

**Draft Screening Assessment
Petroleum Sector Stream Approach**

Coal Tars and Their Distillates

**Environment Canada
Health Canada**

2015

Synopsis

Pursuant to section 68 and 74 of the *Canadian Environmental Protection Act, 1999* (CEPA 1999), the Ministers of the Environment and of Health have conducted a screening assessment of coal tars and their distillates, including the following substances:

CAS RN ^a	DSL ^b name
8007-45-2	Tar, coal
65996-82-9 ^c	Tar oils, coal
65996-91-0 ^c	Distillates (coal tar), upper
65996-90-9	Tar, coal, low-temperature
65996-89-6 ^c	Tar, coal, high-temperature
65996-93-2	Pitch, coal tar, high-temperature

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^b DSL, *Domestic Substances List*.

^c This substance was not identified under subsection 73(1) of CEPA 1999 but was included in this assessment as it was considered a priority.

Coal tars are the condensation products obtained by cooling, to approximately ambient temperature, the gas evolved in the destructive distillation (pyrolysis) of coal that occurs at integrated steel mills and are 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 refiner, and include both the fractions obtained from the distillation tower as well as the residue (pitch) remaining following distillation. Coal tars and their distillates are considered to be 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.

During the categorization exercise, coal tars and their distillates under six Chemical Abstracts Service Numbers (CAS RN) 8007-45-2 (Tar, coal), 65996-90-9 (Tar, coal, low-temperature), 65996-89-6 (Tar, coal, high-temperature) 65996-93-2 (Pitch, coal tar, high-temperature), 65996-82-9 (Tar oils, coal) and 65996-91-0 (Distillates (coal tar), upper) were identified as priorities for assessment, as they met the categorization criteria under subsection 73(1) of the *Canadian Environmental Protection Act, 1999* (CEPA 1999) and/or were considered as a priority based on other human health concerns. Data obtained on these six coal tars and their distillates were used to assess the risk from all coal tars and their distillates as defined above. As such, the conclusions of this assessment are considered to cover coal tars and their distillates including but not limited to the six priority CAS RNs.

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. 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.

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 of coal tars are produced annually in Canada; from this the coal tar distillates are produced, of which an estimated 82 to 100 kilotonnes per year is coal tar pitch and an unknown quantity is coal tar oils and coal tar upper distillates.

This screening assessment considers both the risk from releases at facilities and their associated processes, which captures all coal tars and their distillates released by the integrated steel and/or coal tar refining facilities, as well as risks from releases of the substances during their transportation and/or use.

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 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.

Quantitative analyses comparing predicted environmental concentrations of PAHs in soil, resulting from releases to air of coal tars and their distillates, from the processing, storage and handling of these substances at a coal tar refining facility, with no-effect levels for PAHs from the Canadian Soil Quality Guidelines, determined that the concentrations of coal tars and their distillates in soil are likely to exceed levels that elicit adverse effects in organisms in the vicinity of such facilities. In addition, releases of coal tar substances to water and sediment from the application and use of coal tar-based pavement sealants are likely to exceed levels that elicit adverse effects in organisms based on estimated releases of PAHs.

Considering all lines of evidence presented in this draft screening assessment, there is a risk of harm to organisms, but not to the broader integrity of the environment, from releases of coal tars and their distillates. It is proposed to conclude that coal tars and their distillates meet the criteria under paragraph 64(a) of CEPA 1999, 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 proposed to conclude that coal tars and their distillates do not meet the criteria under paragraph 64(b) of CEPA 1999, 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 limited general population exposure to these high-hazard volatile constituents of coal tars and their distillates in the vicinity of coal tar producers and refiners. 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 estimates. Accordingly, it is proposed to conclude that coal tars and their distillates 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 proposed to conclude that coal tars and their distillates meet one or more criteria as set out in section 64 of CEPA 1999.

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1. Introduction

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

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

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

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

In order to conduct the screening assessments, each high-priority petroleum substance was placed into one of five categories ("Streams") depending on its production and uses in Canada:

Stream 0: substances not produced by the petroleum sector;

Stream 1: site-restricted substances, which are substances that are not expected to be transported off refinery, upgrader or natural gas processing facility sites;¹

Stream 2: industry-restricted substances, which are substances that may leave a petroleum-sector facility and may be transported to other industrial facilities (for example, for use as a feedstock, fuel or blending component), but that do not reach the public market in the form originally acquired;

Stream 3: substances that are primarily used by industries and consumers as fuels;

¹ For the purposes of the screening assessment of PSSA substances, a "site" is defined as the boundaries of the property where a facility is located.

Stream 4: substances that may be present in products available to the consumer. An analysis of the available data determined that the six coal-tar-based substances should be evaluated under Stream 0, as described above. These six substances are being included in the PSSA because they arise from processes that are similar to those used in the petroleum sector and are considered to be UVCBs.

During the categorization exercise, six coal tars and their distillates under Chemical Abstracts Service Numbers (CAS RN) 8007-45-2 (Tar, coal), 65996-90-9 (Tar, coal, low-temperature), 65996-89-6 (Tar, coal, high-temperature), 65996-93-2 (Pitch, coal tar, high-temperature), 65996-82-9 (Tar oils, coal) and 65996-91-0 (Distillates (coal tar), upper) were identified as priorities for assessment, as they met the categorization criteria under subsection 73(1) of the *Canadian Environmental Protection Act, 1999* (CEPA 1999) and/or were considered as a priority based on other human health concerns.

Screening assessments focus on information critical to determining whether a substance meets the criteria set out in section 64 of CEPA 1999. Screening assessments examine scientific information and develop conclusions by incorporating a weight-of-evidence approach and precaution.²

This screening assessment encompasses all coal tars and their distillates as described in the Substance Identity and Uses sections. Included in this screening assessment is the consideration of information on chemical properties, uses, exposure and effects, including information provided by stakeholders for CAS RNs 8007-45-2, 65996-90-9, 65996-89-6, 65996-93-2, 65996-82-9 and 65996-91-0. Data relevant to the screening assessment of these substances were identified in original literature, review and assessment documents, and in stakeholder research reports and from recent literature searches, up to January 2014 for both the environmental and the health effects sections. Key studies were critically evaluated and modelling results were used to inform proposed 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

² A determination of whether one or more of the criteria under section 64 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 the use of consumer products. A conclusion under CEPA 1999 is not relevant to, nor does it preclude, an assessment against the hazard criteria specified in the *Controlled Products Regulations*, which are part of the regulatory framework for the Workplace Hazardous Materials Information System for products intended for workplace use. Similarly, a conclusion based on the criteria contained in section 64 of CEPA 1999 does not preclude actions being undertaken under other sections of CEPA 1999 or other acts.

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 upon which the proposed conclusion is based.

This screening assessment was prepared by officials in the Existing Substances Programs at Health Canada and Environment Canada, and incorporates input from other programs within these departments. The human health and ecological portions of this assessment have undergone external written peer review/consultation. Comments on the technical portions relevant to human health were received from Dr. Spencer Williams, Baylor University, Dr. Miriam Diamond, 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, University of Toronto, and from Dr. Geoff Granville, GCGranville Consulting Corp. While external comments were taken into consideration, the final content and outcome of the screening assessment remain the responsibility of Health Canada and Environment Canada.

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

2. Substance Identity

This assessment addresses on coal tars and their distillates.

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) that occurs at integrated steel mills and are 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 refiner, and include both the fractions obtained from the distillation tower as well as the residue (pitch) remaining 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 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 process. Given their complex and variable compositions, they could not practicably be formed by simply combining individual constituents.

Six coal tars and distillates were identified as priorities during the categorization exercise, and for which information was obtained from stakeholders. For the purposes of this document, the following common names will be used for these six substances:

- CAS RN 8007-45-2, coal tar
- CAS RN 65996-90-9, low-temperature coal tar
- CAS RN 65996-89-6, high-temperature coal tar
- CAS RN 65996-82-9, coal tar oils
- CAS RN 65996-91-0, coal tar upper distillates
- CAS RN 65996-93-2, high-temperature coal tar pitch or HTCTP

The plural term, 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 (less than 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 greater than 700°C (European Commission 1976; NCI 2010). Coal tar (CAS RN 8007-45-2) is a broader name that can include both low- and high-temperature coal tars.

Coal tar oils (CAS RN 65996-82-9), coal tar upper distillates (CAS RN 65996-91-0) and HTCTP (CAS RN 65996-93-2) are distilled from high-temperature coal tar (Blümer and Sutton 1998; Ruetgers 2013a – see Figures 1 and 2 of Environment Canada 2015a). Coal tar oils have an approximate distillation range of 130–250°C, while the upper distillates have an approximate distillation range of 220–450°C (NCI 2010). HTCTP (CAS RN 65996-93-2) is the solid distillation residue and has an approximate softening point of 30–180°C (NCI 2010).

The composition and properties of coal tars depend upon 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 1 summarizes the main components of the six priority coal tars and distillates.

CAS RN	Substance	Composition
8007-45-2	Coal tar	Complex combination of aromatic hydrocarbons, phenolic compounds, nitrogen bases and thiophene.
65996-90-9	Low-temperature coal tar	Composed primarily of polycyclic aromatic hydrocarbons, phenolic compounds, aromatic nitrogen bases and their alkyl derivatives.
65996-89-6	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	Coal tar oils	Composed primarily of naphthalene, alkylnaphthalenes, phenolic compounds and aromatic nitrogen bases.
65996-91-0	Coal tar upper distillates	Composed primarily of three- to four-membered polycyclic aromatic hydrocarbons and other hydrocarbons.
65996-93-2	High-temperature coal tar pitch	Composed primarily of a complex combination of three- or more-membered polycyclic aromatic hydrocarbons.

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 (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 (Ruetgers 2013a), are expected to contain only one PAH (i.e., naphthalene), as their boiling point range of

168–202°C (Table 3) 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 (Ruetgers 2013a), contain the following substances based on their boiling point range of 307–365°C (Table 4): 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 (e.g., 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 2015b).

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, slightly alkaline and 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 2 through 5. 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 is a source of uncertainty in this assessment.

Low-temperature coal tar has less polyaromatic character than high-temperature coal tar, as it contains higher proportions of phenolic substances, such as cresols, xylenols and high boiling point tar acids (Environment Canada 2015b).

Table 1. Estimated physical and chemical properties of coal tars (CAS RNs 8007-45-2, 65996-90-9 and 65996-89-6) (R4CC 2011; ECHA 2013)¹

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	

¹ Based on read-across from CAS RN 65996-89-6

Table 2. Estimated physical and chemical properties of coal tar oils (CAS RN 65996-82-9) (R4CC 2011; ECHA 2013)¹

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	

Based on read-across from related CAS RN 84650-03-3

Table 3. Estimated physical and chemical properties of coal tar upper distillates (CAS RN 65996-91-0) (R4CC 2011; ECHA 2013; NCI 2010)¹

Property	Value	Temperature (°C)
Physical state	Solid depending on composition	20
Melting point (°C)	92	
Boiling point (°C)	307 to 365	
Distillation range (°C)	220 to 450	
Density (g/cm ³)	1.11	20
Vapour pressure (Pa)	1.1	20
Vapour pressure (Pa)	10.1	50
Vapour pressure (Pa)	174	100

Water solubility (mg/L)	0.518	20
Flash point (°C)	140	
Auto flammability (°C)	> 540	

¹Based on read-across from related CAS RN 90640-80-5

Table 5. Physical and chemical properties of HTCTP (CAS RN 65996-93-2) (ECHA 2013; 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	

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 2015b). 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 have been 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 (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). Additionally, mixture components, which are normally solid under environmental conditions, may have lower melting points (and therefore be in a liquid state) and increased vapour pressure and water solubility (Banerjee 1984). 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 152–178 g/mol, and high-molecular-weight (HMW) PAHs being those with four to seven rings and molecular weights in the range of 202–278 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 constituents, 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 pressures, 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. Both the vapour pressure and the water solubility decrease, and the partition coefficients increase with the addition of benzene rings or increased alkylation to the molecule (e.g., pyridine, quinoline, carbazole and acridine).

The physical-chemical properties of major PAH and non-PAH coal tar constituents are available in Environment Canada (2015b). Throughout this screening assessment, where information specific to the coal tar product is not available, information relating to its major components, such as 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 (CCPA 2010). 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 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). Low-temperature coal tar (CAS RN 65996-90-9) has not been reported to be

manufactured in or imported into Canada in 2011, as further described below, and no information was found on its use in Canada.

An estimated 165 to 220 kilotonnes (kT) of coal tar are produced and refined annually in Canada (Sutton 2008). 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 leads directly to changes in the production of coke and coal tars as by-products. Distillation of the 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 Sutton's (2008) estimate of Canadian coal tar production.

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.

Information on the manufacturing 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 under section 71 of CEPA 1999 (Canada, Dept. of the Environment 2012). Canadian manufacture and import of coal tar oils (CAS RN 65996-82-9) was reported by steel mills and coal tar refiners in the range of 100 to 1000 kT in 2011. No manufacture or import of low-temperature coal tar was reported. Coal tar upper distillates (CAS RN 65996-91-0) were reported to be imported, but not manufactured produced by coal tar refiners in the range of 1–100 kT in 2011.

