg/L (nominal). Although 90% to 100% mortality was observed at the highest test concentrations of coal tar pitch, acute LC₅₀ values were not determined due to extremely low extraction efficiencies (i.e., the range of measured to nominal concentration ratios in the water fraction was 0.13% to 0.3%). A comparison of whole-solution (dissolved and insoluble portions of the test substances in water) versus dissolved-portion-only testing determined that the highest toxicities were consistently observed in whole-solution testing, suggesting that precipitates existing in the whole solution may dissolve gradually into the test water or may be directly absorbed by the test organisms (Tadokoro et al. 1991).

In whole-sediment testing, weathered coal tar collected from a riverbed located downstream from a former gas works site was acutely toxic to embryos and larvae of shortnose sturgeon, *Acipenser brevirostrum*, producing 95% embryo-larval mortality over a 21-day exposure period (Kocan et al. 1996). Similar impacts were not seen following exposure to elutriates prepared from the sediment, leading the researchers to conclude that the observed adverse effects were the result of direct contact with the contaminated sediment rather than via exposure to soluble hydrocarbons. The authors noted that aspects of the sturgeon life cycle, such as juvenile burrowing behaviour and benthic feeding habits in juveniles and adults, increased the likelihood of physical contact with sediment, thereby increasing the probability of exposure to coal tar present in the sediment. The study also found that coal tar that had been submerged in sediment for between 40 and 140 years retained its toxicity to sturgeon early life stages,

suggesting that continued exposure to the sediment could lead to a decline in the sturgeon population as a result of decreased reproductive success.

River sediments collected in the vicinity of a former gas company site and contaminated with coal tar residue resulted in significantly reduced cell counts in the green alga, *Pseudokirchneriella subcapitata*, and increased mortality in *Daphnia magna* and the midge, *Chironomus tentans* (Oberholster et al. 2005). Standard 48-hour and 10-day whole-sediment tests were conducted on *D. magna* and *C. tentans*, respectively; the algal test was a standard 96-hour screening test using 100% (undiluted) filtered pore water collected from the test-site sediments. Algal cell counts were approximately 50% lower in sediments collected near the gas site than those in sediments collected at nearby reference sites, while *Daphnia* and midge survival was reduced by 40% to 100% at the test location (estimated from graphical data). Evidence of toxicity in the water-soluble component of the coal tar and the coal tar residue itself indicated that both can produce adverse effects in water column and sediment species.

9.1.2 Coal-tar-based sealant studies

Bryer et al. (2006) monitored survival and development in African clawed frog, *Xenopus laevis*, embryos exposed to one of four nominal aqueous concentrations of total PAHs (TPAH; sum of 16 PAHs) from coal-tar-based pavement sealant (CTPS) over a 52-day period. The treatments were prepared by adding dried CTPS flakes to conditioned tap water. The nominal TPAH concentrations were estimated based on the theoretical maximum amount of TPAH contained in the CTPS. Complete mortality occurred at the highest treatment level of 300 ppm TPAH (nominal) by day 6 of the study, and reduced rates of development were observed in the 3 and 30 ppm TPAH (nominal) exposure groups. By the end of the study, the low-dose group had reached a stage of development equivalent to that of the controls (i.e., they had metamorphosed), while none of the remaining medium-dose animals had reached metamorphosis. The study concluded that frogs exposed to coal-tar-based sealant at concentrations of 3 ppm TPAH and higher took longer to hatch and were smaller and developmentally delayed.

Adult eastern newts, *Notophthalmus viridescens*, exposed to concentrations of 15 to 1500 mg dried CTPS/kg dry weight (dw) sediment (nominal) for 28 days exhibited significantly decreased ability to right themselves at all test concentrations and diminished liver enzyme activity at concentrations of 125 mg/kg dw (nominal) and higher (Bommarito et al. 2010a). Abilities associated with the righting response include cognition (i.e., animal recognizing that it is upside down), muscular strength and coordination. Therefore, a reduced righting ability could result in reduced survival if food capture or the ability to escape from predators is impacted negatively (Bommarito et al. 2010a). The observed reduction in enzyme activity was considered to indicate possible hepatic damage in the exposed animals.

A similar study was conducted using larvae of the spotted salamander, *Ambystoma maculatum*, and dried sealant concentrations of 60, 280 and 1500 mg/kg dw in sediments (Bommarito et al. 2010b). No significant mortality occurred during the 28-day

experiment; however, dose-dependent decreases in growth rate and swimming ability (speed, distance and duration) were observed at all dose levels. These effects could negatively impact the ability of the salamanders to capture prey or escape predators (Bommarito et al. 2010b).

Mahler et al. (2015) investigated the acute toxicity of simulated runoff collected from pavement test plots sealed with a coal-tar-based sealant or an asphalt-based sealant thought to contain about 7% coal tar sealant (based on elevated PAH concentrations and profile) to neonate water flea, *Ceriodaphnia dubia*, and larval fathead minnow, *Pimephales promelas*. The test plots were areas of active use for parking and driving over the course of the study, allowing for incorporation of the potential contribution of PAHs from dried sealant abraded by vehicle tires (Mahler et al. 2014), as well as from other sources. Test organisms were exposed for 48 hours to runoff collected from 5 hours to 111 days following application of the coal-tar-based sealant and from 4 hours to 36 days after application of the asphalt-based sealant. Exposure concentrations of 100% runoff (undiluted runoff), 10% runoff (1:10 dilution with control water) and 0% runoff (100% control water) were used. At the end of the 48-h exposure period, half of the test organisms were transferred to control water and exposed to an environmentally relevant level of ultraviolet (UV) light for 4 hours to assess potential photoactivated toxicity, followed by an additional 20-h recovery period under ambient light conditions. Survival was 0% in the undiluted coal tar sealant runoff samples collected 3 days (*C. dubia*) or 36 days (*P. promelas*) following sealant application. Survival in the 10% coal tar sealant runoff samples over this time period ranged from 93% to 100% for *C. dubia* and from 98% to 100% for *P. promelas*. UV exposure reduced the survival of organisms exposed to coal tar sealant runoff (both 100% and 10%) relative to that in the controls. Survival in organisms irradiated with UV light following 48 hours in control water ranged from 95% to 100% for both species over the course of the study, while that in UV-exposed organisms following 48 hours in 10% coal tar sealant runoff was 0% to 30% for *C. dubia* and 35% to 100% for *P. promelas*. Survival was 0% for both species following UV light exposure in the undiluted runoff samples. Much lower toxicity was evident with organisms exposed to the asphalt-based sealant containing an estimated 7% coal tar sealant. Survival for both species ranged from 60% to 100% in the undiluted runoff samples and from 88% to 100% in the 10% runoff samples prior to irradiation with UV light. After irradiation, survival in the organisms exposed to undiluted runoff was 0% to 10% for *C. dubia* at all sampling times and 0% to 90% in *P. promelas*. Survival for UV-exposed organisms following 48 hours in 10% asphalt-based sealant runoff was between 65% to 100% for both species. The results showed that coal tar sealant runoff remains acutely toxic for weeks to months following application. Coal tar sealant runoff was substantially more toxic than the runoff from the asphalt-based sealant used in the study and survival generally decreased with the addition of UV irradiation in both sealants.

Kienzler et al. (2015) also used runoff samples from coal-tar-based sealant and asphalt-based sealant to examine genotoxicity and DNA repair capacity impairment endpoints, using the rainbow trout RTL-W1 fish liver cell line. Samples were collected from 5 hours to 36 days following sealant application and diluted to either 10% or 1%. For controls,

simulated stormwater runoff was prepared as a mixture of groundwater and deionized water. Cells were exposed to the media for 24 hours in the dark or with co-exposure to UV-A light for the first 2 hours of the 24 hours. Relative to control conditions, genotoxic effects, as measured using the Formamido pyrimidine glycosylase (Fpg)-modified comet assays,³ were significant for both 10% and 1% dilutions of coal-tar-based sealant runoff collected at 5 hours, 26 hours and 7 days with co-exposure to UV-A. No significant effect was measured in the absence of UV-A exposure. Significant genotoxicity following co-exposure with UV-A was also measured for 10% dilutions of the asphalt-based sealant (with 7% coal tar sealant) runoff at 26 hours, 7 days and 36 days and for the 1% dilution at 7 days following sealant application. DNA repair capacity, as assessed using a base excision repair comet (BERc) assay, was significantly impaired for the 10% coal-tar-based sealant runoff samples both with and without UV-A exposure, and for the 10% asphalt-based sealant runoff only in the absence of UV-A. The results indicated that co-exposure to runoff from coal-tar-sealed pavement and UV-A has the potential to cause DNA damage and impair DNA repair capacity. Deleterious effects were measured for samples of runoff collected as long as 36 days following coal-tar-based sealant application. Exposure to runoff from asphalt-based sealant containing an estimated 7% coal tar sealant also demonstrated genotoxic potential and BER impairment, but to a lesser extent (Kienzler et al. 2015).

McIntyre et al. (2016) examined the lethal and sublethal toxicity of runoff from a coal-tar-based sealcoated test plot using juvenile coho salmon, *Oncorhynchus kisutch*, and embryo-larval zebrafish, *Danio rerio*. The sealcoated plot was subject to natural sunlight and rainfall, with four simulated storm events providing runoff at intervals from 2 hours to 7 months post-application. The unfiltered runoff from the first storm event (collected 2 hours after sealant application) was highly toxic to juvenile coho salmon, with no salmon surviving after 5 hours of exposure. Mortality decreased substantially for subsequent simulated storms events, where mortality was 20% for the second event (7 days post-application) and 10% for the third event (13 days post-application), as determined by standard 96-h toxicity tests. An elevated mortality of 55% occurred in salmon exposed to runoff collected from the fourth simulated storm event (207 days post-application). This was attributed to the possible photomodification of PAHs present in the coal tar sealant runoff to more toxic chemical species due to the higher UV index recorded on that day as compared with earlier events (McIntyre et al. 2016).

Runoff from the first simulated rainfall event was also acutely lethal to zebrafish embryos, resulting in 100% mortality within 48 hours of exposure (McIntyre et al. 2016). No significant difference in mortality relative to controls (embryo system water) was observed for zebrafish exposed to runoff from rainfall events 2, 3, and 4. Zebrafish exposed for 48 hours during embryonic development to sublethal concentrations of unfiltered runoff (diluted runoff from the first event and undiluted runoff from the second to fourth event) displayed a range of cardiovascular abnormalities, including pericardial edema and cranial hemorrhaging, and exhibited significant induction of molecular

³ The standard and Fpg-modified comet assays measure primary DNA damage (i.e., single and double strand breaks, base oxidation and alkylation) on the basis of differential migration of the damaged DNA in an electrophoresis gel (Kienzler et al. 2015).

markers for PAH exposure and cardiac stress. The results showed that fresh coal-tar-based sealants are a source of hazardous chemical contaminants in urban stormwater runoff. As well, mortality and sublethal toxicity were observed in coal-tar-based sealant runoff up to 7 months after application.

Adverse impacts to freshwater benthic macroinvertebrate communities have also been reported following exposure in a field setting to coal-tar-based sealants. Scoggins et al. (2007) reported a significant decrease in the health of benthic macroinvertebrate communities situated downstream of parking lots coated with CTPS, as measured using biological indices, such as species richness (number of taxa) and density, changes in the abundance of individual species (an effect attributable to differences in species tolerance), and altered species dominance. The downstream communities exhibited decreases of up to 50% in species richness and density as compared with similar benthic communities at sites upstream of the parking lots. Changes in species dominance and abundance were also observed.

Bryer et al. (2010) investigated the effect of CTPS on a freshwater benthic macroinvertebrate community by exposing sediment organisms to four treatment groups of sealant added at TPAH concentrations (total of 16 EPA priority pollutant PAHs; U.S. EPA 2013) of 0.1, 7.5, 18.4 and 300 mg/kg dw. At the end of the 24-day exposure period, the total abundance and number of taxa were significantly lower in the high-treatment group compared to the control. Changes in the abundance of individual species, considered to be an indicator of individual species' tolerance to the test substances, were also evident. The results confirm that CTPS contains bioavailable PAHs that can alter benthic communities and adversely affect aquatic organisms.

9.1.3 Read-across data for coal tar oils and upper distillates

The ECHA (c2007-2017) database reports ecotoxicity data for distillates (coal tar) light oils (CAS RN 84650-03-3) as acceptable for use in read-across for coal tar oils, and anthracene oil (CAS RN 90640-80-5) and wash oil (no CAS RN provided) as acceptable for use in read-across for the upper distillates. Physical and chemical property data for distillates (coal tar) light oils and wash oil were presented as read-across data for tar oils and upper distillates, respectively (Tables 3-2 and 3-3).

Acute toxicity data for testing performed using water-accommodated fractions (WAFs) of distillates (coal tar) light oils indicate there is potential for toxicity in aquatic species. WAFs are laboratory-prepared aqueous media derived from low-energy mixing of a poorly soluble test material, in this case distillates (coal tar) light oils. They are essentially free of particles of bulk material, containing only the fraction that is dissolved or present as a stable dispersion or emulsion (Singer et al. 2001). Median effect levels based on loading rates (EL_{50s})⁴ of the distillates (coal tar) light oils WAF were 13 mg/L in 48-h testing with the water flea, *Daphnia* sp. and ranged from < 5 to 39 mg/L in 72-h testing with the green alga, *Desmodesmus subspicatus* (ECHA c2007-2017b).

⁴ The EL_{50} is defined as the loading rate of test substance resulting in a specified effect (e.g., immobilization, growth) in 50% of the test species exposed to the WAF.

Similar studies conducted using WAFs of anthracene oil and wash oil as surrogates for coal tar upper distillates yielded 48-h *Daphnia* sp. EL₅₀ values of 2.7 mg/L (wash oil) to 137 mg/L (anthracene oil) and 72-h EL₅₀ values of 20 mg/L (wash oil) to 48 mg/L (anthracene oil) for testing with *D. subspicatus* (ECHA c2007-2017e). As well, acute median lethal loading rates (LL_{50s}) of 79 mg/L (wash oil) to > 100 mg/L (anthracene oil) were obtained in 96-h testing with the zebrafish, *D. rerio* (ECHA c2007-2017e). Considered together, the results demonstrate potential for toxicity in aquatic species.

9.1.4 Toxicity of PAH and non-PAH coal tar component classes

As UVCBs, coal tars and their distillates contain a large and variable number of components that may contribute to toxicity. Most adverse ecological effects associated with exposure to coal tar substances are attributed to the PAHs present as major components of the substances. This screening assessment primarily considers the potential for adverse effects in relation to PAHs, and in particular the 16 U.S. EPA Priority PAHs. However, these 16 PAHs form only a subset of the total number of components that may be present in a coal tar substance. Some non-PAH components may also contribute to toxicity, particularly when present in higher proportions, such as in the coal tar oils and upper distillates. The following two sections provide a general overview of the toxicity of major PAH and non-PAH components of coal tars and their distillates. Summary tables of selected ecotoxicity endpoint values for the PAH and non-PAH representative structures are available in Environment Canada (2015b).

9.1.4.1 Toxicity of PAHs

PAH toxicity has been well studied and is still an area of active research, with much information available in the published literature. Extensive summaries of PAH toxicity can be found in assessment reviews such as Environment Canada, Health Canada (1994), WHO (1998), Douben (2003) and European Commission (2008).

PAHs exert toxicity through various means, including narcosis, mutagenesis and/or carcinogenesis, reproductive toxicity, impairment of growth and development, and disruptions to hormonal and immunological function (Uthe 1991; Den Besten et al. 2003; Payne et al. 2003). Some PAHs are phototoxic, resulting in adverse impacts through photosensitization (production of reactive singlet oxygen that damages cells) or photomodification (formation of new compounds, usually via oxidation processes, that can exert toxicity) (Ankley et al. 2003; European Commission 2008). Metabolism of some PAHs, such as benzo[a]pyrene, can result in the formation of metabolites with higher toxicity than the parent compound. There is also evidence that PAHs or their metabolites may interact synergistically with other environmental contaminants, notably some metals, resulting in enhanced toxicity (Babu et al. 2001; Xie et al. 2006). Low molecular weight PAHs with two- to three-ring structures are more likely to exert acute toxicity and be non-carcinogenic, while higher molecular weight PAHs having four or more aromatic rings are generally not as acutely toxic but have higher carcinogenic potential (Neff 1979; Moore and Ramamoorthy 1984; Goyette and Boyd 1989). For

toxicity to aquatic organisms, PAHs are expected to have a narcotic mode of action and thus are expected to have additive toxicity (Di Toro et al. 2000).

Most PAHs present as major components in coal tars and their distillates are highly hazardous to aquatic organisms (e.g., acute LC/EC₅₀ values \leq 1.0 mg/L and/or chronic no observed effect concentrations (NOECs) \leq 0.1 mg/L (Environment Canada, Health Canada 1994; WHO 1998; Douben 2003; European Commission 2008). In addition, some PAH components may elicit adverse effects in terrestrial species, including acute toxicity in soil nitrifying bacteria and significantly reduced growth or reproduction in invertebrates and terrestrial plants (Douben 2003; European Commission 2008).

9.1.4.2 Toxicity of non-PAH components

Many non-PAH components of coal tars and their distillates are more water soluble than the PAHs and, together with moderate octanol-water partition coefficients, this suggests that these components may be more bioavailable to organisms. They may also be more mobile in the environment, with the potential to migrate further from the contaminant source (Gray 1984).

Short-term acute effects are more likely to be the primary concern for volatile and water-soluble non-PAH constituents because, unless deposition of the coal tar substance is fresh or continuous, exposure concentrations of these substances can be expected to decrease rapidly through the processes of dissipation and/or degradation. An exception is groundwater contamination, where biodegradation rates and opportunities for loss through volatilization are much reduced (Irwin et al. 1997). In addition, the acute toxicity of some non-PAH constituents may adversely affect microbial communities, resulting in reduced biodegradation rates for all coal tar constituents (Gray 1984).

Table 9-1 summarizes toxicity endpoint ranges for each of the major chemical classes of non-PAH components. The ranges are based on selected endpoints for each class, as described in Environment Canada (2015b), and do not represent all ecotoxicity data available for substances within the group. Consequently, the ranges provided in Table 9-1 demonstrate general trends for the chemical classes in order to allow a qualitative comparison of ecotoxicity among the classes.

Table 9-1. Summary of ecotoxicity data for major non-PAH components of coal tars and their distillates

Chemical class	Testing type	Organism type	Range of endpoint values^a
MAHs ^b	Acute	Pelagic ^c	0.72–86 mg/L
MAHs	Chronic	Pelagic	3.9–41 mg/L
MAHs	Acute	Benthic ^d	9.5–100 mg/L ^e
MAHs	Chronic	Soil ^f	3–835 mg/kg dw soil
Phenolics ^g	Acute	Pelagic	0.20–> 99 mg/L
Phenolics	Chronic	Pelagic	0.07–175 mg/L

Chemical class	Testing type	Organism type	Range of endpoint values ^a
Phenolics	Acute	Benthic	0.85–187 mg/L ^e
Phenolics	Chronic	Soil	67–472 mg/kg dw soil
Heterocyclics ^h	Acute	Pelagic	0.11–575 mg/L
Heterocyclics	Chronic	Pelagic	0.18–11 mg/L
Heterocyclics	Acute	Benthic	0.07–182 mg/L ^e
Heterocyclics	Chronic	Soil	23–> 4000 mg/kg dw soil

^a Toxicity endpoints used to determine ranges for water column and sediment testing were the median lethal concentration, LC₅₀, and the median effect concentration, EC₅₀. Terrestrial toxicity endpoints used were the EC₅₀ and EC₁₀ (concentration causing an effect in 10% of test organisms), LC₅₀ and LC₂₅ (concentration lethal to 25% of test organisms), IC₂₅ (concentration causing a 25% reduction in a quantitative biological measurement), and LOEC (lowest observed effect concentration). Endpoints were selected based on the quantity and reliability of available data.

^b Monocyclic aromatic hydrocarbons include benzene, toluene, styrene, ethylbenzene and xylenes.

^c Pelagic species were fish, water fleas, mysid shrimp and algae.

^d Benthic species were marine and freshwater amphipods, larval insects and blackworms.

^e Water-only exposure.

^f Soil species were earthworm, springtails, plants and soil bacteria.

^g Includes phenol, cresols, xylenols and naphthols.

^h Includes pyridine, quinoline, carbazole, acridine, dibenzothiophene and dibenzofuran.

Note: No chronic sediment or acute terrestrial toxicity testing data were found for the selected components.

Monocyclic aromatic hydrocarbons (MAHs) generally exhibit low to moderate toxicity in aquatic and terrestrial organisms, with most acute and chronic aquatic median effective and lethal concentration (EC₅₀/LC₅₀) values falling in the range of 1 to 100 mg/L and most chronic terrestrial values generally falling in the range of 50 to 100 mg/kg dw of soil (Table 9-1; Environment Canada 2015b).

Phenolic compounds exhibit slightly higher toxicity than MAHs, although most acute and chronic aquatic endpoint values remain in the range of 1 to 10 mg/L (Environment Canada 2015b). However, some species have relatively high sensitivity to phenolic compounds. For example, a 96-hour LC₅₀ of 0.20 mg/L was reported for the mysid *Americamysis bahia* exposed to 1-naphthol (Union Carbide 1986), and 27-day LC₅₀ values of 0.15 and 0.07 mg/L were determined for rainbow trout, *Oncorhynchus mykiss*, exposed to phenol and 2-naphthol, respectively (Black et al. 1983). Terrestrial toxicity endpoint values for the phenolics are comparable to those of the MAHs, with the lowest values falling in the range of 50 to 100 mg/kg dw of soil (Environment Canada 2015b).

Heterocyclic compounds generally have low to moderate acute and chronic toxicity (e.g., aquatic endpoint values in the range of 1 to 100 mg/L; Environment Canada 2015b), although there is also evidence for marked sensitivity of some species to members of this chemical class. Acute (96-hour) LC₅₀ values for fathead minnow, *Pimephales promelas*, of 0.44 mg/L (Millemann et al. 1984) and 0.93 mg/L (Brooke 1991) were determined for quinoline and carbazole, respectively. Black et al. (1983) reported 7-day bass, *Micropterus salmoides*, and 27-day rainbow trout, *Oncorhynchus mykiss*, LC₅₀ values of 1.02 and 0.32 mg/L, respectively, following exposure of the fish to acridine, while growth EC₅₀ values for acridine and green algae are 0.27 to 0.90 mg/L for the microalga *Pseudokirchneriella subcapitata* (Blaylock et al. 1985; Dijkman et al. 1997) and 0.32 to 0.41 mg/L for the alga *Scenedesmus acuminatus* (Van Vlaardingen et

al. 1996). A pattern of increased toxic potential with increasing number of aromatic rings is evident, so that quinoline, which has two rings, is significantly less toxic than acridine, which has three rings. This increase in toxicity is likely attributable to increased lipophilicity of the molecule, which facilitates bioavailability and uptake potential (Black et al. 1983; Millemann et al. 1984; Bleeker et al. 1998). In addition, some heterocyclics, including acridine and quinoline, can elicit adverse effects by mechanisms such as reproductive toxicity, photo-induced toxicity and/or metabolic transformation to more biologically active substances, leading to significantly enhanced toxicity (Bleeker et al. 2002).

9.1.5 Derivation of critical toxicity values

Complex mixtures, such as coal tars and their distillates, cannot be analytically measured in environmental media. Thus, components of the mixtures, such as PAHs, are frequently measured as being representative of the entire mixture, as they are important components of coal tars and their distillates. For these reasons, the critical toxicity values (CTVs) used in evaluating the ecological risk of coal tars and their distillates are based on PAHs.

In 1994, an assessment conducted by Environment Canada and Health Canada determined that PAHs met the criteria for “toxic” as defined in the *Canadian Environmental Protection Act* (CEPA) (Environment Canada, Health Canada 1994). Based on this decision, PAHs were added to Schedule 1 of CEPA (i.e., the List of Toxic Substances). In response to the need for risk management, Canadian Environmental Quality Guidelines (CEQGs) for PAHs were developed by the Canadian Council of Ministers of the Environment (CCME), including water and sediment quality guidelines for the protection of aquatic life and soil quality guidelines for the protection of the environment and human health (CCME 1999a, 1999b, 2010) (Table 9-2).

CEQGs from the CCME were used to determine CTVs for aquatic, sediment and soil organisms (Table 9-2). CCME environmental quality guideline values have been derived for 9 PAHs for water (CCME 1999a), for 12 PAHs for sediment (CCME 1999b), and for 15 PAHs for soil (CCME 2010). As the CEQG values have been derived to be protective of water, sediment and soil on a long-term basis, they are also used as the predicted no-effect concentrations (PNECs) for the risk assessment, without use of application factors. Where available, soil quality guidelines for the protection of freshwater life were used as PNECs for the soil compartment as contamination present in soil can migrate to groundwater. If there are surface water bodies nearby, then life in these surface water bodies may be affected by the contamination, particularly if there is a permeable aquifer connecting the contamination with the surface water body. The soil exposure scenario for this screening assessment (see section 9.2.2.1) examined PAH emissions from a facility situated close to a water body, and for this reason, soil guidelines derived from impact on aquatic life as a result of runoff to freshwater were preferentially used as PNECs.

Table 9-2. Canadian Environmental Quality Guidelines for PAHs in water, sediment and soil (CCME 1999a, 1999b, 2010)

PAH	Freshwater quality guideline ($\mu\text{g/L}$) ^a	Sediment quality guideline (mg/kg dw) ^{a,b}	Soil quality guideline ^c (mg/kg dw)
Acenaphthene	5.8	0.00671	0.28 ^d
Acenaphthylene	N/A	0.00587	320 ^d
Anthracene	0.012	0.0469	2.5
Benz[a]anthracene	0.018	0.0748 (M); 0.0317 (F)	0.1 ^e
Benzo[a]pyrene	0.015	0.0888 (M); 0.0319 (F)	0.7 ^e
Benzo[b]fluoranthene	N/A	N/A	0.1 ^e
Benzo[k]fluoranthene	N/A	N/A	0.1 ^e
Chrysene	N/A	0.108 (M); 0.0571 (F)	6.2 ^f
Dibenz[a,h]anthracene	N/A	0.00622	0.1 ^e
Fluoranthene	0.04	0.113 (M); 0.111 (F)	50
Fluorene	3	0.0212	0.25 ^d
Indeno[1,2,3-cd]pyrene	N/A	N/A	0.1 ^e
Naphthalene	1.1, 1.4 ^g	0.0346	0.013 ^d
Phenanthrene	0.4	0.0867 (M); 0.0419 (F)	0.046 ^d
Pyrene	0.025	0.153 (M); 0.053 (F)	0.1 ^e

N/A, no guideline value available

^a Interim guidelines

^b Values are for both freshwater (F) and marine/estuarine (M) sediments, unless otherwise stated.

^c The most protective guideline was selected from those for the various land-use types (agricultural, residential, commercial/industrial).

^d Guideline value for protection of freshwater life.

^e Interim guideline value (CCME 1991) or provisional guideline value (CCME 1997).

^f Guideline value for soil and food ingestion. A full guideline value is not available.

^g Marine guideline (mg/L).

9.2 Ecological exposure assessment

9.2.1 Scope of the ecological assessment

As coal tars and their distillates are complex mixtures that cannot be easily measured, the releases of PAHs, major components of coal tars and their distillates, from coal-tar-related sources were used to estimate the potential for ecological exposure from coal tars and their distillates.

Based on information regarding releases of PAH components of coal tars and their distillates into the environment, three exposure scenarios were identified as presenting the highest potential releases to the environment, and therefore quantitative exposure scenarios were developed for them. The first scenario considers deposition of PAHs onto soil from air emissions from the only coal tar refinery in Canada; the second scenario considers releases from the coal tar refinery to the sewer system; the third scenario examines releases from coal-tar-based pavement sealants.

Concentrations of PAHs in air resulting from industrial activities related to coal tar production, refining or transport are not examined in the context of ecological effects in this report, as these emissions are considered to be of potential concern primarily from a human health perspective. A report by the European Commission's PAH Working Group, which examined the need for air quality standards for PAHs, states as follows: "There does not appear to be a body of data suggesting that there is a significant impact on non-human fauna, [or] flora...requiring the establishment of objectives for ambient air quality other than those designed for the protection of human health" (European Commission 2001).

The ecological exposure assessment given below for the coal tars and their distillates includes, but is not limited to, the six substances with CAS RNs given in Table 2-1.

9.2.2 Releases from industry

9.2.2.1 Releases to air depositing on soil

The deposition of PAHs from coal tars and their distillates to soil in the Hamilton, Ontario, area as a result of air emissions from the only coal tar refinery in Canada was examined as a potential route of exposure to the environment and is discussed below. The coal tar refinery is situated close to Hamilton Harbour, on the shore of Lake Ontario. Most of the substances of primary concern in coal tars—PAHs—have low to negligible volatility, with the exception of naphthalene and phenanthrene, which are moderately volatile. In the atmosphere, PAHs can be removed by wet or dry deposition to water or soil (Environment Canada, Health Canada 1994). It is noted that PAHs, as well as other components of coal tars and their distillates from coal tar production and refining industries, may also be deposited into Lake Ontario, thus contributing to the pollutant load of this lake, though this is not quantitatively assessed here.

Coal tars and their distillates all contribute to the releases of PAHs from the coal tar refinery. The deposition of selected PAHs to soil resulting from air emissions in the Hamilton area from the coal tar refinery was modelled. The annual air emissions of naphthalene, phenanthrene and acenaphthene, which are the PAHs released in the greatest quantities, as reported to the National Pollutant Release Inventory in 2012 (NPRI 2014), were converted to emissions rates based on this facility operating 24 hours/day, 365 days/year. The maximum 1-year deposition rates to soil resulting from the atmospheric emissions of the PAHs were then modelled using AERMOD (2009) for an area approximately 1 km downwind of the facility based on 4 years of

atmospheric data. The 1-year deposition rates were converted to soil concentrations after 10 years of deposition at this rate using a spreadsheet program based on the work of Baes and Sharp (1983), U.S. EPA (1999) and ECHA (2012):

$$C_s = D [1.0 - e^{-kt}] / Z_s \cdot B_d \cdot k$$

where,

C_s = concentration of substance in soil after total time period of deposition ($\mu\text{g/g}$)

100 = units conversion factor ($[10^6 \mu\text{g/g} / [10^4 \text{cm}^2/\text{m}^2]]$)

D = annual deposition rate to soil (g/m^2)

k = soil loss rate constant (yr^{-1})

t = total time period of deposition (years)

Z_s = soil mixing depth (cm)

B_d = soil bulk density (g/cm^3)

This calculation considered losses from both biodegradation and physical processes, such as soil leaching, runoff and volatilization. The soil loss rate constants were based on the average half-lives in soil for the PAHs estimated by Mackay et al. (1992).

The results of this analysis are presented in section 9.3.1.1.

9.2.2.2 Releases to water

All process water and effluent generated from coal tar storage, precipitation and further processing at the coal tar refinery is collected and treated to comply with the sewer use by-law discharge limits of the City of Hamilton, Ontario (City of Hamilton 2013).

The relevant City of Hamilton limits for discharge to the sewer system for various components of coal-tar-related substances are listed in Table 9-3. It is assumed that these limits are the maximum concentration of each component found in the effluent from the coal tar refinery and are used to derive concentrations for each component in wastewater entering Hamilton's Woodward Avenue wastewater treatment plant (WWTP) (Table 9-3), based on the equation below.

$$C_1 \times V_1 = C_2 \times V_2$$

or

$$C_2 = C_1 \times V_1 / V_2$$

where,

C_1 = the discharge limit concentration for the component

C_2 = the maximum estimated aquatic concentration of the component after dilution in the sewer system (i.e., the concentration in the influent to the WWTP)

V₁ = the average daily discharge to the sewer system by the coal tar refinery (311 m³/day) (e-mail from City of Hamilton, Environmental Monitoring & Enforcement, to Ecological Assessment Division, Environment Canada, dated 2013, unreferenced)
V₂ = the average daily flow of the Woodward Avenue WWTP (278 146 m³/day) (Environment Canada 2013b)

Table 9-3. Maximum estimated aquatic concentrations of coal tar components discharged by the coal tar refinery to the sewer system as compared to the Canadian Water Quality Guidelines

Parameter	City of Hamilton, Ontario, sewer discharge limits (µg/L)	Maximum estimated aquatic concentration from the coal tar refinery at influent to WWTP (µg/L)	CCME guidelines ^a (µg/L)
Phenolic compounds	1000	1.1	4 ^b
Benzene	10	0.011	370
Ethylbenzene	160	0.18	90
Toluene	16	0.018	2
Total xylenes	1400	1.5	N/A
Total PAHs	5	0.0056	N/A ^c

N/A, no guideline value available

^a Canadian freshwater quality guidelines for aquatic life, CCME (2014)

^b Guideline is for mono- and di-hydric phenols.

^c No CCME guideline value available for total PAHs. Guidelines for individual PAHs range from 0.12 to 5.8 µg/L.

9.2.3 Releases from the use of coal-tar-based pavement sealant (CTPS)

CTPS contains approximately 15% to 30% coal tar pitch emulsified in water (EHS 2010; see Uses section). Since coal tar substances cannot be measured directly in the environment because they are UVCBs, PAHs, which are the primary constituents of coal tar substances, are used to evaluate the environmental exposure to coal tar substances.

9.2.3.1 Spills data

Of the 29 reported spills in the categories of “tar,” “tar and water mixture,” “tar-based caulking,” “driveway sealant,” “asphalt sealer,” “sealant (N.O.S.)” and “coal tar distillates (N.O.S.)” to the non-industrial sites, to land or surface water in Ontario during the 5 years between 2008 and 2012 (Ontario 2013), 25 are considered as CTPS and total 2534 L. Spills from roofing companies/jobs were not included in the total here, as only spills that could be CTPS were considered here. This is equivalent to an average of 5.2 spills/year and approximately 500 L/year. These products may, but do not necessarily,

contain coal tar and/or its distillates. Diamond Environmental Group (2011) found that 23% of the driveways sampled in the Toronto area were coated with CTPS. Applying this proportion to the spills data, it is estimated that CTPS products would account for about 1.2 spills/year and a volume of about 120 L/year. These are considered to be high-end estimates, as the sampling from the Diamond Environmental Group (2011) study was biased towards sampling driveways and parking lots that appeared to have sealcoat (personal communication, e-mail from M. Diamond to Ecological Assessment Division, Environment Canada, dated 2014, unreferenced). As well, only 13 of the spills from the Ontario spills database were listed as asphalt sealer or driveway sealer or sealant (N.O.S.). With only one exception, the other 13 spills were “tar” and “tar and water mixture,” which probably represents an even lower fraction of coal-tar-based products, as many were probably from road construction/maintenance, which does not use CTPS (see Uses section).

9.2.3.2 Releases of CTPS to water

Based on the use of CTPS in Canada (see Uses section), stormwater runoff from paved areas coated with CTPS is expected to enter the aquatic environment. Four studies relevant to Canadian conditions were used to estimate the concentration of PAHs in runoff from areas sealed with CTPS (Watts et al. 2010; Rowe and O’Connor 2011; Mahler et al. 2014; and McIntyre et al. 2016). The studies differed in their duration, number of coats of CTPS applied, whether traffic was allowed, and the number of water samples taken to determine concentrations over time (ECCC 2018). There was also variability in precipitation events, where one study measured actual events (Watts et al. 2010) and the others involved simulated events (Rowe and O’Connor 2011; Mahler et al. 2014; McIntyre et al. 2016). Results from the four studies were used to provide a weight of evidence due to uncertainties within each study. Each study showed a higher PAH concentration in runoff associated with the first precipitation event after the lot was sealed and lower PAH concentrations in runoff from subsequent precipitation events (ECCC 2018). For this reason, concentrations were separated into concentrations in runoff taken during the initial precipitation event (C_{IP}) and concentrations from the remaining events (C_R). From these four studies, C_{IP} and C_R were obtained for use in calculating predicted environmental concentrations (PECs) in the exposure assessment. When multiple concentrations were available for a given study, an average value was calculated. This approach ensured that each study contributed equally to the overall average used to determine the PECs.

PAH concentrations were calculated over a 2-year period, which represents the lower end of the 2- to 5-year resurfacing time generally recommended by manufacturers and companies that apply sealcoats (Goosey et al. 2011). All four studies used sealant products that contained HTCTP at the high end of the 10% to 30% content range reported in the literature, thereby maximizing the PAH content of the sealant. Technical information available on CTPS products used in the studies indicate that the commercial formulation used by both Watts et al. (2010) and Mahler et al. (2014) contained 25% to 35% coal tar product (Neyra 2015), while that used by McIntyre et al. (2016) contained 30% to 33% HTCTP (Surface Coatings Company 1999). Although Rowe and O’Connor

(2011) do not provide the name of the commercial product used in their study, their report includes a National Institute of Standards and Technology (NIST) Certificate of Analysis for coal tar standard reference material that states that coal tar emulsion sealants can contain up to 35% refined coal tar. PAH concentrations reported by Rowe and O'Connor (2011) are consistent with those of the other three studies, suggesting that the HTCTP content of the product used in that study was comparable to that used in the other three studies.

Using data from these studies, PECs for individual and total PAHs were estimated for surface waters and bottom sediment in 10 Canadian urban centres (ECCC 2018). Total PAH estimates were based on the 16 U.S. EPA priority pollutant PAHs, although it is recognized that this represents only a subset of the total PAH and non-PAH components expected to be present in the runoff (U.S. EPA 2013). Of the 10 urban centres selected, 6 were large, 2 were medium-sized and 2 were small. It was assumed that all runoff goes into the storm sewer system and into downstream water bodies without any treatment. While stormwater runoff is sometimes routed to a wastewater treatment system (WWTS) (e.g., in older combined sewer infrastructures), it is more commonly released directly into the environment without prior treatment.

The following data were used in the estimations. Approximately $10\,500 \pm 50\%$ tonnes of CTPS are sold annually in Canada (see Uses section; EHS 2010). The upper end of this range, or 15 750 tonnes per year of CTPS, was used to estimate the quantity applied annually in Canada. The quantity estimate is corroborated by newly collected data (ECCC 2019), which are higher but within the same order of magnitude. The amount of CTPS used in each urban centre was proportioned based on its population relative to the total urban population of Canada (approximately 27 million). The mass of PAHs in unfiltered samples of runoff was scaled to the Canadian urban centres based on their land area and expected area of pavement covered with CTPS. The total mass of PAHs was then used to determine the concentration of individual PAHs, based on their percentage contribution to the total PAHs concentration. A full description of the estimation method and calculations is provided in ECCC (2018). The results for one large urban centre are provided in Table 9-5 of section 9.3.1, while PECs for all of the 10 selected locations within Canada are provided in ECCC (2018).

9.2.3.3 Partitioning to sediment

The PECs for PAHs in bottom sediment were calculated using a sediment-water partitioning model. This approach is based on an equilibrium partitioning principle described by the European Chemicals Agency (ECHA 2012) and incorporates two additional calculation methods. The first step is to estimate the substance's concentration in the aqueous phase (truly dissolved) of the overlying water from its total concentration, according to studies by Gobas (2007 and 2010). The second step is to estimate the substance's concentration in bottom sediment from its concentration in the aqueous phase of the overlying water based on an equilibrium partitioning assumption between bottom sediment and overlying water, as described by the U.S. EPA's National Center for Environmental Assessment (U.S. EPA 2003). At equilibrium, the PEC in

bottom sediment can linearly correlate with the concentration in the aqueous phase of the overlying water. Sediment exposure scenarios were developed as an extension of the runoff release scenarios described above to determine equilibrium sediment exposure concentrations.

The sediment PECs have been standardized to 1% organic carbon to be comparable with the PNEC values (CCME 1999b), which have also been standardized to this value (CCME 1995). This allows for appropriate risk quotient analyses. However, typical values of organic carbon in bottom sediments are 1% to 3% for rivers and estuaries and 2% to 4% for lakes (Gobas 2010).

Additional information on the approach is available in ECCC (2018). The resulting PECs in bottom sediment for one large urban centre are presented in Table 9-6 of section 9.3.1 (Risk quotient analysis) below, and the results for all substances are presented in ECCC (2018).

9.3 Characterization of ecological risk

The approach taken in this ecological screening assessment was to examine assessment information and develop conclusions using a weight-of-evidence approach and precaution. Evidence was gathered to determine the potential for coal tars and their distillates to cause harm in the Canadian environment. Lines of evidence considered include those that support the characterization of ecological risk.

9.3.1 Risk quotient analysis

Risk quotient analyses were performed by comparing the various estimates of exposure (PECs; see section 9.2, Ecological exposure assessment) with ecotoxicity information (PNECs; section 9.1, Ecological effects assessment) to determine whether there is potential for ecological harm in Canada. Risk quotients (RQs) were calculated by dividing the PEC by the PNEC for relevant environmental compartments and associated exposure scenarios. RQs were determined for PAHs only, as they are the major components of coal tar substances and can be used to represent the toxicity of the coal tar mixture as a whole. The environmental quality guideline for individual PAHs in the appropriate media was used as the PNEC (see section 9.1.5).

In order to determine the combined risk of total PAHs in an environmental media, a sum of RQs (i.e., sum of PEC/PNEC) approach was followed (Calamari and Vighi 1992; Backhaus and Faust 2012) as PAHs have a narcotic mode of action and are considered to be concentration additive (Di Toro et al. 2000; Di Toro and McGrath 2000). In this approach, the sum of RQs for individual components (i.e., individual PAHs) of the substance are added together to obtain an overall RQ for the substance. This broad-based approach is similar to the concentration addition approach using toxic units for a mixture (Vighi et al. 2003) as it compares an exposure concentration (PEC) to an effect or no-effect level (PNEC) for substances with similar modes of action. However, the

sum of RQs approach differs from the toxic unit approach in that the toxicity data for each substance in the mixture must refer to the same biological endpoint and organism (Backhaus and Faust 2012). The sum of RQs approach is considered more conservative than the toxic unit approach. Nevertheless, where disparate toxicity data with regard to biological endpoints and organisms are only available for mixture components, as in the case with PAHs, it is the only useable approach (Backhaus and Faust 2012).

It is acknowledged that the number of PAHs for which environmental quality guidelines are available does not represent the total number of polyaromatic components present in coal tar substances, nor does the sum of RQs for PAHs take into account the potential ecotoxicity contribution of non-PAH components within coal tar substances. Therefore, the sum of RQs obtained based on the environmental quality guidelines may underestimate the potential for effects. However, the PAHs examined in the RQ analysis are those likely present in the highest proportions in the coal tar substance.

9.3.1.1 Releases from industry

9.3.1.1.1 Releases to soil from air deposition

The estimated soil concentrations resulting from 10 years of deposition of the selected PAHs to soil 1 kilometre downwind from the industrial area in Hamilton, Ontario, where the coal tar refinery is located were compared to the Canadian Soil Quality Guidelines (CCME 2010) to calculate the RQs for soil for each PAH considered (Table 9-4). This estimate was based on air emission data for 2012 and does not reflect past, higher emission levels or future changes in emissions. While comparison of NPRI data for the years 2012 to 2016 shows some fluctuation in annual quantities for the individual PAHs examined in this analysis, overall releases to air from this facility have remained relatively constant over time. These fluctuations are therefore not expected to affect the outcome of this analysis. As the coal tar refinery has been in operation at this location for over 20 years, a PEC based on 10 years of deposition at 2012 emission levels was considered to be a reasonable estimate of the impact from long-term releases.

Table 9-4. PECs, PNECs and RQs for PAHs in soil resulting from industrial air releases from coal tar refining

PAH	Soil PEC (after 10 years of deposition) (mg/kg dw)	Soil PNEC (CCME guidelines) (mg/kg dw)	Soil RQ (PEC/PNEC)
Phenanthrene	0.053	0.046	1.15
Naphthalene	0.0074	0.013	0.57
Acenaphthene	0.0089	0.28	0.03
RQ sum	-	-	1.8

Based on results obtained for the three PAHs emitted in highest quantities by the coal tar refinery in 2012, it is considered that PAHs depositing to soil from industrial air

releases during coal tar refining have the potential to cause adverse effects. The soil CCME guidelines for these three PAHs took runoff waters into consideration and were derived for the protection of freshwater life. Therefore, the above results indicate that there is a potential risk of harm to aquatic organisms that might be exposed to PAHs through runoff from the surrounding contaminated terrestrial environment. This analysis considered only the top three PAHs emitted by the coal tar refiner; if additional PAHs had been included, the sum of RQs, and thus the estimated risk, would have been even greater. Also not quantitatively considered here were releases of PAHs from coal tar storage at the two Hamilton-area steel mills and other coal-tar-related components released by the coal tar refinery, such as cresols and quinoline, which were reported to be released in quantities in the same range as phenanthrene in 2012 (NPRI 2014). Inclusion of releases of PAHs from coal tar storage at the steel mills would result in additional PAH loading and increased risk. However, given that a risk to the soil environment was indicated based on the releases of only three PAHs from the coal tar refinery, it was not considered necessary to quantitatively consider the releases from coal tar storage at the steel mills or of other coal-tar-related components released by the coal tar refinery for this scenario.

9.3.1.1.2 Releases to water

The maximum estimated concentrations of various coal tar components discharged by the coal tar refinery to the sewer system result in WWTP influent concentrations that do not exceed the Canadian Water Quality Guideline values for these parameters (Table 9-3). The concentrations of coal tar components in the WWTP effluent will be even lower than those in the influent due to physical-chemical treatment processes at the WWTP. Therefore, these releases are not in themselves considered to present a risk to the environment, although they do contribute to total loading from all sources.

9.3.1.2 Releases from use of coal-tar-based pavement sealant

Tables 9-5 and 9-6 show the estimated aquatic and sediment PECs, PNECs and RQs based on runoff from CTPS-coated surfaces entering receiving water bodies for one of the large urban centres considered. PECs, PNECs and RQs for individual PAHs for the other nine urban areas considered are tabulated in ECCC (2018).

Table 9-5. Summary of aquatic PECs, PNECs and RQs for PAHs from CTPS runoff in receiving water for one large urban centre in Canada

PAH	PEC (µg/L)	PNEC ^a (µg/L)	Aquatic RQ (PEC / PNEC)
Naphthalene	<0.001	1.1	<0.01
Acenaphthene	0.004	5.8	<0.01
Fluorene	0.004	3.0	<0.01
Phenanthrene	0.020	0.40	0.05
Anthracene	0.006	0.012	0.52
Fluoranthene	0.019	0.04	0.48

PAH	PEC (µg/L)	PNEC ^a (µg/L)	Aquatic RQ (PEC / PNEC)
Pyrene	0.015	0.025	0.59
Benzo[a]anthracene	0.006	0.018	0.32
Benzo[a]pyrene	0.005	0.015	0.35
Acenaphthylene	<0.001	N/A	_ ^b
Chrysene	0.006	N/A	_ ^b
Dibenz[a,h]anthracene	0.001	N/A	_ ^b
Sum of RQs	-	-	2.3

N/A – not applicable; no Canadian Water Quality Guideline was available for this PAH.

^a Aquatic PNECs are the Canadian Water Quality Guidelines for the protection of aquatic life (CCME 1999a).

Table 9-6. Summary of sediment PECs, PNECs and RQs for PAHs from CTPS runoff in receiving water for one large urban centre in Canada

PAH	Sediment PEC (µg/kg dw)	Sediment PNEC ^a (µg/kg dw)	Sediment RQ (PEC / PNEC)
Naphthalene	0.001	34.6	<0.01
Acenaphthene	0.148	6.71	0.02
Fluorene	0.194	21.2	<0.01
Phenanthrene	3.12	41.9	0.07
Anthracene	0.911	46.9	0.02
Fluoranthene	5.45	111	0.05
Pyrene	4.59	53.0	0.09
Benzo[a]anthracene	2.32	31.7	0.07
Benzo[a]pyrene	2.54	31.9	0.08
Acenaphthylene	0.004	5.87	<0.01
Chrysene	2.02	57.1	0.04
Dibenz[a,h]anthracene	0.523	6.22	0.08
Sum of RQs	-	-	0.53

^a Sediment PNECs are the Canadian Sediment Quality Guidelines (CCME 1999b).

RQs for individual PAHs were less than 1 for both the aquatic and sediment compartments at all urban centres examined in the analysis (ECCC 2018). Table 9-7 summarizes the risk quotient data for each of the 10 urban centres considered in the analysis. The sum of the RQs for individual PAHs at each urban centre ranged from 0.6 to 2.3 in the aquatic compartment and from 0.1 to 0.5 in the sediment compartment. Aquatic RQs were greater than 1 at 9 of the 10 urban centres considered, indicating a potential for risk, while sediment RQs were less than 1 at all urban centres examined.

Table 9-7. The sum of RQs for total PAHs from CTPS calculated for ten urban centres in Canada

Urban centre	Sum of RQs (aquatic)	Sum of RQs (sediment)
Location 1 (L) ^a	2.3	0.5

Urban centre	Sum of RQs (aquatic)	Sum of RQs (sediment)
Location 2 (L) ^a	1.9	0.4
Location 3 (L) ^a	1.7	0.4
Location 4 (L) ^a	1.5	0.4
Location 5 (L) ^a	1.2	0.3
Location 6 (L) ^a	1.7	0.4
Location 7 (M) ^b	0.6	0.1
Location 8 (M) ^b	1.3	0.3
Location 9 (S) ^c	1.5	0.3
Location 10 (S) ^c	1.3	0.3

^a L = large urban centre (population of 100 000 and over)

^b M = medium-sized urban centre (population of 30 000 to 99 999)

^c S = small urban centre (population of 1000 to 29 999)

For all studies, highest PAH concentrations occurred in runoff originating from the initial precipitation event or wetting of the coal-tar-sealed pavement, with lower concentrations measured in runoff from subsequent wetting events. PAH concentrations were especially high in the initial runoff from Watts et al. (2010; 5890 µg/L) and McIntyre et al. (2016; 1180 µg/L) compared with those of Rowe and O'Connor (2011; 277 µg/L) and Mahler et al. (2014; 190 to 409 µg/L). The reason for the higher concentrations measured in Watts et al. (2010) and McIntyre et al. (2016) relative to those of Rowe and O'Connor (2011) and Mahler et al. (2014) is uncertain, but indicates that PAH concentrations released into the environment in CTPS runoff can vary greatly. While highest concentrations were observed in first flush runoff, the contribution to the overall amount of PAH being released to the environment was much higher for the combined total of post-initial precipitation events (ECCC 2018). For example, for the large urban centre considered in Tables 9-5 and 9-6 above, the mass of PAH released in the first flush event was calculated to be 34.5 kg, while the total mass of PAH released in subsequent wetting events was 300 kg/yr over the 2-year period considered in the analysis (ECCC 2018). It is acknowledged that some PAH degradation would also be expected to occur over this 2-year period and this could not be quantitatively incorporated into the analysis.

The results of the risk quotient analysis indicate that PAHs in runoff from CTPS-coated areas have the potential to cause adverse effects in surface water organisms in Canada. However, PAH concentrations in CTPS runoff are unlikely to reach levels causing adverse effects in sediment organisms. While RQs for the sediment compartment were less than 1, it should be noted that the persistence of some PAHs in sediment suggests there is potential for long-term accumulation of PAHs in the sediment bed that could lead to adverse effects in sediment organisms, as well as exposure to pelagic species should sediment re-suspension occur.

9.3.2 Weight of evidence for determining potential to cause harm to the Canadian environment

Based on empirical and modelled data, most PAHs that are major components of coal tars and their distillates are expected to biodegrade slowly and may therefore persist in water, soil and sediment. While PAHs are rapidly degraded in air, the presence of some PAHs in remote regions, such as the Arctic, provides evidence that these substances have the potential to persist for long periods in air adsorbed to particulate matter and may undergo long-range transport to areas far from their point of release. High-temperature coal tars and their distillates also contain substantial proportions of non-PAH components, including phenolic compounds and heterocyclics. In general, the phenolic compounds are not expected to persist in the environment for long periods, while the heterocyclics may persist in the environment longer as they do not biodegrade rapidly. Some volatile components, such as pyridine and dibenzofuran, have the potential to remain in air for longer periods of time.

The combined evidence of empirical and modelled data shows that coal tars and their distillates contain components that have the potential to be highly bioaccumulative in aquatic organisms. This includes some three-ring PAHs, as well as several four-, five- and six-ring PAHs. Most fish species are able to efficiently metabolize PAHs, reducing the potential for bioaccumulation in fish and biomagnification in the food web. However, PAHs are not well metabolized in some invertebrates and can bioaccumulate to high levels in these species. Most non-PAH components of coal tars and their distillates are not highly bioaccumulative; monocyclic aromatic hydrocarbons and phenols have low bioaccumulation potential, while some heterocyclic representative structures may have some bioaccumulation potential. Most PAH and non-PAH components of coal tars and their distillates are not expected to biomagnify in aquatic or terrestrial food webs, largely because a combination of metabolism, low dietary assimilation efficiency and growth dilution allows the elimination rate to exceed the uptake rate of these compounds from the diet. However, Harris et al. (2011) found evidence that some alkylated PAHs may biomagnify in the sea otter food web.

Coal tars demonstrate moderate to high acute toxicity in laboratory testing with fish, aquatic invertebrates and algae. Toxicity is associated with both whole (unfiltered) solutions and the dissolved-only fractions, as well as with direct sediment contact, indicating that both water column and sediment species may be at risk following entry of a coal tar substance into the aquatic environment. In addition, whole sediment testing with early-life stage sturgeon confirmed that coal tar submerged in sediment for between 40 and 140 years retains its toxicity to this species, and therefore possibly to others.

PAHs are the primary components of coal tars and their distillates and most PAHs present as major coal tar components have demonstrated acute and chronic toxicity to aquatic and terrestrial organisms. These substances are therefore considered to pose high ecological hazard potential.

Considering the release of coal tars and their distillates from facilities and their associated processes, an analysis to estimate concentrations in soil resulting from the deposition from air of three PAHs released from a coal tar refining facility predicted that

levels of phenanthrene exceed the no-effect level specified in the CCME Canadian Soil Quality Guidelines (CCME 2010). As well, the three PAHs modelled as a group (naphthalene, phenanthrene and acenaphthene) were determined to pose a risk based on a combined RQ of 1.8. While not quantified in this report, other PAHs released from coal tars and their distillates from the coal tar refining facility would add to this combined risk. Therefore, releases of coal tar and their distillates at coal tar refining facilities depositing to soil have the potential to exceed levels that elicit adverse effects in freshwater organisms exposed through runoff from the surrounding terrestrial environment.

In addition, a risk quotient analysis was performed to determine the potential for risk from the release of PAHs present in runoff from coal-tar-sealed paved surfaces. The analysis considered PAH concentrations over a 2-year period using results from four different studies to provide a weight of evidence due to uncertainties within each study. For all studies, highest PAH concentrations occurred in runoff originating from the initial precipitation event or wetting of the coal-tar-sealed pavement, with lower concentrations measured in runoff from subsequent wetting events. However, while highest concentrations were observed in initial precipitation event runoff, the contribution to the overall amount of PAH being released to the environment for the combined total of post-initial precipitation events was much higher (ECCC 2018). The analysis determined that predicted environmental concentrations of individual coal-tar-related PAHs did not exceed the no-effect levels for pelagic and sediment organisms set out in the CCME Canadian Water and Sediment Quality Guidelines (CCME 1999a, 1999b) for all 10 urban centres examined. Risk quotients calculated as the sum of individual PAH risk quotients at each urban centre considered ranged from 0.6 to 2.3 in the aquatic compartment and from 0.1 to 0.5 in the sediment compartment. Aquatic RQs were greater than 1 at 9 of the 10 urban centres, indicating a potential for risk, while sediment RQs were less than 1 at all 10 urban centres. The quantitative analysis considered a subset of the total PAH and non-PAH coal-tar-based components that are expected to be present in the runoff and does not include all components that could contribute to the toxicity of the whole substance. It is acknowledged that the quantitative analysis presented in this assessment could demonstrate an increased risk if all components within the coal tar substance were considered.

Considered together, the information presented in this screening assessment indicates that coal tars and their distillates have the potential to cause ecological harm in Canada, as a result of the deposition onto soil of coal-tar-related substances in air emissions from a coal tar refinery located in Canada, as well as from the use of coal-tar-based pavement sealants in Canada. Releases of coal-tar-related substances from the coal tar refinery to water entering wastewater treatment systems are not likely to be causing ecological harm in Canada.

9.3.3 Sensitivity of the conclusion to key uncertainties

The application of modelling approaches to estimate the physical and chemical properties, persistence, and bioaccumulation potential of major coal tar components

introduces uncertainty into the analysis of these characteristics in the mixture. However, while coal tars and their distillates are themselves UVCBs, their components are discrete substances that can be effectively modelled and, for this reason, model estimates for individual components were deemed to be reliable. In addition, when reliable empirical data related to the modelled parameters were available (e.g., provided in EPI Suite (2000–2010)), these values were used instead of the modelled data.

HTCTP is known to be used as an asphalt rejuvenator, while coal tar pitch is used in roof resurfacing activities. However, not enough information was available to characterize the environmental releases from these uses in Canada. Uses of these products are believed to be infrequent and were not considered to be a significant source of exposure to the environment, relative to other sources considered in this assessment. Releases from such activities would result in incremental increases in environmental concentrations.

In terms of the estimation of the quantity of PAHs in runoff from CTPS-treated pavements, the values used in the exposure calculations are based on measured values for a 2-year period from four different studies to provide a weight of evidence due to uncertainties within each study. However, because all four studies used sealant products that contained HTCTP at the high end of the 10% to 30% content range, the estimate for the release of PAHs from asphalt sealant to water is based only on data from sealants with high HTCTP, which might overestimate the PEC. It is unclear whether CTPS products containing HTCTP at the lower end of the 10% to 30% range are currently in use in Canada and, if so, whether PAH concentrations released into the environment from surfaces coated with these products would be lower than those measured in the four studies used for PEC determination. No information was found on these lower-HTCTP products, and PECs were therefore calculated using the available data.

The bioavailability of PAHs in the environment is also an important consideration. Empirical evidence indicates that the PAHs that predominate in coal tars and their distillates may be less available for uptake by organisms than would be expected based solely on equilibrium partitioning principles, due to strong binding of the PAH with the carbon matrix of the coal tar product (Erickson et al. 1993; Paine et al. 1996; CCME 1999b; Alexander 2000; Ghosh et al. 2001; Neuhauser et al. 2006 and others). The sorption of PAHs to solid media such as organic particulates present in water and sediment and the effect of this sorption on bioavailability of the PAH could not be quantitatively considered in the analysis of risk.

Conversely, risk from the use of CTPS considered only a limited number of PAHs, i.e., those for which Canadian Environmental Quality Guidelines could be used as a basis for PNECs. Coal tars and their distillates are UVCBs, containing a multitude of PAHs and other non-PAH components, all of which can contribute to the hazard of the coal tar substances. Consideration of only a subset of components (i.e., 16 PAHs) in the estimation of risk from these substances underestimates the hazard and risk of CTPS. However, the assumption that PAHs are the primary components responsible for toxicity

is reasonable based on their predominance in coal tar substances and is also consistent in approach with other jurisdictions (e.g., European Commission 2008).

The risk analysis examines PAH concentrations as the coal tar substance first enters the environment and does not incorporate consideration of the continuous release of PAH and non-PAH components over time. This could lead to accumulation of the more stable and persistent PAHs, potentially leading to concentrations sufficient to cause adverse effects in organisms.

10. Potential to cause harm to human health

10.1 Exposure assessment

The assessment focuses on potential exposures to substances released during storage, handling and refining of coal tars in the vicinity of the site of the industrial facilities. The potential for exposures from products used by consumers, such as coal-tar-based sealants, is also considered. Due to the complex and variable nature of the targeted substances, it is difficult to estimate exposure to whole coal tar, coal tar oil, coal tar upper distillate and coal tar pitch. Exposure is characterized by choosing benzene, benzo[a]pyrene (B[a]P) and, more generally, the U.S. EPA priority PAHs as a marker for coal tars. PAHs and benzene are regarded as high-hazard components present in coal tar substances and have been included on the List of Toxic Substances under Schedule 1 of CEPA 1999. In addition to the six substances with the CAS RNs given in Table 2-1, other coal tars and their distillates are expected to have similar use patterns.

Industrial releases from the processing, handling and storage of coal tars and their distillates at a coal tar refinery

Releases of benzene and PAH constituents are associated with the storage and handling of coal tars and their related products and represent high-hazard constituents of concern. Releases of these components derived from coal tar at the site of the coal tar refinery are reported to NPRI. Analysis of NPRI data for 2012 reveals that a total of 4100 kg of benzene was released to air from stack and fugitive emissions (NPRI 2014). It has been determined from information provided by the refinery that it is not possible to definitively conclude the exact substance from which these emissions are originating due to extensive vapour capture and emissions control systems that are interlinked throughout the facility (personal communication, email from Ruetgers Canada Inc. to Risk Management Bureau, Health Canada, dated August 2013, unreferenced). Releases will therefore be considered to apply to all substances associated with coal tars and their distillates.

Dispersion modelling was used to determine concentrations of coal-tar-derived benzene to which the general population may be exposed in the vicinity of a coal tar refining site. Emission rates were derived based on the NPRI data and were used in SCREEN3

(1996) calculations to determine benzene dispersion at various distances from the respective industrial facility. Relevant input parameters for SCREEN3 modelling scenarios are presented in Appendix A (Table A-1 with results presented in Table A-2). Releases were considered area releases rather than point releases, given the size, potential points of emissions and location of the facility involved.

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). 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 1 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 (U.S. EPA 1992a). Similarly, for exposure events happening over the span of 1 year, it can be expected that the direction of the prevalent winds will be more variable and uncorrelated with the wind direction for a single event; thus, the maximum amortized exposure concentration for 1 year is determined by multiplying the maximum 1-hour exposure by a factor of 0.08. Such scaling factors are not used for non-point source emissions. However, to prevent underestimation of the exposures originating from area sources, a scaling factor of 0.2 was used to obtain the annual amortized concentration from the value of the maximum 1-hour exposure concentration determined by SCREEN3.