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% of light oil, 2% of carbolic oil, 10% of naphthalene oil, 10% of wash oil, 10% of anthracene oil, 12% of base oil and 50% of pitch (European Commission 2008). Note that coal tar oils include tar acid oil and naphthalene oil, and that coal tar upper distillates include wash oil and heavy aromatic oil (Environment Canada 2015a). Coal tar oil (CAS RN 65996-82-9) and coal tar upper distillates (CAS RN 65996-91-0) are used as feedstocks for industrial processes. Coal tar oil (CAS RN 65996-82-9) is used in organics recovery and to produce naphthalene as well as naphthalene sulphonates for use in superplasticizers (Ruetgers 2013a). Coal tar upper distillates are 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 can also be used as a binder in graphite electrodes, as an adhesive/binder in clay pigeons and briquettes, to strengthen and impregnate refractories (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). Use of coal tar pitch for pavement and roofing applications has been decreasing, as it is replaced by asphalt sealants that have a lower PAH content (European Commission 2008). Coal tar pitch can also be used as fuel in blast furnaces of the steel industry and in surface coatings for industrial applications, such as pipe linings and harsh climate protection (European Commission 2008).

Coal tar pitch may be used in roofing systems for buildings with flat roofs in Canada (Conestoga 2014). One HTCTP-containing roofing product material safety data sheet was identified, which states that the product contains 100% HTCTP (Koppers Inc. 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 (2014 e-mail from CRCA to Products Division, Chemicals Sector Directorate, Environment Canada, unreferenced).

Coal-tar-pitch-based epoxy coatings, containing 10–30% w/w of 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 coating 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, 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 because their use is limited to specialized industrial applications.

Coal tar-based pavement sealants (CTPS) constitute one of several types of pavement sealants and may contain coal tars and/or their distillates (Ruetgers 2013a, EHS 2010, Pavement Rejuvenation International 2014, Neyra Industries Inc. 2011, 2014). CTPS are available in the Canadian retail market and have 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 ± 50% tonnes/year of CTPS are sold in Canada by a small number of companies, mostly in the retail sector (EHS 2010).

CTPS generally contain 15 to 30% coal tar pitch emulsified in water (EHS 2010). The coal tar pitch in CTPS contains approximately 70 000 mg/kg dry wt. for the sum of the 16 U.S. EPA priority PAHs (Scoggins et al. 2009).

Diamond Environmental Group (2011) found that 23% of 92 driveways and parking lots sampled 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.

None of the six priority coal tars and distillates 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 remaining priority coal tars and their distillates identified during categorization 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).

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 temperature; thus, there is potential for release of coal tars or their components from relief valves and 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. Coal tar oil, upper distillates and pitch are formed at this facility 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 (Ruetgers 2013b). 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 leave the coal tar processing facility and are 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 also in naphthalene refining and the production of naphthalene sulphonates (superplasticizers) (Ruetgers 2013a). Coal tar upper distillates are used to produce creosote and carbon black (Environment Canada 2015a). 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 being considered, 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 1–10% coal tar (Health Canada 2006), which limits 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 as a binder in graphite electrodes (CHEMINFO 2010), 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 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, as identified by CHEMINFO (2010), 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 using 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 very low (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 were obtained for the province of Ontario, where the only coal tar refiner in Canada and all the Canadian integrated steel mills are located, for the five years from 2008 to 2012 (Ontario 2013). The following spills were reported: a total of 16 spills of coal tars and their distillates (average of 3.2 spills/year), all at industrial sites, for a reported total of 39 862 L over five years (average of 7972 L/year). All of the industrial spills were to land, except one to air, albeit one 115 L spill of wash oil was reported as spilled on the pier at a port.

There were also a total of 29 spills (average of about 6 spills/year) of tar, tar and water mixture and driveway sealer, totalling 22 556 L over five years (average of about 4500 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 a fuel for heating and lighting; coal tars therefore exist as contaminants at many former coal gasification sites in Canada. A national inventory performed 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 (Montréal), 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

When coal tar or 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 weathering 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.

As noted previously (see Physical and Chemical Properties section), 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 petroleum hydrocarbons.

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 2015b) 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 168–202°C (Table 3), are expected to contain the representative structures naphthalene, phenols, cresols and xlenols. The coal tar oils are liquid at ambient temperatures, have a density slightly less than water (0.99 g/cm³), a low to moderate water solubility range of 2–60 mg/L and a high vapour pressure of 1700 Pa (Table 3). 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 not heavily sorbed to sediment. They have moderate vapour pressures, meaning that they will volatilize to some extent.

Based on the boiling point range of 307–365°C (Table 4), 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 4), have a density slightly greater than that of water (1.11 g/cm³), a moderate vapour pressure of 1.1 Pa and low water solubility of 0.51 mg/L (Table 4), 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. They mostly have high log K_{oc} values, meaning that they will tend to sorb to organic matter, such as that contained in soil or sediment.

The distillate products, 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 4.8–6.3) (Appendix 3 of Environment Canada 2015b) indicate that when released into water, these substances will readily sorb to suspended solids and bed sediment. Based on 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 0.00026–0.26 mg/L at 25°C; Appendix 3 of Environment Canada 2015b), 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 the 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 (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 constant values (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 a suite of representative structures is characterized based on empirical and/or modelled data. All of the representative structures are discussed within this section, though 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 for the substance. For example, tar oils will contain a higher proportion of LMW components, such as monoaromatics and LMW PAHs, while the upper distillates and coal tar pitch will contain a low proportion of LMW components and high proportions of HMW components, such as PAHs and heterocyclic structures.

The following sections provide a summary of the empirical and modelled data for persistence and bioaccumulation. More detailed descriptions of the empirical data, as well as tabulated modelled data, are available in Environment Canada (2015b).

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 the supporting document (Environment Canada 2015b).

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 (Environment Canada 2015b) and have atmospheric half-lives of less than 2 days. Exceptions are benzene, toluene, ethylbenzene, pyridine and dibenzofuran, which have

estimated atmospheric half-lives greater than 2 days (Environment Canada 2015b), and thus may undergo long-range atmospheric transport to regions remote from their source. As well, many three- to six-ringed PAHs sorb to particulate matter in the atmosphere and thus undergo long-range atmospheric transport (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 2015b). 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 diet, which predominates for substances with log K_{ow} greater than about 4.5 (Arnot and Gobas 2003).

In addition to fish BCF and BAF data, bioaccumulation data for aquatic invertebrate species were also examined. Biota-sediment/soil accumulation factors (BSAFs), trophic magnification factors (TMFs) and biomagnification factors (BMFs) were also considered in characterizing bioaccumulation potential.

Empirical and modelled bioaccumulation data for major coal tar components, as well as the weighing of information, can be found in Environment Canada 2015b.

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 HMW 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, benzo[ghi]perylene and dibenz[a,h]anthracene (Environment Canada 2015b). Non-PAH coal tar components, including monocyclic aromatic hydrocarbons, phenolics and heterocyclics, are not highly bioaccumulative (Environment Canada 2015b). 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 the diet (Environment Canada 2015b). 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 one, which is consistent with evidence for efficient metabolism in these organisms.

It is possible that BSAFs will be greater than one 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

Only limited ecotoxicity data are available for coal tar and coal tar pitch, and no information was found on the toxicity of tar oils and upper distillates. In addition, some limited data are available for coal tar-based pavement sealants.

9.1.1 Coal tar studies

The acute toxicity of coal tar and coal tar pitch were 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 7.33–12.1 mg/L (nominal), and 48-hour LC₅₀ values for the water flea were 4.44–11.2 mg/L (nominal). Although 90–100% 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 0.13–0.3%). 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 early life stages of the sturgeon, 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 produced 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 compared to those in sediments collected at nearby reference sites, while *Daphnia* and midge survival was reduced by 40–100% 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 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).

Adverse impacts to freshwater benthic macroinvertebrate communities have also been reported following exposure 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 up to 50% decreases 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 Toxicity of PAH and non-PAH coal tar component classes

Most adverse effects associated with exposure to coal tars and their distillates are attributed to the PAHs present as the major components of the substances. 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 (2015c).

9.1.4 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 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 ≤1.0 mg/L and/or chronic no-observed-effect concentrations (NOECs) ≤0.1 mg/L (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).

9.1.5 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 6 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 (2015c), and do not represent all ecotoxicity data available for substances within the group. Consequently, the ranges provided in Table 6 demonstrate general trends for the chemical classes in order to allow a qualitative comparison of ecotoxicity among the classes.

Table 6. 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
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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
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 includes 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 EC/LC₅₀ values falling in the range of 1–100 mg/L and most chronic terrestrial values generally in the range of 50–100 mg/kg dw of soil (Table 6; Environment Canada 2015c).

Phenolic compounds exhibit slightly higher toxicity than MAHs, although most acute and chronic aquatic endpoint values remain in the range of 1–10 mg/L (Environment Canada 2015c). 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 rainbow trout, *Oncorhynchus mykiss*, LC₅₀ values of 0.15 and 0.07 mg/L were determined for phenol and 2-naphthol, respectively (Black et al. 1983). Terrestrial toxicity endpoint values for the phenolics are comparable with those of the MAHs, with the lowest values falling in the range of 50–100 mg/kg dw of soil (Environment Canada 2015c).

Heterocyclic compounds generally have relatively low acute and chronic toxicity (e.g., aquatic endpoint values in the range of 1–100 mg/L; Environment Canada 2015c); however, 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 EC_{50} values for acridine and green algae are 0.27–0.90 mg/L for *Pseudokirchneriella subcapitata* (Blaylock et al. 1985; Dijkman et al. 1997) and 0.32–0.41 mg/L for *Scenedesmus acuminatus* (Van Vlaardingen et al. 1996). A pattern of increased toxic potential with increasing number of aromatic rings is evident, so that quinoline with 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 such as acridine and quinoline can elicit adverse effects by mechanisms such as reproductive toxicity, photo-induced toxicity and/or metabolic transformation to more biologically active products, leading to significantly enhanced toxicity (Bleeker et al. 2002).

9.1.6 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 “toxic” criteria as defined in the *Canadian Environmental Protection Act* (CEPA) (Canada 1994). Based on this decision, PAHs were added to Schedule 1 of CEPA (i.e., the List of Toxic Substances) and major sources of anthropogenic PAHs, such as aluminum smelters and steel mills, are now subject to risk management. In response to the need for risk management, Canadian Environmental Quality Guidelines for PAHs were developed by the Canadian Council of Ministers of the Environment (CCME) for aquatic, sediment and soil organisms (CCME 1999a, 1999b, 2010) (Table 7).

Canadian Environmental Quality Guidelines (CEQGs) from the CCME were used to determine CTVs for aquatic, sediment and soil organisms (Table 7). CCME guideline values have been derived for 9 PAHs for aquatic life (CCME 1999a), for 12 PAHs for sediment quality (CCME 1999b), and for 15 PAHs for soil quality (CCME 2010). As CEQG values have been derived to be protective of wildlife on a long-term basis, these guideline values are also used as the predicted no effect concentrations (PNECs) for the risk assessment, without use of application factors.

Table 7. 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 ^e
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
2-methylnaphthalene	N/A	0.020	N/A

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. A full guideline value is not available.

^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 emissions of PAHs, major components of coal tars and their distillates, from coal-tar-related sources were used to evaluate 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 releases from the only coal tar refiner in Canada; the second scenario considers releases from the coal tar refiner to the sewer system; the third scenario examines releases from the application of 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)

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 refiner in Canada was examined as a potential route of exposure to the environment, and is discussed below. The substances of primary concern in coal tars—PAHs—mostly 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 (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 refiner. The deposition of selected PAHs to soil resulting from air emissions in the Hamilton area from the coal tar refiner was modelled. The coal tar refiner is situated close to Hamilton Harbour, on the shore of Lake Ontario. The annual air emissions of naphthalene, phenanthrene and acenaphthene, which are the PAHs released in the greatest quantities, as reported to the National Pollutant Releases Inventory in 2012 (NPRI 2014), were converted to emissions rates based on this facility operating 24 hours/day, 365 days/year. The maximum one-year deposition rate 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 four years of atmospheric data. The one-year deposition rates were converted to soil concentrations

after ten 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 = yearly 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 biodegradation and from 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 8. It is assumed that these limits are the maximum concentration of each component found in the effluent from the coal tar refiner and are used to derive concentrations for each component in wastewater entering Hamilton's Woodward Avenue wastewater treatment plant (WWTP) (Table 8), using 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)

V_1 = the average daily discharge to the sewer system by the coal tar refiner (311 m³/day) (e-mail from City of Hamilton, Environmental Monitoring & Enforcement, to Ecological Assessment Division, 2013, unreferenced)

V_2 = the average daily flow of the Woodward Avenue WWTP (278 146 m³/day) (Environment Canada 2013)

Table 8. Maximum estimated aquatic concentrations of coal tar components discharged by the coal tar refiner to the sewer system as compared to the Canadian Water Quality Guidelines

Parameter	City of Hamilton, ON, sewer discharge limits µg/L	Maximum estimated aquatic concentration from the coal tar refiner at influent to WWTP µg/L	CCME guidelines ¹ µg/L
Phenolic compounds	1000	1.1	4 ²
Benzene	10	0.011	370
Ethylbenzene	160	0.18	90
Toluene	16	0.018	2
Total xylenes	1400	1.5	N/A ³
Total PAHs	5	0.0056	N/A ⁴

N/A – not available

¹ Canadian freshwater quality guidelines for aquatic life, CCME (2014)

² Guideline is for mono- and di-hydric phenols

³ No guideline value exists available

⁴ No guideline value available. 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 one or more coal tars and their distillates. Since coal tar substances cannot be measured in the environment as such because it is a UVCB, 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

There were 26 reported spills of “tar,” “tar and water mixture,” “tar-based caulking,” “driveway sealant,” “asphalt sealer,” “sealant (not otherwise specified (N.O.S))” and “coal tar distillates (N.O.S)” to land or surface water in Ontario during the five years between 2008 and 2012 (Ontario 2013), totalling 2534 L (spills from roofing companies/jobs were not included in the total here, unlike the spills total given in the Releases section, 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 their distillates. Diamond Environmental Group (2011) found that 23% of the driveways sampled in the Toronto area were coated with CTPS. Applying this estimate 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 seal coat (e-mailed letter from M. Diamond to Ecological Assessment Division, Environment Canada, 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).