The results of the dispersion modelling indicate elevated levels of benzene at a distance of 1000 m from the source of release compared to the average background level in Canada. Map analysis has confirmed that residences exist within this distance. Benzene concentrations at 1000 m were estimated to be $1.45 \mu\text{g}/\text{m}^3$. This concentration is greater than the 2008 Canadian average background concentration of benzene in ambient air of $0.88 \mu\text{g}/\text{m}^3$ (NAPS 2008). As well, the modelled benzene air concentration of $1.45 \mu\text{g}/\text{m}^3$ exceeds the Ontario Ministry of Environment's ambient air quality criteria of $0.45 \mu\text{g}/\text{m}^3$ for annual air concentrations (Ontario 2012, 2016). The region around the coal tar refinery has a local monitoring network that monitors volatiles, including benzene. The annual average values at the three monitoring stations were between 1.6 and $3.3 \mu\text{g}/\text{m}^3$ for 2012, with maximum values ranging from 4.6 to $54.5 \mu\text{g}/\text{m}^3$ (HAMN 2012). The annual average values for 2018 at the three monitoring stations were between 1.2 and $1.9 \mu\text{g}/\text{m}^3$, with maximum values ranging from 2.4 to $6.3 \mu\text{g}/\text{m}^3$ (HAMN 2018). The generally higher values are associated with monitoring stations bounding on the city which, when combined with wind data, indicate that emissions from industry can disperse in the direction of the urban population as well as the Great Lakes (HAMN 2011). Other sources would be expected to contribute to the

levels measured at these monitoring sites in addition to the industrial releases considered here.

Masses of PAHs released in 2012 as a result of coal tar refining are reported to NPRI (2014). While comparison of NPRI data for the years 2012 to 2016 shows some fluctuation in annual quantities for the individual PAHs examined in this analysis, overall releases to air from this facility have remained relatively constant over time. These fluctuations are therefore not expected to affect the outcome of this analysis. These masses were modelled for their dispersion using SCREEN3. Generally, between 80% and almost 100% of PAHs with five rings or more (which are predominantly particle-bound in the atmosphere) can be found associated with particles having an aerodynamic diameter of less than 2.5 µm. For many emission sources, particulate PAHs are predominantly observed in fractions of fine particles with a diameter ranging between 0.01 and 0.5 µm, but can approach values of several microns in ambient air (Ontario 2011; European Commission 2001). During normal exposure to PAH-containing aerosols, a major fraction (probably > 80%) of the inhaled PAHs is expected to be deposited on the thin alveolar epithelium (epithelium thickness of 1 to 2 µm) and is rapidly absorbed into the blood (Ontario 2011). These considerations support the dispersion modelling of PAHs and potential inhalation exposure for those residing in the vicinity of a release source.

Relevant parameters used in the modelling are presented in Health Canada (2014). The results of the modelling at 1000 m from the site of the area release are presented in Table 10-1, with further details in Health Canada (2014). The concentration at 1000 m was used to characterize long-term general population exposure for those residing in the vicinity of the facility.

Table 10-1. SCREEN3 modelled air concentrations of PAH releases from a coal tar refinery

Compound	Annual concentration maximum at 1000 m (ng/m³)
Acenaphthene	480
Acenaphthylene	2.8
Acethracene	43
Benzo[a]anthracene	7.2
Benzo[a]pyrene	0.71
Benzo[b]fluoranthene	5.0
Benzo[g,h,i]perylene	1.4
Benzo[k]fluoranthene	1.9
Dibenzo[a,h]anthracene	0.28
Fluoranthene	66
Fluorene	74
Indeno[1,2,3-c,d]pyrene	1.5
Phenanthrene	180

Pyrene	44
Naphthalene	3110

The resulting air concentration of B[a]P was determined to be 0.71 ng/m³. The modelled concentration is greater than the 2012 Canadian average background concentration of B[a]P in ambient air of 0.14 ng/m³ (NAPS 2012). Recent air monitoring activities in close proximity to the coal-tar-related and other industrial sites have shown B[a]P annual means ranging from 0.08 to 1.84 ng/m³, which exceeds the Ontario Ministry of Environment’s ambient air quality criteria for annual air concentrations of 0.01 ng/m³ (HAMN 2011; Ontario 2012). Furthermore, the 24-hour average air concentration was exceeded 35 times, with maximum 24-hour air concentrations reaching 3.7 to 7.0 ng/m³ at three different stations. It is noted that the principle source of B[a]P in Hamilton is coke oven emissions, a process that produces coal tars (HAMN 2011). Ambient concentrations of eight PAHs were also monitored at three stations in Hamilton and are presented for context and validation of SCREEN3 results in Table 10-2 (HAMN 2012). Other sources would be expected to contribute to the levels measured at these monitoring sites in addition to the industrial releases considered here.

Table 10-2. Ambient air concentrations of PAHs in Hamilton

Compound	Annual average concentration in 2012 (ng/m ³) ¹	Annual average concentration in 2017 (ng/m ³) ²
Benzo[a]anthracene	1.41	0.40
Chrysene	1.92	0.73
Benzo[b]fluoranthene	2.06	0.92
Benzo[k]fluoranthene	1.56	0.60
Benzo[a]pyrene	1.40	0.49
Indeno[1,2,3-cd]pyrene	1.15	0.62
Dibenzo[a,h]anthracene	0.36	0.17
Benzo[g,h,i]perylene	1.16	0.67

¹ HAMN 2012

² HAMN 2017

SCREEN3 is recognized as being a conservative dispersion model, compared to more advanced models that require highly detailed inputs. Thus, AERSCREEN (U.S. EPA 2011a) was also used, with parameters that are considered to be site-specific. AERSCREEN is the screening model based on AERMOD (U.S. EPA 2011a). The model will produce estimates of “worst-case” 1-hour concentrations for a single source, without the need for hourly meteorological data and also includes conversion factors to estimate “worst-case” 3-hour, 8-hour, 24-hour and annual concentrations. AERSCREEN is intended to produce concentration estimates that are equal to or greater than the estimates produced by AERMOD, without a fully developed set of meteorological and terrain data (U.S. EPA 2011a). Benzene emissions modelled with AERSCREEN for a coal tar refinery produced estimates of 1.14 µg/m³ at 1000 m, a difference of 0.31 µg/m³ compared to SCREEN3, which produced a value of 1.45 µg/m³ in Health Canada (2014). Additionally, AERSCREEN (U.S. EPA 2011a) was used to model B[a]P

dispersion with parameters that are considered to be site-specific. The resulting concentration at 1000 m from the site of release was estimated to be 0.55 ng/m³ after the 1-hour concentration was converted to an annual concentration, a difference of 0.16 ng/m³ compared to SCREEN3, which produced a value of 0.71 ng/m³ (Health Canada 2014). AERSCREEN results suggest that, while conservative, the values generated by SCREEN3 are valid.

Industrial releases from the handling and storage of coal tar at integrated steel mills

Emissions of volatile compounds are associated with industries that produce and refine coal tars. Benzene emissions from industrial releases arise from coal tar storage and handling following the coking process at steel mills. Releases from steel mills are reported to NPRI, with benzene considered to be a substance of concern to human health for those residing in the vicinity of such operations. Analysis of the 2012 NPRI data for the four integrated steel mills in Canada revealed that a combined total of approximately 96 tonnes of benzene was released to air from stack, handling and fugitive emissions (NPRI 2014). It was reported from information provided by the steel sector that a weighted average of 8.03% of site-wide benzene emissions are specific to coal tar storage and handling (personal communication, email from Canadian Steel Producers Association to the Risk Management Bureau, Health Canada, dated February 2014, unreferenced). The largest reported benzene emission from an individual steel mill was 39 tonnes (NPRI 2014), resulting in a coal-tar-specific benzene emission value of 3132 kg.

Dispersion modelling was used to determine concentrations of coal-tar-derived benzene to which the general population may be exposed in the vicinity of coal tar storage tanks at steel mills. Emission rates were derived based on the NPRI data and the industry-submitted information on release percentage. SCREEN3 (1996) was used to determine benzene dispersion at various distances from the respective industrial facilities.

Relevant input parameters for SCREEN3 modelling scenarios are presented in Appendix B (Table B-1 with results presented in Tables B-2 and B-3). All releases were considered area releases rather than point releases, given the size and locations of the facilities involved. Emissions rates from steel mills for benzene were scaled to 8.03% of the reported values to account for emissions from coal tar storage and handling, which could be considered attributable to the substances under assessment.

The modelled results show benzene levels below or slightly above that of background, i.e., a maximum of 0.11 to 1.11 µg/m³ at 1000 m from the source of release. The concentration profile of benzene emissions for the largest emitter reaches the average background of 0.88 µg/m³ at 1200 m. Map analysis shows that residential homes exist within 1 km of release sites.

While no emissions of PAHs were attributed to coal tar storage and handling, an estimation of potential exposure from releases of PAHs was determined based on the ratio of benzene to B[a]P equivalents (a value obtained from the coal tar refinery scenario, and using potency equivalency factors). This is described in further detail in the Characterization of Risk section, Table 10-6.

Coal tar transport from integrated steel mills

Releases from washing or cleaning transportation vessels are not considered in this screening assessment, as tanks or containers for transferring coal tar substances are typically dedicated vessels and therefore washing or cleaning is not required on a routine basis (U.S. EPA 2008).

The total volatiles released from coal tar were estimated using the physical-chemical properties of coal tar (ArcelorMittal 2010) and AP-42 emissions equations (U.S. EPA 2008) for each mode of transport. The approximate amount of benzene contained in raw coal tar, as produced by integrated steel mills, was reported to be between 0.06 and 0.29 wt% (ArcelorMittal 2010). To ensure a conservative estimate for determining the potential benzene exposure values in the vicinity of transit sites, the upper bound of the range was selected for the mass of benzene present. A breakdown of the transport types was provided by industry to be 40% to 50% by marine, 40% to 50% by truck, and 3% to 7% by rail for transport to a coal tar refinery (personal communication, email from Ruetgers Canada Inc. to Risk Management Bureau, Health Canada, dated August 2013, unreferenced).

A conservative estimate for these transit losses may be calculated by using stationary storage tank formulas, adapted to typical dimensions of truck and train tanks. Even at this level of conservatism, due to the low volatility of coal tars, the evaporative emissions from marine, truck and train transit are small. It has been assumed that releases would occur over 1 day of travel time based on the close proximity of the integrated steel mills to the coal tar refinery.

Total annual releases of coal tar volatiles to air from all marine transport were estimated to be 139 kg, with less than 1 kg considered to be benzene based on the acknowledged weight percent. Truck transport is associated with 43 kg of total emissions, and less than 1 kg of benzene per year. Releases to air from rail transport are expected to be small due to the presence of pressure safety valves, and the small proportion of coal tar transferred by rail. This was estimated to be approximately 7 kg of total volatiles and less than 1 kg of benzene.

Coal tar product transport from a refinery

Releases of volatiles can also occur during the loading and transport of coal tar products.

Information on the percentage of distilled products transported by railcar (40% to 60%) or tanker truck (30% to 50%) was provided by industry to estimate the amount of each product transported. The total volatiles released from coal tar products were estimated using the physical-chemical properties of the substances at relevant transport temperatures and AP-42 emissions equations (U.S. EPA 2008) for each mode of transport. The approximate percentage of product streams was considered as 50% HTCTP (65996-93-2) and 35% tar oils (65996-82-9 and 6599691-0) (Blumer and Sutton 1998), which was combined with an estimated input of 200 kt per year to determine the mass of each product produced.

Transport of the products was considered for three priority distillates (CAS RNs 65996-91-0, 65996-82-9 and 65996-93-2). The analysis of the transport of HTCTP at 50 °C did not lead to an appreciable generation of volatile releases given its extremely low vapour pressure and solid state. Consideration of the vapour pressure at 200 °C, which would allow for a liquid state, led to the generation of approximately 210 kg and 320 kg of total volatiles for truck and railcar, respectively. Assuming a tanker volume of 30 000 L, releases per tank car are estimated to be from 0.16 kg to 0.24 kg during 1 day of transit, in the absence of pressure valves and venting to the atmosphere. The transport of other distillates including coal tar oils (CAS RN 65996-82-9) at 20 °C led to the generation of approximately 22 kg and 165 kg of total volatiles for truck and railcar, respectively. Assuming a tanker volume of 30 000 L, releases per tank car are estimated to be from 0.17 kg to 0.26 kg during 1 day of transit, in the absence of pressure valves and venting to the atmosphere. The low vapour pressure associated with the remaining distillate (CAS RN 65996-91-0) resulted in an estimated total release of less than 22 kg for truck transport and 28 kg for railcar transport at 100 °C, based on 1 day of transit time.

In summary, transient exposures associated with the transportation of coal tars and their distillates are minor given the small amount of volatiles released by each mode of transport, as well as the fact that the releases of volatiles that occur during the transit process occur continuously from a moving source (a line source) rather than from a stationary point source. Consequently, the actual concentration of the coal-tar-derived vapours around a moving line source, for any given location, will be considerably lower than that at the site of production and processing, which occurs at a stationary site and is associated with a larger amount of air releases. Thus, it is not possible to reliably establish the concentration to which the general population would be exposed, except to consider it to be much less than the potential exposures associated with production and refining.

Products used by consumers – coal-tar-based pavement sealants (CTPS)

Dust exposure from CTPS

As noted in the Uses section, coal tars and their distillates are used in pavement sealants (typically 15% to 30% w/w) in Canada, which are generally applied to residential driveways and small commercial or residential parking lots (EHS 2010). A study conducted in Texas (Mahler 2010) analyzed PAH content in dust from 23 ground-

floor apartments and their parking lots, 11 of which were parking lots to which CTPS had been applied (herein called “CTPS parking lots”). The concentration of total PAHs was calculated as the sum of 16 parent PAHs—corresponding to the 16 priority PAHs identified by the U.S. EPA—measured in house dust from apartments with CTPS parking lots and was determined to be significantly higher than that measured in dust from apartments with parking lots of other pavement surface types (median concentrations of 129 and 5.1 µg/g, respectively; Table 10-3). Of the 17 variables tested for relation to total PAH levels in dust, pavement surface type was the most dominant single factor affecting the concentration of PAHs in dust, accounting for 48% of the observed variance.

Table 10-3. Median concentrations in dust (mg/kg) from 23 ground-floor apartments in Austin, Texas (Mahler et al. 2010)

Substance	With CTPS-based parking lots	With parking lots of other pavement surface types
Total PAH ^a	129	5.1
Benzo[a]pyrene	4.5	0.44

^a Determined as the sum of 16 parent PAHs, corresponding to the 16 priority PAHs identified by the U.S. EPA (see Table C-1 in Appendix C).

In the dust study described above, the median concentration of B[a]P in dust from apartments with CTPS parking lots (4.5 mg/kg) was 10 times higher than that in the dust from apartments with parking lots of other pavement surface types (0.44 mg/kg). The average concentrations of the 16 individual PAHs found in house dust (Mahler et al. 2010) were considered for exposure (see Tables C-1, C-2 and C-3 in Appendix C). It was assumed that all soil and dust ingested contain the same concentration of PAHs attributed to CTPS. However, it is noted that soils near a sealcoated parking area contain a higher concentration of PAHs (UNHSC 2010).

Research conducted at the University of New Hampshire on simulated sealcoated surfaces resulted in findings similar to those of Mahler et al. (UNHSC 2010). Pavement dust collected from sealcoated surfaces was found to contain up to 1192 mg/kg total PAHs, compared to less than 2 mg/kg collected from unsealed surfaces (UNHSC 2010). Similarly, soils adjacent to sealcoated lots were consistently found to have total PAH concentrations exceeding 90 mg/kg, compared to 5 mg/kg at sampling control sites located some distance from the lot (UNHSC 2010). Concentrations of as high as 411 mg/kg total PAHs were detected in soil directly adjacent to the sealcoated parking surfaces, with the highest value of B[a]P (29 mg/kg) exceeding the U.S. EPA regional screening level preliminary remediation guideline of 0.21 mg/kg for surface soil at industrial locations (UNHSC 2010).

Inhalation exposure from CTPS

The potential for inhalation exposures to volatile PAHs for do-it-yourself application from coal-tar-based pavement sealants has been investigated for total PAHs, defined as the sum of 18 PAHs (similar to the 16 PAH priority pollutants identified by the U.S. EPA).

Concentrations of total PAHs in wet samples (in bucket) of two sealcoats ranged from 90 000 to 120 000 mg/kg (Diamond Environmental Group 2011). Following a 48-hour drying process, the concentrations were again measured and found to contain approximately 32 000 mg/kg, or a loss of approximately 70% of the PAH mass. Most lower molecular weight PAHs were lost as the sealcoat dried. The potential acute inhalation exposure to various PAHs was determined from the air concentrations following application of a CTPS. Of the 18 PAHs measured, 6 were below the detection limit of either 1 or 2 ng/m³. A further 2 PAH values were estimated from the data rather than directly determined, and were therefore not considered (Van Metre 2012a, 2012b). Three of the remaining 10 PAHs had values of less than 4 ng/m³, which were considered to be of minor consequence. The values of the remaining 7 measured PAHs are presented in Table 10-4.

Table 10-4. Ambient air concentrations at 1.28 m height 1.6 hours following CTPS application (Van Metre 2012a)

PAH	Concentration (ng/m ³)
Phenanthrene	4330
Anthracene	499
Fluoranthene	392
Pyrene	208
4,5-methylene-phenanthrene	190
1-methyl-phenanthrene	53.3
2-methyl-anthracene	21.3

Dermal exposure from CTPS

The potential for dermal exposure is considered to be relevant for the general population during the sealcoating of a driveway with a coal-tar-based product. Exposure associated with a homeowner using CTPS on their driveway is considered to be the most likely exposure to coal tars from products used by consumers. Thus, the do-it-yourself driveway sealcoating scenario is used to characterize dermal risk. During application, dermal exposure to CTPS may occur by way of spills, splashes and handling of the container or painting apparatus.

For the purpose of this assessment, contact with a small amount of sealcoat is assumed to occur via the skin of the palms of the hands. The dermal load associated with such exposure is derived using the EPA-Versar thin film approach (U.S. EPA 2011b). This approach characterizes the 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 a CTPS density of 1.19 g/cm³, with an upper limit of 28% w/w comprising pure coal tar and 227.5 cm² (or one quarter of each hand) as the exposed skin surface area, the dermal load was estimated to be 151.6 mg per exposure event. 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 estimated to be 2.1 mg/kg bw. This dose represents a

typical incidental dermal exposure event to coal tar that could reasonably occur during application of CTPS and was considered to occur once every 2 to 3 years or longer.

Other products used by consumers

Coal tars, crude and refined, are described on the Cosmetic Ingredient Hotlist as prohibited as ingredients in cosmetic products in Canada. The Hotlist is an administrative tool used by Health Canada to communicate to manufacturers and others that certain substances, when present in a cosmetic, may contravene (a) the general prohibition found in the *Food and Drugs Act* or (b) a provision of the *Cosmetic Regulations*.

Exposure to coal tar pitch from roof resurfacing activities is infrequent, as such activities are performed in the outdoor environment and usually by a professional contractor. Built-up roofing systems containing coal tar pitch (for flat and low-slope roofs) are not considered to be a significant source of exposure to pitch for the Canadian general population.

In a risk assessment of high temperature coal tar pitch (HTCTP) conducted by the European Commission, the use of this substance as an adhesive/binder in clay pigeons and charcoal briquettes was indicated (European Commission 2008). Clay pigeons are considered to be of hobbyist usage, and therefore exposure is expected to be limited and not representative of the general population. Exposure would be limited to direct dermal contact while handling the pigeon. However, because the pitch acts as a binder, the ability for transfer and uptake by the skin would be limited. With respect to charcoal briquettes, pitch is used in the production process as a binder. This use is limited to industrial applications in blast furnaces and foundries that require higher mechanical strength, limiting exposures to occupational settings. Barbecue briquettes used by the general public for cooking employ starch (typically corn-based) as a binder instead of coal tar pitch and thus exposures are not expected for this use (FAO 1987; e-mail from Kingsford Product Company to Existing Substances Risk Assessment Bureau, Health Canada, 2012, unreferenced).

Exposures from therapeutic products

Coal tar is listed in the Drug Product Database as an active ingredient present in human and veterinary drugs marketed for the management of psoriasis, eczema and dermatitis. These products are in the form of creams, lotions, shampoo and other topical solutions (DPD 2014). There is the potential for limited direct exposure to humans while using veterinary drugs intended for animal care.

Coal tar USP used in drugs is prepared by mixing a mass of coal tar with alcohol, polysorbate and washed sand, followed by seven days of mixing. The resulting solution is filtered and diluted with alcohol (U.S. Pharmacopia 2008–2010). The composition of coal tar USP therefore differs significantly from the coal tars considered in this assessment. Use of coal tar therapeutic products is recognized by the United States

Food and Drug Administration as Category I (safe and effective) for over-the-counter drug ingredients and for use in the treatment of dandruff, seborrhea and psoriasis (Health Canada 2006; CIR 2008; CFR 2013). Because these products have been considered acceptable therapeutic treatment options for certain skin conditions by international agencies and because they have been authorized and assigned Drug Identification Numbers (DINs) by Health Canada, their therapeutic use is not considered in this screening assessment (ATSDR 2002).

Potential general population exposure to the PAHs present in these products from post-consumer use is expected to be minimal, as these products represent a small fraction of sales in their respective product categories in Canada. Most coal-tar-containing drugs are formulated to contain approximately 0.5% to 10% coal tar, which limits the amounts entering receiving water (Health Canada 2006). Additionally, wastewater treatment plants are able to remove an average of 73% of PAHs conveyed to the plant, further limiting post-consumer exposure from receiving water (Pham and Proulx 1997). The low PAH levels from therapeutics will be further reduced through environmental biodegradation and/or drinking water treatment prior to consumption. The concentration in a water source is also significantly reduced via dilution, as it is released into waterways.

10.2 Health effects assessment

A critical effect for coal tars and their distillates was carcinogenicity, based primarily on classifications by international agencies. The European Commission, the International Agency for Research on Cancer (IARC) and the U.S. National Toxicology Program (NTP) have classified coal tars as carcinogens. The European Commission has classified coal tar (CAS RN 8007-45-2) and high-temperature coal tar (CAS RN 65996-89-6) as Category 1 carcinogens (*substances known to be carcinogenic to man*; R45: may cause cancer) and high-temperature coal tar pitch (HTCTP; CAS RN 65996-93-2) as a Category 2 carcinogen (*substances which should be regarded as if they are carcinogenic to man*; R45: may cause cancer) (European Commission 1994; ESIS c1995-2011). The Globally Harmonized System of Classification and Labelling of Chemicals (GHS) has classified coal tar and high-temperature coal tar as Category 1A carcinogens (*known to have carcinogenic potential for humans, classification is largely based on human evidence*; H350: may cause cancer) and HTCTP as a Category 1B carcinogen (*presumed to have carcinogenic potential for humans, classification is largely based on animal evidence*; H350: may cause cancer) (European Commission 2008). IARC has classified coal tars and coal tar pitch as Group 1 carcinogens (*carcinogenic to humans*), based on sufficient evidence in humans (occupational exposure) and experimental animals (IARC 1985, 1987a, b). The NTP has classified coal tars and coal tar pitches as *known to be human carcinogens*, based on sufficient evidence of carcinogenicity in humans (NTP 2011, 2016).

The European Union has also notified the World Trade Organization's Committee on Technical Barriers to Trade of plans to restrict HTCTP (CAS RN 65996-93-2) in

products used by consumers and to impose the labelling requirement “restricted to professional users” (WTO 2013). This restriction resulted from the recently agreed-upon harmonized classification of this substance by the European Union as carcinogenic, mutagenic or reprotoxic (CMR), Category 1A or 1B.

The European Commission has classified low-temperature coal tar (CAS RN 65996-90-9) as a Category 1 carcinogen and coal tar upper distillates (CAS RN 65996-91-0) as Category 2 carcinogens when the concentration of B[a]P is greater than 0.005% w/w. Coal tar oils (CAS RN 65996-82-9) are classified as both Category 2 carcinogens and Category 2 mutagens (*substances which should be regarded as if they are mutagenic to man*; R46: may cause heritable genetic damage) when the concentration of benzene is greater than 0.1% w/w (European Commission 1994; ESIS c1995-2011). The GHS classification of low-temperature coal tar is Category 1A carcinogen. Coal tar upper distillates are classified as Category 1B carcinogen when containing more than 0.005% B[a]P w/w. Coal tar oils are classified as both Category 1B carcinogens and Category 1B mutagens (*chemicals which should be regarded as if they induce heritable mutations in germ cells of humans*; H340: may cause genetic defects) when the concentration of benzene is greater than 0.1% w/w (European Commission 2008).

Summary of health effects

Appendix D contains a summary of available health information for high-priority coal tar substances, including coal tar, high-temperature coal tar, high-temperature coal tar pitch, coal tar oils, low-temperature coal tar, and upper coal tar distillates. This health effect information is considered representative of the characteristics of all coal tars and their distillates, including, but not limited to the six substances given in Table 2-1. Because these substances share similar physical-chemical properties, their toxicological properties can be similar. The health effects data from these six substances were used to construct a toxicological profile considered representative of all coal tar substances.

For the exposure scenarios involving volatile emissions from industrial facilities and house dust from CTPS, exposure to coal tar substances occurs in the form of exposure to components found within coal tar, rather than exposure to the whole coal tar substances (i.e., the parent substances). Components of coal tars and their distillates can be released from facilities during substance processing and/or storage and can enter house dust from the weathering of coal tar driveway sealcoat. Because certain components of coal tar substances are carcinogenic (e.g., certain PAHs and benzene), these high-hazard components form the basis of the exposure estimates and critical health effects considered in this assessment.

Acute toxicity of coal tar substances in rodents is low. Oral median lethal dose (LD₅₀) values ranged from 3300 to greater than 15 000 mg/kg bw in rats (European Commission 2008), and dermal LD₅₀ values ranged from greater than 400 mg/kg bw in rats to greater than 7950 mg/kg bw in rabbits (i.e., a dermal LD₅₀ value was not established) (European Commission 2008; ENTTOX 2005). A single dermal application

of 2041 mg/kg bw resulted in induction of aryl hydrocarbon hydroxylase (AHH) in the skin and liver of neonatal rats (Bickers et al. 1982; Mukhtar et al. 1982). Induction of AHH activity (two- to five-fold over control) was also observed in the skin of humans after a single application of 0.286 mg/kg bw (Bickers and Kappas 1978). There were no acute inhalation studies identified.

Few short-term toxicity studies were identified. The lowest observed adverse effect level (LOAEL) for orally administered coal tar substances was 37.5 mg/kg bw/day based on early mortality and degenerative changes in the liver of pigs exposed to high-temperature coal tar pitch (CAS RN 65996-93-2) for 2 or 5 days (Graham et al. 1940). An additional oral effect level of 1067 mg/kg bw/day was identified based on decreases in both body weight and food consumption in mice exposed to coal tar for 28 days (Culp and Beland 1994). The only inhalation study identified a lowest observed adverse effect concentration (LOAEC) of 30 mg/m³ in rats for lung histiocytosis (male and female), increased relative liver weight (female), and a decreased eosinophil count (male) after exposure to coal tar aerosol for 5 weeks (Springer et al. 1986). One dermal study reported that crude coal tar (10 mg of a 0.1% solution) caused comedogenicity (acne-like conditions) in rabbits after 3 weeks of exposure (Kligman and Kligman 1994).

Subchronic toxicity studies were also limited. Only one oral study was identified that tested manufactured gas plant residue. No adverse effects were observed in mice when exposed to maximum doses of 462 mg/kg bw/day (male) and 344 mg/kg bw/day (female) for 94 or 185 days (Weyand et al. 1994). Comedogenicity was observed after 10 mg of a 10% crude coal tar sample was applied to rabbits for 15 weeks (Kligman and Kligman 1994). Application of high-temperature coal tar pitch (CAS RN 65996-93-2) to mice for 31 weeks resulted in LOAEL values of 68 mg/kg bw/day (male) and 85 mg/kg bw/day (female) based on early mortality (Wallcave et al. 1971). Skin tumours were observed in these dermal studies. A LOAEC of 0.2 mg/m³ was identified based on decreased growth rate in several rodent species exposed to coal tar aerosol for 90 days (Kinkead 1973). An additional inhalation effect level of 30 mg/m³ was identified based on histiocytosis of lung tissue in male and female rats, increased relative liver weight (males), decreased volume of packed red cells (males), and decreased eosinophil and monocyte counts (females) after exposure to coal tar aerosol for 13 weeks (Springer et al. 1986).

Reproductive and developmental effects were observed in laboratory animals from exposure to coal tar substances. These effects often occurred at doses that were also maternally toxic. A developmental LOAEL of 140 mg/kg bw/day was identified based on an increase in anomalous fetuses, following oral exposure of rat dams to coal tar on gestational days 12 to 16. In the same study, a reproductive LOAEL of 180 mg/kg bw/day was identified based on an increase in the number of fetal resorptions (Hackett et al. 1984). Inhalation exposure of rats to coal tar aerosol for 13 weeks resulted in a reproductive LOAEC of 140 mg/m³ based on increased relative testis weight (Springer et al. 1986). A reproductive LOAEC of 660 mg/m³ was based on an increased incidence of mid- and late-gestational fetal resorptions after exposure to heavy distillate aerosol during gestational days 12 to 16 (Springer et al. 1982). Springer et al. (1982) also

reported a developmental LOAEC of 660 mg/m³ based on reduced fetal size and weight, as well as an increased incidence of litters with reduced ossification. In a dermal study in rats and mice, reproductive and developmental LOELs of 500 mg/kg bw/day were identified. Rodents were exposed to heavy distillate during gestational days 11 to 15, and developmental effects included decreased fetal size and weight, increased incidences of small lungs, cleft palate, edema, mid-cranial lesion, dilated ureter and renal pelvic cavitation, and reduced cranial ossification. Reproductive effects included increased mid- and late-gestational resorptions, decreased number of live fetuses per litter, and decreased placental and uterine weights (Zangar et al. 1989).

The majority of *in vitro* genotoxicity studies investigated the mutagenicity of coal tar substances using the Salmonella reverse mutation (Ames) assay. The 17 identified Ames studies all found positive responses, primarily with the addition of an exogenous metabolic activation system (see Appendix D for more details). The potential for coal tar substances to cause DNA adducts was also investigated in five studies using a variety of mammalian cell lines; all studies reported positive findings (Koganti et al. 2000; Leadon et al. 1995; Mahadevan et al. 2004, 2005, 2007). Other studies observed mixed results depending on the test system and conditions used (Casto et al. 1981; Curren et al. 1981; Mitchell et al. 1981).