9.2.3.1 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. Two studies relevant to Canadian conditions were identified. Watts et al. (2010a, 2010b) and Rowe and O'Connor (2011) measured concentrations of PAHs in stormwater runoff collected from parking areas coated with CTPS, in the states of New Hampshire and New Jersey, respectively, which are considered to be similar to conditions in some Canadian cities. As well, Watts et al. (2010a, 2010b) measured PAH concentrations in aquatic runoff from the coated parking lots, and in sediments downstream of the parking areas in a stormwater swale and nearby wetland, over an 855-day period.

Watts et al. (2010a, 2010b) obtained much higher peak concentrations of PAHs in the stormwater runoff (642 and 5890 µg/L, in two different lots) than the study by Rowe and O'Connor (2011) (maximum of 288 µg/L). This may have been influenced by only one coat of CTPS applied to the parking lot studied by Rowe and O'Connor (2011), instead of the two coats used by Watts et al. (2010a). Manufacturers of CTPS recommend that two coats be applied (Rowe and O'Connor 2011). Therefore, the data from Watts et al. (2010a, 2010b) were used to develop the aquatic exposure scenario described below.

Concentrations of PAHs were measured as whole water concentrations, though Watts et al. (2010a, 2010b) also measured some filtered samples for dissolved concentrations. The total PAH concentrations comprised the 16 EPA priority pollutant

PAHs (U.S. EPA 2013) in each study, though Watts et al. also analyzed for 46 PAHs in some of the water and sediment samples (UNHSC 2010). PAH concentrations in the filtered subset of samples were reduced by 41–98% relative to the unfiltered splits, showing that PAHs tend to partition to particulates.

In both Watts et al. (2010a, 2010b) and Rowe and O'Connor (2011), the highest total PAH concentrations were initially observed in the runoff from the first rain event that occurred approximately one day after the application of the CTPS. In the second rain event, the total PAH concentrations fell by more than half in each case.

Using the aquatic emission factor from Watts et al. (2010a), realistic estimates were made of the mass of PAHs that would be transferred to urban runoff on an annual basis as a result of CTPS application in ten urban centres in Canada, including six large, two medium and two small urban areas. It was conservatively assumed that all runoff goes into the storm sewer system and into downstream water bodies without any treatment, which is the case with separated sewer systems (separate storm sewers and sanitary sewers). Some of the older combined sewer infrastructure remains in parts of many Canadian cities, where all of the storm and sanitary runoff is routed to the wastewater treatment system (WWTS). However, in cases of large storms, the WWTS often does not have enough capacity to treat all of the storm and sanitary flow, so part of the flow is not treated but routed directly to downstream water bodies.

It is noted that the alkylated PAHs may represent a significant fraction of the total PAH mass in CTPS, as shown by the runoff data of Watts et al. (2010b), who analyzed 46 PAHs, including alkylated naphthalenes, phenanthrenes and fluorenes. The alkylated PAHs are not given further consideration in this exposure assessment, as Canadian Environmental Quality Guidelines have not been developed for them (with the exception of 2-methylnaphthalene); however, it is acknowledged that all PAHs will contribute additively to toxicity.

The following data were used in the estimations. Approximately 15 500 tonnes per year, as the upper threshold of CTPS, are sold in Canada (EHS 2010). The amount of CTPS used in each urban centre was proportioned based on its population as compared to the total urban population of Canada (approximately 27 million). The mass of PAHs in runoff as measured in Watts et al. (2010a, 2010b) in unfiltered water samples was scaled to the Canadian urban centres based on their land area and expected area of pavement covered with CTPS. Then, the total mass of PAHs was used to determine the concentration of individual PAHs, based on the percentage contribution of these individual PAHs to the total PAHs concentration as measured by Watts et al. (2010a, 2010b). The concentrations of each PAH calculated with this method are considered as the aquatic predicted environmental concentrations (PECs). A full description of the estimation method and calculations is available in Environment Canada (2015a). The results of the calculations for one large urban centre are provided in Table 10 in the Risk Characterization section. The PECs for all of the ten selected locations within Canada are provided in Environment Canada (2015a).

9.2.3.2 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 USEPA's National Center for Environmental Assessment (USEPA 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 (OC) 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 OC in bottom sediments are 1–3% for rivers and estuaries and 2–4% for lakes (Gobas 2010).

Additional information on the approach is available in supporting documentation (Environment Canada, 2015a). The resulting PECs in bottom sediment for one large urban centre are presented in Table 11 in the Risk Characterization section, below.

9.3 Characterization of ecological risk

The approach taken in this ecological screening assessment was to examine the available scientific information and develop conclusions based on a weight-of-evidence approach, as required under CEPA 1999. Lines of evidence considered in the assessment relate to the environmental stability, bioaccumulation potential, toxicity and exposure potential of coal tar, its distillates and their major components. Risk quotients ($RQ = PEC/PNEC$) were calculated for exposure scenarios deemed to be significant potential sources of coal tar products and/or their components to the environment. The RQ results are an important line of evidence in evaluating the potential for risk to the environment.

As the toxicity of PAHs and other non-polar narcotic substances is additive (DiToro et al. 2000; DiToro and McGrath 2000), a toxic units approach, as described in Appendix 4 of Environment Canada (2015c), was employed to determine the risk of total PAHs to soil, aquatic and sediment organisms in the scenarios described in the following sections. The toxic units for each PAH in the substance is determined by dividing its concentration by a low effects level (in this case the Canadian water, sediment or soil

quality guidelines) and then summing the toxic units for the individual PAHs to obtain the overall toxic units for PAHs in the substance. As the risk quotients here are determined by dividing the PECs for each PAH by their PNEC (the Canadian water, sediment or soil quality guideline values), the risk quotients are the same as the toxic units for each PAH. Therefore, the RQs can be summed to obtain the overall toxic units for the PAHs.

9.3.1 Releases from industry

9.3.1.1 Releases to soil from air deposition

The estimated soil concentrations resulting from ten years of deposition of the selected PAHs to soil one kilometre downwind from the industrial area containing the coal tar refiner in Hamilton, Ontario, were compared to the Canadian Soil Quality Guidelines (CCME 2010) to calculate the RQs for soil for each PAH considered (Table 9). This estimate was based on recent (2012) air emission data and does not reflect past, higher emission levels nor future changes in emissions. As the coal tar refiner has been in operation at this location for over 20 years, an estimate 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. 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 ^a	1.15
Naphthalene	0.0074	0.013 ^b	0.57
Acenaphthene	0.0089	0.28	0.03
Toxic units sum	-	-	1.8

^a Interim guideline value (CCME 1991) or provisional guideline value (CCME 1997)

^b Guideline value for protection of freshwater life. Full guideline value not available.

A toxic units approach, as described above, was used to determine that the combined toxicity of the three PAHs listed above is approximately 1.8 toxic units. This is considered to pose a risk to the environment, as the threshold level for concern is 1.0 toxic unit. This analysis only considered the top three PAHs emitted by the coal tar refiner; had additional PAHs been included, the toxic units, and thus the 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 refiner, 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 refiner, 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 refiner for this scenario.

9.3.1.2 Releases to water

The maximum estimated concentrations of various coal tar components discharged by the coal tar refiner to the sewer system result in WWTP influent concentrations that do not exceed the Canadian Water Quality Guideline values for these parameters (Table 8). The concentrations of coal tar components in the WWTP effluent will be even lower than in the influent due to physical-chemical treatment processes at the WWTP. Therefore, these releases are not considered to present a risk to the aquatic environment.

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

Tables 10 and 11 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 Environment Canada (2015a) and the RQs are summarized in Table 12.

Table 10. Summary of aquatic PECs, PNECs and RQs for PAHs from CTPS runoff in receiving water for one large urban centre in Canada

PAH	Aquatic PEC (µg/L)	Aquatic PNEC ^a (µg/L)	Aquatic RQ (PEC / PNEC)
Naphthalene	<0.01	1.1	<0.01
Acenaphthene	0.025	5.8	<0.01
Fluorene	0.027	3.0	<0.01
Phenanthrene	0.12	0.4	0.30
Anthracene	0.035	0.012	2.9
Fluoranthene	0.11	0.04	2.7
Pyrene	0.082	0.025	3.3
Benzo[a]anthracene	0.031	0.018	1.7
Benzo[a]pyrene	0.028	0.015	1.9
Acenaphthylene	<0.01	N/A	N/A
Chrysene	0.033	N/A	N/A
Dibenz[a,h]anthracene	<0.01	N/A	N/A

^a Aquatic PNECs are the Canadian Water Quality Guidelines for the protection of aquatic life (CCME 1999a). N/A – not applicable; no Canadian Water Quality Guideline (CCME 1999a) was available for this PAH.

Table 11. Summary of sediment PECs, PNECs and RQs for PAHs from CTPS runoff in receiving water bodies for one large urban centre in Canada

PAH	Sediment PEC (µg/kg dw)	Sediment PNEC ^a (µg/kg dw)	Sediment RQ (PEC / PNEC)
Naphthalene	<0.01	34.6	<0.01
Acenaphthene	0.95	6.71	0.14
Fluorene	1.29	21.2	0.06
Phenanthrene	22.7	41.9	0.54
Anthracene	6.23	46.9	0.13
Fluoranthene	47	111	0.42
Pyrene	41.2	53.0	0.78
Benzo[a]anthracene	24.4	31.7	0.77
Benzo[a]pyrene	33.3	31.9	1.0
Acenaphthylene	0.026	5.87	<0.01
Chrysene	18.9	57.1	0.33
Dibenz[a,h]anthracene	6.82	6.22	1.1

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

Table 12 summarizes the risk quotient data for individual PAHs for the ten urban centres. Many of the urban centres have aquatic RQs greater than one for each of anthracene, fluoranthene, pyrene, benzo[a]anthracene, and benzo[a]pyrene. Sediment RQs for individual PAHs were below one except for benzo[a]pyrene and dibenz[a,h]anthracene at two locations. Naphthalene, acenaphthene, fluorene and phenanthrene did not exceed levels of concern at any locations.

Table 12. Number of urban centres (out of ten) characterized with aquatic or sediment RQs greater than one

PAH	Number of centres with aquatic RQ >1	Number of centres with sediment RQ >1
Naphthalene	0	0
Acenaphthene	0	0
Fluorene	0	0
Phenanthrene	0	0
Anthracene	7	0
Fluoranthene	7	0
Pyrene	8	0*
Benzo[a]anthracene	6	0*
Benzo[a]pyrene	6	2
Acenaphthylene	N/A	0
Chrysene	N/A	0
Dibenz[a,h]anthracene	N/A	2

* One centre had RQ of >0.9.

A toxic units approach, as described earlier, was employed to determine the risk of total PAHs to the aquatic and sediment environments in each of the ten Canadian urban areas considered (Table 13). It can be seen that at these urban centres, toxic units range from 1.2 to 15 for the aquatic compartment and from 0.5 to 6.3 for the sediment compartment. All ten urban centres characterized have toxic units for PAHs greater than one for the aquatic compartment and eight urban centres have toxic units greater than one for the sediment compartment.

It is noted that these toxic unit calculations are underestimations of the total toxic units from PAHs contributed by CTPS to the environment, as only a limited set of 9 PAHs for the aquatic environment and 12 PAHs for sediment were considered, which included only those PAHs with a CEQG value developed for them (Table 7). Based on the data of Watts et al. (2010b), CTPS contains a high proportion of alkyl-PAHs, which have not been considered here.

Table 13. Toxic units^a for total PAHs from CTPS for each urban centre

Urban centre	Toxic units (aquatic)	Toxic units (sediment)
Location 1 (L) ^b	12.9	5.3
Location 2 (L) ^b	8.5	3.5
Location 3 (L) ^b	4.0	1.6
Location 4 (L) ^b	15.2	6.3
Location 5 (L) ^b	9.5	3.9
Location 6 (L) ^b	8.0	3.3
Location 7 (M) ^c	1.2	0.50
Location 8 (M) ^c	5.1	2.1
Location 9 (S) ^d	2.2	0.92
Location 10 (S) ^d	10.4	4.3

^a Aquatic toxic units include 9 PAHs (see Table 10) and sediment toxic units include 12 PAHs (see Tables 11 and 12).

^b L = large urban centre (population greater than 100 000)

^c M = medium urban centre (population between 30 000 and 100 000)

^d S = small urban centre (population between 1000 and 30 000)

The results indicate that PAHs in runoff from CTPS-coated areas can reach levels that are high enough to exert toxicity to aquatic and sediment organisms in receiving water bodies, both through aggregate exposure, and also for individual PAHs, in some cases. Long-term accumulation of PAHs in the sediment bed could cause toxic effects in sediment organisms, as well as exposure to pelagic species should sediment re-suspension occur.

9.4 Consideration of lines of evidence and conclusion

Based on empirical and modelled data, most PAHs that are major components of coal tars, coal tar oils, coal tar upper distillates and high-temperature coal tar pitch 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

remain for long periods in air sorbed 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 remain for long periods in the environment, while the heterocyclics are, 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.

Based on the combined evidence of empirical and modelled data, coal tars and their distillates contain components that have the potential to be highly bioaccumulative in fish and/or invertebrates with BCF/BAF values greater than 5000. These include the three-ring PAH phenanthrene as well as several other four-, five- and six-ring PAHs, such as anthracene, fluoranthene, pyrene, benzo[a]anthracene, benzo[k]fluoranthene, benzo[a]pyrene, benzo[ghi]perylene and dibenz[a,h]anthracene. Some invertebrate species are not able to metabolize PAHs, which may cause them to bioaccumulate these substances to high levels. As high molecular weight PAHs tend to persist in sediments, benthic invertebrates may be continuously exposed to them. Most non-PAH components of coal tars and their distillates are not highly bioaccumulative; monocyclic aromatic hydrocarbons and phenols have low bioaccumulation potential; the heterocyclic representative structures have low to moderate 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.

Considering the release of coal tars and their distillates from facilities and their associated processes, an analysis of estimated concentrations in soil resulting from the deposition from air of three coal-tar-related PAHs released from a coal tar refining facility determined that levels of phenanthrene are predicted to exceed the no-effect level 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 to the soil environment, based on their combined toxicity of approximately 1.8 toxic units, which is equivalent to 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, increasing the toxic units. Therefore, releases of coal tar and their distillates at coal tar refining facilities and depositing to soil are likely to exceed levels that elicit adverse effects in soil organisms in Canada.

There is an average of about six spills/year in Ontario of potential coal tar-containing substances (e.g., tar, tar and water mixture, and driveway sealer), with an average total volume of about 4500 L/year (Ontario 2013). However, many of these spills likely do not contain coal tar. Based on Ontario spills data, and the Diamond Environmental Group (2011) study of the prevalence of CTPS use in the Toronto area, it is estimated that

there is about one spill/year of CTPS in the province of Ontario, with an estimated total volume of 120 L/year as a high-end estimate (see Ecological Exposure Assessment section). Thus, there is low risk of harm to the environment due to releases of coal tar substances as a result of spills.

In addition, an analysis was performed of the potential risk from the release of coal tar to water and sediment in Canada resulting from the application and use of coal tar-based pavement sealants, and its subsequent wash-off due to precipitation and abrasion. This analysis determined that predicted exposure concentrations of individual coal-tar-related PAHs (i.e., anthracene, fluoranthene, pyrene, benzo[a]anthracene and benzo[a]pyrene in water, plus benzo[a]pyrene and dibenz[a,h]anthracene in sediment) exceed no-effect levels for pelagic and sediment organisms in the CCME Canadian Water and Sediment Quality Guidelines (CCME 1999a, 1999b). As the toxicity of PAHs and other non-polar narcotic substances is additive (Di Toro et al. 2000; Di Toro and McGrath 2000), all PAHs in the water and sediment contribute to the risk, and a toxic units approach was employed to describe the risk of PAHs as a group to the aquatic and sediment environments in each of the ten Canadian urban centres considered. The toxic units for PAHs for the urban centres ranged from 1.2 to 15 in water and 0.5 to 6.3 in sediment. These toxic units are based only on those PAHs for which aquatic or sediment quality guidelines were available and not the total number of individual PAHs that are expected to be present. Additional PAHs would also contribute to toxicity and, therefore, the risk from coal tar in CTPS is actually higher than presented above. Therefore, based on the risk characterization using select PAHs as being representative of releases from CTPS, there is a risk of harm to aquatic and sediment organisms in urban centres across Canada due to the use of CTPS.

Considering all lines of evidence presented in this draft screening assessment, there is a risk of harm to organisms, but not to the broader integrity of the environment, from releases of coal tars and their distillates. It is proposed to conclude that coal tars and their distillates meet the criteria under paragraph 64(a) of CEPA 1999, 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 proposed to conclude that coal tars and their distillates do not meet the criteria under paragraph 64(b) of CEPA 1999, 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.

9.5 Uncertainties in evaluation of ecological risk

The application of modelling approaches to estimate 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.

For the exposure scenarios, the quantity of PAHs entering the soil and aquatic environments was estimated based on a number of assumptions, such as the amount of CTPS used in Canada, the emission factor for PAHs in stormwater runoff obtained from Watts et al. (2010a), and the method of estimating the PAHs concentration in sediments using the equilibrium partitioning approach. The latter of these is believed to underestimate the concentration of substances in sediments (Fu et al. 1994; Gobas and MacLean 2003; Neff et al. 2005; Zeng and Tran 2002) and thus, the risks might be greater than estimated herein.

The exposure scenarios only considered a limited set of PAHs, ones for which Canadian environmental quality guidelines have been developed. It is noted that Watts et al. (2010a,b) measured a suite of 46 PAHs in CTPS runoff. Monitoring data for other substances in coal tars and coal tar pitch, such as monocyclic and heterocyclic aromatic compounds, were not found, so these substances were also not considered in the quantitative risk characterizations for the aquatic environment. NPRI emissions data from the coal tar refiner were available for other substances besides PAHs, which could have been used in the soil deposition scenario. However, given that a risk to the soil environment was indicated based on the releases of only three PAHs, releases of other substances for the soil deposition scenario were not quantified. If more PAHs and other substances had been considered in the risk assessment scenarios, this would have increased the level of risk identified for coal tars and their distillates or their components released to soil, or to water and sediment. Given that several of the substances in coal tars and their distillates (e.g., PAHs, MAHs) have a common mode of action (non-polar narcotic), the risk of these substances is predicted well by additive toxicity (Di Toro et al. 2000; Backhaus and Faust 2012).

10. Potential to Cause Harm to Human Health

10.1 Exposure assessment

The assessment focuses on potential exposures to substances released during storage and handling of coal tars and refining of coal tars in the vicinity of those industrial sites. 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.

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

Releases of benzene and PAHs 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 refiner 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 refiner 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 (Ruetgers 2013b). Thus, releases will 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 1 (Table A1.1 with results presented in Table A1.2). Releases were considered as area 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 one hour after a given release event. During a 24-hour period, for point emission sources, the maximum one-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 a year, it can be expected that the direction of the prevalent winds will be more variable and uncorrelated to the wind direction for a single event; thus, the maximum amortized exposure concentration for one year is determined by multiplying the maximum one-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 yearly amortized concentration from the value of the maximum one-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 µg/m³. This concentration is greater than the 2008 Canadian average background concentration of benzene in ambient air of 0.88 µg/m³ (NAPS 2008). As well, the modelled benzene air concentration of 1.45 µg/m³ exceeds the Ontario Ministry of Environment criteria (Ambient Air Quality Criteria) for annual air concentrations of 0.45 µg/m³ (Ontario 2012). The region around the coal tar refiner has a local monitoring network for which volatiles, including benzene, are monitored. The annual average values at the three monitoring stations were between 1.6 and 3.3 µg/m³ for 2012, with maximum values ranging from 4.6 to 54.5 µg/m³ (HAMN 2012). 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 as a result of coal tar refining are reported to NPRI (2014). 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 1–2 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 14, 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 14. SCREEN3 modelled air concentrations of PAH releases from a coal tar refiner

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 criteria (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 15 (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 15. Ambient air concentrations of PAHs in Hamilton

Compound	Annual average concentration (ng/m ³) ¹
Benzo[a]anthracene	1.41
Chrysene	1.92
Benzo[b]fluoranthene	2.06
Benzo[k]fluoranthene	1.56
Benzo[a]pyrene	1.40
Indeno[1,2,3-cd]pyrene	1.15
Dibenzo[a,h]anthracene	0.36
Benzo[g,h,i]perylene	1.16

¹ (HAMN 2012)

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 refiner produced estimates of $1.14 \mu\text{g}/\text{m}^3$ at 1000 m, a difference of $0.31 \mu\text{g}/\text{m}^3$ compared to SCREEN3, which produced a value of $1.45 \mu\text{g}/\text{m}^3$ 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 $0.55 \text{ ng}/\text{m}^3$ after the 1-hour concentration was converted to an annual concentration, a difference of $0.16 \text{ ng}/\text{m}^3$ compared to SCREEN3, which produced a value of $0.71 \text{ ng}/\text{m}^3$ in 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 (CSPA 2014). 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 2 (Table A2.1 with results presented in Table A2.2). All releases were considered as area 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; a maximum of 0.11 to 1.11 $\mu\text{g}/\text{m}^3$ 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 $\mu\text{g}/\text{m}^3$ 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 19.

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 AP-42 emissions equations (U.S. EPA 2008) for each mode of transport, as well as the physical-chemical properties of coal tar (ArcelorMittal 2010). 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 refiner (Ruetgers 2013b).

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 one day of travel time based on the close proximity of the integrated steel mills to the coal tar refiner.

Total yearly 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 refiner

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 AP-42 emissions equations (U.S. EPA 2008) for each mode of transport, as well as the physical-chemical properties of the substances, at relevant transport temperatures. 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 kilotonnes 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 one day of transit, in the absence of pressure valves and venting to the atmosphere. The transport of other distillates including tar oils (coal) (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 tankcar are estimated to be from 0.17 kg to 0.26 kg during one 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 one 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 is used in pavement sealants (typically 15–30% 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 (here 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 16). 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 16. Median concentrations in dust (µg/g) 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 ¹	129	5.1
Benzo[a]pyrene	4.5	0.44

¹ Determined as the sum of 16 parent PAHs, corresponding to the 16 priority PAHs identified by the U.S. EPA (see Table A.3.1 in Appendix 3).

In the dust study described above, the median concentration of B[a]P in dust from apartments with CTPS parking lots (4.5 µg/g) was ten times higher than that in the dust from apartments with parking lots of other pavement surface types (0.44 µg/g). The average concentrations of the 16 individual PAHs found in house dust (Mahler et al. 2010) were considered for exposure (see Table A3.1A-C in Appendix 3). 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 greater 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, soil concentrations of total PAHs adjacent to seal coated lots were found to consistently contain concentrations greater than 90 mg/kg, compared to 5 mg/kg at sampling control sites located some distance from the lot (UNHSC 2010).

Concentrations of up to 411 mg/kg total PAHs were detected in soil directly adjacent to the seal coated parking surfaces, with the largest 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, determined 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, six were below the detection limit of either 1 or 2 ng/m³, with a further two PAH values being estimated from the data rather than directly determined, and so were not considered (Van Metre 2012a, 2012b). Three of the remaining ten PAHs had values of less than 4 ng/m³, which were considered to be of minor consequence. The values of the remaining seven measured PAHs are presented in Table 17.

Table 17. Ambient air concentrations at 1.28 m, 1.92 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/cm^3 , with an upper limit of 28% w/w comprising pure coal tar and 227.5 cm^2 (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 therefore 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 per two to three 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 that Health Canada uses to communicate to manufacturers and others that certain substances, when present in a cosmetic, may contravene (a) the general prohibition found in 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 these 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, the pitch acts as a binder, which would limit the ability for transfer and uptake by the skin. During the charcoal briquette production process, pitch has been used 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 greatly 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, seborrhoea and psoriasis (CIR 2008; CFR 2013). As these products have been considered acceptable therapeutic treatment options for certain skin conditions by international agencies, and Drug Identification Numbers (DINs) have been authorized for these products 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

Basis for categorization

A critical effect for coal tars and their distillates was carcinogenicity, based primarily on classifications by international agencies. The European Commission, IARC and NTP have classified coal tars as carcinogens. The European Commission 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). High-temperature coal tar pitch (HTCTP; CAS RN 65996-93-2) was classified 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 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). HTCTP was classified 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 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 classified coal tars and coal tar pitches as *known to be human carcinogens*, based on sufficient evidence of carcinogenicity in humans (NTP 2011).

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 classified low-temperature coal tar (CAS RN 65996-90-9) as a Category 1 carcinogen. Coal tar upper distillates (CAS RN 65996-91-0) are classified 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 reclassified 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 4 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. 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 LD₅₀'s ranged from 3300 to greater than 15 000 mg/kg-bw in rats (European Commission 2008), and dermal LD₅₀'s 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; ENTOX 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 two or five 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 five 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 three 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 12–16. 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 12–16 (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 LOAELs of 500 mg/kg-bw/day were identified. Rodents were exposed to heavy distillate during gestational days 11–15, 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 4 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 4 for more details).