The ability of coal tar substances to induce DNA adduct formation was also investigated *in vivo*. Fifteen oral studies were identified, with significant adduct formation observed in the lung, liver, forestomach and small intestine. Eight dermal studies were identified, with significant adduct formation observed primarily in the skin and lung. DNA adduct formation in human skin has also been observed in cases where coal tar was applied for therapeutic purposes (see Appendix D for more details).

Three oral carcinogenicity studies were identified for coal tar substances. Culp et al. (1998) exposed mice for 2 years to two separate coal tar mixtures that were mixed into the feed at doses ranging from 0 to 1300 mg/kg bw/day for Coal Tar Mixture 1 (CAS RN 8007-45-2; a composite from seven manufactured gas plant waste sites) and 0 to 346 mg/kg bw/day for Coal Tar Mixture 2 (CAS RN 8007-45-2; a composite from two of seven waste sites plus a third site having a high B[a]P content). A significant increase in early mortality was observed in both groups at the higher doses. Tumours were observed at multiple sites, particularly in the liver, lung and forestomach. Similar findings were observed in Culp et al. (1996) and Weyand et al. (1995) with doses ranging from 0 to 2000 mg/kg bw/day (2-year exposure) and 0 to 236 mg/kg bw/day (260-day exposure), respectively.

Several dermal carcinogenicity studies were conducted for coal tar substances, with the majority of the studies demonstrating skin tumours (Kligman and Kligman 1994; Brandon et al. 2009; Gorski 1959; Hueper and Payne 1960; Niemeier et al. 1988; Wallcave et al. 1971; Emmett et al. 1981; Kireeva 1968; Mukhtar et al. 1986b; Wright et al. 1985; Robinson et al. 1984; Phillips and Alldrick 1994). Various skin tumours developed on the ears of rabbits when dermally exposed to 10 mg of 10% to 100% coal tar (doses up to 5 mg/kg bw/day) for 15 weeks (Kligman and Kligman 1994). Mice also

developed skin tumours when exposed to both coal tar and coal tar pitch at doses of up to 833 mg/kg bw/day for 22 to 80 weeks (Gorski 1959; Robinson et al. 1984; Niemeier et al. 1988; Wallcave et al. 1971; Emmett et al. 1981). Study designs assessing the ability to initiate tumour development were also positive. A single dose of coal tar pitch in mice with doses ranging up to 20 mg/kg bw showed increased skin tumour incidence at all doses (Robinson et al. 1984). Single doses of 833 or 1333 mg/kg bw coal tar also resulted in tumours, as did repeated doses of 25 mg/kg bw (Phillips and Alldrick 1994; Wright et al. 1985; Mukhtar et al. 1986b).

Several inhalation carcinogenicity studies exposed mice and rats to coal tar aerosols (MacEwen et al. 1977; Kinkead 1973; McConnell and Specht 1973; Horton et al. 1963; Heinrich et al. 1994a, 1994b; Schulte et al. 1994). Squamous cell carcinomas of the lung were typically observed. Schulte et al. (1994) and Heinrich et al. (1994a, 1994b) observed dose-related lung tumour development after exposing rodents to coal tar pitch aerosol at concentrations of 0 to 2.6 mg/m³ for up to 20 months. Several studies using whole-body inhalation chambers resulted in skin tumour development, indicating dermal contact as another route of exposure in these studies. MacEwen et al. (1977) observed lung tumour formation in rodents, but also a dose-related response for skin tumours in mice following a 90-day whole body exposure to coal tar aerosols ranging up to 10 mg/m³.

A number of studies were identified regarding occupational exposures to coal tar that indicated increased relative risk estimates and mortality rates due to cancer for exposed workers. The European Commission conducted a meta-analysis for relative risk of lung and bladder cancers from various coal tar industry-related exposures (European Commission 2008). For lung cancer, an overall unit relative risk (URR) estimate of 1.20 (95% CI = 1.11–1.29) per unit of 100 µg/m³·year cumulative B[a]P exposure was determined, and a URR specific to the aluminum smelter industry was found to be 1.16 (95% CI = 1.05–1.28). For bladder cancer, an overall URR of 1.33 (95% CI = 1.17–1.51) per unit of 100 µg/m³·year cumulative B[a]P exposure was determined, and a URR specific to the aluminum smelter industry was found to be 1.42 (95% CI = 1.23–1.65). The U.S. EPA (1989) reported significantly increased mortality due to lung cancer in coke oven workers, and similar effects have been noted in steel workers. However, these effects are difficult to attribute specifically to coal tars given the variety of substances present at these facilities.

The Government of Canada previously completed a risk assessment of certain PAHs, including benzo[a]pyrene (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 1999.

The U.S. EPA previously identified PAHs that may be carcinogenic in animals and humans (U.S. EPA 1992b), ultimately listing 16 substances that became known as the

'priority pollutant' PAHs (Menzie et al. 1992). They are naphthalene, acenaphthylene, acenaphthene, fluorene, phenanthrene, anthracene, fluoranthene, pyrene, benz[a]anthracene, chrysene, benzo[b]fluoranthene, benzo[k]fluoranthene, benzo[a]pyrene, indeno[1,2,3-cd]pyrene, dibenz[a,h]anthracene and benzo[g,h,i]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) (CCME 2008; Collins et al. 1998; Krewski et al. 1989, Meek et al. 1994; Muller 1997; Nisbet and LaGoy 1992; U.S. EPA 1993). PEFs as developed by Nisbet and LaGoy (1992) were used in this assessment. 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 (European Commission 2008; WHO 1998, 2001). The PEF approach has been adopted herein as a method to characterize a systemic carcinogenic risk from oral exposure to PAHs derived from coal tars and their distillates.

Using U.S. EPA Benchmark Dose Software (BMDS 2.3.1) and a LogLogistic model, 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 (Appendix 5; Health Canada, unpublished). This reference value, referred to as an oral BMDL₁₀, was calculated to be 0.562 mg/kg bw/day and is based on forestomach papillomas and/or carcinomas in female B6C3F1 mice. This is of the same order of magnitude as oral BMDL₁₀ 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 BMDL₁₀ range for B[a]P of 0.1 to 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 drinking water guidelines recommend a maximum acceptable daily intake of 10 ng/L (0.010 ppb) B[a]P (Health Canada 1988, 2012).

Additionally, Health Canada previously developed estimates of carcinogenic potency associated with the inhalation of B[a]P. A tumorigenic dose (TD₀₅) was calculated to be 1.57 mg/m³ from the animal study by Thyssen et al. (1981) based on respiratory tract tumours and a multi-stage model (Canada 1994).

Benzene is also a component of coal tars and their distillates. Benzene was assessed under CEPA (Environment Canada, Health Canada 1993) and was determined to be a carcinogen. It was therefore added to the *List of Toxic Substances* in Schedule 1 of CEPA 1999. International organizations have drawn similar conclusions. For example, IARC classifies benzene as Group 1 (*carcinogenic to humans*) (IARC 1987a, 2004, 2011). The Government of Canada has previously developed estimates of carcinogenic potency associated with inhalation exposure to benzene. A 5% tumorigenic concentration (TC₀₅) for benzene was calculated to be 14.7 × 10³ µg/m³ (Environment Canada, Health Canada 1993) from the epidemiological investigation of Rinsky et al. (1987) based on acute myelogenous leukemia in Pliofilm workers. The TC₀₅ value is the

air concentration of a substance associated with a 5% increase in incidence or mortality due to tumours (Health Canada 1996). Reference values for benzene from other international agencies (U.S. EPA [2000], WHO [2000]) are similar to the TC₀₅ used below in this screening assessment for the characterization of risk to human health.

Non-cancer health effects endpoints are relevant for consideration for the characterization of potential short-term inhalation exposures to vapour during application of CTPS. Studies on the short-term health effects of PAH exposure are limited. However, a health effects database for naphthalene was available and considered. Naphthalene was previously assessed under CEPA (Environment Canada, Health Canada 2008), and a LOAEC of 7.86 mg/m³ was reported by Phimister et al. (2004) based on point-of-contact nasal olfactory epithelium injury in mice after a 2-hour exposure. This effect was also seen in short-term and subchronic exposure studies in rats that reported LOAECs of 5 and 10 mg/m³, respectively.

10.3 Characterization of risk to human health

Risk from the processing, handling and storage of coal tars and their distillates at a coal tar refinery

Although exposure to total coal tar cannot be quantified with any certainty, quantifying exposures to specific components known to be present in the whole substance can be verified with measured data. High-hazard components are used to determine the exposure hazard of complex mixtures and associated risk. Confidence in the ability to accurately characterize risk from coal tar exposure is significantly higher for its high-hazard components than for coal tar itself.

The estimate of carcinogenic potency for benzene, previously developed by Health Canada (TC₀₅), was used to calculate margins of exposure (MOEs) associated with evaporative emissions from raw coal tar handling/loading and storage of the resulting products at a coal tar refinery. The TC₀₅ value is the concentration of a substance associated with a 5% increase in incidence or mortality due to tumours and has a value of 14.7 × 10³ µg/m³ (Environment Canada Health Canada 1993). The resulting MOEs for this exposure scenario are presented in Table 10-5.

Table 10-5. MOEs for benzene-based modelled data for a coal tar refinery

Model	SCREEN3	AERSCREEN
Benzene inhalation concentration (µg/m ³)	1.45 (1000 m)	1.14 (1000 m)
TC ₀₅ benzene (µg/m ³)	14.7 × 10 ³	14.7 × 10 ³
MOE	10 140	12 890

The MOEs for inhalation of benzene emissions from a coal tar refinery are considered potentially inadequate to address uncertainties related to the health effects and exposure databases. The modelled concentrations of benzene from the facility reached

the equivalent of the background level of 0.88 µg/m³ at a distance of between 1200 m and 1500 m (NAPS 2008).

Releases of PAHs are associated with both the production and the refinement of coal tars. For the purposes of long-term exposure to PAHs, the modelled concentrations at 1000 m were converted into B[a]P equivalents using PEFs developed by Nisbet and LaGoy (1992). The resulting sum of the B[a]P equivalents was 8.07 ng/m³, based on emissions data from NPRI (see Table 10-6).

Table 10-6. Modelled air concentrations of PAH releases for a coal tar refinery

Compound	Annual concentration maximum at 1000 m (µg/m ³)	Potency equivalency factor	Benzo[a]pyrene equivalents (ng/m ³)
Acenaphthene	0.48	0.001	0.48
Acenaphthylene	0.0028	0.001	0.0028
Acethracene	0.043	0.01	0.43
Benzo[a]anthracene	0.0072	0.1	0.72
Benzo[a]pyrene	0.00071	1	0.71
Benzo[b]fluoranthene	0.0050	0.1	0.5
Benzo[g,h,i]perylene	0.0014	0.01	0.014
Benzo[k]fluoranthene	0.0019	0.1	0.19
Dibenzo[a,h]anthracene	0.00028	5	1.4
Fluoranthene	0.066	0.001	0.066
Fluorene	0.074	0.001	0.074
Indeno[1,2,3-c,d]pyrene	0.0015	0.1	0.15
Phenanthrene	0.18	0.001	0.18
Pyrene	0.044	0.001	0.044
Naphthalene	3.11	0.001	3.11
Sum benzo[a]pyrene equivalents			8.07

An estimation of potential exposure to PAHs from a coal tar refinery was determined to be 8.07 ng/m³ B[a]P equivalents at 1000 m, compared to the average Canadian background concentration of 0.14 µg/m³ (NAPS 2012). Using the TD₀₅ for B[a]P, the resulting MOE ($1.57 \text{ mg/m}^3 \div 8.07 \times 10^{-6} \text{ mg/m}^3$) is 195 000. This margin is considered adequate to address the uncertainties in the exposure and health effects databases.

Using AERSCREEN, the concentration of B[a]P was determined to be $5.5 \times 10^{-4} \text{ µg/m}^3$, a difference of 0.16 ng/m³ compared to the SCREEN3 calculation (0.55 ng/m³ versus 0.71 ng/m³). These results indicate that site-specific modelling and parameters will have a minor effect on PAH concentration.

Risk from the production, handling and storage of coal tar at integrated steel mills

The TC₀₅ value of $14.7 \times 10^3 \mu\text{g}/\text{m}^3$ for benzene (Environment Canada, Health Canada 1993) was also used to calculate the MOEs associated with evaporative emissions from the production, handling and storage of coal tar at steel mills. The resulting MOEs for this exposure scenario are presented in Table 10-7.

Table 10-7. MOEs for benzene-based modelled data for steel mills

Benzene inhalation concentration ($\mu\text{g}/\text{m}^3$)	1.11 (1000 m)	0.11 (1000 m)
TC₀₅ benzene ($\mu\text{g}/\text{m}^3$)	14.7×10^3	14.7×10^3
MOE	13 240	133 640

The MOEs for inhalation of benzene emissions from steel mills, using the upper bound, are considered potentially inadequate to address uncertainties related to health effects and exposure databases. The modelled concentrations of benzene from the facility reached the equivalent of the background level of $0.88 \mu\text{g}/\text{m}^3$ at a distance of 1200 m (NAPS 2008). Although the modelling estimates of exposure are considered conservative, there is the potential for increased long-term exposure for those residing in the vicinity of this industry. This is supported by monitoring data, as well the 1-hour concentrations, which are several times higher than the annual averages.

While no emissions of PAHs were attributed to coal tar storage and handling, an estimation of potential exposures from releases of PAHs was determined based on the ratio of benzene to the toxicological equivalents of B[a]P from the coal tar refining scenario (see Table 10-6). The ratio to determine a relative release for steel mills was completed as follows: 4100 kg benzene to $8.07 \text{ ng}/\text{m}^3$ B[a]P equivalents compared to 3132 kg benzene to unknown amount of B[a]P equivalents. The resulting concentration, by proportion, is $6.16 \times 10^{-6} \text{ mg}/\text{m}^3$ B[a]P equivalents or $6.16 \text{ ng}/\text{m}^3$ at 1000 m for the largest amount of releases, assuming comparable conditions of dispersion. This is compared to the average Canadian background concentration of $0.14 \text{ ng}/\text{m}^3$ (NAPS 2012). The low end of the range was determined to be $0.58 \text{ ng}/\text{m}^3$ at 1000 m. The concentration profile for the upper bound reaches this background level beyond the modelled distance of 3000 m. The TD₀₅ for B[a]P, previously developed by Health Canada (Canada 1994), is $1.57 \text{ mg}/\text{m}^3$. The resulting MOE ($1.57 \text{ mg}/\text{m}^3 \div 6.16 \times 10^{-6} \text{ mg}/\text{m}^3$) is 255 000. The margin is considered adequate to address uncertainties related to the health effects and exposure databases.

Risk from the transport of coal tars and their distillates

The transient exposure associated with transporting coal tars and their distillates is minor given the small amount of volatiles released by each mode of transport and the fact that the releases occur continuously from a moving source (a line source) rather than from a stationary point source. Consequently, the actual concentration of the coal tar vapours around a moving line source, for any given location, will be considerably lower than that represented by the production and processing of the material, which

occurs at a stationary site. The transportation of coal tars and their distillates is not considered to be a risk to human health.

Risk from PAHs in house dust associated with use of coal-tar-based pavement sealants

The average concentrations of the 16 individual PAHs found in house dust (Mahler 2010) were converted into B[a]P equivalents using established carcinogenic PEFs proposed by Nisbet and LaGoy (1992). The resulting concentrations were summed to give a B[a]P equivalent value of 20.1 µg/g. Detailed data on the average concentrations used and the potency factor applied can be found in Tables C-1, C-2 and C-3 (Appendix C). Using the 20.1 µg/g concentration, upper-bounding estimates of daily exposure to B[a]P (via house dust originating from coal-tar-based sealcoated parking lots) were derived.

Combined soil and dust ingestion rates adapted from the work by Wilson et al. (2013) are based on the arithmetic means from a probabilistic approach. An exposure estimate was derived based on the ingestion of soil and indoor dust. The following age groups were assumed to ingest the indicated amounts incidentally each day: 0–6 months (assumed to weigh 7.5 kg), 38 mg of soil and dust per day; 0.5–4 years (assumed to weigh 15.5 kg), 55 mg of soil and dust per day; 5–11 years (assumed to weigh 31.0 kg), 52 mg of soil and dust per day; 12–19 years (assumed to weigh 59.4 kg), 3.6 mg of soil and dust per day; 20–59 years (assumed to weigh 70.9 kg), 4.1 mg of soil and dust per day; 60+ years (assumed to weigh 72.0 kg), 4.0 mg of soil and dust per day (Wilson et al. 2013). B[a]P intakes for each age group and average daily dose are presented in Table 10-8.

Table 10-8. Soil and dust intakes and exposure by age group

Age group	0–0.5 yr	0.5–4 yr	5–11 yr	12–19 yr	20–59 yr	60+ yr
Soil and dust intake rates (mg/day)	38	55	52	3.6	4.1	4.0
Body weights (kg)	7.5	15.5	31.0	59.4	70.9	72.0
Intake mass (µg/day)	0.76	1.11	1.05	0.072	0.082	0.080
Dose (µg/kg bw/day)	0.102	0.071	0.033	0.0012	0.0012	0.0011
Time-weighted intake (µg/kg bw/day)	0.00073	0.0036	0.0034	0.00014	0.00066	0.00018
Lifetime average daily dose (µg/kg bw/day)	0.0086					

Concentration B[a]P in dust (µg/mg)	0.0201
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The lifetime average daily dose of coal-tar-based B[a]P to which the population could be exposed was determined from the above intakes and determined to be 0.0086 µg/kg bw/day. Young children are considered to represent a susceptible subpopulation in this particular scenario, based on greater exposure potential associated with their extensive hand-to-mouth action; crawling on floors and surfaces that accumulate dust (Van Metre et al. 2013). Accordingly, the age-dependant adjustment factors⁵ recommended by the U.S. EPA were considered and adjusted to the Health Canada age groups (see Table 10-9). These factors were then applied to the MOEs for each age group (U.S. EPA 2011b).

Table 10-9. Age-dependent adjustment factors and age groups

Life stage	Age range	Adjustment factor
Infant	0–0.5 years	10
Toddler	0.5–4 years	5 ^a
Children	5–11 years	3
Teenager	12–19 years	2 ^b
Adult	20+ years	1

$$^a \text{ADAF}_{0.5 \text{ to } 4 \text{ yr}} = (\text{ADAF}_{0 \text{ to } < 2} \times D_{0.5 \text{ to } 1} / D_{0.5 \text{ to } 4}) + (\text{ADAF}_{2 \text{ to } 4} \times D_{2 \text{ to } 4} / D_{0.5 \text{ to } 4}) \\ = (10 \times 1.5 / 4.5) + (3 \times 3 / 4.5) = 5, D_i = \text{exposure duration (years)}$$

$$^b \text{ADAF}_{12 \text{ to } 19 \text{ yr}} = (\text{ADAF}_{12 \text{ to } < 16} \times D_{12 \text{ to } 15} / D_{12 \text{ to } 19}) + (\text{ADAF}_{16+} \times D_{16 \text{ to } 19} / D_{12 \text{ to } 19}) \\ = (3 \times 4 / 8) + (1 \times 4 / 8) = 2, D_i = \text{exposure duration (years)}$$

The point of departure for determining a MOE for each age group was the lower limit of a one-sided 95% confidence interval (BMDL₁₀) for B[a]P, which was calculated by Health Canada (Health Canada 2014) to be 0.562 mg/kg bw/day.

The lifetime adjusted margin of exposure is calculated as follows:

$$\text{MOE}_{\text{ADJ lifetime}} = 1 / \Sigma[1/\text{MOE} \times \text{averaging time}]$$

$$\text{MOE}_{\text{ADJ lifetime}} = 1 / \Sigma[(1/552 \times 0.7/70) + (1/1576 \times 3.5/70) + (1/5556 \times 7/70) + (1/230672 \times 8/70) + (1/483507 \times 40/70) + (1/503284 \times 11/70)]$$

$$\text{MOE}_{\text{ADJ lifetime}} = 15\,500$$

The resulting MOEs for each age group were then weighted according to their time length; the resulting lifetime adjusted MOE is 15 500. The MOEs associated with ingestion of house dust by children are considered potentially inadequate to protect these susceptible subpopulations.

⁵ Age-dependent adjustment factor (ADAF): In cases where age-related differences in toxicity occur, differences in both toxicity and exposure need to be integrated across all relevant age intervals by the use of age-dependent potency adjustment factors (ADAFs). This is a departure from the way cancer risks have historically been calculated based on the premise that risk is proportional to the daily average of the long-term adult dose.

A large, one-time ingestion of soil contaminated with CTPS associated PAHs is not considered to be of risk to human health. A toddler's exposure was considered for this event, using a body weight of 15.5 kg and the concentration of B[a]P equivalents used in the house dust exposure scenario (20.1 µg B[a]P/g soil). A mass of 1 gram of soil was found to yield a dose of 1.30 µg B[a]P/kg bw. Very few acute duration studies for PAHs are available, as the focus for PAHs is primarily on chronic cancer research. However, several acute-duration (1–3 day exposure) cancer studies were identified, and these doses resulted in forestomach, liver, lung and mammary tumours in rodents (Klein 1963; Neal and Rigdon 1967; McCormick et al. 1981). However, the doses required to produce such tumours (approximately 35 to 100 mg/kg bw/day) are much higher than expected environmental concentrations.

Risk from inhalation exposure to coal-tar-based pavement sealant application

Exposure to volatiles from the application of CTPS was investigated (see Table 10-7). Given the periodic nature of inhalation exposure for individuals sealcoating their driveway (once per year is conservatively assumed, though the interval between sealcoatings is likely to be 2 to 3 years), along with the short duration of exposure (expected to be 2 to 3 hours), the potential for non-cancer effects was considered. Analysis of available studies in the health effects database for acute and short-term exposure to PAHs was inadequate to derive a margin of exposure. However, the few available studies indicate that short-term health effects are limited, localized and generally reversible (e.g., nasal epithelium damage in mice; Phimister et al. [2004]). Longer-term repeated exposure studies in rats support this notion.

Risk from dermal exposure resulting from coal-tar-based pavement sealants

Dermal exposure from the application of a driveway CTPS was estimated using a “thin film” approach (U.S. EPA 2011b). The exposure dose for an average Canadian from this use was determined to be low (2.1 mg/kg bw), and this exposure event is considered to be very infrequent (conservatively once every year, but likely less than this). Given the infrequent nature of the exposure and the limited duration for which it is expected to occur, the conclusion is that incidental dermal exposure to CTPS does not constitute a human health concern.

10.4 Uncertainties in assessing risk to human health

Because coal tars and their distillates are UVCBs, their specific composition is only broadly defined and can vary depending on the producer and specifications. The proportion of component compounds can vary depending on the operating conditions, feedstocks and processing units.

Additional uncertainty arises from the fact that the Screen 3 modelling for the coal tar refinery and integrated steel mills used an area source release as opposed to stack

release. The area use may result in an underestimation if a majority of releases come from a stack.

The screening assessments are based on consideration of the available data and include various conservative exposure scenarios that account for both the general population and vulnerable populations in Canada. There is limited information available on the application of pavement sealants at schools, and this potential exposure is acknowledged as an uncertainty in the assessment.

Additional substances that are potentially present in coal tars, including alkylated PAH and those containing heteroatoms—atoms other than carbon or hydrogen in an organic molecule—have not been characterized due to limited detailed information on coal tar substance composition, exposure monitoring and hazard properties. The margin of exposure is based on the compositional analysis of a suite of 16 PAHs. Recent data analyzing a larger suite of 60 PAHs reveals a higher concentration and proportion of potentially harmful PAHs to which the general population may be exposed, which would further reduce the margin of exposure.

Although the use of PEFs is a common method for determining the hazard potential of a complex mixture, there is uncertainty regarding how well this approach estimates the true hazard of the mixture.

There are many different PEFs that have been proposed for the 16 PAHs analyzed in this assessment, as well as for many additional alkylated PAHs for which exposure data were not available. The selection of the suite of PAHs to include and their PEFs has the potential to either overestimate or underestimate risk.

There is conservative, and therefore upper-bounding estimates of PAHs ingested given the uncertainty in the PAH concentration differences between dust and soil. However, these concentrations were assumed to be equal in the exposure estimations.

11. Conclusion

Considering all available lines of evidence presented in this screening assessment, there is risk of harm to the environment from coal tars and their distillates. It is concluded that coal tars and their distillates, including the six substances in Table 2-1, meet the criteria under paragraph 64(a) of CEPA as they are entering or may enter 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. However, it is concluded that coal tars and their distillates do not meet the criteria under paragraph 64(b) of CEPA as they are not entering the environment in a quantity or concentration or under conditions that constitute or may constitute a danger to the environment on which life depends.

PAHs and benzene are regarded as high-hazard components present in coal tar substances. There may be exposure to these high-hazard volatile constituents of coal tars and their distillates for the general population living in the vicinity of coal tar producers and refineries. The margins of exposure between estimates of exposure to benzene and estimates of cancer potency previously developed for inhalation exposure to benzene are considered potentially inadequate to address uncertainties related to health effects and exposure estimates. As well, the margins of exposure for the ingestion of house dust containing PAHs associated with the use of coal-tar-based sealants and estimates of cancer potency are considered potentially inadequate to address uncertainties related to health effects and exposure databases. Accordingly, it is proposed to conclude that coal tars and their distillates, including the six substances in Table 2-1, meet the criteria under paragraph 64(c) of CEPA 1999, as they are entering or may enter the environment in a quantity or concentration or under conditions that constitute or may constitute a danger in Canada to human life or health.

It is therefore concluded that all coal tars and their distillates meet one or more of the criteria set out in section 64 of CEPA.

References

AERMOD [Computer Model]. 2009. Version 09292. Research Triangle Park (NC): U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards, Emissions, Monitoring, and Analysis Division.

Agurell E, Stensman C. 1992. Salmonella mutagenicity of three complex mixtures assayed with the microsuspension technique. A WHO/IPCS/CSCM study. *Mutat Res.* 276(1-2):87-91. [cited in ATSDR 2002].

Alexander M. 2000. Aging, bioavailability, and overestimation of risk from environmental pollutants. *Environ Sci Technol.* 34(20):4259-4265.

[AMAP] Arctic Monitoring and Assessment Programme. 2004. AMAP Assessment 2002: Persistent Organic Pollutants in the Arctic [PDF]. Oslo, Norway; xvi + 310 pp.

Ankley GT, Burkhard LP, Cook PM, Diamond SA, Erickson RJ, Mount DR. 2003. Assessing risks from photoactivated toxicity of PAHs to aquatic organisms. In: Douben PET, editor. *PAHs: an ecotoxicological perspective*. Hoboken (NJ): Wiley. p. 277-296.

ArcelorMittal. 2015. Chemical Product and Company Identification Information; Coal Tar [PDF]. [MSDS]. Chicago (IL): ArcelorMittal USA Inc.

Arey J, Atkinson R. 2003. Photochemical reactions of PAHs in the atmosphere. In: Douben PET, editor. *PAHs: an ecotoxicological perspective*. Hoboken (NJ): Wiley. p. 47-63.

Arnot JA, Gobas FAPC. 2003. A generic QSAR for assessing the bioaccumulation potential of organic chemicals in aquatic food webs. *QSAR Comb Sci.* 22(3):337-345.

[ASTM] American Society for Testing and Materials. 2011. ASTM D490-92. Standard specification for road tar. West Conshohocken (PA): ASTM International.

[ATSDR] Agency for Toxic Substances and Disease Registry. 1995. Toxicological profile for polycyclic aromatic hydrocarbons. Atlanta (GA): Agency for Toxic Substances and Disease Registry, Division of Toxicology, Toxicology Information Branch.

[ATSDR] Agency for Toxic Substances and Disease Registry. 2002. Toxicological profile for wood creosote, coal tar creosote, coal tar, coal tar pitch, and coal tar pitch volatiles. Atlanta (GA): Agency for Toxic Substances and Disease Registry, Division of Toxicology, Toxicology Information Branch.

Babu TS, Marder JB, Tripuranthakam S, Dixon DG, Greenberg BM. 2001. Synergistic effects of a photooxidized polycyclic aromatic hydrocarbon and copper on photosynthesis and plant growth: evidence that *in vivo* formation of reactive oxygen species is a mechanism of copper toxicity. *Environ Toxicol Chem.* 20(6):1351-1358.

Backhaus T, Faust M. 2012. Predictive environmental risk assessment of chemical mixtures: a conceptual framework. *Environ Sci Technol*. 46(5):2564-2573.

Baes CF, Sharp RD. 1983. A proposal for estimation of soil leaching and leaching constants for use in assessment models. *J Environ Qual*. 12(1):17-28.

Banerjee S. 1984. Solubility of organic mixtures in water. *Environ Sci Technol*. 18(8):587-591.

Barański B, Palus J, Rogaczewska T, Szymczak W, Spiechowicz E. 1992. Correlation between polycyclic aromatic hydrocarbons concentration and airborne particle mutagenicity in the rubber factory. *Pol J Occup Med Environ Health*. 5(4):357-362. [cited in ATSDR 2002].

[BCFBAF] [Bioaccumulation Program for Microsoft Windows \[estimation model\]](#). 2010. Ver. 3.01. Washington (DC): US Environmental Protection Agency, Office of Pollution Prevention and Toxics; Syracuse (NY): Syracuse Research Corporation.

Becker S, Halsall CJ, Wlodek T, Hung H, Attewell S, Blanchard P, Li H, Fellin P, Stern G, Billeck B, Friesen S. 2006. Resolving the long-term trends of polycyclic aromatic hydrocarbons in the Canadian Arctic atmosphere. *Environ Sci Technol*. 40(10):3217-3222.

Beland FA, Churchwell MI, von Tungeln LS, Chen S, Fu PP, Culp SJ, Schoket B, Gyorffy E, Minarovits J, Poirier MC, Bowman ED, Weston A, Doerge DR. 2005. High-performance liquid chromatography electrospray ionization tandem mass spectrometry for the detection and quantitation of benzo[a]pyrene-DNA adducts. [Abstract]. *Chem Res Toxicol*. 18(8):1306-1315. [cited in PubMed 1996].

Betts WD. 2000. Tar and pitch. In: Kirk-Othmer encyclopedia of chemical technology. Hoboken (NY): John Wiley and Sons, Inc. 31 pp.

Bickers DR, Kappas A. 1978. Human skin aryl hydrocarbon hydroxylase. Induction by coal tar. *J Clin Invest*. 62(5):1061-1068.

Bickers DR, Wroblewski D, Tapu-Dutta-Choudhury, Mukhtar H. 1982. Induction of neonatal rat skin and liver aryl hydrocarbon hydroxylase by coal tar and its constituents. *J Invest Dermatol*. 78(3):227-229.

Black JA, Birge WJ, Westerman AG, Francis PC. 1983. Comparative aquatic toxicology of aromatic hydrocarbons. *Fundam Appl Toxicol*. 3:353-358.

Blaylock BG, Frank ML, McCarthy JF. 1985. Comparative toxicity of copper and acridine to fish, *Daphnia* and algae. *Environ Toxicol Chem*. 4:63-71.

Bleeker EAJ, van der Geest, Kraak MHS, de Voogt P, Admiraal W. 1998. Comparative ecotoxicity of NPAHs to larvae of the midge *Chironomus riparius*. *Aquat Toxicol.* 41:51-62.