Three oral carcinogenicity studies were identified for coal tar substances. Culp et al. (1998) exposed mice for two 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 (two-year exposure) and 0–236 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 well as 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 tumours 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 relative risk estimate (URR) 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 aluminium 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. Similar effects were 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 human health 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 US EPA previously identified PAHs that may be carcinogenic in animals and humans (US EPA 1992b), ultimately listing 16 substances that became known as the ‘priority pollutant’ PAHs (Menzie et al. 1992). They are naphthalene, acenaphthylene, acenaphthene, fluorene, phenanthrene, anthracene, fluoranthene, pyrene, benz[a]anthracene, chrysene, benzo[b+j]fluoranthene, benzo[k]fluoranthene, benzo[a]pyrene, indeno[1,2,3-cd]pyrene, dibenz[a,h]anthracene and benzo[ghi]perylene.

Several authors have created toxic or potency equivalency factors (PEFs) for numerous PAH species (i.e., estimates of their carcinogenic potency relative to that of B[a]P) (CCME 2008; Collins et al. 1998; Krewski et al. 1989, Meek et al. 1994; Muller 1997; Nisbet and LaGoy 1992; U.S. EPA 1994). 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 US 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 0.1-0.23 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 tumourigenic 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 has been assessed by Health Canada under CEPA, 1999 (Environment Canada Health Canada

1993), was determined to be a carcinogen and was added to the *List of Toxic Substances* in Schedule 1 of CEPA 1999. International organizations have drawn similar conclusions; 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% tumourigenic concentration (TC₀₅) for benzene was calculated to be $14.7 \times 10^3 \mu\text{g}/\text{m}^3$ (Environment Canada Health Canada 1993) from the epidemiological investigation of Rinsky et al. (1987), based on acute myelogenous leukaemia in Pliofilm workers. The 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 by Health Canada (Environment Canada, Health Canada 2008), and a LOAEC of $7.86 \text{ mg}/\text{m}^3$ was reported by Phimister et al. (2004) based on point-of-contact nasal olfactory epithelium injury in mice after a two-hour exposure. This effect was also seen in short-term and subchronic exposure studies in rats, that reported LOAECs of 5 and $10 \text{ mg}/\text{m}^3$, 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

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 $14.7 \times 10^3 \mu\text{g}/\text{m}^3$ (Environment Canada Health Canada 1993). The resulting MOEs for this exposure scenario are presented in Table 18.

Table 18. MOEs for benzene-based modelled data for a coal tar refiner

Model	SCREEN3	AERSCREEN
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Benzene inhalation concentration ($\mu\text{g}/\text{m}^3$)	1.45 (1000 m)	1.14 (1000 m)
TC₀₅ benzene ($\mu\text{g}/\text{m}^3$)	14.7×10^3	14.7×10^3
MOE	10 140	12 890

The MOEs for inhalation of benzene emissions from a coal tar refiner 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 $0.88 \mu\text{g}/\text{m}^3$ 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 $8.07 \text{ ng}/\text{m}^3$, based on emissions data from NPRI (see Table 19).

Table 19. Modelled air concentrations of PAH releases for a coal tar refiner

Compound	Annual concentration maximum at 1000 m ($\mu\text{g}/\text{m}^3$)	Potency equivalency factor	Benzo[a]pyrene equivalence (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 $8.07 \text{ ng}/\text{m}^3$ B[a]P equivalents at 1000 m, compared to the average Canadian

background concentration of $0.14 \mu\text{g}/\text{m}^3$ (NAPS 2012). Using the TD_{05} for B[a]P, the resulting MOE ($1.57 \text{ mg}/\text{m}^3 \div 8.07 \times 10^{-6} \text{ mg}/\text{m}^3$) 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 $5.5 \times 10^{-4} \mu\text{g}/\text{m}^3$, a difference of $0.16 \text{ ng}/\text{m}^3$ compared to the SCREEN3 calculation ($0.55 \text{ ng}/\text{m}^3$ versus $0.71 \text{ ng}/\text{m}^3$). 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 TC_{05} 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 20.

Table 20. 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)
TC_{05} 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 one-hour concentrations, which are several times larger 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 19). 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 TD_{05} for B[a]P, previously developed by Health Canada (Canada 1994), is $1.57 \text{ mg}/\text{m}^3$. The resulting MOE ($1.57 \text{ mg}/\text{m}^3 \div 6.16 \times 10^{-6}$

mg/m³) 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 Table A3.1A-C (Appendix 3). 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 (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 2013). B[a]P intakes for each age group and average daily dose are presented in Table 21.

Table 21. 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					

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 factors³ recommended by the U.S. EPA were considered and adjusted to the Health Canada age groups (see Table 22). These factors were then applied to the MOEs for each age group (U.S. EPA 2011b).

Table 22. 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
AdultAdult	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, calculated by Health Canada (Health Canada 2014) to be 0.562 mg/kg-bw/day.

³ 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.

The lifetime adjusted margin of exposure is calculated as follows:

$$MOE_{ADJ} \text{ Lifetime} = 1 / \Sigma[1/MOE \times \text{Averaging Time}]$$

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

$$MOE_{ADJ} \text{ Lifetime} = 15\,500$$

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 is considered potentially inadequate to protect these susceptible subpopulations.

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 17). 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 two to three years), along with the short duration of exposure (expected to be two to three 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 substances that are potentially present in coal tars, such as those containing heteroatoms (heteroatom: any atom 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.

In the health effects studies that were reviewed, certain details on laboratory animals (i.e., sex, strain, body weight and minute volume) were not always reported and were obtained from laboratory standard data as a result. Therefore, the use of standardized data may not be entirely representative of the physical features of the actual test animals used in the studies.

There is conservatism given the uncertainty in the estimates of PAHs ingested due to PAH concentration differences between dust and soil; however, these concentrations were assumed to be equal in the exposure estimations.

11. Conclusion

Quantitative analyses comparing predicted environmental concentrations of PAHs in soil, resulting from releases to air of coal tars and their distillates from the processing, storage and handling of these substances at a coal tar refining facility, with no-effect levels for PAHs from the Canadian Soil Quality Guidelines, determined that the concentrations of coal tars and their distillates in soil are likely to exceed levels that elicit adverse effects in soil organisms in the vicinity of such facilities. In addition, releases of coal tar and their distillates from CTPS to water and sediment from the application and use of CTPS are likely to exceed levels that elicit adverse effects in organisms based on estimated releases of PAHs.

Considering all lines of evidence presented in this draft screening assessment, there is a risk of harm to organisms, but not to the broader integrity of the environment, from releases of coal tars and their distillates. It is proposed to conclude that coal tars and their distillates meet the criteria under paragraph 64(a) of CEPA 1999, 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 proposed to conclude that coal tars and their distillates do not meet the criteria under paragraph 64(b) of CEPA 1999, 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 limited general population exposure to these high-hazard volatile constituents of coal tars and their distillates in the vicinity of coal tar producers and refiners. 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 estimates. Accordingly, it is proposed to conclude that coal tars and their distillates 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 proposed to conclude that coal tars and their distillates meet one or more criteria as set out in section 64 of CEPA 1999.

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.
- Aguirell 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]
- [AMAP] Arctic Monitoring and Assessment Programme. 2004. AMAP Assessment 2002: Persistent Organic Pollutants in the Arctic. Oslo, Norway; xvi + 310 pp. Available at: <http://www.amap.no/documents/doc/amap-assessment-2002-persistent-organic-pollutants-in-the-arctic/96>
- 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. pp. 277–96.
- ArcelorMittal. 2010. Chemical Product and Company Identification Information; Coal Tar. [MSDS]. Chicago (IL): ArcelorMittal USA Inc. Available from: <http://www.arcelormittalna.com/facilities/americas/ArcelorMittal+USA/documents/Coal%20Tar03-01-10.pdf>
- 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): 337–45.
- [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): 1351–8.
- Backhaus T, Faust M. 2012. Predictive environmental risk assessment of chemical mixtures: a conceptual framework. *Environ Sci Technol* 46: 2564-73.
- 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): 587–91.
- 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): 357–62. [cited in ATSDR 2002]
- 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): 3217–22.
- 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): 1306–15. [cited in PubMed 1996]
- Betts WD. 2000. Tar and pitch. [Internet]. In: Kirk-Othmer encyclopedia of chemical technology. [Internet] 1999-2014. John Wiley and Sons, Inc. Available from: <http://mrw.interscience.wiley.com/emrw/9780471238966/kirk/article/allykneu.a01/current/pdf> [restricted access]
- Bickers DR, Kappas A. 1978. Human skin aryl hydrocarbon hydroxylase. Induction by coal tar. *Journal of Clinical Investigation* 62(5): 1061–8.
- 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. *Journal of Investigative Dermatology* 78(3): 227.
- Black JA, Birge WJ, Westerman AG and Francis PC. 1983. Comparative aquatic toxicology of aromatic hydrocarbons. *Fund Appl Toxicol* 3: 353–8.
- 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* 81:187–93.
- Bommarito T, Sparling DW, Halbrook RS. 2010b. Toxicity of coal-tar pavement sealants and ultraviolet radiation to *Ambystoma maculatum*. *Ecotoxicology* 19: 1147–56.

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. *Toxicological Science* 56(1): 37–48.

Bos RP, Jongeneelen FJ, Theuvs JLG, Henderson PT. 1985. Detection of volatile mutagens in creosote and coal tar. *Mutation Research* 156 (3): 195–8. [also cited in ATSDR 2002]

Bos RP, Prinsen WJ, van Rooy JG, Jongeneelen FJ, Theuvs JL, Henderson PT. 1987. Fluoranthene, a volatile mutagenic compound, present in creosote and coal tar. *Mutation Research* 187(3): 119–25. [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. *Toxicologic Pathology* 37 (6): 733–40.

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, oxychlorane, 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: 241–7.

Bryer PJ, Scoggins M, McClintock NL. 2010. Coal-tar based pavement sealant toxicity to freshwater macroinvertebrates. *Environ Pollut* 158: 1932–7.

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. pp. 35–45.

Canada. 1999. *Canadian Environmental Protection Act, 1999*. S.C., 1999, c. 33, Canada Gazette Part III, vol. 22, no. 3. Available from: <http://www.gazette.gc.ca/archives/p3/1999/index-eng.html>

Canada. 2003. *Natural Health Products Regulations*. Available from: <http://gazette.gc.ca/archives/p2/2003/2003-06-18/html/sor-dors196-eng.html>

Canada, Dept. of the Environment. 2012. *Canadian Environmental Protection Act, 1999: Notice with respect to certain substances on the Domestic Substances List*. Canada Gazette, Part I, vol. 146, no. 48. Available from: <http://www.gazette.gc.ca/rp-pr/p1/2012/2012-12-01/html/sup-eng.html>

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): 403–9. [cited in IARC 1985]

[CCME] Canadian Council of Ministers of the Environment. 1991. Interim Canadian Environmental Quality Criteria for Contaminated Sites. CCME, Winnipeg. [cited in CCME 2010]

[CCME] Canadian Council of Ministers of the Environment. 1995. Protocol for the Development of Canadian Sediment Quality Guidelines for the Protection of Aquatic Life. CCME, Winnipeg. Available from:

http://www.ccme.ca/en/resources/canadian_environmental_quality_guidelines/scientific_supporting_documents.html

[CCME] Canadian Council of Ministers of the Environment. 1997. Recommended Canadian Soil Quality Guidelines. CCME, Winnipeg. [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). Winnipeg (MB). Available from: <http://ceqg-rcqe.ccme.ca/en/index.html#void>

[CCME] Canadian Council of Ministers of the Environment. 1999b. Canadian Sediment Quality Guidelines for the Protection of Aquatic Life: Polycyclic Aromatic Hydrocarbons (PAHs). Winnipeg (MB). Available from: <http://ceqg-rcqe.ccme.ca/en/index.html#void>

[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.

[CCME] Canadian Council of Ministers of the Environment. 2010. Canadian Soil Quality Guidelines for the Protection of Environmental and Human Health: Carcinogenic and Other PAHs. Available from: http://www.ccme.ca/en/resources/canadian_environmental_quality_guidelines/scientific_supporting_documents.html

[CCME] Canadian Council of Ministers of the Environment. 2014. Canadian Water Quality Guidelines for the Protection of Aquatic Life [Internet]. Accessed 5 August 2014. Available from: <http://st-ts.ccme.ca/>

[CCPA] Canadian Chemical Producers' Association. 2010. Responsible Care® Re-verification of VFT Canada Inc. [Internet]. [cited 2011 Jan]

[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.

[CHEMINFO] Cheminfo Services Inc. 2010. Technical and Socioeconomic Background Study on Coal Tars. Confidential Final Report. Confidential Contract Report. Coal Tars. Protected B. March 2010. Submitted to Health Canada. [CIR] Cosmetic Ingredient Review. 2008. Final Safety Assessment of Coal Tar as Used in Cosmetics. *International Journal of Toxicology* 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. *Environmental and Molecular Mutagenesis* 43(3): 159–68.

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, 453–65. [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): 819–23. [cited in CIR 2008]

Cloverdale Paint. 2013. Coal Tar 22 – Coal Tar Epoxy Product Information. Available from: http://www.cloverdalepaint.com/contractor_professional/product.asp?code=83022

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

[Conestoga] Conestoga Roofing and Sheet Metal Ltd. 2014. “Flat Roofing Systems”. Website page. Accessed 15 May 2014. Available from: <http://conestogaroofing.com/FlatRoofSystems.html>

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

[CSPA] Canadian Steel Producers Association. 2014. Communication to Health Canada Risk Management Bureau on Coal Tar Related Benzene Releases. February 2014.

Culp SJ, Beland FA. 1994. Comparison of DNA adduct formation in mice fed coal tar or benzo[a]pyrene. *Carcinogenesis* 15(2): 247–52. [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. *Polycyclic Aromatic Compounds* 11(1): 161–8. [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. Dietz WA et al. 1952. Properties of High Boiling Petroleum Products: Carcinogenicity Studies. *Ind Eng Chem* 44:1818–1827. [cited in ATSDR 2002]

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): 411–5. [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. pp. 127–46.

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):121–6.

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): 1951–70.

Di Toro DM, McGrath JA. 2000. Technical basis for narcotic chemicals and polycyclic aromatic hydrocarbon criteria. II. Mixtures and sediments. *Environ Toxicol Chem* 19:1971–82. [cited in DiToro et al. 2000]

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, Huebne 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. pp. 138–48. [cited in ATSDR 2002]

[DPD] Drug Product Database. 2014. [database on the Internet]. Ottawa (ON): Health Canada. [cited 2014 June]. Available from: <http://webprod5.hc-sc.gc.ca/dpd-bdpp/index-eng.jsp>

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

[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. Available from: <http://echa.europa.eu/web/guest/guidance-documents/guidance-on-information-requirements-and-chemical-safety-assessment>

[ECHA] European Chemicals Agency. 2013. Information on Chemicals – Registered Substances Database. Available from: <http://echa.europa.eu/web/guest/information-on-chemicals/registered-substances>

[ECJRC] European Commission Joint Research Centre. 2008. European Union risk assessment report: CAS No.: 65996-93-2: Coal-tar pitch, high temperature. May 2008. Luxembourg (NL):

Office for Official Publications of the European Communities. Available from:
<http://echa.europa.eu/documents/10162/433ccfe1-f9a5-4420-9dae-bb316f898fe1>

[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., Her Majesty the Queen in Right of Canada, as represented by the Minister of the Environment, and the Ministry of Environment, Province of Ontario. 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, and Her Majesty the Queen in Right of Ontario, as represented by the Minister of the Environment. Available from: <http://www.ec.gc.ca/epe-epa/default.asp?lang=En&n=F2E8DE70-1>

Emmett EA, Bingham EM, Barkley W. 1981. A carcinogenic bioassay of certain roofing materials. *America Journal of Industrial Medicine* 2(1): 59-64.

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

Environment Canada. 2001. Environmental Code of Practice for Integrated Steel Mills – CEPA 1999 Code of Practice. 1st edition. Environmental protection series: EPS 1/MM/7. Minister of Public Works and Government Services Canada. Available from: <http://www.ec.gc.ca/lcpe-cepa/default.asp?lang=En&n=E034D992-1>
Available from: <http://www.ec.gc.ca/lcpe-cepa/default.asp?lang=En&n=E034D992-1>

Environment Canada. 2013. Effluent Regulatory Reporting Information System (ERRIS) database. Accessed Oct. 2013.

Environment Canada. 2015a. Supporting Documentation: Ecological Exposure Assessment of Coal Tar Pitch-based Pavement Sealant (CTPS) to the Aquatic Environment. Information in support of the Draft Screening Assessment for coal tars and their distillates. Gatineau, QC: Environment Canada, Ecological Assessment Division. Available upon request from: substances@ec.gc.ca

Environment Canada. 2015b. Supporting Documentation: Identity, compositional information, physical and chemical properties, environmental persistence data and aquatic bioaccumulation data for coal tar and its distillates. Information in support of the Draft Screening Assessment for coal tars and their distillates. Gatineau, QC: Environment Canada, Ecological Assessment Division. Available upon request from: substances@ec.gc.ca

Environment Canada. 2015c. Supporting Documentation: Summary of empirical aquatic and terrestrial toxicity data for representative structures of coal tars and their distillation products and an explanation of the Toxic Units Approach. Information in support of the Draft Screening Assessment for coal tars and their distillates. Gatineau, QC: Environment Canada, Ecological Assessment Division. Available upon request from: substances@ec.gc.ca

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

Environment Canada; Health Canada. 1994. Polycyclic Aromatic Hydrocarbons. Ottawa (ON): Environment Canada; Health Canada. (Priority Substances List Assessment Report). Available from: http://www.hc-sc.gc.ca/ewh-semt/pubs/contaminants/psl1-lsp1/hydrocarb_aromat_polycycl/index-eng.php

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

[EPI Suite] Estimation Programs 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. Available from: www.epa.gov/oppt/exposure/pubs/episuitedi.htm

[ESIS] European Chemical Substances Information System [database on the Internet]. c1995-2011. Database developed by the European Chemicals Bureau (ECB). Accessed 19 January 2011. Available from: <http://esis.jrc.ec.europa.eu/>

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. 76/769/EEC. Official Journal L 262, 27.9.1976, pp. 201. Available from: <http://eurlex.europa.eu/LexUriServ/LexUriServ.do?uri=CONSLEG:1976L0769:20071003:EN:PDF>

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). Position Paper. Office for Official Publications of the European Communities: Luxembourg. Available from: http://ec.europa.eu/environment/air/pdf/pp_pah.pdf

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] Available from: <http://echa.europa.eu/documents/10162/433ccfe1-f9a5-4420-9dae-bb316f898fe1>

[FAO] 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. Available from: <http://www.fao.org/docrep/X5328E/X5328E00.htm>

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

Fu G, Kan AT, Tomson M. 1994. Adsorption and desorption hysteresis of PAHs in surface sediment. *Environ Toxicol Chem* 13(10): 1559–67.

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. *Polycyclic 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.

Gobas F, MacLean L. 2003. Sediment-water distribution of organic contaminants in aquatic ecosystems: the role of organic carbon mineralization. *Environ Sci Technol* 37(4): 735–41.

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): 767–73. [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): 537–43. [cited in ATSDR 2002]

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):1325–30. [cited in ATSDR 2002]

Gorski T. 1959. Experimental investigations on the carcinogenic properties of some pitches and tars produced from Silesian pit coal. *Medycyna Pracy* 10: 309–17. [cited in ECJRC 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. *Journal of the American Veterinary Medical Association* 96: 135–49. [cited in ECJRC 2008]

Gray RH. 1984. Health and environmental effects of coal-liquefaction processes: Environmentally acceptable technology development. *Energy* 9(4): 315–22.

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. Available from: <http://www.hamnair.ca/hamn-annual-reports.aspx>

[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. Available from: <http://www.hamnair.ca/hamn-annual-reports.aspx>

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): 2184–93.

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. [Internet]. Ottawa (ON): Health Canada, Environmental Health Directorate. Available from: <http://www.hc-sc.gc.ca/ewh-semt/pubs/contaminants/approach/index-eng.php>

Health Canada. 1996. Health-based tolerable daily intakes/concentrations and tumorigenic doses / concentrations for priority substances. Cat. H46-2/96-194E. Ottawa (ON): Minister of Supply and Services Canada. Available from <http://dsp-psd.pwgsc.gc.ca/Collection/H46-2-96-194E.pdf>

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

Health Canada. 2006. Anti-Dandruff Products monograph. Natural Health Products Ingredients Database. [Internet]. Ottawa (ON): Health Canada. [cited 2014 June 27]. Available from: http://webprod.hc-sc.gc.ca/nhpid-bdipsn/atReq.do?atid=antidandruff_anitpelliculaire&lang=eng

Health Canada. 2008. Guidance Document: Classification of Products at the Cosmetic-Drug Interface. Accessed 16 October 2013. Available at: http://www.hc-sc.gc.ca/cps-spc/pubs/indust/cosmet_drug_guide-droque-ref/index-eng.php

Health Canada. 2012. Guidelines for Canadian Drinking Quality. Ottawa (ON): Health Canada, Federal-Provincial-Territorial Committee on Drinking Water. Available from: http://www.hc-sc.gc.ca/ewh-semt/pubs/water-eau/2012-sum_guide-res_recom/index-eng.php

Health Canada. 2013. List of Permitted Food Additives. [Internet]. Ottawa (ON): Health Canada. [cited 2014 June]. Available from: <http://www.hc-sc.gc.ca/fn-an/securit/addit/list/index-eng.php>

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 on request 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: 155–6. [also 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): 351–6. [also 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. *Journal of Neural Transmission* 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. *Journal of the National Cancer Institute* 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: 372–84. [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: 1261–7.

[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. [also cited in ECJRC 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. pp. 175–6.

[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. pp. 174–5.

[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 1–102 (alphabetical order). Lyon (FR): International Agency for Research on Cancer. Available from: <http://monographs.iarc.fr/ENG/Classification/ClassificationsAlphaOrder.pdf>

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. *Mutation Research* 204(2): 195–20.

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): 267–72. [cited in ATSDR 2002]

Kinkead ER. 1973. Toxicity of coal tar. *In: Proceedings of the Fourth Annual Conference on Environmental Toxicology Held at Fairborn, Ohio on 16, 17 and 18 October 1973, Aerospace Medical Research Laboratory, Wright-Patterson Air Force Base, Ohio.* pp. 177–188. [accessed 2010 July 6].

Kireeva IS. 1968. Carcinogenic properties of coal-tar pitch and petroleum asphalts used as binders for coal briquettes. *Hyg. Sanit.* 33: 180–6. [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: 361–2.

Klein M. 1963. Susceptibility of strain B6AFI-J hybrid infant mice to tumorigenesis with 1,2-benzanthracene, deoxycholic acid and 3-methylcholanthrene. *Cancer Res* 23: 1701–7. [cited in ATSDR 1995]

Kligman AM, Kligman LH. 1994. Carcinogens show comedogenic activity: a potential animal screen for tumorigenic substances. *Cancer Letters* 87(2): 171–8. [also 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: 161–5.

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): 1601–9. [also 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. *Environmental Science and Technology* 35(13): 2704–9.
- Koppers Inc. 2013. MSDS Sheet, Coal tar roofing pitch. Issued March 14, 2013. Accessed 15 May 2014. Available from: <http://www.chemadvisor.com/koppers/ohsquery.pl>
- Krewski D, Thorslund T, Withey J. 1989. Carcinogenic risk assessment of complex mixtures. *Toxicology and Industrial 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, Sumerel 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): 3021–6. [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: 1801–14.
- Lewis RJ Sr., editor. 2001. *Hawley's Condensed Chemical Dictionary*. 14th edition. New York (NY): John Wiley & Sons, Inc.
- [LNHPD] 2014. Licensed Natural Health Products Database. [database on the Internet]. Ottawa (ON): Health Canada. [cited 2014 June]. Available from: <http://webprod5.hc-sc.gc.ca/lnhpd-bdpsnh/index-eng.jsp>
- 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 ECJRC 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. *Environmental and Molecular Mutagenesis* 44(2): 99–107.
- 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): 224–31.
- 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): 1161–8. *Environ Sci Technol.* 44(3): 894–900.

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): 1721–32. [also 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. pp. 189–98.

McCormick DL, Burns FJ, Alberg RE. 1981. Inhibition of benzo[a]pyrene-induced mammary carcinogenesis by retinyl acetate. *J Natl Cancer Inst* 66(3): 559–64. [cited in ATSDR 1995]

Meek ME, Chan PKL, Bartlett S. 1994. Polycyclic aromatic hydrocarbons: Evaluation of risks to health from environmental exposures in Canada. *Journal of Environmental Science and Health – Part C: Environmental Carcinogenesis & Ecotoxicology Reviews* 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*. New York (NY): Springer-Verlag. pp. 67–87. [cited in Environment Canada 1999]

Mukhtar H, Link CM, Chorniack 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. *Toxicology and Applied Pharmacology* 64(3): 541–9.

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 Letters* 33(3): 287–94.

Mukhtar H, Das M, Bickers DR. 1986b. Skin tumor initiating activity of therapeutic crude coal tar as compared to other polycyclic aromatic hydrocarbons in SENCAR mice. *Cancer Letters* 31(2): 147–51.

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]. Available from: <http://www.ec.gc.ca/rnspa-naps/>

[NAPS] National Air Pollution Surveillance Network. 2012. Data for PAHs in 2012. Ottawa (ON): Environment Canada. Available from: <http://maps-cartes.ec.gc.ca/rnspa-naps/data.aspx?lang=en>

[NCI] National Chemical Inventories [database on CD-ROM]. 2010. Issue 1. Columbus (OH): American Chemical Society. [cited 2011 Jan]. Available from: <http://www.cas.org/products/other-cas-products/nci-on-cd>

Neal J, Rigdon RH. 1967. Gastric tumors in mice fed benzo[a]pyrene: A quantitative study. *Tex Rep Biol Med* 25(4): 553–7. [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 ECJRC 2008]

Neff JM, Stout SA, Gunster DG. 2005. Ecological risk assessment of polycyclic aromatic hydrocarbons in sediments: identifying sources and ecological hazard. *Integrated Environ Assess and Manag* 1(1): 22–3.

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): 425–9. [cited in IARC 1985]

Neyra Industries Inc. 2011. Material Safety Data Sheet: Tarconite Coal Tar Pavement Sealer. Cincinnati (OH): Neyra Industries Inc. [Issued: 2011 May 23. Cited: 2015 April 20]. Available from: <http://neyra.com/resources/technical-information>.