Bleeker EAJ, Wiegman S, de Voogt P, Kraak M, Leslie HA, de Haas E, Admiraal W. 2002. Toxicity of azaarenes. *Rev Environ Contam Toxicol.* 173:39-83.

Blümer GP, Sutton M. 1998. Tar – only a by-product of the coke plant? *Cokemaking International.* 10(1):55-60.

Bommarito T, Sparling DW, Halbrook RS. 2010a. Toxicity of coal-tar and asphalt sealants to eastern newts, *Notophthalmus viridescens*. *Chemosphere.* 81:187-193.

Bommarito T, Sparling DW, Halbrook RS. 2010b. Toxicity of coal-tar pavement sealants and ultraviolet radiation to *Ambystoma maculatum*. *Ecotoxicology.* 19:1147-1156.

Bordelon NR, Donnelly KC, King LC, Wolf DC, Reeves WR, George SE. 2000. Bioavailability of the Genotoxic Components in Coal Tar Contaminated Soils in Fischer 344 Rats. *Toxicol Sci.* 56(1):37-48.

Bos RP, Jongeneelen FJ, Theuws JLG, Henderson PT. 1985. Detection of volatile mutagens in creosote and coal tar. *Mutat Res.* 156(3):195-198. [cited in ATSDR 2002].

Bos RP, Prinsen WJ, van Rooy JG, Jongeneelen FJ, Theuws JL, Henderson PT. 1987. Fluoranthene, a volatile mutagenic compound, present in creosote and coal tar. *Mutat Res.* 187(3):119-125. [cited in ATSDR 2002; abstract cited in PubMed 1996].

Brandon JL, Conti CJ, Goldstein LS, DiGiovanni J, Gimenez-Conti IB. 2009. Carcinogenic effects of MGP-7 and B[a]P on the hamster cheek pouch. *Toxicol Pathol.* 37(6):733-740.

Brooke LT. 1991. Results of freshwater exposures with the chemicals atrazine, biphenyl, butachlor, carbaryl, carbazole, dibenzofuran, 3,3'-dichlorobenzidine, diclorovos, 1,2-epoxyethylbenzene (styrene oxide), isophorone, isopropalin, oxychlordane, pentachloroanisole, propoxur (baygon), tetrabromobisphenol A, 1,2,4,5-tetrachlorobenzene, and 1,2,3-trichloropropane to selected freshwater organisms. University of Wisconsin, Superior (WI): Center for Lake Superior Environmental Studies, 110 pp. [cited in ECOTOX 2006].

Bryer PJ, Elliott JN, Willingham EJ. 2006. The effects of coal tar based pavement sealer on amphibian development and metamorphosis. *Ecotoxicology.* 15:241-247.

Bryer PJ, Scoggins M, McClintock NL. 2010. Coal-tar based pavement sealant toxicity to freshwater macroinvertebrates. *Environ Pollut.* 158:1932-1937.

Burgess RM, Ahrens MJ, Hickey CW. 2003. Geochemistry of PAHs in aquatic environments: source, persistence and distribution. In: Douben PET, editor. PAHs: an ecotoxicological perspective. Hoboken (NJ): Wiley. p. 35-45.

Calamari D, Vighi M. 1992. A proposal to define quality objectives for aquatic life for mixtures of chemical substances. *Chemosphere*. 25(4):531-542.

Canada. 1999. [Canadian Environmental Protection Act, 1999](#). S.C. 1999, c. 33, Canada Gazette Part III, vol. 22, no. 3.

Canada. 2003. [Natural Health Products Regulations](#). SOR/2003-196. Government of Canada. Ottawa (ON).

Canada, Dept. of the Environment. 2012. [Canadian Environmental Protection Act, 1999: Notice with respect to certain substances on the Domestic Substances List \[PDF\]](#). Canada Gazette, Part I, vol. 146, no. 48. Supplement.

Casto BC, Hatch GG, Huang SL, Lewtas J, Nesnow S, Waters MD. 1981. Mutagenic and carcinogenic potency of extracts of diesel and related environmental emissions: *In vitro* mutagenesis and oncogenic transformation. *Environ Int*. 5(4-6):403-409. [cited in IARC 1985]

[CCME] Canadian Council of Ministers of the Environment. 1991. Interim Canadian environmental quality criteria for contaminated sites. Winnipeg (MB): CCME. [cited in CCME 2010].

[CCME] Canadian Council of Ministers of the Environment. 1995. [Protocol for the derivation of Canadian sediment quality guidelines for the protection of aquatic life \[PDF\]](#). Winnipeg (MB): CCME.

[CCME] Canadian Council of Ministers of the Environment. 1997. Recommended Canadian soil quality guidelines. Winnipeg (MB): CCME. [cited in CCME 2010].

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

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

[CCME] Canadian Council of Ministers of the Environment. 2008. Canadian soil quality guidelines for carcinogenic and other polycyclic aromatic hydrocarbons (environmental and human health effects). Scientific Supporting Document. Winnipeg (MB): CCME.

[CCME] Canadian Council of Ministers of the Environment. 2010. Canadian soil quality guidelines: carcinogenic and other polycyclic aromatic hydrocarbons (PAHs) (environmental and human health effects) [PDF]. Scientific criteria document (revised). Winnipeg (MB): CCME.

[CCME] Canadian Council of Ministers of the Environment. 2014. Canadian environmental quality guidelines summary table. Winnipeg (MB): CCME.

[CFR] Code of Federal Regulations. 2013. Title 21, Volume 5, Food and Drugs, Part 358 – Miscellaneous External Drug Products for Over-the-Counter Human Use. Subpart H. Washington (DC). United States Food and Drug Administration.

[CIR] Cosmetic Ingredient Review. 2008. Final Safety Assessment of Coal Tar as Used in Cosmetics. *Int J Toxicol*. 27(Supplement 2):1-24.

City of Hamilton. 2013. Proposed New Sewer Use By-Law (PW13061) – (City Wide), Public Works Department, Hamilton Water Division, September 3, 2013.

Cizmas L, Zhou G, Safe SH, McDonald TJ, Zhu L, Donnelly KC. 2004. Comparative in vitro and in vivo genotoxicities of 7H-benzo[*c*]fluorene, manufactured gas plant residue (MGP), and MGP fractions. *Environ Mol Mutagen*. 43(3):159-168.

Claxton L, Huisingsh JL. 1980. Comparative mutagenic activity of organics from combustion sources. In: *Pulmonary Toxicology of Respirable Particles*. Sander CL, Cross FT, Dagle GE, Mahaffey JA, eds. DOE Symposium Series, Vol 53. Washington, DC: Technical Information Center, Department of Energy, p. 453-465. [cited in IARC 1985].

Clonfero E, Zordan M, Cottica D, Venier P, Pozzoli L, Cardin EL, Sarto F, Levis AG. 1986. Mutagenic activity and polycyclic aromatic hydrocarbon levels in urine of humans exposed to therapeutic coal tar. *Carcinogenesis*. 7(5):819-823. [cited in CIR 2008].

Cloverdale Paint. 2013. ClovaTar 22 – Coal Tar Epoxy. Product Information (TDS and SDS).

Collins JF, Brown JP, Alexeeff GV, Salmon AG. 1998. Potency equivalency factors for some polycyclic aromatic hydrocarbons and polycyclic aromatic hydrocarbon derivatives. *Regul Toxicol and Pharmacol*. 28(1):45-54. [cited in CCME 2008].

[Conestoga] Conestoga Roofing and Sheet Metal Ltd. 2014. Flat Roofing Systems. [accessed 2014 May 15].

[CRCA] Canadian Roofing Contractors' Association. ca. 2001. 2000–2001 Annual Market Survey.

Culp SJ, Beland FA. 1994. Comparison of DNA adduct formation in mice fed coal tar or benzo[a]pyrene. *Carcinogenesis*. 15(2):247-252. [cited in ATSDR 2002].

Culp SJ, Gaylor DW, Sheldon WG, Goldstein LS, Beland FA. 1996. DNA adduct measurements in relation to small intestine and forestomach tumor incidence during the chronic feeding of coal tar or benzo[a]pyrene to mice. *Polycycl Aromat Compd*. 11(1):161-168. [cited in ATSDR 2002].

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(1):117-124.

Culp SJ, Warbritton AR, Smith BA, Li EE, Beland FA. 2000. DNA adduct measurements, cell proliferation and tumor mutation induction in relation to tumor formation in B6C3F1 mice fed coal tar or benzo[a]pyrene. *Carcinogenesis*. 21(7):1433-1440.

Curren RD, Kouri RE, Kim CM, Schechtman LM. 1981. Mutagenic and carcinogenic potency of extracts from diesel related environmental emissions: Simultaneous morphological transformation and mutagenesis in BALB/c 3T3 cells. *Environ Int*. 5(4-6):411-415. [cited in IARC 1985].

Curry P, Kramer G, Newhook R, Sitwell J, Somers D, Tracy B, Oostdam JV. 1993. Reference values for Canadian populations. Ottawa (ON): Environmental Health Directorate Working Group on Reference Values, Health Canada.

Den Besten PJ, Ten Hulscher D, Van Hattum B. 2003. Bioavailability, uptake and effects of PAHs in aquatic invertebrates in field studies. In: Douben PET, editor. PAHs: an ecotoxicological perspective. Hoboken (NJ): Wiley. p. 127-146.

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

Dijkman NA, van Vlaardingen PLA, Admiraal WA. 1997. Biological variation in sensitivity to N-heterocyclic PAHs: effects of acridine on seven species of micro-algae. *Environ Pollut*. 95(1):121-126.

Di Toro DM, McGrath JA, Hansen DJ. 2000. Technical basis for narcotic chemicals and polycyclic aromatic hydrocarbon criteria. I. Water and tissue. *Environ Toxicol Chem*. 19(8):1951-1970.

Di Toro DM, McGrath JA. 2000. Technical basis for narcotic chemicals and polycyclic aromatic hydrocarbon criteria. II. Mixtures and sediments. *Environ Toxicol Chem*. 19:1971-1982. [cited in DiToro et al. 2000].

Di Toro DM, McGrath JA, Stubblefield WA. 2007. Predicting the toxicity of neat and weathered crude oil: toxic potential and the toxicity of saturated mixtures. *Environ Toxicol Chem.* 26:24-36.

Donnelly KC, Brown KW, Markiewicz KV, Anderson CS, Manek DJ, Thomas JC, Giam CS. 1993. The use of short-term bioassays to evaluate the health and environmental risk posed by an abandoned coal gasification site. *Haz Waste Haz Mater.* 10(1):59-70. [cited in ATSDR 2002].

Donnelly KC, Phillips TD, Onufrock AM, Collie SL, Huebner HJ, Washburn KS. 1996. Genotoxicity of model and complex mixtures of polycyclic aromatic hydrocarbons. In: Bengtson DA, Henshel DS, eds. *Environmental toxicology and risk assessment: Biomarkers and risk assessment.* ASTM STP. Conshohocken, PA: ASTM. p. 138-148. [cited in ATSDR 2002].

[DPD] Drug Product Database [database]. 2014. Ottawa (ON): Health Canada. [cited 2014 June].

Douben PET, editor. 2003. *PAHs: an ecotoxicological perspective.* Hoboken (NJ): Wiley.

Canada, Dept. of the Environment. 2018. Notice to provide information for the risk management of certain coal tars and their distillates [PDF]. *Canada Gazette, Part 1*, vol. 152, no. 48. p. 4089-4103.

[ECCC] Environment and Climate Change Canada. 2018. Supporting documentation: ecological exposure assessment of coal tar pitch-based pavement sealant (CTPS) to the aquatic and sediment environment. Gatineau (QC): ECCC. Information in support of the screening assessment for coal tars and their distillates. Available from: eccc.substances.eccc@canada.ca.

[ECCC] Environment and Climate Change Canada. 2019. Data for certain coal tars and their distillates collected under the *Canadian Environmental Protection Act, 1999, section 71: Notice to provide information for the risk management of certain coal tars and their distillates*. Data prepared by Environment and Climate Change Canada, Health Canada; Existing Substances Program.

[ECCC, HC] Environment and Climate Change Canada, Health Canada. [modified 2017 Mar 12]. Categorization of chemical substances. Ottawa (ON): Government of Canada.

[ECHA] European Chemicals Agency. 2012. Guidance on information requirements and chemical safety assessment. Chapter R.16: Environmental exposure estimation. Version 2.1, Oct. 2012. Helsinki, Finland.

[ECHA] European Chemicals Agency. 2013. Information on Chemicals – Registered Substances [database].

[ECHA] European Chemicals Agency. c2007-2017a. Registered substances database [database]; Tar, coal, high-temp; CAS RN 65996-89-6. Helsinki (FI): ECHA. [updated 2017 Jul 28; accessed 2017 Aug 1].

[ECHA] European Chemicals Agency. c2007-2017b. Registered substances database [database]; Distillates (coal tar), light oils; CAS RN 84650-03-3. Helsinki (FI): ECHA. [updated 2017 Jul 28; accessed 2017 Aug 1].

[ECHA] European Chemicals Agency. c2007-2017c. Registered substances database [database]; Distillates (coal tar), upper; CAS RN 65996-91-0. Helsinki (FI): ECHA. [updated 2017 Jul 28; accessed 2017 Aug 1].

[ECHA] European Chemicals Agency. c2007-2017d. Registered substances database [database]; Pitch, coal tar, high-temp; CAS RN 65996-93-2. Helsinki (FI): ECHA. [updated 2017 Jul 28; accessed 2017 Aug 1].

[ECHA] European Chemicals Agency. c2007-2017e. Registered substances database [database]; Anthracene oil; CAS RN 90640-80-5. Helsinki (FI): ECHA. [updated 2017 Jul 28; accessed 2017 Aug 1].

[ECOTOX] [ECOTOXicology database \[database\]](#). 2006. Version 4. Washington (DC): US Environmental Protection Agency, Office of Research and Development; National Health and Environmental Effects Research Laboratory, Mid-Continent Ecology Division.

[EHS] Environmental Health Strategies. 2010. Technical and economic study of VOC emissions from coal tar-based pavement sealants. Final report. Prepared for Products Division, Chemical Sectors, Environmental Stewardship Branch, Environment Canada. Toronto (ON): Environmental Health Strategies Inc.

[EMA] Environmental Management Agreement. 1997. Environmental Management Agreement among Dofasco Inc. (“Dofasco”); Her Majesty the Queen in right of Canada, as represented by the Minister of the Environment (“EC”); and the Ministry of Environment, Province of Ontario (“MOE”). 14 pp.

[EMA] Environmental Management Agreement. 2000. Environmental Management Agreement between Algoma Steel Inc. and Her Majesty the Queen in Right of Canada, as represented by the Minister of the Environment (EC), and Her Majesty the Queen in Right of Ontario, as represented by the Minister of the Environment (MOE).

Emmett EA, Bingham EM, Barkley W. 1981. A carcinogenic bioassay of certain roofing materials. *Am J Ind Med.* 2(1):59-64.

[ENTOX] Encyclopedia of Toxicology. 2005. 2nd ed. Elsevier, Oxford, UK. p. 628-631.

Environment Canada. 2001. Environmental Code of Practice for Integrated Steel Mills – CEPA 1999 Code of Practice [PDF]. 1st edition. Environmental protection series: EPS 1/MM/7. Ottawa (ON): Minister of Public Works and Government Services Canada.

Environment Canada. 2013a. DSL Inventory Update data collected under the *Canadian Environmental Protection Act, 1999, section 71: Notice with respect to certain substances on the Domestic Substances List*. Data prepared by: Environment Canada, Health Canada; Existing Substances Program.

Environment Canada. 2013b. [ERRIS] Effluent Regulatory Reporting Information System [database]. [accessed 2013 Oct].

Environment Canada. 2015a. Supporting documentation: identity, compositional information, physical and chemical properties, environmental persistence data and aquatic bioaccumulation data for coal tar and its distillates. Gatineau (QC): Environment Canada. Information in support of the screening assessment for coal tars and their distillates. Available from: eccc.substances.eccc@canada.ca.

Environment Canada. 2015b. Supporting documentation: summary of empirical aquatic and terrestrial toxicity data for representative structures of coal tars and their distillation products. Gatineau (QC): Environment Canada. Information in support of the screening assessment for coal tars and their distillates. Available from: eccc.substances.eccc@canada.ca.

Environment Canada, Health Canada. 1994. Canadian Environmental Protection Act: Priority Substance List report: polycyclic aromatic hydrocarbons. Ottawa (ON): Environment Canada, Health Canada.

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. 2011. Screening assessment: Quinoline. Chemical Abstracts Service Registry Number 91-22-5. Ottawa (ON): Government of Canada.

[EPI Suite] Estimation Program Interface Suite for Microsoft Windows [estimation model]. 2000–2010. Version 4.1. Washington (DC): U.S. Environmental Protection Agency, Office of Pollution Prevention and Toxics; Syracuse (NY): Syracuse Research Corporation.

Erickson DC, Loehr RC, Neuhauser EF. 1993. PAH loss during bioremediation of manufactured gas plant site soils. *Water Res.* 27(5):911-919.

[ESIS] European Chemical Substances Information System [database]. c1995-2011. Database developed by the European Chemicals Bureau (ECB).

European Commission. 1976. Council Directive of 27 July 1976 on the approximation of the laws, regulations and administrative provisions of the Member States relating to restrictions on the marketing and use of certain dangerous substances and preparations [PDF]. 76/769/EEC. Official Journal L 262, 27.9.1976, p. 201.

European Commission. 1994. Commission Directive 94/69/EC of 19 December 1994. Annex II. Official Journal of the European Communities. 31.12.94. L 381, Vol. 37. European Commission, 21st ATP.

European Commission. 2001. Ambient Air Pollution by Polycyclic Aromatic Hydrocarbons (PAH) [PDF]. Position Paper. Office for Official Publications of the European Communities: Luxembourg.

European Commission. 2003. IUCLID Dataset 1-naphthol, CAS No. 90-15-3. Ispra (IT): European Commission, Joint Research Centre, Institute for Health and Consumer Protection, European Chemicals Bureau.

European Commission. 2008. 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 (NL): Office for Official Publications of the European Communities. [cited 2014].

Food and Agriculture Organization of the United Nations. 1987. FAO Forestry Paper 41, Simple technologies for charcoal making. Rome (IT): Food and Agriculture Organization of the United Nations.

[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 [PDF]. WHO Technical Report Series: 930. Geneva: World Health Organization. [cited 2014].

Fysh J M, Andrews LS, Pohl LR, Nebert DW. 1980. Differing degrees of coal-tar shampoo-induced mutagenesis in the Salmonella/liver test system in vitro. Pharmacology. 20(1):1-8. [cited in CIR 2008; IARC 1985].

Genevois C, Brandt HCA, Bartsch H, Obrecht-pflumio S, Wild CP, Castegnaro M. 1996. Formation of DNA adducts in skin, lung and lymphocytes after skin painting of rats with undiluted bitumen of coal-tar fume condensates. Polycycl Aromat Compd. 8(2-3):75-92. [cited in ATSDR 2002].

Gobas F. 2007. Development and review of a generic water–sediment modeling framework for organic chemicals. Report prepared for Environment Canada. Burnaby (BC): Simon Fraser University, Faculty of Environment. March 26, 2007.

Gobas F. 2010. Comments on approach to sediment exposure approach. Report prepared for Environment Canada. Burnaby (BC): Simon Fraser University, Faculty of Environment. March 25, 2010.

Godschalk RWL, Ostertag JU, Moonen EJ, Neumann HA, Kleinjans JC, van Schooten FJ. 1998. Aromatic DNA adducts in human white blood cells and skin after dermal application of coal tar. *Cancer Epidemiol Biomarkers Prev.* 7(9):767-773. [cited in ATSDR 2002].

Godschalk RWL, Ostertag JU, Zandsteeg AM, van Agen B, Neuman HA, van Straaten H, van Schooten FJ. 2001. Impact of GSTM1 on aromatic-DNA adducts and P53 accumulation in human skin and lymphocytes. *Pharmacogenetics.* 11(6):537-543. [cited in ATSDR 2002].

Ghosh U, Talley JW, Luthy RG. 2001. Particle-scale investigation of PAH desorption kinetics and thermodynamics from sediment. *Environ Sci Technol.* 35:3468-3475.

Goldstein LS, Weyand EH, Safe S, Steinberg M, Culp SJ, Gaylor DW, Beland FA, Rodriguez LV. 1998. Tumors and DNA adducts in mice exposed to benzo[a]pyrene and coal tars: Implications for risk assessment. *Environ Health Perspect Suppl.* 106(6):1325-1330. [cited in ATSDR 2002].

Goosey E, Diamond M, Zhu J, Feng Y-L, Csiszar S. 2011. Reconnaissance study of sealcoat application in Toronto, Canada and an estimate of related PAH emissions. Poster presented at the 32nd annual meeting of the Society of Environmental Toxicology and Chemistry North America held November 13-17, 2011, Boston MA.

Gorski T. 1959. Experimental investigations on the carcinogenic properties of some pitches and tars produced from Silesian pit coal. *Medycyna Pracy.* 10:309-317. [cited in European Commission 2008; IARC 1985].

Goyette D, Boyd J. 1989. The relationship between polycyclic aromatic hydrocarbon (PAH) concentrations in sediment and the prevalence of liver lesions in English sole (*Parophrys vetulus*) from Vancouver Harbour 1985/86 and 1987. Vancouver (BC): Environment Canada, Conservation and Protection. [cited in CCME 1999b].

Graham R, Hester HR, Barkey W. 1940. Coal-tar-pitch poisoning in pigs. *J Am Vet Med Assoc.* 96:135-149. [cited in European Commission 2008].

Gray RH. 1984. Health and environmental effects of coal-liquefaction processes: Environmentally acceptable technology development. *Energy.* 9(4):315-322.

Hackett PL, Rommereim DN, Sikov MR. 1984. Developmental toxicity following oral administration of a high-boiling coal liquid to pregnant rats. *J Appl Toxicol.* 4(1):57-62.

[HAMN 2011] Hamilton Air Monitoring Network. 2011. HAMN Annual Air Quality Reports. Hamilton (ON): Hamilton Air Monitoring Network.

[HAMN 2012] Hamilton Air Monitoring Network. 2012. Specific Air Pollutants Reporting. Polyaromatic Hydrocarbons and Volatile Organic Compounds Monitoring Station Reports. Hamilton (ON): Hamilton Air Monitoring Network.

[HAMN 2017] Hamilton Air Monitoring Network. 2017. Specific Air Pollutants Reporting. Polyaromatic Hydrocarbons and Volatile Organic Compounds Monitoring Station Reports. Hamilton (ON): Hamilton Air Monitoring Network.

Harris KA, Nichol LM, Ross PS. 2011. Hydrocarbon concentrations and patterns in free-ranging sea otters (*Enhydra lutris*) from British Columbia, Canada. *Environ Toxicol Chem.* 30(10):2184-2193.

Health Canada. 1988. Guidelines for Canadian Drinking Quality: Benzo[a]pyrene. Ottawa (ON): Health Canada, Federal-Provincial-Territorial Committee on Drinking Water. Guidelines are under revision and will be updated in 2014 after public consultations.

Health Canada. 1994. Human Health Risk Assessment for Priority Substances. Ottawa (ON): Health Canada, Environmental Health Directorate.

Health Canada. 1996. Health-based tolerable daily intakes/concentrations and tumorigenic doses / concentrations for priority substances [PDF]. Cat. H46-2/96-194E. Ottawa (ON): Minister of Supply and Services Canada.

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

Health Canada. 2006. Anti-Dandruff Products monograph. Natural Health Products Ingredients Database [database]. Ottawa (ON): Health Canada. [cited 2014 June 27].

Health Canada. 2008. Guidance Document: Classification of Products at the Cosmetic-Drug Interface. [accessed 2013 Oct 16].

Health Canada. 2012. Guidelines for Canadian drinking water quality. Ottawa (ON): Health Canada, Federal-Provincial-Territorial Committee on Drinking Water. [Updated 2017]

Health Canada. 2013. List of Permitted Food Additives. Ottawa (ON): Health Canada. [cited 2014 June].

Health Canada. 2014. Supporting document: Tables of modelling parameters and resulting dispersion concentrations and distances. Derivation of an Oral BMDL₁₀ for Benzo[a]pyrene Ottawa (ON): Health Canada. Available from: substances@ec.gc.ca.

Heinrich U, Pott F, Roller M. 1994a. Estimation of a lifetime unit lung cancer risk for benzo[a]pyrene (BAP) based on tumor rates in rats exposed to coal tar/pitch condensation aerosol (CTP). *Zentral Hyg Umwelt*. 195:155-156. [cited in ATSDR 2002].

Heinrich U, Dungworth DL, Pott F, Peters L, Dasenbrock C, Levsen K, Koch W, Creutzenberg O, Schulte A. 1994b. The carcinogenic effects of carbon black particles and tar-pitch condensation aerosol after inhalation exposure of rats. *Ann Occup Hyg*. 38 (Suppl. 1):351-356. [cited in ATSDR 2002].

Himpel S, Bartels J, Zimdars K, Huether G, Adler L, Dawirs RR, Moll GH. 2006. Association between body weight of newborn rats and density of serotonin transporters in the frontal cortex at adulthood. *J Neural Transm*. 113(3):295-302.

Horton AW, Tye R, Stemmer KL. 1963. Experimental carcinogenesis of the lung. Inhalation of gaseous formaldehyde or an aerosol of coal tar by C3H mice. *J Natl Cancer Inst*. 30:31-43. [cited in TOXLINE 2009; IARC 1985; Chang et al. 1992].

Hueper WC, Payne WW. 1960. Carcinogenic studies on petroleum asphalt, cooling oil, and coal tar. *Arch Pathol*. 70:372-384. [cited in IARC 1985].

Hughes NC, Pfau W, Hewer A, Jacob J, Grimmer G, Phillips DH. 1993. Covalent binding of polycyclic aromatic hydrocarbon components of coal tar to DNA in mouse skin. *Carcinogenesis*. 14(1):135-144.

Hyun S, Park H, Ahn M-Y, Zimmerman AR, Jafvert CT. 2010. Fluxes of PAHs from coal tar-impacted river sediment under variable seepage rates. *Chemosphere*. 80:1261-1267.

[IARC] International Agency for Research on Cancer. 1985. Monographs on the evaluation of the carcinogenic risk of chemicals to humans. Polynuclear aromatic compounds, part 4, bitumens, coal-tars and derived products, shale oils and soots. Vol. 35, Lyon, France: World Health Organization, International Agency for Research on Cancer. [cited in European Commission 2008].

[IARC] International Agency for Research on Cancer. 1987a. Monographs on the evaluation of the carcinogenic risk of chemicals to humans. An updating of IARC monographs volumes 1-42. Coal-tars. IARC Monographs Supplement 7. Lyon, France: World Health Organization, International Agency for Research on Cancer. p. 175-176.

[IARC] International Agency for Research on Cancer. 1987b. Monographs on the evaluation of the carcinogenic risk of chemicals to humans. An updating of IARC monographs volumes 1-42. Coal-tar pitches. IARC Monographs Supplement 7. Lyon,

France: World Health Organization, International Agency for Research on Cancer. p. 174-175.

[IARC] IARC Working Group on the Evaluation of Carcinogenic Risks to Humans. 2004. Overall evaluations of carcinogenicity to humans: as evaluated in IARC Monographs volumes 1-88 (a total of 900 agents, mixtures and exposures). Lyon (FR): International Agency for Research on Cancer.

[IARC] IARC Working Group on the Evaluation of Carcinogenic Risks to Humans. 2011. Agents classified by the IARC monographs, Volumes 1-122 [PDF]. Lyon (FR): International Agency for Research on Cancer.

Irwin RJ, Van Mouwerik M, Stevens L, Seese MD, Basham W. 1997. Environmental contaminants encyclopedia. Entry for BTEX and BTEX compounds. July 1, 1997. Fort Collins (CO): National Park Service, Water Resources Division, Water Operations Branch.

Jongeneelen FJ, den Akker VW, Bos RP, Anzion RBM, Theuws JLG, Roelofs HMJ, Henderson PT. 1988. 1-Hydroxypyrene as an indicator of the mutagenicity of coal tar after activation with human liver preparations. *Mutat Res.* 204(2):195-201.

Kesik K, Janik-Spiechowicz E. 1997. Comparison of the mutagenicity of chemical agents released during coke production. *Int J Occup Med Environ Health.* 10(3):267-272. [cited in ATSDR 2002].

Kienzler A, Mahler BJ, Van Metre PC, Schweigert N, Devaux A, Bony S. 2015. Exposure to runoff from coal-tar-sealed pavement induces genotoxicity and impairment of DNA repair capacity in the RTL-W1 fish liver cell line. *Sci Total Environ.* 520:73-80.

Kinkead ER. 1973. Toxicity of coal tar. In: Proceedings of the Fourth Annual Conference on Environmental Toxicology, held at Fairborn, Ohio, October 16-18, 1973. Aerospace Medical Research Laboratory, Wright-Patterson Air Force Base, Ohio. p. 177-188. [accessed 2010 Jul 6].

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 IARC 1985].

Kleffner HW, Talbiersky J, Zander M. 1981. Simple scheme for the estimation of concentrations of polycyclic aromatic hydrocarbons in tars. *Fuel.* 60:361-362.

Klein M. 1963. Susceptibility of strain B6AF1-J hybrid infant mice to tumorigenesis with 1,2-benzanthracene, deoxycholic acid and 3-methylcholanthrene. *Cancer Res.* 23:1701-1707. [cited in ATSDR 1995].

Kligman AM, Kligman LH. 1994. Carcinogens show comedogenic activity: a potential animal screen for tumorigenic substances. *Cancer Lett.* 87(2):171-178. [cited in ATSDR 2002].

Kocan RM, Matta MB, Salazar SM. 1996. Toxicity of weathered coal tar for shortnose sturgeon (*Acipenser brevirostrum*) embryos and larvae. *Arch Environ Contam Toxicol.* 31:161-165.

Koganti A, Singh R, Rozett K, Modi N, Goldstein LS, Roy TA, Zhang FJ, Harvey RG, Weyand EH. 2000. 7H-benzo[c]fluorene: a major DNA adduct-forming component of coal tar. *Carcinogenesis.* 21(8):1601-1609. [cited in ATSDR 2002].

Koganti A, Singh R, Ma BL, Weyand EH. 2001. Comparative Analysis of PAH: DNA Adducts Formed in Lung of Mice Exposed to Neat Coal Tar and Soils Contaminated with Coal Tar. *Environ Sci Technol.* 35(13):2704-2709.

Krewski D, Thorslund T, Withey J. 1989. Carcinogenic risk assessment of complex mixtures. *Toxicol Ind Health.* 5(5):851-867. [cited in CCME 2008].

LaFlamme RE, Hites RA. 1978. The global distribution of polycyclic aromatic hydrocarbons in recent sediments. *Geochim Cosmochim Acta.* 42:289-303.

Leadon SA, Sumnerel J, Minton TA, Tischler A. 1995. Coal tar residues produce both DNA adducts and oxidative DNA damage in human mammary epithelial cells. *Carcinogenesis.* 16(12):3021-3026. [cited in ATSDR 2002].