Neyra Industries Inc. 2014. Material Safety Data Sheet: Tarconite Cool Weather Formula. Cincinnati (OH): Neyra Industries Inc. [Issued: 2014 March 19. Cited: 2015 April 20]. Available from: <http://neyra.com/resources/technical-information>.

[NHPID] Natural Health Products Ingredients Database. 2014. [database on the internet]. Ottawa (ON): Health Canada. [cited 2014 June]. Available from: <http://webprod.hc-sc.gc.ca/nhp-id-bdipsn/search-rechercheReq.do?lang=eng>

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: Polynuclear Aromatic hydrocarbons: A decade of progress. pp. 609–47. [cited in ATSDR 2002]

Nisbet ICT, LaGoy PK. 1992. Toxic Equivalency Factors (TEFs) for Polycyclic Aromatic Hydrocarbons (PAHs). *Regulatory Toxicology and Pharmacology* 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 on the Internet]. 2014. Gatineau (QC). Environment Canada. [cited 2014 January]. Available from: <http://www.ec.gc.ca/inrp-npri/>

[NTP] National Toxicology Program. 2005. Substance profiles: coal tars and coal tar pitches. In: 11th report on carcinogens (RoC) [Internet]. 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) [Internet]. Research Triangle Park (NC): U.S. Department of Health and Human Services, National Toxicology Program. [cited 2012 Sep 17]. Available from: <http://ntp.niehs.nih.gov/ntp/roc/twelfth/profiles/CoalTars.pdf>

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: 4913–24.

[Ontario] Ontario Standards Development Branch, Ministry of the Environment. 2011. Ontario Air Standards for Benzo[a]pyrene as a Surrogate for Polycyclic Aromatic Hydrocarbons. Toronto (ON): Ministry of the Environment. Available from: http://www.downloads.ene.gov.on.ca/envision/env_reg/er/documents/2011/010-6213.pdf

[Ontario] Ontario Standards Development Branch, Ministry of the Environment. 2012. Ontario Ambient Air Quality Criteria. [cited June 2012]. Toronto (ON): Ministry of the Environment. Available from: <http://www.airqualityontario.com/press/publications.php>

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

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): 1569–73. [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): 95–9. [cited in ATSDR 2002]

Pavement Rejuvenation International. 2014. Material Safety Data Sheet: Rejuvaseal [Internet]. Buda (TX): Pavement Rejuvenation International, LP. [Updated: 2014 February 2. Cited: 2015 April 30]. Available from: <http://www.rejuvaseal.com/technical-info/specifications>

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. pp. 192–224.

Peters AJ, Gregor DJ, Teixeira CF, Jones NP and Spencer C. 1995. The recent historical depositional trend of polycyclic aromatic hydrocarbons and elemental carbon to the Agassiz Ice Cap, Ellesmere Island, Canada. *Sci Tot Environ* 160/161: 167–79.

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 Research* 31(8): 1887–96.

Phillips DH, Alldrick AJ. 1994. Tumorigenicity of a combination of psoriasis therapies. *British Journal of Cancer* 69(6): 1043–5.

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. *Toxicological Sciences* 82(1): 268–78. [also cited in Environment Canada, Health Canada 2008]

PubMed. 1996. [database on the Internet]. Bethesda (MD): National Library of Medicine (U.S.). [updated 2011 Jan 1; cited 2013 Jan 10]. Available from: <http://www.ncbi.nlm.nih.gov/pubmed>

[R4CC] REACH for Coal Chemicals. 2011. Substance Inventory Attachment 2: Table of UVCB Substances. Available from: http://www.r4cc.org/index-Dateien/R4CC_INVENTORY.htm

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*. *Environmental Mutagenesis* 1(2): 105–12. [cited in IARC 1985]

[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 epidemiological risk assessment. *N Engl J Med* 316(17): 1044–50. [also 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): 1015–8. [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):1023–36.

Rowe AA, O'Connor, TP. 2011. Assessment of Water Quality of Runoff from Sealed Asphalt Surfaces. National Risk Management Research Laboratory, Office of Research and Development, U.S. Environmental Protection Agency, Cincinnati, OH. EPA/600/R-10/178.

[Ruetgers] Ruetgers Canada Inc. 2013a. Coal Tar Product Mapping Schematic. Diagram. E-mail to Risk Management Bureau, Health Canada, Aug. 2013.

[Ruetgers] Ruetgers Canada Inc. 2013b. Follow-up questions related to coal tar substances. Document, 4 pp. E-mail to Risk Management Bureau, Health Canada, Aug. 2013. [contains

some confidential business information]

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. *Mutation Research* 334(2): 117–224. [cited in ATSDR 2002]

Saperstein MD, Wheeler LA. 1979. Mutagenicity of coal tar preparations used in the treatment of psoriasis. *Toxicology Letters* 3(6): 325–9. [also 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): 329–34. [also 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. *Environmental Mutagenesis* 3(2): 181–95.

Schocket 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): 1253–8.

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): 415–21. [cited in ECJRC 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.

Scoggins M, Ennis T, Parker N, Herrington C. 2009. A photographic method for estimating wear of coal tar sealcoat from parking lots. *Environmental Science and Technology* 43: 4909–14, Supporting information.

[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. Available from: http://www.epa.gov/scram001/dispersion_screening.htm

[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 ECJRC 2008]

Sporstøl S, Gjøes N, Lichtenthaler RG, Gustavsen KO, Urdal K, Oreld F. 1983. Source identification of aromatic hydrocarbons in sediments using GC/MS. *Environ Sci Technol* 17: 282–6.

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): 261–5.

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): 112–31.

Steel Times International. 2013. SGL closes Canadian electrode plant. Oct. 23, 2013. Accessed 20 May 2014. Available from: <http://www.steeltimesint.com/news/view/sgl-closes-canadian-electrode-plant>

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. Available from: <http://www.accci.org/industry.html>

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. *Ecotox 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): 575–7. [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 EC 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. 1995a. Great Lakes Water Quality Initiative Technical Support Document for the Procedure to Determine Bioaccumulation Factors. Report No. EPA-820-B-95-005. Washington (DC): U.S. EPA, Integrated Risk Information System (IRIS). Available from: <http://www.epa.gov/IRIS/subst/0395.htm>

[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): US EPA. Available from: http://cfpub.epa.gov/ncea/iris_drafts/recordisplay.cfm?deid=54787

[U.S. EPA] United States Environmental Protection Agency. 1994. Coke oven emissions (CAS RN 8007-45-2). Washington (DC): U.S. EPA, Integrated Risk Information System (IRIS). Available from: <http://www.epa.gov/iris/subst/0395.htm>

[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). Washington (DC): U.S. EPA, Integrated Risk Information System (IRIS). Available from: www.epa.gov/iris/subst/0276.htm

[U.S. EPA] United States Environmental Protection Agency. 2003. Procedures for the Derivation of Equilibrium Partitioning Sediment Benchmarks (ESBs) for the Protection of Benthic Organisms: PAH Mixtures. EPA-600-R-02-013. Office of Research and Development. Washington, DC 20460

[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. Washington (DC): U.S. Environmental Protection Agency. Available from: <http://www.epa.gov/ttn/chief/ap42/ch05/final/c05s02.pdf>

[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. Available from: http://www.epa.gov/ttn/scram/dispersion_screening.htm#aerscreen

[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 19 March 2014. Available from: <http://water.epa.gov/scitech/methods/cwa/pollutants.cfm>

U.S. Pharmacopoeia. 2008–2010. [database on the Internet]. Coal Tar Topical Solution, USP 29. Available from: http://www.pharmacopeia.cn/v29240/usp29nf24s0_m19140.html

Uthe JF. 1991. Polycyclic aromatic hydrocarbons in the environment. *Can Chem News* Aug: 25–7. [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. *Atmospheric Environment* 51: 108–15.

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. *Environmental Science and Technology* 47: 1101–9.

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): 2035–42.

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. *Toxicology and Applied Pharmacology* 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):1017–22.

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. Available from: <http://pubs.acs.org/journal/esthag>

Watts AW, Ballester TP, Rosen RM, Houle JP. 2010a. Polycyclic aromatic hydrocarbons in stormwater runoff from sealcoated pavements. *Environ Sci Technol* 44(23): 8849–54.

Watts AW, Ballester TP, Rosen RM, Houle JP. 2010b. Supporting information for Watts et al. 2010a. Available at: <http://pubs.acs.org>

[WCI] World Coal Institute. 2007. Coal & steel [Internet]. [cited 2011 Jan]. Available from: [http://www.worldcoal.org/bin/pdf/original_pdf_file/coal_steel_report\(03_06_2009\).pdf](http://www.worldcoal.org/bin/pdf/original_pdf_file/coal_steel_report(03_06_2009).pdf)

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. *Polycyclic Aromatic Compounds* 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. *Chemical Research in Toxicology* 8(7): 955–62.

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. *Chemical Research in Toxicology* 4(4): 466–73. [also cited in ATSDR 2002]

Weyand EH, Wu Y, Patel S, Goldstein LS. 1994. Biochemical effects of manufactured gas plant residue following ingestion by B6C3F1 mice. *Journal of Toxicology and Environmental Health* 42(1): 89–107. [also 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. *Chemical Research in Toxicology* 8(7): 949–54. [also 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] Available from: <http://www.inchem.org/documents/ehc/ehc/ehc202.htm>

[WHO] World Health Organization. 2000. Air Quality Guidelines. Evaluation of Human Health Risk – Benzene (Chapter 5.2). Copenhagen (DK): Copenhagen WHO Regional Office for Europe. Available from:
http://www.euro.who.int/data/assets/pdf_file/0017/123056/AQG2ndEd_5_2benzene.pdf

[WHO] World Health Organization. 2001. Polycyclic aromatic hydrocarbons. In: Background material to WHO Regional Publications, European series, no. 91 (Air Quality Guidelines for Europe, Second Ed. 2000). Chapter 5.9: pp. 1-24. WHO Regional Office for Europe, Copenhagen, Denmark.

Wilson. 2013. Revisiting Dust and Soil Ingestion Rates Based on Hand-to-Mouth Transfer. *Human and Ecological Risk Assessment: An International Journal* 19: 158–88.

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: 887–94.

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: 717–28.

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. *Journal of Applied Toxicology* 5(2): 80–8. [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 17 June 2013]. [Accessed 9 Dec 2013].

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): 613–22.

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): 662–9.

Zeng E, Tran K. 2002. Distribution of chlorinated hydrocarbons in overlying water, sediment, polychaete, and hornyhead turbot (*Pleuronichthys verticalis*) in the coastal ocean, Southern California, USA. *Environ Toxicol Chem* 21(8): 1600–8.

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): 171–9. [cited in ATSDR 2002]

Appendix 1. Industrial Release from Coal Tar Processing, Handling and Storage for Coal Tar Refineries

Table A1.1 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 one-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 A1.2. Ambient concentrations of benzene in the vicinity of a coal tar refiner from SCREEN3

Distance (m)	One-hour concentration (µg/m³)	Annual concentration (µg/m³)
1	12.07	2.41
100	19.93	3.99
200	27.62	5.52
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
230	28.76	5.75

Appendix 2. Industrial Release from Coal Tar Production, Handling and Storage for Steel Mills

Table A2.1. Variable inputs to SCREEN3 for coal tar storage (benzene) at steel mills

Variables	Input
Source type	Area
Effective emission area ^a	200 m x 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 A2.2. Ambient air concentrations of benzene in the vicinity of coal tar storage and loading at steel mills

Distance (m)	Upper-bounding benzene emissions – one-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
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
230	21.97	4.39

Distance (m)	Lower-bounding benzene emissions – one-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
230	0.42	0.08

Appendix 3. Exposures from Products Used By Consumers

Table A3.1.A PAH concentrations (µg/g) and benzo(a)pyrene equivalents in house dust

Compound ¹	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 ²	0.001	1	0.001	0.001	0.1
B[a]P equivalents	0.00097	8.27	0.0268	0.000297	0.551

¹ Concentration data from Mahler (2010)

² Potency equivalency factors from Nisbet and LaGoy (1992)

³ Total B[a]P concentration from the sum of the potency equivalent PAH concentrations

Table A3.1.B PAH concentrations (µg/g) and benzo(a)pyrene equivalents in house dust

Compound ¹	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 ²	0.1	0.001	0.001	5	0.1
B[a]P Equivalents	0.7977	0.00977	0.000439	7.99	0.625

¹ Concentration data from Mahler (2010)

² Potency equivalency factors from Nisbet and LaGoy (1992)

³ Total B[a]P concentration from the sum of the potency equivalent PAH concentrations

Table A3.1.C PAH concentrations (µg/g) and benzo(a)pyrene equivalents in house dust

Compound ¹	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 (ug/m ³)	9.77	0.402	0.164	15.6	7.35	14.35182
Potency factor ²	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 BaP (ug/g) ³	20.1					

¹ Concentration data from Mahler (2010)

² Potency equivalency factors from Nisbet and LaGoy (1992)