Ledesma EB, Kalish MA, Nelson PF, Wornat MJ, Mackie JC. 2000. Formation and fate of PAH during the pyrolysis and fuel-rich combustion of coal primary tar. *Fuel.* 79:1801-1814.

Lewis RJ Sr., ed. 2001. *Hawley's Condensed Chemical Dictionary*. 14th edition. New York (NY): John Wiley & Sons, Inc.

[LNHPD] Licensed Natural Health Products Database [database]. 2019 Ottawa (ON): Health Canada. [cited 2014 June].

MacEwen JD, Hall A III, Scheel LD. 1977. Experimental oncogenesis in rats and mice exposed to coal tar aerosols. Proceedings of the annual conference on environmental toxicology. Aerospace Medical Research Lab, Wright-Patterson AFB, OH. AMRL-TR-76-125. [cited in ATSDR 2002].

Mackay D, Shiu WY, Ma KC. 1992. Illustrated handbook of physical-chemical properties and environmental fate for organic chemicals. Volume II. Lewis Publishers, Michigan 1992. [cited in European Commission 2008 and WHO 1998].

Mahadevan B, Parsons H, Musafia T, Sharma AK, Amin S, Pereira C, Baird WM. 2004. Effect of artificial mixtures of environmental polycyclic aromatic hydrocarbons present in coal tar, urban dust, and diesel exhaust particulates on MCF-7 cells in culture. *Environ Mol Mutagen.* 44(2):9-107.

Mahadevan B, Marston CP, Dashwood WM, Li Y, Pereira C, Baird WM. 2005. Effect of a standardized complex mixture derived from coal tar on the metabolic activation of carcinogenic polycyclic aromatic hydrocarbons in human cells in culture. *Chem Res Toxicol.* 18(2):24-231.

Mahadevan B, Marston CP, Luch A, Dashwood WM, Brooks E, Pereira C, Doehmer J, Baird WM. 2007. Competitive inhibition of carcinogen-activating CYP1A1 and CYP1B1 enzymes by a standardized complex mixture of PAH extracted from coal tar. *Int J Cancer.* 120(6):1161-1168.

Mahler BJ, Van Metre PC, Foreman WT. 2014. Concentrations of polycyclic aromatic hydrocarbons (PAHs) and azaarenes in runoff from coal-tar- and asphalt-sealcoated pavement. *Environ Pollut.* 188:81-87.

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

Mayura K, Huebner HJ, Dwyer MR, McKenzie KS, Donnelly KC, Kubena LF, Phillips TD. 1999. Multi bioassay approach for assessing the potency of complex mixtures of polycyclic aromatic hydrocarbons. *Chemosphere.* 38(8):1721-1732. [cited in ATSDR 2002].

McConnell EE, Specht HD. 1973. Lesions found in animals exposed to coal tar aerosols. In: Proc. Fourth Annual Conference on Environmental Toxicology, October 16–18, Fairborn, OH, Paper No. 14. p. 189-198.

McCormick DL, Burns FJ, Alberg RE. 1981. Inhibition of benzo[a]pyrene-induced mammary carcinogenesis by retinyl acetate. *J Natl Cancer Inst.* 66(3):559-564. [cited in ATSDR 1995].

McIntyre JK, Edmunds RC, Anulacion BF, Davis JW, Incardona JP, Stark JD, Scholz NL. 2016. Severe coal tar sealcoat runoff toxicity to fish is prevented by bioretention filtration. *Environ Sci Technol* 50:1570-1578.

Meek ME, Chan PKL, Bartlett S. 1994. Polycyclic aromatic hydrocarbons: Evaluation of risks to health from environmental exposures in Canada. *J Environ Sci Health – Part C: Environ Carcinogenesis Ecotoxicol Rev.* 12(2):443-452. [cited in CCME 2008].

[MENVIQ] Ministère de l'Environnement du Québec. 1988. Les cokeries au Québec, rapport d'étape juin 1988. Québec (QC): Ministère de l'Environnement du Québec,

Direction des Substances dangereuses. [cited in Environment Canada, Health Canada 2011].

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

Milleman RE, Birge WJ, Black JA, Cushman RM, Daniels KL, Franco PJ, Giddings JM, McCarthy JF, Stewart AJ. 1984. Comparative acute toxicity to aquatic organisms of components of coal-derived synthetic fuels. *Trans Am Fish Soc.* 113:74-85.

Mitchell AD, Evans EL, Jotz MM, Riccio ES, Mortelmans KE, Simmon VF. 1981. Mutagenic and carcinogenic potency of extracts of diesel and related environmental emissions: *In vitro* mutagenesis and DNA damage. *Environ Int.* 5(4-6):393-401. [cited in IARC 1985].

Moore JW, Ramamoorthy S. 1984. Aromatic hydrocarbons – Polycyclics. In: DeSanto RS, editor. *Organic chemicals in natural waters: Applied monitoring and impact assessment.* New York (NY): Springer-Verlag. p. 67-87. [cited in CCME 1999b].

Mukhtar H, Link CM, Cherniack E, Kushner DM, Bickers DR. 1982. Effect of topical application of defined constituents of coal tar on skin and liver aryl hydrocarbon hydroxylase and 7-ethoxycoumarin deethylase activities. *Toxicol Appl Pharmacol.* 64(3):541-549.

Mukhtar H, Asokan P, Das M, Santella RM, Bickers DR. 1986a. Benzo[a]pyrene diol epoxide-I-DNA adduct formation in the epidermis and lung of SENCAR mice following topical application of crude coal tar. *Cancer Lett.* 33(3):287-294.

Mukhtar H, Das M, Bickers DR. 1986b. Skin tumor initiating activity of therapeutic crude coal to as compared to other polycyclic aromatic hydrocarbons in SENCAR mice. *Cancer Lett.* 31(2):147-151.

Muller P. 1997. Scientific Criteria Document for Multimedia Standards Development Polycyclic Aromatic Hydrocarbons (PAH); Part 1: Hazard Identification and Dose-Response Assessment. Standard Development Branch, Ontario Ministry of Environment and Energy. [cited in CCME 2008].

[NAPS] National Air Pollution Surveillance Network. 2008. Annual data summary for 2005 and 2006. Ottawa (ON): Environment Canada. [cited 2009 Nov].

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

[NCI] National Chemical Inventories [database]. 2010. Issue 1. Columbus (OH): American Chemical Society. [cited 2011 Jan].

- Neal J, Rigdon RH. 1967. Gastric tumors in mice fed benzo[a]pyrene: A quantitative study. *Tex Rep Biol Med.* 25(4):553-557. [cited in ATSDR 1995].
- Neff JM. 1979. Polycyclic aromatic hydrocarbons in the aquatic environment. Sources, fate and biological effects. London (GB): Applied Science Publishers. [cited in European Commission 2008].
- Nesnow S, Lewtas J. 1981. Mutagenic and carcinogenic potency of extracts of diesel and related environmental emissions: summary and discussion of the results. *Environ Int* 5(4-6):425-429. [cited in IARC 1985].
- Neuhauser EF, Kreitinger JP, Nakles DV, Hawthorne SB, Doherty FG, Ghosh U, Khalil MF, Ghosh RS, Jonker MTO, van der Heijden SA. 2006. Bioavailability and toxicity of PAHs at MGP sites. *Land Contam Recl.* 14(2):261-266.
- Neyra Industries Inc. 2011. Material Safety Data Sheet: Tarconite Coal Tar Pavement Sealer. Cincinnati (OH): Neyra Industries Inc. [Issued 2011 May 23; cited 2015 Apr 20].
- Neyra Industries Inc. 2014. Material Safety Data Sheet: Tarconite Cool Weather Formula. Cincinnati (OH): Neyra Industries Inc. [Issued 2014 Mar 19; cited 2015 Apr 20].
- Neyra. 2015. Tarconite Coal Tar Pavement Sealer Safety Data Sheet. Issued June 30, 2015. Cincinnati (OH): Neyra Industries, Inc.
- [NHPID] Natural Health Products Ingredients Database [database]. 2014. Ottawa (ON): Health Canada. [cited 2014 June].
- Niemeier RW, Thayer PS, Menzies KT, von Thuna P, Moss CE. 1988. A comparison of skin carcinogenicity of condensed roofing asphalt and coal tar pitch fumes. In: Cooke M, Dennis A, editors. *Polynuclear aromatic hydrocarbons: A decade of progress.* p. 609-647. [cited in ATSDR 2002].
- Nisbet ICT, LaGoy PK. 1992. Toxic equivalency factors (TEFs) for polycyclic aromatic hydrocarbons (PAHs). *Regul Toxicol Pharmacol.* 16(3):290-300.
- Novotny M., Strand JW, Smith SL, Wiesler D, Schwende FJ. 1981. Compositional studies of coal tar by capillary gas chromatography/mass spectrometry. *Fuel.* 60(3):213-220.
- [NPRI] National Pollutant Release Inventory [database]. 2014. Gatineau (QC). Environment Canada. [cited 2014 Jan].
- [NPRI] National Pollutant Release Inventory. 2013-2016. NPRI Datasets: NPRI On-line Facility Data Search. Ottawa (ON): Government of Canada. Search results for CAS RNs 83-32-9, 85-01-8 and 91-20-3. [modified 2017 Sep 14; accessed 2018 May 2].

[NTP] National Toxicology Program. 2005. Substance profiles: coal tars and coal tar pitches. In: 11th report on carcinogens (RoC). Research Triangle Park (NC): U.S. Department of Health and Human Services, National Toxicology Program. [cited 2011 Jan].

[NTP] National Toxicology Program. 2011. Substance profiles: coal tars and coal tar pitches. In: 12th report on carcinogens (RoC). Research Triangle Park (NC): U.S. Department of Health and Human Services, National Toxicology Program. [cited 2012 Sep 17].

[NTP] National Toxicology Program. 2016. Substance profiles: coal tars and coal tar pitches. In: 14th report on carcinogens (RoC). Research Triangle Park (NC): U.S. Department of Health and Human Services, National Toxicology Program. [accessed 2018 Sep 12]

Oberholster PJ, Botha AM, Cloete TE. 2005. Using a battery of bioassays, benthic phytoplankton and the AUSRIVAS method to monitor long-term coal tar contaminated sediment in the Cache la Poudre River, Colorado. *Water Res.* 39:4913-4924.

[Ontario] Ontario Standards Development Branch, Ministry of the Environment. 2011. Ontario Air Standards for Benzo[a]pyrene as a Surrogate for Polycyclic Aromatic Hydrocarbons [PDF]. Toronto (ON): Ministry of the Environment.

[Ontario] Ontario Standards Development Branch, Ministry of the Environment. 2012. Air quality in Ontario [PDF]. Toronto (ON): Ministry of the Environment. [accessed 2018 Sep 12].

[Ontario] Ontario Standards Development Branch, Ministry of the Environment. 2016. Air quality in Ontario [PDF]. Toronto (ON): Ministry of the Environment. [accessed 2018 Sep 12].

[Ontario] Ontario Ministry of the Environment Spill Database [database]. 2013. Data for years 2008–2012. Toronto (ON): Ontario Ministry of the Environment. Unpublished.

Paine MD, Chapman PM, Allard PJ, Murdoch MH, Minifie D. 1996. Limited bioavailability of sediment PAH near an aluminum smelter: Contamination does not equal effects. *Environ Toxicol Chem.* 15(11):2003-2018.

Pavanello S, Levis AG. 1992. Coal tar therapy does not influence in vitro benzo[a]pyrene metabolism and DNA adduct formation in peripheral blood lymphocytes of psoriatic patients. *Carcinogenesis.* 13(9):1569-1573. [cited in ATSDR 2002].

Pavanello S, Levis AG. 1994. Human peripheral blood lymphocytes as a cell model to evaluate the genotoxic effect of coal tar treatment. *Environ Health Perspect.* 102(9):95-99. [cited in ATSDR 2002].

Pavement Rejuvenation International. 2014. Material Safety Data Sheet: Rejuvaseal. Buda (TX): Pavement Rejuvenation International, LP. [updated 2014 Feb 2; cited 2015 Apr 30].

Payne JF, Mathieu A, Collier TK. 2003. Ecotoxicological studies focusing on marine and freshwater fish. In: Douben PET, editor. PAHs: an ecotoxicological perspective. Hoboken (NJ): Wiley. p. 192-224.

[PCTC] Pavement Coatings Technology Council. 2018. [ACCCI RT-12 Product Description](#). Alexandria (VA): PCTC.

Peters AJ, Gregor DJ, Teixeira CF, Jones NP and Spencer C. 1995. The recent depositional trend of polycyclic aromatic hydrocarbons and elemental carbon to the Agassiz Ice Cap, Ellesmere Island, Canada. *Sci Total Environ*. 160-161:167-179.

Pham TT, Proulx S. 1997. PCBs and PAHs in the Montreal Urban Community (Quebec, Canada) wastewater treatment plant and the effluent plume in the St. Lawrence River. *Water Res*. 31(8):1887-1896.

Phillips DH, Alldrick AJ. 1994. Tumorigenicity of a combination of psoriasis therapies. *Br J Cancer*. 69(6):1043-1045.

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(1):268-278. [cited in Environment Canada, Health Canada 2008].

PubMed [database]. 1996. Bethesda (MD): National Library of Medicine (U.S.). [updated 2011 Jan 1, cited 2013 Jan 10].

[R4CC] REACH for Coal Chemicals. 2011. Substance Inventory Attachment 2: Table of UVCB Substances.

Rao TK, Young JA, Weeks CE, Slaga TJ, Epler JL. 1979. Effect of the co-carcinogen benzo[e]pyrene on microsome-mediated chemical mutagenesis in *Salmonella typhimurium*. *Environ Mutagen*. 1(2):105-112. [cited in IARC 1985].

RejuvaSeal. c2018. RejuvaSeal® The global leader in pavement rejuvenation: what is RejuvaSeal? Buda (TX): Pavement Rejuvenation International, LLC. [accessed 2018 May 24].

[RDRC] Resources Development Research Centre. 1987. National overview of abandoned coal gasification works in Canada. Prepared for the Conservation and Protection Service, Environment Canada, by the Resources Development Research

Centre, Carleton University, Ottawa, Ontario. 41 pp. Contract Report No.: KE145-6-0728.

Rinsky RA, Smith AB, Hornung R, Filloon TG, Young RJ, Okun AH, Landrigan PJ. 1987. Benzene and leukemia: an epidemiologic risk assessment. *N Engl J Med.* 316(17):1044-1050. [cited in Environment Canada, Health Canada 1993; U.S. EPA 2000].

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 Appl Toxicol.* 4(1):49-56.

Rojas M, Godschalk R, Alexandrov K, Cascorbi I, Kriek E, Ostertag J, van Schooten FJ, Bartsch H. 2001. Myeloperoxidase--463A variant reduces benzo[alpha]pyrene diol epoxide DNA adducts in skin of coal tar treated patients. *Carcinogenesis.* 22(7):1015-1018. [cited in ATSDR 2002].

Rostad CE, Pereira WE, Hult MF. 1985. Partitioning studies of coal-tar constituents in a two-phase contaminated ground-water system. *Chemosphere.* 14(8):1023-1036.

Rowe AA, O'Connor, TP. 2011. Assessment of water quality of runoff from sealed asphalt surfaces. Cincinnati (OH): U.S. Environmental Protection Agency, Office of Research and Development. Report No.: EPA/600/R-10/178.

Santella RM, Perera FP, Young TL, Zhang YJ, Chiamprasert S, Tang D, Wang LW, Beachman A, Lin JH, DeLeo VA. 1995. Polycyclic aromatic hydrocarbon-DNA and protein adducts in coal tar treated patients and controls and their relationship to glutathione S-transferase genotype. *Mutat Res.* 334(2):117-224. [cited in ATSDR 2002].

Saperstein MD, Wheeler LA. 1979. Mutagenicity of coal tar preparations used in the treatment of psoriasis. *Toxicol Lett.* 3(6):325-329. [cited in CIR 2008; IARC 1985].

Sarto F, Zordan M, Tomanin R, Mazzotti D, Canova A, Cardin EL, Bezze G, Levis AG. 1989. Chromosomal alterations in peripheral blood lymphocytes, urinary mutagenicity and excretion of polycyclic aromatic hydrocarbons in six psoriatic patients undergoing coal tar therapy. *Carcinogenesis.* 10(2):329-334. [cited in CIR 2008; ATSDR 2002].

Schoeny R, Warshawsky D, Hollingsworth L, Hund M, Moore G. 1981. Mutagenicity of products from coal gasification and liquefaction in the Salmonella/microsome assay. *Environ Mutagen.* 3(2):181-195.

Schoket B, Hewer A, Grover PL, Phillips DH. 1988. Covalent binding of components of coal-tar, creosote, and bitumen to the DNA of the skin and lungs of mice following topical application. *Carcinogenesis.* 9(7):1253-1258.

Schulte A, Ernst H, Peters L, Heinrich U. 1994. Induction of squamous cell carcinomas in the mouse lung after long-term inhalation of polycyclic aromatic hydrocarbon-rich exhausts. *Exp Toxicol Pathol.* 45(7):415-421. [cited in European Commission 2008].

Scoggins M, McClintock NL, Gosselink L, Bryer P. 2007. Occurrence of polycyclic aromatic hydrocarbons below coal-tar-sealed parking lots and effects on stream benthic macroinvertebrate communities. *J N Am Benthol Soc.* 26(4):694-707.

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

[SDS] Safety Data Sheet. 2013. Coal tar roofing pitch. SDS ID: 00228355. Issue date March 14, 2013. Pittsburgh (PA): KOPPERS Inc.

Singer MM, Aurand DV, Coelho GM, Bragin GE, Clark JR, Sowby M and Tjeerdema RS. 2001. Making, measuring, and using water-accommodated fractions of petroleum for toxicity testing. *International Oil Spill Conference Volume 2. International Oil Spill Conference Proceedings: March 2001, Volume 2001, No. 2, p. 1269-1274.*

[SLV] St. Lawrence Vision 2000. 1996. QIT-Fer et Titane inc. Ottawa (ON): Ministry of Supply and Services Canada. 4 pp. Fact Sheet No. 28.

Solorzano MF, Barcon OD, Lopez ME. 1993. Informe sobre caracterización de residuos (residuo: brea). (1/13/0578 no. 2). *Anencia del Medo Ambiente*. [cited in European Commission 2008].

Sporstøl S, Gjøns N, Lichtenthaler RG, Gustavsen KO, Urdal K, Oreld F. 1983. Source identification of aromatic hydrocarbons in sediments using GC/MS. *Environ Sci Technol.* 17:282-286.

Springer DL, Poston KA, Mahlum DD, Sikov MR et al. 1982. Teratogenicity following inhalation exposure of rats to a high-boiling coal liquid. *J Appl Toxicol.* 5(2):261-265.

Springer DL, Miller RA, Weimer WC, Ragan HA, Buschbom RL, Mahlum DD. 1986. Effects of inhalation exposure to a high-boiling (288 to 454°C) coal liquid. *Toxicol Appl Pharmacol.* 82(1):112-131.

Staikova M, Messih P, Lei YD, Wania F, Donaldson DJ. 2005. Prediction of subcooled vapor pressures of nonpolar organic compounds using a one-parameter QSPR. *J Chem Eng Data.* 50:438-443.

Steel Times International. 2013. [SGL closes Canadian electrode plant](#). Oct. 23, 2013. [accessed 2014 May 20].

Surface Coatings Company. 1999. SealPave Driveway Sealer Material Safety Data Sheet. Effective date August 31, 1999. Auburn Hills (MI): Surface Coatings Company.

Sutton M. 2008. Coal tar pitch markets in Europe & North America. Presentation at the 12th Annual Met Coke World Summit October 22-24, 2008, Chicago, Illinois. Hamilton (ON): VFT Canada Inc.

Tadokoro H, Maeda M, Kawashima Y, Kitano M, Hwang D-F, Yoshida T. 1991. Aquatic toxicity testing for multicomponent compounds with special reference to preparation of test solution. *Ecotoxicol Environ Saf.* 21:57-67.

Thyssen J, Althoff J, Kimmerle G, Mohr U. 1981. Inhalation Studies with Benzo[a]pyrene in Syrian Golden Hamsters. *J Natl Cancer Inst* 66(3):575-577. [cited in Canada 1994].

Union Carbide. 1986. Acute toxicity of alpha-naphthol to mysids *Mysidopsis Bahia*. Report No. BW-86-8-2134, Study No. 565.03866122.510. Wareham (MA): Springborn Bionomics, Inc. [cited in European Commission 2003].

[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.

[U.S. EPA] United States Environmental Protection Agency. 1989. Coke oven emissions. Integrated Risk Information System (IRIS)

[U.S. EPA] United States Environmental Protection Agency. 1992a. Screening Procedures for Estimating the Air Quality Impact of Stationary Sources, Revised. Office of Air Quality. Washington (DC): U.S. EPA.

[U.S. EPA] United States Environmental Protection Agency. 1992b. Peer Consultation Workshop on Approaches to Polycyclic Aromatic Hydrocarbon (PAH) Health Assessment. Washington (DC): U.S. EPA.

[U.S. EPA] United States Environmental Protection Agency. 1993. Provisional guidance for quantitative risk assessment of polycyclic aromatic hydrocarbons. Cincinnati (OH): U.S. EPA, Environmental Criteria and Assessment Office.

[U.S. EPA] United States Environmental Protection Agency. 1999. Screening level ecological risk assessment protocol for hazardous waste combustion facilities. Volume 1, Chapter 3: Air dispersion and air deposition modelling. U.S. EPA, Office of Solid Waste. Peer Review Draft. Aug. 1999. EPA530-D-99-001A.

[U.S. EPA] United States Environmental Protection Agency. 2000. Benzene (CAS RN 71-43-2) [PDF]. Washington (DC): U.S. EPA, Integrated Risk Information System (IRIS).

[U.S. EPA] United States Environmental Protection Agency. 2003. Procedures for the derivation of equilibrium partitioning sediment benchmarks (ESBs) for the protection of benthic organisms: PAH mixtures. Report No.: EPA-600-R-02-013. Washington (DC): U.S. Environmental Protection Agency, Office of Research and Development.

[U.S. 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 [PDF]. Washington (DC): U.S. Environmental Protection Agency.

[U.S. EPA] United States Environmental Protection Agency. 2011a. Technology Transfer Network – Support Centre for Atmospheric Modelling – Screening Models – Aerscreen. Research Triangle Park (NC): U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards, Emissions, Monitoring, and Analysis Division.

[U.S. EPA] United States Environmental Protection Agency. 2011b. Exposure Factors Handbook 2011. National Center for Environmental Assessment. Office of Research and Development. U.S. Environmental Protection Agency, Washington, DC 20460.

[U.S. EPA] United States Environmental Protection Agency. 2013. Priority Pollutants. Webpage. [accessed 2014 Mar 19].

U.S. Pharmacopia [database]. 2008–2010. Coal Tar Topical Solution, USP 29.

Uthe JF. 1991. Polycyclic aromatic hydrocarbons in the environment. *Can Chem News*. Aug: 25–27. [cited in CCME 1999a].

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

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

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 Vlaardingen PLA, Steinhoff WJ, de Voogt P, Admiraal WA. 1996. Property-toxicity relationships of azaarenes to the green alga *Scenedesmus acuminatus*. *Environ Toxicol Chem*. 15(11):2035-2042.

Vighi M, Altenburger R, Arrhenius A, Backhaus T, Boedeker W, Blanck H, Consolaro F, Faust M, Finizio A, Froehner K, Gramatica P, Grimme LH, Grönvall F, Hamer V, Scholze M, Walter H. 2003. Water quality objectives for mixtures of toxic chemicals: problems and perspectives. *Ecotoxicol Environ Saf*. 54:139-150.

Wallcave L, Garcia H, Feldman R, Lijinsky 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(1):41-52.

Wang R, Tao S, Wang B, Yang Y, Lang C, Zhang Y, Hu J, Ma J, Hung H. 2010a. Sources and pathways of polycyclic aromatic hydrocarbons transported to Alert, the Canadian High Arctic. *Environ Sci Technol.* 44(3):1017-1022.

Wang R, Tao S, Wang B, Yang Y, Lang C, Zhang Y, Hu J, Ma J, Hung H. 2010b. Supporting information for Wang et al. 2010a. 14 pp.

Watts AW, Ballester TP, Rosen RM, Houle JP. 2010. Polycyclic aromatic hydrocarbons in stormwater runoff from sealcoated pavements. *Environ Sci Technol.* 44(23):8849-8854.

[WCI] World Coal Institute. 2020. Coal & steel. [cited 2021, May].

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(3-4), 927-935.

Weyand EH, Wu Y. 1994. Genotoxicity of manufactured gas plant residue (MGP) in skin and lung of mice following MGP ingestion or topical administration. *Polycycl Aromat Compd.* 6(1):35-42.

Weyand EH, Wu Y. 1995. Covalent binding of polycyclic aromatic hydrocarbon components of manufactured gas plant residue to mouse lung and forestomach DNA. *Chem Res Toxicol.* 8(7):955-962.

Weyand EH, Wu Y, Patel S, Taylor BB, Mauro DM. 1991. Urinary excretion and DNA binding of coal tar components in B6C3F1 mice following ingestion. *Chem Res Toxicol.* 4(4):466-473. [ccited in ATSDR 2002].

Weyand EH, Wu Y, Patel S, Goldstein LS. 1994. Biochemical effects of manufactured gas plant residue following ingestion by B6C3F1 mice. *J Toxicol Environ Health.* 42(1):89-107. [cited in ATSDR 2002].

Weyand EH, Chen YC, Wu Y, Koganti A, Dunsford HA, Rodriguez LV. 1995. Differences in the tumorigenic activity of a pure hydrocarbon and a complex mixture following ingestion: Benzo[a]pyrene vs manufactured gas plant residue. *Chem Res Toxicol.* 8(7):949-954. [cited in ATSDR 2002].

[WHO] World Health Organization. 1998. Selected non-heterocyclic polycyclic aromatic hydrocarbons. Environmental Health Criteria 202. Geneva (CH): International Programme on Chemical Safety. [cited 2014].

[WHO] World Health Organization. 2000. Air Quality Guidelines. Evaluation of Human Health Risk – Benzene (Chapter 5.2) [PDF]. Copenhagen (DK): Copenhagen WHO Regional Office for Europe.

[WHO] World Health Organization. 2001. Polycyclic aromatic hydrocarbons. In: Background material to WHO Regional Publications, European Series, No. 91 (Air Quality Guidelines for Europe, 2nd ed. 2000). Chapter 5.9: p. 1-24. WHO Regional Office for Europe, Copenhagen, Denmark.

Wilson R, Jones-Otazo H, Petrovic S, Mitchell I, Bonvalot Y, Williams D, Richardson M. 2013. Revisiting dust and soil ingestion rates based on hand-to-mouth transfer. *Hum Ecol Risk Assess.* 19:158-188.

Wise SA, Benner, BA, Byrd BD, Chesler SN, Rebbert RE, Shantz MM. 1988. Determination of polycyclic aromatic hydrocarbons in a coal tar standard reference material. *Anal Chem.* 60:887-894.

Wise SA, Poster DL, Leigh SD, Rimmer CA, Mössner S, Schubert P, Sander LC, Schantz MM. 2010. Polycyclic aromatic hydrocarbons (PAHs) in a coal tar standard reference material—SRM 1597a updated. *Anal Bioanal Chem.* 398:717-728.

Wright CW, Later DW, Pelroy RA, Mahlum DD, Wilson BW. 1985. Comparative chemical and biological analysis of coal tar-based therapeutic agents to other coal-derived materials. *J Appl Toxicol.* 5(2):80-88. [cited in CIR 2008].

[WTO] World Trade Organization. Committee on Technical Barriers to Trade – Notification – European Union – Chemical Substances. 2013. G/TBT/N/EU/118. Document #: 13-3135. [Published 2013 June 17]. [accessed 2013 Dec 9].

Xie R, Koziar SA, Lampi MA, Dixon DG, Norwood WP, Borgmann U, Huang X-D, Greenberg BM. 2006. Assessment of the toxicity of copper, 9,10-phenanthrenequinone, and phenanthrene to *Daphnia magna*: Evidence for a reactive oxygen mechanism. *Environ Toxicol Chem.* 25(2):613-622.

Zangar RC, Springer DL, Buschbom RL, Mahlum DD. 1989. Comparison of fetotoxic effects of a dermally applied complex organic mixture in rats and mice. *Fundam Appl Toxicol.* 13(4):662-669.

Zhang YJ, Li Y, DeLeo VA, Santella RM. 1990. Detection of DNA adducts in skin biopsies of coal tar-treated psoriasis patients: Immunofluorescence and 32 P-postlabeling. *Skin Pharmacol.* 3(3):171-179. [cited in ATSDR 2002].

Appendix A. Industrial release from coal tar processing, handling and storage for coal tar refineries

Table A-1. Variable inputs to SCREEN3 for coal tar refinery benzene dispersion

Variables	Input variables
Source type	Area
Effective emission area ^a	200 m × 275 m
Emission rate of benzene (g/s)	0.130
Receptor height ^b	1.74 m (average adult height)
Source release height ^a	10 m
Adjustment factor ^c	0.2 (average wind direction over a 1-year period)
Urban–rural option	Urban
Meteorology ^d	1 (full meteorology)
Minimum and maximum distance	0–3000 m

^a Professional judgement

^b Curry et al. (1993)

^c U.S. EPA (1992a)

^d Default value in SCREEN3

Table A-2. Ambient concentrations of benzene in the vicinity of a coal tar refinery from SCREEN3

Distance (m)	1-hour concentration ($\mu\text{g}/\text{m}^3$)	Annual concentration ($\mu\text{g}/\text{m}^3$)
1	12.07	2.41
100	19.93	3.99
200	27.62	5.52
230	28.76	5.75
300	25.54	5.11
400	19.88	3.98
500	15.94	3.19
600	13.17	2.63
700	11.13	2.23
800	9.55	1.91
900	8.28	1.66
1000	7.27	1.45
1100	6.44	1.29
1200	5.76	1.15
1300	5.19	1.04
1400	4.71	0.94
1500	4.30	0.86
1600	3.95	0.79
1700	3.65	0.73
1800	3.39	0.68
1900	3.16	0.63
2000	2.95	0.59
2100	2.77	0.55
2200	2.61	0.52
2300	2.46	0.49
2400	2.33	0.47
2500	2.21	0.44
2600	2.10	0.42
2700	2.00	0.40
2800	1.91	0.38
2900	1.83	0.37
3000	1.75	0.35

Appendix B. Industrial release from coal tar production, handling and storage for steel mills

Table B-1. Variable inputs to SCREEN3 for coal tar storage (benzene) at steel mills

Variables	Input
Source type	Area
Effective emission area ^a	200 m × 275 m
Emission rate upper-bound (g/m·s ²)	1.806×10^{-6}
Emission rate lower-bound (g/m·s ²)	1.713×10^{-7}
Receptor height ^b	1.74 m
Source release height ^a	10 m
Adjustment factor for annual ^c	0.2
Urban–rural option	Urban
Meteorology ^d	1 (full meteorology)
Minimum and maximum distance to use	1–3000 m

^a Professional judgement

^b Curry et al. (1993)

^c U.S. EPA (1992a)

^d Default value in SCREEN3 (1996)

Table B-2. Upper bounding ambient air concentrations of benzene in the vicinity of coal tar storage and loading at steel mills

Distance (m)	Upper-bounding benzene emissions – 1-hour concentration ($\mu\text{g}/\text{m}^3$)	Upper-bounding benzene emissions – annual concentration ($\mu\text{g}/\text{m}^3$)
1	9.22	1.84
100	15.22	3.04
200	21.10	4.22
230	21.97	4.39
300	19.51	3.90
400	15.19	3.04
500	12.18	2.44
600	10.06	2.01
700	8.50	1.70
800	7.29	1.46
900	6.33	1.27
1000	5.55	1.11
1100	4.92	0.98
1200	4.40	0.88
1300	3.96	0.79
1400	3.60	0.72
1500	3.29	0.66
1600	3.02	0.60
1700	2.79	0.56
1800	2.59	0.52
1900	2.41	0.48
2000	2.26	0.45
2100	2.12	0.42
2200	1.99	0.40
2300	1.88	0.38
2400	1.79	0.36
2500	1.69	0.34
2600	1.61	0.32
2700	1.53	0.31
2800	1.46	0.29
2900	1.40	0.28
3000	1.34	0.27

Table B-3. Lower bounding ambient air concentrations of benzene in the vicinity of coal tar storage and loading at steel mills

Distance (m)	Lower-bounding benzene emissions – 1-hour concentration ($\mu\text{g}/\text{m}^3$)	Lower-bounding benzene emissions – annual concentration ($\mu\text{g}/\text{m}^3$)
1	0.87	0.17
100	1.44	0.29
200	2.00	0.40
300	1.85	0.37
400	1.44	0.29
500	1.16	0.23
600	0.95	0.19
700	0.81	0.16
800	0.69	0.14
900	0.60	0.12
1000	0.53	0.11
1100	0.47	0.09
1200	0.42	0.08
1300	0.38	0.08
1400	0.34	0.07
1500	0.32	0.06
1600	0.29	0.06
1700	0.26	0.05
1800	0.25	0.05
1900	0.23	0.05
2000	0.21	0.04
2100	0.20	0.04
2200	0.19	0.04
2300	0.18	0.04
2400	0.17	0.03
2500	0.16	0.03
2600	0.15	0.03
2700	0.15	0.03
2800	0.14	0.03
2900	0.13	0.03
3000	0.13	0.03

Appendix C. Exposures from products used by consumers

Table C-1. PAH concentrations ($\mu\text{g/g}$) and benzo[a]pyrene equivalents in house dust

Compound ^a	Anthracene	Benzo[a]pyrene	Fluoranthene	Naphthalene	Benzo[k]fluoranthene
Conc.	0.32	3.42	9.04	0.22	2.24
Conc.	1.17	15.2	39	0.47	9.47
Conc.	1.05	10.9	32.1	0.49	7.07
Conc.	0.28	4.04	9.79	0.21	2.73
Conc.	2.4	14.3	44.2	0.23	10.4
Conc.	0.15	1.21	4.42	0.14	0.8
Conc.	0.11	1.41	2.76	0.05	1.14
Conc.	0.91	7.33	32.7	0.49	5.04
Conc.	0.63	4.5	13.8	0.15	3.09
Conc.	0.5	4.44	36.3	0.19	3.43
Conc.	3.18	24.2	70.7	0.63	15.2
Average	0.97	8.27	26.8	0.297	5.51
Potency factor ^b	0.001	1	0.001	0.001	0.1
B[a]P equivalents	0.00097	8.27	0.0268	0.000297	0.551

^a Concentration data from Mahler (2010)

^b Potency equivalency factors from Nisbet and LaGoy (1992)

Table C-2. PAH concentrations ($\mu\text{g/g}$) and benzo[a]pyrene equivalents in house dust

Compound ^a	Benz[a]anthracene	Pyrene	Fluorene	Dibenzo[a,h]anthracene	Indeno [1,2,3-cd]pyrene
Conc.	3.17	2.48	0.11	0.57	2.13
Conc.	13.2	12.4	0.45	2.44	10.1
Conc	7.7	11.8	0.41	2.12	8.27
Conc.	4.01	1.98	0.11	0.84	3.4
Conc	14.7	16.2	1.08	2.54	11.3
Conc	0.93	1.22	0.07	0.28	0.98
Conc	3.99	0.8	0.04	0.5	2.05
Conc	6.24	15.3	0.76	1.26	5.33
Conc	4.15	5.63	0.27	0.84	3.11
Conc	4.05	14.4	0.22	0.91	3.38
Conc.	20.8	25.3	1.31	5.27	18.7
Average	7.977	9.77	0.439	1.6	6.25
Potency factor ^b	0.1	0.001	0.001	5	0.1
B[a]P equivalents	0.7977	0.00977	0.000439	7.99	0.625

^a Concentration data from Mahler (2010)

^b Potency equivalency factors from Nisbet and LaGoy (1992)

Table C-3. PAH concentrations ($\mu\text{g/g}$) and benzo[a]pyrene equivalents in house dust

Compound ^a	Phenanthrene	Acenaphthene	Acenaphthalene	Benzo[b]fluoranthene	Benzo [g,h,i] perylene	Chrysene
Conc.	2.48	0.06	0.06	5.66	2.38	5.21
Conc.	12.4	0.6	0.19	25.5	12.3	20.6
Conc.	11.8	0.22	0.23	20.8	10.5	15.6
Conc.	1.98	0.1	0.05	8.51	4.53	6.75
Conc.	16.2	0.76	0.32	28.5	12.3	24.7
Conc.	1.22	0.06	0.05	2.7	1.22	2
Conc.	0.8	0.5	0.04	2.73	2.01	6.87
Conc.	15.3	0.78	0.26	14.7	6.53	15.2
Conc.	5.63	0.22	0.16	8.33	3.23	6.94
Conc.	14.4	0.19	0.14	15.9	3.69	15.7
Conc.	25.3	0.93	0.3	38.4	22.2	38.3
Average ($\mu\text{g}/\text{m}^3$)	9.77	0.402	0.164	15.6	7.35	14.35182
Potency factor ^b	0.001	0.001	0.001	0.1	0.01	0.01
B[a]P equivalents	0.00977	0.000402	0.000164	1.56	0.0735	0.143518
Sum B[a]P ($\mu\text{g}/\text{g}^3$) ^c	20.1					

^a Concentration data from Mahler (2010)

^b Potency equivalency factors from Nisbet and LaGoy (1992)

^c Total B[a]P equivalents from the sum of the potency equivalent PAH values

Appendix D. Critical health effects information on coal tar substances

Table D-1. Critical health effects information on coal tar substances

Endpoints	CAS RN	Effect levels ^a /Results
Acute health effects	65996-93-2	Oral LD₅₀ = 3300 mg/kg bw (Wistar rat) (European Commission 2008)
Acute health effects	65996-93-2	Oral LD₅₀ = > 15 000 mg/kg bw (rat) (European Commission 2008)
Acute health effects	65996-93-2	Dermal LD₅₀ = > 400 mg/kg bw (Sprague-Dawley rat) (European Commission 2008)
Acute health effects	8007-45-2/ 65996-89-6	Dermal LD₅₀ = > 7950 mg/kg bw (rabbit) (ENTOX 2005)
Acute health effects	8007-45-2/ 65996-89-6	Dermal LOEL = 0.286 mg/kg bw based on induction of enzyme activities in humans. A single topical application of 100 µL (20 mg ^b or 0.286 mg/kg bw ^c) coal tar solution (U.S. Pharmacopeia) was applied to a 1-cm diameter section of clinically unaffected skin of 9 humans; a second skin area > 10 cm away was left untreated or was treated with 100 µL of the vehicle. 6-mm-punch biopsies were taken afterwards. 2- to 5-fold induction of cutaneous aryl hydrocarbon hydroxylase (AHH) occurred at the treated skin over the untreated areas (Bickers and Kappas 1978).
Acute health effects	8007-45-2/ 65996-89-6	Dermal effect level = 2041 mg/kg bw based on induction of enzyme activities in neonatal rats. A single topical application of 100 µL (20 mg ^b or 2041 mg/kg bw ^d) standard coal-tar solution (U.S. Pharmacopeia) was applied to neonatal rats (4–6 days old, 6–8 animals), which were sacrificed 24 hours later; 4-day old neonatal rats treated with topically applied acetone (100 µL) served as controls. Significant induction of AHH in the skin and liver (15- and 8-fold over controls, respectively). AHH activity of 3.69 ± 0.42 pmol 3-OH BP/min/mg protein in the skin (control: 0.24 ± 0.03 pmol 3-OH BP/min/mg protein); AHH activity of 192.73 ± 5.82 pmol 3-OH BP/min/mg protein in the liver (control: 23.22 ± 1.41 pmol 3-OH BP/min/mg protein) (Bickers et al. 1982; similar study/results by Mukhtar et al. 1982).

Short-term repeated-exposure health effects (< 90 days)	65996-93-2	Oral LOAEL = 37.5 mg/kg bw/day based on early mortality and degenerative changes in the liver. Pigs (9 weeks old) exposed to liquid coal tar by capsule at a dose of 3000 mg/day (37.5 mg/kg bw/day ^e) for 5 days (3 animals) or 2 days (2 animals). No gross pathology changes seen in the diet-control group. In the 5-day treatment group, 3 of 3 animals showed diffuse degenerative changes in the liver, and 3 of 3 animals died within 10–18 days. In the 2-day treatment group, 1 of 2 animals showed pseudomelanosis of the colon, and 1 of 2 animals died in 38 days (Graham et al. 1940).
Short-term repeated-exposure health effects (< 90 days)	8007-45-2/ 65996-89-6	Oral effect level = 1067 mg/kg bw/day based on decreases in body weight and food consumption. Male B6C3F1 mice (8 animals per dose) exposed to 0, 0.10, 0.25, 0.50, 1.0 or 2.0% (0, 197, 410, 693, 1067 or 1750 mg/kg bw/day) ^f coal tar in the diet for 28 days. Authors note average body weight and food consumption were significantly less for the 1% and 2% dose groups ($p < 0.05$), and effects were dose-related (Culp and Beland 1994; also cited in ATSDR 2002).
Short-term repeated-exposure health effects (< 90 days)	8007-45-2/ 65996-89-6	Inhalation LOAEC = 30 mg/m³ based on histiocytosis of lung tissue in both sexes, increased relative liver weight in females, and a decreased eosinophil count in males. Fischer-344 rats (42 animals per sex per concentration) exposed to 0, 30, 140 or 690 mg/m ³ coal tar aerosol, 6 hours/day, 5 days/week for 5 weeks. <i>All effects were significant ($p < 0.05$) and concentration-related.</i> $\geq 30 \text{ mg/m}^3$ = Histiocytosis of lung tissue (m/f); increase in relative liver weight (f) and relative kidney weight (m); decrease in eosinophils (m). $\geq 140 \text{ mg/m}^3$ = Decrease in body weight (m/f), increase in relative kidney weight (f); decrease in RBCs, hemoglobin, volume of packed red cells (m/f) and triglycerides (m), increase in reticulocytes and serum cholesterol (f). 690 mg/m^3 = Increase in relative liver weight (m), decrease in relative thymus

		weight (f), decrease in eosinophils (f), increase in reticulocytes and serum cholesterol (m). Other effects observed at the highest dose were significant, but did not follow a concentration-response trend, including decrease of megakaryocytes in spleen (m/f), hepatopathy (m/f), thymus atrophy (m), epithelial hyperplasia and chronic inflammation in cecum (m) (Springer et al. 1986).
Short-term repeated-exposure health effects (< 90 days)	8007-45-2/ 65996-89-6	Dermal LOEL = 10 mg of 0.1% based on comedogenicity. Male Australian albino rabbits (3 animals per dose) exposed to 10 mg of 0.001, 0.01, 0.1 or 1% (0.00005, 0.0005, 0.005 or 0.05 mg/kg bw/day) ^g crude coal tar applied to the region just exterior to the ear canal, 5 consecutive days/week for 3 weeks. None of the control vehicles were comedogenic when applied up to 20 weeks. Authors stated that the threshold for comedogenicity was < 0.1% (severity grade not reported). ATSDR (2002) identifies a LOEL of 0.1% for comedogenicity (Kligman and Kligman 1994; also cited in ATSDR 2002).
Subchronic repeated-exposure health effects (≥ 90 days)	8007-45-2/ 65996-89-6	Oral NOAEL = 462 mg/kg bw/day based on no adverse effects observed on the lungs, bone marrow, glodular, stomach, liver weight, kidney, bladder, salivary glands, pancreas, thymus, parathyroid, adrenal glands or body weight. B6C3F1 mice (24 animals per sex per dose) fed coal tar (Manufactured Gas Plant residues) at doses of 0, 0.05, 0.25 or 0.5%, or 0, 51, 251 or 462 mg/kg bw/day (males) and 0, 42, 196 or 344 mg/kg bw/day (females). ^f Half of the animals were sacrificed after 94 days of treatment and all organs were examined for gross lesions; the remaining animals were sacrificed after a total treatment of 185 days (with 94 previous and 91 additional treatment days) (Weyand et al. 1994; also cited in ATSDR 2002).
Subchronic repeated-exposure health effects (≥ 90 days)	8007-45-2/ 65996-89-6	Inhalation LOAEC = 0.2 mg/m³ based on decreased growth rate for every animal species and age group. Female Sprague-Dawley yearling rats, male and female

		<p>Sprague-Dawley weanling rats, male ICR mice and male CAF-1 mice (approximately 8 animals per group) exposed, in a chamber, to 0, 0.2, 2.0, 10 or 20 mg/m³ coal tar aerosol, 23.75 hours/day for 90 days. Male Golden Syrian hamsters and female New Zealand albino rabbits were exposed to 20 mg/m³ only (approximately 10 animals per group). After 90 days, 10% of the animals in each group were sacrificed for examination, the remainder were observed for various lengths of time up to their natural lifetime. [In order to generate a coal tar aerosol, benzene was added to the coal tar to separate out solids, where the benzene added could be removed by fractional distillation; the light oil fraction was also removed, which contains BTX (benzene, toluene, xylene); most aerosols were noted to be ≤ 5µm 95% of the time]. During exposure, body weight gain was significantly decreased compared to control (and observed a concentration-related trend). Although weights increased post-exposure, the response was still observed 7 months post-exposure. Coal tar deposition in the lungs was noted, and pneumonia was found in animals of all species that died during and after exposure (Kinkead 1973).</p>
<p>Subchronic repeated-exposure health effects (≥ 90 days)</p>	<p>8007-45-2/ 65996-89-6</p>	<p>Inhalation effect level = 30 mg/m³ based on histiocytosis of lung tissue in both sexes, increased relative liver weight and decreased volume of packed red cells in males, and decreased eosinophil and monocyte counts in females. Fischer-344 rats (42 animals per sex per concentration) exposed to 0, 30, 140 or 690 mg/m³ coal tar aerosol, 6 hours/day, 5 days/week for 13 weeks.</p> <p><i>All effects were significant (p < 0.05) and concentration-related.</i></p> <p>≥ 30 mg/m³ = Histiocytosis of lung tissue (m/f); increase in relative liver weight (m); decrease in volume of packed red cells (m), eosinophils and monocytes (f).</p> <p>≥ 140 mg/m³ = Decrease in body weight and relative thymus weight (m/f), increase in relative kidney weight (m/f) and relative liver</p>

		<p>weight (f); decrease in hemoglobin (m/f), RBCs and eosinophils (m).</p> <p>690 mg/m³ = Decrease in lymphocytes, total WBCs (m/f), RBCs, volume of packed red cells (f) and monocytes (m), increased triglycerides (m/f), serum cholesterol (m) and reticulocytes (f). Other effects observed at the highest dose were significant, but did not follow a concentration-response trend, including hypocellular bone marrow and decrease of megakaryocytes in bone marrow and spleen (m/f), hepatopathy (m/f), thymus atrophy (m/f), and epithelial hyperplasia, ulcers and chronic inflammation of the cecum (m/f) (Springer et al. 1986).</p>
Subchronic repeated-exposure health effects (≥ 90 days)	8007-45-2/ 65996-89-6	<p>Dermal LOEL = 10 mg of 10% based on comedogenicity. Male Australian albino rabbits (3 animals per dose) exposed to 10 mg of 10, 25 or 100% (0.5, 1.25 or 5 mg/kg bw/day)⁹ crude coal tar applied to the region just exterior to the ear canal, 3 times a week for 15 weeks. None of the control vehicles were comedogenic or carcinogenic when applied up to 20 weeks. Crude coal tar was both comedogenic and carcinogenic at each applied dose (severity grade not reported) (Kligman and Kligman 1994; also cited in ATSDR 2002).</p>
Subchronic repeated-exposure health effects (≥ 90 days)	65996-93-2	<p>Dermal LOAEL = 68 mg/kg bw/day based on early mortality in males. Swiss albino mice (15 animals per sex) exposed to 25 µl of a 9% coal tar pitch solution in benzene (1.7 mg coal tar pitch per treatment; equivalent to 68 mg/kgbw/day for males weighing 25 g, and 85 mg/kg bw/day for females weighing 20 g), applied to 1 square inch (shaved) of the back, twice a week for 31 weeks. 15 males and 15 females were painted with benzene only and served as controls. Animals were sacrificed when moribund or when at advanced cancer stage. At 68 mg/kg bw/day in males (85 mg/kg bw/day in females), decreased mean survival time was observed (31 weeks as compared to 82 weeks for control animals). Skin tumours also observed (Wallcave et al. 1971).</p>

Developmental and reproductive health effects	8007-45-2/ 65996-89-6	<p>Oral developmental LOAEL = 140 mg/kg bw/day based on increases in the proportion of litters with anomalous fetuses, and anomalous fetuses per litter.</p> <p>Oral reproductive LOAEL (female) = 180 mg/kg bw/day based on increases in total resorptions per implants and litters with resorptions occurring during mid- and late-gestation. Pregnant CD rats (16–36 animals per dose) exposed to 0, 90, 140, 180, 370 or 740 mg/kg bw/day coal tar, via gavage, on gestational days 12–16. Developmental and reproductive effects were significant ($p < 0.05$) and dose-related. Initial maternal toxicity observed at 90 mg/kg bw/day; significant mortality observed in dams at highest dose (Hackett et al. 1984).</p>
Developmental and reproductive health effects	8007-45-2/ 65996-89-6	<p>Inhalation reproductive LOAEC (male) = 140 mg/m³ based on increased testis weight (relative to body weight). Fischer-344 rats (42 animals per sex per concentration) exposed to 0, 30, 140 or 690 mg/m³ coal tar aerosol, 6 hours/day, 5 days/week for 13 weeks. Effect was significant ($p < 0.05$) and concentration-related; significant decrease in relative ovary weight observed at the highest concentration (Springer et al. 1986).</p>
Developmental and reproductive health effects	8007-45-2/ 65996-89-6	<p>Inhalation reproductive LOAEC (female) = 660 mg/m³ based on increased incidence of mid- and late-gestational resorptions.</p> <p>Inhalation developmental LOAEC = 660 mg/m³ based on reduced fetal size and weight, as well as increased incidence of litters with reduced ossification. Pregnant Sprague-Dawley rats (23–25 animals per concentration) exposed to 0, 17, 84 or 660 mg/m³ heavy distillate (the highest-boiling material derived from the solvent refined coal-II process), 6 hours/day, on gestational days 12–16. Resorptions were significant ($p < 0.01$ for mid-gestational, and $p < 0.055$ for late-gestational); reduced fetal size and weight were significant ($p < 0.01$); reduced ossification was significant and concentration-related ($p < 0.05$). Significant decrease in</p>

		maternal body weight also observed at the highest concentration (Springer et al. 1982).
Developmental and reproductive health effects	8007-45-2/ 65996-89-6	Dermal developmental and reproductive (female) LOAEL = 500 mg/kg bw/day based on decreased fetal size and weight, increased incidences of small lungs, cleft palate, edema, mid-cranial lesion, dilated ureter and pelvic cavitation, as well as reduced cranial ossification; increased mid- and late-gestational resorptions, decreased number of live fetuses per litter, and decreased placental and uterine weights. Pregnant Sprague-Dawley rats (16–17 animals per dose) and CD-1 mice (7 animals per dose) exposed to 0, 500 or 1500 mg/kg bw/day heavy distillate (high-boiling, coal-derived, complex organic mixture) applied to the shaved back, on gestational days 11–15. Majority of developmental and reproductive effects observed in rats; all effects were significant (p < 0.05). Significant decrease in maternal body weight observed in rats on gestational day 20 for both low- and high-dose exposures (Zangar et al. 1989).
Endpoint	CAS RN	Oral
Carcinogenicity	8007-45-2/ 65996-89-6	Lowest effect level = 100 mg/kg bw/day (0.1%) based on statistically significant and dose-related increased incidence of lung tumours. Female A/J mice (7 weeks of age at beginning of dosing; 30 animals per dose) exposed to doses of 0%, 0.1% or 0.25% (0, 100 or 236 mg/kg bw/day) ^f manufactured gas plant residue in basal gel diet, <i>ad libitum</i> , for 260 days. 0%: Lung tumour incidence = 19% 0.1%: Lung tumour incidence = 70% 0.25%: Lung tumour incidence = 100% (Weyand et al. 1995; also cited in ATSDR 2002)
Carcinogenicity	8007-45-2/ 65996-89-6	Effect level = 200 mg/kg bw/day (0.1%) based on increased incidence of forestomach tumours. Female B6C3F1 mice (5–6 weeks of age at beginning of dosing; 48 animals per dose) exposed to doses of 0%, 0.01%, 0.03%, 0.1%, 0.3%, 0.6% or 1% (0, 20, 63,

		<p>200, 628, 1364 or 2000 mg/kg bw/day)^f coal tar in diet, <i>ad libitum</i>, for 2 years.</p> <p><i>Forestomach tumours</i> (papillomas, squamous cell carcinomas) occurred in all treated groups (tumour incidence in 0.1% and 0.3% groups was 6% and 30%, respectively).</p> <p><i>Small intestine adenocarcinomas</i> occurred in 0.6% and 1.0% groups (tumour incidence: 61% and 88%, respectively; 0% observed in controls).</p> <p>Increased mortality and early mortality observed in higher dose groups (Culp et al. 1996; also cited in ATSDR 2002).</p>
Carcinogenicity	8007-45-2/ 65996-89-6	<p>Effect level = 333 mg/kg bw/day (0.3%) based on statistically significant and dose-related increased incidences of liver, lung and forestomach tumours. Female B6C3F1 mice (5 weeks of age at beginning of dosing; 48 animals per dose) exposed to doses of 0%, 0.01%, 0.03%, 0.1%, 0.3%, 0.6%, 1.0% (0, 12, 33, 117, 333, 739, 1300 mg/kg/day)^f <i>coal tar mixture 1</i> (CAS RN 8007-45-2; composite from seven manufactured gas plant waste sites) or doses of 0%, 0.03%, 0.1%, 0.3% (0, 40, 120, 346 mg/kg/day)^f <i>coal tar mixture 2</i> (8007-45-2; composite from two of seven waste sites plus a third site having a very high B[a]P content) in diet, <i>ad libitum</i>, for 2 years.</p> <p>Coal Tar Mixture 1 results: <i>0 mg/kg bw/day</i>: Range of tumour incidence was 0–4%. <i>Liver neoplasms</i> (hepatocellular adenomas, carcinomas) occurred in all treated groups. A significant dose-related trend was observed, and statistical significance occurred in the 0.3% group (tumour incidence: 31%). <i>Lung neoplasms</i> (alveolar/bronchiolar adenomas, carcinomas) occurred in all treated groups. A significant dose-related trend was observed, and statistical significance occurred in the 0.3%, 0.6% and 1.0% groups (tumour incidence: 57%, 53% and 47%).</p>

		<p><i>Forestomach neoplasms</i> (papillomas, carcinomas) occurred in all treated groups. A highly significant dose-related trend was observed, and statistical significance occurred in the 0.3% and 0.6% groups (tumour incidence: 30% and 33%).</p> <p>Coal Tar Mixture 2 results: <i>0 mg/kg bw/day</i>: Range of tumour incidence was 0–4%. <i>Liver neoplasms</i> (hepatocellular adenomas, carcinomas) occurred in all treated groups. A significant dose-related trend was observed, and statistical significance occurred in the 0.3% group (tumour incidence: 22%). <i>Lung neoplasms</i> (alveolar/bronchiolar adenomas, carcinomas) occurred in all treated groups. A significant dose-related trend was observed, and statistical significance occurred in the 0.1% and 0.3% groups (tumour incidence: 21% and 49%). <i>Forestomach neoplasms</i> (papillomas, carcinomas) occurred in all treated groups. A highly significant dose-related trend was observed, and statistical significance occurred in the 0.3% group (tumour incidence: 30%).</p> <p>Both Coal Tar Mixtures: <i>Small intestine adenocarcinomas</i> were observed, as well as dose-related increases in hemangiosarcomas, histiocytic sarcomas and sarcomas. Increased mortality and early mortality observed in higher dose groups (Culp et al. 1998; also cited in ATSDR 2002).</p>
Endpoint	CAS RN	Dermal (<i>chronic</i>)
Carcinogenicity	8007-45-2/ 65996-89-6	Lowest effect level = 10 mg of 10% (0.5 mg/kg bw/day) based on increased incidence of skin tumours. 10 mg of 10%, 25% or 100% (0.5, 1.25 or 5 mg/kg bw/day) ⁹ of coal tar applied to the region just exterior to the ear canal of male Australian albino rabbit (3 animals per dose), 3 times a week for 15 weeks. None of the control vehicles were comedogenic or carcinogenic when applied

		up to 20 weeks. Skin tumours (papillomas, squamous cell carcinomas, keratoacanthomas, and cutaneous horns) developed, with latency as early as 1–2 weeks following the last treatment at 0.5 mg/kg bw/day (Kligman and Kligman 1994; also cited in ATSDR 2002).
Carcinogenicity	8007-45-2/ 65996-89-6	Effect level = 208 mg/kg bw/day based on skin tumour incidence. Two groups of 30 mice (each with 16 males, 14 females) were exposed to coal tar (as a benzene extract) via a single drop, twice a week for 22 weeks. Cumulative doses of 0.2 and 0.6 g of coal tar after 4 months (275 and 825 mg after 22 weeks; 208 or 625 mg/kg bw/day) ^h . No benzene control group was reported. Tumours were observed after 7 months. <i>208 mg/kg bw/day</i> : skin tumour incidence = 6 of 22 or 27% <i>625 mg/kg bw/day</i> : skin tumour incidence = 8 of 26 or 31% (Gorski 1959).
Endpoint	CAS RN	Dermal (<i>initiation</i>)
Carcinogenicity	8007-45-2/ 65996-89-6	Effect level = 25 mg/kg bw/day based on significantly increased incidence of skin tumours. 50 mg of a 1.5% coal tar ointment (25 mg/kg bw/day ⁱ ; assuming a w/w percentage) applied to the shaved dorsal skin of female CD-1 mice (30 animals), 5 consecutive days/week for 2 weeks. One week later, 0.1% dithranol cream (50 mg per treatment) was applied 3 times a week for 40 weeks. Negative controls not treated with initiator, but subsequently treated with dithranol promoter. Significant increase in mice with papillomas (4 of 27 treated animals versus 0 of 28 control animals) (Phillips and Alldrick 1994).
Carcinogenicity	8007-45-2/ 65996-89-6	Effect level = 833 mg/kg bw based on increased incidence of skin tumours. 25 mg (833 mg/kg bw ⁱ) of each test substance: 1) an industrial coal tar from the National Bureau of Standards, and 2) a coal tar pharmaceutical stock material (20% coal tar in alcohol), having been diluted (1:1) in methylene chloride, applied to the shaved skin of female CD-1 mice (30 animals per group), in 50 µl

		volumes as a single dose. Two weeks later, 50 µl of PMA (0.1 mg/ml in acetone) was applied twice a week for 6 months. Positive controls initiated with 50 µg B[a]P, negative controls initiated with 50 µl MeCl ₂ . Results for both test substances were similar to each other. Latency period of approximately 8 weeks (post-initiation), with 100% tumour incidence at approximately 22 weeks. Authors state that the test substances were similar to the positive control (Wright et al. 1985).
Carcinogenicity	8007-45-2/ 65996-89-6	Effect level = 1333 mg/kg bw based on increased incidence of skin tumours. 200 µl (1333 mg/kg bw ^j) of therapeutic crude coal tar (USP) applied (neat) to the shaved and depilated skin of female SENCAR mice (20 animals) as a single dose. 7 days later, TPA (3.24 nmol administered in 0.2 ml of acetone) was applied twice a week until termination of the experiment. Negative controls treated with acetone. First appearance of tumours (10%) at 6 weeks (post-initiation), with 100% tumour incidence at 11 weeks (Mukhtar et al. 1986b).
Endpoint	CAS RN	Dermal (<i>chronic</i>)
Carcinogenicity	65996-93-2	Effect level = 2 mg/kg bw/day based on increased incidence of skin tumours. 2.0 µl (2 mg/kg bw/day ^k) of coal-tar-based paint coating (<i>Coal Tar E</i> containing 67% coal tar pitch) applied to the shaved dorsal skin of female SENCAR mice (40 animals), once a week for 30 weeks. Negative controls treated with 200 µl mineral spirits, positive controls treated with 100 µg B[a]P. Tumour response for <i>Coal Tar E</i> comparable to that of B[a]P. <i>Mineral spirits</i> : 3 of 40 (8%) mice with papillomas; 0 of 40 mice with carcinomas. <i>Coal Tar E</i> : 33 of 40 (83%) mice with papillomas; 4 of 40 (10%) mice with carcinomas (Robinson et al. 1984).
Carcinogenicity	65996-93-2	Effect level = 50–140 mg/kg bw/day based on skin tumour incidence. 50 µl of a 30–84 mg/ml solution (50–140 mg/kg bw/day ^l) of coal tar pitch volatile (condensed fumes from coal tar) applied to an unspecified area of the skin of male Swiss CD-1 and pigmented C3H/HeJ mice (number of animals not