³ Total B[a]P equivalents from the sum of the potency equivalent PAH values

Appendix 4. 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) (ECJRC 2008)
	65996-93-2	Oral LD₅₀ = > 15 000 mg/kg-bw (rat) (ECJRC 2008)
	65996-93-2	Dermal LD₅₀ = > 400 mg/kg-bw (Sprague-Dawley rat) (ECJRC 2008)
	8007-45-2/ 65996-89-6	Dermal LD₅₀ = > 7950 mg/kg-bw (rabbit) (ENTOX 2005)
	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 nine 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).
	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, 3/3 animals showed diffuse degenerative changes in the liver, and 3/3 animals died within 10–18 days. In the 2-day treatment group, 1/2 animals showed pseudomelanosis of the colon, and 1/2

		animals died in 38 days (Graham et al. 1940).
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).
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).
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). [†] 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).
	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 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/m ³ 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/m ³ 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).
	8007-45-2/ 65996-89-6	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

		<p>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 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); epithelial hyperplasia, ulcers and chronic inflammation of the cecum (m/f) (Springer et al. 1986).</p>
	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/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>
	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/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, 2 times/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</p>

		observed (31 weeks as compared to 82 weeks for control animals). Skin tumours also observed (Wallcave et al. 1971).
Developmental and reproductive health effects	8007-45-2/ 65996-89-6	Oral developmental LOAEL = 140 mg/kg-bw/day based on increases in the proportion of litters with anomalous fetuses, and anomalous fetuses per litter. 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).
	8007-45-2/ 65996-89-6	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/m ³ 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).
	8007-45-2/ 65996-89-6	Inhalation reproductive LOAEC (female) = 660 mg/m³ based on increased incidence of mid- and late-gestational resorptions. 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/m ³ 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 maternal body weight also observed at the highest concentration (Springer et al. 1982).
	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).
Carcinogenicity		Oral
	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 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)
	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, 200, 628, 1364 or 2000 mg/kg-bw/day) ^f coal tar in diet, <i>ad libitum</i> , for 2 years. <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). <i>Small intestine adenocarcinomas</i> occurred in 0.6 and 1.0% groups (tumour incidence: 61 and 88%, respectively; 0% observed in controls). Increased mortality and early mortality observed in higher dose groups (Culp et al. 1996; also cited in ATSDR 2002).
	8007-45-2/ 65996-89-6	Effect level = 333 mg/kg-bw/day (0.3%) based on statistically significant and dose-related increased incidences of liver, lung and forestomach tumours.

		<p>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:</p> <p><i>0 mg/kg-bw/day</i>: Range of tumour incidence was 0–4%.</p> <p><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%).</p> <p><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:</p> <p><i>0 mg/kg-bw/day</i>: Range of tumour incidence was 0–4%.</p> <p><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%).</p> <p><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%).</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% group (tumour incidence: 30%).</p>
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		<p>Both Coal Tar Mixtures:</p> <p><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>
		Dermal (chronic)
	8007-45-2/ 65996-89-6	<p>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)^g of coal tar applied to the region just exterior to the ear canal of male Australian albino rabbit (3 animals per dose), 3 times/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).</p>
	8007-45-2/ 65996-89-6	<p>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, 2 times/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.</p> <p>208 mg/kg-bw/day: skin tumour incidence = 6/22 or 27%</p> <p>625 mg/kg-bw/day: skin tumour incidence = 8/26 or 31% (Gorski 1959).</p>
		Dermal (initiation)
	8007-45-2/ 65996-89-6	<p>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/week for 40 weeks. Negative controls not treated with initiator, but subsequently treated with dithranol promoter. Significant increase in mice with papillomas (4/27 treated animals versus 0/28 control animals) (Phillips and Alldrick 1994).</p>
	8007-45-2/	Effect level = 833 mg/kg-bw based on increased

	65996-89-6	incidence of skin tumours. 25 mg (833 mg/kg-bw ^l) 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, 2 times/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).
	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 ^l) 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, 2 times/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).
		Dermal (chronic)
	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), 1 time/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> : 3/40 (8%) mice with papillomas; 0/40 mice with carcinomas. <i>Coal Tar E</i> : 33/40 (83%) mice with papillomas; 4/40 (10%) mice with carcinomas (Robinson et al. 1984).
	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), 2 times/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).
	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), 2 times/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 53/58 or 91.4% (control: 1/26 or 3.8%). Skin tumours observed (31 squamous cell carcinomas, 53 papillomatous growths). Early mortality also observed (Wallcave et al. 1971).
	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), 2 times/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: 48/49 or 98% (malignant: 45/49, 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).
		Dermal (initiation)
	65996-93-2	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

		<p>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/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> 0.2 ml: 4/23 mice with squamous cell tumours; 4/23 mice with squamous cell papillomas; 0/23 mice with squamous cell carcinomas.</p> <p><i>Coal Tar E</i> 0.2 µl: 12/23 mice with squamous cell tumours; 10/23 mice with squamous cell papillomas; 3/23 mice with squamous cell carcinomas. 0.6 µl: 24/26 mice with squamous cell tumours; 13/26 mice with squamous cell papillomas; 20/26 mice with squamous cell carcinomas. 2.0 µl: 14/18 mice with squamous cell tumours; 10/18 mice with squamous cell papillomas; 10/18 mice with squamous cell carcinomas. 6.0 µl: 19/20 mice with squamous cell tumours; 14/20 mice with squamous cell papillomas; 11/20 mice with squamous cell carcinomas. 20.0 µl: 14/14 mice with squamous cell tumours; 10/14 mice with squamous cell papillomas; 7/14 mice with squamous cell carcinomas (Robinson et al. 1984).</p>
		<p>Inhalation</p>
	<p>65996-93-2</p>	<p>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/m³ 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 (0/40, 38/40 and 39/40, respectively), and squamous metaplasia in 6/40 animals at the high-concentration. Statistically significant increases in the incidence of lung adenomas (5/40, 40/40 and 40/40, respectively), lung adenocarcinomas (6/40, 10/40 and 33/40, respectively), and lung squamous cell carcinomas (0/40, 0/40 and 6/40, respectively). In addition, one adenosquamous</p>

		carcinoma was found in the high-concentration group (Schulte et al. 1994).
	65996-93-2	<p>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/ m³ 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.</p> <p><i>10 month exposure</i> 0 mg/m³: 0% 1.1 mg/m³: 4.2% 2.6 mg/m³: 39%</p> <p><i>20 month exposure</i> 0 mg/m³: 0% 1.1 mg/m³: 33% 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>
Genotoxicity – <i>in vivo</i>		Oral
	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> 0% (control): 2.20 ± 0.71 adducts/10⁸ nucleotides 0.01%: 2.85 ± 0.48 adducts/10⁸ nucleotides 0.03%: 5.80 ± 0.70 adducts/10⁸ nucleotides (p < 0.05) 0.1%: 31.2 ± 5.86 adducts/10⁸ nucleotides (p < 0.05) 0.3%: 102 ± 5.86 adducts/10⁸ nucleotides (p < 0.05)</p>

		<p>0.6%: 239 ± 38.8 adducts/10⁸ nucleotides (p < 0.05) 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 0.01%: 6.18 ± 1.60 adducts/10⁸ nucleotides 0.03%: 6.92 ± 1.19 adducts/10⁸ nucleotides 0.1%: 15.1 ± 3.45 adducts/10⁸ nucleotides 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)</p> <p><i>DNA adducts in the forestomach</i></p> <p>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)</p> <p><i>DNA adducts in the small intestine</i></p> <p>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>
	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, 0.03, 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.</p> <p><i>DNA adducts in the lung</i></p> <p>0% (control): 2.20 ± 0.71 adducts/10⁸ nucleotides 0.03%: 8.38 ± 1.08 adducts/10⁸ nucleotides (p < 0.05) 0.1%: 26.2 ± 6.38 adducts/10⁸ nucleotides (p < 0.05) 0.3%: 123 ± 29.8 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 0.03%: 10.2 ± 2.22 adducts/10⁸ nucleotides 0.1%: 17.7 ± 8.41 adducts/10⁸ nucleotides (p < 0.05) 0.3%: 39.9 ± 7.96 adducts/10⁸ nucleotides (p < 0.05)</p>

		<p><i>DNA adducts in the forestomach</i> 0% (control): 6.76 ± 3.00 adducts/10⁸ nucleotides 0.03%: 13.5 ± 3.84 adducts/10⁸ nucleotides 0.1%: 9.98 ± 2.95 adducts/10⁸ nucleotides 0.3%: 25.5 ± 11.0 adducts/10⁸ nucleotides (p < 0.05)</p> <p><i>DNA adducts in the small intestine</i> 0% (control): 0.53 ± 0.81 adducts/10⁸ nucleotides 0.03%, 0.1%: not tested 0.3%: 16.7 ± 23.9 adducts/10⁸ nucleotides (p < 0.05) (Culp et al. 2000).</p>
	8007-45-2/ 65996-89-6	<p>Positive for presence of DNA adducts in the small intestine and forestomach 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> Control: 17 ± 25 fmol adduct/mg DNA 0.01%: 69 ± 32 fmol adduct/mg DNA 0.3%: 461 ± 189 fmol adduct/mg DNA 0.6%: 1438 ± 200 fmol adduct/mg DNA 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>
	8007-45-2/ 65996-89-6	<p>*[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]</p>
		Dermal
	8007-45-2/ 65996-89-6	<p>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-bwⁱ) of crude Manufactured Gas Plant residue-designated Site 4 (MGP-4) applied to 4 cm² of the shaved back for 24 hours. Dose-related DNA adducts detected in the skin and lungs through ³²P-post-labelling. At the highest dose, the relative adduct labelling (RAL) × 10⁹ was 514 in skin. MGP-4 was noted by the authors to cause fewer lung adducts than skin DNA adducts (Cizmas et al. 2004).</p>
	8007-45-2/ 65996-89-6	<p>Positive for presence of DNA adducts in the skin and lung Male Parkes mice (4–6 weeks old at beginning of</p>

		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, 2 times/week (1 st and 4 th days; 3 days apart) for up to 5 weeks. One group of four animals was sacrificed weekly, 24 hours after last treatment (5 th 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).
	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) 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. <i>DNA adducts 24 hours after single dose (assuming background adduct levels subtracted, therefore control value is set to zero)</i> 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 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 (Schoket et al. 1988).
	8007-45-2/ 65996-89-6	Positive for presence of DNA adducts in the skin 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 ⁱ ; 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).
	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]
		Dermal – therapeutic use
	8007-45-2/	Positive for presence of DNA adducts in skin

	65996-89-6	Patients dermally treated with 3–5% coal tar ointments for ≥ 7 days. Increased incidence of DNA adducts from skin biopsies (Zhang et al. 1990).
	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).
	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).
	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).
	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).
	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).
	8007-45-2/ 65996-89-6	*[Similar findings observed in: Godschalk et al. 1998, 2001].
Genotoxicity – <i>in vitro</i>		DNA Adducts
	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).
	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).
	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).
	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,l]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).
	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).
		Mutagenicity – therapeutic coal tar
8007-45-2/ 65996-89-6		Positive in <i>S. 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> <i>Zetar® Emulsion:</i> 7.0 ± 0.6 <i>Estar®:</i> 3.8 ± 0.6 <i>Lavatar:</i> 2.0 ± 0.2 <i>Coal tar solution (USP):</i> 1.4 ± 0.1 (Saperstein and Wheeler 1979; also cited in CIR 2008 and IARC 1985).
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).
		Mutagenicity – crude coal tar
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).
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

		<p>values.</p> <p><i>Mutagenic potencies for TA98 (revertant colonies/μg coal tar)</i></p> <p><i>Kidney donor 1: 3.99</i></p> <p><i>Kidney donor 2: 1.85</i></p> <p><i>Kidney donor 3: 1.40</i></p> <p><i>Kidney donor 4: 0.69</i></p> <p><i>Kidney donor 5: 0.76</i></p> <p><i>Non-induced rat: 7.81</i></p> <p><i>Aroclor-induced rat: 19.5 (Jongeneelen et al. 1988).</i></p>
	8007-45-2/ 65996-89-6	<p>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.</p> <p><i>Mutagenic potencies (revertant colonies/μg coal gasification tar product)</i></p> <p><i>TA98: 6.8</i></p> <p><i>TA100: 6.5</i></p> <p><i>TA1538: 11.2 (Schoeny et al. 1981).</i></p>
	8007-45-2/ 65996-89-6	<p>*[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]</p>
		Mutagenicity – crude coal tar
	65996-89-6	<p>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: 67%; Coal tar F: 47%; Coal tar G: 37%; Coal tar H: 39%</i>) 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).</p>
	65996-89-6	<p>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; Barrett M-30</i>] was tested (Rao et al. 1979; IARC 1985).</p>
	65996-89-6	<p>Positive in <i>S. typhimurium</i> TA1537, TA1538, TA98 and TA100 with S9 exogenous metabolic activation</p>

		(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 Huisingh 1980 and Nesnow and Lewtas 1981; IARC 1985).
	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).
	65996-89-6	*[Similar findings observed in: Kesik and Janik-Spiechowicz 1997]

^a LC₅₀, median lethal concentration; LD₅₀, median lethal dose; LOAEC, lowest-observed-adverse-effect concentration; LOAEL, lowest-observed-adverse-effect level; LOEL, lowest-observed-effect level; NOAEC, no-observed-adverse-effect concentration; 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 ECJRC 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).