		provided), twice a week for 78 weeks. Negative controls treated with 50 µl of cyclohexane/acetone (1:1) vehicle. Benign skin tumours (including papillomas, fibromas and epitheliomas) and malignant tumours (including squamous cell carcinomas and fibrosarcomas) were observed. CD-1 mice had a much lower incidence of malignant tumours than C3H/HeJ mice (approximately 5% versus 60%), and latency for CD-1 mice was 47.4–76.5 weeks versus 39.5–56.1 weeks for C3H/HeJ mice (Niemeier et al. 1988).
Carcinogenicity	65996-93-2	Effect level = 68 mg/kg bw/day based on increased incidence of skin tumours in males. 25 µl of a 9% coal tar pitch solution in benzene (1.7 mg coal tar pitch per treatment; equivalent to 68 mg/kg bw/day for males weighing 25 g, and 85 mg/kg bw/day for females weighing 20 g), applied to 1 square inch (shaved) of the back of Swiss albino mice (15 animals per sex), twice a week for 31 weeks. 15 males and 15 females were painted with benzene only and served as controls. Animals were sacrificed when moribund or when at advanced cancer stage. At 68 mg/kg bw/day in males (85 mg/kg bw/day in females), the number of tumour-bearing animals was 53% of 58 or 91.4% (control: 1 of 26 or 3.8%). Skin tumours observed (31 squamous cell carcinomas, 53 papillomatous growths). Early mortality also observed (Wallcave et al. 1971).
Carcinogenicity	65996-93-2	Effect level = 833 mg/kg bw/day based on significantly increased incidence of skin tumours. 25 mg (833 mg/kg bw/day ⁱ) of traditional coal tar pitch, having been dissolved 1:1 in toluene, was applied to the clipped backs of male weaning C3H/HeJ mice (50 animals), twice a week for 80 weeks or until a skin lesion was diagnosed as a papilloma. Negative controls treated with 50 mg of toluene, positive controls treated with 50 mg of 0.1% B[a]P. Skin tumour incidence: 48 of 49 or 98% (malignant: 45 of 49, benign: 3/49), with average latency period of 18

		weeks. Authors state that the test substance was significantly more carcinogenic than the positive control; no mice developed tumours for negative control (Emmett et al. 1981).
Endpoint	CAS RN	Dermal (<i>initiation</i>)
Carcinogenicity	65996-93-2	<p>Lowest effect level = 0.2 mg/kg bw based on increased incidence of skin tumours. 0, 0.2, 0.6, 2.0, 6.0 or 20.0 µl (0, 0.2, 0.6, 2, 6 or 20 mg/kg bw)^k of coal-tar-based paint coatings formulated with varying concentrations of coal tar pitch (<i>Coal tar E</i>: 67%; <i>Coal tar F</i>: 47%; <i>Coal tar G</i>: 37%) applied to the shaved dorsal skin of female SENCAR mice (30 animals per dose) as a single dose (diluted in acetone to give a dosing volume of 0.2 ml). Two weeks later, 1.0 µg of TPA (in 0.2 ml of acetone) was applied 3 times a week for 20 weeks. Negative controls initiated with 0.2 ml acetone. Clear tumour dose-response observed, and <i>Coal Tar E</i> displayed the maximum tumour yield.</p> <p><i>Acetone</i></p> <p><i>0.2 ml</i>: 4 of 23 mice with squamous cell tumours; 4 of 23 mice with squamous cell papillomas; 0 of 23 mice with squamous cell carcinomas.</p> <p><i>Coal Tar E</i></p> <p><i>0.2 µl</i>: 12 of 23 mice with squamous cell tumours; 10 of 23 mice with squamous cell papillomas; 3 of 23 mice with squamous cell carcinomas.</p> <p><i>0.6 µl</i>: 24 of 26 mice with squamous cell tumours; 13 of 26 mice with squamous cell papillomas; 20 of 26 mice with squamous cell carcinomas.</p> <p><i>2.0 µl</i>: 14/18 mice with squamous cell tumours; 10 of 18 mice with squamous cell papillomas; 10 of 18 mice with squamous cell carcinomas.</p> <p><i>6.0 µl</i>: 19 of 20 mice with squamous cell tumours; 14 of 20 mice with squamous cell papillomas; 11 of 20 mice with squamous cell carcinomas.</p> <p><i>20.0 µl</i>: 14 of 14 mice with squamous cell tumours; 10 of 14 mice with squamous cell</p>

		papillomas; 7 of 14 mice with squamous cell carcinomas (Robinson et al. 1984).
Endpoint	CAS RN	Inhalation
Carcinogenicity	65996-93-2	Lowest effect level = 0.5 mg/m³ based on statistically significant increased incidence of lung tumours. Female NMRI/BR mice (newborn; 40 animals per concentration) exposed to concentrations of 0, 0.5 (±0.85) or 2.44 (±0.40) mg/m ³ of coal tar pitch aerosol, generated by pyrolyzing preheated CTP in nitrogen atmosphere at 750–800 °C and diluting with fresh air, 16 hours/day, 5 days/week for 44 weeks. Treatment induced multiple foci of bronchiolo-alveolar hyperplasia in almost all mice (0 of 40, 38 of 40 and 39 of 40, respectively), and squamous metaplasia in 6 of 40 animals at the high-concentration. Statistically significant increases in the incidence of lung adenomas (5 of 40, 40 of 40 and 40 of 40, respectively), lung adenocarcinomas (6 of 40, 10 of 40 and 33 of 40, respectively), and lung squamous cell carcinomas (0 of 40, 0 of 40 and 6 of 40, respectively). In addition, one adenosquamous carcinoma was found in the high-concentration group (Schulte et al. 1994).
Carcinogenicity	65996-93-2	Effect level = 1.1 mg/m³ based on increased incidence of lung tumours. Female Wistar rats (10 weeks of age at beginning of treatment; 72 animals per concentration) exposed to concentrations of 0, 1.1 or 2.6 mg/ m ³ high-temperature coal tar pitch aerosol, generated by heating hard coal tar pitch to 750 °C under nitrogen and diluting with clean air, 17 hours/day, 5 days/week for 10 months (followed by exposure to clean air for 20 months) or for 20 months (followed by clean air for 10 months). Most tumours were benign and malignant keratinizing squamous cell tumours; some broncho-alveolar adenomas and adenocarcinomas were also found. No tumours were observed in other organs. <i>10 month exposure</i> <i>0 mg/m³: 0%</i> <i>1.1 mg/m³: 4.2%</i>

		<p>2.6 mg/m³: 39%</p> <p>20 month exposure</p> <p>0 mg/m³: 0%</p> <p>1.1 mg/m³: 33%</p> <p>2.6 mg/m³: 97.2%</p> <p>Increased mortality observed (particularly in the high-concentration group) due to development of large, multiple tumours in the lung (Heinrich et al. 1994a, 1994b; also cited in ATSDR 2002).</p>
Endpoint	CAS RN	Oral
Genotoxicity – <i>in vivo</i>	8007-45-2/ 65996-89-6	<p>Positive for presence of DNA adducts in the lung, liver, forestomach and small intestine</p> <p>Female B6C3F1 mice (5–6 weeks of age at the beginning of dosing; 4 animals per dose) exposed to doses of 0%, 0.01%, 0.03%, 0.1%, 0.3%, 0.6% or 1.0% (0, 12, 33, 117, 333, 739 or 1300 mg/kg bw/day^f) <i>coal tar mixture 1</i> (CAS RN 8007-45-2; composite from seven manufactured gas plant waste sites) in diet, <i>ad libitum</i>, for 4 weeks. DNA adducts detected by ³²P-post-labelling.</p> <p><i>DNA adducts in the lung</i></p> <p>0% (control): 2.20 ± 0.71 adducts/10⁸ nucleotides</p> <p>0.01%: 2.85 ± 0.48 adducts/10⁸ nucleotides</p> <p>0.03%: 5.80 ± 0.70 adducts/10⁸ nucleotides (p < 0.05)</p> <p>0.1%: 31.2 ± 5.86 adducts/10⁸ nucleotides (p < 0.05)</p> <p>0.3%: 102 ± 5.86 adducts/10⁸ nucleotides (p < 0.05)</p> <p>0.6%: 239 ± 38.8 adducts/10⁸ nucleotides (p < 0.05)</p> <p>1.0%: 227 ± 49.4 adducts/10⁸ nucleotides (p < 0.05)</p> <p><i>DNA adducts in the liver</i></p> <p>0% (control): 5.47 ± 2.48 adducts/10⁸ nucleotides</p> <p>0.01%: 6.18 ± 1.60 adducts/10⁸ nucleotides</p> <p>0.03%: 6.92 ± 1.19 adducts/10⁸ nucleotides</p> <p>0.1%: 15.1 ± 3.45 adducts/10⁸ nucleotides</p>

		<p>0.3%: 43.2 ± 9.15 adducts/10⁸ nucleotides (p < 0.05) 0.6%: 54.4 ± 14.3 adducts/10⁸ nucleotides (p < 0.05) 1.0%: 55.8 ± 20.5 adducts/10⁸ nucleotides (p < 0.05) <i>DNA adducts in the forestomach</i> 0% (control): 6.76 ± 3.00 adducts/10⁸ nucleotides 0.01%: 7.67 ± 5.07 adducts/10⁸ nucleotides 0.03%: 9.49 ± 6.39 adducts/10⁸ nucleotides 0.1%: 9.30 ± 3.92 adducts/10⁸ nucleotides 0.3%: 17.6 ± .41 adducts/10⁸ nucleotides 0.6%: 33.1 ± 13.0 adducts/10⁸ nucleotides (p < 0.05) 1.0%: 48.4 ± 3.98 adducts/10⁸ nucleotides (p < 0.05) <i>DNA adducts in the small intestine</i> 0% (control): 0.53 ± 0.81 adducts/10⁸ nucleotides 0.01%: 2.22 ± 1.02 adducts/10⁸ nucleotides 0.03%, 0.1%: not done 0.3%: 15.4 ± 5.40 adducts/10⁸ nucleotides (p < 0.05) 0.6%: 36.6 ± 23.4 adducts/10⁸ nucleotides (p < 0.05) 1.0%: 11.3 ± 9.12 adducts/10⁸ nucleotides (Culp et al. 2000).</p>
Genotoxicity – <i>in vivo</i>	8007-45-2/ 65996-89-6	<p>Positive for presence of DNA adducts in the lung, liver, forestomach and small intestine Female B6C3F1 mice (5–6 weeks of age at the beginning of dosing; 4 animals per dose) exposed to doses of 0%, 0.03%, 0.1% or 0.3% (0, 40, 120 or 346 mg/kg bw/day^f) <i>coal tar mixture 2</i> (8007-45-2; composite from two of seven waste sites plus a third site having a very high B[a]P content) in diet, <i>ad libitum</i>, for 4 weeks. DNA adducts detected by ³²P-post-labelling. <i>DNA adducts in the lung</i> 0% (control): 2.20 ± 0.71 adducts/10⁸ nucleotides</p>

		<p>0.03%: 8.38 ± 1.08 adducts/10^8 nucleotides ($p < 0.05$)</p> <p>0.1%: 26.2 ± 6.38 adducts/10^8 nucleotides ($p < 0.05$)</p> <p>0.3%: 123 ± 29.8 adducts/10^8 nucleotides ($p < 0.05$)</p> <p><i>DNA adducts in the liver</i></p> <p>0% (control): 5.47 ± 2.48 adducts/10^8 nucleotides</p> <p>0.03%: 10.2 ± 2.22 adducts/10^8 nucleotides</p> <p>0.1%: 17.7 ± 8.41 adducts/10^8 nucleotides ($p < 0.05$)</p> <p>0.3%: 39.9 ± 7.96 adducts/10^8 nucleotides ($p < 0.05$)</p> <p><i>DNA adducts in the forestomach</i></p> <p>0% (control): 6.76 ± 3.00 adducts/10^8 nucleotides</p> <p>0.03%: 13.5 ± 3.84 adducts/10^8 nucleotides</p> <p>0.1%: 9.98 ± 2.95 adducts/10^8 nucleotides</p> <p>0.3%: 25.5 ± 11.0 adducts/10^8 nucleotides ($p < 0.05$)</p> <p><i>DNA adducts in the small intestine</i></p> <p>0% (control): 0.53 ± 0.81 adducts/10^8 nucleotides</p> <p>0.03%, 0.1%: not tested</p> <p>0.3%: 16.7 ± 23.9 adducts/10^8 nucleotides ($p < 0.05$) (Culp et al. 2000).</p>
Genotoxicity – <i>in vivo</i>	8007-45-2/ 65996-89-6	<p>Positive for presence of DNA adducts in the small intestine and forestomach</p> <p>Female B6C3F1 mice (5–6 weeks of age at the beginning of dosing; 4 animals per dose) exposed to doses of 0%, 0.01%, 0.03%, 0.1%, 0.3%, 0.6% or 1.0% (0, 20, 63, 200, 628, 1364 or 2000 mg/kg bw/day^f) coal tar in diet, <i>ad libitum</i>, for 28 days. DNA adducts detected by ³²P-post-labelling.</p> <p><i>Small intestine DNA adduct levels</i></p> <p>Control: 17 ± 25 fmol adduct/mg DNA</p> <p>0.01%: 69 ± 32 fmol adduct/mg DNA</p> <p>0.3%: 461 ± 189 fmol adduct/mg DNA</p> <p>0.6%: 1438 ± 200 fmol adduct/mg DNA</p> <p>1.0%: 529 ± 282 fmol adduct/mg DNA</p> <p><i>Adduct levels in forestomach not reported</i> (Culp et al. 1996; also cited in ATSDR 2002).</p>

Genotoxicity – <i>in vivo</i>	8007-45-2/ 65996-89-6	*[Similar findings observed in: Goldstein et al. 1998; Bordelon et al. 2000; Beland et al. 2005; Culp and Beland 1994; Koganti et al. 2000, 2001; Weyand and Wu 1994, 1995; Weyand et al. 1991, 1994]
Endpoint	CAS RN	Dermal
Genotoxicity – <i>in vivo</i>	8007-45-2/ 65996-89-6	Positive for presence of DNA adducts in the skin and lung Female ICR mice (≥ 3 animals per dose) exposed to 0, 0.48, 1.2 or 3.0 mg (0, 16, 40 or 100 mg/kg bw ⁱ) of crude Manufactured Gas Plant residue-designated Site 4 (MGP-4) applied to 4 cm ² of the shaved back for 24 hours. Dose-related DNA adducts detected in the skin and lungs through ³² P-post-labelling. At the highest dose, the relative adduct labelling (RAL) × 10 ⁹ was 514 in skin. MGP-4 was noted by the authors to cause fewer lung adducts than skin DNA adducts (Cizmas et al. 2004).
Genotoxicity – <i>in vivo</i>	8007-45-2/ 65996-89-6	Positive for presence of DNA adducts in the skin and lung Male Parkes mice (4–6 weeks old at beginning of dosing; 4 animals per group) exposed to 0 or 6 mg (0 or 200 mg/kg bw/day ⁱ) pharmaceutical grade coal tar (20% <i>liquor picis carbonis</i> stock solution in ethanol; dose applied in a 150 µl aliquot) applied to the shaved dorsal skin, twice a week (1st and 4th days; 3 days apart) for up to 5 weeks. One group of four animals was sacrificed weekly, 24 hours after last treatment (5th day); skin and lung samples were removed at sacrifice. An overall rise in DNA adduct levels was observed (values shown graphically in publication); adduct levels for skin were higher than those for the lung (Schoket et al. 1988).
Genotoxicity – <i>in vivo</i>	8007-45-2/ 65996-89-6	Positive for presence of DNA adducts in the skin Male Parkes mice (4–6 weeks old at beginning of dosing; 4 animals per dose) exposed to 0, 6 or 30 mg (0, 200 or 1000 mg/kg bw ⁱ) pharmaceutical grade coal tar (20% <i>liquor picis carbonis</i> stock solution in ethanol; doses applied in 150 µl aliquots)

		<p>applied as a single dose to the shaved dorsal skin. Animals were sacrificed after 24 hours; skin samples taken at sacrifice. A dose-related increase in DNA adduct levels was observed.</p> <p><i>DNA adducts 24 hours after single dose (assuming background adduct levels subtracted, therefore control value is set to zero)</i></p> <p>0 mg/kg bw: 0 fmol adducts/μg DNA 200 mg/kg bw: 0.14 fmol adducts/μg DNA 1000 mg/kg bw: 0.38 fmol adducts/μg DNA</p> <p>DNA</p> <p>Highest dose of coal tar also tested for persistence of DNA adducts. Results shown graphically in publication, but maximum levels were observed after 24 hours, followed by a rapid loss of adducts up to 1 week after treatment, followed by a slower removal of damage over the next 25 days (Schocket et al. 1988).</p>
Genotoxicity – <i>in vivo</i>	8007-45-2/ 65996-89-6	<p>Positive for presence of DNA adducts in the skin</p> <p>Female CD-1 mice (7–8 weeks old at beginning of dosing; 4 animals per group) exposed to 0 or 50 mg of a 1% coal tar ointment (16.7 mg/kg bw/day; assuming a w/w percentage) applied to the shaved dorsal skin, 5 consecutive days/week for 2 weeks. Animals sacrificed 12 days after final treatment. DNA adducts detected in the skin of treated animals; not observed in untreated animals (Phillips and Alldrick 1994).</p>
Genotoxicity – <i>in vivo</i>	8007-45-2/ 65996-89-6	*[Similar findings observed in: Genevois et al. 1996; Hughes et al. 1993; Weyand and Wu 1994; Mukhtar et al. 1986a]
Endpoint	CAS RN	Dermal – therapeutic use
Genotoxicity – <i>in vivo</i>	8007-45-2/ 65996-89-6	<p>Positive for presence of DNA adducts in skin</p> <p>Patients dermally treated with 3%–5% coal tar ointments for ≥ 7 days. Increased incidence of DNA adducts from skin biopsies (Zhang et al. 1990).</p>
Genotoxicity – <i>in vivo</i>	8007-45-2/ 65996-89-6	Positive for presence of DNA adducts in skin

		Patients dermally treated with 3%–10% coal tar for 1 week. Increased incidence of B[a]P-DNA adducts from skin biopsies. Study also found that a mutant myeloperoxidase genotype reduced the formation of B[a]P-DNA adducts (Rojas et al. 2001).
Genotoxicity – <i>in vivo</i>	8007-45-2/ 65996-89-6	Positive for presence of DNA adducts in blood Psoriasis patients dermally treated with 20–100 g coal tar/day. Increased incidence of B[a]P-DNA adducts in blood (Santella et al. 1995).
Genotoxicity – <i>in vivo</i>	8007-45-2/ 65996-89-6	Positive for presence of DNA adducts in lymphocytes Patients dermally treated with 50% coal tar paste, 2% coal tar ointment, or a combination of pure coal tar and 2% ointment for 3–17 days. Increased incidence of B[a]P-DNA adducts in lymphocytes, determined through ELISA. Adduct levels were noted to be higher during therapy than 2–5 months after treatment (Pavanello and Levis 1994).
Genotoxicity – <i>in vivo</i>	8007-45-2/ 65996-89-6	Negative for presence of DNA adducts in lymphocytes Psoriasis patients dermally treated with 50% coal tar paste for 8 days. No difference observed in the number of B[a]P-DNA adducts in the peripheral blood lymphocytes of patients versus levels in untreated individuals. Furthermore, no statistical difference in DNA adduct levels were noted in patients before, during or 16 days after treatment (Pavanello and Levis 1994).
Genotoxicity – <i>in vivo</i>	8007-45-2/ 65996-89-6	Negative for presence of DNA adducts in lymphocytes Male psoriasis patients (4 people in total) dermally treated with 50% coal tar paste for 4–10 days. No difference observed in the number of B[a]P-DNA adducts of patients versus levels in untreated individuals. Furthermore, no statistical difference in DNA adduct levels were noted in patients before and after treatment (Pavanello and Levis 1992).
Genotoxicity – <i>in vivo</i>	8007-45-2/ 65996-89-6	*[Similar findings observed in: Godschalk et al. 1998, 2001].

Endpoint	CAS RN	DNA Adducts
Genotoxicity – <i>in vitro</i>	8007-45-2/ 65996-89-6	Positive for induction of DNA adducts in calf thymus DNA with S9 exogenous metabolic activation (no data without S9). Exposure to coal tar (1, 2, 5 or 10 µg dissolved in 5 µl DMSO; mixture of three samples obtained from coal gasification sites). Highest adducts occurred from exposure to 1 µg coal tar (Koganti et al. 2000; also cited in ATSDR 2002).
Genotoxicity – <i>in vitro</i>	8007-45-2/ 65996-89-6	Positive for induction of DNA adducts in human mammary epithelial cells without S9 exogenous metabolic activation (no data with S9) (Leadon et al. 1995).
Genotoxicity – <i>in vitro</i>	8007-45-2/ 65996-89-6	Positive for induction of DNA adducts in human mammary carcinoma derived (MCF-7) cells. Exposure to 400 µg of an artificial coal tar mixture (50 µl of an 8 mg/ml mixture composed of weak and non-carcinogenic coal tar components prepared using the proportions present in SRM 1597 [the standard reference mixture for coal tar]) continuously for 24 or 48 hours. The artificial coal tar mixture induced low, but detectable levels, of PAH-DNA adducts compared to B[a]P alone (average of 9 times less adducts elicited by the coal tar mixture than B[a]P). DNA adducts were identified through ³³ P-post-labelling (Mahadevan et al. 2004).
Genotoxicity – <i>in vitro</i>	8007-45-2/ 65996-89-6	Positive for induction of DNA adducts in human mammary carcinoma-derived (MCF-7) cells. Exposure to 400 µg of a standardized coal tar mixture (SRM 1597; 95.8±5.8 mg B[a]P/kg or 82.9±5.3 µg/ml) continuously for 6, 12, 24, 48, 72, 96, 120, 144, 168 or 192 hours. Maximum level of DNA adducts formed in treated cells was observed to be 11.6 pmol/mg DNA detected at 144 hours and 10 pmol/mg DNA detected at 72 hours (two different experiments). Co-treatment of SRM 1597 with other PAHs (B[a]P or dibenzo[a,]pyrene) resulted in significant inhibition of PAH-DNA adduct formation compared to treatment with these PAHs alone. DNA adducts were identified through ³³ P-post-labelling (Mahadevan et al. 2005).

Genotoxicity – <i>in vitro</i>	8007-45-2/ 65996-89-6	Positive for induction of DNA adducts in V79 Chinese hamster lung cells, expressing human CYP1A1 or CYP1B1. Exposure to 400 µg (20 µg/ml) of a standardized coal tar mixture (SRM 1597; 95.8±5.8 mg B[a]P/kg or 82.9±5.3 µg/ml) continuously for 6, 12, 24, 48 or 72 hours. SRM 1597-mediated adducts only observed in CYP1A1-expressing cells. Co-treatment of SRM 1597 with other PAHs (B[a]P or dibenzo[a,l]pyrene) resulted in a decrease of PAH-DNA adduct formation compared to treatment with these PAHs alone. DNA adducts were identified through ³³ P-post-labelling (Mahadevan et al. 2007).
Endpoint	CAS RN	Mutagenicity – therapeutic coal tar
Genotoxicity – <i>in vitro</i>	8007-45-2/ 65996-89-6	Positive in <i>Salmonella typhimurium</i> TA98, TA100 and TA1538 with S9 exogenous metabolic activation (no data without S9), using the Ames assay. Four therapeutic coal tar preparations (Zetar® Emulsion, Estar®, Lavatar, Coal Tar Solution USP) tested at concentrations of 10, 25, 50, 100, 150 or 200 µg/plate (4–6 plates per concentration). CIR (2008) reported that all of the coal tar preparations were mutagenic, and a significant increase in <i>his+</i> revertants was observed in all three strains, but TA98 was the most sensitive. <i>Mutagenic potency for TA98 (revertant colonies/µg test substance):</i> Zetar® Emulsion: 7.0 ± 0.6 Estar®: 3.8 ± 0.6 Lavatar: 2.0 ± 0.2 Coal tar solution (USP): 1.4 ± 0.1 (Saperstein and Wheeler 1979; also cited in CIR 2008 and IARC 1985).
Genotoxicity – <i>in vitro</i>	8007-45-2/ 65996-89-6	Positive in <i>S. typhimurium</i> TA98 and TA100 with S9 exogenous metabolic activation (negative without S9), using the Ames assay. Coal tar preparation (therapeutic shampoo) tested at concentrations of 1, 10, 100 or 500 µg coal tar in 100 µl DMSO. Two-fold increase over the spontaneous revertant level observed following exposure to 10 µg/plate on TA98 and 16 µg/plate on TA100 (Clonfero et al. 1986).

Endpoint	CAS RN	Mutagenicity – <i>crude coal tar</i>
Genotoxicity – <i>in vitro</i>	8007-45-2/ 65996-89-6	Positive in <i>S. typhimurium</i> TA98 and TA100 with exogenous metabolic activation (Aroclor-induced male Wistar rat liver S9; negative without S9), using the taped plate assay where coal tar was heated* to 37 °C and the cells exposed to the vapour. Coal tar tested at concentrations of 25, 50, 100, 200 or 500 µg/plate (3 plates per concentration) (Bos et al. 1985; also cited in ATSDR 2002).
Genotoxicity – <i>in vitro</i>	8007-45-2/ 65996-89-6	Positive in <i>S. typhimurium</i> TA98 with exogenous metabolic activation (Aroclor-induced and non-induced male Wistar rat liver S9, 5 human kidney transplant donor S9; no data without S9), using the Ames assay. High-temperature coal tar (CAS RN 65996-89-6) tested at concentrations of 10, 20, 30, 40, 50, 60 or 70 µg/plate (7 plates per concentration). Even the less active kidney donors (4 and 5) showed a 3-fold increase of revertant colonies at 70 µg/plate compared to the background values. <i>Mutagenic potencies for TA98 (revertant colonies/µg coal tar)</i> <i>Kidney donor 1:</i> 3.99 <i>Kidney donor 2:</i> 1.85 <i>Kidney donor 3:</i> 1.40 <i>Kidney donor 4:</i> 0.69 <i>Kidney donor 5:</i> 0.76 <i>Non-induced rat:</i> 7.81 <i>Aroclor-induced rat:</i> 19.5 (Jongeneelen et al. 1988).
Genotoxicity – <i>in vitro</i>	8007-45-2/ 65996-89-6	Positive in <i>S. typhimurium</i> strains TA98, TA100 and TA1538 with exogenous metabolic activation (Aroclor 1254-induced male Sprague-Dawley rat liver S9; negative without S9 in the aforementioned strains and in TA1535), using the Ames assay. Coal gasification tar product diluted in DMSO tested at concentrations of 10, 100 or 1000 µg/plate ^m . <i>Mutagenic potencies (revertant colonies/µg coal gasification tar product)</i> <i>TA98:</i> 6.8 <i>TA100:</i> 6.5 <i>TA1538:</i> 11.2 (Schoeny et al. 1981).

Genotoxicity – <i>in vitro</i>	8007-45-2/ 65996-89-6	*[Similar findings observed in: Fysh et al. 1980; Sarto et al. 1989; Agurell and Stensman 1992; Baranski et al. 1992; Bos et al. 1987; Donnelly et al. 1993, 1996; Mayura et al. 1999]
Endpoint	CAS RN	Mutagenicity – crude coal tar
Genotoxicity – <i>in vitro</i>	65996-89-6	Positive in <i>S. typhimurium</i> TA1537, TA1538, TA98 and TA100 with S9 exogenous metabolic activation (negative without S9 in the aforementioned strains and in TA1535), using the Ames assay. Four coal-tar-based paint coatings formulated with varying concentrations of coal tar pitch (<i>Coal tar E</i> : 67%; <i>Coal tar F</i> : 47%; <i>Coal tar G</i> : 37%; <i>Coal tar H</i> : 39%) were tested at concentrations of 0.005, 0.01, 0.1, 1.0, 5.0 or 10 µl/plate. All test substances elicited mutagenic activity, with the highest mutagenic responses observed on TA98 and TA100 (Robinson et al. 1984).
Genotoxicity – <i>in vitro</i>	65996-89-6	Positive in <i>S. typhimurium</i> TA1537, TA98 and TA100 with S9 exogenous metabolic activation (negative without S9), using the Ames assay. DMSO extract of coal tar pitch [<i>Class 7.3: Unspecified or other coal tar pitches</i> ; Barrett M-30] was tested (Rao et al. 1979; IARC 1985).
Genotoxicity – <i>in vitro</i>	65996-89-6	Positive in <i>S. typhimurium</i> TA1537, TA1538, TA98 and TA100 with S9 exogenous metabolic activation (negative without S9 in the aforementioned strains and in TA1535), using the Ames assay. Dichloromethane extract of emissions from a roofing tar pot was tested. IARC (1985) considers the test substance to be <i>Class 7.3: Unspecified or other coal tar pitches</i> (Claxton and Huisingsh 1980 and Nesnow and Lewtas 1981; IARC 1985).
Genotoxicity – <i>in vitro</i>	65996-89-6	Positive in <i>S. typhimurium</i> TA100 with and without S9 exogenous metabolic activation and in TA98 with activation (negative without S9), using the Ames assay. Coal tar pitch was tested at concentrations of 0.05, 0.25, 2.5 or 5 mg/plate) (Solorzano et al. 1993).
Genotoxicity – <i>in vitro</i>	65996-89-6	*[Similar findings observed in Kesik and Janik-Spiechowicz 1997]

- ^a LD₅₀, median lethal dose; LOAEC, lowest observed adverse effect concentration; LOAEL, lowest observed adverse effect level; LOEL, lowest observed effect level; NOAEL, no observed adverse effect level.
- ^b Dose calculated assuming the standard coal tar solution (USP) used consisted of 20% coal tar (w/v) (i.e., 20 g/100 ml) (United States Pharmacopoeia USP30-NF25, p. 1817).
- ^c Dose calculated based on average weight of Canadian adult (70 kg) (Health Canada 1994).
- ^d Dose calculated based on average weight of neonatal rats being 9.80 ± 2.13 g; obtained from averaging the two means of the highest and lowest birth weights in a laboratory experiment (9.14 ± 0.97 g and 10.45 ± 1.16 g) (Himpel et al. 2006).
- ^e Doses calculated using reference value for body weight in pigs (80 kg) (Health Canada 1994).
- ^f Converted dose(s) provided in ATSDR 2002.
- ^g Doses calculated using the value provided for average weight of rabbits (2 kg) (Kligman and Kligman 1994).
- ^h Doses calculated based on the assumption that animals were treated for 22 weeks, that the 4-month “cumulative dose period” consisted of 16 weeks, and that the average cumulative applied amount of the test substance would increase in a direct, linear fashion (Gorski 1959, as cited in European Commission 2008 and IARC 1985). Doses converted using reference value for body weight in mice (0.03 kg) (Health Canada 1994).
- ⁱ Dose calculated using reference value for body weight in mice (0.03 kg) (Health Canada 1994).
- ^j Dose calculated assuming the standard coal tar solution (USP) used consisted of 20% coal tar (w/v) (i.e., 20 g/100 ml) (United States Pharmacopoeia USP30-NF25, p. 1817) and a reference value for body weight in mice of 0.03 kg (Health Canada 1994).
- ^k Dose(s) calculated using the lowest stated substance density of 30 mg/ml for coal tar pitch (Niemeier et al. 1988 as cited in ATSDR 2002), as well as the reference value for body weight in mice (0.03 kg) (Health Canada 1994).
- ^l Dose range calculated using the given substance density range of 30–84 mg/ml for coal tar pitch (Niemeier et al. 1988, as cited in ATSDR 2002), as well as the reference value for body weight in mice (0.03 kg) (Health Canada 1994).
- ^m Assuming the 3 log concentration range consisted of the stock concentration of 10 mg/ml, as well as 1 and 0.1 mg/ml (Schoeny et al. 1981).